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MEDICINE

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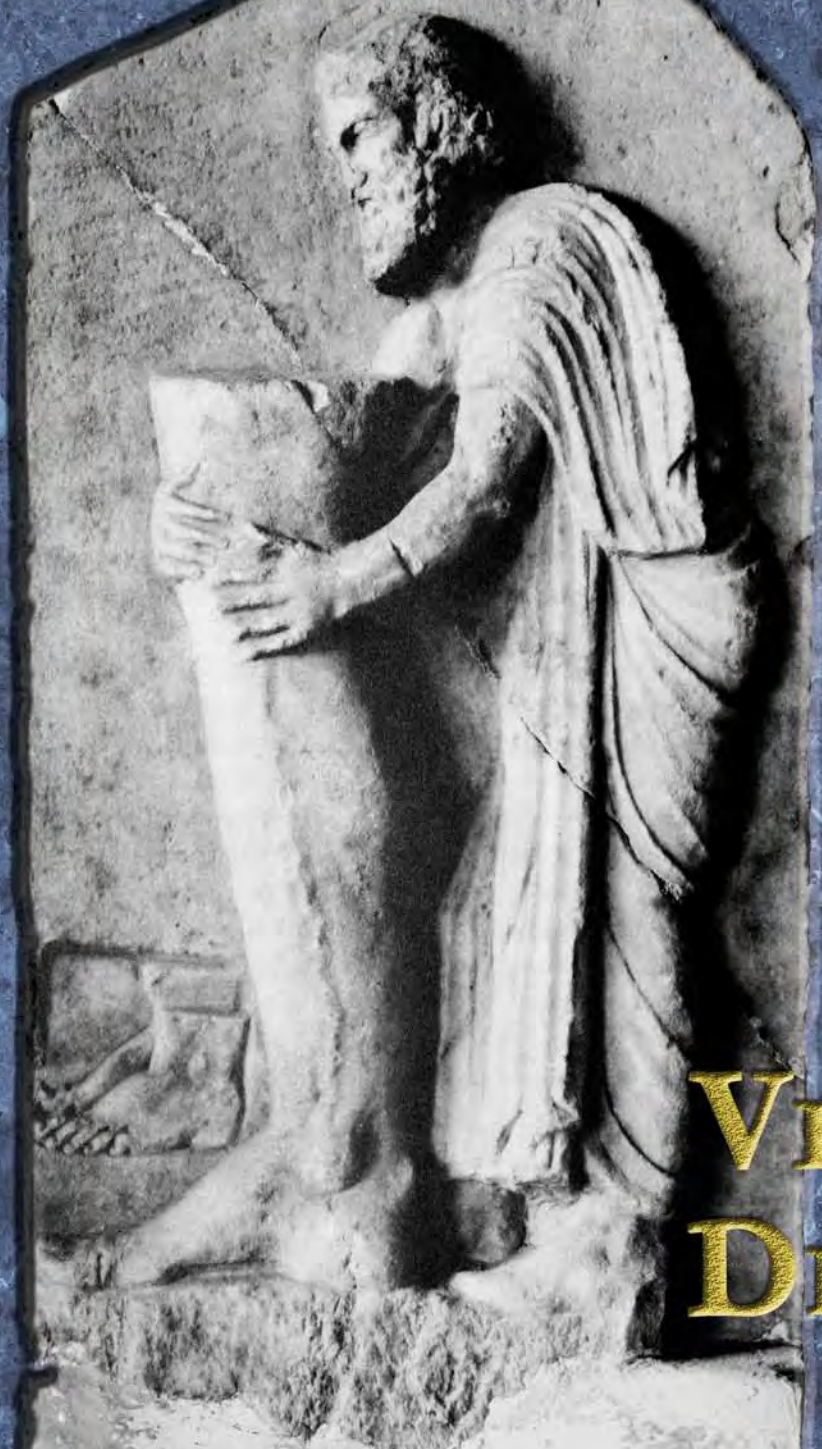

VENOUS DISEASE

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COVER: Votive offering to Greek god of medicine, Asclepius. Sculpture at National Museum of Athens in Greece. Used with permission.

Inside this issue of

Northeast Florida Medicine

Features

Special Articles

Departments

And the Survey Says...

The mission of the Duval County Medical Society (DCMS) is to promote the delivery of and access to high quality, ethical medical care for the community, and to serve as an advocate for physician members and their patients.

The *Northeast Florida Medicine* journal (NEFM) supports the DCMS mission and serves as a corner stone in the value proposition it brings to its members. Letters from the President, Executive Vice President, other leaders in the medical community, and special articles provide important communication to and from the members of DCMS. They highlight all the efforts by DCMS leadership to promote the delivery and access to ethical high quality care and how the DCMS serves as an advocate for patients and physicians. Our peer reviewed educational articles along with free Continued Medical Education to DCMS' members help physicians stay informed of the latest advancements in medicine. Therefore, it supports and promotes the delivery of high quality of care to our community.

Raed Assar, MD, MBA
Editor-in-Chief
Northeast Florida Medicine

In a continuing effort to bring the highest quality to its readership, NEFM conducted a survey in November 2011. DCMS members were asked to take the survey to see if the journal continues to meet their needs. There was a 5% response (97 members). The results indicate we are doing a lot well, but there is room for improvement. I would like to take some time and discuss the results and the actions taken in response.

- Print journal or online? – A print journal was preferred by the majority of respondents (53%) 12% preferred online, and 26% preferred both.
- Value of peer review? – Most felt that the journal is more valuable because it is peer reviewed (69% rated this quality as important or very important).
- Need more color pages? – Most do not want a change in the color per page ratio (82% asked for no change in color ratio).
- Quality of articles? – Scientific articles (71% rated high quality), Editorials 67% rated high quality), CME articles (80% rated high quality), Special Features (62% rated high quality).
- Production quality? – Format and Layout (64% rated high quality), Graphics (64% rated high quality), and Cover (73% rated high quality).
- CME submission? – The majority preferred submitting CME tests online (62%).
- Patient focused content? – One question asked whether or not the journal should have patient focused information to enhance readership in waiting rooms. Forty percent (40%) of the respondents rated this as important or very important, and 37% rated it as somewhat important.

Because of the low response rate, the results are cautiously reassuring. However, it seems that we are on a positive track. In order to make the journal more attractive, we are adding visual enhancements such as summary boxes to highlight important points, improve the ease of reading, and the overall visual attraction of articles. The DCMS Journal and Communication Committee is also considering including a one-page patient-focused "health literacy" sheet or even resource lists with websites, etc. that patients can access. We will take caution to make sure any patient focused health information is accurate and not misleading in any way.

We plan to continue to produce print journals. However, we will need additional sources of funding through advertisement to ensure the financial survival of this valuable publication. Please consider advertising in the journal and ask others to do so. The details are online at dcmsonline.org under "NEFM" and "Advertising" or contact the Managing Editor at llegacy@dcmsonline.org. Our EVP, Bryan Campbell, has considered engaging a local agency that would secure advertising for the journal, however the cost would be less if our own members and local institutions stepped up to support the journal. Besides the print ad, all advertising appears in the online virtual edition and reaches even more people. It would also be possible in the future to poly bag the journal with booklets or brochures that advertisers want to distribute. All options and avenues to fund the journal are being considered.

In conclusion, the results of the survey are reassuring. However, it is likely that future demands will change how the journal continues to meet your expectations. If you have additional suggestions, please contact me or DCMS EVP Bryan Campbell. Feel free to let us know how we are doing as a medical journal for our northeast Florida readership. Our community needs a DCMS that is healthy and strong. One of our strengths is *Northeast Florida Medicine*. Through print and virtual editions, we plan to continue to inform our members and serve our community.

Bullets Dodged During Legislative Session and Beyond

I knew the 2012 Florida Legislative Session would be a challenge, and it was. In a national election year, it can be tough to keep state legislators focused on local political issues. As I have had time to reflect on major issues that were overlooked and key bills that were not passed, I have discussed some of my frustrations with DCMS EVP Bryan Campbell. His upbeat perspective is "We dodged some bullets, and the best thing is that bad things didn't happen."

A real win was the passage of HB 291 sponsored by our own Ronald "Doc" Renuart, DO. Focused on Youth Athletes/Head Trauma, the bill requires the Florida High School Athletic Association and independent youth sanctioning authorities to develop "return to play" policies for athletes who sustain a traumatic head injury.

Ashley Booth Norse, MD
2012 DCMS President

Further, the bill defines the membership of the sports medicine advisory committee of the Florida High School Athletic Association as follows: eight M.D.s or D.Os., one dentist, one podiatrist, one chiropractor, one retired coach, and three athletic trainers. Chiropractors' efforts to amend the bill so that they would be authorized to clear athletes to return to play were unsuccessful.

Also passed this year was PIP reform. This billed banned payments by PIP for massage and limited payments for non-emergency treatment — typically done by chiropractors — to $2,500 unless they can show they are treating an "emergency medical condition" resulting from an accident. The previous max was $10,000. The bill also requires accident victims to seek medical treatment within 72 hours from a licensed M.D., preferably in a hospital emergency room.

As we head into this 2012 Presidential election year, let me encourage all of us to keep politically attune to what is happening, especially as it concerns health care. We will all have the opportunity to voice our views to colleagues, family, friends and maybe even patients. Believe it or not, those groups will listen to our opinions! The battles to repel IPAB (Independent Payment Advisory Board) and fix the SGR are uphill challenges that must be won and we need patients on our side.

Let me thank those at the Florida Medical Association and our own Duval County Medical Society who worked diligently during the 2012 legislative session on behalf of our profession.

More need to get involved. All of us need to come forward and be counted. You might have to dodge a few stones, but when was the last time you were bruised because you took the risk to get in the way of someone's opposing opinion?

Watch DCMS publications and the website (dcmsonline.org) as the national 2012 political climate heats up this summer and into the fall elections. Let me know what you are thinking or write an editorial and submit it. DCMS is your organization. We do want your political views and insights.

Let me close this rather philosophical piece with a statement I made in my inaugural speech when I became your DCMS president. I said, "Medicine in America is at a crossroads and crossroads are challenging. However, it is the challenges, that while daunting, allow us to evolve and thrive."

Well, DCMS is all about "thriving." If we have to dodge bullets, fine. We have done it for 159 years. Why not continue?

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Finding Your Personal Medical Society

I am privileged and honored to continue the tradition of "From the Executive Vice-President's Desk" series of editorials in *Northeast Florida Medicine.* This journal is one of the great benefits of membership in your county medical society, and I hope to use this space to provide you valuable information on important issues, open your eyes to issues which may be on the horizon, and hopefully bring you at least one smile in your day. Since I first occupied the EVP desk for the Duval County Medical Society (DCMS) in February, I have seen such a wide variety of meetings, events and opportunities provided to members by the Society. There truly is something for every physician.

I'll start with this journal. This issue of *Northeast Florida Medicine* provides DCMS members and Clay County subscribers access to free CME credits. That goes for every quarterly issue of the Journal. And now, getting those free CME credits is easier than ever. Members and Clay County subscribers can take the CME post test online (http://dcmsonline.org/CME/cme_DomesticViolenceUpdate2012.html) and send it digitally to be graded. A CME certificate will then be emailed.

Bryan Campbell
DCMS Executive Vice President

AMA Presidential Visit

The reach and the impact of the DCMS have been inspiring for me to witness. On April 16, the Society hosted Dr. Jeremy Lazarus, the 2012-2013 President of the American Medical Association (AMA). I was fortunate to travel with Dr. Lazarus and several members of the DCMS Board of Directors to meetings across the city, culminating with a wonderful membership gala at Epping Forest Yacht Club. Dr. Lazarus spoke of the importance of repealing the broken Sustainable Growth Rate formula for funding Medicare, protecting access to care for patients regardless of the U.S. Supreme Court's decision on the Affordable Care Act, and finally about a new strategic plan for the AMA which will be revealed later this year.

Dr. Lazarus' visit was the latest in a tradition that dates back more than a quarter-century of the AMA President visiting Jacksonville. The tradition started with Dr. Sanford Mullen in the 1980s and continues today. DCMS is one of the only counties in the nation which still hosts the AMA President annually. The DCMS would like to thank FPIC/The Doctor's Company and Florida Blue for their support of this meeting.

Play Ball!

On April 25, DCMS hosted what is becoming a new tradition, a night with the Jacksonville Suns. More than 110 members, their families, and their staff joined together for a night of food, fun and baseball. The group sat right behind first base, filling up the section from the first row until about half-way up the stadium. The fans who stuck through to the end saw the Suns squeak out a win, 6-5, against the Pensacola Wahoos. The MVP of the evening was clearly Dr. Cynthia Anderson, who brought virtually her entire office to the event. "I just thought it was a fun way to give back to the staff members and their families," she said. Very special thanks to our sponsors for this event: American Enterprise Bank of Florida, Associated Medical Office Experts, Haven Hospice, Jacksonville Health Experts, and Waddell and Reed.

Around the First Coast

As many of you know, I am not just the Executive Vice-President to the Duval County Medical Society, I also serve as the EVP for the Medical Societies in Nassau, St. Johns, Clay and Putnam counties. Each of these counties has a rich and unique physician community and I have been lucky enough to experience a variety of events and meetings. From CME presentations to information on protecting your assets in from malpractice lawsuits, there is valuable education for every medical professional.

One of the most exciting events was the Annual Clay County Medical Society Retreat and Golf Tournament on May 5 at the Orange Park Country Club. I am not much of a golfer myself, (just ask the doctor who played with me), but I truly enjoyed the opportunity to spend a day with the leaders of the Clay County medical community. Congratulations to Drs. Neel Karnani, Jeffrey Harris, and Gene McCoskey, as well as yours truly, for winning the team title at the event. Ron Christopher won the Longest Drive Contest, and Doug Deen won the Closest to the Hole Challenge. Many thanks to all of our wonderful sponsors for this event.

What do YOU Want Your Medical Society to Be?

These programs are just a few of the great ideas that your County Medical Society can give back to you and your practice. It is important to thank our supporters, because without them, many of these programs would just not be possible. So now it's your turn. What more do you want from your medical society. What education can we provide? What networking opportunities can we create for you? The only limit to what we can do, is what we can envision. Please take 30 seconds to send me a personal email. (bcampbell@dcmsonline.org) Tell me what your favorite part of membership in the Medical Society is. Tell me what you'd like to see us focus on in the future. Together, we can make your ideas a reality that can help your fellow doctors across Northeast Florida.

Editor's Note: In an effort to connect more Duval County Medical Society members with residents, in each 2012 issue there will be a "Residents' Corner" with information about a residency program in the area, details about research being done and/or a list of achievements/accomplishments of the program's residents. This "Residents' Corner" features the University of Florida's Oral & Maxillofacial Surgery Residency Program.

Overview of Residency Program

The University of Florida (UF) Division of Oral and Maxillofacial Surgery provides an extensive array of specialized medical services at Shands Jacksonville and has a fully accredited residency program in oral and maxillofacial surgery and a microvascular surgery fellowship under the guidance of the division's faculty. The division is comprised of nationally recognized experts with specialty and subspecialty training and certification. The division's UF faculty physicians offer the entire spectrum of contemporary maxillofacial surgery: a comprehensive facial trauma and reconstruction practice, pediatric craniomaxillofacial surgery, facial aesthetic surgery, TMJ and corrective jaw surgery, head and neck oncology and microvascular rehabilitation and reconstruction of head and neck defects.

Medical services provided within Oral and Maxillofacial Surgery are dental implants, dento-alveolar surgery, oral surgery, orthgnathic surgery (corrective jaw), removal of wisdom teeth and temporomandibular joint (TMJ) surgery. In Maxillofacial Trauma Surgery there is repair of facial features and soft tissue injuries of the head and neck, and secondary reconstruction of trauma patients. The Cranio-Maxillofacial Surgery (Pediatric) performs cleft lip and palate surgery, craniofacial distraction osteogenesis, cranio-synostosis surgery (skull reshaping), and maxillofacial and craniofacial surgery. Facial Aesthetic Surgery consists of brow and forehead lift, Cervicoplasty (neck lift), cheek and chin implants, laser resurfacing, Otoplasty (ear surgery), Rhinoplasty (nasal surgery) and Rhytidectomy (face lift). Finally, oncology (head and neck cancer) deals with cysts and tumors of the jaw, head and neck pathology, head and neck reconstruction and skin cancers of the head and neck.

Miscellaneous Services offered are for conscious sedation, microvascular reconstruction and sleep apnea.

Research

The following research is taking place:

1. "Novel Uses of Resorbable Anchoring Pins in Maxillofacial Surgery" - Drs. J. Thakker, T. Fattahi, A. Adamec and B. Steinberg. Submitted for publication to JOMS.
2. "Evaluation of Reconstructive Techniques for Anterior Skill Base Defects Following Tumor Ablation" - Drs. J. Thakker, R. Fernandes. Presenting at 2012 UF Research Day and AAOMS Meeting (MORS)
3. "Diagnostic Value of Ultrasound vs. MRI and Arthoscopy in the Management of Temporomandibular Joint Internal Derangements" - Drs. J. Thakker, G. Kaeley, L. Vega. In progress.
4. "The Need for Resorbable Plates in Repair of Craniosynostosis: A Case of Endocranial Migration of Titanium Hardware and Review of Literature" - Dr. J. Thakker, B Steinberg. Submitted for publication to JOMS.

Additional research is being done in Zygomatic Implants, TMJ Arthorscopy Outcomes and Endoscopic Procedures in Maxillocacial Surgery.

Awards and Honors

Exemplary Teacher Awards to Dr. Tirbod Fattahi, Dr. Rui Fernandes Dr. Luis Vega, Dr. Howard Schare, Dr. Nelson Goldman, Dr. Barry Steinberg, Dr. Philip Pirgousis. (all are faculty)

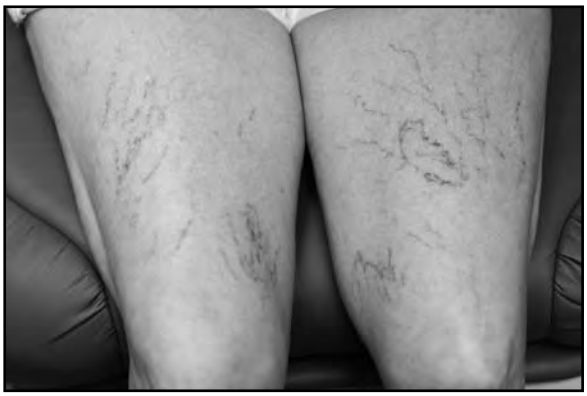
Additional honors go to Dr. Rui Fernandes who was elected chair of the Educational Committee for International Oral and Maxillofacial Surgery Society and to Dr. Luis Vega who received the Faculty Educational Development Award (given annually to exemplary young academicians).

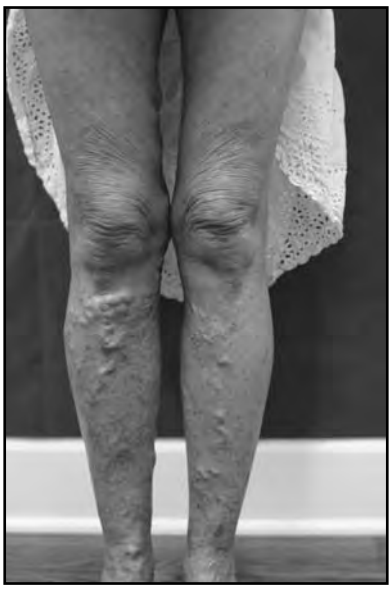
Bouali Amoli graduated of the University of Kentucky with an undergraduate degree in Philosophy and Biology. In 2002 he received a dental degree (DMD) and practiced as a dentist for two years before entering medical school in 2005 and completing his MD in 2009 at the University of Kentucky. He is currently a PGY3 in a 4-year track in oral and maxillofacial surgery at the University of Florida. In July he will do a one-year clinical fellowship in Tulsa, OK in general cosmetic surgery. He plans to return to Jacksonville to reside and practice medicine.

What do you know about Varicose Veins?

Statistics

- More than 30 million Americans suffer from chronic vein disease.
- Only 1.9 million of those seek treatment annually while the remaining go undiagnosed and untreated.
- They are not just a cosmetic problem. Varicose Veins are caused by chronic venous insufficiency (CVI).
- Heredity is the number one contributing factor that causes varicose and spider vein. While pregnancy, menopause and other hormonal factors put women at a higher risk, men can be affected as well. Some other predisposing factors include aging, standing occupations, obesity and leg injury/surgery.





Smaller varicose veins (above) and bulging veins (left)

Signs & Symptoms of Varicose Veins

(For further information, please consult your personal physician. For a list of physicians, you can go to the "DCMS Physician Directory" at http://db.dcmsonline.org/directory/. Find more information at http://veinforum.org/patients.aspx.)

- **Varicose and/or spider veins**
- **Swelling of the ankles**
- **Leg fatigue**
- **Leg pain, aching or cramping**
- **Burning, itching, tingling or numbing of the skin on the legs**
- **Leg ulcers, open wounds or sores**
- **Skin discoloration**

Complications

Varicose veins can lead to complications such as blood clots, bleeding, rashes and ulceration.

Treatment

- **Two general types of treatment options: conservative measures, such as compression stockings and leg elevation; and corrective measures, such as endovenous thermal ablation, chemical ablation, surgery and sclerotherapy.**
- **Technological advances in treatment methods allow spider and varicose veins to be treated more effectively and safely than ever before.**
- **Successful treatment depends on two things: careful assessment of the problem, and the skill of the physician providing the treatment.**

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Venous Disease: Misunderstood, Misdiagnosed & Mistreated

"It is ironic that medical education does not cover three of the most common medical problems: back pain, hemorrhoids, and varicose veins." *P. Fujimura, MD, Surgical Intern, University of California School of Medicine*

Dr. Fujimura is right. Venous disease is often misunderstood, misdiagnosed and therefore mistreated even though it is among the most common medical conditions to affect mankind. Approximately 1-3% of the population of the Western world is estimated to have a severe venous problem at some point in their lives. Although it is generally acknowledged that venous disorders have an enormous socioeconomic impact, the diagnosis and treatment of such conditions have suffered from a lack of interest and support, especially in academic centers.

James St. George, MD
Medical Director/CEO
St. Johns Vein Center

Ironically, venous disorders have affected patients for thousands of years. An early method of treating varicose veins consisted of making offerings to the gods for help. The cover of this issue displays one such offering from 300 BC. According to an inscription found on the West side of the Acropolis, this was a votive offering to the Greek god of medicine, Asclepius. It was commissioned by Lysimachidis of Archarnes, who was probably suffering from varicose veins, and dedicated to Dr. Amynos, a local physician.

The first description of medical treatment appears in the writings of Hippocrates in the 4th century BC. He describes treating varicose veins by traumatizing them with "a slender instrument of iron" to cause thrombosis. Celsus (30 BC to AD 30) practiced stripping and cauterization.

Plutarch described the first varicectomy on the Roman Consul Gaius Marius (157-86 BC.) He recorded, "For having, as it seems, both his legs full of great tumours, and disliking the deformity, he determined to put himself into the hands of an operator, when, without being tied, he stretched out one of his legs, and slightly, without changing countenance, endured most excessive torments in the cutting, never either flinching or complaining; but when the surgeon went to the other, he declined to have it done, saying, 'I can see the cure is not worth the pain."

Until recently, surgical stripping of incompetent leg veins remained the primary treatment for incompetent leg veins and, like poor Gaius Marius, many patients felt that the treatment was worse than the disease. Fortunately, many recent advances in the field of Phlebology have revolutionized the treatment of venous disorders.

Since venous disorders frequently start with acute thrombosis, in "Endovascular Management of Iliofemoral Deep Vein Thrombosis," **David M. Sella, MD,** and **Gregory Frey MD,** review the technique, benefits, and medical evidence of catheter-directed thrombolysis for the treatment of DVT. **Shannon L. Beardsley, MD,** reviews the pathophysiology, diagnosis and treatment of Pelvic Congestion Syndrome in "Endovascular Therapy for Pelvic Congestion Syndrome: Making the Diagnosis and Getting the Patient Treated." This syndrome may affect as many as 17,000 women in Duval County alone. Chronic venous disease of the lower extremity has a tremendous impact on the health and quality of life for a large number of people so in "Evaluation and Treatment of Lower Extremity Superficial Venous Insufficiency," I explain the tools necessary for practitioners to help their patients with this condition. This is also the CME article.

David S. Ross, MD, addresses the evaluation and treatment of venous ulceration in "Venous Stasis Ulcers: A Review," since 50% of ulcers remain unhealed for over a year due to inadequate or incomplete treatment. **Rickie Sander, MD, FCAP,** provides an overview of primary and secondary lymphedema, which are difficult to diagnose and manage, in "Lymphedema-Pervasive and Chronic, but Treatable" An Overview." A mainstay treatment for venous insufficiency, venous ulceration and lymphedema is compression therapy. **Sonya M. Casey, RVS,** and I provide a brief primer for the prescribing physician with "An Introduction to Compression Therapy." Congenital venous anomalies may be disfiguring and disabling for both the pediatric and the adult patient. **Ricardo Paz-Fumagalli, MD,** addresses congenital venous anomalies in "Venous Malformations: Basic Concepts and Interventional Radiologic Management."

A condition dating from 300 BC and written about by Hippocrates and Plutarch, should be better understood today. It is my hope that this issue provides some tools to physicians so they can dramatically improve modern day patients' quality of life and venous health.

Endovascular Management of Iliofemoral Deep Vein Thrombosis

David M. Sella, MD and Gregory Frey, MD

Abstract *Acute deep venous thrombosis (DVT) is a common entity encountered in clinical practice with an overall lifetime risk of between 2-5% in the population. In the majority of cases, conventional therapy with anticoagulation remains the gold standard of treatment. Certain cases of DVT may require additional therapy. Patient evaluation is paramount to this process. The decision to attempt endovascular therapy is primarily based on location of thrombus and symptoms related to the DVT. DVT associated with arterial compromise may lead to ulceration, PE or death and should be treated emergently. A secondary goal of endovascular therapy is to reduce the incidence of post-thrombotic syndrome (PTS), which can significantly impact quality of life. For iliofemoral DVT 1 year patency rates following endovascular therapy are 63-90% for the iliac vein and 40-47% for the femoral vein. Data regarding incidence of PTS following catheter-directed thrombolysis (CDT) is suggestive of benefit, but randomized data is scant. The NIH funded ATTRACT trial, which is currently underway, is designed to evaluate this relationship.*

Introduction

Acute deep venous thrombosis (DVT) is a common entity encountered in clinical practice with an overall lifetime risk of between 2-5% in the population.[1] The primary impetus for DVT therapy over the last 50 years has been the prevention of pulmonary embolism using anticoagulant therapy.[2] Although effective, anticoagulants do not actively eliminate venous thrombus and therefore are not always sufficient at preventing DVT complications.[3] Despite the appropriate use of anticoagulant therapy, 25-50% of proximal DVT patients develop significant quality of life impairment from post-thrombotic syndrome (PTS).[4] The symptoms of PTS usually take months to years to develop after the acute DVT. Early signs include chronic leg fatigue/heaviness, paresthesias, aching, and swelling (worse with standing and improved with leg elevation). Late changes include skin thickening, stasis dermatitis, and erythema, characteristically just above the medial malleolus. In severe cases ulceration may develop.[5] The strongest predictor for PTS is recurrent DVT. Other factors include obesity, gender, and thrombophilic states. PTS is more likely to occur when recanalization of the vein is delayed or slow, rethrombosis occurs, or reflux into the popliteal or saphenous vein ensue.[5]

Location, Location, Location

Isolated iliofemoral DVT by definition includes thrombus found in the common femoral and iliac veins. It accounts for 20% of lower extremity DVT.[6] Specific etiologies of iliofemoral DVT include propagation of femoropopliteal thrombus, pelvic mass, and May-Thurner syndrome. Another name for the latter is left iliac vein compression syndrome, which is a condition caused by anatomic constriction of the left common iliac vein by the right common iliac artery. *(Figure 1,p.14)*

Iliofemoral DVT is unique in that it is relatively resistant to conventional anticoagulation, rarely completely recanalizes, and more frequently results in post-thrombotic syndrome.[7] Chronically diseased veins are found in over one-half of patients with acute iliofemoral DVT. This number is significantly higher than found in femoropopliteal or isolated calf vein thrombosis.[8] Rarely, extensive peripheral and iliofemoral DVT cause obstruction of all venous collateral channels blocking nearly all the venous return from the leg. The patient then may suffer from an extremely swollen, cyanotic and painful extremity, sometimes with compromise of arterial inflow (phlegmasia cerulea dolens). This extremely serious condition may lead to venous gangrene, massive PE and death. Phlegmasia cerulean dolens is a medical emergency that requires rapid treatment.[9]

Catheter-Directed Iliofemoral Venous Thrombolysis

Fortunately, DVT does not always embolize. As the clot organizes it becomes attached to the vein wall over several weeks. The process of endogenous fibrinolysis then occurs, beginning at the center of the lumen. If the clot resolves entirely, the vein wall remains structurally intact with functioning valves. However, if clot resolution is incomplete, residual thrombus along the vein wall leads to wall thickening, an irregular intraluminal channel, and subsequent incompetence of the deep and perforating vein valves. Multiple studies have demonstrated that preservation of valve competence is a function of time to thrombus resolution.[10-12] Some literature shows that despite optimal therapy including anticoagulation, over 25% of individuals with a proximal leg DVT will progress to post-thrombotic syndrome. The main feature of PTS is ambulatory venous hypertension.[5]

The development of PTS is directly related to the continued presence of thrombus within the deep venous system during the initial weeks and months after an acute DVT. This occurs via two primary pathways: (1) residual thrombus physically obstructs blood flow/venous return and (2) the thrombotic state stimulates inflammation which directly damages the venous valves, leading to valvular incompetence. For this reason, rapid thrombus elimination and restoration

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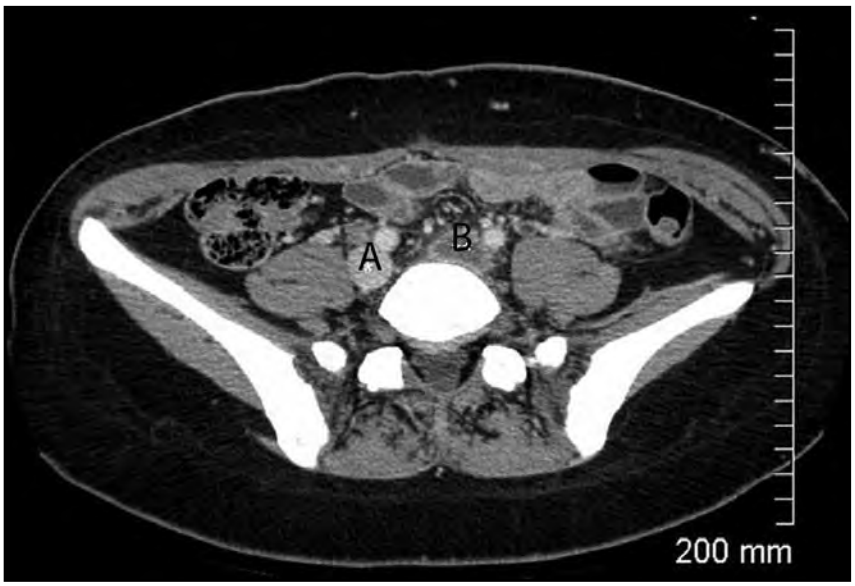
Figure 1 Contrast Enhanced Abdominal CT



This contrast enhanced abdominal CT demonstrated a patent right iliac vein (A) and acute thrombus in the left iliac vein (B) in the setting of May-Thurner's disease.

of unobstructed deep venous flow using catheter directed thrombolysis should rapidly improve initial DVT symptoms and prevent late valvular reflux, venous obstruction and PTS. Systemic thrombolytic infusion has been utilized, but has low effectiveness. Furthermore, bleeding complications are more frequent compared to catheter-directed thrombolysis.[3]

Catheter Directed Thrombolysis

It is important to remember that the benefits of Catheter Directed Thrombolysis (CDT) have not been evaluated in randomized controlled trials relative to the associated risks, inconveniences and costs.[3] CDT is sometimes classified into 3 basic categories: Urgent first-line CDT, Non-urgent second-line CDT, and Non-urgent first-line CDT.[3]

Urgent first-line CDT is used in adjunct with anticoagulation to prevent life, organ, or limb-threatening circulatory compromise. This includes (i) acute limb-threatening circulatory compromise, (ii) extensive IVC thrombosis placing a patient at high risk for fatal PE and (iii) acute renal failure from thrombus extension into the suprarenal IVC/renal veins.

Nonurgent second-line CDT may be performed in patients with symptomatic proximal DVT demonstrating anatomic or clinical progression on anticoagulation. This includes rapid iliocaval thrombus extension, persistence or exacerbation of lower-extremity symptoms, and failure to experience sufficient symptom relief to allow ambulation.[3]

Nonurgent first-line CDT is used as an adjunct to anticoagulation to provide faster symptom relief and/or prevention of post-thrombotic syndrome. This includes functional patients with acute (<14-21 days) iliofemoral deep venous thrombosis.

CDT is considered, if symptoms and/or clinical manifestations merit aggressive therapy. The patient must be evaluated for the risk of bleeding complications, ambulatory status and life-expectancy. Venous Duplex US of the lower extremity veins is utilized to evaluate the extent of thrombus and plan treatment. Evaluation of the iliac veins with CT scan or magnetic resonance may be necessary. Preprocedure laboratory evaluation should include serum creatinine, hemoglobin/hematocrit, platelet count, international normalized ratio (INR), partial thromboplastin time, and pregnancy test in women of childbearing potential.

As with most endovascular procedures, there are multiple techniques, devices, and personal biases. Conscious sedation is generally utilized with dedicated monitoring by a nurse trained in sedation techniques. Some operators may place a prophylactic IVC filter before thrombolysis if the clinical consequence of even a small embolus is significant or if there is a large thrombus burden in the IVC. Pulmonary embolism rates are low with pharmacological (thrombolytic only) CDT, and IVC filters are probably not necessary. A retrievable IVC filter may be placed based on operator preference, particularly if pharmacomechanical CDT (combined thrombolysis and thrombectomy) techniques are used, as PE risk in this group is not well established.[3]

Venous access is typically obtained via the ipsilateral popliteal vein, utilizing ultrasound guidance with the patient in the prone position. The venous occlusion is traversed with a guidewire and contrast venography is performed to determine the extent of the thrombus. *(Figure 2, a-d, p.15)* Multiple techniques are employed to speed the resolution of clot. The most common involves establishing an overnight infusion of a thrombolytic agent such as tissue plasminogen activator (tPA) via a multisidehole catheter at about 1 mg/hr. The catheter is designed to deliver the medication within the thrombosed venous segment at multiple levels (intra-thrombic infusion).

As with other thrombolytic procedures, the patient will require overnight admission to an intensive care unit for observation. The patient is maintained in a recumbent position with the leg elevated during the infusion. The patient is

Figure 1 Contrast Venography

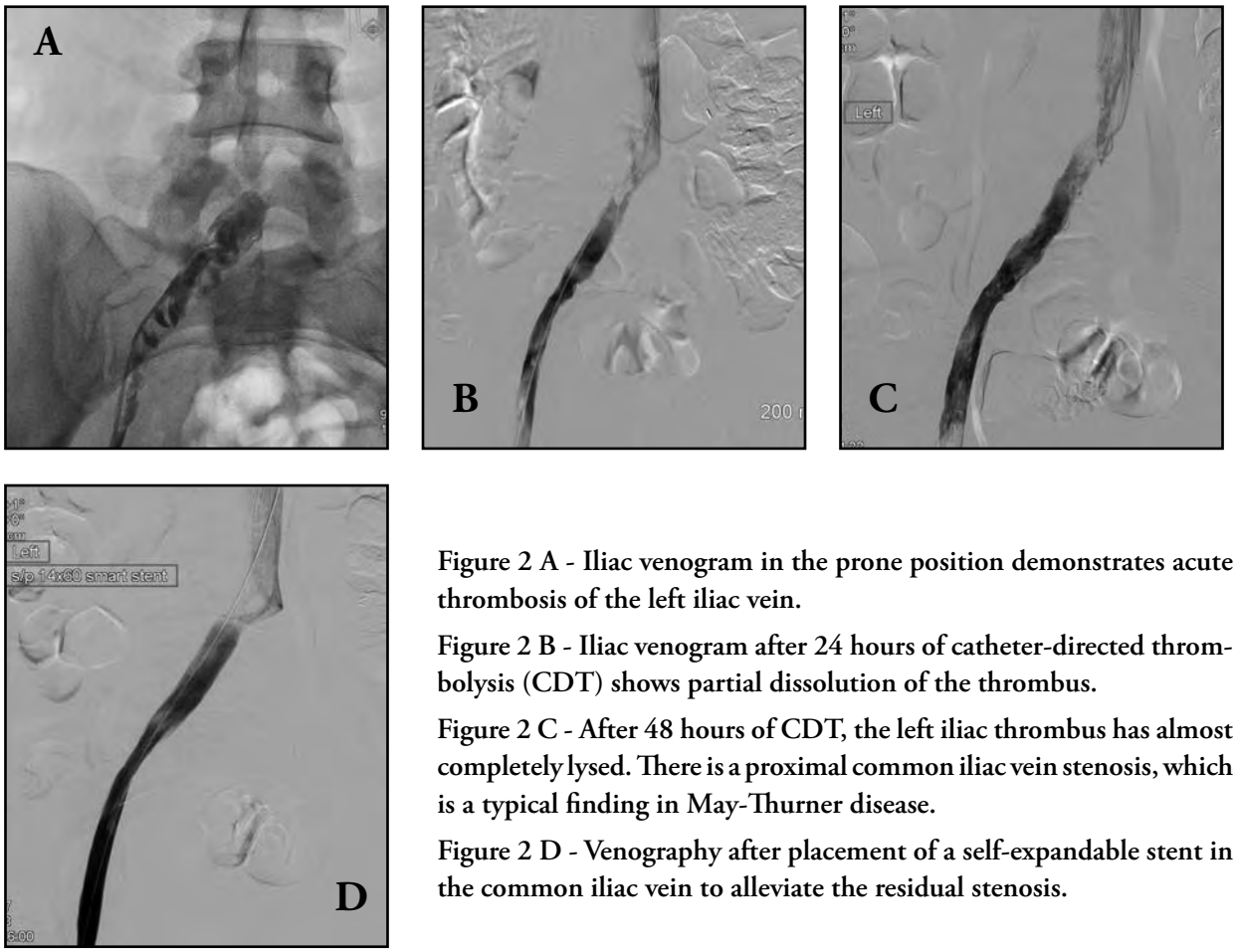


Figure 2 A - Iliac venogram in the prone position demonstrates acute thrombosis of the left iliac vein.

Figure 2 B - Iliac venogram after 24 hours of catheter-directed thrombolysis (CDT) shows partial dissolution of the thrombus.

Figure 2 C - After 48 hours of CDT, the left iliac thrombus has almost completely lysed. There is a proximal common iliac vein stenosis, which is a typical finding in May-Thurner disease.

Figure 2 D - Venography after placement of a self-expandable stent in the common iliac vein to alleviate the residual stenosis.

allowed a clear liquid diet as the thrombolysis may last 24-48 hours with serial follow-up venograms to assess response. Serial laboratory studies are drawn to assess for developing coagulopathies and subsequent bleeding risk. [3]

There are a variety of devices and techniques utilized to shorten thrombolysis time. These include ultrasound-assisted thrombolysis with the EKOS catheter (EKOS Corporation, Bothell, WA), the Trellis infusion system (Bacchus Vascular, Santa Clara, CA), Trerotola thrombectomy device (Arrow/Teleflex Medical, Research Triangle Park, NC), and Angiojet mechanical thrombectomy (Medrad, Inc., Warrendale, PA.) These all aim to achieve rapid thrombectomy at a single session, particularly in individuals at high risk for bleeding.

For the most part, mechanical thrombectomy alone is not successful and infusion of a thrombolytic is necessary. The earlier mentioned thrombectomy devices are used to initially reduce the thrombus burden and "jumpstart" the thrombolyis. Alternatively, they may be employed after tPA administration to eliminate residual thrombus. After the bulk of thrombus has been removed, balloon angioplasty or self-expanding stent placement may be performed to treat residual disease and underlying stenoses. At completion, the venous sheath is removed and hemostasis is achieved utilizing manual pressure. The patient then undergoes a short period of bedrest with the treated leg immobile and elevated for approximately 4 hours. Early ambulation is desirable.[3]

Therapeutic anticoagulation is resumed immediately after hemostasis with either unfractionated heparin or low molecular weight heparin (LMWH). Warfarin is started immediately with a target INR of 2 to 3. In patients with cancer-related DVT, LMWH monotherapy is preferred. The patient is generally placed on aggressive anticoagulation for at least 6 months and sometimes indefinitely based upon the associated risk factors and etiology of the thrombus. The retrievable IVC filter may be removed at any time after the thrombolysis is completed. It is not necessary to stop anticoagulation for filter retrieval unless the INR is highly supratherapeutic. A venogram is performed prior to removal to assess for thrombus within the filter. Knee-high compressive elastic stockings (30-40 mmHg) are also strongly recommended. Daily use of compression therapy has been shown to reduce the rate of PTS by up to 50%.[8] One month follow-up is generally performed along with appropriate monitoring of the patient's INR.[3]

Results – What We Know So Far

Endovascular therapies have largely replaced open thrombectomy procedures except in rare cases of venous gangrene. Technical success is reported in 80-95% of cases in which

the thrombosis is acute.[5] Stand-alone infusion-first CDT, which involves overnight infusion of thrombolytics, dissolved acute thrombus in 80% of patients with acute DVT in a 473 patient prospective multicenter registry and in a pooled analysis of 19 observational studies comprising over a thousand patients.[10,11] To date there are several studies that suggest CDT can prevent post-thrombotic syndrome and improve health-related quality of life in patients with acute iliofemoral DVT. Unfortunately these studies had serious design flaws due to single center performance, small sample size, and non-randomized design. One must note that there are equally no comparison studies arguing against the benefits of CDT. One could certainly maintain that patients with acute iliofemoral DVT be routinely considered for CDT for PTS prevention and at least be educated on the potential benefits.[3]

The risks of CDT are a necessary part of the discussion. Major complications of bleeding occurred in 11% of cases according to the international venous registry; however, the majority were access site complications.[10,11] It is difficult to extrapolate this data to today's techniques as the majority of venous access today is performed with ultrasound-guidance and the doses of thrombolytics are generally lower. The most feared complication is intracranial hemorrhage, which occurred in 0.4% of patients in the venous registry. Symptomatic PE occurred in 1.3% of patients and fatal PE in 0.2%.[14]

Primary patency rates at 1 year vary from 63-90% for the iliac vein and 40-47% for the femoral vein.[5] The majority of data on CDT is derived from non-randomized trials. Therefore the actual risk-benefit ratio of endovascular DVT therapy in preventing PTS is not clear. The recently reported CaVenT trial is the first randomized controlled trial to evaluate the efficacy of CDT in patients with DVT. Thrombolysis reduced PTS compared to anticoagulation and elastic compression stockings alone, with a relatively small risk of additional bleeding. The risk of PTS was reduced by 15% at 24 months and the iliofemoral patency was increased for the CDT group at 6 months. Both findings were statistically significant.[12] In 2009, the National Institutes of Health launched a multidisciplinary multicenter randomized clinical study "Acute Venous Thrombosis: Thrombus Removal With Adjunctive Catheter-Directed Thrombolysis (ATTRACT)." The purpose of this study is to determine if adjunctive pharmacomechanical catheter directed thrombolysis can prevent PTS in patients with symptomatic proximal DVT as compared with optimal standard DVT therapy alone. To date the study is not complete and results are pending.

Summary

Acute DVT remains a common entity in daily clinical practice. Although anticoagulant therapy remains the standard of care, it is insufficient in many cases at preventing serious DVT complications. PTS remains a potentially debilitating complication. The endovascular community is working diligently to provide additional evidence to help determine the long-term benefit of CDT.

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Endovascular Therapy for Pelvic Congestion Syndrome: Making the Diagnosis and Getting the Patient Treated

Shannon L. Beardsley, MD

Abstract: *Pelvic congestion syndrome (PCS) is an often misunderstood, misdiagnosed and therefore mistreated condition which may be responsible for chronic pelvic pain in as many as 17,000 women living in Duval county today. For accurate diagnosis and treatment, it is important that those involved with delivery of healthcare to women be aware of the disease itself, understand its pathophysiology, know how the diagnosis is made, and refer the diagnosed patient for appropriate consultation and therapy.*

Chronic Pelvic Pain

Chronic pelvic pain (CPP) can be defined as pelvic pain occurring over at least a 6 month period of time and can include deep pelvic pain of a noncyclical nature, dyspareunia, and dysmenorrhea. The prevalence of CPP is as high as 15% in women between the ages of 18 and 50 years.[1] When a woman makes an outpatient appointment with her gynecologist, the purpose of the appointment is to ask for help with the diagnosis and management of CPP in 10% to 40%.[1] These numbers are staggering when one looks at the population as a whole with over 10.5 million women affected in the United States (based on 2010 US Census Bureau Statistics).

The high prevalence and nature of the problem translates into a significant economic burden to society as well with an estimated $14 billion of lost productivity each year due to 15% of affected women missing an average of 14.8 hours of work per month. Including such hidden costs as potentially unnecessary medical, surgical, and psychiatric care or hospitalization, it is estimated that the total cost of care for women with CPP reaches $39 billion per year.[2,3]

A World Health Organization (WHO) systematic review of prevalence of CPP found it to be the single most common indication for referral to women's health services. Unfortunately, despite the relatively high prevalence, significant personal burden and public economic toll, the underlying causes for CPP still remain overlooked and under diagnosed gynecologic conditions.[4]

Pelvic Congestion Syndrome

PCS is one possible cause of chronic pelvic pain and has been defined as a condition characterized by visible congestion of the pelvic veins on selective ovarian venography in multiparous, premenopausal women with a history of chronic pelvic pain for more than 6 months.[5] Other conditions responsible range from gastrointestinal tract problems to other gynecological diseases and urologic abnormalities.[1]

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In 1857, Richet first described the collection of symptoms in his description of a patient who had a "tubo-ovarian varicocele." Then, in 1949 and the following decade, Dr. Howard Taylor described the syndrome and the diagnosis gained some credibility. This early progress was thwarted, however, due to many physicians focusing on what they believed to be a strong psychosexual component of the illness along with the fact that stress usually resulted in aggravation of the pain. Conceptualizing this syndrome within a framework of mental health disease or as a psychosomatic illness meant that its evaluation and management was largely left in hands of the psychiatric community.

Little progress was made in understanding this disease in the ensuing years. Fortunately in the 1970's and continuing to today, Dr. Beard's group at St. Mary's Hospital in London began to systematically address the clinical characteristics, methods and criteria for diagnosis of the condition, while maintaining a respect for the psychological components.[6]

Signs and symptoms of PCS typically occur after pregnancy. The pain can become more intense either before or during menses and, similar to varicocele in men, is increased with those activities which generally increase venous congestion within the pelvis, including prolonged standing, fatigue and coitus.[7]

PCS as a cause of chronic pelvic pain has certain classifications.[8] They are:

Anatomic Dysfunction – It is well known pelvic congestion syndrome and pelvic varicocities are associated with venous reflux and flow reversal in the left ovarian vein. To understand various anatomic factors, a quick review of pelvic venous anatomy is provided. The ovaries, supported within the broad ligament extending from the uterus to its lateral support on the pelvic sidewall, drain cranially via the internal ovarian veins and caudally via the peri-uterine venous plexus. The left ovarian vein inserts into the left renal artery and the right ovarian vein inserts into the inferior vena cava directly, caudal to the right renal vein insertion. Crossing from side to side within the uterus are the arcuate veins. Finally, the periuterine venous plexus itself drains via multiple venous pathways but mainly into branches of the internal iliac venous system. Also contributing to the overall network of veins within this pelvic system are labial and vaginal drainage branches.

Pelvic varicosities are most commonly associated with venous reflux and flow reversal in the left ovarian vein. Pregnancy results in significant increase in pelvic venous flow and it is likely that dilation of the ovarian veins during pregnancy is a likely cause of subsequent venous incompetence.[9]

Koc Z studied right ovarian vein drainage variants in a retrospective study to try to uncover an association with pelvic varicosities.[10] They looked at routine abdominal

multidetector-row computed tomography studies for 324 women and found thirty-two (9.9%) with right ovarian vein variants that drained directly into the right renal vein. The remainder of women had right ovarian vein drainage into the inferior vena cava. Pelvic varicosities were found in 59 women and 97% of these also exhibited ovarian vein reflux. Despite the nearly 10% incidence of right ovarian vein variants, only a single woman had reflux predominantly in the right ovarian vein. They concluded there was no association between these right ovarian vein variants and pelvic varicosities.

In an intriguing theory, PCS may actually be just one small part of a wider complex of symptoms resulting from compression of the left renal vein. Pain and function disturbances may be seen in other organs or systems that may attributed to results of renal venous outflow congestion including the vertebral column, skull, brain, spinal medullae, prostate, urinary bladder, rectum, vagina and urethra. Scholbach used the term midline congestion syndrome to describe this collection of etiologically linked symptoms and went on to suggest possible links to chronic back pain, frequent cystitis, enuresis, abdominal pain, flank pain, placental insufficiency, prostate disease and myelopathie.[11]

Orgasmic Dysfunction – Vasocongestion of the pelvic viscera can occur when a female is stimulated up to, but not reaching, orgasm (ie the plateau phase of female sexual response). Associated with this vasocongestion may be pain localized to the pelvic viscera.[8] Although this is known to occur, no studies have looked at whether this can produce permanent vascular changes.

Psychosomatic Dysfunction – When considering psychosomatic conditions as an etiology, one is reminded of the logic dilemma of "the chicken and the egg." And so it is no surprise that despite previous conclusions regarding psychiatric disturbances being associated with pelvic congestion syndrome, there are no clear studies pointing to a causal relationship with pelvic congestion and associated pain as the effect and psychiatric condition as the cause. However, numerous studies looking at chronic pain have found the reverse to be true, namely that chronic pain of a variety of etiologies may result in chronic distress and associated psychological consequences.[8]

Hormonal Dysfunction – Women with PCS have an increased incidence of multi-cystic ovaries, uterine enlargement, and endometrial stripe thickness, all of which can be causally related to hormonal stimulation. Due to almost complete absence of the diagnosis in post-menopausal women, Taylor in 1949 suggested the condition (then tubo-ovarian varicocele) may be related to hormonal insensitivity.[12]

More recently, Foong and Beard used tilt table testing to measure peripheral vascular reactivity during the menstrual cycle among women suffering chronic pelvic pain from congestion and a control group of pain-free women matched for age.[13] They found the congestion group did indeed show abnormal peripheral vascular reactivity which returned to normal after suppression of ovarian activity with medroxyprogesterone acetate, suppression of pituitary LH production with leuprorelin, or oopherectomy. Additionally, they found significant improvements in pelvic pain during an acute attack if the woman was given the intravenous venoconstrictor, dihydroergotamine tartrate.[14] They concluded that pelvic congestion is likely secondary to ovarian dysfunction and associated reductions in reactivity of the pelvic circulation.

Iatrogenically Induced Dysfunction – One study found that 60% of patients diagnosed with pelvic varicosities had a history of tubal ligation and of these, up to 39% were symptomatic. Various studies have found at least an association of PCS and the use of intrauterine devices.[8] No significant study has validated these preliminary findings.

Diagnosis of PCS

Women presenting with chronic pelvic pain must first have non-PCS causes excluded including endometriosis, pelvic adhesions, atypical menstrual pain, urologic problems, spastic colon syndrome and psychosomatic disorders.[15] Using transabdominal ultrasound without the benefit of a tilt table results in a difference in ovarian vein diameter of as little as 2 mm in women appropriately diagnosed with PCS.[15] The mean vein diameters were 4.9 mm +/- 1.5 mm in the control group and only 7.9 mm +/- 2.3 mm in the affected group.[15] Other ultrasound findings of ovarian and pelvic varicosities are tortuous and dilated pelvic venous plexuses around the ovary and uterus with a reversed caudal flow of the ovarian vein.[15] CT findings of PCS consist of retrograde filling of incompetent dilated ovarian veins from the left renal vein in the arterial phase and varicose veins around the uterus and ovaries.[16] The presence of dilated pelvic veins greater than 5 mm in diameter is indicative of pelvic varicosities, and greater than 8 mm in diameter is indicative of PCS.[15]

In the past, confirmatory support for the diagnosis could have involved invasive methods such as transuterine venography, vulvar vein cannulation and venography, or retrograde venography.[17] Today, one may instead perform confirmatory ovarian venography together with subsequent definitive embolization at the same procedural session.

Treatment

The generally accepted treatment for PCS today is minimally invasive transcatheter embolization of the dysfunctional ovarian vein(s) and possibly associated contribution from incompetent internal iliac veins. As mentioned previously, this is often done at the same session as confirmatory venography.

The procedure is indicated when a woman suffers from chronic pelvic pain of at least 6 months duration with radiographic evidence of pelvic congestion and additionally, negative workup for other possible etiologies. Radiologic evidence for pelvic congestion may consist of any of a number of accepted modalities including computed tomography, MRI, or ultrasound plus or minus tilt table evaluation, or catheter venography. Often the latter is performed as final confirmation immediately prior to embolization.

The procedure should not be performed in situations of significant bleeding diathesis or uncorrected coagulopathy, severe contrast allergy, elevated creatinine, or current pregnancy.

Access to the ovarian vein may typically be from right internal jugular vein or common femoral vein approach. This author typically selects the right common femoral vein due to patient's negative perception of approach from the neck. A 5 French or 6 French hemostatic, vascular sheath is advanced into the chosen vein and an appropriately shaped catheter is used to select the left renal vein. If tilt table is not available, the patient is carefully instructed on the performance of a Valsalva maneuver and asked to do so during injection of contrast. If reflux is present then at least the ostium and beginning several centimeters of the vein will be visualized as contrast passes in retrograde direction toward the pelvis.

With the catheter secured into the proximal several centimeters of the vein, an additional run is done to further visualize the extent of the venous involvement. It is then a relatively simple process to pass a flexible guide wire down the vein to the level of the inferior margin of the sacroiliac joint. The vein and tributary points are then embolized using any of a variety of embolic particles or sclerosing agents, depending on personal preference and local availability.

An attempt is then made to locate and cannulate the right ovarian vein although if not incompetent, this can be quite difficult or even impossible. If reflux into the right ovarian vein is demonstrated, then the right vein is embolized in a similar fashion to the left.

Finally, both right and left internal iliac vein tributaries are explored with superselective angiography and if there is apparent contribution to the pelvic varicosities, these too are embolized.

The patient is then discharged home 4-6 hours later after explaining that she is to expect to experience mild pelvic discomfort and possibly low grade fever over the following 5 to 7 days. She is also instructed to abstain from straining or heavy lifting over the next week due to theoretical concerns of coil migration.

Procedure Risks

Risks of catheter directed embolization may include vessel injury or bleeding at the puncture site, venous bleeding from variceal perforation by guidewire, allergic reaction to intravenous contrast, acute renal failure from contrast nephropathy, unsuccessful procedure and radiation exposure.

Technical and Clinical Success

Technical and clinical success varies widely in the literature. Initial technical success ranges between 93 – 100% with partial symptomatic relief in approximately 10% and total relief of symptoms in close to 60% (to 19.9 months).[18,19,20] In another study with average 3.8 year follow-up, the average McGill Pain Score was 43.3% before the procedure and 3.3% on long term follow-up.[19] Visual Analog Score (VAS) study of 56 patients showed a mean decease of 65% in follow up between 6 and 38 months (mean 22.1 months).[20] At least one study looked at the effect of ovarian vein and concomitant internal iliac vein embolization on menstrual cycle and found no appreciable change.[20] Further research will be needed to effect improved clinical success by improved patient selection or to determine causes for persistent or recurrent symptoms in those patients in whom the procedure was performed with technical success and at least initial symptomatic relief.

Alternative Therapies

There is a depth of experience with stenting for treatment of symptomatic compression of the left iliac vein by the right common iliac artery as it crosses the pelvic promontory into the pelvis. Some endovascular specialists are beginning to approach left renal vein compression by the superior mesenteric artery (nutcracker syndrome) in similar logic. In support of this strategy, follow-up ultrasound in stented women has been found to show return of ovarian vein blood flow to normal antegrade direction toward the left renal vein along with significant improvement in symptoms. Unfortunately these studies have seen significant problems with stent migration or malposition which typically results in return of symptoms and further interventions either to deal with the stent or to retreat due to the pain.[21]

Although widely used previously, hormonal therapy or treatment with the vein constricting drug ergotamine for long term control of symptoms is not as commonly used today due to poor effectiveness at control of symptoms. However, treatment with analgesics is common and generally effective. Surgical treatment with hysterectomy and oophorectomy or vein ligation plays a role in select patients but requires longer recovery.

Conclusion

Pelvic congestion syndrome is one possible cause of chronic pelvic pain, a highly prevalent condition with significant personal and public burden. The syndrome is characterized by chronic pelvic pain attributed to dilated, engorged pelvic veins within the pelvis and usually is seen in association with multiparous women with incompetent left ovarian veins and reversal of flow toward the pelvis. Due to the common presence of dilated pelvic veins in asymptomatic women, other causes of chronic pelvic pain must be excluded. Confirmatory diagnosis is usually performed in conjunction with definitive endovascular treatment. Endovascular treatment is directed toward eliminating the abnormal venous flow patterns by occluding the incompetent ovarian veins and possibly internal iliac vein divisions that may also show reversed flow. Technical success is high and clinical success is moderate with the majority of women expressing significant improvement in symptoms. Further research is needed to improve patient selection and long term clinical success.

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Evaluation and Treatment of Lower Extremity Superficial Venous Insufficiency

Background - Benefits that Matter!

The Duval County Medical Society (DCMS) attempts to provide its members with the benefits that consistently meet your professional needs. One example of how this is being accomplished is by providing to DCMS members free Continuing Medical Education (CME) opportunities in the subject areas mandated/and or suggested by the State of Florida Board of Medicine to obtain and retain medical licensure. The DCMS would like to thank the St. Vincent's Healthcare (SVHC) Committee on CME for reviewing and accrediting this activity in compliance with the Accreditation Council on Continuing Medical Education (ACCME). Helena Karnani, MD, Chair of the CME Committee; Betsy Miller, Director, Medical Staff, Quality Management; and Cindy Williamson, CME Coordinator, from SVHC deserve special recognition for their work on behalf of DCMS.

This issue of *Northeast Florida Medicine* includes an article, "Evaluation and Treatment of Lower Extremity Superficial Venous Insufficency" authored by James St. George, MD (*see pp. 23-28)*, which has been approved for 1.0 AMA PRA Category 1 credit(s).™ For a full description of CME requirements for Florida physicians (MD/DO), please visit the DCMS website (http://www.dcmsonline.org/cme_requirements.aspx).

Faculty/Credentials: James St. George, MD, is the Medical Director/CEO of St. Johns Vein Center in Jacksonville, FL. Dr. St. George received his MD degree from the University of Massachusetts Medical School and besides his internship, he did a Radiology Residency at Albany Medical College, and a Nuclear Medicine Residency at Yale-New Haven Hospital at Yale University School of Medicine and the Hospital of the University of Pennsylvania. He also completed an Interventional Fellowship in Cardiovascular/Interventional Radiology at Brigham and Women's Hospital, Harvard Medical School in Boston, MA.

Objectives for CME Journal Article

1. Understand the pathophysiology, risk factors, and symptoms of venous insufficiency
2. Understand the clinical presentation and diagnosis of venous insufficiency
3. Understand current technologies and options for treatment of venous insufficiency

Date of Release: June 7, 2012 Date Credit Expires: June 7, 2014. Estimated time to complete: 1 hr.

Methods of Physician Participation in the Learning Process

1. Read the "Evaluation and Treatment of Lower Extremity Superficial Venous Insufficiency" article pages 23-28
2. Complete the Post Test and Evaluation on p.22. Members or non-members may fax the Post Test to DCMS (FAX) 904-353-5848 **OR** members can also go to www.dcmsonline.org & submit the test online. ***Non-members must arrange for the CME fee payment before submitting the post test by fax. Call 904-355-6561 x106 or fax.***

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Faculty Disclosure Information

Dr. St. George reports no significant relationships to disclose, financial or otherwise with any commercial supporter or product manufacturer associated with this activity.

Disclosure of Conflicts of Interest

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Evaluation and Treatment of Lower Extremity Superficial Venous Insufficiency

CME Questions & Answers (Circle Correct Answer) /Free-DCMS Members/$50.00 charge non-members*

(Return by June 7, 2014 by FAX: 904-353-5848, by mail: 555 Bishopgate Lane, Jacksonville, FL 32204 OR online: www.dcmsonline.org)

1. Which is NOT a risk factor for venous insufficiency:
   a. Obesity
   b. Heredity
   c. Prior limb surgery
   d. Tobacco use
   e. Gender

2. Which is NOT a typical symptom of venous insuffiency involving the lower extremity:
   a. Edema
   b. Limb heaviness and fatigue
   c. Pain with ambulation, relieved by rest
   d. Restless legs
   e. Sensory changes

3. Regarding the clinical examination of venous insufficiency of the lower extremity patients, which is true:
   a. Spider veins may be the only cutaneous finding (30-40%)
   b. Reticular veins are subdermal veins < 1mm in diameter
   c. Bulging varicose veins are found in most patients
   d. The clinical history & examination is usually diagnostic

4. Which is NOT a finding in venous stasis of the leg:
   a. Erythema
   b. Hyperpigmentation
   c. Lymphedema
   d. Compartment syndrome

5. Which may be associated with venous insuffiency of the lower extremity:
   a. Venous insufficiency of the deep venous system
   b. Lymphedema
   c. Iliac vein obstruction or stenosis
   d. Pelvic Congestion syndrome
   e. All of the above

6. Which statement best describes the duplex evaluation of venous insufficieny:
   a. Duplex ultrasonograpy can reliably assess the iliac veins
   b. Exam is essentially identical to a DVT evaluation
   c. Equal incidence of venous insufficiency within the great and small saphenous veins
   d. Duplex shows flow reversal within a saphenous vein for more than 0.5 seconds while patient is standing

7. Regarding conservative treatment of venous insufficiency with compression hose, which statement is true:
   a. Compression therapy will reverse chronic venous insufficiency
   b. Compression therapy will prevent progression of chronic venous insufficiency
   c. Compression therapy is palliative
   d. 80% of patients will comply with prescribed compression therapy

8. Which is NOT a treatment for lower extremity venous insufficiency:
   a. Compression therapy
   b. Endovenous thermal ablation
   c. Endovenous chemical ablation (sclerotherapy)
   d. Saphenous vein bypass surgery

9. Which is NOT a relative contraindication to saphenous vein thermal ablation:
   a. Deep venous obstruction
   b. Therapeutic anticoagulation
   c. Hypercoagulability syndrome
   d. Inability to ambulate

10. Which statement regarding sclerotherapy is true:
    a. Sclerotherapy is not necessary following endovenous thermal ablation of a saphenous vein
    b. Foam Sclerotherapy with sodium tetradecyl sulfate is FDA-approved
    c. Hyperpigmentation may occur in 10-40% of patients
    d. Sclerotherapy is the preferred treatment for large bulging varicose veins

Evaluation questions & CME Credit Information

(Please evaluate this article. Circle one number using this scale: 1= Strongly Agree to 5= Strongly Disagree)

The article met the stated objectives:	1	2	3	4	5
The article was appropriate to my practice:	1	2	3	4	5
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Evaluation and Treatment of Lower Extremity Superficial Venous Insufficiency

James St. George, MD

Abstract: *Chronic venous disease of the lower extremity is frequently not diagnosed, misdiagnosed or untreated. Clinical findings are often minimized as cosmetic or simply attributable to the aging process. This is one of the greatest misnomers among both medical providers and the lay public. This article will review the pathophysiology, clinical findings, diagnosis, and treatment of lower extremity venous insufficiency.* *(see color figures on p.51.)*

Introduction

Lower extremity chronic venous disease (CVD) is a condition whose spectrum ranges from abnormalities such as "spider veins" to severe edema, skin ulceration, and major disability. The importance of chronic venous disease is related to the large number of people with the disease and the socioeconomic impact of its more severe manifestations. More important is the significant adverse effect on an individual's quality of life (QOL).[1] Chronic venous disease is often overlooked by providers because of an under appreciation of the magnitude and impact of the problem. These are the patients who repeatedly appear in the emergency room at 2 a.m. with leg pain yet have no evidence of deep venous thrombosis. These patients may present with moderate to severe chronic edema; however, the lack of a proper diagnosis leads to an incorrect treatment with chronic diuretics. Symptoms such as leg discomfort, fatigue, and restless legs, are often minimized by providers or relegated to vague diagnoses such as fibromyalgia or restless leg syndrome. Many patients do not seek or receive care until development of venous ulceration. The aim of this article is to update the clinician regarding current diagnosis and treatment.

Normal Venous Anatomy and Function

The veins of the lower extremity are divided into the superficial and deep venous systems connected by a series of perforator veins. The deep venous system is located below the muscular fascia and serves as the primary conduit for venous return. The deep veins follow the course of the major arteries and return greater than 90% of blood volume from the lower extremity. The superficial venous system is located above the muscular fascia layer and functions as a reservoir and conduit to the deep venous system. The principal veins of the superficial system are the great and small saphenous veins. By International Consensus, the word "great" replaces "greater" or "long." The word "small" replaces "lessor" or "short." The great saphenous vein runs from the medial ankle along the medial leg to join the common femoral vein. The small saphenous vein runs along the midline of the calf between the medial and lateral gastrocnemius muscles and typically joins the popliteal vein. Innumerable small cutaneous veins arborize to join superficial branches, which drain into the truncal great, and small saphenous veins. The superficial veins are connected to the deep system by numerous perforating veins in the thigh and leg that pass through anatomic fascial spaces.[2]

In the erect position, venous blood return must rise against both gravity and fluctuating thoracoabdominal pressures. A large number of one-way bicuspid valves function in concert with the lower extremity muscle pump to facilitate venous return. The muscle pump, primarily the calf, forces blood out of the venous plexus and up the deep venous system. The valve system insures that blood moves only in the cephalic direction and blocks gravitational forces from pulling blood back to the feet. This prevents a significant increase in hydrostatic pressure within the distal veins of the lower leg and feet. Perforator valves prevent transmission of high pressure and flow from the deep system to the superficial system.

Venous Pathophysiology and Dysfunction

Venous pathology develops when the return of blood from the lower extremity is impaired and venous pressure is increased. In the majority of cases, this results from venous valve incompetence. When the bicuspid valves do not correctly oppose, retrograde flow of blood occurs. This is known as venous insufficiency or venous reflux and may occur in the deep, superficial and perforator veins. Venous reflux results in venous hypertension. Dysfunction of the deep venous valves is most often a consequence of deep venous thrombosis (DVT.) Incompetence of the valves in the superficial venous system may be Primary due to preexisting weakness of the vessel wall or valve leaflets, or Secondary due to direct injury, superficial phlebitis, or excessive venous distention.[3,4]

When this occurs locally in small cutaneous veins, it can be manifested as telangiectasias (spider veins.) Visually, these appear as fine cutaneous red, purple or blue veins less than 1mm in diameter. These are often seen in relation to larger 1-3 mm subdermal blue or blue-green veins known as reticular veins. *(Figure 1, p.51)* While these entities are often considered cosmetic, patients may experience symptoms such as itching, burning or achiness. These symptoms are often exacerbated by factors promoting vasodilation such as heat or elevated hormone levels (menstrual cycle, pregnancy.)

Failure of valves located at the saphenous-femoral and saphenous-popliteal junctions substantially increases hydrostatic pressure within the superficial veins. There is usually

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concomitant valve failure in larger secondary and tertiary branches of the saphenous vein. The markedly elevated venous pressures may result in progressive venous dilation and thickening of large venous branches, which manifest visually as varicose veins. "Varicose veins" are defined as any vein abnormally dilated greater than 3 mm.

The effects of venous hypertension are transmitted to the microcirculation and eventually result in venous microangiopathy. The normal reabsorption of perivascular fluids by osmotic and pressure gradients is impaired, resulting in accumulation of perivascular and lymphatic fluid. This leads to edema, fibrosis, and impaired oxygenation of surrounding tissue.[5] The disruption of normal vascular and lymphatic flow of the lower extremities may result in the symptoms of chronic venous insufficiency and lymphedema.

Venous obstruction, deep vein reflux, muscle pump failure and congenital abnormalities are less common causes. Venous obstruction and deep venous reflux are the most common secondary causes of CVD and are almost always the result of DVT.[6] Iliac venous stenosis or obstruction may be detected in 10-30% of patients with lower extremity swelling. Venous insufficiency of ovarian, parauterine, and internal iliac veins (Pelvic Congestive Syndrome) may contribute significantly to lower extremity symptoms.

Clinical Manifestations

Chronic venous Insufficiency (CVI) is the clinical entity that results from chronic venous hypertension. Numerous risk factors contribute to the development of venous insufficiency *(Table 1.)*[7-9] Hereditary factors play a major role. If a single parent experienced venous disease, there is a 50% likelihood of the child presenting with a similar condition and nearly 100% probability if both parents had CVD. Many patients report their first telangiectasia as a teenager. The incidence of venous insufficiency increases with age. Varicose veins occur in 8 % of women aged 20-29 years, increasing to 41% in the fifth decade and 72% in the seventh decade of life. Prolonged hormonal influences related to female gender, pregnancy, and birth control pills increase the incidence. It has been estimated that almost 70% of women develop telangiectasias during pregnancy. Men are affected less than women (41% versus 72% in the seventh decade.) Prolonged standing/sitting, obesity, limb trauma and limb surgery are other risk factors.

Patients often present with concerns regarding the cosmetic appearance of their leg veins. They frequently do not recognize CVI symptoms *(Table 2)* due to the insidious progression of venous insufficiency. They have acclimated to their condition, incorrectly attribute their symptoms to another cause, or consider them part of the aging process. Directed specific questioning is required.

Complaints of lower extremity fatigue or heaviness are common. Ankle or calf edema is also common and may be severe *(Table 3. p.25)*; however, early evidence may only be calf fullness and is often not recognized by the patient. Edema is typically described as completely resolved upon awakening and progressively worsens throughout the day. Patients may report cramping or pain that often awakens them. They may describe chronic aching or throbbing. Other complaints include skin itching, burning, tingling, and numbness. Patients frequently complain of restless legs and venous insufficiency symptoms are these are often mischaracterized as "restless leg syndrome." Generally all these symptoms are worse with prolonged standing or sitting and are most noticeable at the end of the day. Leg elevation, walking and exercise improve symptoms.

Table 1 Risk Factors for Chronic Venous Insufficiency

Heredity
Female gender
Pregnancy
Hormone therapy
Age
History of prolonged standing or sitting
Obesity
Leg trauma or surgery

Table 2 Leg Symptoms Associated with Chronic Venous Insufficiency

Arching
Throbbing
Cramping/Night Cramps
Heaviness
Fatigue
Swelling
Restless legs
Itching, Burning, Numbness, Tingling

The patient should be evaluated while standing to allow maximal distention of the veins and elicit dependent venous congestion. Calf edema is best evaluated viewing the posterior calves from a distance of 4-5 feet. *(Figure 2, p.51)* Direct careful attention to the skin of the medial ankle as this region is the most dependent and vulnerable to the effects of long-term venous hypertension. If saphenous reflux is severe, there may be focal or diffuse increased pigmentation from hemosiderin deposition. *(Figure 3, p.51)* There may be erythema, rubor or a dusky appearance to the feet. Long-standing venous hypertension and distal venous stasis leads to more advanced skin changes within the distal leg, ankle, and foot. These include eczema, lipodermatosclerosis, atrophe blanche and

Table 3 Differential Diagnosis of Lower Extremity Edema

Venous obstruction
Chronic right-sided heart failure
Pericardial effusion
Venous insufficiency
Tricuspid stenosis
Tricuspid regurgitation
Pericarditis
Cirrhosis
Cellulitis
Premenstrual fluid accumulation
Low albumin
Medications (Calcium channel blockers)
Lymphatic obstruction
Preeclampsia–eclampsia
Myxedema
Compartment syndrome
Malignancy
Pelvic tumor
Pregnancy
Deep venous thrombosis
Fluid overload

ulceration. *(Figure 4, p.51)* Lipodermatosclerosis is a dark leather-like fibrotic thickening of the skin. Atrophe blanche is focal hypopigmentation resulting from skin ischemia/infarct.[10,11] Uncorrected, advanced CVI patients are at risk for ulceration and non-healing wounds as well infection and lymphedema.

Venous ulcers from great saphenous vein insufficiency usually occur in the medial supramalleolar area and in the lateral malleolar area from small saphenous vein insufficiency. Long-standing CVI may also lead to the development of secondary lymphedema, representing a combined disease process. Lymphatic dysfunction is present in up to one third of patients with chronic venous insufficiency and may resolve with correction of the venous abnormalities.[12]

Large tortuous bulging varicose veins are not present in most patients with venous insufficiency. Scattered spider veins or reticular veins may be the only cutaneous finding in 30–40 percent of patients. While the history and physical exam may be suggestive, diagnosis requires a lower extremity venous duplex examination.

Duplex Ultrasound

Advances in affordable venous duplex ultrasound technology have revolutionized the treatment of venous disease. A properly performed duplex ultrasound is the primary diagnostic test to identify and characterize venous insufficiency.[13-16] This has replaced other modalities such as hand-held Doppler, photoplethysmography, air plethysmography, and contrast venography. Venous duplex imaging combines grey scale imaging of the deep and superficial veins with pulsed Doppler assessment of venous flow. The aim of duplex ultrasound is to define all the incompetent superficial venous pathways. The deep venous system is assessed to exclude DVT or venous obstruction.

It is very important to recognize that a duplex ultrasound for CVD is very different than a study of the lower extremity for DVT. The patient must be examined while standing which requires a platform or a tilt table. Duplex studies for DVT are performed with the patient supine and venous reflux is usually not detected. A thorough knowledge of the superficial venous system anatomy and common variants is necessary. Unfortunately, most imaging centers and hospitals lack these capabilities. Many patients experience significant worsening of their lower extremity pain, cramping, and swelling in the late evening. They present to the emergency room where a venous duplex is performed with the patient supine. DVT is excluded; however, the true diagnosis is missed. Even most vascular technologists lack sufficient training and familiarity with duplex testing of the superficial venous system. One credentialing body, Cardiovascular Credentialing International (CCI,) has addressed this deficiency by offering an examination to vascular ultrasound technologists to be certified as a Registered Phlebology Sonographer (RPhS.)

Any significant venous flow toward the feet is diagnostic of venous insufficiency. Venous reflux is diagnosed when there is reversal of flow from the expected cephalic direction for more than 0.5 seconds following a provocative maneuver such as calf/foot compression by the examiner, dorsiflexion by the patient, or a Valsalva maneuver.[16] Significant reflux is identified in the great saphenous vein or one of its primary tributaries in 70-80% of patient's with CVI symptoms. Small saphenous pain reflux is found in 10-20% of patients and tributary non-saphenous superficial reflux is identified in 10-15%.[17,18]

Duplex ultrasonography cannot reliably assess the iliac veins and the inferior vena cava. Additional imaging such as computed tomography venography, magnetic resonance venography, or contrast venography may be required to characterize venous obstruction, stenosis, or a venous anomaly in the pelvis. The iliac veins should be assessed in patients with post–thrombotic disease. It should also be considered in patients with non-thrombotic disease if the clinical presentation is incongruent with the duplex findings.[19,20]

Treatment

Conservative Treatment-Compression Leg Garments - The initial treatment of CVI involves conservative measures to reduce symptoms and help prevent the development of secondary complications. Elevating the legs, exercise or walking, flexion/extension of the feet helps reduce edema and symptoms. Graded compression garments have been the mainstay of conservative treatment.[21]

Most 3rd party payers require 90 days of compression therapy prior to authorizing more definitive treatment. There is no data supporting this requirement; It has not been proven that stockings will prevent progression of venous disease. Compression hose treatment is palliative. The patient's chronic venous disease will remain and the clinical benefits are only realized while wearing the garment. Symptomatic relief is incomplete and patient compliance is very poor. The reported rates of non-compliance range from 30-65%. The stockings are hot, difficult to put on, difficult to remove, may be uncomfortable, may fall down the leg, or may cause skin irritation. The elderly, who usually present with the most advanced venous disease, are often unable to adequately utilize standard compression garments due to frailty and arthritis. It is even more difficult to obtain adequate fitting in the obese patient. It is the rare patient who will comply with compression hose for more than a few days or weeks during a hot Florida summer.

Endovenous Thermal Ablation - For many years, definitive treatment required saphenous vein stripping and high ligation. Recovery from this treatment was long and painful. Consequently, many patients who required intervention avoided surgery. The second revolution in venous disease treatment was venous ablation. Endovenous thermal ablation (EVTA) of the great and small saphenous veins was FDA approved in 1999 and has replaced routine surgical stripping. This procedure uses either a radiofrequency catheter or a laser fiber to create a full-thickness burn of the incompetent vein wall. This results in irreversible occlusion and fibrosis of the vein.

The first device approved was a catheter that creates radiofrequency (RF) energy to heat the vein wall. Patents of the procedure make Covidien (formerly VNUS, Mansfield, MA) the sole vendor of RF technology in venous ablation. Laser fibers were subsequently developed to administer thermal energy to the vein wall and there are numerous laser vendors. Successful ablation rates of the great saphenous vein have been reported between 85% and 100%. Most recent studies report greater than 95% successful occlusion with both technologies. There are no prospective randomized comparisons between RF and laser, although several retrospective analyses have demonstrated similar occlusion and complication rates. Initially, the RF technique had significantly less post-procedure pain and bruising; however, this advantage has been eliminated with newer laser wavelengths. Endoluminal laser closure has a lower disposable cost and the fibers may be sterilized for reuse. Both technologies apply heat; it becomes a matter of preference and cost.

Procedures are performed in the physician's office with local anesthetic and require no sedation. Relative contraindications for EVTA are outlined in *Table 4*. It is not necessary to discontinue therapeutic anticoagulation.[22] Following a sterile preparation and drape of the extremity, intravenous access into the mid-calf saphenous vein is obtained using ultrasound guidance. The RF catheter requires a 7-French hemostatic vascular introducer. The laser only requires a 4-French catheter. The RF catheter or a laser fiber is inserted into the great or small saphenous vein and advanced under ultrasound-guidance. The device is positioned 2cm proximal to the saphenous junction.

Table 4 Relative Contraindications for EVTA

Pregnancy or nursing
Obstructed deep venous system inadequate to support venous return after EVTA
Liver dysfunction or allergy limiting local anesthetic use.
Severe uncorrectable coagulopathy or hypercoagulability syndromes
Inability to wear compression stockings secondary to inadequate arterial circulation, hypersensitivity to the compressive materials, or musculoskeletal or neurologic limitations to donning the stocking.
Inability to adequately ambulate after the procedure
Sciatic vein reflux

A mixture of 50 cc of 1% lidocaine with epinephrine in 500 cc normal saline is utilized as a tumescent anesthesia. This solution is instilled into the venous perivascular space under ultrasound guidance. The tumescent provides local anesthesia, insulates the adjacent soft tissues, and compresses the vein lumen. When activated, the RF catheter uses a standard protocol to heat the vein wall to 120 degrees Celsius for 20 seconds. The RF catheter treats the vein sequentially in 7 cm segments. Lasers treatment protocols and wavelengths vary; in general, the laser fiber is withdrawn continuously at 3-20 cm/minute depending upon the laser wavelength and wattage. The goal is to apply at least 70 J/cm. The entire procedure is performed in less than 30 minutes.

Following the procedure, the leg is wrapped with a compressive elastic bandage. The patient is fully ambulatory and may immediately return to routine activities. Elderly patients easily tolerate the minimally invasive treatment and the most difficult component of treatment is 20-30 mmHg compression hose for 1-3 weeks post-procedure.

Complications from EVTA are relatively minor.[23] There may be minor ecchymosis. Approximately one week after EVTA, the treated vein may develop a feeling of tightness and there may be transient discomfort related to inflammation in the treated vein segment. The latter is easily ameliorated with

non-steroidal anti-inflammatory drugs. Superficial phlebitis of branch varicosities may occur in 5% of patients. Irritation or injury to sensory nerves may result in paresthesia or dysesthesia. This has been reported in 0-15% of cases and is usually temporary. Skin burns have been reported but are rare and avoidable. DVT following EVTA is unusual. The reported rates vary widely; pooled data indicates a 0.3% to 0.4% incidence. A duplex ultrasound is performed 2-7 days after a procedure to exclude extension of saphenous thrombus into the deep system

Several studies have documented significant and durable improvements in QOL following EVTA.[24-29] Many patients enjoy prompt symptomatic relief. In all reported series, most treatment failures occurred within the first 6 months and all have occurred within the first 12 months. Late clinical recurrence of venous insufficiency is due to progression of CVD. Treatment can only address current incompetent veins. The patient's individual risk factors will determine progression with advancing age.

Reflux in the truncal saphenous veins must be treated before addressing any visible abnormality. Clinical success after EVTA is predicated by the ability of the treating physician to identify and eliminate all incompetent pathways with adjunctive procedures. Unfortunately, many physicians do not provide comprehensive treatment and treat with only EVTA. While there may be an initial symptomatic improvement, the patient is unlikely to achieve lasting benefit and distal venous stasis will likely persist.

Sclerotherapy - Patients require adjuvant measures to eliminate incompetent tributaries.[30] Eradication of residual incompetent saphenous branches is important to complete the hemodynamic correction, maximize symptomatic improvement, eliminate incompetent reservoirs that could facilitate the development of new incompetent pathways, and complete cosmetic improvement of the treated limb. Elimination may also improve the success of EVTA and possibly slow the progression of venous disease in parallel venous trunks. Therefore associated varicose tributaries, reticular veins, and telangiectasias are treated with adjuvant sclerotherapy and ambulatory phlebectomy .

Sclerotherapy is the injections of a medication into a vein to irreversible occlude the vein. This is usually done with a 1cc or 3 cc syringe and a 27 or 30-gauge needle. The preferred sclerosant agents are polidocanol, sodium tetradecyl sulfate and 50% glycerin. These agents are FDA approved, have an excellent safety profile, and have been in use for over 50 years. Some practitioners still use hypertonic saline (23.4%); however, this is painful and has higher risk of skin ulceration.

The size of the vein determines the sclerosant type and concentration. Higher concentrations (1.5-5%) of either sodium tetradecyl sulfate or polidocanol are used for obliteration of varicose veins and residual great or small saphenous vein remnants. The latter are treated with ultrasound guidance. The concentration is progressively and significantly reduced with decreasing vein size. For example, 0.5% to 0.75% of polidocanol is used for reticular veins while a concentration of 0.25% is used for spider veins. And 50% glycerin is popular for the treatment of telangiectasias, particularly when they involve the thin skin of the ankle and foot.

Injecting the liquid agent into the vein is known as "liquid sclerotherapy." Agitating a sclerosant medication with air or physiologic gas creates a foam for "foam sclerotherapy." The foam promotes prolonged contact with the vein wall and is more effective; however, the FDA has not approved this method. Twenty-six percent of patients have a residual patent foramen ovale and there are reports of transient ischemic attacks presumably due to the embolization of foam bubbles.

Some authors have used sclerotherapy for the treatment of truncal saphenous vein insufficiency however, there is a high rate of recannulization. Sclerotherapy is the primary treatment of recurring neovascularization following surgical ligation and stripping.

A common complication (10-40%) of sclerotherapy is hyperpigmentation of the surrounding skin from hemosiderin degradation. This is more common in individuals with darker skin pigmentation and usually resolves within 6 months to one year. Inadvertent backwash into a small arteriole will result in focal skin ulceration. Inadvertent direct injection of an artery will cause acute arterial occlusion. Allergic reactions occur infrequently.[30]

Ambulatory Phlebectomy - Ambulatory phlebectomy (AP), also known as microsurgical phlebectomy or stab avulsion phlebectomy, is a procedure by which small segments of veins are removed with minimal skin damage. This procedure is performed for incompetent tributary branches of the great or small saphenous veins, reticular veins and varicose veins. Pudendal and labial varicose veins are also appropriate for AP. As with EVTA, the procedure is performed as an outpatient in the office.[30,31]

The procedure is performed either in conjunction with or shortly after EVTA. The veins to be treated are marked both upright and supine. Following a sterile preparation and drape, the tissue surrounding the veins is infiltrated with the tumescent. Cutaneous 1-3 mm incisions are made and the vein is snared with various vein hooks. The vein is brought to the skin surface, grasped with a clamp and progressively freed from the subcutaneous tissues until avulsed. This is repeated along the entire course of the marked veins at 1cm-5cm intervals depending upon the size, length, tortuosity, and type of vein. Long courses of veins often require greater than 20 or more individual stabs.

Absolute contraindications for AP include infectious dermatitis or cellulitis in the area to be treated, severe peripheral edema, and lymphedema. The vast majority of complications are minor. There may be skin blisters from dressings, changes in skin pigmentation, minor scaring, infections, rare allergy to the local anesthetic, post-procedure venous bleeding, and sensory nerve paresthesia.[31]

Conclusion

The prevalence of symptomatic CVD may be as high as 60-70% in the elderly population. CVI can severely impair an individual's activities of daily living and quality of life. Painful surgical stripping has been replaced by endovenous ablation, which is a minimally invasive venous procedure performed in the physician's office and allows an immediate return to daily activity. Additional treatment with adjuvant methods is extremely important to eliminate all incompetent venous disease and obtain lasting clinical benefit.

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Venous Stasis Ulcers: A Review

David S. Ross, MD, FACEP

Abstract: *The most common etiology of chronic leg ulcers is venous disease. It often occurs after trauma, and heals slowly due to the venous insufficiency. The classic signs of venous disease may not always be present. Work up should include venous duplex scans that evaluate superficial venous insufficiency as well as evaluation of the deep system. Other possible co-morbidities must be addressed. Compression is the mainstay of treatment as well as prevention. It should only be done when there is little risk of significant arterial disease. Regular debridement and maintenance of a moist wound bed will encourage wound healing. Venous insufficiency wounds and ulcers that do not show significant progress within 4 weeks should be considered for referral to a specialized wound care center. Other wounds requiring referral are those involving deep structures, any limb threatening wounds, and those with significant co-morbidities. Numerous advanced therapies are available which have revolutionized the treatment of venous stasis ulcers.* ***(see color figures for this article on p. 51)***

Introduction

Over 70% of chronic wounds in the lower extremities result from venous disease.[1] The wounds are often initially traumatic and the underlying pathology may not be obvious. They have traditionally been difficult to treat with frequent recurrence. Diagnostic and treatment options have often been limited. Patient compliance with prevention protocols is often poor. Finally, the cost and variety of even basic dressings and treatment modalities can be prohibitive. As a result, these wounds and ulcers have represented a challenge to physicians. Due to the multiple co-morbidities, a multidisciplinary approach is essential.

The purpose of this article is to provide an organized approach for the physician, an update on diagnostic and treatment options, and rational criteria for when to refer to a specialized wound care center.

Varied Presentation of Venous Stasis Ulcers and Wounds

Venous stasis ulcers may occur spontaneously. However, most often, they appear after a history of trauma, which may be minimal. These traumatic wounds are less likely to be recognized as a complication of venous stasis disease unless a high index of suspicion is maintained. The signs may not be obvious and the only tip-off may be the delayed wound healing.

The classic signs of venous disease are pitting edema, varicosities, spider veins (telangectasias), reticular veins (dilated bluish sub dermal veins less than 3 mm in diameter). Corona phlebectatica is a fan-shaped pattern of numerous small intradermal veins below the maleoli. Symptoms may include dependent, aching leg pain, heavy, tired feeling legs, and cramping. Restless leg syndrome may be present.

Long-standing advanced venous disease leads to chronic findings of venous stasis. Brawny edema is most commonly seen. There is often anterior or circumferential inflammation and erythema of the distal leg. It can be indurated and often mimics cellulitis. Microscopic subcutaneous bleeding breaks down leaving a brown hemosiderin deposition. There is often scaly dry exfoliating skin (eczema), particularly seen as the edema decreases. The flaking skin may be thick and waxy, particularly around a wound.

In severe venous stasis disease, the legs may weep and blister. Lipodermatosclerosis occurs and there is loss of adipose tissue. The "inverted bottle" or "champagne bottle" leg may be seen. Long standing venous insufficiency causes atrophic changes, which can be focal or generalized. Atrophic white skin, is known as "atrophie blanche". Severe, long standing venous insufficiency will produce enough edema to overwhelm the lymphatics and compress them causing "secondary lymphedema" .Recurrent ulcers and wounds are a prominent feature in advanced severe disease.[2]

Venous Ulcers and Wounds Are Seldom Classic

The most common location for venous stasis ulcers is the distal medial lower leg. Lateral leg wounds are less common. Venous ankle and foot ulcers are rare. Atypical locations are usually due to traumatic causes. The pathology delaying wound healing is the same.

Venous stasis ulcers are usually irregular shaped, relatively flat and rarely have undermined margins. They may have a red or pink granulation base and may have a violacious border. They often have white to yellow slough and they almost never produce a black eschar. Surrounding "peri-wound" tissue is often red inflammatory changes often mimicking cellulitis.

Anatomy of a Venous Ulcer

Veins in the lower extremities are classified as the deep system, the superficial system, and the perforator veins, which usually carry venous flow from the superficial to the deep systems.[3] Of primary importance are the venous valves, which are present in most veins. Since the venous return is a low-pressure system, it requires an intact network of valves. It also requires the calf and foot "muscle pumps", which increases the intravenous pressure and drives the venous return. If the valves are not functioning (incompetent), then the venous

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return will reflux retrograde upon standing. This leads to increased venous engorgement, venous hypertension, and edema. Likewise, muscle weakness and inactivity will lead to an impaired "muscle pump" and edema.

Edema, low flow rates, and venous engorgement may predispose to deep vein thrombosis (DVT.) Previous DVT may, in turn, cause a worsening of the venous stasis. There is a history of DVT in 37% of people with venous insufficiency.[4]

Statistics/Epidemiology

Some key statistics on conditions described earlier are:

- Estimate of venous disease prevalence in the population is 10-35%
- The incidence of venous leg ulcers in the elderly is 4%
- While venous disease and venous leg ulcers are increased in the elderly, they are seen all ages: 61% of patients with venous leg ulcers have their first episode < 65 Yrs; 40% of patients with venous leg ulcers have their first episode < 50 Yrs;13% of patients with venous leg ulcers have their first episode < 30 Yrs [5]
- Recurrences rates of venous leg ulcers is 72%[4]
- The annual cost of venous leg ulcers in the U.S. is $1.9-$2.5 billion[5]
- 2 million work days are lost yearly[2]

Differential Diagnoses Equals Co-Morbitities

The differential diagnosis of lower extremity ulcers includes the major causes of delayed wound healing: 1) Diabetic Ulcers; 2) Arterial Insufficiency Ulcers; 3) Pressure Sores; 4) Infected Wounds; 5) Traumatic Wounds and 6) Post-Surgical Wounds.

Less common causes of delayed wound healing may be osteomyelitis, chronic steroid use, occult foreign bodies, delayed effects of radiation (soft tissue radio necrosis), malnutrition, and occult malignancy. Chronic edema due to lymphedema, CHF, hypoalbuminemia, cirrhosis, and renal failure may delay wound healing.

Co-morbidity plays a large part in delaying wound healing. Chronic wounds are often multi-factorial. Diabetes increases the risk of infection and can accelerate peripheral arterial disease. Diabetic neuropathy can lead to an occult pressure injury. Arterial insufficiency can be found in nearly 20% of patients with venous leg ulcers.[6]

A final part of the differential diagnosis is the "pseudo-wound". This is the lesion, which represents a dermatitis that appears to be a wound, but has intact epithelium.

An Organized Evaluation

An appropriate history and physical is important in any chronic wound work-up. While clinical evaluation can give good direction, certain testing is recommended. HgbA1c is appropriate to screen for diabetes in patients with an elevated blood sugar or a family history. While the clinical findings may indicate venous insufficiency, this author has been impressed with the number of times that venous insufficiency is not clinically evident, yet it is found on a venous duplex scan.

In order to diagnose venous insufficiency, a venous duplex scan of the superficial saphenous veins is required. This is available at specialized vein centers, in most vascular surgeons' offices, some radiology departments, and some cardiologists' and podiatrists' offices. A venous duplex scan of the deep system to diagnosis DVT is the commonly ordered study in most hospitals. Do not assume that a standard venous duplex scan has evaluated the superficial system or has evaluated the veins for incompetence.

There are two vital reasons to evaluate for arterial insufficiency. The first is the frequency of co-morbidity and the inherent risk of limb loss and possible mortality. In addition, arterial insufficiency may appear minimal or be sub-clinical but can be precipitated by compressive stockings and dressings. This is especially true when using venous compression of 30-40 mm Hg or greater.

An appropriate simple evaluation is an ABI or ankle brachial index, with exercise. The index is the ratio of the maximum systolic BP at the ankle to the maximum brachial systolic BP.

$$\textbf{ABI} = \frac{\textbf{ankle SBP}}{\textbf{brachial SBP}}$$

ABI:	0.9 - 1.1	are considered normal
ABI:	0.7 – 0.9	are mildly decreased
ABI:	0.5 – 0.7	indicate arterial insufficiency
ABI:	< 0.5	severe arterial insufficiency
ABI:	>1.1	calcified poorly compressible arteries with Diabetes Mellitus

A complete non-invasive arterial evaluation of the lower extremity includes segmental limb pressure measurements, which can help to localize stenosis and occlusion, pulse-volume recordings, which can measure the blood flow and record pulse volume waveforms and arterial duplex ultrasonography, which can evaluate for arterial stenosis and occlusion. If these screening tests are abnormal, a CT angiogram may be obtained.

Wound cultures are often taken but the results may be misleading. Chronic wounds are often colonized by multiple bacteria since they have lost the protection of epithelium and have a wealth of available of nutrients. The greatest impediment to healing is bacteria invading tissue, i.e. cellulitis. It is important to assess for the typical signs of infection: pain, tenderness, fever, erythema, induration, exudate, odor, and systemic symptoms. In the wound care center, tissue cultures with actual cellular material are often sent, not just slough or surface debris. An intermediate situation called critical colonization may occur when there are multiple species, virulent species or an immunocompromised patient.

Finally, biopsy is important if there is a possibility of

malignancy. This should be considered if there is a history of previous malignancy or if the patient shows features of malignancy.

Compression/Treatment and Prevention

Compression has been well established as the mainstay of treatment for venous insufficiency ulcers. All types of compression have been shown to be effective. Random controlled trials have shown the benefit although further comparisons are recommended.[7] One of the most commonly used methods is the Unna's boot. It is a cotton bandage impregnated with zinc and various other ingredients. It is most effective when covered with a second elastic wrap. Newer dressings with additional layers have been developed. These will usually have two elastic layers with the other layers providing various methods of absorption. The key to proper dressing application is proper training in order to apply the recommended amount of overlap and compression. The principle of compression requires greater pressure exerted at the distal leg/ankle with a graduated decrease in pressure proximally. In a large review of randomized controlled trials, it was shown that: 1) Ulcer healing improved with compression vs. non-compression dressings; 2) Multi-layered systems were more effective than single-layered systems; 3) There was no difference between different types of multi-layered dressings; and 4) High compression is more effective than low compression.[8]

It is important to remember that the use of higher-pressure dressings requires exclusion of significant arterial insufficiency. As a general guideline, Unna's Boot and other long stretch elastic wraps are safe with an ABI above 0.8. Some short stretch compression wraps may be used safely with lower ABI's, however great caution is recommended.

Compressive stockings can be used with some dry venous insufficiency wounds but are more helpful in preventing ulcers. Attention to prevention cannot be over emphasized. The major obstacle to effective long-term use of compression is patient compliance. Velcro wrap graded compression garments are available.

Non-prescription knee-high support stockings providing less than 20 Mm Hg may be best for mild, early edema. T.E.D. stands for "thrombo-embolic deterrent" and represents one brand of low-pressure garments to prevent DVT; however, these stockings are not graduated and are designed for use in the non-ambulatory supine patient. They are not appropriate in the treatment of venous insufficiency.

Compression stockings and dressings work by providing a gradient pressure that assists in venous return and therefore edema, weeping and exudate are reduced. Graduated compression is most effective in the ambulatory patient who assists venous return by using the muscle pump. Patients are advised to walk. What should be avoided is standing stationary. Elevation in the inactive patient is helpful as well to counteract the effects of gravity. Diuretics, on the other hand, are less effective and not recommended for venous insufficiency, since it is not a state of volume overload.

Compression pumps may be needed for refractory cases of venous insufficiency and for lymphedema. If done at home, careful patient education is needed. Specialized lymphedema clinics are available for ongoing treatment.

A Moist Wound Heals Best

The process that the body uses to clean up chronic wounds and remove dead and dying cells is called autolysis. It requires a warm, moist environment.[9] It occurs in injured cells or dying tissue. Autolysis is initiated by the lysosomes releasing digestive enzymes. It is slowed by dry environments and is one of the reasons that wet to dry dressings are becoming less popular.

Excessive moisture in a wound can cause maceration and tissue breakdown. There are numerous dressings with varying properties that can correct both extremes. Alginates, foams, hydrocolloids, hydrofibers, and hydrogels are excellent absorbent dressings. When moistened, they provide an appropriate level of hydration.

Decrease the Bioburden

Chronic venous stasis wounds that have critical colonization or cellulitis should be treated with systemic antibiotics. Topical antibiotics such as mupirocin are an effective adjunct to treat of gram positive organisms.

Silver ion dressings and ointments and cadexomer iodine have an excellent topical antibacterial effect. They are helpful in decreasing the bioburden. Silver has been found to increase re-epithelialization across meshed skin grafts.[10]

Older iodine preparations such as povidone iodine will inhibit fibroblast growth.[11]The cellular toxicity of hydrogen peroxide has been shown to exceed its bactericidal potency.[12]

Debridement/Surgical Treatment

A basic surgical principle is to remove necrotic tissue. This should be done for all venous insufficiency ulcers. The only exception is in the arterial insufficiency wound. There may not be enough viable substrate available for the wound to heal if the protective eschar or scab is removed. Once adequate circulation is established, debridement is appropriate.

Debridement removes tissue that is physically blocking cell migration. Chronic venous insufficiency wounds are "out of balance". There is excess bioburden and biofilm. Inflammatory cells and enzymes may impede wound healing. Dying tissue is a source of fuel for more bacteria. One hypothesis is that debridement creates acute changes in the wound that stimulate the body's response to new tissue injury. Fibroblasts are stimulated to lay down new collagen, blood vessels and local tissue growth factors.

The practitioner who is skilled in the technique may undertake sharp surgical debridement. Following basic surgical principles, removing necrotic tissue, slough, and biofilm, as well as "saucerizing" the wound in order to obtain a more favorable shape will stimulate wound healing. [13]

One time tested debridement technique has been called into question.[13, 14] This is the wet to dry dressing change. Saline is applied to sterile dressings which are applied to wounds. As the saline evaporates, debris, fibrin, bioburden, and any moist tissue sticks to the bandage. As the bandage is removed, so is the debris. There is the rub; wet to dry dressings are indiscriminate in which tissue is pulled out of the wound. There have been no randomized controlled trials to evaluate the effectiveness of wet to dry dressings. They are labor intensive and inconsistently applied.[14] There may be increased pain. Cotton fibers may be left in the wound. As saline migrates to the outer dressing, it becomes a conduit for bacteria into the wound. Finally, in the dry stages, the body's own autolysis is hindered. All of these factors may combine to slow wound healing or make the treatment less acceptable to the patient. Since the practice of wound care is directed towards healing wounds rapidly, this technique has fallen into disfavor in the treatment of chronic wounds. Nevertheless, this author has seen some "stalled" wounds respond only to wet to dry dressings.

Debridement can be accomplished with minimally invasive mechanical cleansing of the wound. Irrigation at pseudo high pressures can be accomplished with a syringe and saline, or with a mechanical or even an ultrasonic irrigation system Use of non-cytotoxic detergents may be helpful. Whirlpool baths have come into disfavor for venous stasis ulcer due to lack of random controlled trials, the risk of nosocomial infection, and the potential for damaging newly epithelialized tissue.[15]

As surgical debridement is effective on the macroscopic level, enzymatic debridement represents an approach that removes dead tissue on the microscopic level. Collagenase is the single enzymatic product currently available in the U.S. It is deactivated by many silver containing dressings and iodine. Medicinal maggot therapy is available for selected refractory cases. This process leads to remarkably clean wounds. The primary limiting factor in its use is patient and practitioner acceptance.

Stimulation of the Wound Bed

Several dressings have been developed that contain a composite matrix of a freeze-dried composite of collagen and oxidized regenerated cellulose (ORC) with or without silver. These reduce the activity of inflammatory proteases, cytokines and free radicals in chronic wounds, thereby restoring a healing balance in the wound. The silver reduces the bioburden. These dressings have been shown to increase the wound healing in chronic venous insufficiency wounds.[16] These dressings should stay in place for 3 days, or shorter periods if severe exudate warrants.

When to Refer Wounds

Physicians are encouraged to become skilled in chronic wound care. It is recommended that the following wounds be referred to a specialized wound care center:

1. Venous insufficiency wounds that have failed to show significant progress despite four weeks of standard care
2. Wounds that involve deep tissue structures
3, Any limb threatening wounds
4. Wounds complicated by significant co-morbidities: Peripheral vascular disease, Persistent edema, Persistent infection, Prior radiation treatment, Compromised immune status
5. Any wound that physicians would like the wound care center to follow.

Advanced Treatment Modalities

Advanced therapies should be initiated on any venous insufficiency wound that has not decreased in size (area) by 30% after four weeks of conventional treatment. If properly documented, these advanced treatments may be covered by most carriers.

The following advanced treatment modalities may be considered:

APLIGRAF® was the first living, cell-based tissue regeneration product. It was approved by the FDA in 1998. It was shown to improve the rate of wound healing in chronic venous insufficiency ulcers.[17] It is a bi-layered human skin equivalent with tissue cultured neonatal foreskin over bovine collagen lattice. It provides most human tissue growth factors. Immunogenicity is lost as fibroblasts are grown in tissue culture. There is no known graft vs. host reaction as the graft does not "take", but is lysed by the native cells. There is no increased incidence of wound infection. It is applied every 2 to 6 weeks.

OASIS® WOUND MATRIX is a natural extracellular matrix derived from porcine small intestine submucosa. It was initially developed in 1995. OASIS contains key components of the dermal extracellular matrix that are reported to stimulate fibroblasts, keratinocytes and vascular endothelial growth factor (VEGF). It has been shown to improve wound management and the percentage of healed venous insufficiency ulcers.[18] It is applied weekly.

Other bioengineered skin substitutes are being studied to establish their efficacy.

Negative-pressure wound therapy (NPWT) is a treatment that uses a vacuum applied through a sealed dressing to promote healing in chronic or acute wounds. It was first approved in 1995. It has been shown to be effective in healing venous insufficiency wounds among other indications.[19] It works by drawing wound edges together, deforming the wound which

stimulates fibroblasts and angiogenesis, removing exudate and infectious materials, reducing edema, and promoting granulation tissue formation.[20] It can be used over bioengineered skin substitutes. It cannot be used over exposed blood vessels, anastomotic sites, organs, nerves, malignancies, untreated osteomyelitis, fistulas, or undebrided necrotic tissue It must have an opening of greater than 1 cm x 1 cm.

Hyperbaric Oxygen Treatment (HBOT) is a medical treatment in which the patient is entirely enclosed in a pressurized chamber breathing 100% oxygen at greater than one atmosphere pressure, typically between 2 and 3 atmospheres. HBOT works by dissolving oxygen in serum creating hyperoxygenation which kills bacteria, reduces edema (by vasoconstriction), stimulates angiogenesis by direct stimulation of fibroblasts and oxygen dependant collagen production, and release of tissue growth factors. [21]

The Undersea and Hyperbaric Medical Society and Centers for Medicare & Medicaid Services (CMS) have recommended HBOT treatment for 14 medical conditions, including ischemic/hypoxic wounds, certain diabetic foot ulcers, refractory osteomyelitis, compromised and failing skin grafts or flaps. [21,22] In general, venous insufficiency wounds are not covered unless there is significant co-morbidity as noted earlier.

Other Treatment Modalities

Venous Ablation of incompetent superficial and perforator veins by radiofrequency (RFA) and endovenous laser treatment (EVLT) offers additional options for some refractory cases and are indicated to prevent recurrent ulcers.[23] Split Thickness skin grafts (STSG) are indicated in some refractory cases. In rare cases a full thickness flap may be required if complications have led to exposure of underlying structures. Pentoxifyline improves the effectiveness of compression dressings.[24]

Conclusion

Venous insufficiency wounds and ulcers represent a common problem for the physician. A thorough evaluation with attention to co-morbidities and a multidisciplinary approach is most effective. The specialized wound care center is poised to aid the primary physician in management of these wounds. . Venous insufficiency wounds and ulcers that do not show significant progress within 4 weeks should be referred to a specialized wound care center. Other venous wounds requiring referral are those with significant co-morbidities, any limb threatening wounds, and those involving deep structures. Many new advanced treatment modalities are available including bioengineered skin substitutes, negative-pressure wound therapy, and hyperbaric oxygen therapy in selected cases. Venous ablation of incompetent superficial and perforator veins, and the application of split thickness skin grafts may also be effective. ***(see color figures for this article on p.51)***

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Lymphedema - Pervasive and Chronic, but Treatable: An Overview

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Abstract: *Lymphedema as a pathologic condition is presented in this article. Its clinical presentation, incidence, and causes are described. The morbidity associated with lymphedema and the benefits of treatment are stressed.*

Introduction

Lymphedema is poorly known and poorly understood.[1] It is swelling of a body part caused by arterial capillary filtration exceeding lymphatic transport capacity.[2] There are three major categories of lymphatic pathophysiological abnormalities. These include obstruction, reflux and overproduction of lymph fluid. Obstruction is the most common mechanism producing lymphedema. It accounts for nearly all cases of secondary lymphedema and most cases of primary lymphedema. Obstruction of lymphatic flow may result from cancer, surgery, trauma, inflammation, and infection. In 10% of patients there is a presumption of chronic reflux of lymphatic fluid in a retrograde direction when lymphatic vessels are chronically dilated and the lymphatic valves no longer function. Overproduction of lymphatic fluid occurs with increased vascular permeability at the capillary level further increasing the interstitial fluid and associated proteins.

Lymphedema of the upper extremity may present with symptoms such as tight jewelry, or a tired or thick, heavy feeling of the arm.[3] Lymphedema of the lower extremity can have swelling, heaviness, tightness, and skin problems.[4] It can be primary (congenital) caused by abnormal development of the lymphatic system, or secondary (acquired) caused by injury to the lymphatic system. Secondary lymphedema is more common except in the pediatric population.[5,6]

Diagnosis

Lymphedema can present in various stages. Acutely it can present within a few hours of interruption of lymphatic drainage. It is reversible, in that it can recede readily by elevation of the edematous body part. The edema is pitting at this stage. Dimpling, pitting, and swelling may give a peau d'orange (like the skin of an orange) appearance to the skin. The skin creases become prominent with broadened skin folds of the toes and fingers, which cannot be easily lifted (Stemmer's sign). Untreated, the lymphedema becomes chronic, nonpitting, and irreversible.

Lymphedema is best confirmed by lymphoscintigraphy.[1] A radionuclide is injected between the fingers or toes and transported by the lymphatic system. Abnormal accumulation of the radionuclide in the subcutaneous tissues indicates lymphedema. Lymphedema can also be diagnosed with bioimpedance technology (bioelectrical impedance spectroscopy)[7-9] It can also be assessed using limb circumference measurements, volume displacement measurements such as water displacement, an infrared optoelectronic perometer for volume measurement, computerized tomography (CT), and magnetic resonance imaging (MRI).[10,11] Self reporting of swelling can also be taken into account. The method of diagnosis can make a rather substantial difference in the statistics of prevalence.[12] As a practical matter, in orderto get insurance reimbursement for the diagnostic testing of lymphedema, extensive justification is often required.

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Pediatric Patient Characteristics

Primary lymphedema is categorized at the time of onset. If the lymphedema appears in the newborn, it is called congenital lymphedema. If it appears after birth but before the age of 35 years, it is called lymphedema praecox. If it develops after 35 years of age, it is called lymphedema tardum. Infancy was the time of development in 49.2% of the patients. Childhood was the time of development in 9.5%, and adolescence was the time in 41.3%. The lymphedema involved an extremity 95.7% of the time and the genitalia 18.1% of the time. The lower extremity was affected 91.7% of the time with 52.9% having bilateral disease. Only 11.7% had familial disease or a syndrome which includes lymphedema.[6] While lymphedema often presents as a large lower extremity, in the pediatric age group this can be caused by several conditions besides pure lymphedema. These conditions include: microcystic/macrocystic lymphatic malformation, noneponymous combined vascular malformation, capillary malformation, Klippel-Trenaunay syndrome, hemihypertrophy, posttraumatic swelling, Parkes Weber syndrome, venous malformation, rheumatologic disorder, infantile hemangioma, kaposiform hemangioendothelioma, and lipofibromatosis.[13] Lipedema is a bilateral symmetrical limb enlargement caused by the deposition of adipose tissue.

Some cases of primary lymphedema have been associated with mutations in certain specific genes[14] with some of these genes (GJC2) affecting gap junctions which could disrupt lymphatic flow.[15] Primary lymphedema of the lower extremities has been associated with hypoplasia of ipsilateral inguinal lymph nodes on lymphoscintigraphy.[16] Lymphangiography by MRI shows dilated lymphatic vessels in most cases of lower extremity lymphedema.[17]

Secondary Lymphedema

Filariasis is the most common cause of lymphedema worldwide.[18] In developed countries, cancer and its treatment are a common cause of lymphedema. One review of multiple cancer studies indicated the incidence of lymphedema after cancer treatment as follows: 16% for melanoma (5% for upper extremity and 28% for lower extremity), 20% for gynecologic cancers, 10% for genitourinary cancers, 4% for head and neck cancers, and 30% for sarcomas. There is an increased incidence after pelvic dissection or radiation therapy.[19]

Lower Extremity Lymphedema

Lymphedema of the lower extremity can occur as a direct result of cancer and can be the presenting symptom, such as for cancer of the prostate.[20] It can also occur as a result of pelvic lymph node dissection.[21,22] Lymphedema of the lower extremities occurs in 7 to 23% of patients who have had ilio-inguinal (pelvic) lymph node dissection for melanoma.[10]

Venous obstruction and chronic venous insufficiency can also cause or exacerbate lymphedema.[23] These result in increased lymphatic water load and often overwhelm the lymphatic transport capacity resulting in lymphedema. As with other forms of lymphedema, it is exacerbated by obesity.

Lymphedema is also exacerbated by pregnancy in approximately 11% of cases.[24] Rarely, rheumatoid arthritis, Kaposi sarcoma, retroperitoneal fibrosis, and immunosuppressive agents can cause lymphedema.[18] Some drugs that are known to cause edema can induce or exacerbate lymphedema. These include pioglitazone (Actos),[25] and calcium channel blockers such as amlodipine (Norvasc) and nifedipine (Procardia).[26] Spina bifida increases the risk of lymphedema by almost 100 times over the general population to 9.2%. A history of trauma, cellulitis, cancer, obesity, decreased mobility, wounds, and hypertension increase the risk even further.[27]

Upper Extremity Lymphedema

Lymphedema occurs in the upper extremity on the side of breast cancer treatment in 28% to 42% [18,28] of breast cancer survivors and is the most common cause of lymphedema in most Western countries.[19] Most cases (80%) of lymphedema after breast cancer treatment occur in the first two years after treatment.[29,30] Four to five years after surgery, it occurred in 5 to 7% of women after sentinel lymph node biopsy and in 16 to 21% after axillary lymph node dissection.[31-33] It was more common in women who had more than five lymph nodes removed, had a higher stage cancer, or had metastases in the lymph nodes. [28,29] The incidence of lymphedema after breast cancer treatment increases if the patient is overweight, [28,30,32,33] had a mastectomy, chemotherapy, lymph nodes with metastases, residual cancer, full axillary node dissection, injury in the extremity, or had treatment-related complications.[34-36] The estimated pooled risk ratio from 98 separate studies indicated that radiation therapy increases the risk of developing lymphedema.[36] The presence of a high BMI, infection, and increased hand use can increase the incidence of lymphedema from 7% to 94%.[37] Lower socioeconomic status, having a partner, greater child care responsibilities, participation in regular activity, an age greater than 80 and having good upper body function have also been associated with a lower incidence of lymphedema.[8,28]

Surgery after the cancer has been treated can also affect the incidence or extent of lymphedema. Tissue expander breast reconstruction after mastectomy can reduce the incidence of lymphedema.[38] Autologous breast reconstruction can minimally increase the incidence of lymphedema but it sometimes improves preexisting lymphedema.[39]

Lymphedema of Other Areas

Lymphedema after head and neck cancer treatment can affect superficial areas such as the face or neck and internal areas such as the larynx, pharynx, or oral cavity.[40] Lymphedema can occur in the treated breast or associated chest area after cancer treatment.[41] Lymphedema of the vulva can be associated with being overweight.[7] Lymphedema of the male external genitalia is usually associated with lymphedema of the lower limbs. It can be primary, or it can occur after bladder or prostate or rectum cancer, lymphoma, aorto-bifemoral bypass grafting, or biopsy of inguinal lymph nodes.[16]

Effects of Lymphedema

Lymphedema can cause significant morbidity and mortality.[1] It is associated with a poor quality of health, poor quality of life and disability. It results in decreased joint mobility, poor physical functioning, decreased strength and range of motion, and pain. Physical and emotional role limitations, lower vitality, and poor social functioning, leads to poor mental health and high psychological stress.[28,33,42-46] The worse the lymphedema, the worse the quality of health and the health outcome.[47,48] Lymphedema associated with cancer is also worse than lymphedema not associated with cancer.[47] Lymphedema after head and neck cancer treatment can influence speech, breathing, swallowing, and cervical range of motion.[40] Lymphedema is often associated with pain.[23] Lymphedema occurring after breast cancer treatment affects physical, psychological, and sexual functioning in significant ways.[2] Erisipelas and lymphangitis are frequent complications of lymphedema.[46] In a claims analysis for two years after starting treatment for breast cancer, lymphedema doubled the incidence of cellulitis or lymphangitis and increased the cost of care $15,000 to $23,000 which included health care services, diagnostic imaging, and visits with moderate or high complexity.[34] Lymphedema in the pediatric population also was associated with cellulitis in 18.8% with most of these requiring hospitalization.[6] Rarely, angiosarcoma occurs in the affected limb (Stewart-Treves syndrome).[49]

Treating Lymphedema

Following the guidelines from the National Lymphedema Network for risk reduction may decrease lymphedema risk.[2] Patients should avoid trauma, limb constriction and temperature extremes. They should wear appropriate compression garments and optimize activity and weight. Increased knowledge about lymphedema increases the adherence to risk-reducing behaviors.[50] Most lymphedema therapists recommend avoiding cuts and scratches, insect bites, sunburn, and excessive exercise.[4]

Lymphedema treatment aims to remove excess plasma proteins from interstitial fluid. Treatment should decrease the size of the affected body part, improve skin texture, prevent complications such as skin infections, improve function, and improve overall sense of psychological well-being.[5] Referral to specially trained healthcare professionals is recommended to ensure optimal treatment.[1] Treatment consists of complete (or complex) decongestive therapy which includes skin care, external pressure, isotonic pressure, and manual lymphatic drainage with compression.

Manual lymphatic drainage is done by a specially trained massage, occupational, or physical therapist. It consists of a gentle stretching of the skin which enhances lymph formation, then a gentle pressure in the direction of desired lymphatic flow, and then a release of pressure causing refilling of the lymphatic vessels before repeating the sequence approximately every second. The motion can be circular, linear, or of various patterns depending upon the contour and area of the body being treated. Treatment is often started proximally to decongest areas into which the peripheral fluid can be moved. Various paths over the body are used to move the fluid based upon the anatomic lymphatic flow and the area of obstruction or slow drainage. The lymphedematous area is then wrapped with various short-stretch materials to create a pressure gradient which will continue moving the fluid proximally. Padding is used to even the pressure in areas with an uneven contour. This treatment reduces the volume of lymphedema fluid, decreases the fear of activity, increases the quality of life, improves general well-being, and is usually very effective.[3,46,51,52,53] Women with mild lymphedema after breast cancer treatment are three times more likely to develop moderate to severe lymphedema than women without lymphedema,[3,54] so treatment can be of benefit for even mild lymphedema.

Complete decongestive therapy involves moving fluid from the interstitial space into the intravascular space so that the kidneys can remove it. This requires adequate cardiac function to move the intravascular fluid and adequate renal function to remove the fluid. Thus, uncontrolled congestive heart failure and end-stage renal disease are contraindications to complete decongestive therapy as well as such conditions as deep vein thrombosis and acute cellulitis.

Surgery can reduce the amount of lymphedema in some patients with lower extremity lymphedema by lymphaticovenular bypass [55,56] Iliac vein stenting can reduce the swelling and discomfort associated with lymphedema of the lower extremities when venous obstruction is involved in the lymphedema.[23] Lymphedema of the male external genitalia can be helped by circumcision or penile or scrotal cutaneous excision.[16]

The treatment for lymphedema is rarely curative so that a maintenance regimen is usually needed. This can include intermittent pneumatic compression (lymphedema pumps,) compression garments, manual lymphatic drainage with wraps by the patient or an acquaintance, proper exercise, proper diet, and skin care.[46] The pumps should apply pressure in a sequential manner to move the fluid proximally.

Compression garments need to be properly fitted without interrupting lymphatic flow. Wearing an elastic compression garment during the day and a multilayer low-stretch bandage at night helps maintain a lower volume of lymphedema in the affected limbs. Certain types of exercise can be done which help physical and psychosocial recovery without worsening the lymphedema.[57] This can include weight lifting in a controlled and monitored setting while wearing well-fitted compression garments. This will result in fewer exacerbations of lymphedema, reduced symptoms, and increased strength.[58] Younger age, higher weight, and higher BMI are associated with greater recurrence of the lymphedema.[59]

Summary

Lymphedema is swelling of a body part which can be caused by lymphatic obstruction, reflux, or overproduction of lymph fluid. It can be acute or chronic in its presentation. It can be primary (congenital) or secondary (acquired). It can present at any time of life – from the newborn to the elderly. Secondary lymphedema can result from infection, such as filariasis, or cancer and its treatment, or from venous obstruction or chronic venous insufficiency. Lymphedema can cause significant morbidity and mortality. There are guidelines for reducing the risk of lymphedema and its consequences. Treatment is available in the form of complete decongestive therapy by trained professionals that can reduce the lymphedema and decrease the morbidity leading to a better quality of life.

Resource: *Textbook of Lymphology for Physicians and Lymphedema Therapists.* Foldi M, Foldi E, Kubik S. (EDS.) Urban & Fischer**;** Munchen, Germany. 2003.

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An Introduction to Compression Therapy

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Abstract: *Several studies have confirmed the hemodynamic benefits of compression therapy in patients with chronic venous insufficiency. In patients with venous ulcers, compression is effective in both healing and preventing recurrence of ulceration. This article is intended to provide the clinician with a practical guide to the principles, methods, and prescription of external compression therapy.*

From the Dawn of Man to Boston Marathon

The use of compression in the treatment of venous diseases is not a new idea. The earliest evidence of compression dates back to the beginning of mankind. Mural paintings in the Tassili caves (Sahara) dating back to the Neolithic Age (5000 – 2500 B.C) depict illustrations of what is believed to be compression dressings.[1] Hippocrates wrote about compression treatment in the 4th century BC. Roman soldiers who marched for days at a time learned that applying tight strappings to the calves reduced leg fatigue. 2000 years later, calf compression sleeves are extremely popular with long-distance runners. In 1839, Dr. John Watson reported on using elastic stockinsg to treat varicose veins in a 23-year-old woman with Klippel-Trenaunay syndrome.[2] Chronic Venous Insufficiency (CVI), affects up to 13 million people in the United States. Peak incidence occurs in women aged 40-49 and men aged 70-79 years, however these numbers are probably very underestimated due to misdiagnosis, lack of knowledge and education.[3]

Basic Principles of Compression

In a standing individual the venous hydrostatic pressure, which equals the weight of the blood column between the foot and right atrium, is about 80-100 mmHg. During walking blood flow is accelerated by the combined action of the calf muscle pump and the foot pump, which decreases the volume of venous blood and reduces venous pressure to about 10-30 mmHg.

If the valves in the large veins become incompetent due to primary degeneration or post-thrombotic damage, blood will oscillate up and down in those segments lacking functional valves. The resulting retrograde (backward) flow in the veins of the lower leg (venous reflux) leads to venous hypertension. This causes fluid loss into the tissues, edema, and chronic venous stasis tissue changes. Compression of veins with incompetent valves provides a ridged sheath around the vessels so that blood flow will be propelled upward toward the heart instead of laterally against the wall. The objective is to oppose the hydrostatic forces of venous hypertension. Bandages and, to a lesser degree, graduated compression stockings provide the external support needed to produce this effect.

The application of adequate compression reduces the diameter of major veins, which increases blood flow velocity.[4] The clinical significance depends upon the relationship between the intravenous hydrostatic pressure and the applied external compression. In a supine individual, a pressure of 10 mmHg applied to the calf is sufficient to reduce venous stasis by producing a marked decrease in blood volume accompanied by a corresponding increase in blood velocity. This is the basis of anti-embolism T.E.D. (Thrombo Embolic Deterrent) stockings, which are designed for the non-ambulatory supine patient. However, T.E.D. compression is not graduated and is ineffective in the treatment of ambulatory venous insufficiency. In the upright position, the pressure in the lower leg fluctuates during walking between 20-100 mmHg, and therefore much higher levels of compression are required to exert a marked effect upon blood flow.[5]

Compression initiates a variety of complex physiological and biochemical effects involving the venous, arterial and lymphatic systems.[4,6,7] Several studies have confirmed the hemodynamic benefits of compression therapy in patients with CVI. If the correct technique and materials are used, edema and pain are reduced. In patients with venous ulcers, compression is effective in both healing and preventing recurrence of ulceration.

Microcirculation

Compression also accelerates blood flow in the microcirculation and normalizes cutaneous blood flow. Improvement in cutaneous oxygenation has been demonstrated with the use of compression in patients with venous stasis after only 10-15 min.[8] Capillary filtration is also reduced and reabsorption is increased due to enhanced tissue pressure. In lipodermatosclerotic areas where skin perfusion may be reduced due to high tissue pressure, the use of compression therapy can increase this gradient and improve blood flow. This leads to softened skin. Effects on mediators involved in the local inflammatory response may explain both the immediate pain relief that occurs with good compression and subsequent ulcer healing.

Compression bandages and walking exercises can improve lymph transport. The morphological changes of the lymphatics in lipodermatosclerotic skin can be normalized with long-term compression. The dramatic reduction of edema by compression therapy can be explained by the reduction of lymphatic fluid in the tissue, rather than by an improvement of lymphatic transport.[9]

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Compression Bandages and Compression Stockings

A number of compression methods are available. (*Table 1*) The main categories of compression concern the elastic properties of the materials. Extensibility is the ability of the material to increase in length in response to an applied force.

In general, compression bandages are able to achieve higher pressures than compression stockings.[10] Bandages are best indicated when temporary compression is required, such as in the acute phase of DVT, superficial phlebitis, venous ulcers, lymphedema, and phlebolymphedema. Another benefit of bandages is that they can be reapplied to maintain the optimal compression as edema in the affected limb is reduced.

Graduated compression stockings (GCS) provide a convenient method of maintaining pressure while allowing ambulation. The graduated compression garment, combined with the pumping effect of the leg muscles (especially the calf muscles), aids venous and lymphatic return. Graduated compression applies the maximum amount of pressure to the ankles, which gradually tapers off over the length of the stocking, e.g. a 20-30mmHg knee-high stocking will apply 30mmHg to the ankle and taper to 20mmHg at the knee.

Table 1 Types of Compression Devices

Graduated Compression Stockings
- Ready-made retail stockings manufactured in fixed sizes
- Custom-made stockings

Bandages
- Inelastic
  - No stretch (~zero extensibility) e.g. Unna boot, Circ-Aid
  - Short stretch (<100% extensibility)
- Elastic
  - Long stretch (100% extensibility)
  - Cohesive, adhesive
  - Single component or multi-layer

Compression boots
- Water, air
- Inelastic band devices

Intermittent Pneumatic Compression
- Single chamber
- Sequential chambers

(Adapted from Reference 12)

Prescription of a Stocking

A prescription for elastic compression stockings requires information about both tension and length. Proper fitting of the stockings is essential and requires accurate limb diameter measurements. Individual measurements for a compression stocking should be taken while the patient is standing and at the beginning of the day when the leg is less edematous. The most important measurement is at the ankle, where a graduated stocking exerts the greatest pressure. Measurements should conform to the manufacturers' guidelines. Compression stockings are widely available. Most vein centers keep a supply in the office. They can also be obtained by prescription at a medical supply store. The stockings can be bought at some pharmacies; however, it is not advisable to purchase over-the-counter stockings due to the lesser quality of the material and improper fit.

Proper fit of compression stockings is imperative. Patient compliance and satisfaction is higher with quality, properly sized stockings. Vein centers that supply stockings have been educated on measuring patients for an accurate fit, as have medical supply stores.

Treatment with 20-30 mmHg compression (Class II) is adequate for mild to moderate venous insufficiency. Patients with more advanced disease may require Class III 30-40 mmHg compression. (*Table 2, p.41*)

Stocking Styles and Lengths

Ready–made, off-the-shelf stockings are manufactured in fixed sizes. Most manufacturers have numerous sizes, varying in both length and width at various points on the ankle, calf, and thigh. Although the sizes are somewhat standardized, there may be considerable variations between the different manufacturers. Up to six styles of medical compression stockings are available, depending on the manufacturer: Knee–high, mid–thigh, or high–thigh, pantyhose, one–leg pantyhose, thigh with waist attachment, and maternity pantyhose. Some manufacturers have open–toed hose available, especially the single-leg, thigh–thigh variety. Regardless of the style, most stockings are available in three lengths: Knee-high, mid thigh, and high thigh.

The most common length is knee–high because patient adherence is greater and symptom relief may be adequate; however, some feel that knee–high stockings have a tourniquet effect on the great saphenous venous return and may actually worsen proximal venous congestion. Thigh-high or waist-high stockings provide a greater benefit; however, the stockings are more difficult to use and compliance is reduced. Stockings need to be changed every 6-9 months if worn daily to avoid loss of the elastic tension.

Patient Compliance

Noncompliance is the most important factor limiting the use of compression stockings. Patient's compliance in wearing their compression stockings is frequently underestimated by their physicians. The reported rates of non-compliance range from 30-65%.[11] The stockings are hot, difficult to put on, difficult to remove, may be uncomfortable, may fall down the leg, or may cause skin irritation. The elderly,

Table 2 CVI, DVT, PTS Treatment

8-15mmHg	15-20mmHg (Class I)	20-30mmHg (Class II)	30-40mmHg (Class III)	>40mmHg (Class IV)
	Minor varicose	Moderate to severe varicose	Severe varicose	Severe varicose
Minor swelling	Minor swelling	Moderate edema	Lymphatic edema	Lymphatic edema
DVT prophylaxis	DVT prophylaxis	DVT prophylaxis	DVT prophylaxis	DVT prophylaxis
	Post-Sclerotherapy	Post-Sclerotherapy	Post-Sclerotherapy	
		CVI	CVI	CVI
		Prevent ulcer recur	Manage active ulcer or Prevent ulcer recur	Manage active ulcer
		Post-venous procedure	Post-venous procedure	
		Superficial thrombophlebitis	Manage PTS	Manage PTS
		Orthostatic hypotension	Orthostatic hypotension	

(Adapted from Reference 12)

who usually present with the most advanced venous disease, are often unable to adequately utilize standard compression garments due to frailty and arthritis. It is difficult to obtain adequate fitting in the obese patient. Some obese patients will need custom manufactured stockings, which are expensive, and not covered by insurance. Patients who cannot tolerate class II stockings should be fitted with class I stockings; mild compression is better than no compression.

Compression hose treatment is palliative. The patient's chronic venous disease will remain and the clinical benefits are only realized while wearing the garment. Patients with symptomatic venous insufficiency should be referred for definitive treatment; this usually requires endovenous thermal ablation and adjuvant sclerotherapy. Most 3rd party payers require 45 to 90 days of compression therapy prior to authorizing treatment. There is no data supporting this requirement, and it has not been proven that stockings will prevent progression of venous disease.

Another potential obstacle to compression is arterial disease. Although it is accepted that compression should never be allowed to impede arterial inflow, there is currently no convincing clinical data to indicate the level of compression that may be safely applied to a limb with arterial insufficiency. It is accepted that a systolic ankle pressure below 50-80 mmHg is a contradiction for high compression therapy, as is an ankle-brachial pressure index (ABPI) of less than 0.5.

Conclusion

The physiological basis of compression therapy on the lymphatic, venous and arterial systems is well established. Patients may experience substantial improvements in pain, mobility, and quality of life. It remains a mainstay treatment for venous ulceration. However, compression treatment remains plagued by practical limitations and poor patient compliance; however, the literature is also clear that any compression is more effective than no compression.

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Venous Malformations: Basic Concepts and Interventional Radiologic Management

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Abstract: *Venous malformations (VM) are congenital vascular anomalies with multiple clinical presentations, are sometimes of difficult to diagnose, and are often ineffectively or incompletely treated. Multiple diagnostic classifications in the past have contributed to misunderstanding of the condition, and consequently interfered with the development and implementation of effective therapeutic approaches that minimize procedural morbidity. Within the larger universe of vascular anomalies, venous malformations represent a distinct group and will be the focus of this article. Depending on the type of vascular tissue involved, vascular anomalies can be capillary, venous, arterial, lymphatic, or mixed. True arteriovenous malformations (AVM) and distinct lymphatic anomalies will not be addressed.*

Introduction

Venous malformations (VM) are congenital lesions caused by abnormal vascular morphogenesis that present clinically during childhood and adolescence, but not usually at birth. They consist of conglomerates of abnormally developed veins that become distended and painful with very slowly flowing blood. Though venous tissue predominates, VMs can be mixed with dysmorphic capillary or lymphatic tissue. Erroneously but commonly referred to as "hemangiomas", VMs are not neoplasms and exhibit mature cellular components with normal turnover.

Symptoms and signs include skin discoloration, pain, swelling, coagulopathy, and hemorrhage. Pulsatility and thrills are absent.VMs are important components of various syndromes. The most common and best known example is Klippel-Trenaunay. This syndrome is a rare condition that is present at birth. The syndrome usually involves port wine stains, excess growth of bones and soft tissue, and varicose veins. Diagnosis is based on the clinical presentation, physical examination and imaging characteristics. MRI is by far the most useful diagnostic imaging modality, but ultrasound, fluoroscopy and direct puncture venography are essential for percutaneous therapy.

Management is best accomplished with a multi-disciplinary team of surgeons (plastic, orthopedic, otolaryngologists), interventional radiologists, dermatologists, and physical therapists among others. Relatively few lesions can be completely resected without major tissue loss, and incomplete surgery can lead to very symptomatic recurrences. Percutaneous sclerotherapy in most cases is essential in preparation for surgical resection or as the primary treatment modality. Absolute ethanol is the most effective sclerosant, but carries the greatest risks, including tissue necrosis, nerve palsies, rarely severe cardiopulmonary complications, and even death. Milder sclerosants are available and preferred when ethanol is considered too risky. Of the patients treated with percutaneous sclerotherapy 70-90% see improvement in pain, bulk symptoms and appearance.

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Classification

Perhaps the greatest hurdle in understanding vascular anomalies and establishing effective treatment has been the use of inadequate classifications. Since ancient times, these entities have been recognized and grouped by the external appearance. Skin involvement has influenced observers and terms such as "port wine stain" or "strawberry" have been perpetuated. The mass-like nature of many malformations has led to the use of names with tumoral or neoplastic implications. Commonly and inappropriately called "hemangiomas", venous malformations need to be classified with embryologic genesis and hemodynamic characteristics in mind.

Mulliken and Glowacki proposed a classification based on biologic and pathologic differences divided in two subgroups, hemangiomas and vascular malformations.[1] Hemangioma referred to lesions of infancy characterized by proliferating and involuting phases with increased cellular turnover. The term vascular malformation was used to refer to congenital lesions that grow along with the child and exhibit vascular spaces with normal cellular turnover that could be capillary, venous (venous malformation), arterial (arteriovenous malformation), lymphatic or fistulous. These lesions were further characterized according to flow dynamics into slow-flow and high-flow anomalies, a designation of great value in planning and executing therapeutic intervention.[2,3] *Table 1* (p.43) summarizes a commonly used classification.[4]

Pathology

Vascular malformations are congenital anomalies that may not manifest themselves until later in life and often are undetected at birth or early childhood.[5] Abnormal embryologic vascular development results in persistent remnant islands of primitive vascular tissue. Particular alterations of vascular morphogenesis will determine the dominant vessel, e.g. capillary, venous, arterial, lymphatic or mixed. VMs are abnormal collections of veins, inconsistent in size and with abnormal venous wall structure. The lack of a normal muscular layer explains the tendency for marked enlargement of the vascular channels. As the child grows, blood pooling and luminal distention become more prominent. VMs can be located in almost any part of the body. They can be well-defined or can replace normal tissues in a diffuse, infiltrative pattern. The involved tissue is often atrophic with adipose replacement.

Table 1 ISSVA Classification

Vascular Anomalies		
Vascular Tumors	**Vascular Malformations**	
	Simple (Terms of common usage)	**Mixed**
Infantile hemangioma Congenital hemangioma Tufted angioma Kaposiform hemangioendothelioma Hemangiopericytoma Pyogenic granuloma Spindle-cell Hemangioendothelioma	Capillary (Birth marks and stains) Lymphatic (Cystic hygroma and macro/microcytic lymphangioma) Venous (Hemangioma and cavernous hemangioma) Arterial (Arteriovenous malformation)	Arterivenous fistula Arteriovenous malformation Capillary-venous malformation Capillary-lymphatic-venous malformation Lymphatic-venous malformation Capillary-arterial-venous malformation Capillary-lymphatic-arteriovenous malformation

(Modification of table from Reference 4)

Microscopy reveals thickened dysplastic-appearing vascular channels with mature endothelial cells, as opposed to vascular tumors that have increased cellularity. The muscular layer is disorganized or focally absent. Intravascular thrombosis with formation of calcified phleboliths is common.[5] Infantile hemangiomas, but not VMs, express glucose transporter-1. In contrast, VMs are positive to S-100 neural tissue marker on immunohistochemistry.[6, 7]

Clinical Findings

VMs are present at birth. At first many are undetected. The lesions enlarge during childhood and adolescence and become clinically apparent. Growth slows during adult life, but it can continue. Hidden deep lesions may not become symptomatic until adulthood. Hormonal influences (puberty, pregnancy) and injury may elicit faster growth. Attempts at surgical resection, especially when incomplete, can have an adverse influence on lesion growth and symptomatic worsening. Any part of the body can be affected. VMs can be intraosseous, intramuscular, subcutaneous, mucosal, or can involve multiple tissue layers and deep organs.

On physical examination, superficial lesions usually are readily visible. They can have a capillary component in the dermis, commonly called a "birth mark", or can have a purple to dark blue discoloration caused by the subdermal or dermal dilated venous channels. The lesions are usually soft and spongy, and both gravity and Valsalva maneuver can cause engorgement. Lesions that have thrombus formation can be hard to palpation. Deeper lesions can be undetectable by physical exam or present as soft tissue masses sometimes with a prominent fibro-fatty component. In contrast to true AVMs, VMs are not pulsatile, and do not have a palpable or audible thrill.

The most common symptoms are pain and swelling from blood pooling and are heavily influenced by gravity and exercise. If the lesion exhibits mass effect, nearby structures such as nerves can be affected. Intravascular thrombosis can cause pain and an inflammatory reaction, which leads to tissue hardening and phlebolith formation. Rarely, a chronic coagulopathy can be detected, especially with extensive lesions. Hemorrhage of superficial lesions can be caused by minor trauma or rubbing. Muscular strength and joint function may be affected. Patients commonly do not report symptoms during sleep, but experience gradual onset of pain while upright and walking. The pain is often relieved by rest in the recumbent position and by limb elevation.

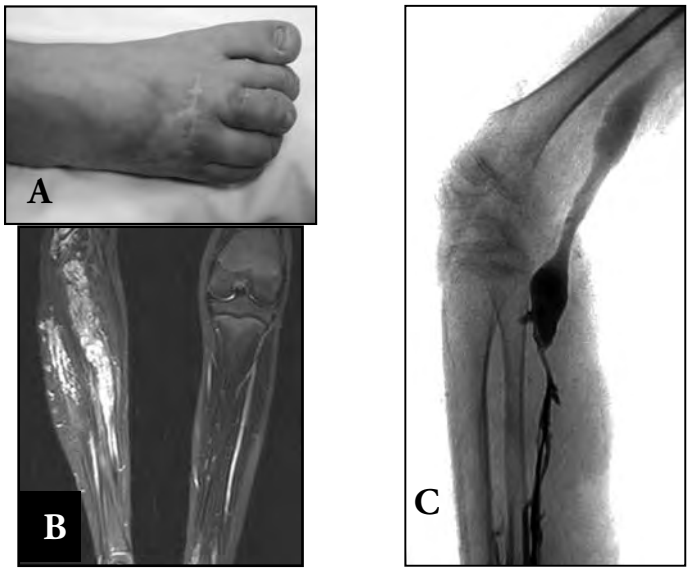
Venous malformations are prominent features of multiples clinical syndromes, and in some entities lymphatic and capillary components are essential for diagnosis. Of these, the best known is Klippel-Trenaunay, characterized by venous malformations, capillary malformations (port wine stain), lymphatic malformations, sometimes lymphedema, and hypertrophy of body parts, typically the limbs *(Figure 1 a-c, p. 44)*. Other such syndromes include blue-rubber bleb nevus, Maffucci's, Proteus, Gorham-Stout, and Bockenheimer.

Diagnostic Imaging

Multiple imaging modalities can contribute to the diagnosis and help formulate a treatment plan. Imaging is necessary to differentiate VM from other entities such as neoplasms. Radiography is of limited value, but the diagnosis can be suggested by a soft tissue mass containing calcified phleboliths. Abnormalities of adjacent bone are common. In cases of clinical syndromes associated with VM, radiography can reveal exostoses and enchondromas in Maffucci's syndrome, limb hypertrophy in Klippel-Trenaunay or bone osteolysis in Gorham's syndrome.

Ultrasonography is extremely valuable for localization and

Figure 1 Klippel-Trenaunay Syndrome



Klippel-Trenaunay Syndrome in a 13-year-old wheelchair-bound male with severe pain and irreversible contractures of the knee and ankle joints from chronic lack of use. There was no response to percutaneous sclerotherapy and very poor orthopedic rehabilitation potential. An amputation was recommended to enable management of pharmacologic dependence and to recover the lost ambulation with a prosthesis.

A. Photograph of the right foot. Note hypertrophy of toes and capillary malformations (birth marks) characteristic of this condition.

B. MRI image of the calves after intravenous administration of contrast. Notice extensive replacement of muscle by the enhancing venous malformation in the right calf compared to the normal contralateral limb.

C. Ascending venogram of the right calf shows venographic abnormalities of Klippel-Trenaunay, including hypoplastic deep venous system and focal venous dilatations, in this case involving the popliteal vein.

characterization, but particularly valuable to guide therapy. Doppler examination effectively establishes the presence of high or low flow within the lesion (differential diagnosis of VM versus AVM), which is critical to determine the therapeutic approach (percutaneous sclerotherapy versus transarterial embolization). Real-time sonographic guidance is essential during the performance of percutaneous phlebography and sclerotherapy.[8,9]

Computed tomography (CT) without and with intravenous contrast enhancement provides much more detailed information than radiography, especially for bone involvement. It is capable in demonstrating the extent of the lesion when done with intravenous contrast. Deep lesions can be targeted for percutaneous sclerotherapy using CT scan for guidance.

MRI is the single most valuable imaging modality. It has the best tissue contrast resolution, can differentiate slow-flow from high-flow lesions, has multiplanar capabilities, and intravascular contrast can provide extremely detailed vascular anatomy. No other modality can show the relationship to adjacent structures, such as neurovascular bundles, as clearly as MRI does. It is invaluable for follow up, demonstrating post-treatment improvement or complications. The MRI characteristics of VMs include high signal in the vascular channels on T2 weighted imaging and lack of flow voids on T1 weighted images, which indicate slow flow.[10] VMs enhance with intravenous contrast and can have mass effect. Involved structures such as muscle and bone often have substance loss or atrophy. A strong fibroadipose component is common. MRI can alert the diagnostician to clues that may indicate a vascular tumor rather than a vascular anomaly.

Angiography has little utility in the diagnosis and treatment of VM (but is important for AVM diagnosis and therapy). VM angiography is helpful only to evaluate an arterial component in mixed high-flow vascular anomalies. Direct puncture venography (phlebography) is not necessary for diagnosis but is an essential first step in the performance of percutaneous sclerotherapy.

Management of Venous Malformations

A multidisciplinary approach to plan and execute the therapy of VM cannot be underestimated. Multiple specialties contribute greatly, but this article emphasizes the role of percutaneous sclerotherapy, usually performed by interventional radiologists. Many lesions are detected early and managed in pediatric institutions, but presentations later in life are managed in adult facilities. Surgeons (plastic, general, vascular, orthopedic, otolaryngology), radiologists (diagnostic and interventional), dermatologists, physical therapists, and nurses are essential members of the team.

The main reasons to treat VM are pain and other congestive symptoms, mass effect, interference with daily activities, and to improve the appearance of the involved body part, especially when very visible (face, neck). Many patients do not need intervention and are satisfied with the reassurance that VM is not a neoplastic process. Congestive symptoms can be managed conservatively with limb elevation, compression stockings and anti-inflammatory medication. However, most patients require more aggressive treatment to control pain, which can be disabling. Intervention is necessary or strongly considered for hemorrhage, symptomatic mass-effect, when a nearby vital organ is at risk (usually in the head or neck), to prevent tissue breakdown and recurrent infection, or in cases of substantial cosmetic problems.[11,12]

VM may be treated with surgery and/or percutaneous sclerotherapy.[13,14] Surgery is ideal if the VM is sharply defined and can be resected without important functional loss or residual deformity. Pre-operative sclerotherapy can facilitate surgery. Usually, the lesion is too poorly defined or infiltrative for surgery without excessive tissue loss. Incomplete resection of a VM may result in an aggressive recurrence with worse symptoms.

Percutaneous sclerotherapy is considered the primary therapeutic modality for slow-flow vascular anomalies, most commonly VM. High-flow lesions such as true arteriovenous malformations typically need transcatheter techniques.[15]

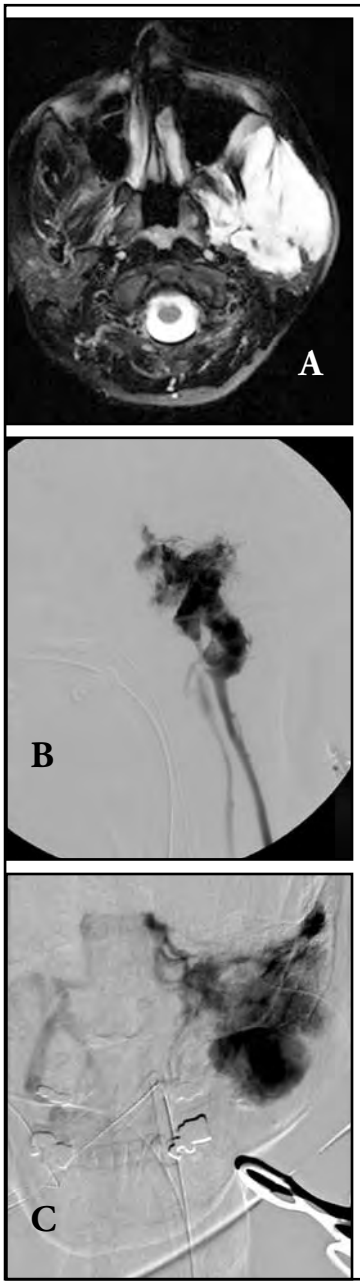
Blood pooling in the abnormal venous channels causes vascular distention and is associated with an inflammatory reaction from intravascular thrombosis, which leads to pain, congestive symptoms and mass effect. If the vascular spaces can be closed, the pain and bulk can improve substantially. This is the rationale for percutaneous sclerotherapy (*Figure 2, a-c*). The essential elements are fluoroscopy, digital subtraction venography, real-time sonography and direct puncture of the lesion to gain access into the lumen of the abnormal veins. Injection of sclerosants induces thrombosis and endothelial denudation that lead to permanent closure of the vascular lumen.[16]

The agents most commonly used are absolute ethanol, sodium tetradecyl sulfate (Sotradecol, STS), polidocanol, and ethanolamine oleate. Ethanol is injected without dilution, while the other agents can be used in their liquid form or can be transformed into foam by vigorously mixing with air. Foam sclerotherapy can be radiodense and visible fluoroscopically if mixed with lipiodol (an oily contrast agent). Ethanol is considered the most effective agent, has wide availability and very low cost; however, the risk of tissue necrosis makes it the most dangerous agent. The treating physician must ensure that the ethanol does not flow or extravasate into non-target vulnerable tissues. Local complications of ethanol are always a concern and include skin necrosis and sloughing, nerve palsy and hemolysis. Systemic complications include alcohol intoxication when large volumes are used, and cardiopulmonary compromise. Ethanol that flows quickly through a vascular anomaly may reach the pulmonary circulation in high concentration. This may potentially cause pulmonary arterial constriction, right heart compromise, bronchospasm, cardiopulmonary collapse and death.[17-19] The intravascular injection of ethanol causes intense pain and therefore mandates general anesthesia. It is prudent to keep the ethanol dose below 0.5 mL/kg, but the maximum recommended is 1 mL/kg. Although considered less effective, STS, ethanolamine and polidocanol have a safer profile than ethanol and are thus preferred by many practitioners for routine use. These agents are especially useful for higher-risk lesions due to involvement of the skin, neurovascular bundles, or delicate structures such as the face or hands.

No specific preparation is needed for the procedure, except for a pre-anesthesia evaluation and NPO status. Pre-procedural blood work is not mandatory, but can include a coagulation profile and platelet count. Prophylactic antibiotic is customary but there is no proof that this is necessary. The lesion is punctured directly by palpation, sonographic, or fluoroscopic guidance. Virtually any kind of needle can be used, but in the author's experience 18 gauge intravenous cannulas and butterfly needles are the most useful.

The direct puncture is followed by subtraction venography for confirmation of reliable intraluminal access, distribution of contrast within the lesion and to understand the venous drainage. The lesion's volume is estimated by the amount of contrast administered during the injection. Multiple punctures are usually required to treat the entire lesion. If extravasation occurs, the access is abandoned and a new one obtained. Rapid drainage into normal veins must be controlled with direct compression to avoid deep vein thrombosis. Orthopedic tourniquets are usually unnecessary for VMs, but can be very helpful for AVMs.

Figure 2 Craniofacial Venous Malformation



42-year-old female with a left craniofacial venous malformation that caused face bulging in the recumbent position and contour excavation in the upright position. Percutaneous ethanol sclerotherapy caused palsy of cranial nerves V and VII, with recovery after 3 months. The patient was very satisfied with the final result but this case underscores the risks of ethanol use.

A. MRI after intravenous administration of contrast showed engorgement of the venous malformation and marked atrophy of the masticator muscular compartment.

B. Initial percutaneous phlebography demonstrated immediate drainage into the internal jugular system, which increases the risk of deep vein thrombosis and cardiopulmonary complications from sudden flow of ethanol into the pulmonary circulation.

C. Repeat percutaneous phlebography with compression of the outflow vein redirected the outflow into the venous channels of the base of the skull. This probably accounts for the cranial nerve compromise.

Once the lesion has been thoroughly treated, all access needles and cannulas are removed and hemostasis is obtained with digital pressure. For large lesions, multiple sclerotherapy sessions may be necessary. Typically the patient is discharged from the hospital after a 3-hour observation. Longer observation or overnight hospital stay may be necessary if the anesthesia recovery is slow, the patient requires intravenous narcotics, or if nausea, vomiting, and problems such as urinary retention occur. Post-procedural pain has an inflammatory component

related to the intravascular thrombotic process induced by the sclerosant. For this reason non-steroidal anti-inflammatory medication can be very helpful.[20]

Inflammation, pain and swelling are expected for one or two weeks. As the acute post-procedural pain subsides, the patient typically notices an improvement from the congestive pain. A decrease in mass-effect and bulk can take 1 to 3 months. Additional sessions of sclerotherapy are needed only if symptoms persist.

Complications

Minor procedure complications include increased pain, swelling and skin discoloration. Skin blisters can resolve quickly and completely. Major complications include full skin necrosis that can require extensive local therapy and several months to completely heal. Skin grafting or other surgical procedures are only rarely necessary. Nerve damage can cause paresthesias, numbness or muscular weakness that typically improves gradually over 3 months. Residual permanent numbness is common with lesions that extensively involve the skin and subcutaneous tissues. If ethanol escapes excessively into the normal venous drainage, deep vein thrombosis is possible. Complications can be expected in 15% of procedures when using alcohol.[21] Other sclerosants are associated with a lower complication rate, but can be less effective.

Outcomes

Scientifically acquired outcomes data for percutaneous sclerotherapy of VM are scant. Most results are reported in vague terms and subjectively. However, most patients can expect an improvement in pain and functionality. In one large series of multiple sessions of ethanol sclerotherapy greater than 90% of patients achieved "fair" to "good" results. A "poor" result was experienced by about 5% of patients.[15] Improvement can be expected in about 70% of cases with sodium tetradecyl sulfate.[16] Quality of life analysis is scant. In a small series of 24 patients, a prospectively quality of life questionnaire showed that 16 of 24 patients demonstrated improve-ment. Poor outcome was predicted by extensive involvement of a muscular compartment.[22]

Conclusion

Venous malformations are an important manifestation of errors in vascular embryogenesis within a larger context of vascular malformations that include arterial, lymphatic, and capillary lesions. Historically this pathology was described and classified based on macroscopic observations, creating a confusing terminology with no consistent reference to the lesion biology and hemodynamic behavior. Much effort has been made in refining diagnostic classifications applying current understanding of the biology of the disease and to provide a framework with helpful therapeutic implications. Appropriate classification combines information obtained with the physical examination and imaging studies that include ultrasound, CT scanning and MRI. The hemodynamic pattern of the lesion is confirmed with percutaneous venography, and the treatment is accomplished with combined venographic/fluoroscopic and ultrasonographic guidance. Though surgical techniques are important in selected cases, the foundation of modern treatment is percutaneous sclerotherapy. In most cases pain, mass effect, hemorrhagic tendency and other clinical manifestations can be expected to improve with this approach.

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Medicare Quality Reporting & the Supreme Court Decision

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In late June the United States Supreme Court will decide the fate of the Patient Protection and Affordable Care Act ("ACA"). The key decision will center on whether or not the individual mandate to purchase insurance is an appropriate exercise of congressional power under the commerce clause of the United States Constitution. If the Court determines that it is not, then the Court will address the issue of severability of the individual mandate from the rest of the ACA. If the individual mandate is severable, then the remainder survives including various provisions impacting physicians serving Medicare beneficiaries.

If, instead, the court strikes down the entire law, physicians will feel an immediate impact from elimination of a variety of programs including certain key Medicare reimbursement and quality reporting reform provisions already implemented. Among the ACA's reimbursement and quality reforms already in place are the extension of the Physician Quality Reporting Initiative (PQRI), the Physician Feedback Program and the Physician Compare Website.

A finding that all of ACA falls would directly affect providers participating in these programs and any additional reimbursement that they may receive from such programs unless they are separately re-authorized through additional Congressional action. This article provides an overview of what these programs are and how physicians are impacted by the programs as well as the decision of the Supreme Court.

Physician Quality Reporting Initiative Extension

The Patient Protection and Affordable Care Act of 2010 (PPACA) made several changes to Medicare's voluntary reporting program known as the Physician Quality Reporting Initiative (PQRI). The key changes include extending the program for eligible physician/provider types and, effectively, making participation mandatory for eligible physician/ provider types beginning in 2015. The program offers bonuses to physicians/providers who report on designated quality measures. Group practices are also eligible to participate. The final rule interpreting the changes to the program from PPACA was issued by the Department of Health and Human Services on November 29, 2010.

PPACA, and rules interpreting the PQRI program accomplish the following: 1.) extend PQRI incentive payments through 2014, 2.) implement a penalty for physicians who do not report quality measures beginning in 2015, 3.) provide for an additional bonus to physicians who meet the requirements of a continuous assessment program known as the Maintenance of Certification Program ("MOCP"), and 4.) implement a subsequent penalty for those who do not meet the MOCP standards post 2015.

In 2012, individual eligible physicians who meet the criteria for participation in and submission of quality measures data will qualify to earn an incentive payment equal to 0.5% of their total estimated Medicare Part B Physician Fee Schedule (PFS) allowed charges for covered professional services furnished during that same reporting period. This is a reduction from 1% in 2011 as provided for in the ACA.

A group practice may also potentially qualify to earn Physician Quality Reporting incentive payments equal to 0.5% of the group practice's total estimated Medicare Part B PFS allowed charges for covered professional services furnished during their 2012 reporting period based on the group practice meeting criteria specified by CMS.

Physicians may now participate for either 6 months or 1 year unlike in previous years and submit quality data on that same time basis.

If the ACA is found unconstitutional in its entirety, three major impacts to the PQRI program occur: 1.) the program effectively ceases to exist, 2.) participating providers no longer have the opportunity to earn additional incentive payments, and 3.) providers will not face the prospect of penalties for failing to participate or meet MOCP standards after 2015. Of immediate concern is what happens to existing providers who are currently participating in the program and who have received advanced incentive payments for a reporting period that goes beyond June of 2012. It is unclear what action, if any, CMS would take regarding such advanced incentive payments.

For more information, go to:https://www.cms.gov/Medicare/Quality-Initiatives-Patient-Assessment-Instruments/PQRS/index.html?redirect=/PQRS/.

Physician Feedback Program

The Physician Feedback Program was designed by CMS to provide comparative performance information to physicians in an effort to improve the quality and efficiency of medical care. The purpose, from CMS's website, is to provide meaningful feedback to physicians and move toward a reimbursement system that rewards value rather than volume. The program was initially created by Medicare Improvements for Patients and Providers Act of 2008 and extended and expanded by the ACA.

The program contains two primary components: **1)The Physician Quality and Resource Use Reports ("QRURs").** The QRURs are intended to provide simple & transparent performance results to encourage more efficient and higher quality clinical practice by physicians. These reports will initially be e-mailed to providers in Iowa, Missouri, Kansas and Nebraska in early 2012 with expansion to the rest of the country at a later date; 2)**The Development and implementation of a Value-based Payment Modifier (VBPM)**. Beginning in 2015, for physicians who will be impacted by the VBPM, the QRURs will contain composite measures of quality and cost that display the bases for the VBPM. By

Continued p.50

Heart Disease Control: Successes and Challenges

James B. Tidwell, MPH; Niketa Walawalkar, MD, MPH; and Robert Harmon, MD, MPH

Heart disease has been the leading cause of death in the United States since 1921. Almost 600,000 or one-fourth of all American deaths were due to heart disease in 2009, according to the CDC.

Although death rates have decreased nationally since 1950, several risk factors for heart disease continue to increase. Innovative treatments have contributed to lowering the death rate, but they have also greatly increased the cost of care. The American Hearth Association estimates that heart disease treatment costs will triple from $273 billion in 2010 to more than $800 billion by 2030. Therefore, the prevention of heart disease must remain a public health priority.

Heart disease is also an important concern locally. It accounted for 1,532 deaths in Duval County and 41,241 deaths statewide in 2010. These represent 22% and 24% of all deaths in those areas. Following national trends, death rates are also declining locally. Though the age-adjusted death rates from heart disease were 280.9 per 100,000 population in Duval County and 247.2 in Florida in 2000, by 2010 the rates had dropped to 170.3 and 147.7, respectively. Rates decreased for both whites and blacks in Duval County during the same time period (from 315.5 to 205.8 for blacks and from 259.6 to 158.4 for whites) as well as for both males and females (from 326.6 to 194.7 for males and from 219.6 to 149.7 for females).

Remarkable progress has occurred in reducing heart disease death rates. Long-term downward trends in some population risk factors for heart disease such as tobacco use and saturated fat intake have reduced its burden. Medical advances including better drugs for preventing and treating heart disease and more effective surgical procedures such as bypass surgery, angioplasty, and the use of stents have also saved lives.

Despite these advances, heart disease remains a leading cause of death. Obesity, poor control of blood pressure, and poor control of cholesterol are some of the major preventable causes of heart disease. The Behavioral Risk Factor Surveillance System (BRFSS) is a national survey of adults that produces county-level estimates of many health behaviors. The BRFSS reports that 28.4% of adults in Duval County were obese and an additional 36.0% were overweight in 2010. Between 2002 and 2010, the percentage of adults considered overweight or obese increased 15.0% in Duval County and those considered obese increased 33.0%.

No local data are available for blood pressure or cholesterol control, but the CDC estimates that half of adults with high blood pressure and nearly two-thirds of adults with high cholesterol do not adequately control their conditions. Heart disease is clearly an enormous public health problem best addressed by strategies targeting the preventable causes of the disease.

The Duval County Health Department (DCHD) has several programs under the Community Cardiovascular Health Program's (CCHP) "Hearts With Spirit" initiative. Started in 2002 through a grant from the CDC, the CCHP began with a survey that found that a large percentage of patients said their physicians had not addressed their risk factors. The chief recommendation was to support the establishment of clinical protocols for physicians to regularly assess their patients' risks. Now it has expanded to include multiple projects aimed at addressing unhealthy behaviors including "Hearts N Motion", "Cardio Kids", "Moving Against Diabetes", the "Gateway Shopping Center Walking Club", "Nutricize", Adult and Youth Smoking Cessation Referrals, "Chain of Survival", and the Signs and Symptoms of Heart Disease survey.

Much still remains to be done to reduce the impact of heart disease in Duval County. There is no good surveillance system that monitors heart disease incidence at either the national or local levels and so the effectiveness of programs is difficult to evaluate. However, community efforts to control high blood pressure, emergency care guidelines to improve heart attack response, and programs that target obesity, exercise, and nutrition have been shown to be effective.[1] The implementation, local adaptation, and evaluation of these programs could have great impact on the prevalence of heart disease.

Additionally, most existing programs through Healthy Jacksonville and DCHD target the behavioral causes of heart disease, but better detection of high blood pressure and high cholesterol and more effective patient education about the control of these conditions are also essential to reducing the problem. Physicians, community organizations, government, health insurance companies, and many other stakeholders all have a role to play and an interest in controlling heart disease. Only a coordinated, long-term effort will be effective in reducing the burden of heart disease in Duval County.

Endnote

[1] CDC, Heart Disease and Stroke Prevention—Addressing the Nation's Leading Killers: At A Glance 2011, accessed from http://www.cdc.gov/chronicdisease/resources/publications/AAG/dhdsp.htm.

Continued from p.48

Medicare Quality Reporting

2017, the ACA applies VBPMs to almost all physicians paid under the Medicare fee for service program.

While there is no immediate impact from a finding that the ACA is unconstitutional for most physicians, the VBPM will be eliminated which means that physician fee for service reimbursement will not be adjusted by its' application starting in 2015. However, QRURs may still be created and mailed although there is no direct statutory authority for such action.

For more information see:https://www.cms.gov/Medicare/Medicare-Fee-for-Service-Payment/PhysicianFeedbackProgram/index.html?redirect=/PhysicianFeedbackProgram/

Physician Compare Website

On January 1, 2011 CMS launched the CMS Physician Compare website. Phase 1 of the website was to provide information to consumers regarding physician's name, address, practice areas, and Medicare participation status. Data on the site comes from the Medicare Provider Enrollment, Chain and Ownership System. Initially, some physicians were concerned with the biographical data as it contained some errors.

Starting January 1, 2013, the ACA requires CMS to publish physicians' quality performance data, based on the prior reporting year. Such performance information to be published on the website includes, but is not limited to:

- measures collected under the PQRI;
- an assessment of patient health outcomes and the functional status of patients;
- an assessment of the continuity and coordination of care and care transitions, including episodes of care and risk-adjusted resource use;
- an assessment of efficiency;
- an assessment of patient experience and patient, care giver, and family engagement;
- an assessment of the safety, effectiveness, and timeliness of care; and
- other information as determined appropriate by the Secretary.

Currently the system is voluntary but, as noted before, quality reporting becomes mandatory starting in 2015. In 2009, over 200,000 physicians and other health care professionals servicing Medicare beneficiaries used the voluntary system. Providers who currently do no participate in the PQRI will have significantly less quality data present on the website for at least the years 2013-2015 which may result in questions from patients who are looking for quality data.

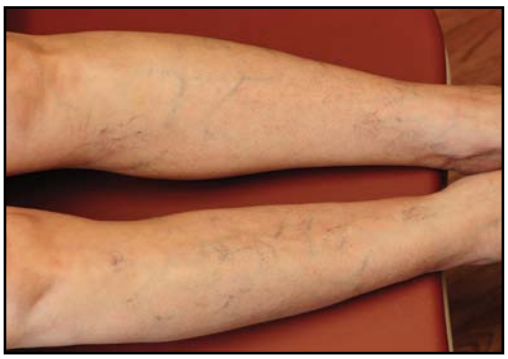
If ACA is found unconstitutional, without reauthorization CMS would not have the statutory authority to expand the Physician Compare website to include the voluntary reporting data outlined above.

To access the current information on the CMS Physician Compare Website, please refer to the following link:http://www.medicare.gov/find-a-doctor/provider-search.aspx.

Color Figures for Earlier Articles

Evaluation and Treatment for Lower Extremity Superficial Venous Insufficiency (p.21)

1



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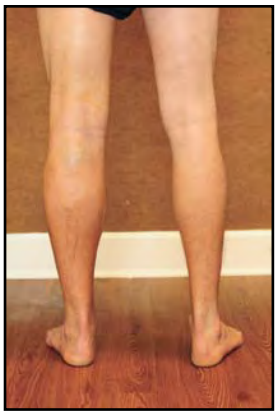

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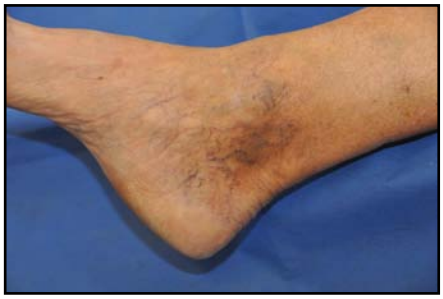

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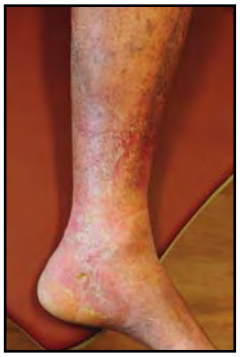

(Top to bottom) Fig. 1 - Telangiectasis (spider veins) and reticular veins of the leg; Fig. 2 - Lower extremity edema (note swollen left calf - best appreciated posteriorly); Fig. 3 - Hyperpigmentation of the medial ankle; and Fig. 4 - Advanced venous stasis skin changes (exzema, hyperpigmentation, lipodermatosclerosis, erythema/rubor and ulceration.

Venous Stasis Ulcers: A Review (p.29)

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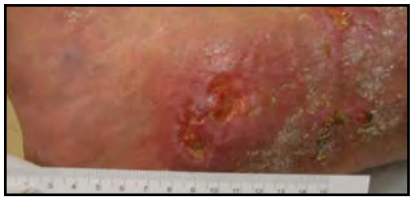

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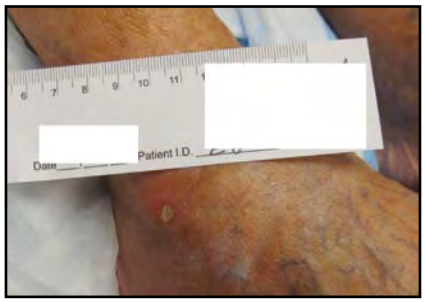

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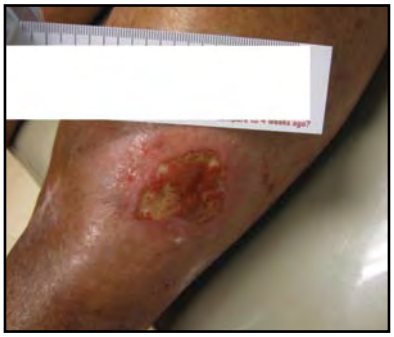

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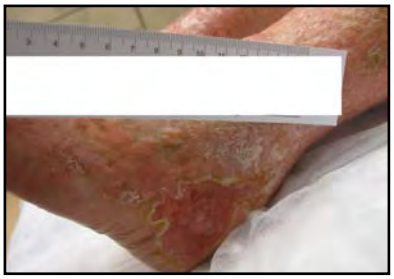

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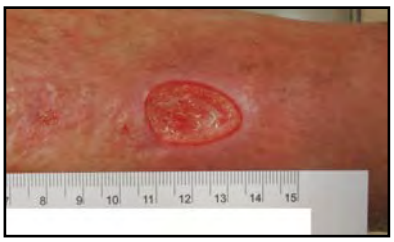


(Top to bottom) Fig. 1 - Shallow irregular venous ulcer with exzematous changes of venous stasis; Fig. 2 - Small, shallow, round, smooth arterial ulcer in patient with venous disease and early sign of corona phlebectatica; Fig. 3 - Venous ulcer with signs of "atrophie blanche", darkened hemosiderin deposits, early lipodermatosclerosis with beginning of "champagne bottle" leg; Fig. 4 - Venous ulcer in an atypical location: lateral heel. It has exzema and signs of beginning lipodermatosclerosis; and Fig. 5 - Smooth, oval ulcer is an atypical arterial ulcer with a moist appearance and redness mimicking a venous ulcer.

These physicians' applications for membership in the Duval County Medical Society are now being processed. Any information or opinions you may have concerning the eligibility of the applicants listed here may be directed to Ashley Booth Norse, MD, DCMS President (904-244-4106) or Barbara Braddock, Membership Director (904-355-6561 x107).

Laura Armas-Kolostroubis, MD
Internal Medicine
UF Rainbow Center
Medical Degree: LaSalle University
Residency: Texas Health Presbyterian Hospital
Fellowship: Dallas VA Medical Center & New York State Department of Health

Jannifer D. Harper, MD
Internal Medicine
BCBS Florida
Medical Degree: Case Western Reserve University
Residency: Case Western Reserve University Hospitals

Jonathan Kantor, MD
Dermatology
North Florida Dermatology
Medical Degree: University of Pennsylvania School of Medicine
Residency: University of Pennsylvania School of Medicine

Rachana A. Patel, MD
Ophthalmology
UF Ophthalmology
Medical Degree: University of Florida College of Medicine
Internship: University of Florida College of Medicine
Residency: Medical College of Georgia
Fellowship: University of Minnesota

Raymond M. Pomm, MD
Addiction Psychiatry/General Adult Psychiatry
River Region Human Services, Inc.
Medical Degree: Meharry Medical College
Internship/Residency: Penn State University

Hagop M. Tabakian, MD
Anesthesiology/Pain Management
Center for Pain Management/UF
Medical Degree: American University Beirut
Residency: St. Agnes Hospital & University of Maryland Medical System
Fellowship: University of Maryland Medical System

J. Michael Walker, MD
Pediatrics/Developmental-Behavioral Pediatrics
UF Developmental Pediatrics
Medical Degree: American University of Beirut
Residency: Medical College of Ohio at Toledo
Fellowship: Cincinnati Children's Hospital Medical Center

Daniel R. Wilson, MD, PhD
Psychiatry
VP Health Affairs/Jacksonville Dean/ UFHSCJ
Medical Degree: University of Iowa
Residency: Mount Auburn Hospital, McLean Hospital, Brigham & Women's Hospital, and Bridgewater State Hospital.

2012 BEALS AND SHAHIN AWARDS

If you have published peer reviewed articles in 2011, you are eligible to submit for the Beals and Shahin Awards. Categories are: Original Investigation, Clinical Observation and Review Articles.

The submission process begins in June, so watch E-News, dcmsonline.org and faxes. All winners will be recognized at the DCMS Annual Meeting in late November.

Questions? Contact Laura Townsend at 355-6561 x101 or email her at ltownsend@dcmsonline.org.

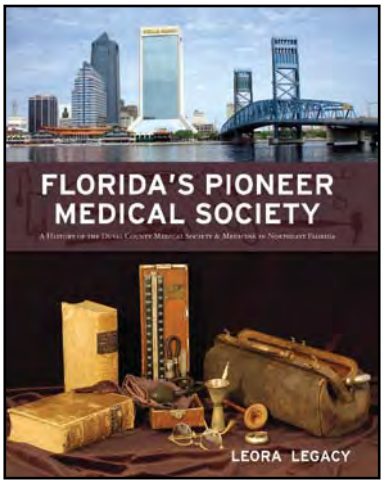
Ole' Time Reunion is Living History






(L to R, top) Attending the 2012 Good Ole' Time Reunion, (for DCMS members who practiced during the "Golden Age" of medicine before 1970) were Dr. George Trotter and Dr. Jim Beale; Dr. & Mrs. Charles McIntosh; and Dr. & Mrs. Howard Hogshead.

(L to R, bottom) Also at the 2012 reunion were Dr. & Mrs. Ferdinand Berley and Mrs. Margaret Ludwig; DCMS Past Presidents at the 2011 reunion were Dr. George Trotter, Dr. C. Ted Montgomery, Dr. Gene Glenn and Dr. Faris Monsour; and DCMSA Past Presidents at the 2011 reunion included Mrs. Jerry Ferguson (unofficial reunion coordinator), Mrs. Ruth Glenn, Mrs. Margaret Fleet and Mrs. Margaret Ludwig.



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