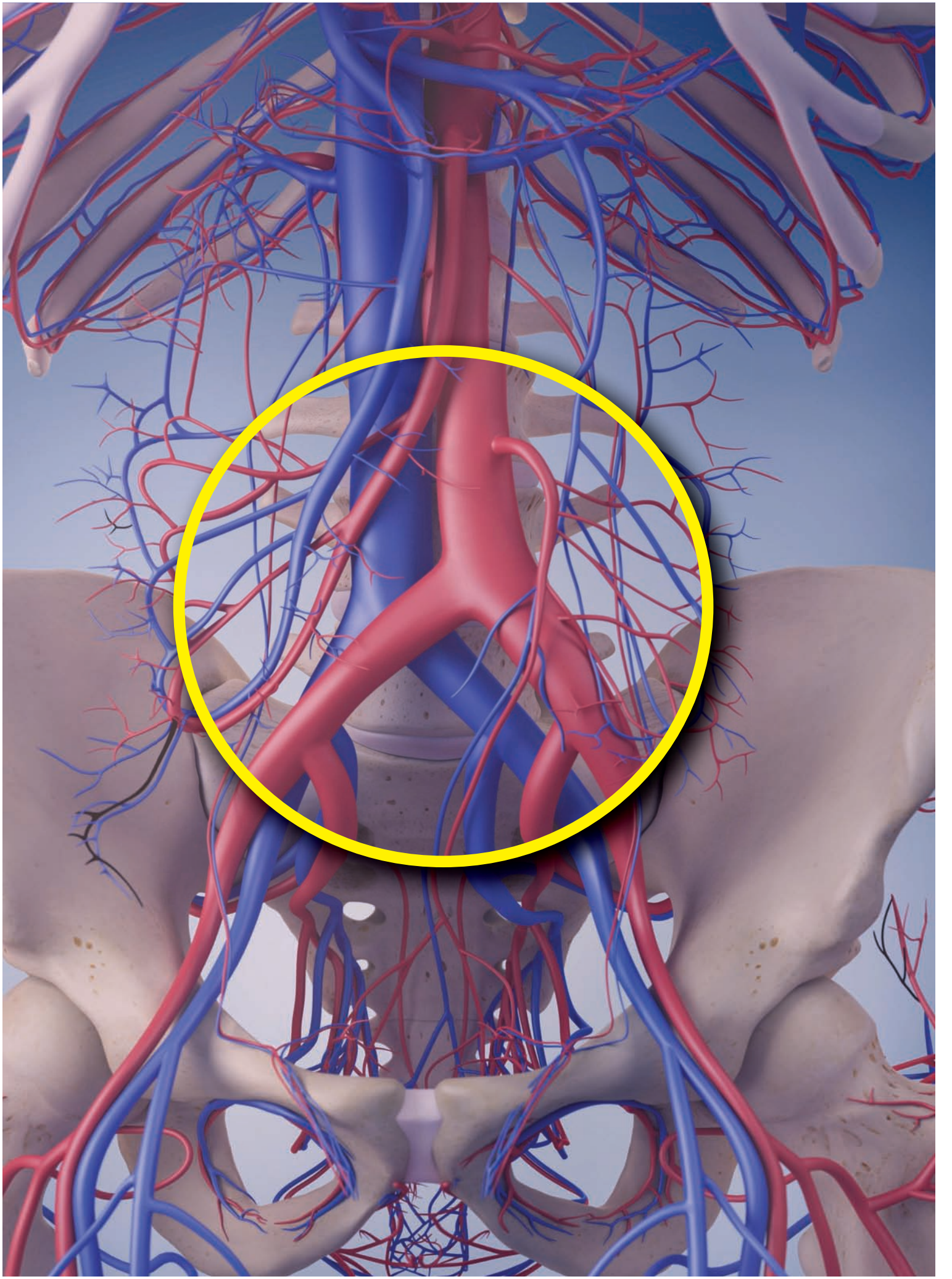

Acute iliofemoral DVT in the presence of

May-Thurner syndrome

By Susan K. Adams, BSN, RN, CCRN, and Inga Sinyangwe, MSN, RN

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MAY-THURNER SYNDROME (MTS), also known as iliac vein compression syndrome, occurs when the left common iliac vein is compressed by the overlying right common iliac artery against the underlying lumbar vertebrae.[1] Patients with this syndrome are predisposed to deep vein thrombosis (DVT) and pulmonary embolism (PE), together known as venous thromboembolism (VTE).

Iliofemoral vein thrombosis accounts for about 25% of all lower-extremity DVTs. Compared with DVTs occurring below the knee, iliofemoral vein thrombosis is associated with a greater risk of PE, malperfusion, and post-thrombotic syndrome.[1] This article discusses the mechanisms of MTS and the interventional therapies available to improve the patient's quality of life and prevent potentially life-threatening complications.

VTE in the news

After 39-year-old reporter David Bloom died of PE in 2003 while covering the Iraqi invasion, VTE gained national attention as media coverage highlighted the cause of this young man's death.[2] Subsequently, The Joint Commission, partnering with the National Quality Forum in 2005, developed guidelines to decrease the incidence of VTE.[3] In 2008, the United States Surgeon General called for further national initiatives to prevent VTE.

The focus of this article is acute iliofemoral DVT caused by MTS. Although the potential for fatal PE is the most concerning risk, chronic venous insufficiency from postthrombotic syndrome (PTS) consumes healthcare dollars, negatively affects quality of life, and predisposes individuals to chronic venous ulcerations and recurrent DVT.[1] (See *Understanding PTS and chronic venous insufficiency*.)

Pathophysiology of VTE

Venous thromboembolic disease may begin from physiologic and/or anatomical abnormalities. The primary components of thrombi are platelets and fibrin. Arterial thrombi differ from venous thrombi in that arterial thrombi usually result from ruptured atherosclerotic plaque or other intimal injury. Because these thrombi, called *white clots*, are platelet-rich, they're treated with anti-platelet drugs. In contrast, venous thrombi (the focus of this article), or *red clots*, are fibrin-rich and contain more red blood cells, so they're treated with anticoagulation.[4]

Most DVTs arise from the venous system in the lower extremities. However, MTS is associated with unprovoked left iliofemoral DVT or chronic venous insufficiency.[4,5]

Several risk factors predispose an individual to VTE. In the 1800s, the physician Rudolf Virchow was the first to note the presence of three abnormalities in blood flow that contribute to an increased risk of venous thrombosis: venous stasis, hypercoagulability, and damage to the endothelial cells within the vascular wall. These factors are known as *Virchow's triad*.[4,6]

Risk factors can also be classified as nonmodifiable and modifiable. Nonmodifiable risk factors include factor V Leiden mutation or other inherited hypercoagulable states such as Protein S deficiency, older age, female gender, and anatomical abnormalities that impede venous flow (such as in MTS).[5] Modifiable risk factors include use of menopausal hormone therapy or oral contraceptives, prolonged immobilization, prolonged standing, and obesity. Surgery, cancer, pregnancy, and acute infection also raise the risk.[4,7,8]

Looking closer at MTS

MTS is one anatomic risk factor contributing to VTE that has been identified through improved diagnostic imaging. Although first documented by Virchow in 1851, it was more fully explained a century later by physicians May and Thurner during extensive autopsy examinations.[9]

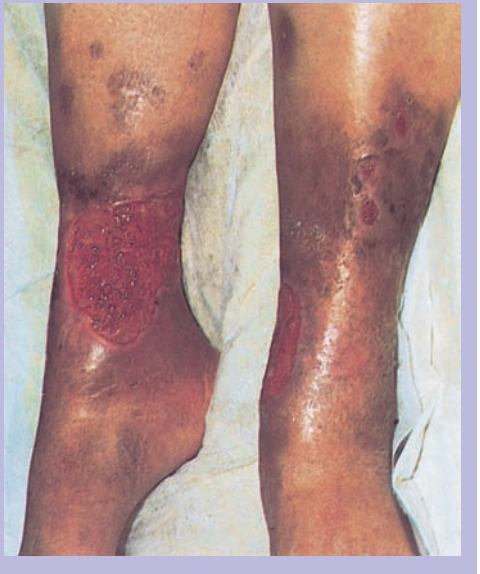
MTS occurs when the right common iliac artery overlies and compresses the left common iliac vein against the underlying lumbar vertebrae, impairing blood flow, causing venous stasis, and potentially resulting in iliofemoral DVT.[10] Further intimal injury occurs in the left common iliac vein from the pulsations of the adjacent right common iliac artery.[10,11]

Although research shows that MTS affects over 20% of the population, it's often not considered in the differential diagnosis of DVT and is frequently overlooked.[12] However, this anatomical variant has become more widely recognized as improved diagnostic and interventional modalities for venous thrombosis have been developed.

Signs and symptoms

The first presentation of MTS may be unprovoked left iliofemoral DVT accompanied by pain and edema of the left lower extremity.[5] Less commonly, if the proximal clot burden is large, arterial compromise

Understanding PTS and chronic venous insufficiency[5,14,20]



Formerly called postphlebitic syndrome, postthrombotic syndrome (PTS) describes development of signs and symptoms of chronic venous insufficiency following DVT, such as pain, venous dilation, edema, hyperpigmentation and other skin problems, and venous ulcers. Risk factors include older age, personal or family history of venous disorders, prolonged standing or sedentary lifestyle, pregnancy, and advancing age. Patients may complain of a heavy feeling or aching in the legs, muscle cramps, and skin discomfort, such as a tight feeling, itching, or dry skin. Contributing factors include reflux from valvular incompetence and venous hypertension from thrombotic obstructions. DVT prophylaxis, early treatment of DVT, and prevention of recurrent DVT help reduce risks associated with this debilitating and costly complication.

called *phlegmasia cerulea dolens* may occur in the affected lower extremity. (See *Phlegmasia cerulea dolens: Rare but ominous*.) Emergent intervention is needed to restore arterial blood flow. If untreated, hypotension and progression to hypovolemic shock may ensue, and the patient may lose the affected limb or die.[13]

As the acute phase of DVT resolves, the patient may experience persistent signs and symptoms as a result of lower extremity chronic venous insufficiency, which significantly negatively impacts economic resources and the patient's health status, psychological well-being, and overall quality of life.[12,14]

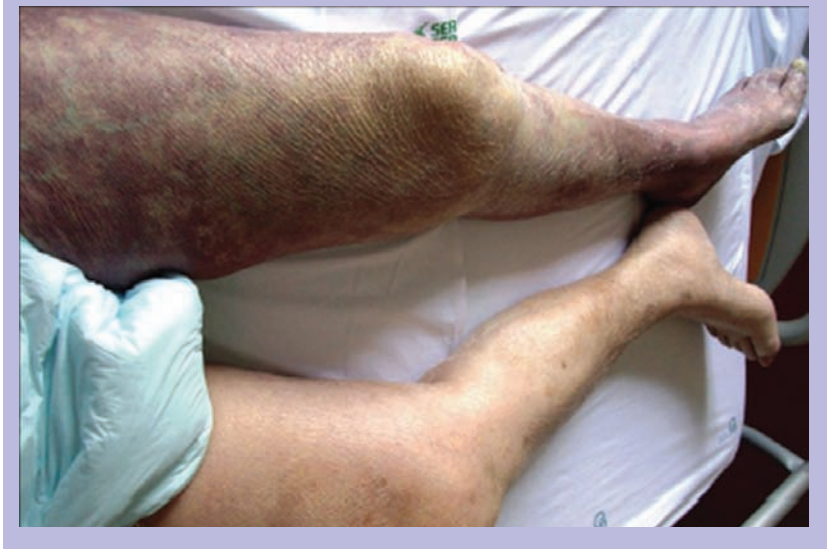
Diagnosis

For patients presenting with signs and symptoms related to chronic venous disease, with or without acute venous thrombosis, clinicians must obtain a detailed health history and physical assessment.[12] The history should include questions pertaining to progression of signs and symptoms, previous DVT, and identification of VTE risk factors. Physical assessment should include careful inspection and palpation of the lower extremities for skin color and temperature changes, unilateral edema, and varicosities. To assess for coexisting arterial disease, palpate and grade femoral, popliteal, and dorsalis pedis and posterior tibial pulses. Also assess deep tendon reflexes, muscle strength, and sensation.[7,10]

Appropriate lab studies include a complete blood cell (CBC) count, basic metabolic profile, coagulation panel, and a d-dimer. A fibrin degradation product, d-dimer elevation is present in nearly all patients with VTE. However, because hospitalized patients often have elevated d-dimer levels for various other reasons, it's not specific for diagnosing VTE and is useful only when negative.[10]

Phlegmasia cerulea dolens: Rare but ominous[15]

Massive iliofemoral venous thrombosis of the lower extremities causes sudden severe leg pain with cyanosis, edema, venous gangrene, compartment syndrome, and arterial compromise. Treatment delay may result in circulatory collapse, shock, loss of the leg, and/or death.



Physical assessment should be accompanied by compression ultrasonography of the lower extremities.[10] One limitation of compression ultrasonography for diagnosing MTS is that it doesn't detect isolated thrombi in the iliac vein or that portion of the femoral vein within the adductor canal. Other imaging studies that may be performed to assess the degree of stenosis and the hemodynamic effects of iliac vein compression in MTS include:[10,12]

- impedance plethysmography
- contrast venography
- computerized tomography (CT) venography
- intravascular ultrasound
- magnetic resonance venography.

Medical management of DVT

Once a diagnosis of acute DVT in the presence of MTS is made, anticoagulation should be started immediately in most patients because a delay in therapy may increase the risk of potentially life-threatening PE.[5,15] This initial anticoagulation should continue for at least 5 days.[15] Treatment options include low molecular weight heparin (LMWH) such as enoxaparin, fondaparinux, I.V. unfractionated heparin (UFH), or subcutaneous UFH. Oral anticoagulation with a vitamin K antagonist such as warfarin may be overlapped with LMWH, fondaparinux, or UFH for 4 to 5 days. Long-term oral anticoagulation should be prescribed for at least 3 months and may be continued for 6 to 12 months or even indefinitely for some patients. Options for long-term oral anticoagulation include direct factor Xa inhibitors such as rivaroxaban, thrombin inhibitors such as dabigatran, and vitamin K antagonists; subcutaneous options include LMWH and fondaparinux.[15]

Prior to discharge, patients must be educated about their prescribed medication therapy and modifiable risk factors should be addressed. For example, patients should be encouraged to optimize weight and follow a regular exercise program. They should develop strategies to interrupt prolonged periods of inactive sitting or standing, and they should wear medical-grade graduated compression stockings unless contraindicated. If a patient is on hormone therapy, its use should be evaluated and discontinued if possible. Although no clear link has been established between the risk of tobacco use and VTE, smoking cessation should be strongly encouraged because smoking impacts overall comorbidities and mortality.[16]

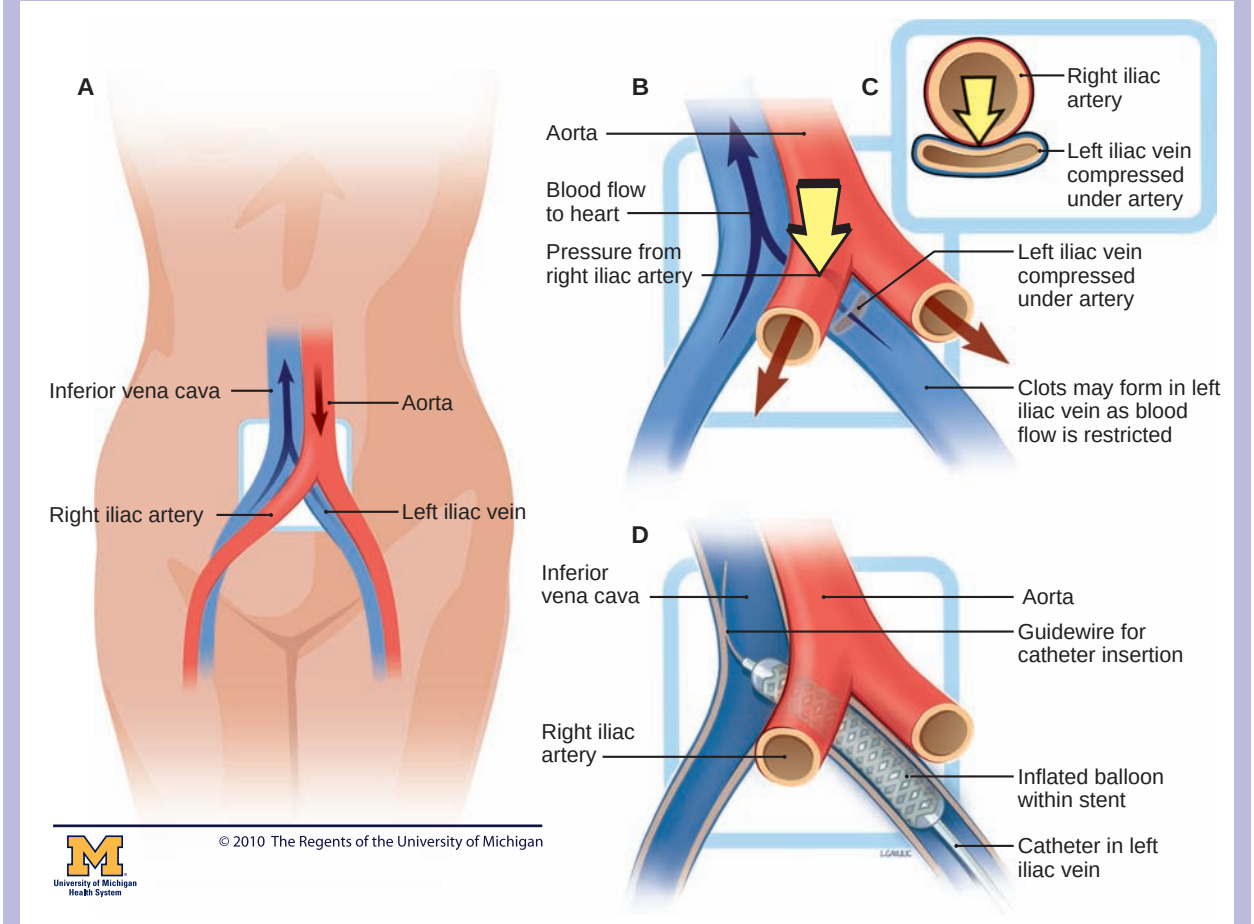
Interventional therapies and nursing care

Uncorrected, the anatomic abnormality associated with MTS may lead to recurrent DVT and other complications.[12] In addition to anticoagulation, catheter-directed fibrinolytic therapy, with or without catheter-directed thrombectomy, may be an option for patients with MTS and acute massive iliofemoral DVT or phlegmasia cerulea dolens with symptoms for less than 2 weeks and good functional status. Balloon angioplasty and endovascular stent placement may be used to correct the stenosis and restore optimal blood flow in the stenosed left common iliac vein.[5] (See *MTS and endovascular stent placement*.)

Preprocedure lab work for patients with MTS undergoing venography with endovascular intervention should include assessment of baseline renal function because of the

MTS and endovascular stent placement

MTS involves the compression of the left iliac vein by the overlying right iliac artery (A, B, and C). Treatment of MTS includes left iliac vein balloon angioplasty and endovascular stent placement, as shown in D.



administration of intravascular contrast media; a CBC count to screen for anemia and thrombocytopenia; and a coagulation panel to determine activated partial thromboplastin time and prothrombin time/international normalized ratio if anticoagulation had been started or if fibrinolytic therapy administration is planned. Patients must also be screened for allergies, including a history of any allergy-like reaction to contrast media. Ensure the patient has a patent peripheral venous access.

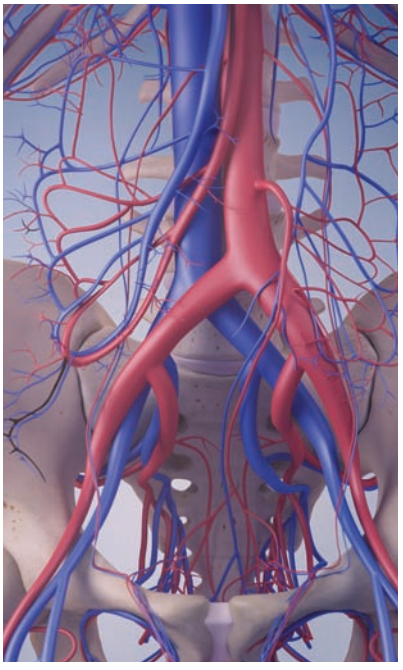
Immediate postprocedure care involves obtaining frequent vital signs and monitoring for signs and symptoms of occult bleeding. Assess the catheter insertion site for active bleeding and signs of infection. Encourage oral fluid intake and administer I.V. fluids as prescribed to prevent dehydration and contrast-induced nephrotoxicity (CIN).[17]

Before discharge, patients should be educated about the need for follow-up lab work as indicated, depending upon the type of anticoagulation therapy prescribed. Teach patients the signs and symptoms of bleeding while on anticoagulant therapy and about the importance of medication adherence, keeping follow-up appointments, and contacting the healthcare provider about potentially serious complications. Patients also need to be taught signs and symptoms of DVT and PE and what to do should these occur. Lifestyle modifications should be promoted as indicated.

Complications

Risks associated with endovascular intervention may include acute adverse reactions to intravascular contrast media, bleeding related to fibrinolytic therapy and anticoagulation, early and late stent thrombosis, stent migration (rare), and CIN.[7]

CIN has been identified by some studies to be the third leading cause of acute kidney injury experienced by patients in the acute care setting.[18] According to the American College of Radiology, there are no standard criteria for the diagnosis of CIN.[17] One of the most commonly used criteria has been an absolute increase of 0.5 mg/dL over a baseline serum creatinine. However, creatinine levels are influenced by other factors such as gender, race, and age. It's also unclear whether acute kidney injury is the direct result of contrast exposure or caused by a combination of factors. Medications such as nonsteroidal anti-inflammatory drugs, metformin, and amphotericin B may all impact renal function in the presence of intravascular contrast media, as can hypovolemia.[17,18] Strategies to prevent CIN include avoidance of volume depletion and nonsteroidal anti-inflammatory drugs, which can increase renal vasoconstriction.[19]



MTS is one anatomic risk factor contributing to VTE that has been identified through improved diagnostic imaging.

Case study

MK, 54, had a history of left iliofemoral DVT at age 24 while pregnant. At that time, she was treated with UFH until the delivery of a healthy boy. Immediately postpartum she was placed on warfarin for 6 months.

During her second pregnancy, MK was placed on subcutaneous UFH every 12 hours for DVT prophylaxis for her second and third trimesters. She continued this regimen for 3 months postpartum with no recurrence of DVT. She remained a nonsmoker, maintained optimal weight and exercised regularly. She had no comorbidities.

At age 40, MK experienced edema, pain, fatigue, and a heavy feeling in her left lower extremity as well as skin color changes and varicosities. CT venography showed a complete occlusion of her left common iliac vein, pelvic venous congestion, and extensive venous collaterals. MTS was diagnosed and MK was referred for intervention.

MK underwent successful endovascular stenting of the left common iliac vein and was discharged on enoxaparin every 12 hours and daily aspirin. She remained on enoxaparin for 6 months poststent placement. After 6 months, she continued to take low-dose daily aspirin as directed. Follow-up at 1 year revealed continued stent patency and the patient reported symptom improvement, increased activity tolerance, and an overall improvement in quality of life.

Achieving successful outcomes

As technologic advancements evolve, quality of life may be improved by patients with MTS who undergo

interventional therapies, but long-term surveillance for ongoing stent patency is crucial. Early intervention, long-term follow-up, and patient education are the keys to continued success. ■

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