HIS REPORT of the development of symptomatic deep-vein thrombosis (DVT) in a physically active 24-year-old male recreational athlete/physical education major was unusual in that its development did not coincide with apparent risk factors. This case demonstrates how a fortuitous second-hand encounter with an athletic training student who recognized the atypical presentation averted a potentially catastrophic outcome. The athlete remains under medical care but has resumed many aspects of his lifestyle.

Deep-vein thrombosis (DVT) is an abnormal blood clot occurring in a deep vein.¹ A pulmonary embolism threatens life when part of the clot breaks off and occludes a pulmonary artery. Two million Americans develop DVT annually, although the number of cases may be underestimated when the number of asymptomatic cases are considered.²,³ Current estimates of DVT are limited by inaccuracies in clinical diagnosis.⁴ Extremity pain is commonly encountered in the clinical practice of athletic training, but vascular compromise is seldom considered during initial evaluation in young athletes.⁵⁻⁶ Trauma is the primary causative factor in lower extremity DVT among athletes. Such injuries are not often reported, resulting in a paucity of sports-medicine literature addressing vascular abnormalities.⁶,⁷

**Case History**

A robust 24-year-old African-American male, who was a physical education major, developed unexplained right-sided back pain. He reportedly lost 27 lb over the previous 12 weeks. His normal activity regimen included preprofessional activity modules, weight lifting, daily recreational basketball, and a daily 3-mile run. He discontinued his cardiovascular and strength regimen but continued his physical education activity modules and began self-administered cryotherapy for his back pain. The short-lived back pain was followed by a noticeable increase in the size of his right leg and thigh from the calf to the groin. Despite a limp and an edematous leg, the athlete was reluctant to seek medical attention. Four days later, his girlfriend described the symptoms to a
that are administered to begin anticoagulation until significant pain.

to the common femoral vein with 3+ edema. He had an emergency room, this temperature was 97.9 °F, pulse was 60 beats/min, blood pressure was 147/82 mmHg, respiratory rate was 14 breaths/min, and oxygen saturation was 100%. Compression ultrasonography revealed an acute DVT extending from the calf to the common femoral vein with 3 + edema. He had no symptoms of pulmonary embolism, a roentgenogram was negative for acute cardiopulmonary disease, and ECG was normal. Computerized tomography of the abdomen and pelvis demonstrated dilation of the right common and external iliac veins and a markedly attenuated left external iliac vein. His social history was negative for drinking, smoking, or drug use. There was no personal or family history of clotting problems, DVT, or pulmonary embolism.

The young man was admitted to the hospital, where he was evaluated and treated for 7 days. He was found to have mild hyperhomocysteinemia and mildly increased anticardiolipin antibody (ACA) levels. Homocysteine is an amino acid that is associated with coronary artery disease when present in the blood at a high level. Folate therapy, administered in the hospital, reduced the homocysteine level. Otherwise, his blood-chemistry values were within normal limits, including Protein S and Protein C. DNA testing indicated that this individual was negative for the Factor V Leiden mutation. Anticoagulant medications were administered immediately, including heparin, enoxaparin, and warfarin sodium. Their simultaneous use indirectly inhibits thrombin by increasing the action of antithrombin. Heparin and enoxaparin are rapidly acting medications that are administered to begin anticoagulation until the warfarin reaches therapeutic levels in 4–5 days. Oral anticoagulation therapy is usually prescribed for a minimum of 3 months after an initial episode of DVT. Patients with an ongoing risk factor may be continued for 6 months or longer.

On the second day, the patient underwent a venogram and iliofemoral angiography under intravenous sedative anesthesia in the operating room. Several attempts were made to place a clot filter in the inferior vena cava through both the left femoral vein and right jugular cava. Although extensive hepatic collaterals were demonstrated, no direct inferior vena cava to superior vena cava communication was demonstrated. The physicians concluded that the anomaly in venous structure provided protection to the patient, which made placement of the vena cava filter unnecessary.

The patient was discharged on the seventh day, once his international normalized ratio (INR) had reached therapeutic levels. His instructions were to avoid green leafy vegetables, avoid contact sports, and continue anticoagulant medication and folate daily. He was also advised not to ingest aspirin or NSAIDs. The records are unclear as to when the folate was discontinued after discharge, but patients typically respond within several weeks. At 3 months after DVT diagnosis, the warfarin was discontinued for 1 week to determine whether or not the INR had reached a criterion level. Because the value remained below the therapeutic threshold, warfarin was continued. At the time this report was written, the patient was still receiving warfarin therapy, with another test of the INR scheduled. The INR value will influence the type and duration of future therapy. Lifelong anticoagulation therapy is a controversial treatment option that may be considered.

**Discussion**

**Relationship of Genetic Risk Factors to DVT**

Genetic risk factors can increase predisposition for DVT including Factor V Leiden, Protein S, and Protein C deficiencies. Factor V Leiden is one of the most common causes of inherited thrombophilia (Quest Diagnostic Nichols Institute, San Juan Capistrano, CA). Inherited thromboembolic disease occurs when the body lacks the ability to regulate the clotting cascade, involving failure to neutralize thrombin or inadequate thrombin production. Deficiencies in Protein S and Protein C can