Acute Deep-Vein Thrombosis in an Active Male College Student: A Case Report

Marsha Grant-Ford, ATC, PhD, and David Middlemas, EdD, ATC • Montclair State University

THIS 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.[5-9] 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

© 2007 Human Kinetics - ATT 12(1), pp. 26-30

senior athletic training student (ATS) during a chance encounter in a parking lot. The ATS, having a high level of suspicion, gave the young lady instruction on how to perform Homan's sign, with advice to seek evaluation and treatment at an emergency room if the test produced a positive finding. Later that afternoon, the young lady passively dorsiflexed the foot and squeezed the calf of her boyfriend as instructed, which elicited significant pain.

When the young man was subsequently evaluated at an emergency room, his 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.[10] Heparin and enoxaparin are rapidly acting medications that are administered to begin anticoagulation until the warfarin reaches therapeutic levels in 4–5 days.[2] 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.[11]

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 routes, but the effort was not successful. The physicians concluded that the patient had anatomical characteristics that interfered with filter placement, which were described as complex venous and inferior vena cava anomalies. Specifically, the patient was found to have no direct communication between the left femoral vein and the inferior vena 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.[12] 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.[2] 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.[5] Deficiencies in Protein S and Protein C can

be responsible for recurrent thromboembolic disease, secondary to the incapability to regulate the clotting cascade. Protein S deficiency can also be acquired from use of oral contraceptives, and it is associated with Type I diabetes.[5] Patients deficient in Protein S or Protein C have a propensity for thromboembolitic challenges to the vascular system[5] (Table 1).

Relationship of Relevant Laboratory Studies to DVT

The INR is a value derived from a laboratory test that compares the time it takes for blood to clot in relation to an established average time (HRSociety).An INR of 2.0–3.0 is often the selected treatment goal. The duration of anticoagulation therapy is based on individual risk and predisposing factors. An INR less than 2.0 indicates inadequate protection from clotting.[13]

Several factors can alter the INR. For example, this patient was cautioned against using aspirin and ibuprofen due to the risk of a decrease in platelet aggregation. He was also advised to avoid consumption of green leafy vegetables. Vitamin K, an important component of the coagulation cascade, is derived from green leafy vegetables.[14] Hyperhomocystinemia, identified in this patient, is also an established risk factor for arterial thrombosis, which has recently been recognized to present an almost threefold increase in risk for initial and recurrent DVT.

Intervention

The rationale for placement of an inferior vena cava filter relates to the fact that a greater risk of developing an embolism exists when a thrombus lies in a femoral vein. A clot with a considerable volume tends to develop because of the large lumen size, and it has a direct pathway to the pulmonary arteries through the iliofemoral circulation to the right side of the heart. When pulmonary arterial flow is occluded, the sequela is failure of the right heart, circulatory collapse, hypoxia, and death.[2] A wire device derived from an inert alloy is inserted to trap an embolus before it occludes the pulmonary arteries.[2,4]

Related Pathology

Primary upper extremity deep-vein thrombosis refers to either effort thrombosis (called Paget-Schroetter syndrome) or idiopathic upper extremity DVT.[7,18] The entity is rare, but known triggers in the athletic population merit mention. In fact, Paget-Schroetter syndrome is reported to be the most common athletic vascular problem.[19] This syndrome can be caused when shear forces, such as the mechanical stretching associated with some repetitive athletic motions, contribute to aneurysm formation that is secondary to a weakening of the venous intima and the activation of the coagulation cascade. The subsequent development of fissures can result in thrombi entering the vascular system, especially when mechanical compression of the vessel occurs.[9,20,21] Rowers, wrestlers, weight lifters, pitchers, and other athletes regularly engage in activities that may traumatize the vessel wall.[18] This syndrome has been the subject of several case reports involving the sports of volleyball, basketball, football, handball, golf, badminton, field hockey, and ice hockey.[20,22]

The most salient risk factor for venous thrombosis is a previous thrombolytic event. Fifty percent of those

TABLE 1. GENETIC RISK FACTORS THAT INCREASE THE PREDISPOSITION FOR DEEP-VEIN THROMBOSIS (DVT)

	Normal Function in Clotting Process	DVT Risk Factor
Factor V	Activated by thrombin, it accelerates and prolongs the clot-formation process.	Factor V Leiden: Genetic abnormality causing increased tendency for clotting.
Protein C	Inactivates Factor V and stimulates fibrinolysis when activated by thrombin.	Deficiency: Does not control Factor V, therefore not slowing cascade of clotting process.
Protein S	Cofactor for activating Protein C in the clotting process dependent on Vitamin K.	Deficiency: Failure to activate Protein C, therefore not slowing cascade of clotting process.
Anticardiolipid antibodies (ACA)		Antibody positive: Patients have approximately twice the risk of DVT as those without elevated ACA.

who experience DVT possess risk factors, and the risk factors are cumulative.[17] Generally, the risk factors comprise one or more of the mechanisms of Virchow's triad,[23] which includes the following components:

- Alteration of blood-flow mechanics
- Blood-composition changes
- Compromise of the blood-vessel lining

Risk for clot development increases with blood stasis from limited movement, whether due to a long airplane flight (economy-class syndrome), car ride or computer work, or orthopedic surgery and immobilization after surgery. Blood-flow mechanics are altered because of venous-pump deactivation and venous pooling.[2] Surgery and trauma precede 40 % of DVT events. A relevant consideration for female athletes using oral contraceptives containing progestin is its association with greater risk for DVT.[10,12,23] Diabetes is also identified as a risk factor in the literature.[2] A contusion or strain involving the calf musculature could also be a precursor of DVT in the physically active population (Table 2).

This case underscores the importance of including vascular conditions in the differential diagnosis when assessing extremity pain in the physically active population. Although DVT is condition that is not generally associated with active young people, failure to recognize its symptoms (Table 3) could result in tragedy.[24] A void exists in the literature regarding return-to-play protocols for athletes after DVT.[6]

Table 2. Nongenetic Risk Factors That Increase the Predisposition to Deep-Vein Thrombosis

Medically Related	Life Event or Behavior
Previous thrombolytic event	Smoking
Cancer	Oral contraceptives
Hormone-replacement therapy	Obesity
Varicose veins	Recent surgery
Immobilization	Pregnancy and childbirth

Table 3. Signs and Symptoms of Deep-Vein Thrombosis

History	Physical Examination
Recent long trip or plane flight	Unilateral edema
Complaints of loss of endurance	Low-grade fever
Fatigue	Warmth in the affected extremity
Exertional pain or swelling in an upper or lower extremity	Skin discoloration (blue or red more common than white)
Constant or intermittent pain in the affected limb	Possible positive Homan's sign
Sensation of tightness or heaviness	

Conclusions

Clinical assessment of lower extremity DVT should include acquisition of a good clinical history.[4] Failure to recognize the signs and symptoms of DVT may result in a limb-threatening or life-threatening situation. Homan's test should be performed as part of an initial injury assessment.[4,23] Pain and/or tenderness during administration of Homan's test is not always an indicator of venous thrombosis; some sources have reported a 70 % false-negative rate.[4] Although rare, DVT can develop secondary to any athletic trauma to an extremity.[25] Otherwise healthy active people (including coaches and officials) with pain or leg swelling should be questioned about trauma, constrictive clothing or equipment, or activities that might compress the veins of the legs.[26]

References

1. Anderson MK, Hall S, Martin M. *Foundations of Athletic Training: Prevention, Assessment, and Management*. Philadelphia, Pa: Williams & Wilkins; 2005.
2. Slaybaugh RS, Beasley BD, Massa EG. Deep venous thrombosis risk assessment, incidence, and prophylaxis in foot and ankle surgery. *Clin Podiatr Med Surg.* 2003;20(2):269-289.
3. Hansson PO, Sorbo J, Erikkson H. Recurrent venous thromboembolism after deep vein thrombosis: incidence and risk factors. *Arch Intern Med.* March 2000;160(6):769-774.

4. Schreiber D. Deep venous thrombosis and thrombophlebitis. *eMedicine*. 2005. Available at: http://www.emedicine.com/emerg/topic122.htm. Accessed June 8, 2005.
5. Wong C, Bracker M. Coagulation presenting as calf pain in a racquetball player. *J Fam Pract*. 1993;37:390-393.
6. Echlin PS, Upshur RE, McKeag DB, Jayatilake HP. Traumatic deep vein thrombosis in a soccer player: a case study. *Thromb J* [serial online]. 2004; Oct 14(2) 1:8. Available at: http://www.thrombosisjournal.com/content/2/1/8. Accessed November 4, 2004.
7. Arco FR, Harris JE, Zarins CK, Olcott IV C. Vascular complications in high performance athletes. *J Vasc Surg*. May 2001;33:935-942.
8. Slawski DP. Deep vein thrombosis complicating rupture of the medial head of the gastrocnemius. *J Orthop Trauma*. 1994;8:263-264.
9. Arco FR, Olcott IV C. Arterial and venous injuries in athletes. *Physician Sportsmed*. April 2003;31(4):41-48.
10. Lee AY, Hirsh J. Diagnosis and treatment of venous thromboembolism. *Ann Rev Med*. 2002;53:15-33.
11. Davidson BL. DVT treatment in 2000: state of the art. *Orthopedics*. June 2000;23(6 suppl):651-654.
12. Joffe HV, Goldhaber SZ. Laboratory thrombophilias and venous thromboembolism. *Vascular Med*. 2002;7:93-102.
13. Heart Rhythm Society. International Normalized Ratio (INR). Heart Rhythm Foundation. 2004. Available at: http://medicine.ucsf.edu/htc/clinicians/clin.inr.html. Accessed July 8, 2005.
14. Booth SL, Centurelli MA. Vitamin K: a practical guide to the dietary management of patients on warfarin. *Nutr Rev*. September 1999;57:288-296.
15. Greenblatt DJ, von Moltke LL. Interaction of warfarin with drugs, natural substances, and foods. *J Clin Pharmacol*. 2005;45:127-132.
16. Booth SL, Suttie JW. Dietary intake and adequacy of vitamin K. *J Nutr*. May 1998;128(5):785-788.
17. Colucciello S. Protocols for deep venous thrombosis (DVT): a state-of-the-art review. Available at: http://www.thrombosis-consult.com./I.htm. Accessed August 3, 2005.
18. Joffe HV, Goldhaber SZ. Upper-extremity deep vein thrombosis. *Circulation*. October 2002;106(14):874-1880.
19. Sotta RP. Vascular problems in the proximal upper extremity. *Clin Sports Med*. 1990;9(2):379-388.
20. Zell L, Kinderman W, Marschall F, Scheffler P, Gross J, Buchter A. Paget-Schroetter in sports activities: case study and literature review. *Angiology*. November 5, 2001;52:337-342.
21. deWeber K. Effort thrombosis with sepsis. *Physician Sportsmed*. May 1999;27(5):74-XX. Available at ttp://www.physsportsmed.com/issues/1999/05_99/deweber.htm. Accessed November 2, 2004.
22. DiFelice GS, Paletta GA, Phillips BB, Wright RW. Effort thrombosis in the elite throwing athlete. *Am J Sports Med*. September-October 2002;30(5):708-712.
23. Watson AS, Gray D, Godfrey J, Muller A. Deep vein thrombosis following sports injury to the calf: a potentially dangerous complication. *Sports Train Med Rehabil*. 1991;2:273-278.
24. Zamorski MA, Opdycke RA. Advances in the prevention, diagnosis, and treatment of deep vein thrombosis. *Am Fam Physician*. 1993;47:457-469.
25. Starky C, Ryan J. *Evaluation of Orthopedic and Athletic Injuries*. 2nd ed. Philadelphia, Pa: FA Davis.
26. Delcau CM. Deep vein thrombosis in umpires. *South Med J*. 1992:85(6):670.

Marsha Grant-Ford is the clinical education coordinator for the athletic training education program at Montclair State University. She has over 15 years of experience as an athletic training educator and has had international clinical experiences, as well as at the secondary school level and all three NCAA division levels.

David Middlemas is the program director for the athletic training education program at Montclair State University. He has over 15 years of experience as an athletic training educator, as well as a college and high school athletic trainer and supervisor of a clinic-based athletic training outreach program.

STATEMENT OF OWNERSHIP, MANAGEMENT, AND CIRCULATION OF *ATHLETIC THERAPY TODAY* (ISSN 1078-7895, as required by 39 U.S. Code 3685:

Athletic Therapy Today (ISSN 1078-7895) is published six times a year. Subscription fees are $44 per year for individuals and $176 per year for institutions.

The owner of *Athletic Therapy Today* is Human Kinetics, Inc., whose office of publication is at 1607 North Market St., Champaign, IL 61820-2200. The editor is Gary Wilkerson, Dept. #6606, UT-Chattanoonga, Chattanooga, TN 37403-2598. The publisher is Rainer Martens, P.O. Box 5076, Champaign, IL 61825-5076. There are no bondholders, mortgagees, or other security holders.

Average number of copies printed per issue (net press run) during the preceding 12 months is 2,705; number of copies nearest to filing date is 2,769. Average number of copies of each issue distributed after mass mailing to subscribers is 17; number of copies nearest to filing date is 23. Average number of copies of each issue distributed in mass mailing to subscribers 2,374; number of copies nearest to filing date is 2,431. Average number of copies of each issue distributed free is 33; number of copies nearest to filing date is 33.