Blood Clots and the Athlete: A Review of Deep Vein Thrombosis in Sports

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A BLOOD CLOT arising in the vein, or deep vein thrombosis (DVT), is a rare but significant event for athletes. Unique circumstances are involved in both increasing and decreasing an athlete’s risk. Upper extremity deep vein thromboses (UEDVT) and lower extremity deep vein thromboses (LEDVT) are obviously similar but have certain important distinguishing features. Prevention and return-to-play issues are critical concepts for the athletic trainer to consider. Blood clots that form in the arteries are often confused with DVT, but are managed differently and are not reviewed in this report.

DVT is a common disorder in the general population with potentially severe consequences. The general incidence of DVT has been reported as 1 in 1,000 people per year, with only 2% of all DVTs involving the upper extremity. Pulmonary embolization and post-thrombotic syndrome are the most serious potential complications. The former is urgent and may be life threatening. Pulmonary emboli have been noted in 20% of LEDVT; only about 12% of UEDVT are thought to create pulmonary emboli.1 Post-thrombotic syndrome occurs in as many as 60% of cases and can be a significant disability leading to chronic discomfort.

Numerous case reports describe a consistent pattern of symptoms.1-5 Edema and discomfort may last from a few hours to several days after onset and usually prompt the athlete to seek help. The edema involves the entire limb circumferentially and is usually tender to the touch. Distended veins and a palpable tender cord are often, but not always, present. Low-grade fever and distal limb cyanosis may also occur. Confirmation requires duplex ultrasound, which is sensitive and specific. However, a strong clinical suspicion with a negative ultrasound requires further studies such as magnetic resonance angiography or venography.6

Athletes actually have some protection from DVTs by the nature of their conditioning. Certain antithrombotic or “anti-clotting” conditions exist with exercise, including increased blood volume, higher blood flow velocities, increased collateral vessels, and diminished stasis. While helpful, these factors are not sufficient to eliminate DVT risk. Initiation of a DVT requires some component of Virchow’s Triad of stasis, endothelial injury, and hypercoagulability. Assessing risk and/or looking for causation entails evaluation of these factors.

Virchow's Triad: Stasis

Venous stasis is not something readily associated with athletic activity, but immobilization associated with an injury can easily disrupt normal venous return. Immobilization for greater than 3 days is associated with a greater prevalence of DVT, and the presence of this risk may persist for more than one month after injury. One study suggests that the only significant predictors of thromboembolism were obesity and duration of immobilization.7

Virchow's Triad: Endothelial Injury

Endothelial injury results in the promotion of several clotting mechanisms derived from the lining of the veins themselves. Platelets are activated, they become sticky, and several chemical mediators are released that activate multiple levels of the clotting cascade.8 A population study (not limited to athletes) displayed a 13-fold increased risk in DVT after some sort of endothelial injury.9
**Virchow’s Triad: Hypercoagulability**

Hypercoagulability is a broad concept involving many factors, including malignancy, but the attention to genetic factors is particularly pertinent. A hypercoagulable disorder adds significant risk to the issues noted below. These factors are numerous, and their relative risk status is listed in the accompanying chart (see Table 1). The fact that some gene frequencies vary in the general population is noteworthy; for example, the prothrombin 20210A mutation is rare in non-Whites.

**Table 1. Risk Status for Factors of Hypercoagulability Disorder**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor V Leiden mutation</td>
<td></td>
</tr>
<tr>
<td>heterozygous mutation</td>
<td>7x</td>
</tr>
<tr>
<td>homozygous mutation</td>
<td>80x</td>
</tr>
<tr>
<td>Prothrombin 20210A mutation</td>
<td>2.8x</td>
</tr>
<tr>
<td>Antithrombin III</td>
<td>(less than 10%)</td>
</tr>
<tr>
<td>Protein C deficiency</td>
<td>(less than 10%)</td>
</tr>
<tr>
<td>Protein S deficiency</td>
<td>(less than 10%)</td>
</tr>
<tr>
<td>Hyperhomocysteinemia</td>
<td>2.5x</td>
</tr>
<tr>
<td>Elevated Factor VIII</td>
<td>5x</td>
</tr>
<tr>
<td>Elevated Factor XI</td>
<td>2.2x</td>
</tr>
</tbody>
</table>

Hypercoagulable states can occur in much less esoteric situations. Estrogen confers a certain degree of hypercoagulability. The sports health-care clinician must keep in mind the potential for DVT in the female athlete on oral contraceptives or who is pregnant. Conversely, the spontaneous onset of DVT in a female mandates a definitive determination of pregnancy status.

Immobilization and flying combine to increase risk. Modern sports often require prolonged travel. Confined seating, transient hypoxia (though mild), and longer duration of flight have been proposed as risks. In a case-control study of DVT risk, recent travel imparted a four times greater risk than that found for control subjects. Flying longer than four hours was an independent risk factor. Specific measures to reduce the risks of airline travel based on expert opinion include making room for leg extension, frequent walks while in-flight, wearing loose clothing, maintaining hydration, and consideration of anticoagulants such as aspirin, based on risk factors.

Strenuous high altitude exercise, such as mountain climbing, presents uniquely risky circumstances. The climbing career of Sir Edmund Hillary, best known for the first ascent of Mount Everest, was shortened by an embolic accident from a DVT. The hypoxia of high altitude causes the body to increase red blood cell production to deliver more oxygen to peripheral tissues. However, more red blood cells thicken the blood, making it more sluggish and prone to stasis, which is one of the key elements in Virchow’s Triad. Low temperatures at high elevations further promote increased blood viscosity. Dehydration associated with low air pressure and the logistical difficulty of providing sufficient fluids for replenishment under strenuous physical conditions bring additional risks. Frostbite may also cause endothelial injuries that can initiate thrombus formation.

**Upper Extremity DVT**

UEDVT is unique, because the initiating factors may not be readily apparent. Paget-Schroetter syndrome is described as “effort thrombosis,” triggered by microtrauma to the axillo-subclavian veins. This microtrauma may result from strenuous exertion seen in throwers or weightlifters. Many cases are idiopathic, as demonstrated by the subgroup of throwers with UEDVT in the nondominant arm. Anatomic risk factors have been documented, including cervical ribs, a long transverse process of the cervical spine, hypertrophied sternocleidomastoid muscles, and clavicular or first rib anomalies.

These anatomic factors are of particular importance in management. Some physicians suggest that treatment is incomplete in UEDVT until an anatomic procedure is undertaken. This may explain outcome differences with anticoagulation treatment in UEDVT compared to LEDVT. Acute thrombolysis with such agents as urokinase, directed near the site of the DVT via a directed catheter, is strongly recommended to prevent the long term effects of post-thrombotic syndrome, seen more often in the upper extremities. Structural abnormalities of the upper extremity may explain the higher occurrence of post-thrombotic syndrome in UEDVT. Fortunately, treatment of anatomic factors does not seem to inhibit eventual return to sport, with a few published exceptions. Up to 90% of patients treated with anticoagulation and surgery...
reported good prognosis. Four elite baseball players who underwent a first rib resection returned to play at their previous levels.¹⁷

**Lower Extremity DVT**

LEDVT responds well to conventional anticoagulants, and with a less complicated course than UEDVT. Compression stockings minimize post-thrombotic syndrome complications. Although microtrauma resulting from mechanical factors can create LEDVT, this seems to occur far less frequently than with UEDVT. Cyclists with their aerodynamic position and repetitive hip flexion create the same venous endothelial disruption seen in the overhead athletes. However, most LEDVT are attributed to severe trauma from surgery and/or immobilization.

**Treatment Options**

Regardless of the acute treatment variations, the primary definitive treatment is anticoagulation. After the acute phase, oral warfare is used to maintain the International Normalized Ratio (INR), used for reporting blood clotting results, between 2.0 and 3.0. Duration of treatment guidelines are fairly well established, with variations depending on the number of prior events, genetic predisposition, the use of stents, and the degree to which the event was life-threatening.¹⁸

Return to play status will vary based on the nature of the sport and limited to noncontact activities while on anticoagulation. This will preclude many sports except perhaps running, track and field, and golf. Exceptions may exist, but must be critically analyzed for risk. At the conclusion of anticoagulation therapy, activity restrictions can be lifted as long as the patient remains asymptomatic. There are no published guidelines on progression of activity while undergoing anticoagulation therapy or after its completion. Roberts and Christie¹⁹ proposed a protocol to allow a female triathlete to resume running; however, the athlete was noncompliant and she resumed training well ahead of the agreed-upon schedule. She developed post-phlebitic syndrome, which later resolved, and she went on the excel and achieve her best performances two years later.¹⁹

Despite certain protections associated with athletics, venous thromboses may occur even in very fit individuals. Treatment may vary based on the location of the disorder, but early diagnosis and expedient management are essential. Return-to-play issues eventually arise, which can be dealt with on a case-by-case basis. Some athletes will be forced make a long-term adaptation by changing exercise and sport participation routines. Such cases require the athletic trainer to be ever-vigilant in monitoring the athlete’s status.

**References**


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