



Case Report

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Deep Vein Thrombosis in a Collegiate Female Soccer Player: A Case & Evidence-Supported Review

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Abstract

Typically, deep vein thromboses (DVTs) affect older individuals with existing comorbidities or known risk factors such as obesity and smoking, or as potential sequelae for certain pharmacological therapies. Although it's expected for clinicians to be leery for DVT pathogenesis in typical patient populations, especially under the auspices of significant lower extremity trauma, the reality is that DVTs are not seen or expected in healthy collegiate athletic populations very often. In fact, there are very few reported cases of DVTs occurring in younger individuals, regardless of the nature of injury or circumstances surrounding the injury, which can lull the unsuspecting clinician into a false sense of security. Failure to appreciate that DVTs can occur in non-typical populations and further failing to recognize the clinical development of a DVT can produce catastrophic consequences for the athlete. A detailed history and clinical exam are prudent for detecting pulmonary thrombosis, regardless of the age and perceived lack of risk factors of the patient and it's important to know that established clinical prediction rules can be very helpful. The authors present a case of an extensive DVT following an isolated osteochondral injury of the tibiofemoral joint in an otherwise healthy collegiate female soccer player, which in the final analysis closely matches the risk criteria for typical DVT patients. Comparing the findings of this case with previously reported prediction rules provides a supporting and systematic approach for recognizing and accurately diagnosing this potentially hazardous condition.

Keywords

Deep vein thrombosis; Collegiate athlete; Clinical prediction rules; Soccer

Introduction

A deep vein thrombosis (DVT) or venous thromboembolism (VTE) is a blood clot that develops within the deep venous structures of the body, typically affecting the thigh or calf but is not limited to the lower extremity [1,2]. Epidemiological studies indicate that a correlation exists between age and incidence, with a 0.005% (<5/100,000 persons/year) occurrence in individuals under the age of 15, and an estimated 0.5% (450-600/100,000/year) occurrence for those over the age of 80 [3]. Sixty years seems to be the critical age threshold as the incidence has been found to increase dramatically

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once the 7th decade begins. Furthermore, only about 30 first time cases of DVT occur for every 100,000 persons in the 25-35 age year range, and despite the concern for the increased risk that oral contraceptives offer, females and males actually suffer from the same rate of occurrence. Regarding race and ethnicity, Caucasians and African Americans suffer similar rates, while Asian-Pacific Islanders and Hispanics have a 2.5-4 fold lower risk [3]. It is estimated that 600,000 new DVT cases are diagnosed each year, with one percent of those cases resulting in death [4]. The cause of death is primarily due to a pulmonary embolism (PE), in which the thrombus dislodges from the vascular wall and becomes trapped within the lungs. The resulting hypoxia causes cardiac failure, which can be fatal if the condition is not diagnosed and treated early. Up to 70 percent of patients diagnosed with PE had an existing lower extremity DVT, emphasizing the significance of accurately diagnosing DVTs in patients and securing early intervention [5].

Case Review

An otherwise healthy, 20-year-old, collegiate female soccer athlete presented in our athletic training room complaining of deep and diffuse knee pain after a non-contact, weight bearing and torque producing mechanism while playing indoor soccer. Her past medical history revealed a successfully reconstructed anterior cruciate ligament following an ACL tear in the right lower extremity status post three years. Upon physical exam, she presented with diffuse swelling and subjective pain as a 3 out of 10 that was aggravated with weight bearing and palpation over the lateral joint line and lateral tibial plateau. Passive range of motion was limited to 90 degrees of flexion, and a 5-degree terminal extension lag was obvious. McMurray's test was negative, and Lachman's test proved to be equivocal. Based on the mechanism of injury and her history, meniscal and ACL pathologies were suspected. Initial treatment involved the PRICE (protection, rest, ice, compression, and elevation) protocol, including the use a compression wrap and of crutches to protect the injured joint and to allow pain free partial weight bearing (PWB) ambulation.

Upon referral to the team physician, magnetic resonance imaging (MRI) was ordered and findings indicated a small osteochondral defect along the lateral femoral condyle, and a subjacent cartilage defect along the lateral tibial plateau (Figure 1). Subchondral bone marrow edema was present in the distal femur and proximal tibia,



Figure 1: Magnetic resonance image of osteochondral defect on left femoral condyle and subjacent chondral defect on athlete's lateral tibial plateau.

and a moderate sized suprapatellar joint effusion was noted. MRI indicated that the anterior cruciate ligament and menisci were normal. Based on the physician's final diagnosis, low level quadriceps contractions and electrotherapy for pain management were added to her initial treatment plan.

Early conservative care resulted in significant improvement with considerable pain relief within one week. The team physician instructed the athlete to continue this treatment plan and to maintain PWB status until a follow-up appointment in ten days. Based on the significant improvements on physical exam, the athlete was cleared by the team physician to travel with her team for an overseas training trip. The athlete traveled with her team on a five-hour bus ride and an additional five-hour flight to the final training destination. Four days after her departure date, she complained of increasing pain in the posterior aspect of the right calf, redness and marked edema in the lower leg. As pain increased, her ambulatory ability deteriorated from weight bearing as tolerated, to non-weight bearing. Upon return to campus three days after the reported increase in symptoms, a follow up evaluation noted a fairly unremarkable physical exam:

- Diffuse edema in the popliteal fossa extending distally into the calf
- Calf pain recorded as 4+/10 (during gait)
- Knee pain decreased to 0/10
- (-) Homan's Sign
- Normal distal pulses (posterior tibialis, dorsal pedis)
- Discolored skin on the anterior and posterior aspects of the lower leg
- Unable to perform toe raises on the affected extremity (secondary to pain)

At this point, concern was raised over the possibility of a DVT, but the patient's overall health and age rendered the suggestion unlikely. Concerned with our mixed findings (negative Homan's Sign in particular), we consulted the literature and found support for our suspicions. According to Well's Clinical Prediction Rule (CPR) for DVT, the presence of 3 or more major, and 2 or more minor criteria indicate with 98% specificity that a DVT may be present [6]. For Well's major criteria, this athlete experienced 1) prolonged immobilization (10 hours of travel), 2) complained of localized tenderness and 3) presented with calf and lower leg swelling. For Well's minor criteria, she had 1) a history of recent trauma and 2) presented with pronounced erythema in her affected calf and lower leg. According

to Wells CPR, the presence of these 5 key features indicates a positive likelihood ratio of 39, and a diagnostic odds ratio of 124. In other words, our clinical findings meant that our young soccer player was extremely likely to be suffering (124 times more likely) from a DVT, and the (-) Homan's Sign was a false negative result (supporting the low evidence for this test). Given such strong evidence, this athlete was referred to the local hematology department for a diagnostic ultrasound to confirm our suspicions. Imaging results indicated an extensive occlusive deep vein thrombosis present within the distal femoral, popliteal, posterior tibial and peroneal veins (Figure 2).

Treatment

Following confirmation of a DVT, the attending hematologist immediately initiated a treatment plan. Anticoagulation therapy consisted of 60 mg dosages of Lovenox (an enoxaparin injection administered subcutaneously adjacent to the naval), twice a day for two weeks, and a daily dosage of 5 mg of Coumadin administered orally. Weekly blood tests were performed to monitor the athlete's International Normalized Ratio (INR) to ensure levels were within a therapeutic range (between 2-3 INR). The athlete was experiencing severe back pain one month after beginning anticoagulation therapy, and follow up blood tests revealed high INR levels. Based on these findings, subsequent modifications were made to the Coumadin dosage, which lowered the INR and resolved the back pain. Coumadin treatments continued for four months until the clot was completely dissolved as determined by Doppler ultrasound. The athlete also maintained a PWB status for four weeks to provide adequate recovery time for the osteochondral pathology.

To decrease the likelihood of developing post thrombotic symptoms (PTS), a 40 mmHg thigh-length compression stocking was prescribed for continuous use. Compression stockings can help reduce blood from pooling, or remaining stagnant in the lower leg. In a study involving the duration of compression stocking use, it was noted that prolonging the compression therapy (therapy lasting longer than six months post diagnosis) had significantly better results than those who had shorter treatments [7]. Limb elevation while sleeping was also prescribed to avoid PTS.

Additionally, the team physician prescribed therapeutic exercises including isometric quadriceps contractions (3 sets of 10 repetitions with 3 second hold) and straight leg raises (2 sets of 20 repetitions) to address the osteochondral trauma and subsequent decreased lower extremity function. One month after diagnosis of the DVT, the athlete was cleared to begin light exercise by the hematologist and team physician. This exercise included the use of an upright stationary bicycle and elliptical machine. Stationary bike was first utilized for 5 minutes during the initial session with subsequent sessions adding 5 minutes per session until 30 minutes was reached. This same progression was utilized for the elliptical machine once 30 minutes of pain free activity was reached with the stationary bike. In addition, the athlete added various closed chain exercises (including leg press, body weight squats) to her rehabilitation during the next couple of months to address her overall lower extremity strength and function. Four months after the initial trauma, the athlete was cleared to begin a pain free return to running.

To decrease the likelihood of future clot formation, the medical team recommended the athlete take several actions. A compression stocking was prescribed for any travel-induced immobilization lasting longer than two hours, as well as air travel of any duration. Also, oral contraceptive use was discontinued due to the increased risk of



Figure 2: Ultrasound image of extensive occlusive DVT in athlete's distal femoral, popliteal and peroneal veins.

Table 1: Known risk factors for deep vein thromboses.

1	Previous DVT or family history of DVT
2	Vein disease (i.e. varicose veins)
3	Limb trauma and/or orthopedic procedures
4	Immobility, such as bed rest or sitting for long periods of time
5	Air Travel (greater than 3000 kilometers or longer than 4 hours)
6	Recent surgery
7	Above the age of 40
8	Hormone therapy or oral contraceptive use
9	Pregnancy or post-partum (specifically 6 weeks post partum)
10	Previous or current cancer
11	Genetic factors that predispose one to clotting
12	Obesity
13	Smoking
14	A central venous catheter

Outstanding risk factors that contributed to the differential diagnosis of this particular case are indicated in bold type [1,2,4].

clot development [8]. At five months following the DVT diagnosis, anticoagulant medication was discontinued and no additional follow up appointments were indicated.

Discussion

It is well accepted that recent trauma and/or surgery, followed by prolonged immobilization in certain, higher risk individuals should raise suspicion for the development of DVTs, especially in the lower extremities. However, there are few documented cases of young and otherwise healthy athletes developing DVTs, but due to the serious and significantly hazardous outcomes, it's essential for clinicians to carefully identify potential risk factors that may predispose certain patients to clot formation, regardless of their demographics. Chiefly, it's prudent for a thorough medical history to be taken in order to recognize those situations in which a DVT might not be suspected, and as demonstrated here, to regard the results of the Homan's Sign with suspicion (Cranley, et al reported a sensitivity of 48%, a specificity of 41% and a DOR of only 0.64, demonstrating its poor diagnostic utility) [9]. Table 1 provides a list of common risk factors associated with the development of a DVT. Those exhibited by the athlete in this case are highlighted for case reference and context.

In reflecting on the particulars of this specific case, our athlete's risk factors were:

- Osteochondral defect along lateral femoral condyle and sub adjacent tibia, as well as subchondral bone marrow edema
- Combined twenty hours of immobilization during the travel period
- Four separate plane flights totaling over ten hours at significant altitude
- Athlete was currently taking Tri-Lo Sprintec, an oral contraceptive
- Blood tests revealed the genetic mutation for Factor V Leiden

These risk factors are explained and reviewed to ensure a better understanding of the etiology specific to this case.

Limb trauma

The torsion forces caused by the mechanism of trauma in this case proved capable of damaging the osteochondral surfaces of both the femur and tibia. This resulted in focal cartilaginous edema about the tibial and femoral articular cartilage of the lateral joint space with a small amount of reactive edema about the lateral femoral condyle. Traumatic events can cause disruption of the endothelium lining of the vein wall, causing platelets to begin to adhere to the venous wall and results in the deposition of leukocytes, erythrocytes, and fibrin [10]. This accumulation of cells and proteins result in a thrombus formation.

Immobility

The combined twenty hours of immobilization from the initial and return trips resulted in decreased muscle pump from the lower extremity, and increased venous stasis. Venous stasis increases the risk of thrombosis by inhibiting activated coagulation factors from being removed through normal blood flow [11]. The higher concentration of coagulation factors facilitates the continued development of the thrombus. Also, the endothelial protein thrombomodulin that is responsible for converting thrombin into an anticoagulant enzyme requires blood flow to travel from the capillaries into the veins to take effect [11].

Air travel

Of the twenty hours of travel immobilization, ten hours were spent via air travel at significant altitude (greater than 3000 kilometers). A previous case report indicated that air travel lasting at least four hours resulted in an increased risk of venous thromboembolic disease [12]. Reasons behind this increased risk have been accredited to the pressure being placed upon the legs while seated cross legged [13]. The popliteal vein has been known to develop "transverse rippling" while seated, which not only facilitates venous stasis, but may cause damage to the endothelial wall [14]. With the increase in altitude the concentration of ambient air decreases, resulting in a decrease in fibrinolysis and increases the risk of clot formation [15]. The literature suggests that deep vein thrombosis development is four times more likely to develop within a given "hazard period," which is two to four weeks following a flight [16]. Interestingly, the DVT in this case developed 3-4 days post travel, which occurred much more rapidly than the typical "hazard period" of air travel.

Oral contraceptives

The athlete had been taking oral contraceptives for over a year prior to the injury. One case report indicated that the risk of DVT increased by a magnitude of four when women were using oral contraceptives (OCs) [8]. The oral contraceptive specific to this case, Tri-Lo Sprintec, is known as third generation progesterone which has a higher associated risk than some OCs [17]. The hormonal effect of both increasing coagulation and decreasing fibrinolysis can explain the genesis of the DVT. Most relevant to our case, a study found that the risk of developing a DVT was increased by a factor of fourteen when combining OCs use and air travel [8].

Genetic factors

After the diagnosis of a DVT was formulated, the hematologist pursued further testing to determine if there were any underlying genetic components contributing to the pathogenesis. Testing consisted of a blood test with DNA analysis, which confirmed a genetic mutation of Factor V. Factor V is a protein found in the blood

that is required for normal clotting to occur in the body. The genetic mutation of interest, Factor V Leiden, is an abnormal version of the clotting factor that is resistant to the action of activated protein C (APC); a substance that inactivates Factor V preventing an excessive growth of the clot [18]. Since the mutation results in APC being unable to inhibit the effects of Factor V, clots are more prone to develop, hence their correlation to the development of DVTs. This statement is supported by a study which indicated that Factor V Leiden mice had faster growing thrombi compared with non-Factor V Leiden mice [19]. There are two forms of the genetic mutation, either heterozygous or homozygous, with an increased risk of DVT found in homozygous carriers (an increased risk of about 25-50 fold) [20]. The athlete in this case tested as a heterozygous carrier of the genetic mutation. The risk of venous thrombosis once again increases in women using oral contraceptive pills or on hormone replacement therapy that also have Factor V Leiden, as there is a 35-fold increased risk of developing a DVT [20].

Conclusion

Each of the previously mentioned risk factors contributed to the etiology of this athlete's extensive DVT, reinforcing the importance of obtaining a thorough medical history to assist the medical team with the diagnosis. Using these risk factors as a guide, the following questions should be asked of an athlete presenting with a lower leg trauma:

- Have you had any period of immobilization or weight-bearing restrictions recently?
- Have you recently traveled or intend to travel in the near future (specifically air travel lasting longer than four hours)?
- Are you currently taking oral contraceptives or receiving hormonal therapy? If so, what kind?
- Do you or any members of your family have a history of DVTs or clotting disorders?

Although young, otherwise healthy athletes are not typical candidates for DVT following mild to moderate orthopedic trauma, the risk for developing thrombi should be at the forefront of a clinician's thought process when other risk and contributing factors are present. After the initial trauma, this athlete presented with only two known risk factors for a DVT (limb trauma and oral contraceptive use). At this point in the diagnostic process, suspicion of a DVT would be limited, but should not be eliminated as a potential sequela to the injury. Yet, the exacerbation of symptoms shortly following a period of prolonged travel should serve as a red flag to the diagnostic team.

Well's Clinical Prediction Rules inform us that a history of cancer, presence of paralysis, and/or lengthy immobilization are major criteria for increasing the risk for DVTs. In young athletic populations, none of these criteria are typically in the mix, nor were they in our case presentation except for the 10 hours of air and bus travel that forced her into immobilization. However, it turned out that the athlete actually had a genetic risk factor for the condition, and presented with 3 of the key clinical features upon her return trip. Collectively, our case supports the robustness of the Well's Clinical Prediction Rules and reinforces the need for the attending clinicians to perform a thorough history and pay attention to subtle clinical findings, regardless of the relatively low risk in college aged athletes. Well's Clinical Prediction Rules can be used as a diagnostic guideline for DVTs in the athletic population, but further development of research-based guidelines for return to play in athletic populations is worthy of future investigation.

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