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# Deep Vein Thrombosis in a Young, Healthy Baseball Catcher: A Case Report and Review of the Literature

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## Summary:

Venous thromboembolism is becoming increasingly recognized as a significant cause of morbidity and mortality in the hospitalized pediatric population. However, young healthy athletes can present with unique risk factors for deep vein thrombosis (DVT) that can be overlooked. Here we report a case of an adolescent male with no inherited risk factors or prior history of DVTs who developed a right femoral vein DVT in the context of playing catcher in baseball after recovering from a bout of streptococcal pharyngitis. We review the evidence that suggests that repetitive squatting-induced compression of the femoral vein can cause the venous stasis and endothelial microtrauma that contributed to the formation of this thrombus.

### Keywords

deep vein thrombosis; adolescent hematology; sports medicine

Deep vein thrombosis (DVT) is an important and increasingly recognized problem in the pediatric population, as it can have devastating consequences including pulmonary embolism, postthrombotic syndrome, and even death. One large multicenter analysis of tertiary children's hospitals in the United States found a 70% increase in venous thromboembolism (VTE) diagnoses from 2001 to 2007, from 34 to 58 cases per 10,000 children.[1] Although the cause of the increasing frequency is likely multifactorial, it has been postulated that it may reflect increased awareness and improved detection of DVTs, increased survival of children with chronic diseases that predispose to DVTs, and increased use of central venous catheters in the pediatric population. VTE is not as common in children as it is in adults, but within the pediatric population, it is most prevalent in infants and adolescents.[1] There are several identifiable risk factors. The most common of these are TV catheter use and underlying chronic conditions such as cardiovascular disease, malignancy, and neuromuscular disease.[2,3] Inherited and acquired thrombophilias along with

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infection and trauma contribute to a smaller but important proportion of the underlying causes.[2,4]

Although much of the pediatric literature is dedicated to discussion of VTE in children who are hospitalized and ill, there is significantly less analysis of this pathology in children who are relatively healthy and even athletic. Although we expect this subset of the pediatric population to be least likely to develop clots, they in fact carry unique risk factors and challenges for diagnosis that warrant increased awareness and investigation. Here we report a case of a young male adolescent with no chronic medical conditions who developed a right femoral DVT in the setting of playing catcher in baseball.

## OBSERVATION

An 18-year-old boy presented with a 1-week history of right thigh pain, calf swelling, and altered sensation of the leg. The patient had a normal developmental history and no significant chronic medical problems. Six months before presentation he was diagnosed with infectious mononucleosis and recovered several weeks thereafter. Three weeks before presentation the patient was diagnosed with and treated for streptococcal pharyngitis, which was complicated by persistent malaise, fever, loss of appetite, intermittent vomiting, and body aches. Two and a half weeks before presentation, these symptoms abruptly evolved into acute chest pain, cough, and lethargy. Evaluation in the emergency department at that time was inconclusive with a mildly elevated erythrocyte sedimentation rate of 23 mm/h and C-reactive protein of 1.3mg/dL, and normal cardiac enzymes and complete blood cell count with differential. He had an unremarkable chest radiograph electrocardiogram, and echocardiogram. He was given a diagnosis of possible mild pericarditis and was discharged home on the same day after his chest pain resolved with an TV fluid bolus and oral analgesics.

After having been ill and sedentary for the next 2 weeks, he reported losing about 15 pounds but that he had regained 12 pounds by the third week, 1 week before admission, as he was feeling and eating better. The patient is a competitive baseball player and plays the position of catcher for his team. He resumed playing 1 week before presentation, when he started feeling better. At the same time, the patient began developing right thigh pain, noticeable only at night and mild in severity. He played catcher for several hours during baseball games for 3 consecutive days. He denied any trauma to his leg during these games. After the last day of baseball games and 4 days before presentation, he noticed that his right calf had painless swelling. He denied any erythema, warmth, or weakness of his leg at the time, however, he did note some occasional tingling sensations in his right foot. This painless swelling of the calf fluctuated over the next few days, and the leg became increasingly more numb. The patient also noted firmness and worsening pain over his right thigh, particularly with exertion. He played baseball once more the day before presentation.

He denied any recent fevers, shortness of breath, chest pain, or cough. He denied any family history of thrombophilia, autoimmune disease, or hematologic malignancy. He denied smoking, alcohol, and drug use. He had just recently graduated from high school and has a college baseball scholarship to start in the next academic year.

The patient presented with normal vital signs and an occlusive right femoral vein thrombus was confirmed via ultrasound with Doppler of the bilateral lower extremities. There was no compromise to perfusion or motor function present on evaluation. No pulmonary embolism was identified on computed tomographic angiogram of the chest. Echocardiogram and electrocardiogram were normal. Laboratory evaluation was significant for D-dimers elevated to 657 ng/mL, C-reactive protein elevated to 1.1 mg/dL, and erythrocyte sedimentation rate of 25 mm. A hypercoagulability panel was sent including prothrombin gene mutation (G20210A), factor V Leiden genetic testing, anticardiolipin IgG/IgM, β-2 glycoprotein IgG/IgM, lupus anticoagulant studies, homocysteine level, protein C and S levels, and antithrombin III level. The only positive finding on this panel was a weakly positive lupus anticoagulant (Dilute Russell Viper Venom Time [DRVVT] of 53.8 s, DRVVT ratio of 1.41), but the anticardiolipin and β-2 glycoprotein antibodies were negative. The patient was started on low–molecular-weight heparin upon admission. He was discharged home after a brief hospital stay on a 3-month course of oral rivaroxaban with close outpatient follow-up.

He was advised to avoid athletic activities that would involve squatting for the first 3 months of anticoagulation and to stay well-hydrated during exercise. Three weeks after discharge he developed swelling and pain in the right calf, causing him to limp, consistent with postthrombotic syndrome. Repeat antinuclear antibody testing, prothrombin time, partial thromboplastin time, and DRVVT were normal within 3 months of discharge from the hospital. A Doppler ultrasound of the area of the original thrombosis after 3 months of treatment with rivaroxaban showed no significant change in the thrombus, so he is continuing the rivaroxaban for another 3 months.

At the time of manuscript submission he is in the second of the additional 3 months on rivaroxaban (20 mg every morning) and has returned to participate in college baseball for the past month, under the care of a National College Athletic Association (NCAA) physician, with a 50/50 mixture of playing catcher and outfielder to avoid prolonged periods of squatting. Physical training has contributed to significant improvement in his postthrombotic syndrome symptoms. Given that rivaroxaban has a half-life of 5 to 9 hours in healthy subjects, the NCAA physician also recommended an anticoagulation treatment plan to hold rivaroxaban for at least half a day before competitive games by advancing his morning dose on game days to the night before or delaying his daily dosing until after the game. He has been able to play baseball without significant bleeding symptoms with this regimen.

## DISCUSSION

This case illustrates an unusual presentation of DVT in a young athlete with no history of chronic medical conditions, trauma, or prior DVT. One informative aspect of this patient's history is his frequent and longstanding athletic activity as a baseball catcher. He played his position repeatedly over several days leading up to his presentation, which was temporally related to the development and worsening of his symptoms. We postulate that a combination of dehydration from athletic activity, a deconditioned and proinflammatory state from his recent systemic illness, and venous stasis from his frequent catching activity in the days before presentation provided the multifactorial combination of stressors that provoked the formation of his right femoral vein thrombus. To our knowledge, this is the first reported

episode of a DVT of the lower extremity associated with baseball catching in a male adolescent. In looking at the Virchow classic triad for venous thrombosis (hypercoagulability, endothelial injury, and venous stasis),[5] the prolonged and repetitive squatting required of baseball catchers may affect several of these components through compression of the blood vessels in the pelvic, inguinal, and popliteal areas.

In assessing for underlying hypercoagulability states, the patient had no known family history of clotting disorders. His lab workup for inherited and acquired thrombophilias was essentially unremarkable aside from a weakly positive lupus anticoagulant (elevated DRVVT) and mildly elevated inflammatory markers. Although elevated DRVVT could be evidence of antiphospholipid antibody syndrome, a mild elevation can often be found transiently without clinical significance in the setting of viral or bacterial illness or associated with certain medication such as penicillin[6,7]; therefore, in the absence of other positive antiphospholipid antibody tests this does not definitively demonstrate that he has an underlying antiphospholipid antibody syndrome. Furthermore, the DRVVT was normal on subsequent testing. Dehydration and inflammation have been known to contribute to a transient hypercoagulable state.[8,9] Therefore, the patient's recent illness and decreased oral intake may have resulted in an underlying predisposition toward clotting. There is little evidence in the literature to suggest that baseball catching itself would directly cause sufficient hypercoagulability to provoke a thrombosis in the absence of other risk factors, although 1 study described an interesting phenomenon of "orthostatic hypercoagulability."[10] Specifically, prolonged standing still in young healthy individuals caused a transient decrease in plasma volume and protein C activity with a corresponding increase in fibrinogen, factor V, factor VIII, prothrombin, tissue factor, and von Willebrand factor levels. One might surmise that similar effects could occur with prolonged squatting in position.

The components of endothelial injury and venous stasis likely played a significant role in this patient's DVT. One meta-analysis found the rate of catheter-related thrombosis in children to be as high as 20%.[3] In this case the patient had no history of IV catheter placement or trauma. He did experience a more sedentary period of time several weeks before presentation due to illness that could have contributed to venous stasis. However, the literature also provides several alternate mechanisms of endothelial injury and venous stasis that can arise in otherwise healthy young individuals.

"Effort thrombosis," otherwise known as Paget-Schroetter disease, is a DVT of the axillary-subclavian vein most commonly seen in athletes who perform repetitive movements of the arm or shoulder, especially overhead activity. The pathophysiology includes underlying thoracic outlet abnormalities (such as a cervical rib) and/or hypertrophied muscles in the costoclavicular region that lead to repetitive compression of the subclavian vein. This repetitive endothelial microtrauma and stagnation of blood flow eventually predisposes the patient to thrombus formation in the subclavian and axillary veins.[11] This phenomenon has been documented in athletes in a variety of sports ranging from baseball to billiards[12] and even among baristas.[13]

May-Thurner syndrome is another compressive mechanism that can lead to DVT in healthy young patients. It involves an anatomic abnormality in which the left common iliac vein is

compressed between an overriding right common iliac artery anteriorly and the lumbosacral vertebra posteriorly. This compression has been shown to cause chronic endothelial irritation leading to hyperplasia and the formation of venous “spurs.” These spurs can eventually cause obstruction of venous flow and result in a left-sided DVT of the iliofemoral veins.[11,14]

Popliteal venous compression is another possible mechanism for initiation of DVT. The popliteal vein can be compressed in the popliteal fossa by a combination of the popliteal artery, the head of the gastrocnemius, and/or popliteal cysts.[11] This compression has been most often illustrated in the context of full knee extension, with lack of data on whether full knee flexion (beyond 90 degrees) would also have a similar compressive effect. Nonetheless, this is another example of a position-dependent etiology of venous compression that can lead to DVT. One study found that when the knees were flexed, characteristic patterns of rippling microtrauma in the walls of the popliteal vein were seen at the site of thrombosis in autopsies of patients with a history of long-lasting flexion or repetitive flexion-extension of the knee.[15]

These venous compression syndromes, while anatomically distinct from this baseball catcher’s right femoral vein thrombosis, may provide clues as to how his repetitive and prolonged squatting led to a lower extremity DVT. Repeated squatting could potentially cause compression of the common or proximal femoral veins similar to the compression of the subclavian, common iliac, or popliteal veins in these syndromes. Prolonged venous stasis and endothelial microtrauma could have contributed to the formation of his thrombus. Squatting has been associated with DVT in one other isolated case report, a healthy 21-year-old carpet fitter who spent much of his time squatting as part of his job who developed acute thromboses in his left common femoral, superficial femoral, and great saphenous veins.[16]

The effect of hip flexion on venous flow has been directly studied in patients placed in stirrups in the operating room to evaluate the best amount of flexion for prevention of DVT.[17] The authors found that hip flexion at 90 degrees as compared with 0 (hips extended) or 30 degrees was associated with significantly reduced vessel diameter in the common femoral vein and proximal femoral vein. A catcher’s resting position typically involves flexion of the hips at significantly > 90 degrees, which would be expected to have an even more occlusive effect than 90 degrees. Furthermore, in the catcher’s position, the knees and ankles are also fully flexed, further decreasing venous blood flow return from the distal lower extremity and increasing the risk of venous stasis. In support of this idea, an anatomic investigation assessing dynamics of venous patency during total hip arthroplasty found that flexion, internal rotation, and adduction of the hip in the supine or lateral positions led to kinking and occlusion of the femoral vein. The site of stenosis generally occurred about 5 to 7.5 cm below the inguinal ligament.[18]

The importance of highlighting this case is not only to analyze the unique pathophysiology of DVT associated with the baseball catcher’s positioning but also to increase the awareness of DVT risks in a population generally considered to be low-risk, young athletes. The increased public level of awareness of DVT risk during prolonged airplane flights has led to behavioral modifications to decrease this risk.[19] Perhaps similar interventions, such as stretching after a certain time holding a fixed position and improved hydration, would be

beneficial for catchers and other athletes with roles that predispose to venous stasis and endothelial injuries through prolonged static positioning. It may also be beneficial to have a lower threshold of suspicion for DVTs in athletic patients who may be subject to repetitive and prolonged squatting or other compressive stances as part of their sport.

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