Ulnar Deep Venous Thrombosis in a Professional Baseball Pitcher

A Case Report

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The repetitive motions in overhead throwing place athletes at risk for specific injuries to the dominant shoulder and elbow. Although uncommon, vascular injuries must be considered in this differential. Examples of these are digital vessel thrombosis, proximal arterial thrombosis with distal embolization, vessel aneurysm, and vessel compression.[7] Paget-Schroetter syndrome, or effort thrombosis, is well described and refers to spontaneous axillosubclavian vein thrombosis secondary to mechanical compression in the costoclavicular space.[11] Multiple case series exist in the literature regarding Paget-Schroetter syndrome in young, healthy athletes.[6,16,17,22,26] It is important, however, to differentiate axillosubclavian vein thrombosis from a distal deep venous thrombosis (DVT). To our knowledge, there are no reported cases of deep venous thrombosis occurring distal to the elbow in throwing athletes. The purpose of this article is to report a case of ulnar vein thrombosis in a professional baseball pitcher and to provide a management model.

CASE REPORT

A 22-year-old male professional baseball pitcher awoke from overnight sleep with complaints of swelling, a feeling of tightness, and mild paresthesias in his dominant hand and forearm. He threw 77 pitches the previous evening and fell asleep wearing a circumferential, elastic, thermal elbow compression sleeve from midhumerus to midforearm. There was no apparent injury or abnormal trauma during the game. He reported no symptoms during the game or in the training room after the outing and denied any previous personal or family history of thrombosis as well as any prior occurrence of these symptoms. He was an otherwise healthy individual without any previous upper extremity surgery. His only medication was Naprosyn, 500 mg, twice daily (taken routinely during the season for various other conditions such as wrist tendinitis). The patient admitted to using occasional chewing tobacco, but denied smoking, alcohol, or illicit drug use, including anabolic steroids or other performance-enhancing substances. Further systems review was negative for shortness of breath, cough, or inspiratory chest pain.

On examination following the onset of symptoms, he was noted to have mild, nonpitting edema of the hand without discoloration. There was no tenderness to palpation across the hand or forearm. The veins of the arm were not distended. Capillary refill was brisk, and his radial pulse was 2+ and symmetric. The distal neurological examination was normal. Range of motion testing of the elbow was unchanged from baseline examinations. Stability testing revealed an intact ulnar collateral ligament (UCL). Radiographs of the elbow and forearm demonstrated some ossification in the UCL, but there were not significant osteophytes, loose bodies, or loss of joint space.

A duplex ultrasound was ordered immediately after the athlete reported his symptoms. It demonstrated a 4 cm, partially occlusive, acute thrombosis of the ulnar vein in the distal forearm (Figure 1). Initial laboratory tests, including a complete blood count, chemistry panel, and coagulation panel, were drawn and were all within normal range. All of the following tests were normal and were included as part of his hypercoaguable workup: D-dimer, fibrinogen, APC (activated protein C) resistance, functional protein C and S, and antithrombin III activity. Factor VIII activity was mildly elevated compared with the

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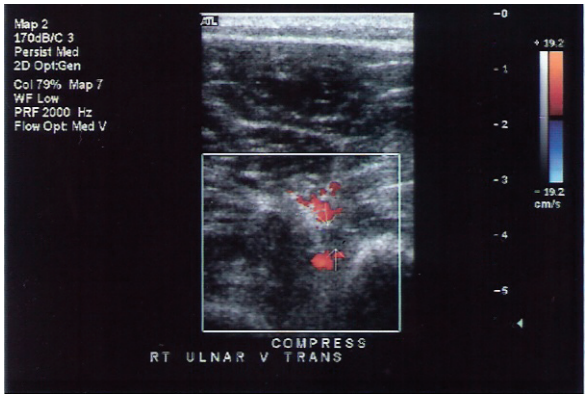

Figure 1. Cross-sectional venous duplex ultrasound of right midforearm. Small arrows outline the walls of the ulnar vein. Red indicates arterial flow toward the transducer. Blue indicates venous flow away from the transducer. Lack of venous flow and incompressibility demonstrate acute deep venous thrombosis.

normal parameters of our institution's laboratory. The pitcher was started on a daily 40 mg dose of subcutaneous enoxaparin sodium injection. He returned for a repeat duplex scan 2 days after diagnosis. There was complete resolution of the thrombus on this study (Figure 2). He continued taking enoxaparin at the prophylactic dose. He was allowed to pitch on the fifth day after diagnosis with the precaution that if he were to sustain a blunt trauma he would immediately exit the game for emergent evaluation. Moreover, his enoxaparin administration was timed to be given approximately 24 hours before his scheduled outing to minimize bleeding risk. He threw 106 pitches in that outing and was asymptomatic. A third duplex was performed in consultation with a vascular surgeon approximately 1 week after diagnosis. It was negative for thrombosis. The patient continued taking enoxaparin for a total of 4 weeks. A follow-up duplex study performed 3 months later was normal.

Overall, the patient did not miss any competition because of his condition. He continues to be asymptomatic approximately 2 years after diagnosis. He has no evidence of postphlebitic syndrome. Had the second duplex examination demonstrated extension of the thrombus, he would have been started on full-dose anticoagulation for 3 to 6 months and held out of competition while taking the anticoagulant.

DISCUSSION

We describe a case of ulnar vein thrombosis in a professional baseball pitcher with a successful outcome. Since the player was clinically symptomatic, and the thrombus was confirmed on duplex, we agreed that it should be treated. The treatment plan was developed by a multidisciplinary team of specialists with the goals of successful medical treatment, patient safety, and continuation of competition.

Why did the player develop this condition? His hypercoaguable workup was negative, and he otherwise had no risk factors. It seems most likely that the elbow compression sleeve worn to bed that evening created an environment of venous stasis. Many pitchers routinely put on an elbow sleeve after throwing to keep the elbow warm (as in this case), although they are not supposed to wear them to sleep. The player changed his postgame routine after this diagnosis, and we do not recommend compressive sleeve wear for prolonged periods of time after throwing.

We do not believe that anabolic steroids or other performance-enhancing substances contributed to this condition. The relationship between anabolic steroids and DVT is unclear in the literature. Many of the intended effects and side effects are well known. Some of the more deleterious effects include hepatic cellular damage, testicular atrophy, and psychological disturbance.[25] From a vascular standpoint, studies have focused on the association between steroid use and developing an atherogenic blood lipid profile with endothelial dysfunction leading to an overall increased risk of atherosclerosis.[25]

Interestingly, studies have shown that androstenedione supplementation has been shown to cause a significant

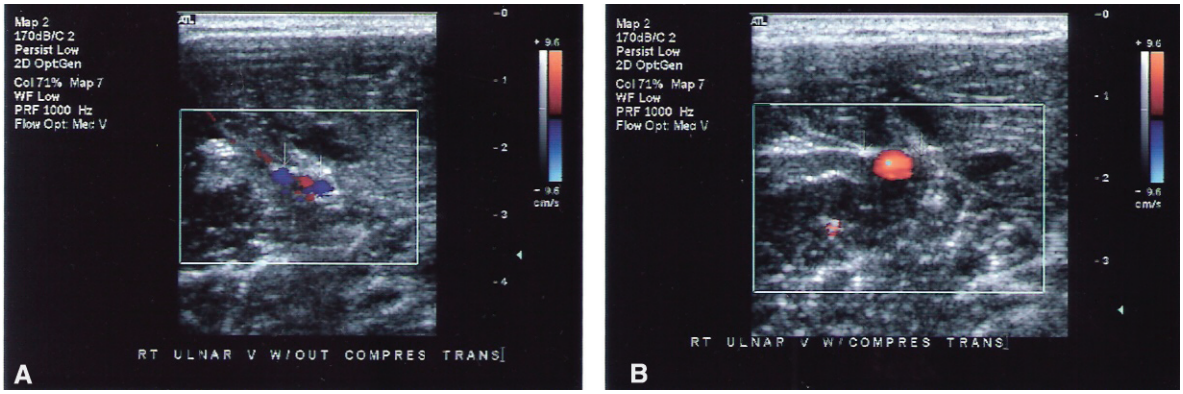

Figure 2. Repeat cross-sectional venous duplex ultrasounds taken in same plane 2 days later showed resolution of thrombus. (A) Red and blue flow indicates patent arterial and venous lumens in absence of compression. (B) Venous lumen obstructed by compression with ultrasound probe head.

increase in estrogen concentration.[2,3,14] Moreover, the link between hormone replacement and DVT is well known.[10] One could then infer that androstenedione could be a risk factor for the development of DVT, but in our case the patient denied use. Last, although his overall hypercoaguable workup was negative, the risk still exists for recurrent thrombosis. One study showed a 2.2% rate of recurrent DVT at 3 months despite treatment, although the patient population was quite different.[20]

What happened to the thrombus in the 2 days between the initial positive duplex and the subsequent negative study? First, we believe that thrombus did occur. The patient exhibited clinical signs and symptoms, and it was documented on duplex examination and reconfirmed. The follow-up duplex, which was negative, was repeated by the same technologist with the aid of the images from the index ultrasound. Therefore, the thrombus most likely embolized. This is not surprising given that pulmonary embolism (PE) may be the presenting manifestation in up to 36% of patients.[19] This is similar to the rate of silent PE associated with lower extremity DVT (40%-50% in one study).[15] Fortunately, in this situation, the embolism was small and subclinical given the athlete's pulmonary fitness. It is unlikely that it lysed on therapy given that the main effect of low-molecular-weight heparins (LMWH) is to prevent clot progression. However, LMWH has been shown to reduce thrombus size.[9]

Low molecular weight heparins have been recommended as a form of treatment for upper extremity DVT in multiple sources, including the American College of Chest Physicians (ACCP).[4,20] Since there are no published treatment guidelines for this situation in athletes, a treatment plan was formed by extrapolating from the pertinent available literature. Despite the location of the thrombus in the deep venous system of the forearm, it was categorized as superficial because of its small size. Once that judgment was made, the results from the Superficial Thrombophlebitis Treated by Enoxaparin Study Group were applied. In that study, 40 mg of subcutaneous enoxaparin reduced the incidence of lower extremity DVT and embolic events in patients with documented acute symptomatic superficial vein thrombosis of the legs.[23] The next decision involved duration of treatment. The optimal duration of anticoagulant therapy after a first episode of venous thromboembolism remains controversial in the medical literature.[1,18] Applying published recommendations to this case was even more challenging. Keeping in mind that this thrombus was categorized as "superficial," 4 weeks of enoxaparin therapy was chosen based on ACCP guidelines for the treatment of superficial thrombophlebitis.[4] In addition, because the repeat Doppler 3 days after diagnosis was negative, and the patient was asymptomatic at that time, the duration of treatment was thought to be adequate.

Treatment was deemed important to prevent complications of DVT, including postphlebitic syndrome (PTS). The frequency of PTS after upper extremity DVT ranges from 7% to 46%.[8] Pain, heaviness, swelling, and skin ulceration are common signs. Risk factors for occurrence of PTS appear to be ipsilateral recurrence of DVT and poor quality of initial anticoagulation for the treatment of DVT.[12] Therefore, it is important to adequately treat the current DVT and try to prevent DVT recurrence with adequate duration and intensity of anticoagulation.[13] Again, 4 weeks of treatment was deemed necessary to prevent this complication.

Multiple studies have shown that the use of enoxaparin is safe in terms of major bleeding complications.[20,21,23] In this situation, there was minimal risk involved in playing while taking anticoagulants. According to published drug information, enoxaparin is an LMWH with a peak onset of 3 to 5 hours and duration of approximately 12 hours. It is metabolized by the liver and excreted in the urine. It is considered advantageous to other forms of prophylaxis or treatment because of its longer plasma half-life, lesser variability in the anticoagulant response to a fixed dosage, and better efficacy-to-safety ratio.[21] Additionally, the medication is easy to use in the outpatient setting and does not require partial thromboplastin time or heparin level monitoring.[5] Although no studies exist regarding athletic safety while on enoxaparin, the medication dosing was timed such that most of the drug was eliminated before competition. In addition, had trauma occurred, for example, being struck with a baseball, a comprehensive plan of action that was previously communicated to all involved would have been executed. This included on-field evaluation by training staff, prompt removal from competition, and transfer to a designated medical facility for thorough evaluation and observation. If LMWH reversal had been necessary, slow intravenous protamine sulfate (1% solution) has been shown to neutralize 60% of Anti-Factor Xa activity at a dose of 1 mg protamine per 1 mg of enoxaparin.[24] The pitcher's risks were further minimized because of the designated-hitter rule in the American League. In conclusion, LMWH was chosen because of its efficacy, ease of use, compliance, player safety, and predictable duration of action.

We can make the following observations regarding this case. First, it appears that rapid diagnosis and prompt treatment from a multidisciplinary medical team resulted in no time lost from competition. Second, we recommend avoiding extended use of a compressive sleeve after pitching, given that this may contribute to venous stasis. Finally, the possibility exists that this problem may go undiagnosed, with resolution or subclinical embolization before recognition. Conversely, embolization may occur even when the condition is promptly recognized and treated. It is still important to initiate and complete treatment to prevent recurrence and PTS. In our opinion, this case demonstrates a challenging clinical problem that was successfully managed with extrapolation of evidence-based medicine from multiple disciplines.

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