

Unique Treatment Regimen for Effort Thrombosis in the Nondominant Extremity of an Overhead Athlete: A Case Report

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Objective: To advise athletic trainers on the potential for effort thrombosis to occur in nonthrowing athletes and to underscore the importance of early recognition and treatment.

Background: An 18-year-old offensive lineman presented with a 1-day history of diffuse shoulder pain with no specific history of injury; swelling and erythema involved the entire left upper extremity. He was immediately referred to the team physician, who suspected deep vein thrombosis and sent the athlete to an imaging center. Duplex ultrasound was obtained on the day of presentation, and he was admitted to the hospital that evening.

Differential Diagnosis: Deep vein thrombosis, thoracic outlet syndrome, shoulder tendinitis.

Treatment: Anticoagulation with heparin was administered at the hospital, and he was sent home the next day on subcutaneous enoxaparin sodium, followed by a 5-mg daily

dose of oral warfarin sodium. Oral anticoagulants were continued for a total of 4 weeks. The athlete began upper body lifting and was released 5 weeks postinjury to gradually return to football without restrictions.

Uniqueness: Effort thrombosis is typically seen in the dominant arm of athletes, and the current treatment protocol calls for thrombolysis or surgical intervention. This athlete, whose position required repeated elevation of his arms in forward flexion, sustained the injury in his nondominant arm, was treated with anticoagulation only, and had a full return to football. At 18-month follow-up, he had no recurrence of symptoms.

Conclusions: Early recognition and treatment of athletes with effort thrombosis is paramount to a successful clinical outcome and prompt return to play.

Key Words: Paget-Schroetter syndrome, football injuries

Primary venous thrombosis of the upper extremity can be idiopathic or activity related, as occurs in effort thrombosis (or Paget-Schroetter syndrome). The term *Paget-Schroetter syndrome* was first used by Hughes in 1949¹ to recognize 2 of the first cases of upper extremity thrombosis reported by Paget in 1858² and Schroetter in 1899.³ Since that time, the evolution of clinical treatment for effort thrombosis has been well documented in the literature.⁴⁻¹⁰ Classically, the treatment of this disorder consisted of bed rest, extremity elevation, and anticoagulation therapy.⁴⁻⁶ High recurrence rates and residual symptoms led to the implementation of more aggressive management with thrombolytics.^{4,5,7} More recently, the importance of achieving venous patency by surgical decompression of the axillosubclavian system has been advocated to prevent long-term symptoms.⁸⁻¹⁰

Multiple case reports and case series have documented the evolution of clinical recognition and treatment of this disorder.^{4-6,9-22} The most common presentation is acute swelling of the dominant extremity; antecedent trauma or repetitive overhead activity has occasionally occurred.^{5,6,16} Early recognition and aggressive treatment of this disorder has been recommended to avoid the long-term sequelae associated with chronic venous obstruction.^{12,16,23} Most reports of effort thrombosis associated with sport participation document occurrence of this disorder in the dominant extremity of overhead throwing athletes.¹⁶ Furthermore, several authors^{6,24} recommended thrombolytic therapy and surgical decompression in the young active population. We present a unique treatment regimen

for effort thrombosis in the nondominant extremity of a nonthrowing athlete who was successfully treated acutely with anticoagulation therapy alone.

CASE REPORT

A male, right-hand dominant, 18-year-old National Collegiate Athletic Association Division I collegiate football offensive lineman presented to his athletic trainer with a 1-day history of left shoulder pain. He was referred to the team's primary care physician and was seen the same day. He did not report a specific inciting event or injury but noted diffuse upper extremity pain that started during football practice the day before. He had no prior episode of a similar problem and no family history of thrombotic disorders. Physical examination revealed erythema and nonpitting edema involving the entire left upper extremity. At a level 14 cm distal to the acromioclavicular joint, the diameter of the left upper extremity was 4.6 cm larger than the right side at the same level. Radial pulses were 2+ on both the uninvolved and involved sides. He denied any chest pain or shortness of breath. The neurologic examination was normal.

Upper extremity deep vein thrombosis (DVT) was suspected, and the patient was immediately referred to an imaging center for further evaluation, where duplex ultrasound was obtained. Absence of flow in the subclavian vein and abnormal flow distal to the obstruction, including the axillary, brachial, cephalic, and basilic veins, was noted. He was admitted to a local hospital overnight for

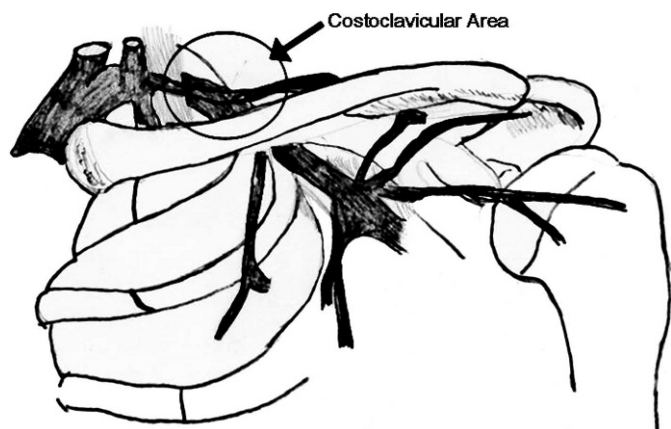


Figure 1. Costoclavicular area. Narrowing of costoclavicular gate (see circle) results in impingement of artery and vein.

anticoagulation with heparin. Upon discharge, the patient was sent home on subcutaneous enoxaparin sodium, dosed by weight at 1 mg/kg 2 times per day for DVT prophylaxis, followed by a 5-mg daily dose of oral warfarin sodium. The patient's international normalized ratio (INR) was therapeutic (2.0–3.0) upon discharge. Enoxaparin sodium treatment was complete 5 days after hospitalization, and the warfarin sodium was continued. A subsequent hypercoagulability workup including factor V Leiden, prothrombin gene mutation, protein C activity, protein S activity, antithrombin III, and methylenetetrahydrofolate reductase mutations failed to demonstrate abnormalities. The patient's INR was assessed twice during the first week of discharge from the hospital (values = 1.9 and 2.2; reference range, 2.0–3.0). At this time, the patient began lower extremity weight lifting and moderate cardiovascular exercise (stairstepping machine and light jogging). After a final hematology consult at 4 weeks posttreatment, the warfarin sodium was discontinued and the patient was cleared to begin upper body weights with close symptom monitoring. With no recurrent symptoms, he was cleared for limited football participation 5 weeks after treatment began. He returned to unrestricted participation 4 months postinjury. At 9-month follow-up, the patient was asymp-

tomatic without sequelae and participated in football without restriction. Unrelated to this injury, he was redshirted the next season. He practiced the entire year without any further problems and at 18 months postinjury, he remained asymptomatic.

DISCUSSION

Anatomy

A basic understanding of the anatomy of the axillosubclavian venous system is necessary for the recognition and treatment of effort thrombosis. The most commonly recognized site of compression of the axillosubclavian vein is the costoclavicular area (Figure 1). In this region, the first rib lies inferior and posterior, the anterior scalene muscle lies posterior, and the clavicle, costoclavicular ligament, and subclavius muscle lie anterior to the vein.²⁵ Additionally, during shoulder abduction, the pectoralis minor tendon or the head of the humerus may compress the axillary vein.²⁶

Clinical Diagnosis

Although some patients may be completely asymptomatic, many have the clinical presentation of localized swelling of the involved extremity with possible associated erythema. Patients may complain that the extremity feels “heavy” and experience a dull ache in the shoulder, neck, or axillary regions. Physical examination may reveal dilation of the superficial veins of the involved extremity, but often the neurologic examination is normal. Occasionally, external compression of the axillosubclavian arterial system may be recognized with provocative maneuvers such as the Adson (Figure 2) and Wright (Figure 3) tests. The diagnosis of effort thrombosis should be suspected clinically and confirmed with contrast venography or, as in this case, duplex ultrasonography.^{27–29}

Two unique aspects of this case deserve mention. First, primary effort thrombosis is a rare disorder (2 in 100 000 persons per year) and usually affects the dominant extremity of active individuals.³⁰ Offensive linemen are



Figure 2. In the Adson maneuver, A, the clinician extends the patient's arm on the affected side and monitors the radial pulse. B, As the patient extends the neck and rotates the head toward the same side, the clinician gradually elevates the arm. With the patient taking a deep inspiration, a decrease in the radial pulse indicates compression of the subclavian artery.



Figure 3. In the Wright maneuver, the patient hyperabducts the arm to 180° with the elbow slightly flexed, and the clinician monitors the radial pulse. Loss of the radial pulse constitutes a positive test.

required to elevate their arms in the forward-flexed position to maintain a pass block. We theorize that this repeated action could be the mechanism of venous intimal injury and subsequent axillosubclavian venous thrombosis in the absence of an identifiable external compression. If that is truly the case, we would expect the side of occurrence to be random, as both extremities are subjected to this repeated action. Therefore, the occurrence of a nondominant-side effort thrombosis should not be entirely unexpected in this population of athletes.

Secondly, the decision to treat this patient with anticoagulation only (without thrombolysis or surgical decompression) deserves discussion. Advocates of surgery for effort thrombosis have noted that lysis of the thrombus alone, anticoagulation therapy alone, or a combination of the two is “inadequate treatment.”⁹ Aggressive treatment, such as thrombolysis and surgical decompression, is thought to be most effective for the prevention of long-term problems in young and active patients.^{6,24} Judicious thrombolysis followed by a course of anticoagulation has also been advocated, with surgical decompression reserved for refractory conditions.²² In cases of external compression, such as an element of thoracic outlet syndrome or clavicular impingement, acute correction of the anatomy would be advocated by most vascular surgeons.^{9,12} In their literature review that included more than 2500 patients, Sajid et al³⁰ found no superiority of treatment between anticoagulation therapy alone and thrombolysis for the general treatment of upper extremity DVT. Of the reported cases, only 20% were primary upper extremity DVTs that occurred due to activity-related venous trauma.

The patient described in this case report had no evidence of traumatic injury and no source of external compression. The decision to treat the patient with only 4 weeks of

anticoagulation therapy alone was made by a multidisciplinary team of physicians based on the clinical evaluation and the patient's symptoms. This patient presented with a unique case, and this treatment should not be generalized to all patients with effort thrombosis.

CONCLUSIONS

Effort thrombosis of the upper extremity is a condition that usually affects the dominant arm of overhead athletes. This disorder should be suspected in athletes performing repetitive overhead activities who develop acute-onset swelling. As noted above, the disorder should also be suspected in athletes who perform repeated arm movements and present with symptoms characteristic of effort thrombosis. Treatment of this disorder varies and depends on multiple presenting factors, including chronicity and the presence of external venous compression. Early recognition and treatment of athletes with effort thrombosis is paramount to a successful clinical outcome and prompt return to play.

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