[Primary Care]

Effort Thrombosis Presenting as Pulmonary Embolism in a Professional Baseball Pitcher

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Context: Effort thrombosis, or Paget-Schroetter's syndrome, is a rare subset of thoracic outlet syndrome in which deep venous thrombosis of the upper extremity occurs as the result of repetitive overhead motion. It is occasionally associated with pulmonary embolism. This case of effort thrombosis and pulmonary embolus was in a 25-year-old major league professional baseball pitcher, in which the only presenting complaints involved dizziness and shortness of breath without complaints involving the upper extremity—usually, a hallmark of most cases of this condition. The patient successfully returned to play for 5 subsequent seasons at the major league level after multimodal treatment that included surgery for thoracic outlet syndrome.

Objective: Though rare, effort thrombosis should be included in the differential diagnosis of throwing athletes with traditional extremity-focused symptoms and in cases involving pulmonary or thoracic complaints. Rapid diagnosis is a critical component of successful treatment.

Keywords: effort thrombosis, upper extremity thrombosis, pulmonary embolism, Paget-Schroetter's syndrome, professional pitcher, baseball, deep venous thrombosis

Effort thrombosis, also called Paget-Schroetter's syndrome,[49] is the primary occurrence of a deep venous thrombosis in the upper extremity secondary to repetitive activity involving the shoulder. It is a rare variant of thoracic outlet syndrome. Patients are often involved in activity requiring hyperabduction and external rotation.[1] Symptoms focus on the extremity and include pain, swelling, weakness, color or temperature differences, and a sense of "heaviness" or a "dead arm."

CASE REPORT

A 25-year-old right-hand dominant major league starting pitcher left the game after the third inning complaining of increasing dizziness and shortness of breath. During warm-up, he had noted mild dizziness and mild shortness of breath but had not reported these symptoms to the training staff or coaches. These symptoms worsened during the course of the first 3 innings of the game, and he was removed after pitching in the third inning. Upon questioning, he did admit to shortness of breath while going up stairs over the previous 3 days. Five days before the game in which his symptoms became problematic, he had pitched a complete game with no symptoms at all. On physical examination at the ballpark, he was comfortable at rest but noted to be mildly dyspneic while walking. He was tachycardic, with a pulse in the 120s. Examination of his right upper extremity showed full range of motion with no evidence of objective weakness, instability, swelling, discoloration, or other problems. Neurologic and vascular examinations yielded normal findings symmetric to the left side, with normal 2+ pulses and reflexes, full strength in all major muscle groups, and full sensation in the distribution of all major peripheral nerves and dermatomes. The game was played at an altitude of more than 5200 feet (1560 m) above sea level, possibly explaining the shortness of breath for a nonacclimated player. The presentation was still concerning, however, because this was the player's home ballpark and he was well acclimated to the high altitude. He was therefore sent to the emergency room for further evaluation.

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On presentation to the emergency room, his oxygen saturation (breathing room air) was measured at 87%; pulse, 100; and blood pressure, 154/71 mm Hg. With auscultation, lung fields were noted to be clear. Laboratory evaluation (including complete blood count, chemistry, coagulation, and cardiac enzyme panels) were normal, with the exception of a mildly elevated creatine kinase and D-dimer. Electrocardiogram and chest radiograph examinations were normal. His clinical findings warranted a pulmonary computed tomography angiogram. This study showed segmental pulmonary emboli in both upper and lower lobes and a small infiltrate in the left lower lobe. The patient was admitted to the intensive care unit and started on intravenous heparin for the pulmonary emboli. A lower extremity ultrasound was performed that showed no evidence of deep venous thrombosis. An ultrasound of the upper extremities revealed acute deep venous thrombosis involving the right subclavian and axillary veins—consistent with a diagnosis of effort thrombosis.

The following morning, a transthoracic echocardiogram showed a normal ejection fraction with normal right heart pressures. Extensive laboratory evaluation for a clotting disorder was negative. The patient underwent an ultrasound-guided venogram with mechanical thrombectomy and catheter-directed venolysis with tissue plasminogen activator. Most of the axillary vein and all of the subclavian vein were thrombosed. An infusion catheter was left in place for continued tissue plasminogen activator venolysis.

The following day, repeat venography showed a small amount of residual mural thrombus in the subclavian vein. When the patient fully flexed his shoulder, complete occlusion of the right subclavian vein occurred, with shunting of venous return into superficial collaterals leading to the right internal jugular vein—thus confirming the suspected diagnosis of effort thrombosis. Thrombolytic therapy with tissue plasminogen activator was continued overnight to remove the remaining thrombus. Consultation with a vascular surgeon led to the recommendation for first rib resection due to the occlusion seen with forward shoulder flexion during venography. The patient was discharged home on oral warfarin after a clear ultrasound, and plans were made for the surgery.

One month later, the patient underwent a right paraclavicular thoracic outlet decompression. This included radical anterior and middle scalenectomy, brachial plexus neurolysis, complete resection of the first rib (Figure 1), circumferential external subclavian vein venolysis, and creation of a right radiocephalic arteriovenous fistula. The fistula was created to decrease risk of thrombus recurrence and to detect early complications. The patient had an uneventful hospital course and was discharged on postoperative day 5. Three months later, he underwent a takedown of the venous fistula.

The patient went through an intense rehabilitation program that progressively focused on range of motion, lower extremity and core strength, cardiac/systemic conditioning, and rotator cuff and periscapular muscular strength. Six months following his pulmonary embolism and 5 months after the thoracic outlet decompression, the patient returned to throwing—beginning with an interval program and gradually advancing to full-speed pitching off the mound. Over the subsequent 3 months, he returned to pitching and resumed his previous competitive level almost 1 year after the initial event. At 5-year follow-up, he continues to pitch at the major league level and was even named the starting pitcher for the All-Star Game. The player gave permission for the production of this case report, and he served as a national spokesman for deep venous thrombosis awareness.

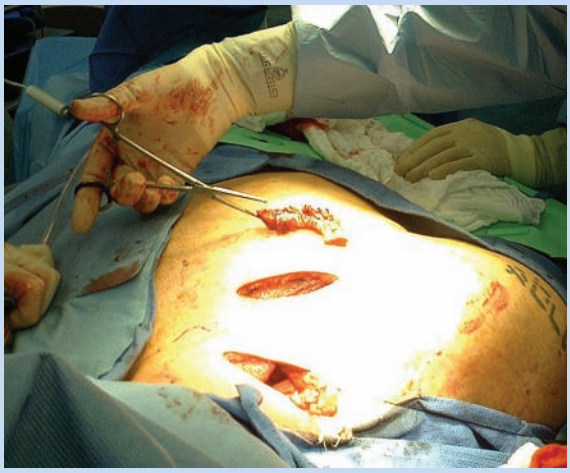

Figure 1. Intraoperative photo of the resection of the first rib.

DISCUSSION

Like its counterpart in the lower extremity, upper extremity deep venous thrombosis can occur for various reasons (Table 1).[10,11] It typically occurs as a result of intimal damage to the subclavian vein in a predisposed individual. Penetrating trauma, extravascular compression, vascular intervention, and caustic substances can cause intravascular irritation resulting in thrombosis. Hypercoagulability, congenital venous malformations, drug abuse, upper extremity trauma, and repetitive overhead shoulder activities can predispose to this condition.[21] The thrombosis may initially develop in the subclavian vein and extend distally into the axillary vein.

Authors coined the term *effort thrombosis* to refer to a "forceful event producing direct or indirect injury to the vein."[3] This event can be a single simple incident—spanking of a child.[8] Effort thrombosis occurs more commonly as a culmination of chronic injury resulting from repetitive microtrauma.[33] Most cases involve young, healthy, athletic men participating in overhead athletic or occupational activities, such as throwing, swimming, racquet sports, pass blocking,

Table 1. Causes of upper extremity thrombosis.

- I. Intimal injury
  - a. Penetrating trauma
    - i. Intravenous catheter placement
    - ii. Stab wound
    - iii. Vascular surgery procedure
  - b. Blunt trauma
    - i. Sudden blow to shoulder/arm
    - ii. Sustained pressure from positioning during surgery or while otherwise unconscious
  - c. Extrinsic compression
    - i. Thoracic outlet syndrome
      1. Cervical rib
      2. Clavicular exostosis
      3. Anterior scalene hypertrophy
      4. Musculofascial banding
      5. Sternoclavicular joint arthrosis
    - ii. Clavicle fractures
    - iii. Local compressive neoplasm or mass
    - iv. Repetitive overhead activity
      1. Normal motion with anatomic predisposition
      2. Physiologic extreme motion with either normal or abnormal anatomy
  - d. Caustic internal injury
    - i. Intravenous medications
    - ii. Radiographic dye
    - iii. Illegal drugs
- II. Venous stasis
  - a. Congestive heart failure
  - b. Radical mastectomy or other procedures altering venous flow
  - c. Local compression of venous system by neoplasm or mass
  - d. Congenital malformations
- III. Hypercoagulability
  - a. Hematologic disorders
  - b. Cancer or other systemic diseases
  - c. Oral contraceptives
  - d. High altitude

repetitive lifting, and chopping wood.[||] Effort thrombosis has been documented most extensively among baseball players (Table 2).[¶]

In addition to repetitive microtrauma, an anatomic abnormality or summation of abnormalities often predispose patients to effort thrombosis—similar to those that cause thoracic outlet syndrome,[29,47] which is caused by compression of the brachial plexus and/or subclavian vessels as they traverse from the neck into the extremity through the costoclavicular space. Approximately 90% of thoracic outlet cases present with neurologic symptoms, with 3% to 5% presenting with purely vascular symptoms.[19,35] The costoclavicular space is bound inferiorly by the first rib and posteriorly by the anterior scalene. Superiorly and anteriorly, it is bound by the costoclavicular ligament, subclavius muscle, and clavicle.[29,35] The shoulder girdle can be involved with compression including the pectoralis minor and subscapularis muscles, coracocostal ligament, pervenous phrenic nerve, cervical ribs, exostosis of the clavicle, and muscular or ligamentous fibrous cords.[15,19,37]

Hyperabduction and external rotation at the shoulder is the most common position during the activities that predispose to effort thrombosis. This position causes the tendon of the pectoralis minor and the head of the humerus to compress the axillary vein with abduction of the shoulder.[1,27,45,47] Meanwhile, compression of the subclavian vein occurs at the intersection of the clavicle and the first rib.[20] This compression can occur in anatomically normal individuals who perform shoulder activities at extremes of motion and power (professional pitching), or it can occur in anatomically abnormal patients within normal physiologic ranges.[1] It can also occur in patients with an anatomic predisposition to compression that engages in activity at physiologic extremes. Pitchers may be at increased risk because of the well-known phenomenon of increased shoulder external rotation and decreased internal rotation.[28,40] Vogel et al described effort thrombosis in a 19-year-old swimmer whose symptoms began after gaining 15 pounds of muscle in an off-season and then returned to a 10-km daily swimming workout.

Patients with effort thrombosis will often complain of arm swelling, heaviness, early fatigue, or aching. Early fatigue was documented in 75% of the cases in Campbell's 1977 series.[13] Typical physical exam findings include venous engorgement, dilated superficial veins, upper extremity swelling of several centimeters, discoloration, palpable axillary cords, and abnormal Adson or Wright maneuvers (Table 3).

Although these symptoms and signs raise suspicion for effort thrombosis, advanced imaging is usually employed to make a definitive diagnosis. Duplex ultrasound of the involved extremity has a sensitivity of 71% to 100%.[9,25,33] Computed tomography angiogram and magnetic resonance angiography can help identify lesions.[43] When thrombosis is suspected, direct contrast venography is considered the gold standard for diagnosis. The timing of intervention is paramount because more established thrombi are less likely to respond to thrombolysis.[34] Cervical radiographs are warranted to rule out cervical ribs as well as an initial thrombophilia screening. Laboratory screening includes clotting tests, antithrombin III, protein C, protein S, antiphospholipid antibodies, anticardiolipin antibodies, serum homocysteine levels, Factor V Leiden, and prothrombin gene mutation.[43]

One unique predisposing factor for hypercoagulability is the occurrence of thrombosis at high altitude.[42] Although this entity has been described in multiple case reports and series, its cause has not been extensively studied.[4-6,18,23,42] Its cause is most likely multifactorial, with high altitude playing a role: hypobaric atmosphere, hypoxia, low humidity, increased solar/UV radiation, and generally lower temperature. Basic hematological laboratory workup in these patients may yield normal findings; hypercoagulability may be due to adaptive hyperfibrinogenemia.[42] Thrombotic complications of sickle cell disease due to professional football at altitude (same city) have

[||]References 3, 8, 16, 19, 22, 31, 33, 35, 44, 47.
[¶]References 3, 12, 19, 26, 31, 33, 35, 40, 44, 47, 49.

Table 2. Reported cases of effort thrombosis in athletic populations.

Author	Age[a]	Sport, n	Comments
Adams[3]	19	Professional baseball	
DiFelice[19]	22[b]	Minor league baseball, 2 Collegiate baseball, 2	Pitchers, 3 Catcher, 1
Vogel[47]	19	Collegiate swimmer	Daily swimming, 10 000 m
Medler[31]	15	High school wrestler	
Snead[44]	18	Collegiate football	Offensive lineman
Melby[33]	20[b]	Baseball, 14 Basketball, 5 Football, 5 Swimming, 3 Fencing, 2 Soccer, tennis, volleyball, 1 each	Men, 29; women, 3; high school, 31%; college, 47%; professional, 22%
Nemmers[35]		Softball	
Zigun[49]		Martial arts	
Hughes[22]		Billiards	
Butsch[12]		Hockey	
Kolodinsky[26]		Backpacking	

[a]If reported.
[b]Average age of patients in series.

Table 3. Common symptoms and signs of effort thrombosis.

I. Symptoms: affected areas include the neck, shoulder, and axillary region
   a. Heaviness or dead arm
   b. Aching
   c. Easy fatigue
   d. Discoloration
   e. Swelling
   f. Tingling
   g. Numbness
II. Signs: affected areas include the neck, shoulder, and axillary region
   a. Edema with or without pitting
   b. Erythema
   c. Mottling or bluish/purple coloring
   d. Dilated superficial veins
   e. Palpable axillary cording
   f. Low-grade fever
   g. Positive Adson maneuver: palpation of radial pulse with rotation of the head to the ipsilateral direction with simultaneous inspiration reveals a dramatic decrease in or disappearance of the pulse or the onset of paresthesias.
   h. Positive Wright maneuver: palpation of the radial pulse with hyperabduction and external rotation of the shoulder reveals progressive decrease in or disappearance of the pulse or the onset of paresthesias.

been reported (unpublished data, A. Yates and G. Eid, 2009). Altitude played a role in this case and should be considered in athletes participating at high elevation.

From 1997 to 2007, Melby et al[33] collected the largest series of competitive athletes with effort thrombosis. This series involved high school, college, and professional athletes with an age range of 16 to 26 years, with 29 males and 3 females. Eighty-one percent of the thromboses occurred in the dominant right arm. The 32 patients underwent adjunctive surgical intervention and returned to their previous level of competition at a mean interval of 3.5 months after surgery. Forty-six percent of patients required axillary-subclavian reconstruction, whereas 56% underwent paraclavicular thoracic outlet decompression with venolysis alone. Melby estimated that 50% of patients with effort thrombosis will have veins that remain patent with decompression and venolysis alone. He also recommended a temporary arteriovenous fistula in the majority of patients. Because venous reconstruction has well-documented success and the long-term efficacy of stent placement in the subclavian area of young athletes is unknown, venous reconstruction is recommended in most scenarios over angioplasty and/or stent placement.[33,34]

Table 4. Historical accounts of pulmonary embolism complicating upper extremity venous thrombosis.

		Cases, n			
Author	Year	UEVT	PE	ET	Comments
Hughes[22]	1949	320	3	0	
Barnett[8]	1951	300	1	1	Violent swinging motion of the arm to spank a child
Aufses[7]	1954	1	1	0	Unknown cause and no precipitating event
Inahara[24]	1968	9	0	0	
Adams[3]	1971	28	3	1	19-year-old professional baseball player
Clagett[14]	1974	1	1	0	Hypercoagulable patient; occurred following extrectory urography (2 risk factors)
Campbell[13]	1977	25	3	0	Two patients with intimal injury, 1 with hypercoagulability
Weinberg[48]	1978	1	1	0	Intravenous drug user with septic PE
Demeter[17]	1982	17	0	0	
Harley[21]	1984	14	5	1	Undocumented scenario

[a]UEVT, upper extremity venous thrombosis; PE, pulmonary embolus; ET, effort thrombosis.

The most notable aspect of this case is that none of the typical findings of effort thrombosis were present. The athlete's only complaints were fatigue, dizziness, and shortness of breath, which were attributed to his pulmonary embolism. Pulmonary embolism, though rare, has been described in association with upper extremity deep venous thrombosis.[20,35] The incidence is between 0% and 36%, with the majority of cases involving scenarios of trauma or hypercoagulability (Table 4).[3,7,8,14,21,22,48] The reported incidence of pulmonary embolism in these cases must be regarded with the historical methods of diagnosis in mind. Early studies utilized clinical findings and ventilation/perfusion scans; subsequent studies added angiography; and more modern reports employ computed tomography angiogram or magnetic resonance angiography in addition to all the earlier methods—representing a spectrum of increasing ability to diagnose.

In 1949, Hughes reported on 320 cases of upper extremity thrombosis, with 285 cases being reviewed from the literature. With his literature review, he uncovered 3 cases of radiographically unconfirmed, clinically suspected pulmonary embolism—none of which involved effort thrombosis. In his opinion, pulmonary embolism was "extraordinarily rare, if indeed it ever occurs."[22] In a series of 22 elite-level throwing athletes, DiFelice et al found 1 athlete who developed a pulmonary embolism.[19] This individual, however, had a coagulation abnormality and all the athletes had upper extremity symptoms.

In 1971, Adams et al reported on a series of 28 patients with upper extremity thrombi. Three patients (11%) were treated with anticoagulation alone, rather than with anticoagulation and surgery, developed pulmonary embolism.[3] Nineteen patients (68%) experienced recurrent swelling and pain after treatment. One of these patients was a 19-year-old professional baseball player with effort thrombosis. Of note, there was 1 patient who developed gangrene of his hand, which ultimately lead to death.[3] This initial negative experience with the results of conservative treatment of thrombosis led Adams to adopt a more aggressive approach including embolectomy.

The series associated with the largest percentage of pulmonary embolism was published by Harley et al in 1984.[21] In this series, comprising 14 patients with upper extremity thrombosis, he noted 5 pulmonary emboli, leading to the largest estimated incidence of 36%. Only 1 of the 5 patients suffered from effort thrombosis, the events surrounding this occurance were not reported.[21] Based on data from Harley and Adams, the incidence of effort thrombosis with pulmonary embolism is between 20% and 30%.[3,21]

Treatment for effort thrombosis has undergone an impressive evolution over the past 60 years, since its initial description. This evolution has mirrored changes in our knowledge, pharmacology, and ability to intervene in the vascular system. Historically, initial treatments were rest, anticoagulation, and elevation. Upon diagnosis, patients received intravenous heparin and transitioned to long-term oral anticoagulation with warfarin. This treatment yielded success rates that varied from 30% to 100%, with residual symptoms still present in up to 75% of patients.[1-3,22] Conventional therapy has still been used successfully, with the addition of low-molecular-weight heparin

sometimes administered to decrease time of hospitalization and the complications associated with warfarin use.[44] In this case, the treating surgeon decided to use warfarin, and no complications occurred. With the advent of angioplasty and stenting, attempts were made to utilize these methods as the sole treatment for thrombi—thus obviating the need for surgical decompression. These methods were abandoned, however, after significant complications were seen—including early postoperative reocclusion and stent fracture.[1,30,32,36,46]

The best outcomes for effort thrombosis result from a multimodal approach based on initial intravenous thrombolysis, followed by oral and/or low-molecular-weight heparin anticoagulation, with later evaluation for thoracic outlet syndrome and vessel integrity. This evaluation after thrombolysis may be repeat venography, computed tomography angiogram, or magnetic resonance angiography. To evaluate the dynamic effect of arm position, it is important that the patient undergo reimaging in the suspected posture of compression.[1,43] Venography offers the best method for evaluation involving forward flexion, abduction, and external rotation. If findings consistent with thoracic outlet syndrome are identified, surgical decompression aimed at relieving venous compression is appropriate. This may involve first rib resection, scalenectomy, and venolysis.[1,29,39,41] If venous integrity is compromised, as in cases of venous stenosis, adjunctive care may include angioplasty, vein reconstruction, and/or formation of a temporary arteriovenous fistula. The creation of a temporary fistula serves 2 purposes: It decreases the incidence of repeat thrombosis and it acts to enhance detection of early complications.[33] When signs of an anatomic abnormality are not found, anticoagulation alone can prove successful.[1,29,43] If a thrombosis is more than 14 days old, success rates with thrombolysis diminish as irreversible fibrosis develops.[34] In these scenarios, axillary-subclavian reconstruction is necessary.

AbuRahma documented his experience with treatment of effort thrombosis in 2 cohorts: (1) patients who received conventional anticoagulation and (2) patients who received a multimodal treatment strategy.[1,2] For the conventional therapy cohort, only 38% of patients reported symptom relief and only 13% had total venous recannulization. The multimodal cohort, however, had 87% symptom relief and 80% total venous recannulization.[1]

In summary, this case of asymptomatic effort thrombosis was diagnosed after the development of a symptomatic pulmonary embolism. In athletic populations, effort thrombosis is most prevalent in throwing—particularly, pitching. The evolving management of effort thrombosis has led to a better prognosis for this condition. Athletes can make a full recovery with a treatment regimen based on early thrombolysis, evaluation of underlying cause, temporary anticoagulation, and surgical intervention. There are serious consequences to pulmonary embolism. Physicians caring for throwing athletes should carefully evaluate any individuals with shortness of breath and fatigue and include effort thrombosis in the differential diagnosis.

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