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Case Report

A Rare Case of Deep Vein Thrombosis: Paget-Schroetter Syndrome

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Abstract

Paget-Schroetter syndrome is a rare form of deep vein thrombosis, occurring in approximately 0.5-1% of all deep vein thrombosis cases, caused by excessive and repetitive upper extremity activities. Therefore, it is also referred to as axillary-subclavian vein effort thrombosis. This condition typically results from chronic compression of the subclavian vein at the thoracic outlet level.

In our case report, we present a 23-year-old male patient who developed pain and swelling in the right shoulder region and upper extremity following weightlifting. There were no risk factors such as trauma, smoking, prior venous catheterization, malignancy, hematological disorders, or a history of chronic disease in the etiology of the deep vein thrombosis detected in this patient. Computed tomographic angiography showed no thrombus in the arterial structures. However, venous Doppler ultrasonography revealed an acute thrombus in the subclavian vein. The patient showed improvement in symptoms following early anticoagulant therapy.

Keywords

Anticoagulant therapy, Deep venous thrombosis, Paget-Schroetter syndrome.

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Effort thrombosis of the axillary-subclavian vein was first described by Sir James Paget in 1875 and Von Schroetter in 1884, and it was named Paget-Schroetter Syndrome in 1948 [1,2]. This syndrome is rare, accounting for only 0.5-1% of all venous thrombosis cases.[3] The affected patient group typically consists of young and healthy individuals.

The most characteristic symptoms include sudden-onset pain, swelling, edema, and cyanosis in the upper extremity, usually occurring in the dominant arm after exercise [3-5]. Due to the presence of pain and cyanosis, it can sometimes be mistaken for acute arterial embolism.

In this case report, we present the treatment of a patient who was referred to our clinic with a preliminary diagnosis of acute arterial embolism due to cyanosis and arm pain after repetitive exercise.

Case Report

A 23-year-old male construction worker presented to the emergency department with complaints of pain, bruising, and swelling in his right arm, which had persisted for approximately one week after lifting heavy weights. He was initially evaluated for arterial embolism, but computed tomographic angiography revealed no occlusion in the arterial structures, leading to his referral to our department.

On physical examination, the patient's blood pressure was 100/60 mmHg, pulse was 78 bpm, and body temperature was 36.8°C. All arterial pulses in the right upper extremity were strong and palpable, with no signs of ischemia. However, there was an approximately 1.5 cm increase in circumference in the right upper extremity compared to the left, along with cyanosis and tightness. Additionally, he reported tenderness in the right shoulder region (Figure 1).

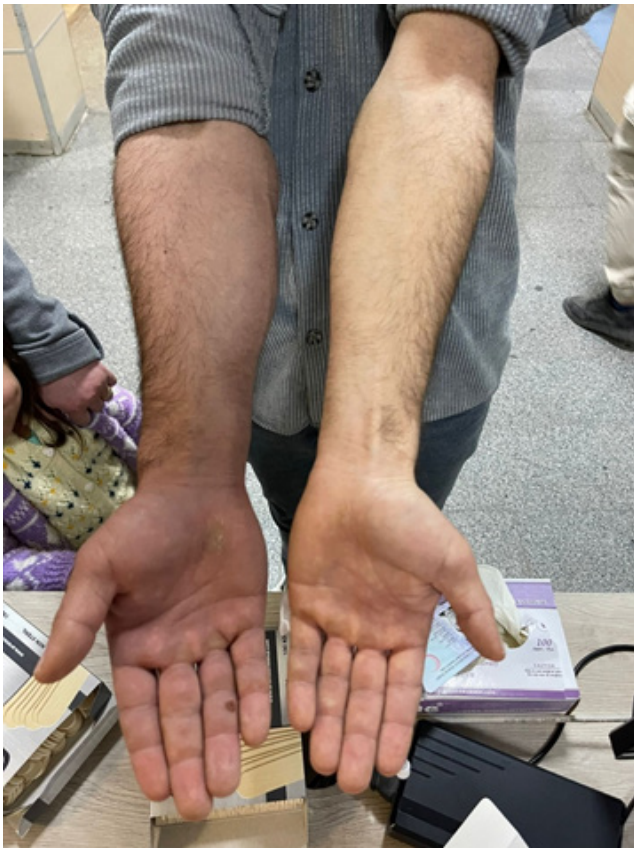

Figure 1: The patient had a significant difference in limb circumference and cyanosis upon their initial presentation to the emergency department.

The patient had no history of trauma, venous catheterization, malignancy, chronic disease, or hematological disorders. His family history was negative for venous thrombosis, pulmonary embolism, or coagulopathy. Venous Doppler ultrasonography of the right upper extremity revealed an acute-to-subacute thrombus in the subclavian vein, obstructing blood flow. The axillary, jugular, cephalic, and basilic veins were patent. A posterior-anterior chest X-ray was normal.

Complete blood count, biochemical parameters, and electrocardiography results were within normal limits. However, the D-dimer level was elevated at 3.35 mg/L. The patient was admitted to the intensive care unit with a diagnosis of right subclavian vein thrombosis. Thrombolytic therapy with tissue plasminogen activator (t-PA; 2 mg/hour), anticoagulant therapy with heparin (1000 U/hour), non-steroidal anti-inflammatory drugs (NSAIDs), and extremity elevation were initiated. After 24 hours, heparin was replaced with low molecular weight heparin (1 mg/kg twice daily).

Further testing for protein C, protein S, homocysteine, and antithrombin III levels revealed normal results. Transthoracic echocardiography showed no signs of right heart enlargement or overload suggestive of pulmonary embolism. During follow-up, the patient's symptoms in the right upper extremity significantly improved. The circumference difference decreased to 0.5 cm, and the tightness and cyanosis regressed (Figure 2). Additionally, the elevated D-dimer level decreased to 0.7 mg/L.

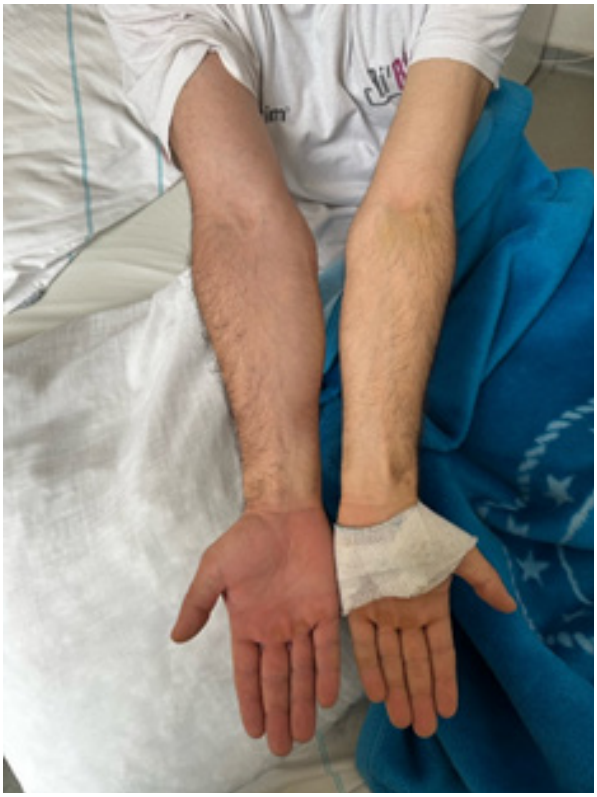

Figure 2: On the seventh day after treatment, the difference in limb circumference significantly decreased.

On the eighth day of hospitalization, the patient was discharged with continued low molecular weight heparin therapy, NSAIDs, arm elevation, and compression therapy.

Discussion

Deep vein thrombosis (DVT) of the upper extremity accounts for approximately 5% of all deep vein thrombosis cases, with Paget-Schroetter syndrome being even rarer [6,7]. Upper extremity DVT, resulting from axillary or subclavian vein thrombosis, can be classified into two groups based on etiology: primary and secondary. While primary upper extremity thrombosis may be associated with Paget-Schroetter syndrome, secondary upper extremity DVT is usually related to central venous catheterization, pacemaker placement, or malignancy.

Paget-Schroetter syndrome is most commonly observed in young individuals aged 15-45 years, with an equal incidence between males and females [8]. This syndrome is more frequently seen in individuals whose occupations or sports activities involve repetitive shoulder movements that provoke hyperabduction, retroversion, and extension of the arm. It is particularly prevalent among athletes involved in swimming, weightlifting, and rowing. Anatomically, the axillary-subclavian vein passes through a tunnel bordered anteriorly by the clavicle, laterally by the scalene muscle, posteriorly by the first rib, and medially by the costoclavicular ligament. Repetitive external mechanical compression in this region, whether congenital or acquired, can lead to venous intimal hypertrophy, chronic inflammation, and perivenular fibrosis, eventually resulting in acute or chronic thrombosis [7]. In most cases, the triggering factor for thrombosis is exercise [9].

The gold standard diagnostic method for Paget-Schroetter syndrome is contrast venography [4]. Other diagnostic tools include venous Doppler ultrasonography (USG), magnetic resonance angiography, and scintigraphy. Venous Doppler USG is the most frequently used diagnostic method due to its non-invasiveness, ease of use, and low cost. In our case, the diagnosis was made based on clinical findings and venous Doppler USG.

The primary goal in the treatment of Paget-Schroetter syndrome is to alleviate symptoms, improve quality of life, and prevent complications and recurrence. Although there is no universally agreed-upon treatment approach, available options include thrombolytic therapy, anticoagulant therapy, percutaneous angioplasty with stenting, and surgical decompression (thoracic outlet decompression). To prevent pulmonary embolism, recurrence, and post-thrombotic syndrome, long-term anticoagulation therapy is necessary.

In accordance with the literature, we initiated early anticoagulant therapy in our patient. Additionally, NSAID treatment, arm elevation, and compression therapy were applied.

In conclusion, Paget-Schroetter syndrome should always be considered in cases of rare upper extremity deep vein thrombosis, particularly in young patients without underlying risk factors. With appropriate treatment, potential complications can be effectively prevented.

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