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Arterial Thromboembolism Secondary to Thoracic Outlet Syndrome

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Abstract: Effective treatment of arterial thoracic outlet syndrome includes early recognition and timely correction, with sufficient decompression of the thoracic outlet. A 56-year-old female suffered recrudescent right upper extremity ischemia due to thromboembolic event, raising suspicion for arterial thoracic outlet syndrome and prompting further imaging studies for confirmation. Decompression of the thoracic outlet by first rib resection and scalenectomy, followed by stenting of the diseased subclavian artery, allowed for resolution of arterial thoracic outlet syndrome.

Keywords: Thoracic Outlet Syndrome, Thromboembolism, Upper Extremity Claudication

1. Case Report

A 56-year-old female was found to have an arterial cause of thoracic outlet syndrome with recrudescent right upper extremity ischemia. The patient initially presented to a primary care physician with a month long history of persistent achiness in the right upper extremity. Employment at a hardwood veneer manufacturer consisted of passing sheets of veneer off the conveyer belt. An outpatient arterial Duplex demonstrated occlusion of the brachial artery at the antecubital fossa, as well as occlusion of the radial and ulnar arteries. The patient was referred to the Emergency Department for Vascular surgical evaluation at which time examination revealed a cool right forearm with poor capillary refill and a non-palpable right radial pulse. A heparin drip was initiated after obtaining a hypercoagulability profile, which ~~was~~ later resulted as negative for clotting disorder. Emergent operative intervention was then performed. An uncomplicated thromboembolectomy of the right brachial artery was completed with return of palpable pulse. The nature of the clot, dark red in color, appeared consistent with acute thrombus. Postoperatively there was resolution of symptoms. A 2-D echocardiogram showed no cardiac etiology of the thromboembolic event. A holter monitor was placed and no arrhythmias were detected. The patient was discharged home on antiplatelet therapy the following day.

Forty-eight hours after discharge the patient returned to the Emergency Department with return of initial symptoms. Arterial Duplex confirmed re-thrombosis of the right brachial artery. Heparin drip was initiated and second thromboembolectomy for recurrent thromboembolism was performed, again with resolution of symptoms. Due to the recurrent episode of thromboembolism, work-up for thoracic outlet syndrome (TOS) was initiated and thoracic surgical evaluation was requested. Magnetic resonance angiography and computed tomography angiography of the neck and right upper extremity were also performed. With the arm in a neutral position by the patient's side there was no evidence of occlusion or stenosis or clot in the right innominate, right subclavian, or right axillary artery. Additionally, there were no findings of aneurysmal dilatation to suggest harbored thrombus. However, duplex ultrasonography with right upper extremity manipulation demonstrated significant compression of the right subclavian artery with diminution of circulatory flow through the brachial artery while the arm was in a hyperabduction, findings consistent with TOS. (Figure 1).

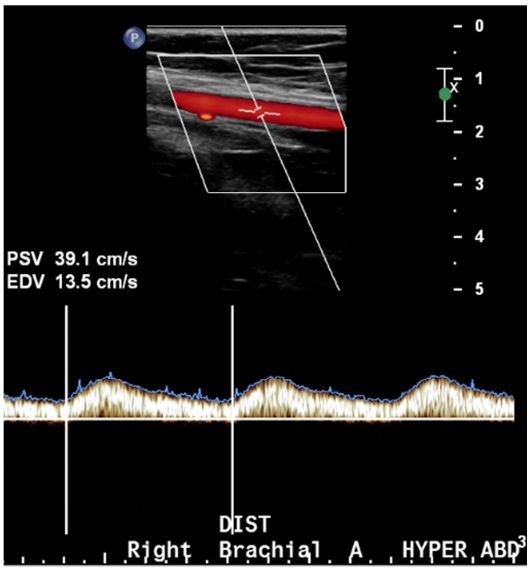

Figure 1. *Low amplitude and diminished flow with hyperabduction of the right upper extremity during duplex ultrasonography.*

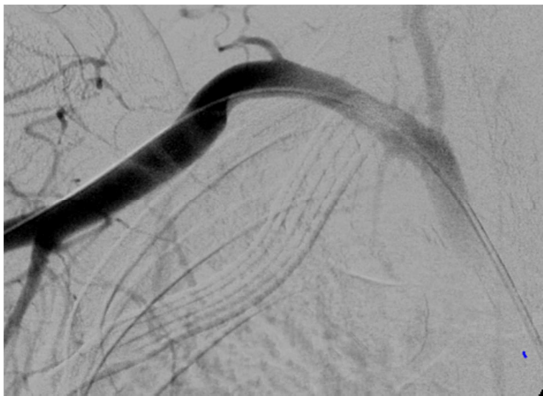

Figure 2. *Arch arteriogram depicts narrowing of the subclavian artery at the level of the resected first rib.*

The thoracic surgery team completed resection of the patient's right first rib along with anterior and middle scalenectomies, and adhesiolysis of fibrous bands. An open infraclavicular approach was used, and exploration revealed hypertrophy of the subclavius and scalene muscles, with neurovascular encroachment of the thoracic outlet. The patient had a palpable distal pulse at the conclusion of the procedure. Immediately post-operatively, while the patient was in the post anesthesia recovery unit, an arterial Duplex was obtained, demonstrating recurrent thrombosis of the right brachial artery. A re-operative thromboembolectomy of the right brachial artery was performed with intraoperative angiogram of the right upper extremity. Completion angiography showed normal flow through the subclavian artery; however, there was a focal luminal narrowing of the subclavian artery at the level of the resected first rib (Figure 2). This stenotic abnormality was accentuated by abduction manipulation of the right upper extremity (Figure 3). A 9mm x 25mm Boston Scientific balloon expandable stent was placed in the right subclavian artery spanning the affected area (Figure 4). A thromboembolectomy of both the radial and ulnar arteries was then performed with injection of Tissue Plasminogen Activator. The patient's postoperative course was uncomplicated, without further thromboembolic events. The patient was discharged home with close follow-up and has had no recurrence of symptoms.

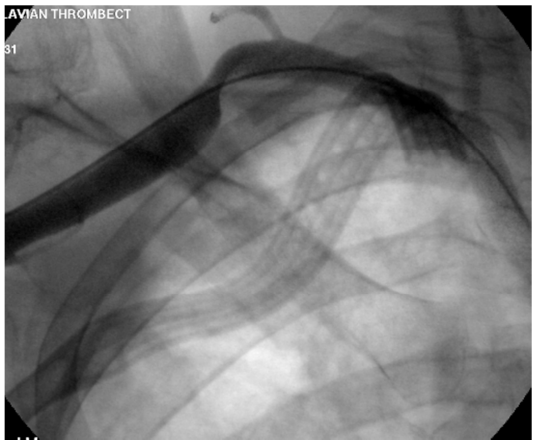

Figure 3. *Angiogram of the same patient during hyperabduction-external rotation maneuver. Significant narrowing of the subclavian artery is present, without occult occlusion or frank extrinsic compression.*

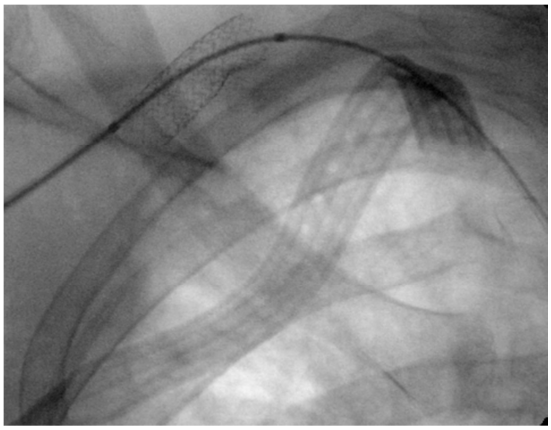

Figure 4. *Balloon expandable stent placement into the right subclavian artery with preservation of thoracodorsal trunk.*

2. Discussion

Arterial thoracic outlet syndrome is the least common of the three defined clinical manifestations of thoracic outlet syndrome (TOS), representing only 1-5% of all cases [1, 2]. The most common classification of thoracic outlet syndrome is neurogenic, followed by venous, accounting for approximately 95% and 4-5 % of cases respectively [3]. The subclavian artery is located in the interscalene triangle and is subject to compression by both the scalene muscles and anomalous fibrous bands, the latter of which is thought to have a more significant effect in increasing potential for neurovascular burden within the thoracic outlet [4]. The interscalene triangle is bordered by the anterior scalene muscle, middle scalene muscle, and the medial surface of the first rib [5]. Repetitive motions narrow the interscalene triangle through muscle hypertrophy and development of fibrous bands, thus compressing the traversing vessels and

nerves. Progressive damage of the arterial wall by repetitive motion or intermittent narrowing of the thoracic outlet can lead to any or all of the following vascular findings: stenosis, fusiform aneurysm with mural thrombosis, local occlusion, or distal embolization [1]. The extensive damage to the endothelium results from turbulent flow and repetitive compression. Without signs of chronic arterial ischemia, it may be difficult to discern symptoms from neurogenic TOS and an arterial pathology may be missed [6]. This patient had a month long history of right upper extremity achiness, initially thought in the outpatient setting to be attributed to an orthopedic cause. The patient did not present with symptoms of an acute thromboembolic phenomenon. In retrospect, this patient's symptoms represented intermittent compression of the thoracic outlet with resultant compromise in arterial flow of the right upper extremity. The palpable pulse after each operation may have represented a transient clearing of the thrombus, and subsequent re-thrombosis was secondary to showering of residual thrombus in the subclavian artery.

Patients often have extensive collateral circulation to compensate for the progressive or intermittent occlusion, leading to claudication symptoms without occult upper limb ischemia. An absence of pulse with provocative compression at the thoracic outlet can be present in asymptomatic patients, and may simply represent a tense thoracic outlet, as opposed to one of chronic arterial compression [6]. Absence of pulse with provocative maneuvers is no longer thought to be a reliable test to make a diagnosis of TOS [7]. Furthermore, if the patient has already developed collaterals due to chronic distal embolization of the arterial and ulnar arteries, an abrupt lack of pulse might be difficult to discern on physical exam. Patients may complain of exertional pain in the affected extremity that worsens with movement and improves with rest [8].

When a patient presents with symptoms suspicious for thoracic outlet syndrome, it is important to exclude other embolic sources, specifically those of cardiac origin including atrial fibrillation, or valvular heart disease [1]. If thoracic outlet compression is suspected, the use of duplex scan or arteriography with inclusion of positional or provocative views can aid in identification of thoracic outlet compression [9]. Changes in peak velocity, vessel diameter, or reproduction of symptoms with provocative maneuvers during duplex ultrasound can be indicative of arterial TOS, as seen in the patient in this study [10], although these variations can sometimes be seen with postural changes in the normal population [11]. MRA and CTA can provide additional information, particularly if they are performed with the arm in both the adducted and abducted positions in order to assist with and optimize surgical planning.

Treatment of arterial thoracic outlet syndrome first involves decompression of the thoracic outlet with resection of the first rib, and cervical rib if present, as well as division of the scalene attachments and any fibrous bands that contribute to the increased neurovascular load within the thoracic outlet. Resection of the first rib is recommended given up to a 30% coexistence with neurogenic TOS [1]. Additionally, the first rib provides attachments for fibrous attachments and failure to resect the first rib may lead to a recurrence [12]. In a study by Aljabri, et. al, 100% of cases of arterial TOS were successful in providing complete relief of vascular symptoms after undergoing thoracic outlet decompression, including resection of cervical and/or first ribs [13]. In this case presentation, duplex ultrasound could have been performed intraoperatively at the conclusion of the patient's thoracic surgery intervention in order to immediately repair any residual vascular deficits. Repair or resection of the artery depends upon any identifiable intrinsic damage as well as the integrity of the vessel. This patient had an identifiable focal luminal narrowing of the subclavian artery at the level of the resected first rib.

3. Conclusion

Effective treatment of arterial thoracic outlet syndrome includes early recognition and timely correction, with sufficient decompression of the thoracic outlet. This patient suffered recurrent thromboembolic events, raising suspicion for arterial thoracic outlet syndrome and prompting further imaging studies for confirmation. Atrial fibrillation and arrhythmias were ruled out after cardiologic work-up. Repetitive motion secondary to employment at a hardwood veneer manufacturer passing sheets of veneer off the conveyer belt may have contributed to the progressive focal narrowing of the subclavian artery. Knowledge of the patient's occupation should have prompted a more inclusive work-up and earlier evaluation of thoracic outlet syndrome. However, decompression of the thoracic outlet by first rib resection and scalenectomy, followed by stenting of the diseased subclavian artery allowed for resolution of the patient's arterial thoracic outlet syndrome.

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