A Case Report: Successful Management of an Aortic Saddle Embolism in a 28-Year-Old Female

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Abstract

The obstruction of blood flow at the aortic bifurcation by an embolus defines aortic saddle embolism (ASE). This rare entity occurs preferentially in individuals with cardiovascular diseases such as the middle aged and the elderly. Conversely, its occurrence is sporadic in younger patients. As a result, the diagnosis of ASE is often overlooked or delayed in this age group; therefore, putting these patients at significant risk of neurologic impairment and potential limb loss. Following an extensive literature review, we have found only one reported case of ASE in a patient younger than 30 years. This patient died within 24 hours of admission and was diagnosed with ASE at autopsy. Here, we report for the first time, a case of a successful management of an ASE in a 28-year-old female who presented at our emergency room with acute weakness and numbness of the lower extremities. After vascular consult, the diagnosis of ASE was made and the patient was treated successfully. A week later, the patient was discharged home in stable condition without complications. The purpose of this report is to raise awareness about this potentially fatal condition and emphasize the importance of rapid assessment and treatment. The treatment options are heparin infusion, thrombolytic therapy, and embolectomy.

Keywords

- ▶ aortic saddle embolism
- ▶ embolectomy
- ▶ thrombolytic
- ▶ adult polycystic kidney disease

Aortic saddle embolism (ASE) is a rare condition that occurs when a blood clot originating from the proximal arterial vasculature stops at the aortic bifurcation and disrupts blood flow to distant organs. In the majority of cases, the clot originates from the left heart, notably from the left atrium.^{1,2} The frequency of the disease is unknown; however, numerous cases have been reported in the literature. Several aspects of this insult make it a unique condition. Its acute nature generates a physiologic stress associated with important hemodynamic changes, which explain the troubling symptoms these patients often have. Furthermore, the sudden arrest of the clot at the level of the aortic bifurcation creates a severe obstruction of blood flow to the pelvis and lower extremities. The resulting ischemia triggers a series of physiologic compensatory mechanisms such as a shift from aerobic to anaerobic cellular metabolism, the release of various cytokines, and the initiation of diverse peripheral nervous

system responses. If the insult persists or intensifies, the protective mechanisms become decompensated. Without swift identification and correction of this abnormal process, irreversible tissue injury and permanent organ dysfunction will occur. Altogether, these events put the patient in great danger of neurologic impairment and potential mortality.³ In general, lower extremity dysfunction is the most common sequelae of ASE, ranging from mild sensory and motor deficits to the loss of the limb.⁴

Here we report for the first time, a case of successful management of ASE in a healthy 28-year-old female patient who presented at our hospital with complaints of bilateral lower extremity weakness and numbness. The patient was successfully treated and discharged home in stable condition on postoperative day seven.

Our main objective in this report is to raise awareness about this potentially fatal condition. ASE must be considered

with acute bilateral neurologic impairment of the lower extremities. In addition, we plan to emphasize the importance of a thorough assessment and expeditious treatment to prevent serious complications including limb loss and potential death. The role of heparin infusion and surgical embolectomy techniques are also discussed. Thrombolytic therapy is the use of fibrinolytic drugs to break up symptomatic clots inside blood vessels. While indicated in the treatment of ASE, the elapsed time from the occurrence of the insult and other systemic factors prevented us from using that option.

Case Presentation

A 28-year-old Hispanic female, college student, presented at our emergency room with the emergency medical services complaining of bilateral lower extremity numbness and weakness that started approximately 30 minutes before. Her ordeal began at school when she collapsed while going up the stairs. The patient described her symptoms as a sudden onset of rapidly progressive numbness and weakness of her legs, associated with a sensation of impending doom. A few minutes later, she became lethargic and confused but did not lose consciousness. Otherwise, she denied additional neurologic symptoms such as dizziness, blurry vision, headache, nausea, or vomiting, and her sphincter function remained intact. Besides the previously described symptoms, the review of systems was negative. Adult Polycystic Kidney Disease (APKD) and aspirin allergy were the only significant findings in the patient's medical history. Her surgical and social histories were noncontributory. She denied tobacco use, as well as alcohol and recreational drug consumption. Also, she was not taking any medications, including birth control pills. Polycystic kidney disease on her maternal side was the only significant finding in her family history. Upon arrival to the hospital, her initial vital signs were within normal limits with blood pressure 116/75, heart rate 77, relative risk 16, temperature 98.8°F, and oxygen saturation at 100%. Her body mass index was around 19 kg/m², and she was noted to be a mesomorphic young woman in mild distress, very cooperative, alert, and oriented to self, space, and time.

On the cardiovascular exam, she had a regular heartbeat and no adventitious sounds. Carotid and upper extremity pulses were normal; the abdominal aorta was not palpable. Focused examination of the lower limbs was negative for any lesions, masses, or deformities. The femoral pulses were unappreciable on palpation, but a monophasic Doppler signal was captured. In addition, progressive coldness was noted from below the knee to the tips of the toes bilaterally. The capillary refill was abnormally prolonged, the distal pulses were absent, and no Doppler signals were registered. Complete loss of sensation and motor functions of the lower extremities were noted on the neurological exam, starting from the hip down to the toes. The patient became confused with a progressively altered mental status. At this point, a cerebrovascular accident (CVA) was entertained as the working diagnosis. The emergency room physician promptly called a stroke code. At the completion of the initial evaluation, the possibility of CVA or acute spinal cord injury was quickly ruled

out after unremarkable computed tomography (CT) scan of the head and magnetic resonance imaging of the spine. Next, vascular surgery was consulted to investigate the bilateral loss of lower extremity pulses. With a high clinical suspicion of an aortic occlusion, the vascular team ordered an immediate CT angiogram of the abdominal aorta with runoff. The scans revealed a large filling defect of the distal abdominal aorta with a large clot overriding the aortic bifurcation extending to both common iliac arteries. There was an absence of opacification distal to the embolus. The findings strongly indicated the diagnosis of an ASE (►Fig. 1). Heparin therapy was started immediately, using the standard protocol for thromboembolism with a bolus of 80 units/kg followed by a maintenance dose of 18 units/kg/h. The diagnosis and the surgical plan were fully discussed with the patient and her relatives. An informed consent was obtained for aortoiliac artery embolectomies under general anesthesia.

Operation

After proper timeouts and induction of anesthesia, two 10-cm vertical femoral incisions were made. The femoral arteries were identified and isolated. Vascular loops and clamps were used for proximal and distal controls. After small incision over the right common femoral artery, a number five Fogarty catheter was used to perform the embolectomy. A linear thrombus measuring about 6 cm in length was removed and flow was reestablished with good Doppler signals. The procedure was repeated on the contralateral side, adequate flow was obtained, however no clots were removed. The vessels were repaired with 6.0 prolene. The patient was extubated and brought to the surgical intensive care unit (SICU) for neurovascular checks and continuation of heparin treatment.

Four hours later, the resident noted a decrease of Doppler signals on exam, which warranted a repeat CT angiography. The results were similar to the initial scan with the saddle embolus present and unchanged. The patient was

Fig. 1 Computed tomography angiography at the level of L3–L4 just above the aortic bifurcation, showing complete occlusion of the aortic artery by the thrombus.

brought back to the operating room for further intervention. After repeat simultaneous bilateral femoral embolectomies, the decision for an open procedure was made after little clot was removed. Through a midline abdominal approach, the aortic bifurcation was isolated with proximal and distal control obtained. A transverse aortotomy was made just above the bifurcation and a large embolus removed in one piece. Strong palpable pulses were returned throughout the lower extremity bilaterally. The patient was returned to the SICU with heparin infusion and neurovascular checks performed as per protocol. The following day, she was extubated and bridged to Coumadin before floor transfer.

Thrombophilia workup including: factor 5 Leiden mutation, homocysteine level, proteins C and S deficiencies, prothrombin gene mutation, and antithrombin 3 deficiency assays performed during her hospital stay were within normal range. Dyslipidemia and other acquired conditions were also ruled out. Although she denied any history of heart disease, a two-dimensional echocardiogram revealed hypokinesia of the left atrioventricular wall. There is no known direct relationship between ASE and APKD but some structural heart abnormalities of the ventricles and the valves have been described as extra renal manifestations of the disease.⁵ A favorable situation exists for the formation of clots in the heart. This case could be the first reported link between ASE and APKD. Otherwise, serial electrocardiograms and cardiac enzymes were unremarkable. The patient was successfully discharged on postoperative day seven ambulating without any difficulty or deficit. A lifetime oral anticoagulant treatment with warfarin was initiated. The patient was appropriately educated on the importance to adhere to the treatment plan.

Discussion

A significant number of ASE-related deaths have been described in the literature. The condition may be directly or indirectly responsible for these fatalities. Primary deaths usually occur as a consequence of cardiovascular collapse or following the systemic inflammatory response caused by the ischemia. ASE may also contribute to death as a result of a rapid decline of preexisting morbidities. Middle-aged smokers with peripheral vascular disease and elderly individuals plagued with advanced cardiovascular disease are the most commonly affected. Less common cases have been reported with trauma and cardiac surgery.^{6,7} Other than a postmortem case involving a 22-year-old woman with atrial myxoma reported by Carter et al in 1959,⁸ this is the only other case report in a young woman.

As it is often the case, a thorough history and physical examination can make the difference in patient care and its importance cannot be overstated. Complications greatly increase when pertinent presenting signs are not appreciated and the diagnosis is delayed or missed. Even in the young patient, ASE must be suspected and investigated in the face of sudden loss of pulses of the lower extremities.

Several clinicians and researchers have dedicated tremendous effort into crafting the best plan of care for patients suffering from this condition. As a result, three major approaches for surgical embolectomy have emerged. A peripheral approach via the groin, an open approach through a midline incision, and thrombolytic therapy via a peripherally inserted catheter are the choices. In this case, the two most commonly used approaches were performed according to the current standard of care. As mentioned earlier, thrombolytic therapy was not an option for this case. Because of the rare occurrence of ASE, no randomized controlled trials are available to dictate the optimal management of this condition. Therefore, a combination of clinical judgment, surgeons' preference, and the limited peer-reviewed data often dictate the approach undertaken. This being said, it is debated among surgeons which of these three techniques is best to use. In this case, similar to other published cases, the abdominal approach seemed to offer the best results. According to current recommendations, all patients with arterial embolic disease should be started on intravenous heparin and discharged with oral anticoagulation, unless there are specific contraindications. Busuttil et al in 1983, found a reembolization rate of 27% after discharge in the majority of patients who were not maintained on Coumadin.^{2,9}

Conclusion

ASE is a potentially fatal disease that can be successfully managed if diagnosed promptly. In reporting this case, we aim to raise awareness about the possibility of this condition in the young and stress the benefit of rapid intervention. The physician facing this challenge must have the appropriate mindset and clinical skills to arrive at the proper diagnosis. Rapid diagnosis and early intervention could make the difference between life and death when a patient is faced with this condition.

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Synopsis

A young woman presented with paraplegia of her lower extremities. She was diagnosed with ASE and was successfully treated.

Financial Disclosure

None.

References

1. Yadav S, Alvarez JM. Catastrophic presentation of atrial myxoma with total occlusion of abdominal aorta. *Interact Cardiovasc Thorac Surg* 2009;9(5):913-915

- 2 Busuttil RW, Keehn G, Milliken J, et al. Aortic saddle embolus. A twenty-year experience. *Ann Surg* 1983;197(6):698–706
- 3 Surowiec SM, Isiklar H, Sreeram S, Weiss VJ, Lumsden AB. Acute occlusion of the abdominal aorta. *Am J Surg* 1998;176(2):193–197
- 4 Jamieson RW, Wallace WA, Din JN, Raza Z. Acute aortic occlusion with sudden paraplegia secondary to *Aspergillus niger* embolism from *Aspergillus niger* aortitis. *J Vasc Surg* 2011;54(5):1472–1474
- 5 Masoumi A, Reed-Gitomer B, Kelleher C, Bekheirnia MR, Schrier RW. Developments in the management of autosomal dominant polycystic kidney disease. *Ther Clin Risk Manag* 2008;4(2):393–407
- 6 Ravdin IS, Wood FC. The successful removal of a saddle embolus of the aorta, 11 days after acute coronary occlusion. *Ann Surg* 1941;114(5):834–839
- 7 Dossa CD, Shepard AD, Reddy DJ, et al. Acute aortic occlusion. A 40-year experience. *Arch Surg* 1994;129(6):603–607, discussion 607–608
- 8 Carter AB, Lowe KG, Hill IG. Cardiac myxomata and aortic saddle embolism. *Br Heart J* 1960;22:502–504
- 9 Ross SA, Hood JM, Barros D'Sa AA. Immediate heparinisation and surgery in the management of saddle embolism. *Eur J Vasc Surg* 1990;4(2):191–194