Sudden-Onset Paraplegia and Ischemia of the Lower Extremities from Acute Aortic Occlusion following Type A Acute Dissection

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Abstract

Herein, we report the case of a 51-year-old man who presented with sudden-onset paraplegia and ischemia of lower extremities, with low back pain, CT scanning revealed Type A acute dissection with occlusion of the infrarenal abdominal aorta and bilateral iliac arteries. A lumbar catheter was inserted for cerebrospinal fluid drainage and we repaired Type A aortic dissection with ascending aorta and total arch replacement combined with stent-graft elephant trunk technique by using innominate cannulation and an aorto-femoral bypass. On postoperative day three, ischaemic symptoms including paraplegia totally disappeared and he was discharged from the hospital on postoperative day 44.

Introduction

Acute aortic occlusion is a rare event in patients with known history of peripheral vascular disease, and is characterized by devastating outcomes, with extremely high morbidity and mortality rates [1]. Its possible causes include in situ thrombosis, aortic saddle embolus, as well as an intimal flap resulting in dissection and thrombosis [2]. The diagnosis can occasionally be mistaken for stroke or a central nervous system lesion [2,3]. Here, we present the unusual case of a patient who presented with sudden-onset paraplegia and ischemia of the lower extremities from acute aortic occlusion following Type A acute dissection.

Case Report

A 56-year-old man was admitted to the emergency department, complaining of sudden onset of low back pain and both lower limbs, which were accompanied by lower limb cyanosis and weakness bilaterally, approximately 4 hours before admittance. Physical examination revealed sensory loss with severe cyanosis of bilateral lower limbs with no pulse of the femoral artery or its distal arteries. The sensory deficit rapidly progressed to full paraplegia. His renal function was normal with myoglobinuria. Computed tomography scanning revealed Type A acute dissection with occlusion of the infrarenal abdominal aorta and bilateral iliac (including common, external, and internal iliac) arteries with thrombus formation (Figure 1). The patient underwent emergent surgical treatment. A lumbar catheter was inserted for cerebrospinal fluid drainage, with a cerebrospinal fluid catheter inserted for cerebrospinal fluid drainage and we repaired Type A aortic dissection with ascending aorta and total arch replacement combined with stent-graft elephant trunk technique by using innominate cannulation. The procedure was carried out by a median sternotomy and total cardiopulmonary bypass with

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the basis of aortic dissection is considered rare [4], the clinical and imaging findings of our case further point out that once the condition is suspected, time is a critical factor and radiologists, neurologists as well as internal specialists should be vigilant for this emergency.

Neurological disorders after aortic dissection are not uncommon, and paraplegia is one manifestation, due to either a spontaneous or iatrogenic compromise of spinal blood supply [5]. If symptoms are not rapidly regressive, cerebrospinal fluid drainage is certainly indicated and may have beneficial effects [5]. There have been some case reports of successful reversal of delayed-onset paraplegia using cerebrospinal fluid drainage after surgical treatment of Type A acute dissection [6]; however, there are few reports of recovery from short time-onset paraplegia. In this report, we described a case of immediate postoperative spinal cord damage that was reversed by cerebrospinal fluid drainage on postoperative day three. In our opinion, the value of cerebrospinal fluid drainage in the resolution of paraplegia is directly related to its implementation before irreversible ischemic damage occurs. The prompt cerebrospinal fluid drainage decreased the cerebrospinal fluid pressure, and the increased spinal perfusion through the collateral circulation prevented irreversible ischemic changes of the spinal cord.

Clinically, ischemia of the lower extremities is most commonly recognized when bilateral or unilateral femoral pulses are lost. Bossone reported that one third of patients with Type A dissection had pulse deficits and that 29% of those patients had limb ischemia [7]. In a
patient with aortic dissection and lower extremity ischemia, one option is to repair the aorta to re-establish flow within the true lumen and restore flow to the lower extremities. However, if this technique is unavailable or unsuccessful, open procedures should be performed. In our patient, after repair of Type A aortic dissection the rapid restoration of flow into the true lumen had improved perfusion of the spinal cord and diminished the right lower-limb ischemia. However, the patient’s left lower-extremity perfusion was not improved, so we performed an aorto-femoral bypass. Although the CT scan before surgery showed embolism of abdominal artery (below the level of renal arteries) and iliac arteries. However, during the surgery we did not find the embolism in the femoral artery. The most likely reason was that the distal blood could not be supplied because of the false lumen instead of thrombosis, so the patient did not receive anticoagulant therapy after the surgery and no residual thromboemboli were identified by the postoperative CT scan.

Paraplegia is caused by occlusion of spinal arteries and occlusion of both renal arteries due to dissection or thrombosis can cause ARF [8]. Aortic occlusion can be complicated by rhabdomyolysis, as in this case, although the renal arteries were patent, contributing to ARF. Haemodialysis and continuous venovenous haemodiafiltration (CVVHDF) are well-established treatment methods in patients with acute kidney injury. It is an efficient method in removing solutes over the course of 24 to 48 hours, just as conventional haemodialysis [9]. The CVVHDF eliminates pro-inflammatory substances, free radicals, endotoxins and cytokines, and this seems to be particularly beneficial in septic patients [10]. Our patient received CVVHDF and his renal function was rescued from dialysis 1 month later. This demonstrated that CVVHDF was important for the patient to depress the risk of further renal insufficiency.

In conclusion, we report a case of acute aortic occlusion following Type A acute dissection that presented as sudden-onset paraplegia and ischemia of the lower extremities. The patient fully recovered after emergent surgery and had no neurologic deficits. Although acute aortic occlusion following Type A acute dissection is rare, it should be considered in the differential diagnosis of patients who present emergently with acute-onset paraplegia and pulseless femoral arteries bilaterally. Prompt diagnosis and intervention can prevent morbidity and death.

References