Case Report

Double Incontinence as a First Symptom of Saddle Embolism of the Aorta Leads to Sudden Paraplegia

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Abstract

An aortic saddle embolus causing cauda equine syndrome followed by paraplegia is an exceedingly rare phenomenon in post-operative period in coronary artery bypass grafting. In non-CABG cases, reported documentation of neurological recovery from this event is even rarer. A 57-year-old male 8 days after uneventful OPCAP presented with severe lower extremity pain and sudden fecal and urinary incontinence, followed by the absence of pulsations in the lower limbs and paraplegia, during 20-minute period. He underwent immediate bilateral transfemoral embolectomy. The postoperative period was uneventful. The paraplegia recovered immediately after embolectomy and recovery from anesthesia. An angiography has been made to verify that a high origin of the great radicular artery above T12 level may be responsible for better recovery of paraplegia when its ostium obstructed by a saddle embolus relieved using embolectomy. Early surgical intervention in restoring the blood flow into the great radicular artery may prevent severe histological changes hitherto responsible for non-recovery from paraplegia in the earlier reports. Three unique characteristics of this article are as follows: 1) Occurrence of this complication in the post-operative period in off-pump CABG surgery; 2) Commencing of emboli with bizarre symptoms of double incontinence; 3) Combination of cauda equine syndrome and complete paralysis.

Keywords: Aortic, cauda equine syndrome, coronary artery bypass grafting, paraplegia, saddle embolus

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Introduction

Post OPCAB spinal cord ischemia remains an exceedingly rare clinical scenario. The main arterial blood supply of the distal spinal cord is from the Adamkiewicz artery (AKA), although in some cases, alternative collateral circulation to distal spinal cord arising from the internal iliac artery. The cauda equine syndrome is known to get most of its blood supply from the lateral and medial sacral arteries originating from the internal iliac artery. This arterial supply mode explains the philosophy of the rarity of paraplegia following saddle emboli.[1] AKA commonly arises between T8 – T12 from vertebral artery and raised, additional arterial blood supply of spinal cord arising segmentally from spinal artery. Saddle emboli lodging at distal aorta may not occlude, conventional higher origin level of the AKA from the aorta. Three important emerging concepts that have an important role in recovery of plegia following saddle emboli article are as follows: 1) Low origin of the AKA artery from vertebral artery; 2) Total ischemic time starting from lodging of emboli and ending to embolectomy procedure; and 3) Potential reversibility of spinal cord damage. However, spinal angiography is a gold standard diagnostic modality and ideally could be performed during preoperative periods;[2] but the emergency nature of the condition did not warrant such time consuming procedures.

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Case Report

A 45-year-old man after an uneventful OPCAB had a sudden episode of severe pelvic pain with radiation to the both extremity in 8th post-operative day of surgery. Ten minutes later, he was affected by paraplegia. His neurological exam was normal, blood pressure was 120/70 and heart rate was 100 and regular. Both lower extremities were pale, cold and calm. His lower extremity sensory and motor function were completely absent to the level of the iliac crest. His femoral and distal pulses were not palpable. Electrocardiography showed normal sinus rhythm. An immediate transthoracic echocardiogram (TTE) revealed severe left ventricular dysfunction (EF, 25%), normal right ventricular pressure, normal right ventricular, and inter-ventricular septum function with absent left ventricular or left atrial clot. While the patient was prepared for operation, emergency angiography was performed via left brachial artery. Abdominal angiography of aorta showed a saddle embolism of the aorta. Aortic dissection, aneurysm or atherosclerosis was not observed. He was received intramuscular petedine and via a large venous line infusion of ringer lactate, along with a bolus of 10000 units of heparin sodium that was started and followed by a drip of 1500 units/h. He was scheduled for embolectomy via bilateral femoral arteries. In operation room, the common, superficial and deep femoral arteries were exposed through a bilateral vertical groin incision (Figures 2 and 3). Scanty back flow bleeding from both pulses less femoral artery was observed. The left and right common femoral artery (CFA) had not any obstructive embolic clot or atherosclerotic plaque. The saddle aortic clot was extracted through a transverse arteriotomy of both femoral arteries with insertion of a No. 5 Fogarty balloon catheter. Exploration of others large, medium size and small size lower

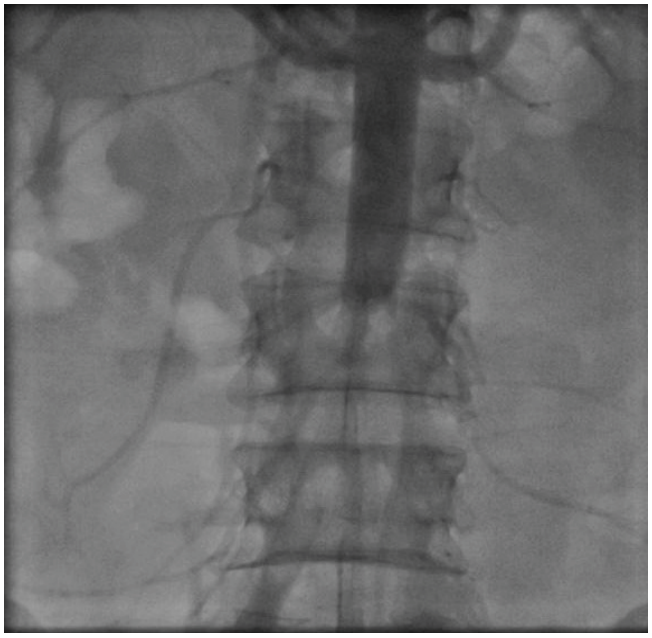

Figure 1. Depicts occlusion of distal aorta.

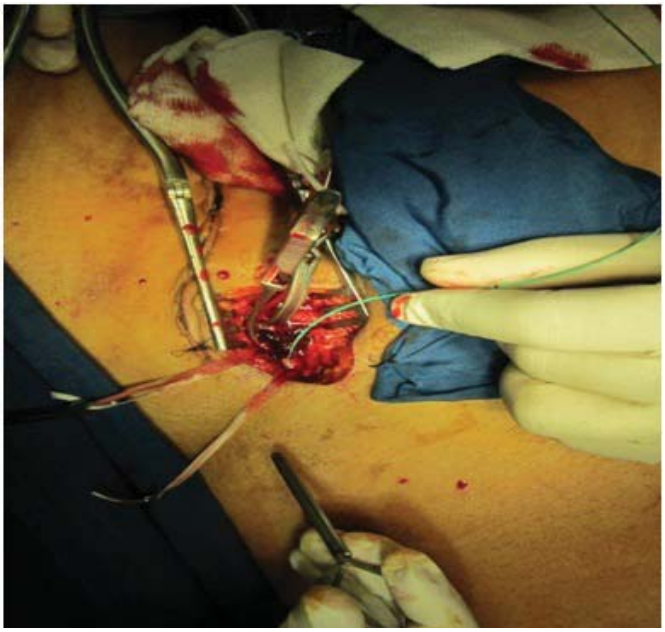

Figure 2. Embolectomy of distal aorta with femoral artery.

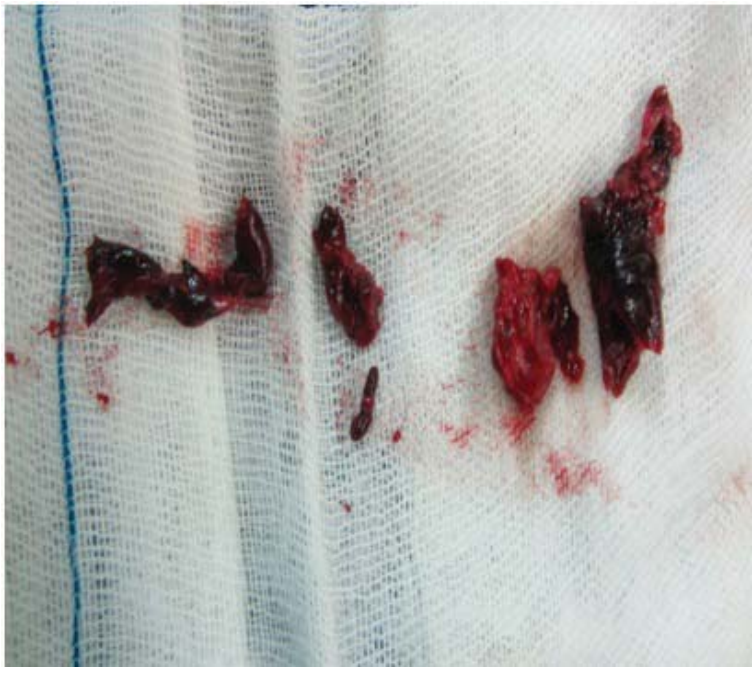

Figure 3. Emboli extracted by Fogarty.

extremity arteries performed accordingly by appropriate size of Fogarty catheter obtained no distal clots and their back bleeding was satisfactory. The time frame from the onset of paraplegia symptoms to reperfusion of the lower extremity by embolectomy was about 5 h, 12 min. He became fully ambulatory within a day and discharged on the 3rd post-operative day.

Discussion

In 85% of patients, the AKA originates on the left side of the aorta from intercostal arteries at segments of T7 – T12. Saddle emboli could not able to occlude origin of these high level arteries to cause paralysis. In 60% of patients, its source comes from a branch of the intercostal or lumbar arteries at the level of the T8 – L4 of the spinal cord. In this state, only long and giant thrombosis of distal aorta that extended upwardly to the level of L4 may able to cause paralysis by occluding fountain of artery as a rare phenomenon.[3] As a very rare anatomic anomaly, AKA artery arises at the level of L3 or L4 – L5 in 1.4% and 0.2% of patients subsequently. A low origin of AKA may be a logical explanation for occurrences of paralysis or cauda equina like symptoms in our patient.[4] Some cases of saddle emboli have been described in the literature among younger patients who had not have advanced atherosclerotic disease or risk factors such as diabetes mellitus, obesity and hypertension.[5–7] Careful literature review revealed no case of saddle emboli with bizarre sign and symptom after off-pump beating coronary bypass surgery. The most important and bizarre symptom in our patient before the occurrence of paralysis

was incontinence that was related to the level of spinal cord injury. Related to the level of spinal cord segment injured by ischemia, there are two distinct patterns in the clinical presentation of bowel or bladder dysfunction: injury above the conus medullaris that results in upper motor neuron bowel or vesicle syndrome and injury at the conus medullaris and cauda equine that results in the lower motor neuron syndrome. This leads to hyper-reflexia of bowel and bladder, which is defined as upper motor neuron syndrome subsequently increased tone of colon and bladder wall and anal sphincter.[8] Spontaneous control of the external anal and bladder sphincter is interrupted and the sphincter remains tight, and retention of stool and urine occurred. With preservation of nerve connections between the spinal cord and the colon and bladder, however coordination and stool propulsion or urine remains intact. The effect of upper motor neuron syndrome on colon is typically associated with colonic content retention due to the hypertonic external anal sphincter activity.[9] Spontaneous stool excretion is not exist in these individuals and evacuation needs reflex activity induced by rectal stimulation by introducing suppositor or finger into the rectum, lower motor neuron syndrome, or a reflexic bowel, is characterized by the loss of spinal cord-mediated peristalsis and slow stool propulsion or urination. In most cases of saddle emboli, the level of spinal cord injury is located between T6 and L1. This is the area commonly fed by AKA.[10] Due to the limited use of echocardiography in our center, routine complications of acute myocardial infarction (MI), severe low ejection fraction or heart failure are not fully assessed; therefore they may go undiagnosed and consequently lead to increased post myocardial infarction complication, as well as the subsequent increase of hospital morbidity and mortality. Thus, it is imperative to bring attention to post MI and left ventricular clot and routinely applied the post MI use of echocardiography in facilitating on time and precise diagnosis in post MI period.

In conclusion, this article has three unique characteristics, including: 1) occurrence of this complication in post-operative period in off-pump CABG surgery; 2) Occurrence of emboli with unusual symptom of double incontinence; 3) Combination of cauda equine syndrome and complete paralysis.

References

1. Griepp RB, Ergin AM, Galla JD, Lansman S, Khan N, Quintana C, et al. Looking for the artery of Adamkiewicz: a quest to minimize paraplegia after operations for aneurysms of the descending thoracic and thoracoabdominal aorta. *J Thorac Cardiovasc Surg*. 1996; 112: 1202 – 1215.
2. Siwach SB, Yadav RK, Singh JP, Dua A. Saddle embolism of aorta. *J Assoc Physicians India*. 1992; 40: 4 – 122.
3. Nagase K, Tamura A, Tatsukawa Y, Kadota J. Aortic saddle embolism caused by left ventricular thrombus in a patient with cardiac sarcoidosis. *Intern Med*. 2007; 46: 145.
4. Jamieson RW, Wallace WA, Din JN, Raza Z. Acute aortic occlusion with sudden paraplegia secondary to Aspergillusniger embolism from Aspergillus nigeraortitis. *J Vasc Surg*. 2011; 54: 4 – 1472.
5. Panetta T, Thompson JE, Talkington CM, Garrett Wilson V, Smith BL. Arterial embolectomy: A 34-year experience with 400 cases. *Surg Clin North Am*. 1986; 66: 339 – 353.
6. Visser CA, Kan G, Meltzer RS, Dunning AJ, Roelandt J. Embolic potential of left ventricular thrombus after myocardial infarction: a two-dimensional echocardiographic study of 119 patients. *J Am Coll Cardiol*. 1985; 5: 1276 – 1280.
7. Sachatello CR, Killen DA. Recurrent saddle and iliac emboli following anterior myocardial infarction. *South Med J*. 1969; 62: 9 – 477.
8. Connolly JE. Prevention of spinal cord complications in aortic surgery. *Am J Surg*. 1998; 176: 92 – 101.
9. Mauney MC, Blackbourne LH, Langenburg SE, Buchanan SA, Kron IL, Tribble CG. Prevention of spinal cord injury after repair of the thoracic and thoracoabdominal aorta. *Ann Thorac Surg*. 1995; 59: 245 – 252.
10. Gharagozloo F, Larson J, Dausmann MJ, Neville RF, Gomes MN. Spinal cord protection during surgical procedures on the descending thoracic and thoracoabdominal aorta: Review of current techniques. *Chest*. 1996; 109: 799 – 809.