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# Acute Paraperisis Due to Acute Aortic Thrombosis in Young Adult- A Mysterious Case

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**Abstract** The occlusion of the Aorta is a rare event, which is potentially life threatening. Acute onset paraplegia is rare event due to sudden aortic thrombosis. We present the case of a 38-year-old man who was referred to the emergency department of our hospital because of sudden onset bilateral lower limb weakness and abdominal pain. He had no history of any disease. He was nonsmoker, nonalcoholic. He had no family history. A computerized tomography angiography of abdominal aorta and bilateral lower limb revealed the thrombosis of the abdominal aorta and of both iliac arteries. After 18 hour of admission he died due to ventricular tachycardia and ventricular fibrillation.

***Keywords:*** *Aortoiliac, paraplegia, young adults, Thrombosis*

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## 1. Introduction

Acute abdominal aortic occlusion is an infrequent, vascular condition that requires emergency surgery. It should be managed promptly due to its fatal outcome. Thrombosis of abdominal aorta is usually insidious onset. Most cases of aortic thrombosis occur in fifth to sixth decades of life but the condition has been reported in patients as young as 29 years. [1] In majority of cases thrombosis is secondary to atherosclerosis or embolism. Less commonly, thrombosis may occur in the sac of an aneurysm and after syphilitic aortitis, [2] pressure by tumors, [3] pelvic peritonitis, and irradiation to the abdomen and/or cardiovascular procedure and trauma.. The following description of a case of aortic thrombosis occurring in a young adult man is of interest because the thrombosis without atheroscrosis and any predisposing condition, is rarely reported.

## 2. Case Report

A 38-year-old male who was admitted to our hospital with sudden onset paralysis of the bilateral lower extremities and abdominal pain. His physical examination revealed that his femoral pulses were both nonpalpable, the bilateral lower extremities were cold and pallor. There was history of pain abdomen radiating to the back. There was no history of bladder and bowel involvement. There was no history of any similar complaint in the past. There was no history of diabetes mellitus, hypertension, tuberculosis, coronary artery disease, trauma, or surgical intervention in the past. The patient was a non-smoker and non alchohlic.

On examination, his pulse rate was 100/min, regular, and equal in both upper limbs. His bilateral lower limb pulses, dorsalis pedis, popliteal and femoral were absent. His respiratory rate was 20/min at the time of examination. His blood pressure was 170/100 mmHg in the right arm in supine position. Systemic examination was not significant.

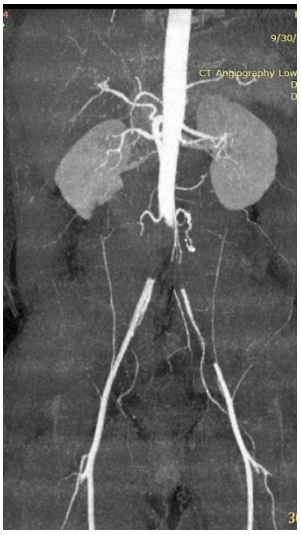

**Figure 1.** Showing complete occlusion of aorta distal to renal arteries

Routine investigations revealed hemoglobin was 11.5gm/dl, total leucocyte counts were 22000 per $mm^3$; platlets counts 3.59 lakh per ml and . His blood urea was 16.86 mg/dl, creatinine 1.08 mg/dl, and serum sodium was 141.2mMol/l, serum potassium was 5.19 mMol/l. Serum cholesterol was 246mg%, random blood glucose was

112mg%. Urine analysis show frequent red blood cells without glucose and albumin.. ECG was normal. CT angiography of abdominal aorta and bilateral lower limb showed thrombus in abdominal aorta with iliac arteries extension. (Figure 1). Figure 2 shows aortic thrombosis with right renal infractionOther investigations including 2-D Echo, protein C and S, were all negative. Homocysteine levels were normal. PT and a PTT were normal.

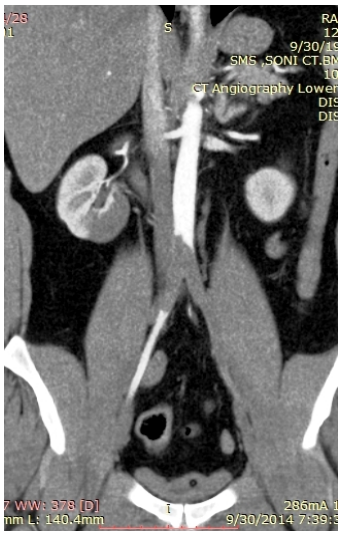

**Figure 2.** Showing aortic thrombosis with right renal infraction

His HIV, HBSagand anti HCV was non-reactive. X ray chest was normal. Spo2 was 98%. His fundi showed no abnormalties. There was no decrease in muscle mass and no evidence of chronic ischemic skin changes.

After 6 hour of admission patient developed difficulty in breathing. Ecg was showing hyperacute T waves. Serum pottasium was done which revealed 9.6 mMol/l. Anti hyperkalemic treatment started immediately. Initial urine output which was 300 ml was not increased. Nephrology consultaion was taken for dialysis. But patient died due to ventricular tachycardia and fibrillation.

## 3. Discussion

Acute abdominal aortic occlusion is a condition that requires emergent surgery. High morbidity and mortality rates occur even when treated in a timely manner. [4] Embolic occlusion and abdominal aortic thrombosis (in situ thrombosis) of the previously diseased aorta are the main pathophysiological mechanisms which underlie this condition. Dossa et al. [4] reported that heart disease and female gender were risk factors for embolization while smoking, diabetes, and a history of intermittent claudication were risk factors for thrombosis. In our case, the patient was male with no risk factors and predisposing conditions.

Although occlusion may occur in any segment of the aorta, the terminal part of the abdominal aorta distal to the renal arteries (infrarenal), especially the iliac bifurcation portion, is the most frequent site of the occlusion. In our patient total thrombotic occlusion was found in the infrarenal abdominal aorta and iliac arteries.

Most emboli originate from the heart, especially from the left atrium, in the setting of atrial fibrillation. Atrial myxoma, bacterial or fungal endocarditis, and prosthetic valve thrombus are infrequent causes of embolization.in our case patient was in sinus rhythm and 2D ECHO was normal. The patient had no other possibilities to account for the embolization. [5,6,7] In situ thrombosis occurs in the setting of severe atherosclerotic occlusive disease in approximately 75% of the cases and is precipitated by conditions such as dehydration, diabetic ketoacidosis, and heart failure that slow the circulation at the vicinity of the stenosis [8].

Different clinical presentations may be observed in patients with involvement of arterial branches of the abdominal aorta. A full-blown case is characterized by the sudden onset of sharp pain located mainly in the lumbar area, abdomen, perineum, and bilateral lower extremities. Later in the course, weakness, numbness, paresthesia, and dysesthesia occur, and in the end, paralysis of the lower limbs may dominate the clinical picture. In the literature, only a few cases have been found that present with the sudden onset of paraplegia or paralysis of the lower extremities without pain due to the occlusion of the Adamkiewicz artery, the major artery supplying blood to the inferior part of the spinal cord. [8,9,10] Our patient was admitted to the hospital complaining of the development of paralysis of the bilateral lower extremities, thus revealing that he might have Adamkiewicz artery involvement. Although the major determinant of the mortality rate has been stated as the time elapsed until revascularization at the previous studies, [8] Dossa et al. [4] reported that the neurological state of the extremities had a more significant effect on the mortality rate than the ischemia time in their study.

Our patient's physical examination revealed cold, pale, cyanotic lower extremities with an absence of femoral and distal pulses. Muscle weakness, absence of deep tendon reflexes, and loss of sensation are some of the manifestations of ischemic neuropathy associated with significant mortality [4].

Although aortography is the gold standard test, the use of Doppler USG, contrast-enhanced CT and magnetic resonance angiography (MRA) can also provide additional information to confirm the diagnosis. [11,12] Since aortography is beneficial for determining the involvement of the renal and mesenteric arteries, it is suggested that patients should have this procedure done preoperatively, especially for those with abdominal pain, hypertension, or anuria. [13] In our case we could not done this test due to early death of the patient.

Since inadequate collateral arterial development occurs, immediate revascularization is necessary for both survival and limb salvage. An embolus can be removed by using balloon-tipped Fogarty catheters through transverse arteriotomies made on common femoral arteries under local anesthesia. In patients with thrombotic occlusion, decisions should be made based on the patient's clinical status and comorbidities. Aortic reconstruction or femoral revascularization via aortofemoral bypass may be undertaken. Several reports [8,9] have advocated aortic reconstruction for all patients with infrarenal aortic occlusion due to the potential risk of propagation of thrombosis at the distal aorta up to the renal and mesenteric arteries; In patients with renal artery involvement, revascularization by reconstruction or an aortofemoral bypass should be performed promptly [13].

In order to maintain sufficient circulation to the distal extremities, relieve the symptoms, and preserve the limbs, we planned urgent surgery, but patient develop acute breathlessness with tacypnea with hypotension and anuria.at the time of admission blood urea, serum creatine and serum electrolytes were normal but after 6 hour of admission serum potassium was 9.6mMol/l blood urea was 54.3mg/dl and serum creatinine was 2.3 mg/dl.Patient died due to refractory ventricular fibrillation.

In conclusion, patients promptly admitted to the hospital because of paralysis of the bilateral lower extremities should be examined there for pulses. If pulselessness or acute aortic occlusion is suspected, additional imaging procedures, including angiography, should be undertaken. In order to prevent the higher rates of morbidity and mortality in cases who develop paralysis in their lower extremities, immediate removal of the obstruction via a percutaneous thrombectomy should be performed using a balloon-tipped Fogarty catheter, and the thrombus material should be retrieved from the aorta. In patients with renal artery involvement, revascularization by reconstruction or aortofemoral bypass should be performed as quickly as possible.

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