ELSEVIER

Case Report

Acute Occlusion of Descending Thoracic Aorta

Cesare Battocchio, Tommaso Dezi, Alessandro D'Andrea, Maurizio Taurino, and Luigi Rizzo, Rome, Italy

Acute aortic occlusion is a rare but potentially devastating clinical event, which requires a prompt diagnosis and emergency treatment. Only 5 cases of native thoracic aorta acute occlusion have so far been reported with different pathologic causes. The clinical features depend on the level of occlusion. Sometimes the diagnosis could be misinterpreted as a stroke or other diseases of the central nervous system. This could lead to a delay in the diagnosis and revascularization procedure, followed by a morbidity or mortality increase. Open surgery has been considered the first-line approach. This study is of a female patient suffering from acute descending thoracic aorta occlusion undergoing, for the first time to our knowledge, endovascular surgical treatment.

Acute aortic occlusion is a rare but potentially devastating clinical event, which requires a prompt diagnosis and emergency treatment.

Despite the recent surgical technique improvements, morbidity and mortality still remain high. Literature data show a mortality range between 30% and 50%.[1–4]

Aortic bifurcation is where acute occlusion most frequently occurs. Suprarenal aorta is an even more rare site of aortic occlusion, with only 8 cases reported in the literature and very high perioperative mortality rate.[5]

Five cases of native thoracic aorta acute occlusion have so far been reported.[6–10]

The clinical features depend on the level of occlusion. Very frequently, an acute ischemia of the lower limbs is present with pain, hypothermia, and a motor or sensory deficit of varying degrees.

Sudden paraplegia is rare and its etiology could be related to acute occlusion of the ostium of radicularis magna or Adamkiewicz artery and supplementary arterial ansa of the conus.[11]

Furthermore, multiple organ ischemic complications could occur, depending on the occlusion site.

Occasionally, the diagnosis could be misinterpreted as a stroke or other diseases of the central nervous system.[12,13]

This could lead to a delay in the diagnosis and revascularization procedure, followed by a morbidity or mortality increase.

The most frequent causes are arterial embolism or thrombosis on a preexisting arterial plaque.

At present, the latter has become comparably more frequent, due to a more effective prevention of cardioembolic events in atrial fibrillation or valve diseases, following anticoagulative therapy.

Furthermore, it has been described as a rare but catastrophic complication as occlusion of abdominal aortic aneurysm.[14]

This study is of a female patient suffering from acute descending thoracic aorta occlusion, 2 months after a mitral valve surgical repair.

Declarations of interest: none.

Vascular and Endovascular Surgery Unit, Sant'Andrea Hospital, Medical and Psychology School, La Sapienza University, Rome, Italy.

Correspondence to: Tommaso Dezi, MD, Vascular and Endovascular Surgery Unit, Sant'Andrea Hospital, Medical and Psychology School, La Sapienza University, Via di Grottarossa, 1035, 1039 Rome, Italy; E-mail: dezitommaso@gmail.com

Ann Vasc Surg 2019; ■: 1.e1–1.e5
https://doi.org/10.1016/j.avsg.2019.03.024

Manuscript received: January 13, 2019; manuscript accepted: March 1, 2019; published online: ■ ■ ■

CASE REPORT

A 64-year-old woman arrived at the first aid of our tertiary referral academic hospital, reporting sudden onset of abdominal and lower limb pain, as well as dyspnea and paraplegia.

Her clinical history was characterized by arterial hypertension, hyperlipidemia, atrial fibrillation under oral anticoagulative therapy, and a recent surgical mitral valvuloplasty in another institution.

At clinical examination, the patient showed plegic marbled lower limbs, hypotension, and absence of femoral pulses.

Laboratory test demonstrated normochromic, normocytic anemia, hypoglycemia, hypermyoglobinemia, and hypercreatininemia. International normalized ratio was not in therapeutic range (Table I). Screening of the thrombophilic pattern was negative for procoagulant alterations.

Arterial blood gas test shows the presence of metabolic acidosis.

She was submitted to an emergency thoracic and abdomen computed tomography (CT) scan with evidence of thrombotic occlusion, superimposed on an atherosclerotic plaque on descending thoracic aorta, like pattern of coral reef aorta, at sixth dorsal vertebra with a late re-entry and perfusion of distal aorta and visceral vessels, although the celiac trunk appeared filiform. No atherosclerotic lesions on iliac and femoral arteries were recorded.

Moreover, bilateral massive pleural effusion with dilatation of pulmonary arteries occurred, mimicking a circulation overload and no signs of pulmonary embolism (Figs. 1 and 2).

At the end of the CT scan, the patient showed severe hypotension and tachycardia. Echocardiogram showed signs of acute right heart failure.

Indication for immediate surgery was given. Surgical access to common femoral arteries was performed. The vessels appeared soft and not pulsating. Five thousand units of heparin were administered intravenously.

An occlusion balloon was inflated, via the left femoral artery, at the origin of common iliac artery to prevent possible peripheral embolism.

An arterial embolectomy by means of 5F and 6F Fogarty catheters (Edwards Lifesciences®, Irvine, CA) was then attempted, via the right femoral artery, without the restoration of a good inflow, probably because of the strong adherence of the thrombus to the arterial plaque.

Subsequently, a standard guidewire 0.035″ (Terumo Medical Corporation®, Somerset, NJ) was passed through the occlusion and an aortic angioplasty was performed by an XXL balloon (Boston Scientific®, Marlborough, MA) with a good result.

Table I. Preoperative laboratory test

Hemoglobin 9.5 g/dL	Myoglobin 1,123 ng/dL
Mean corpuscular volume 88 fl	Creatinine 1.86 mg/dL
MCHC 33 g/dL	Troponin I 34 pg/mL
Platelets 224 × 10^3/μL	INR 2.21
Glycemia 50 mg/dL	D-Dimer 5737 ng/mL

MCHC, mean cell hemoglobin concentration; INR, international normalized ratio.

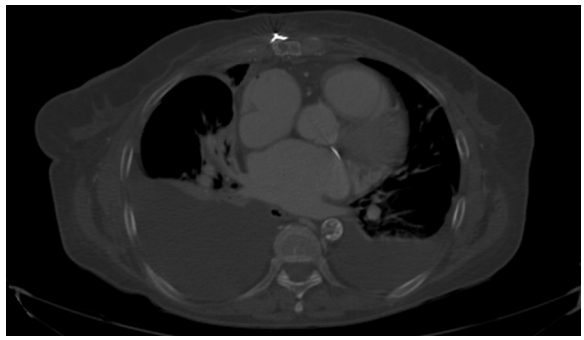

Fig. 1. A computed tomography axial image shows thrombotic occlusion superimposed on an atherosclerotic plaque on descending thoracic aorta.

However, a residual floating thrombus was discovered by transesophageal echo at the level of the aortic lesion. Therefore, a balloon-expandable covered stent, a BeGraft 20 × 48 mm (Bentley Innomed GmbH®, Hechingen, Germany), was deployed via right femoral access.

The completion angiography demonstrated an optimal resolution of the occlusion patency of all visceral vessels and the femoral pulses were restored (Figs. 3 and 4).

The echocardiogram showed an immediate recovery of cardiac performance.

The patient was referred to the intensive care unit in stable hemodynamic conditions.

During the postoperative course, the patient showed an increase in hepatic enzymes bilirubin, lactate dehydrogenase, and serum creatinine, mimicking the onset of an ischemic–reperfusion syndrome.

Forty-eight hours after the intervention the acid–base balance was promptly restored.

Paraplegia was permanent and the neurological status showed a sensitive level at D12 L1.

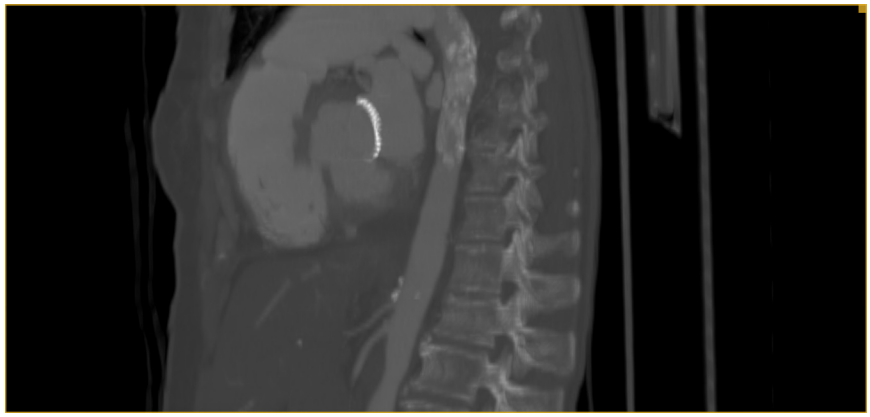

Fig. 2. A computed tomography sagittal image preoperative.

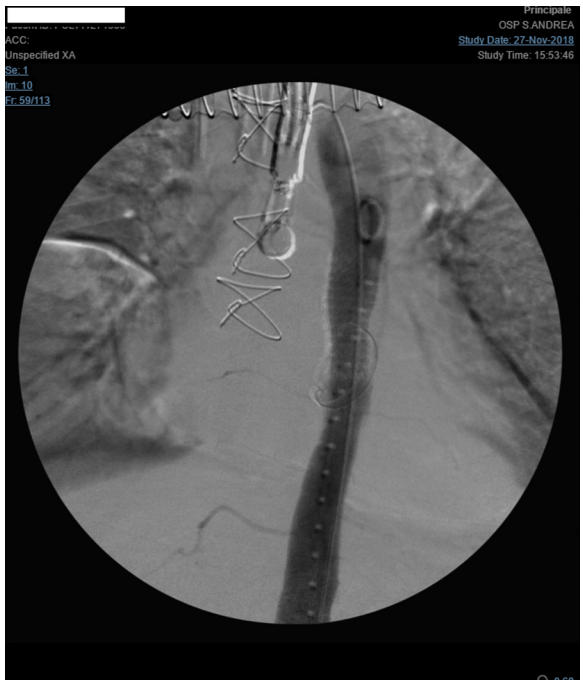

Fig. 3. The control aortography shows the optimal positioning and the endograft patency.

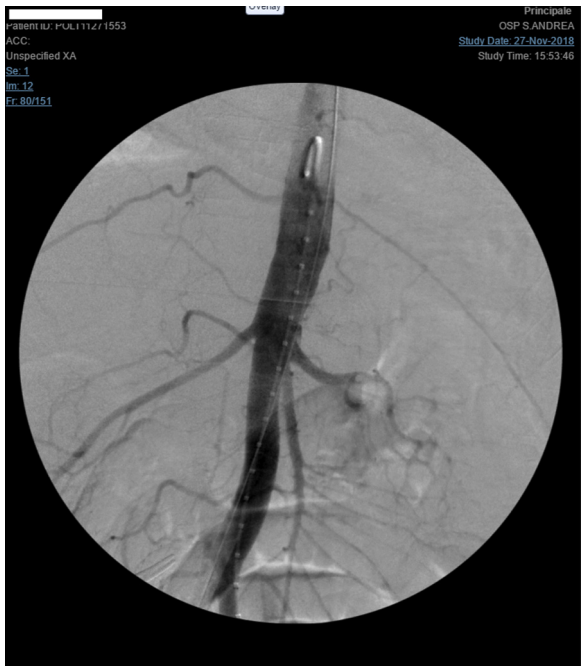

Fig. 4. Abdominal aortography confirmed patency of the visceral arteries.

She was submitted to magnetic resonance encephalic and spinal cord imaging, showing an acute ischemic lesion at D4–D12 (Fig. 5).

On the 15th postoperative day, the CT scan angiography demonstrated good positioning of the aortic endograft and good patency of visceral vessels (Figs. 6 and 7).

On the 25th postoperative day, the patient was discharged and referred to a rehabilitative unit in good general conditions and normal laboratory tests (Table II).

DISCUSSION AND CONCLUSION

Review of the literature showed only 5 cases of acute occlusion of descending thoracic aorta. Two of these died few hours after the diagnosis, and the other 3 cases were operated with open surgical technique. In one case, an axillobifemoral bypass was performed and in 2 cases the entire calcified aortic segment was resected and replaced by a Dacron tube.

Kawahito et al.[9] believe that the direct reconstruction of the aorta with aortobifemoral or an

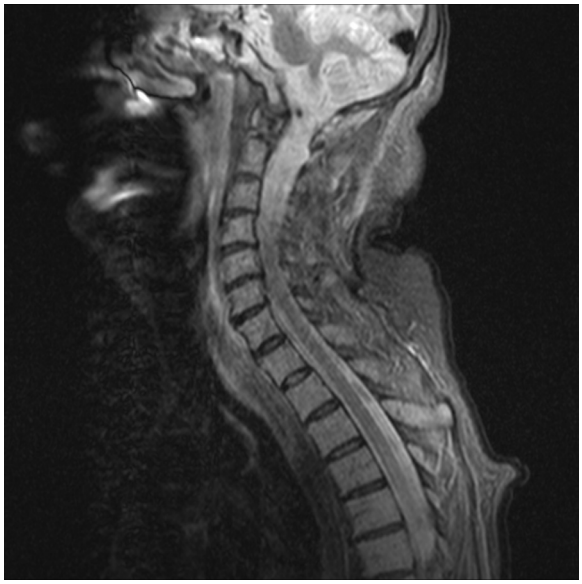

Fig. 5. Magnetic resonance postoperative shows an acute ischemic lesion at D4–D12.

aorto-aortic bypass is the treatment of choice and that extra-anatomical bypass must be reserved in high surgical risk patients.

Tapper et al.[15] believe that extra-anatomic bypass does not work in patients with acute aortic occlusion.

We believe that the first approach to re-establish the patency of the aorta is transfemoral thrombectomy, but, if an adequate reperfusion is not prompt possible, endovascular surgery should be considered, especially in patients unfit for open surgery.

To our knowledge, the present case is the first one reported with acute occlusion of descending thoracic aorta treated by endovascular approach.

Acute aortic occlusion is a rare but critical clinical event. Etiology is mainly due to the superimposition of thrombus on a preexisting atherosclerotic plaque.

Rupture of the fibrous cap is generally responsible of arterial thrombosis.

This process leads to the exposition and release of subendothelial cells and procoagulant factors with subsequent activation of the coagulative cascade and platelet aggregation, determining fresh thrombus on the atherosclerotic plaque.[16]

In the present case, the patient, who had recently been submitted for major cardiac surgery, likely suffered from a thrombosis on a preexisting ''coral reef'' of thoracic aorta and the triggering factor was a low cardiac output syndrome.

Probably that pathology has been underestimated because the preoperative protocol of the cardiac center that performed the operation (another institution) did not provide a tomographic study of the chest.

We believe that in patients undergoing cardiac surgery the entire cardiovascular system should be evaluated and discussed, even in a multidisciplinary meeting.

Paraplegia is a well-known complication in thoracic aortic disease. Sudden onset in acute aortic occlusion is rare and its etiology could be related to acute occlusion of the ostium of radicularis magna or Adamkiewicz artery and supplementary arterial ansa of the conus.

Although cerebrospinal fluid drainage (CSFD) is often utilized as a protective adjunct against spinal cord ischemia, its benefit remains unproven. In addition, other treatments have been described in the literature, such as generalized and local hypothermia and medications such as steroids, naloxone hydrochloride, barbiturates, and papaverine hydrochloride.

In the present case, CSFD could be considered a high risk procedure in an already paraplegic patient undergoing anticoagulant therapy.

Despite the evolution of vascular and endovascular techniques, in the last 20 years, morbidity and mortality are still high.

Most of the data are available after abdominal acute aortic occlusion.

In a recent series of 29 patients, Crawford et al.[4] reported a 30-day mortality rate of 34%, acute renal failure rate of 52%, rhabdomyolysis rate of 34%, permanent neurological deficits in 28%, and a bowel ischemia rate of 14%.

These data are consistent with the most significant experiences reported in the literature.

Better outcomes should be expected with a prompt diagnosis and timely surgical intervention.

The present case shows that, among the various therapeutic options, endovascular technique should be considered a rescue procedure when open surgery is not effective, but hopefully promising even in immediate future.

Further studies are necessary to identify better strategic options for improving the outcomes of patients with acute occlusion of descending thoracic aorta.

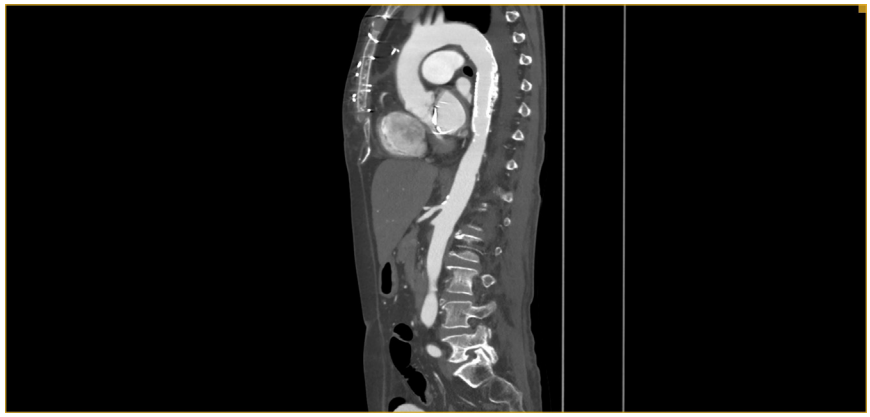

Fig. 6. The CT scan angiography postoperative demonstrated the good positioning of the aortic endograft.

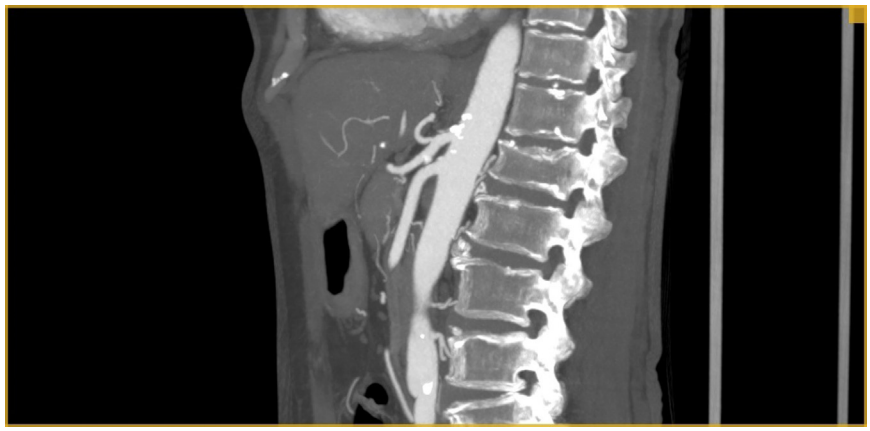

Fig. 7. Good patency of visceral vessels at CT scan postoperative.

Table II. Postoperative laboratory test

Hemoglobin 9.9 g/dL	AST/ALT 44/65 U/L
Mean corpuscular volume 94 fl	Pancreatic amylase 99 U/L
MCHC 31 g/dL	Lipase 129 U/L
Platelets 461 × 10^3/μL	Creatinine 0.5 mg/dL
Glycemia 103 mg/dL	D-Dimer 566 ng/mL

AST, aspartate aminotransferase; ALT, alanine aminotransferase; MCHC, mean cell hemoglobin concentration.

REFERENCES

1. Bell JW. Acute thrombosis of the subrenal abdominal aorta. Arch Surg 1967;95:681–4.
2. Drager SB, Riles TS, Imparato AM. Management of acute aortic occlusion. Am J Surg 1979;138:293–5.
3. Dossa CD, Shepard AD, Reddy DJ, et al. Acute aortic occlusion. A 40-year experience. Arch Surg 1994;129:603–8.
4. Crawford JD, Perrone KH, Wong VW, et al. A modern series of acute aortic occlusion. J Vasc Surg 2014;59:1044–50.
5. Jongkind V, Kievit JK, Wiersema AM. Recovery of renal function after prolonged anuria in acute suprarenal aortic occlusion. Ann Vasc Surg 2016;30:307.e11–4.
6. Axilrod HD. Obstruction of the aortic isthmus by a calcified thrombus. Arch Pathol 1946;41:63–5.
7. Taguschi JT. Obstructing calcified thrombus of the aortic isthmus: a diagnostic roentgenological appearance. Am J Cardiol 1963;12:567–9.
8. Mestres CA, Castella M, Barriuso C, et al. Acute thrombotic occlusion of the suprarenal aorta: importance of early diagnosis and treatment. Br J Surg 1995;82:502–4.
9. Kawahito K, Yamaguchi A, Adachi H, et al. Acute occlusion of the descending thoracic aorta: report of a case. Jpn J Surg 1996;26:652–4.
10. Froelich JJ, Drude L, Klose KJ. Severe calcifying atherosclerosis of the thoracic aorta with symptoms of aortic isthmus stenosis. Case report. Radiologe 1997;37:173–6.
11. Olearchyk AS. Saddle embolism of the aorta with sudden paraplegia. Can J Surg 2004;47:472–3.
12. Webb KH, Jacocks MA. Acute aortic occlusion. Am J Surg 1988;155:405–7.
13. Surowiec SM, Isiklar H, Sreeram S, et al. Acute occlusion of the abdominal aorta. Am J Surg 1998;176:193–7.
14. Haddad F, Yazigi A, El-Rassi I, et al. Acute thrombosis of abdominal aortic aneurysm during cardiac surgery. Ann Thorac Surg 2009;88:1670–1.
15. Tapper SS, Edwards WH, Edwards WH Jr, et al. Suprarenal aortic occlusion. J Vasc Surg 1993;18:372–9.
16. Wolberg AS, Aleman MM, Leiderman K, et al. Procoagulant activity in hemostasis and thrombosis: Virchow's triad revisited. Anesth Analg 2012;114:275–85.