

Aorto-caval fistula resulting from an abdominal aortic aneurysm: A case report from the Emergency Department of Izmir Bozyaka Training and Research Hospital, Turkey

Caglar Alptekin, Hayriye Gonullu, Arife Erdogan, Ismet Parlak

Abstract

The rupture of an abdominal aortic aneurysm into the inferior vena cava with fistula formation is a rare condition but is associated with high mortality. Classical symptoms and findings vary in a wide range, and early diagnosis and intervention can be life-saving. In this study, we present a case of abdominal aortic aneurysm associated with aorto-caval fistula formation accompanied by mortality.

Keywords: Aorto-caval fistula, Abdominal aortic aneurysm, Emergency department.

Introduction

In the United States, the incidence of abdominal aortic aneurysm (AAA) is 2-4% with the elderly men, smokers, and people with a family history of AAA constituting the predisposed group.¹ Aorta-caval fistula (ACF) formation is a rare complication that is caused by the rupture or erosion of AAA into the vena cava. ACF is associated with high mortality if not treated.¹⁻⁴ The treatment options for ACF include conventional open surgery/repair (OR) and endovascular repair (ER), and early diagnosis and treatment are considered to be crucial in reducing mortality.¹⁻⁴ However, the clinical manifestations of ACF may highly vary; therefore, this condition sometimes leads to difficulties in diagnosis.³

In this study, we report a rare case of ACF associated with AAA in a patient that presented to the emergency department with weakness and diarrhoea.

Case Report

A 72-year-old male patient presented to the Emergency Department of Izmir Bozyaka Training and Research Hospital, Turkey, with the complaints of weakness, near-syncope, and diarrhea 3-4 times a day for 2-3 days. The case was seen in December 2016. The patient reported no additional symptoms. According to the routine examination, the vitals of the patient were as follows; arterial blood pressure (ABP) 70/50 mmHg, pulse rate 122

/ min, body temperature 36.6°C, and respiration rate 20 / min. There was no difference between the ABP of the right and left arms. The physical examination of the patient revealed that his general condition was medium, and he was conscious, oriented, and co-operative. The heart sounds were normal except for tachycardia, and no murmur was detected in the examination of the cardiovascular system. Both hemithoraces equally contributed to respiration and no pathological sound was heard in the examination of the respiratory system. Sensitivity, murmurs, and pulsatile masses were not detected in the abdominal examination. All the peripheral pulses were pulsatile. The rectal examination showed liquid stool contamination with normal color. The patient's medical history revealed that he had undergone coronary artery bypass grafting 2 years before. The patient was haemodynamically unstable and therefore electrocardiography (ECG) was performed. ECG showed sinus rhythm at a rate of 120 / min and pathologic Q waves and negative T wave in the leads D2, D3, and AVF. Peripheral vascular access was established and 0.9% NaCl infusion was started. Blood samples were collected for blood typing, cross matching and laboratory tests. Erythrocyte suspension (ES) and fresh frozen plasma (FFP) were prepared. The laboratory parameters were obtained as: Leukocyte: 13000 mm³ (4000-10000), haemoglobin 13 g/dl (13.6-17.2), haematocrit: 40.1% (33-55), urea: 32 mg/dL (17-43), creatinine: 1.5mg/dl (0.67-1.17), troponin I:0.04 ng/mL(0-0.04), D-Dimer 8534 ng/ml (0-240), Ph 7.31 (7.35-7.45), bicarbonate 19 mEq/L (22-26), and lactate 4.7 mmol/L (0.5-1.6). Shock due to ruptured AAA and aortic dissection was considered as a preliminary diagnosis; thus, bedside echocardiography (ECO) and abdominal ultrasonography (USG) were performed for differential diagnosis. The ECO revealed that the left ventricular ejection fraction was 45-50% and the other cardiac structures were normal. The abdominal USG showed an aneurysmal dilatation of 10 cm diameter extending from the infrarenal level to the iliac in the abdominal aorta. A contrasted thoracoabdominal computed tomography (CT) was performed on the patient since the preliminary diagnoses included ruptured AAA and aortic dissection. In the CT, aneurysmal dilatation was observed starting from the level of the renal artery orifices in the abdominal aorta

.....
Department of Emergency Medicine, Izmir Bozyaka Training and Research Hospital, Izmir, Turkey.

Correspondence: Hayriye Gonullu. Email: drhayriyegonullu@gmail.com

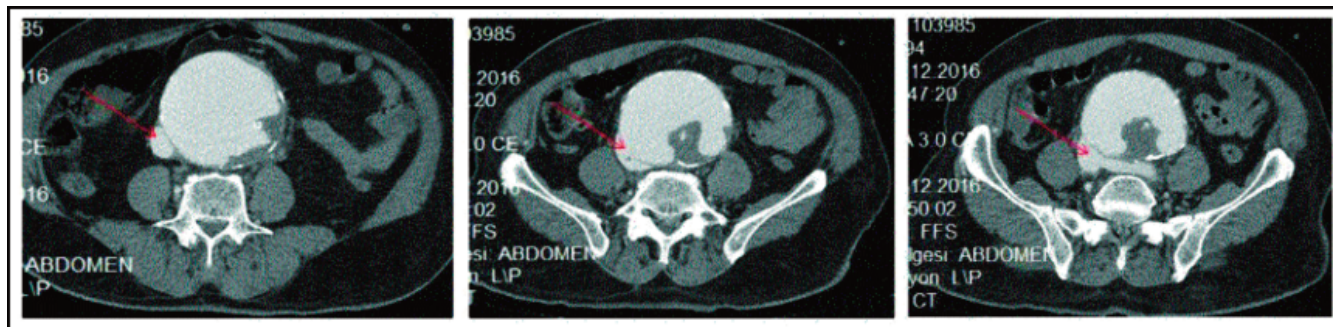


Figure-1: Axial appearance of rupture of aneurysmal fistula into the vena cava.

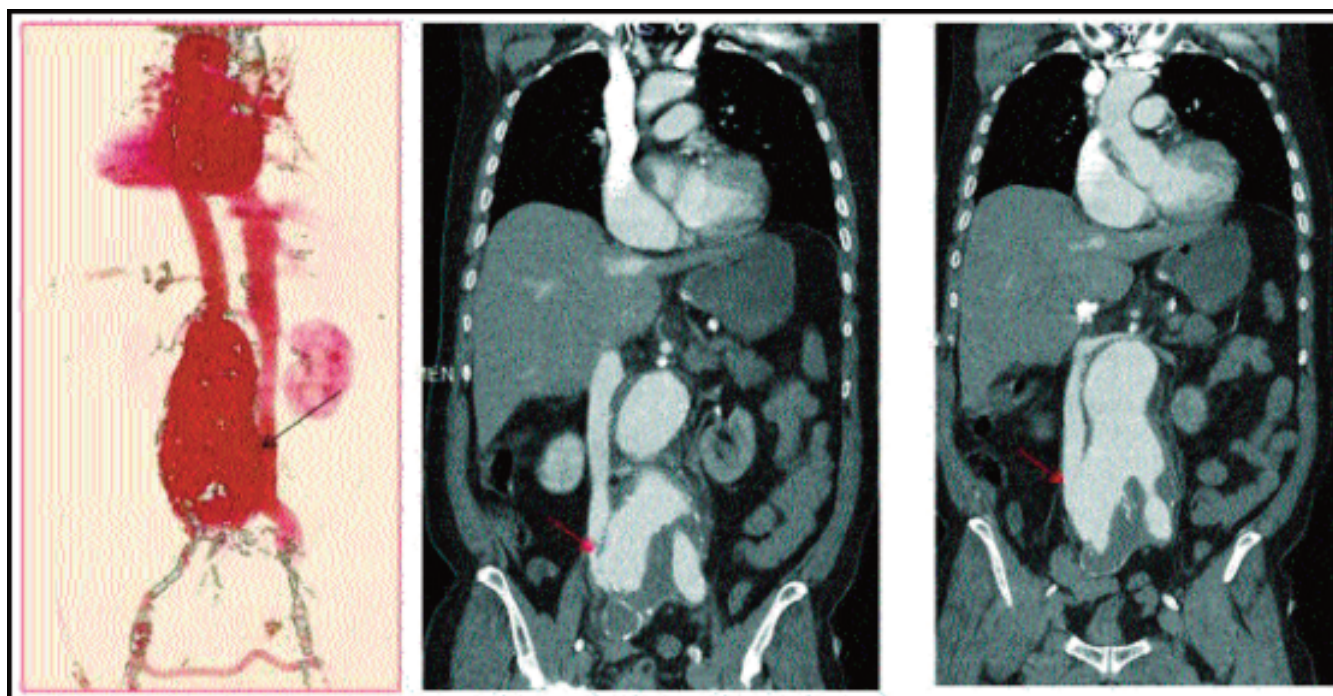


Figure-2: Sagittal appearance of rupture of aneurysmal fistula into the vena cava.

to the iliac artery orifice and covering the right iliac artery as well as the rupture of aneurysmal fistula into the vena cava (Figures-1 to 2). At the 3rd hour of admission, haemoglobin was found to be 9.4 gr / dl, haematocrit was 27.5%, and troponin I was 0.23 ng / mL. No dynamic change was observed in the follow-up serial ECGs. Nasogastric decompression was performed due to the low blood cell count and the digital rectal examination was repeated. There was no evidence of gastrointestinal bleeding. Two units of ES and FFP were transfused and the patient was transported to a suitable center for cardiovascular surgery. The patient underwent open surgical intervention. During the operation, intraabdominal or retroperitoneal haemorrhage was not

observed. AAA was confirmed to be the cause of ACF. The patient died 5 hours postoperatively.

Discussion

The overall prevalence of ACF is very low and it is seen in 2-6% cases with ruptured AAA.^{3,4} According to Wang et al, the most common signs and symptoms were abdominal pain and haemorrhagic shock.³ Similarly, Akwei et al.⁴ reported the classic triad of symptoms as abdominal pain, a palpable mass, and a murmur. In another study, the clinical manifestation of this condition was considered to be in a wide spectrum from intact haemodynamic status to cardiovascular collapse.¹ In other studies, it was suggested that venous pressure increased in ACF cases caused by

ruptured AAA and in these patients, the symptoms of cardiac failure could also be seen due to the increased cardiac overload with hypertension.^{1,3,4} In the current case, the patient was admitted to the emergency service with the complaints of near-syncope and diarrhoea, and was in hypovolaemic shock. Unlike the literature, we did not observe an abdominal palpable mass or abdominal murmur. In addition, there were no symptoms of heart failure or increased ABP in the presence of ACF.

Lau et al.⁵ reported that in ACF cases caused by AAA, the common treatment approach is OR; however, with this treatment, the mortality rate remains to be high at around 67%. In the current literature, ER is considered to be more preferable than OR because it does not cause operational stress and requires a shorter hospital stay.^{4,6} However, in the present case, OR was chosen as the treatment option due to the local availability of the hospital and the haemodynamic status of the patient.

Many studies have demonstrated the positive effects of early diagnosis and treatment of ACF caused by AAA.¹⁻³ More specifically, Akwei et al.⁴ reported that early diagnosis and treatment increased the survival rate from 25% to 50%.⁴ In addition, the authors found that the presence of the poor physiologic reserve, advanced age, and comorbid conditions lead to poor postoperative outcomes.⁴ The current case was fatal despite early diagnosis, resuscitative precautions, and early transport to a specialized center for cardiovascular surgery. This result can be explained by several factors related to the patient's specific situation such as his age, comorbid conditions, the nonspecific complaints that had started several days before, and his delayed admission to the clinic.

An approval was obtained from the patient's family for publication of the case.

Conclusion

Despite the mortal outcome in the postoperative period, our reported case emphasizes the importance of rapid assessment including differential diagnosis and resuscitative approaches in patients with the symptoms of hypovolaemic shock. It should be kept in mind that ACF may be the underlying cause of this clinical picture in patients admitted to the emergency service with the pre-diagnosis of AAA rupture or aortic dissection as well as for those patients for whom rupture or dissection was not detected on CT. Our case also contributes to the literature in this respect.

Disclaimer: None to declare.

Conflict of Interest: None to declare.

Funding Disclosure: None to declare.

References

1. Madsen K, Franz RW. Delayed Endovascular Aneurysm Repair for Aorto-caval Fistula with Correction of Physiologic and Metabolic Abnormalities: A Disease Process Review. *Ann Vasc Surg* 2015; 29: 1456.
2. Kalko Y, U?urlucan M, Ba?aran M, Kafa Ü, Ayd?n Ü, Ya?ar T. Ruptured Abdominal Aortic Aneurysm Associated With Aortocaval Fistula: A Case Report. *Inonu Uni Med Faculty J* 2007; 14: 53-5.
3. Wang T, Huang B , Zhao J , Yang Y, Yuan D. Aortocaval Fistula Resulting From Rupture of Abdominal Aortic Dissecting Aneurysm Treated by Endovascular Repair Delayed: A Case Report. *Medicine (Baltimore)* 2016; 95: e3570.
4. Akwei S, Altaf N, W Tennant, MacSweeney S, Braithwaite B. Emergency endovascular repair. Of aortocaval fistula-a single center experience. *Vasc Endovascular Surg* 2011; 45: 442-6.
5. Lau LL, O'Reilly MJ, Johnston LC, Lee B. Endovascular stent-graft repair of primary aortocaval fistula with an abdominal aortoiliac aneurysm. *J Vasc Surg* 2001; 33: 425-8.
6. Liu M, Wang H. Endovascular stent-graft repair of spontaneous aorto-caval fistula secondary to a ruptured abdominal aortic aneurysm: An emergency management of hostile anatomy. *SAGE Open Med Case Rep* 2016; 4: 2050313X16646526.