Mycotic Aneurysm of the Abdominal Aorta: Early and Late Computed Tomography Findings

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ABSTRACT

We describe the case of a 60 year-old diabetic man that was admitted to our hospital with keto-acidosis and rapidly developed sepsis and a mycotic abdominal aortic aneurysm (AAA) with branch involvement. Early and late findings on serial computed tomography (CT) examinations are presented. Awareness and recognition of early imaging findings associated with infectious aortitis and serial CT examination in cases of patients with sepsis and predisposing factors are essential for prompt diagnosis and treatment.

INTRODUCTION

Infected aortic aneurysms, also known as mycotic aneurysms, are uncommon but life-threatening, accounting for 0.7%-2.6% of all aortic aneurysms.1 They represent abnormal aortic dilatation caused by aortic wall infection and may occur with or without a preexisting aneurysm.2-4 Manifestations of sepsis usually predominate in a patient’s picture and accurate diagnosis may be masked often until aortic rupture. This report emphasizes the role of serial CT evaluation in suspected cases and presents both early and late CT imaging findings.

CASE REPORT

A 60 year-old diabetic man was admitted to our hospital with intermittent fever and malaise for a week. He had a 20-year history of type 2 diabetes mellitus under diet and oral treatment with gliclazide and metformin hydrochloride. On admission, physical examination revealed a body temperature of 38.6 °C, blood pressure of 130/60 mm Hg and regular heart and respiratory rates. Clinical examination was unremarkable. Laboratory tests revealed diabetic keto-acidosis, with hyperglycemia (blood glucose: 340 mg/dL) and abnormal arterial blood gases (PH:7.27, [HCO3-]:8.1 mmol/L, PCO2:17.8 mm Hg, PO2:83 mm Hg, SO2:95%). Abdominal ultrasound examination revealed slightly increased size of both kidneys, which was attributed to the presence of diabetes mellitus and mild right hydronephrosis. The patient was promptly started
on anti-diabetic therapy and antibiotic treatment was initiated with intravenous cefuroxime.

The patient’s clinical status was further complicated over the next few days by fever (38°C) and back pain. Laboratory tests showed persistent hyperglycemia, elevated C-reactive protein (18.5 mg/dL) and leukocytosis (17920/mL) with neutrophilia (89.5%). An initial CT examination of the lumbar spine and abdomen with intravenous administration of iodinated contrast was performed (Toshiba, Aquilion, 16-slice Multi-Detector CT, MDCT). The solid organs of the upper abdomen appeared normal except for mild thickening of the left adrenal gland. The abdominal aorta was of normal caliber, with a thin mural thrombus and wall calcifications and also demonstrated a hazy and slightly thickened wall. The latter was considered to likely represent retroperitoneal fibrosis (Figure 1).

Patient’s sepsis continued for the next six days with development of an abdominal pulsatile mass, a systolic murmur and tarry stools. Laboratory tests revealed a normal hematocrit level, persistent hyperglycemia and leukocytosis, as well as INR 4.45, PT 13%, APTT 92.6 sec, fibrinogen 830 mg/dl, SGOT 1524 IU/L, SGPT 2571 IU/L, LDH 976 IU/L, ALP 206 IU/L, mild elevation of conjugated bilirubin (0.39 mg/dL) and urea (92 mg/dL) and mild hyponatremia (134 mmol/L). Blood cultures were negative. A second abdominal CT examination was performed on an emergency basis (Phillips Single Slice Tomoscan). Both non-enhanced and enhanced CT images were obtained and revealed a fusiform abdominal aortic aneurysm originating cephalad to the celiac artery origin and extending down into the infrarenal aorta, 3 cm cephalad to the aortic bifurcation. Maximum aortic diameter was 5 cm in the infrarenal aorta. The aortic wall was hazy and thickened. Partial thrombosis was apparent in the adjacent inferior vena cava. Small bowel loops were mildly dilated with air-fluid levels and a few intramural air-bubbles (Figure 2a). Several hypoattenuating areas were recognized in the liver, which showed a slightly enhancing rim. The spleen showed no enhancement and appeared diffusely hypointense. The left adrenal gland was enlarged (maximum diameter: 2 cm) with a small central hypoattenuating lesion. The right adrenal gland and kidneys were unremarkable (Figure 2b).

The patient’s clinical status was closely monitored and a new CT examination was performed the next day (Toshiba, Figure 2).

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**FIGURE 1.** Initial CT scan: Transverse contrast-enhanced image at superior mesenteric artery level shows a normal caliber abdominal aorta and thickened aortic wall with calcifications and mural thrombus (arrow).

**FIGURE 2.** Second CT scan: Transverse contrast-enhanced images (a) at level of kidneys hilum show a fusiform aneurysm of the abdominal aorta (black arrow), partial thrombosis of inferior vena cava (grey arrow), mild dilatation of jejunal loops with imperceptible gas in bowel wall (white arrows) (b) at celiac artery level note thickened aortic wall (black arrow), septic infarcts of liver and left adrenal gland, absence of splenic enhancement (white arrows).
Aquilion, 16-slice MDCT), which confirmed the presence of a fusiform mycotic abdominal aortic aneurysm with branch involvement and the aforementioned pathology of the solid organs in the upper abdomen. Marked dilatation of the small bowel and pneumatoysis intestinallis was noted, as well as air in the hepatic portal vein branches (Figures 3a-c). With this information available, the patient was immediately taken to surgery which confirmed all these findings. Unfortunately the patient did not survive the operation.

**DISCUSSION**

The CT appearance of mycotic aneurysms has been extensively described.\(^5\)\(^{-}\)\(^{14}\) To the best of our knowledge, radiographic documentation of the rapid formation of a fusiform abdominal aortic aneurysm (AAA) with branch involvement and accompanying small bowel ischemia and infarcts of the liver, spleen and left adrenal gland has not been previously reported. This case indicates that a mycotic AAA may be life-threatening not only because of rupture, but also because of branch involvement and severe ischemia of bowel and solid abdominal organs.

The term “mycotic aortic aneurysm” was initially used by Sir William Osler in order to describe an aortic aneurysm with pathologic findings of fungal intimal vegetation, in a patient with bacterial endocarditis.\(^2\) To date not only fungi but also other pathogens, commonly Salmonella species, Klebsiella pneumoniae, Streptococcus, Haemophilus, Staphylococcus and Listeria, have been recognized as causative agents in pathologic studies and blood cultures from patients. Nevertheless, blood cultures may be negative in up to 47% of patients and correct diagnosis may be overlooked.\(^15\)

Predisposing factors include bacterial endocarditis, intravenous drug abuse, immunosuppression, alcoholism, diabetes mellitus, arterial catheterization, presence of arterial grafts, vertebral osteomyelitis or an abscess elsewhere.\(^16\)\(^,\)\(^17\) The causative agent may reach the vasa vasorum of the aortic wall through hematogenous seeding, or may implant on damaged intimal layer, arteriosclerotic plaques or mural thrombus. It may also reach the aortic wall either by contiguous spread of adjacent infection or by iatrogenic trauma.\(^1\)

Evolution of a mycotic AAA may occur with or without a pre-existing aneurysm. In both cases, recognition of early imaging findings of infectious aortitis and a high index of clinical suspicion are essential for prompt diagnosis since the clinical profile and laboratory findings are commonly predominated by sepsis. Early surgical treatment and adequate antibiotic therapy are crucial for survival. The overall mortality rate has been reported to be 16-67%.\(^16\)\(^,\)\(^20\)

The clinical picture is characterized by non-specific signs and symptoms, such as sepsis with fever, leukocytosis, septicemia, abdominal and back pain and a palpable abdominal mass.\(^1,\)\(^21\)\(^-\)\(^22\) In the present case, the patient’s clinical picture was complicated by sudden onset of back pain and an abdominal murmur. We assumed that the patient’s infection was followed by septicemia and inoculation of the pathogens in the abdominal aortic wall, probably due to the presence of a thin mural thrombus and atherosclerosis. It is worth mentioning that recently emphasis is put on the presence or absence of the systemic inflammatory response syndrome (SIRS) in patients with mycotic aneurysms, with a significantly higher mortality rate reported for those with SIRS. Patients may be considered to be afflicted by SIRS if two of the following are present: temperature <37º C or >38º C (1), heart rate >90/min (2), respiratory rate >20/min or P\(_{CO_2}^\text{CO_2}<32\) TORR (3), white blood cell count <4.000 ή >12.000 (4).\(^23\)\(^,\)\(^24\) The present case fitted the criteria for SIRS.

In the initial CT scan, the imaging findings of a normal caliber abdominal aorta with thickened and hazy aortic wall may be considered as findings of early infectious aortitis (Fig 1). Identification of CT features of infectious aortitis may be

![FIGURE 3. Third CT scan: Transverse contrast-enhanced images. (a) At level of celiac artery origin, note subtle thickening of celiac artery wall, indicative of branch involvement (arrow) (b) at level between celiac and superior mesenteric arteries, note gas in portal vein branches of the liver (arrow) (c) at level of kidneys notice absence of enhancement and presence of gas in bowel wall (arrows) (c).](image-url)
difficult, as they overlap with retroperitoneal fibrosis, hemorrhage and lymphadenopathy. Several reports mention the presence of subtle periaortic edema, enhancement of periaortic soft tissue, increased periaortic fat density, and paraaortic gas collection as early indicative signs of infectious aortitis, while the aorta remains of normal caliber. Such signs have been deemed to indicate impending rupture, even in the absence of aneurysmal dilatation.

The second and third CT examination performed a few days later, clearly demonstrated the evolution of infectious aortitis to an extensive fusiform AAA, originating cephalad to the origin of the celiac artery and extending infrarenally with a maximum diameter of 5 cm (Fig 2a-b). This is extremely rare, since most previously reported cases of mycotic AAA are described as saccular in shape. Macedo et al, in 2004, retrospectively evaluated CT studies of 27 mycotic aneurysms obtained over a 25-year period and found twenty five (93%) of them to be saccular, and two (7%) that were fusiform. Also, in their study these authors reported rapid development of a mycotic aneurysm in three patients with sequential examinations in as short time as seven days in one case. Nevertheless, at least two of these aneurysms were saccular, whereas the third one was not described with regards to shape and location. Branch involvement has been reported previously to be associated with a mycotic AAA, but in our case it was interesting to notice that both celiac and superior mesenteric arteries were involved, with a slightly thickened aortic wall near their origin (Fig 3a). The development of infarcts of the liver, spleen and left adrenal gland might be due to involvement of the celiac artery or adjacent location to the infected aorta (Fig 2b, 3b). Gonda et al, have previously hypothesized that adjacent location might be responsible for renal infarcts associated with mycotic AAs. The emergence of gas in the bowel wall and in portal vein branches probably resulted from involvement of the superior mesenteric artery (Fig 2b, 3b-c). Finally, the evidence of thrombosis of the inferior vena cava was attributed to adjacent location to the infected aorta (Fig 2a, 3c).

Unfortunately the causative organism was not specified, since blood cultures remained negative, possibly due to antibiotic therapy. However, the diagnosis was established according to the clinical criteria of Oderich et al, taking into account the clinical picture of sepsis and the findings at surgery.

Rapid formation of a fusiform mycotic AAA with associated ischemia of multiple abdominal organs is extremely uncommon. Awareness of early and late imaging findings in serial CT examinations of patients with sepsis and predisposing factors are essential for early diagnosis and therapeutic decision-making.

REFERENCES

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