Infectious aneurysms are almost invariably of bacterial origin. It is a rare life-threatening disease, with a mortality reaching 67% in the absence of an early treatment (1), due to hemorrhage and uncontrolled sepsis.

Clinical and biological findings are not specific and the diagnosis is often delayed. In the presence of fever and sepsis, CT is the imaging method of choice as several features may indicate the infectious origin and distinguish from atherosclerotic aneurysms.

Case report

A 55-year-old woman was admitted in our institution because of fever and abdominal pain.

Upon admission, she presented with tachycardia (110 bpm) and slightly decreased blood pressure (110/70 mmHg). Infection of the lower urinary tract was suspected at first. Blood tests showed an important inflammatory syndrome with increased C-reactive protein (CRP) levels (34 mg/dL, normal: < 1 mg/dL). However, urine analysis did not disclose any abnormal findings. A CT-scan of the whole abdomen was performed (Fig. 1).

CT revealed an important infiltration of the retroperitoneal fat surrounding the distal part of the abdominal aorta as well as the proximal portion of the right iliac artery. The density of the infiltrated fat was evaluated at 20 HU, which was hardly compatible with blood. The infiltration also involved the pelvic retroperitoneal space. After contrast injection (Fig. 2), CT demonstrated a large aneurysm that was located in the proximal part of the right iliac artery and measured 44 x 31 mm in axial view. Based on these clinical and radiological findings, the diagnosis of infected aneurysm of the right iliac artery was rendered. The patient was surgically treated 3 days after admission. She is alive and doing well 2 years after the operation.

Key-word: Aneurysm, mycotic.
Discussion

Infected aneurysms are uncommon disorders. In fact, they represent less than 1% of all aortic aneurysms (2). First described by Sir William Osler in 1895, they were during a long time called “mycotic” aneurysms. They are almost always of bacterial origin. *Staphylococcus aureus* is the most frequent pathogen implicated in this particular disease (MSSA and MRSA), followed by *Salmonella* species and *Streptococcus* species. In contrast, *Salmonella* species are much more common in East Asian countries (2, 3). Notably, fungi are found exceptionally in mycotic aneurysms. The currently accepted term “infected aneurysm” is therefore more appropriate.

Historically, bacterial endocarditis has been the most predisposing condition. However, due to the widespread use of antibiotics, immunosuppression is the main risk factor and is present in 50 to 70% of cases (4): chronic renal failure, diabetes mellitus, corticotherapy or malignancy. Intravenous injection of drugs is another risk.

In most instances, infection produces the aneurysm. Rarely does an infected aneurysm result from the infection of a pre-existing aneurysm. Different mechanisms have been described to explain the entry of the bacteria in the vascular wall, including embolization within the vasa vasorum, direct infection of the intima (owing to a pre-existing intimal defect), infection by contiguity from a neighboring infectious process, and direct inoculation (iatrogenic or traumatic).

Patients are almost always symptomatic, but the diagnosis is often delayed because symptoms are not specific. Pain is the most frequent symptom; observed in 65 to 90% of cases (3-5). Other symptoms include fever of unknown origin (50%), chills, and sepsis. The classic triad of fever, pain and pulsating abdominal mass is very uncommon (3, 4). Blood test often shows elevated CRP and leucocytosis. Blood cultures may be negative in up to 50% of patients. In our particular patient, PB cultures were positive for *Staphylococcus aureus* on two occasions.

In earlier angiographic studies, infected aneurysms did not have specific features that distinguish them from atherosclerotic aneurysms except for sudden appearance, rapid progression or uncommon location (6, 7). Indeed, the infra-renal aorta is a typical localization but combined involvement of other parts of the aorta (thoracic, thoraco-abdominal and supra-renal) account for the majority of cases. In opposition, 85% of atherosclerotic aneurysms are localized in the infra-renal aorta.

At present, multidetector CT angiography is the best imaging method for infected aneurysms, because of its availability and rapidity in emergency cases. In fact, several CT-scan features may indicate an infectious origin in aneurysms: at a very early stage, before aneurysmal formation, the arterial wall becomes irregular. A peri-arterial edema is often present, usually appearing as a hypodensating fat stranding or a concentric rim (2, 8). A mass may develop in the peri-arterial soft tissue, displaying homogeneous or heterogeneous enhancement after contrast injection. Such peri-arterial changes persist after the develop-
ment of the aneurysm and actually represent the most common radiological finding. The shape of mycotic aneurysms is typically saccular, often with lobular contours. The wall of infected aneurysms does not contain atheromatous calcifications in more than 70% of cases, which is in sharp contrast with aneurysms associated with atheromatosis (2, 8). In case of preexisting aortic wall calcifications, a disruption of these calcifications may occur in close proximity to the infected aneurysm. The absence of any identifiable mural thrombus seems to be a reliable sign in favor of an infectious origin, probably due to the rapidity of the aneurysmal process. A less common occurrence is the presence of gas pockets located within the aortic wall or within the surrounding tissues. Although rarely found, this feature is very specific, appearing early before aneurysm formation (9). Vertebral body changes may also be seen but are uncommon. In our patient, many of these characteristics were present (Fig. 1, 2).

Microscopic examination of the resected artery demonstrated a marked destruction of the vascular wall, accompanied by numerous gram-positive cocci, a finding consistent with the presence of Staphylococcus aureus in blood cultures. The tunica media was still present but barely recognizable, due to liquefactive necrosis. Likewise, the internal layer of the adventitia was involved by the necrotic process (Fig. 3).

Infected aneurysm is usually defined as a pseudo-aneurysm, because the artery dilatation does not comprise tree layers like arterial wall, but instead tunica adventitia and adjacent connective tissues. In our patient, the three layers of the vascular wall contained necrotic areas, which resulted in a larger
gical and pathological diagnosis but rupture. Contained rupture is a widely necrotic but still contains the wall of the infected aneurysm is described by the surgeon at the operation. This simply means that contained rupture (1, 5, 10), as usually considered as abscess formation in the retroperitoneum (7). In our patient, the outer part of tunica adventitia was walled off by a fibrous capsule that contained leukocytes mixed with necrotic debris and gram-positive cocci. This is believed to correspond to the peripheral ring enhancement which is more visible at the portal phase (Fig. 4, 5).

The development of infectious aneurysms is usually rapid, within a few weeks or less. As a result, these aneurysms are prone to precocious rupture, which is the initial clinical presentation in 20% of cases with clinically hypovolemic shock and heavy pain in the back area. The aneurysmal free rupture is associated with imaging features: retroperitoneal fat infiltration by hyperattenuating contrast extravasation (10). Moreover, 47 to 75% of aneurysms are in impending or contained rupture (1, 5, 10), as usually described by the surgeon at the operation. This simply means that the wall of the infected aneurysm is widely necrotic but still contains the rupture. Contained rupture is a surgical and pathological diagnosis but not an imaging one: indeed, imaging studies merely illustrate the free rupture of the aneurysm.

To our knowledge, additional investigations that should be performed in case of infected aneurysm are not well defined. However, the origin of bacteriemia should be determined, as far as possible. In this regard, our patient was immunocompetent and did not take any intravenous drug. Transesophageal echography, Tc⁹⁹ white blood cell scan, and oral examination did not reveal any anomaly. Therefore, the origin of bacteriema in our patient still remains unknown. The usefulness of PET-CT in such situation needs to be defined.

Surgical repair is the definitive therapeutic procedure following antibiotic therapy. Perioperative antibiotics usually are administered during 6 weeks. There is no current consensus regarding the benefit of lifelong antibiotics.

The optimal surgical management remains controversial and under debate. Two types of repair are available, including in situ stent graft interposition after extensive debridement, or extranatomatic bypass. The theoretical advantage of extranatomatic bypass is a reconstruction remote from the infection. However, high incidences of complications have been reported in this technique. In situ grafting is therefore considered as a safe option in many patients (5)). Perioperative mortality varies according to series but reaches approximately 20%. In this regard, there is no significant difference between the two types of surgical repair (4). In many studies, in situ repair was preferred, whenever feasible (4, 5, 11, 12). Endovascular aneurysm repair (EVAR) may also represent a less invasive alternative, especially in patients with comorbidity (5).

Our patient was surgically treated with venous in situ homograft, after failure of an extranatomatic graft using a femoro-femoral (left to right) crossover bypass with Goretex.

Conclusion

We reviewed several CT signs that characterize infected aneurysm: a saccular shape, the absence mural calcifications and thrombosis, disruption of calcification wall. We correlated the features observed with histologic findings that appear to be more specific in order to better define them. The peripheral ring enhancement visible at the portal phase corresponds to the fibrous capsule at the outer part of the adventice, the beginning of an abscess formation around the destroyed arterial wall.

These signs explain why it is termed a pseudo-aneurysm, as there remains no functional arterial wall, even if the three layers (intima, media, tunica adventitia) are still present. In fact, every mycotic aneurysm is in “impending rupture” even if no intravenous contrast extravasation is present.

Physicians should understand the seriousness of these features in order to make a correct diagnosis, especially in the early stages of this disease, to ensure prompt therapy.
References