Thoracic Infectious Aortitis

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25.1 Introduction

The infectious attacks of the thoracic aorta remain a rare disease. They are characterized by an endarteritis of infectious origin generally followed by the formation of an aneurysm commonly called mycotic aneurysm. The adjective “mycotic” can be a source of confusion. It could suggest a fungal nature of the aneurysm, whereas bacteria represent the majority of the causes of these infections. This term was introduced by Osler [1] in 1885 to describe an infectious vascular aneurysm taking

the aspect of a “fresh mushroom” in a patient suffering from an infectious endocarditis. It should be understood as indicating any vascular aneurysmal formation of infectious origin.

Before the era of antibiotics, the diagnosis was generally made by autopsy and 86% of the cases were secondary to an infectious endocarditis [2], syphilis excluded. Currently the causes are varied. Each aetiology has particular characteristics with physiopathological, clinical and therapeutic aspects. It is thus currently difficult to regard the infectious attack of the thoracic aorta as a single pathology. It should rather be seen like a collection of different diseases having the same anatomical tropism. Certain causes have a very particular importance, like syphilis, salmonellosis or tuberculosis in addition to the traditional pathogenic bacteria like Staphylococcus aureus or Streptococcus, and will thus be studied separately.

25.2 General Case

25.2.1 Epidemiology

These infections are currently rare. In an autopsic series reporting 22,000 cases realized in Boston between 1902 and 1951, mycotic aneurysms (thoracic and abdominal) accounted for 2.6% of all thoracic aneurysms, themselves being rare (1.5% of the patients) [3]. In another study (20,000 autopsies) of the Mayo Clinic conducted between 1925 and 1954 [4], only six of 178 aortic aneurysms found were of infectious origin. The analysis of four more recent series (between 1946 and 1975) [5–8] found 78 cases of mycotic aortic aneurysms. Lastly, a retrospective study of the Mayo Clinic found between 1976 and 1999 [9] 29 cases of aortic infectious aneurysms, nine of them involving the thoracic aorta and 20 the abdominal aorta. There is a male prevalence in these affections with a sex ratio of 3:1. This must undoubtedly be linked to the significant role that atherosclerosis plays in the genesis of these infections. Athero-
sclerosis is indeed more frequent in men than in women [10]. The average age of occurrence is 65 years old [11–13]. In the particular context of infectious endocarditis, the average age is lower (40 years old) and there is no prevalence of gender [14].

25.2.2 Physiopathology

Four principal mechanisms are found [15]:

1. Secondary aneurysm with the embolism of the vasa vasorum by the germ in question
2. Arterial infection of the intima injured at the time of a bacteremia
3. Traumatism of the arterial wall with direct contamination
4. Infection of the vascular wall by extension of a contiguous infectious site.

The infectious attack on the arterial wall leads to an endarteritis, generally followed by the formation of an aneurysm or a false aneurysm. These aneurysms will generally have a saccular aspect but can also appear fusiform or cupular. The first mechanism of contamination of the arterial wall occurs in particular in the case of infectious endocarditis. The contaminant source is then the valvular vegetation. The germ disseminates in a haematogenous way, embolizes the vasa vasorum and is fixed in the arterial media [14]. An infection appears right inside the arterial wall, which extends in a centrifugal way. The result is major vascular brittleness and the development of an aneurysm with a considerable risk of rupture. It is the same physiopathological mechanism in tuberculosis and especially in syphilis.

The second mechanism involves directly the previously injured arterial wall. The normal intima of the aorta is very resistant to infection, but when it is damaged, infection is likely to develop there. The risk factors of this type of attack are primarily represented by the atherosclerosis with or without an aneurysm, and the intraluminal thrombi. Secondary infections of a pre-existing aneurysm are most commonly found in the abdominal aorta (70%), but 30% of them concern aneurysms of the thoracic aorta (15% for the ascending aorta and 15% for the thoracic descending aorta) [14]. Thus, the intima is the first arterial zone involved in the development of the infection of the interior towards the depth of the vascular wall leading to a thinning of this wall and thus to weakness. The germs in question are classically represented by the germs responsible for infectious endocarditis, the physiopathology of these two infections being very similar. Moreover, salmonellas too are very often found in this type of attack.

In infectious endocarditis, one can see these two mechanisms of aortic attack. More than 70% of mycotic aneurysms found within this pathology concern the proximal part of the thoracic aorta [14]. They are caused by the embolism of the vasa vasorum but can also develop while profiting from an injured zone of the aortic intima and in particular on the supravalvular level where the arterial wall can be deteriorated by the infective flow of blood [16]. These aneurysms are then of small volume and cupular. In parallel, the physiopathology of infectious endocarditis, with an increased susceptibility in the event of preexisting valvular lesions, is very close to the superinfection of atherosclerotic aneurysms and it is easily understood that a germ which was fixed at the level of an injured cardiac valve can also be fixed at an atherosclerotic aneurysm. Lastly, another mechanism of the aortic attack within the framework of this pathology is the attack of the proximal aorta by the extension of the valvular infection.

The acquired lesions of the aorta are thus a factor of risk of superinfection. Certain congenital lesions can also be to blame. Coarctation of the aorta can indeed be the seat of an endarteritis with a mycotic aneurysm developing just above the stenosis [17]. This aneurysm is then of small volume, of saccular aspect and generally develops on the left edge of the aorta [14].

Traumatisms of the thoracic aortic wall leading to an infection are rare. They generally occur in an iatrogenic context (arterial catheterization, surgery) and can in this case involve nosocomial germs. Injuries caused by knives and firearms can also be involved but they remain an exceptional cause of aortitis.

Attacks of the aortic wall by a neighbouring infectious site are more frequent. The causes are mainly thoracic osteomyelitis, pulmonary infections and mediastinitis. The aortic wall is then eroded with an infection developing from outside the artery towards the luminal canal and a major risk of arterial rupture.

Apart from these four large physiopathological mechanisms, two other contexts are to be mentioned. Firstly, cases of prosthetic superinfection of material of the thoracic aorta. These cases are outside the field of infectious aortitis and will not be treated here.

Secondly, real immunological attacks of arteries following an infectious episode are possible, in particular on the level of small arteries. In the thoracic aorta, these postinfectious arteritis remain controversial. However, this type of attack can be found in poststreptococcal acute rheumatoid arthritis. In this pathology, one can indeed in rare cases see an endarteritis of the thoracic aorta resulting in an arterial attack of the media, with oedema and leucocytic infiltrate, also able to affect the adventitia and the vasa vasorum. Aneurysm is, in this context, exceptional. The arterial attack is here of immunological origin with a certain antigenic proximity between the Streptococcus and the arterial media. It is followed by an immune reaction directed against the components of the arterial wall leading to endarteritis [16].