27 Intramural Aortic Hematoma and Aortic Ulcers, Physiopathy
and Natural History

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27.1 Introduction

Intramural aortic hematoma (IAH) and penetrating aortic ulcers (PAU) are part of the so-called acute aortic syndrome (AAS). This new cardiovascular syndrome embraces a heterogeneous group of patients with a similar clinical profile that includes classic aortic dissection, IAH and PAU (Fig. 27.1) [1]. The physiopathological mechanism that precipitates the appearance of each of these entities is different and the natural history of the last two aortic lesions is not well known. Currently, we know that IAH in some patients may evolve into an aortic dissection, that many cases with PAU are accompanied by some degree of intramural hemorrhage, and that occasionally PAU may act as the entrance tear of an aortic dissection (Fig. 27.1) [1–3]. In addition, some patients may exhibit several or all of these lesions. Is, therefore evident, the existence of a link between them. In this chapter the physiopathology and natural history (evolutive patterns) of IAH and PAU are discussed.

Fig. 27.1. The three elements that constitute the acute aortic syndrome (AAS) are depicted. Arrows indicate the possible progression of each of these aortic lesions

27.2 Intramural Aortic Hematoma

27.2.1 Physiopathology

IAH was described by Krukenberg [4] in 1920 as a “dissection without intimal tear.” IAH has been defined as a novel variant of classic aortic dissection characterized by the absence of an entrance tear. It is, therefore, a noncommunicating type of dissection (Fig. 27.2) [5]. Here, the false lumen is created by a hemorrhage into the aortic media, most likely after rhexis of the vasa vasorum that penetrate the outer half of the aortic media from the adventitia and arborize at this level.
27.2.2 Vasa vasorum

In normal circumstances, the intima and the inner part of the aortic media are avascular. It is important to point out that the vasa vasorum are present in the medial layer only when this layer has more than 29 lamellar units and, in such cases, they will only be found in the region of the medial layer that is beyond these 29 units [6]. The region of the medial layer corresponding to the 29 subintimal units is an avascular area and one may presume that the nutrients flow via transintimal diffusion from the aortic lumen. Therefore, we may say that the thoracic aorta has a double, yet precarious, means of nutrient delivery: the adventitia and the outer third of the medial layer depend on the vasa vasorum, while the intima and the inner third of the medial layer are nourished via diffusion [6]. Accordingly, the middle third of the medial layer, where degenerative changes are most frequently seen [7], will nutritionally depend on both sources [7]. Clarke [8] described the aortic wall vasa vasorum being distributed so as to form a deep and superficial plexus. The vessels of the superficial plexus are arterioles 80–100 μm in diameter and they lie at the junction of the media and the adventitia; this superficial plexus leads to a deep plexus of vessels where small tortuous arterioles 10–20 μm in diameter penetrate into the medial layer and arborize in its two outer thirds [8]. Medial vasa vasorum have a larger role in nourishment of the aorta in aortic atherosclerosis, as blood flow through the vasa vasorum in the outer layers of the aortic wall is increased [9]. It appears that increased blood flow in the atherosclerotic aorta cannot be accounted for by dilatation of the existing vasa vasorum and must be produced by proliferation of new vessels in the aortic wall [9]. The morphology and structure of these new vessels differs from that of normal vasa vasorum [10]. Thus, the effectiveness and contribution of these new vasa vasorum to nourishment of the aorta is not well known. Proliferation of these vasa in the atherosclerotic aorta could have unfavorable effects and, in fact, some authors think that these vessel abnormalities may be involved in the pathogenesis of IAH [11].

The development of an IAH may not only be attributed to the spontaneous rupture of “sick” vasa vasorum, it can also be the result of a traumatic rupture of “healthy” vasa vasorum during a traumatism of the aortic wall [12, 13, 14]. A medial hemorrhage secondary to a fracture of an atherosclerotic plaque may also lead to an IAH [3, 5, 15].

27.2.3 PAU Versus Rupture of Vasa Vasorum

Although these aortic lesions (IAH and PAU) are physiopathologically different, in some cases it may be difficult to differentiate between them. Mohr-Kahaly et al. [5] identified 15 patients with IAH by transesophageal echocardiography (TEE) and analyzed the amount of aortic atherosclerosis of these patients. Atherosclerotic lesions were detected in 11 patients (mild in eight, moderate in two, and severe in one); there were no atherosclerotic plaques in the remaining four patients. Accordingly, these authors divided IAH in two physiopathologically different groups: patients with mild aortic atherosclerosis or without aortic atherosclerosis would have had a rupture of the vasa vasorum, whereas in those with severe atherosclerosis a complication of an atherosclerotic plaque was the most likely cause of IAH. This concept is shared by Sheldon et al. [15], who studied 20 patients with IAH identified by TEE; they also had two groups, one with moderate or severe atherosclerosis and another with mild atherosclerosis or without atherosclerosis. Patients from the first group were older and had coronary and peripheral vascular disease more frequently than the others. Sheldon et al.