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Case Report

Penetrating Ulcer of the Ascending Aorta Associated With Intramural Hematoma and Complicated by Dissection: A Case Report

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ARTICLE INFO

Article history:
Received: 21 June, 2023
Accepted: 13 July, 2023
Published: 12 December, 2023

Keywords:
Atheromatous ulcer
intramural hematoma
aortic dissection
ascending aorta

ABSTRACT

Acute aortic syndromes are composed of aortic dissection (AD), intramural hematoma (IMH) and penetrating or atheromatous ulcer of the aorta (PAU). Three entities well individualized on the etiological, pathophysiological and therapeutic level, but whose evolutionary border towards one of the forms is not so clear. We report the case of a patient operated on for PAU associated with IMH complicated with AD.

Introduction

PAU was first described by Shannan in 1934 and subsequently in 1986 Stanton established the currently accepted pathophysiological process [1, 2]. It is one of the acute aortic syndromes with a prevalence between 2.3% and 7.6% [1]. The association of PAU with IMH varies according to the literature from 9 to 14% to 48 to 60% [3-5] and the progression to AD is less than 4% [1]. In this work we report the case of a patient who underwent surgery for PAU associated with IMH with intraoperative discovery of an aortic dissection.

Case Presentation

Mrs. A. K., 65 years old, treated for hypothyroidism with levothyrox, was admitted to the emergency room of the Ibn Sina Hospital in Rabat (Morocco) for chest pain. She reported a history of chronic, intermittent chest pain. Her cardiovascular risk factors were: age, menopause and hypertension under treatment. Clinical examination revealed a conscious patient, well oriented in time and space, eupneic, hemodynamically stable: blood pressure (BP)= 120/65mmHg/ heart rate (HR)=65bpm/ saturation (SpO2) =92% on room air.

Cardiovascular auscultation reveals heart sounds with a regular rhythm, but muffled and without murmurs. Peripheral pulses are perceived symmetrically, and there is an absence of murmur on the path of the major vascular axes as well as the absence of signs of right heart failure. Faced with this clinical picture, an electrocardiogram (ECG), a chest x-ray and a transthoracic echography (TTE) were initially requested, completed by a thoracic angioscan. The rest of the clinical examination was unremarkable.

Thus, it was retained in this patient a PAU of the ascending aorta with aneurysmal dilatation and a him without valvular defect. The management of PAU along with the rest of acute aortic syndrome (AAS) is currently codified [6]. According to U.S. recommendations, there is a class 1 surgical indication for PAU located in the ascending aorta and associated with an IMH.

On this basis the patient was admitted to the operating room. Under general anaesthesia, in dorsal decubitus position, a vertical median sternotomy was performed. Vertical opening of the pericardium, which was free of any symphysis and discovery of a massive blue aorta. Installation of an extracorporeal circulation (ECC) between a femoral arterial cannula and a double stage venous cannula. Aortic clamping flush with the brachiocephalic arterial trunk, followed by vertical

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http://dx.doi.org/10.31487/j.JSCR.2023.03.03

aortotomy and removal of large blackish blood clots. Blood cardioplegia was administered through the coronary ostia followed by local refrigeration with crushed ice. Subsequently, a circulatory arrest in deep hypothermia at 18°C was performed without cerebral perfusion. Inspection of the aorta revealed the intimal lesion of the PAU but also a thrombosed false channel, a consequence of the evolution of the PAU towards a AD. Moreover, the aortic valve, of tricuspid type, presents thin, flexible cusps, without calcifications or symphysis. The aneurysmal portion of the aorta was resected, and a dacron tube was placed in a supracoronary position fixed by proximal and distal 4/0 prolene anastomosis. The postoperative period was marked by a delayed awakening, without any sensory-motor deficit, with clinical improvement of the patient.

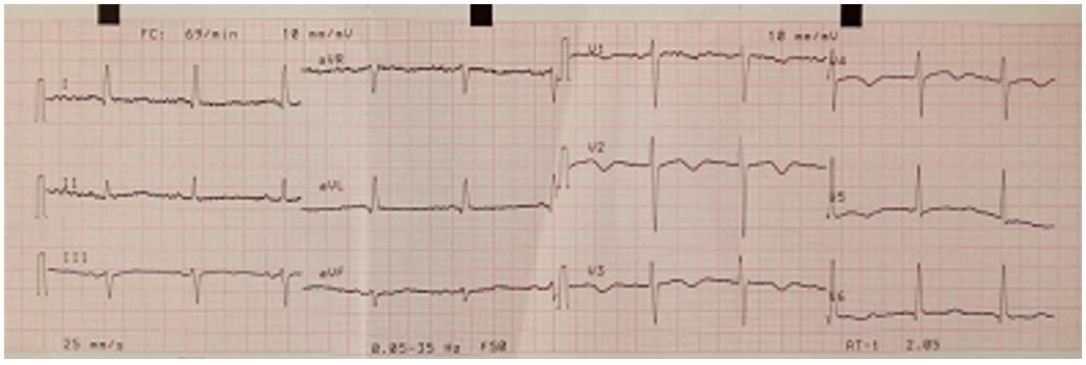

Figure 1: ECG registered in regular sinus rhythm, presence of anterolateral repolarization disorders itou necrosis Q wave.

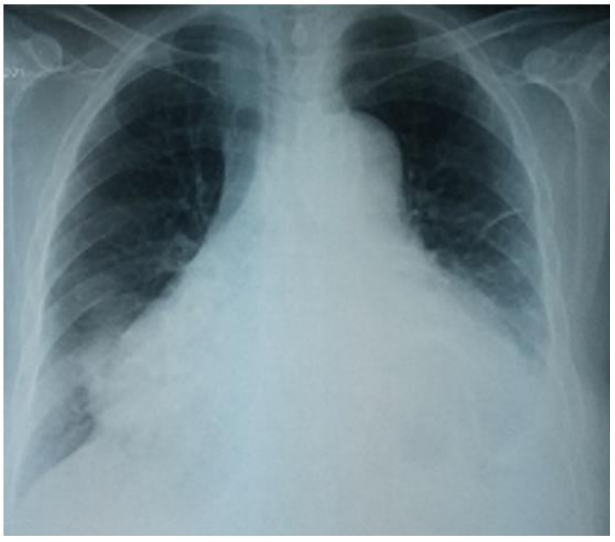

Figure 2: Chest x-ray showing cardiomegaly: cardiothoracic index=0.83 with right overhang and enlargement of the mediastinum.

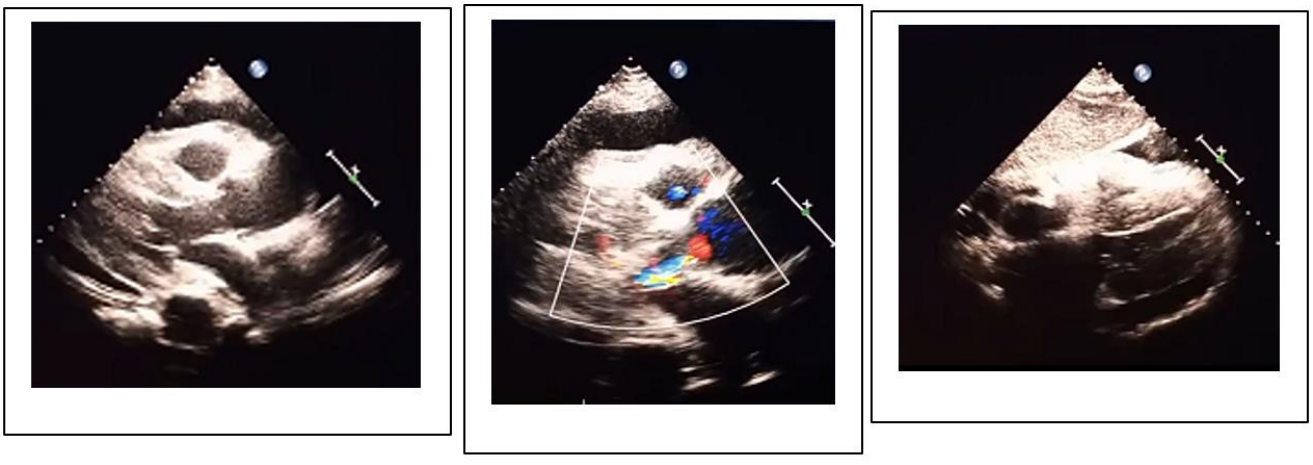

Figure 3: TTE showing dilatation of the ascending aorta to 50mm, a thin and flexible tricuspid aortic valve with minimal aortic insufficiency. The aortic annulus was measured at 21mm and a small to moderate pericardial effusion was noted.

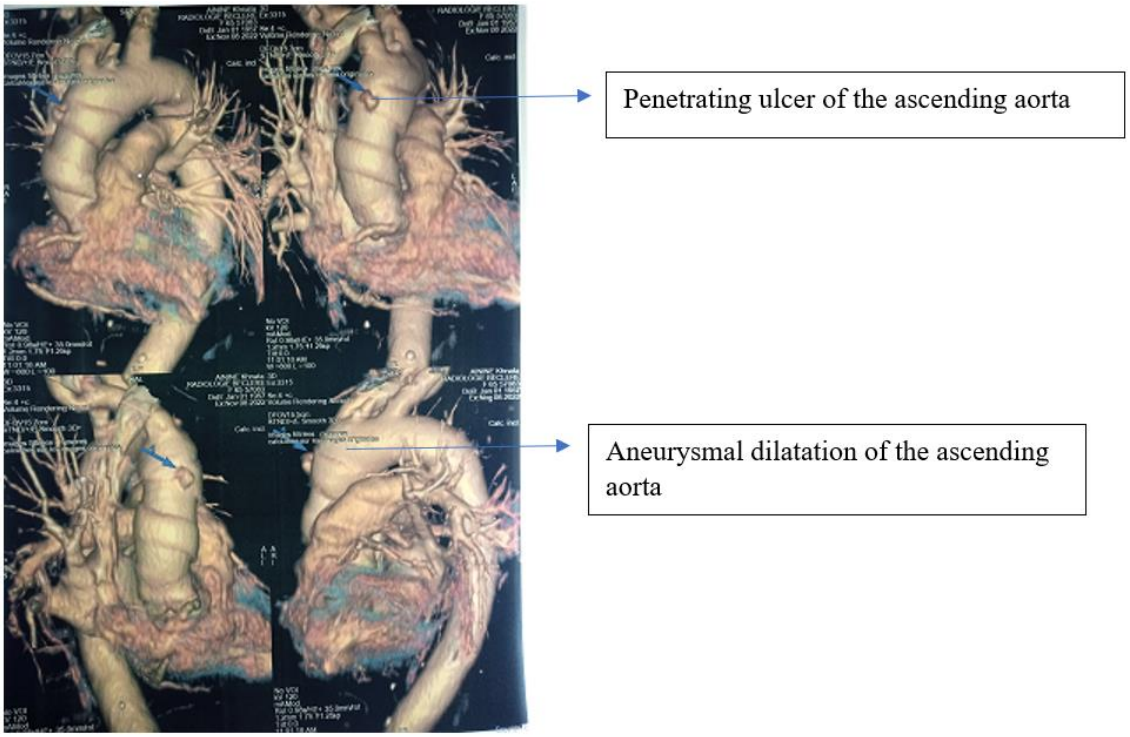

Figure 4: Thoracic angioscan showing aneurysmal dilatation of the ascending aorta and PAU.

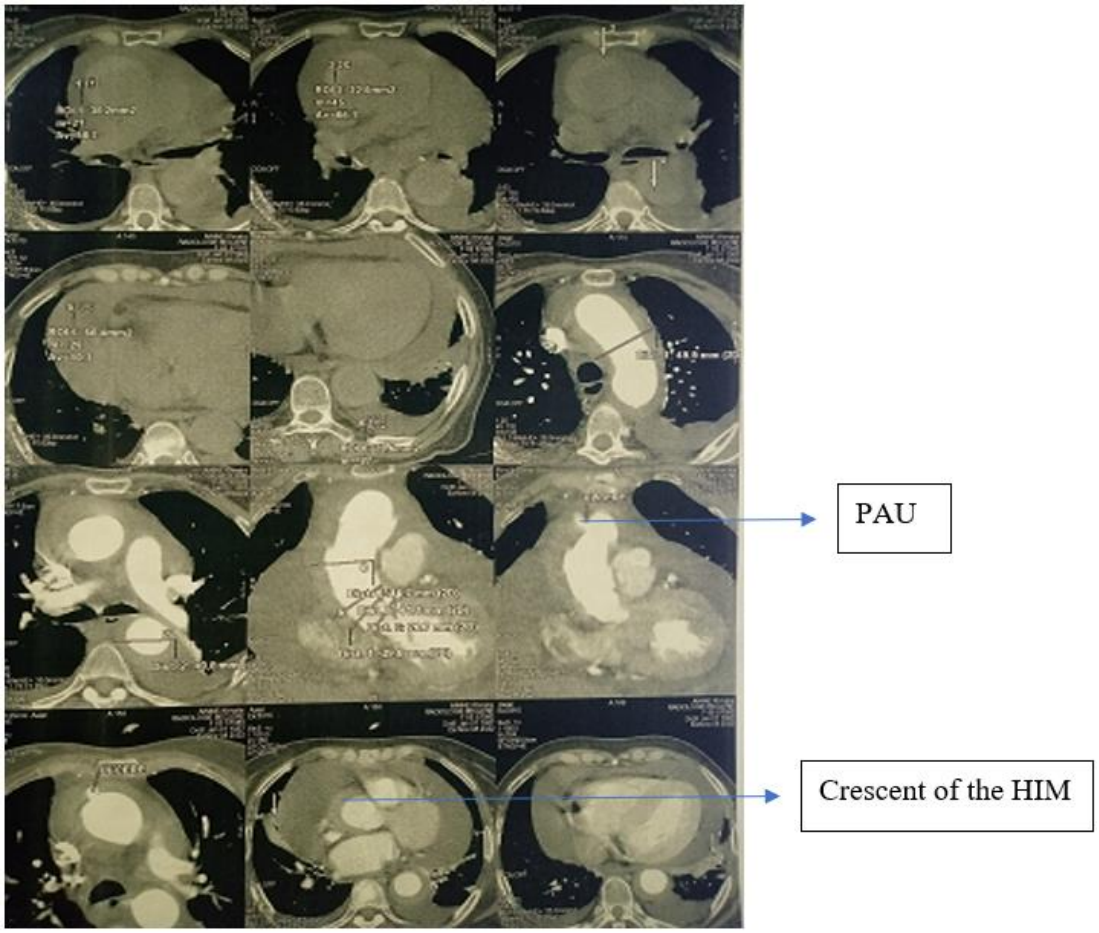

Figure 5: Thoracic angioscanner showing a dilated thoracic aorta without intimal flap.

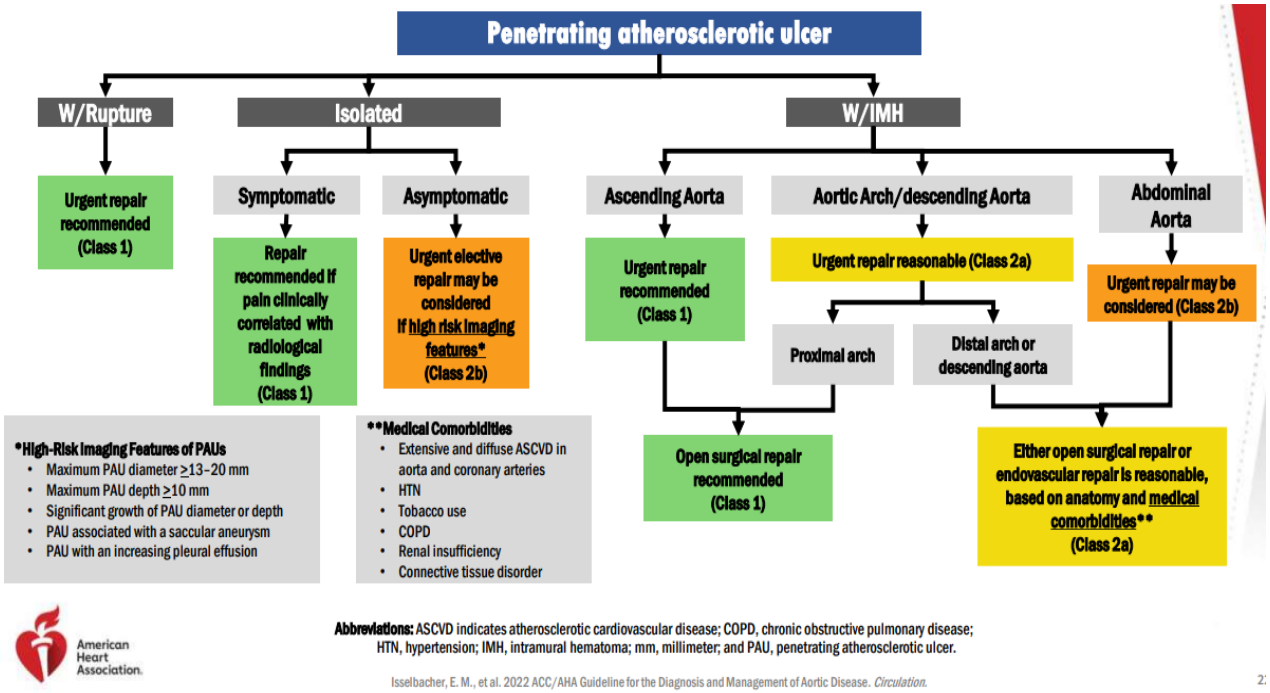

Figure 6: American College of Cardiology (ACC) and American Heart Association (AHA) 2022 guidelines on the management of PAU [6].

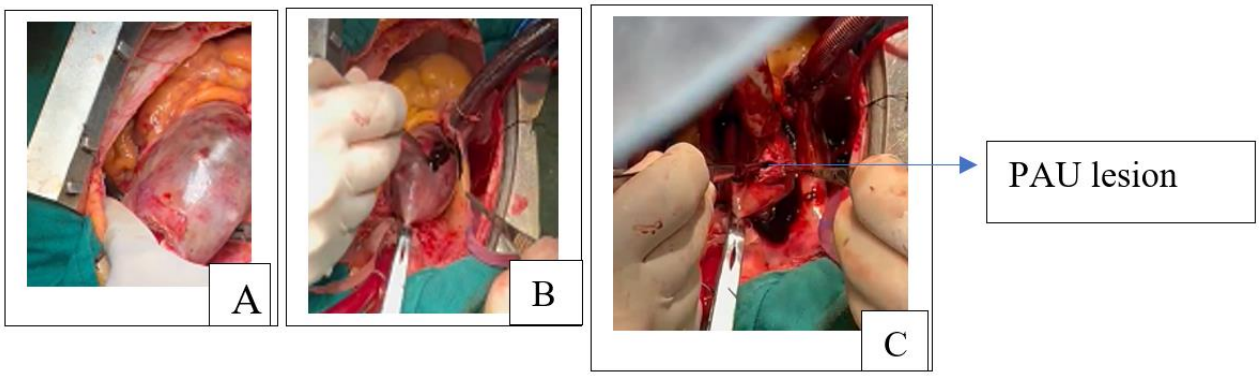

Figure 7: A) Aneurysmal dilatation of the ascending aorta. **B)** Hematoma after aortotomy. **C)** Intimo-medial lesion of the PAU.

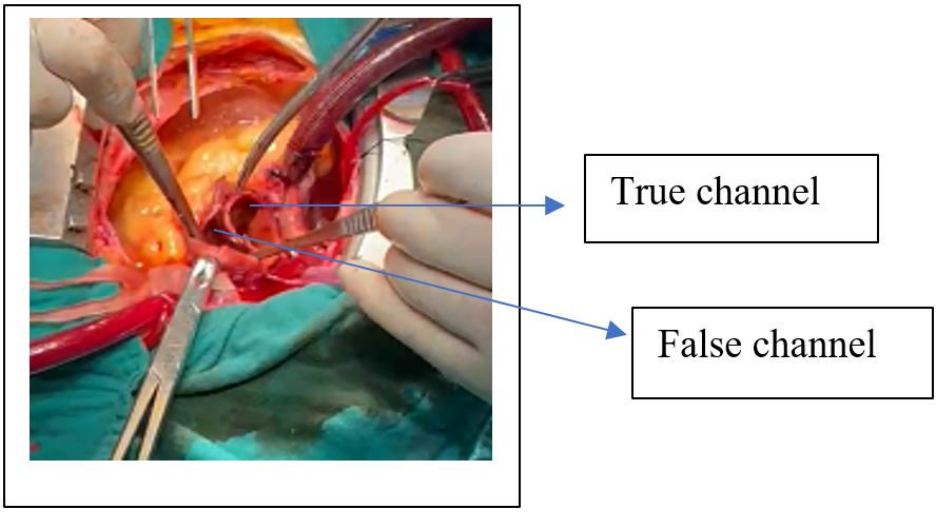

Figure 8: Visualization of the true and false channel after resection of the aneurysmal portion of the ascending aorta and removal of blood clots (the diameter of the false channel is smaller than that of the true channel).

Figure 9: Proximal anastomosis of the supracoronary tube in dacron with 4/0 prolene.

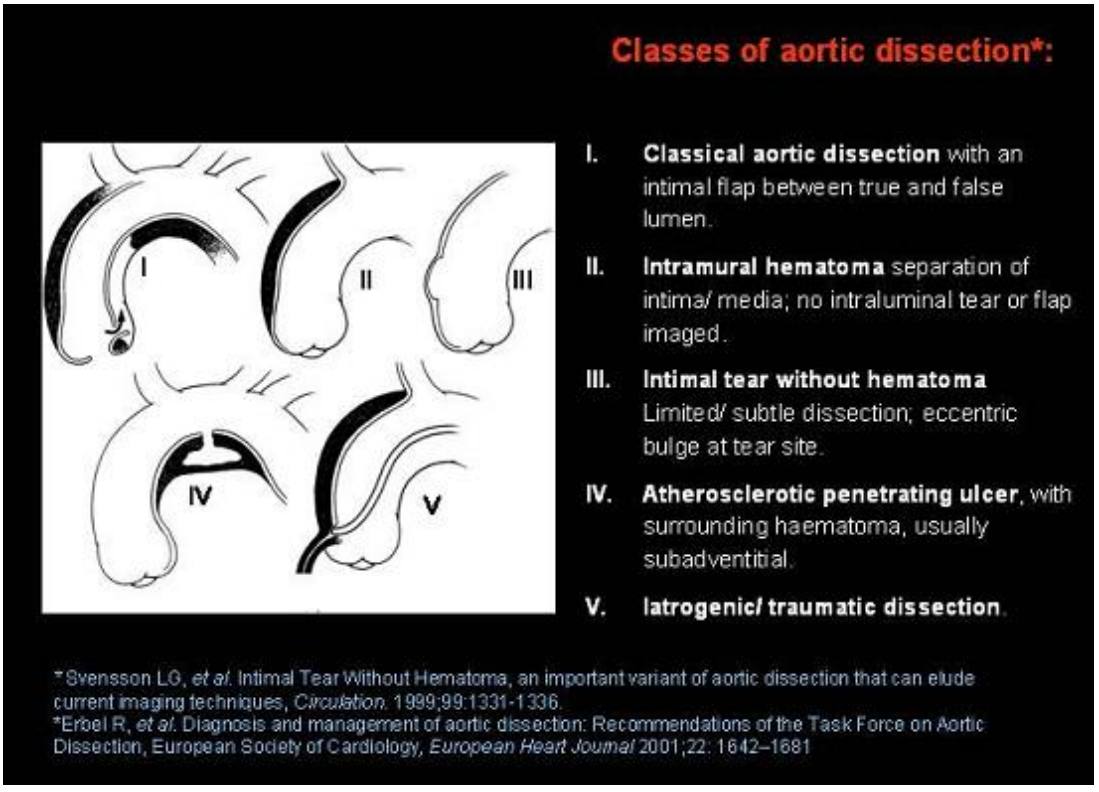

Figure 10: Svensson classification of AD [10].

Discussion

PAU is a clearly individualized anatomic-pathological entity, which is part of the AAS along with IMH and AD. It is most often observed in elderly, hypertensive patients with advanced atheromatous disease. The initial lesion is represented by a thick atheromatous plaque with an irregular surface. The erosion or rupture of the plaque will be the starting point of an extensive ulceration process, affecting the different layers of the aortic wall. Rupture of the internal border leads to the passage of blood into the media and the formation of a hematoma most often associated with a false aneurysm. The descending aorta is more affected by PAU, the location in the ascending aorta is unusual and is mostly seen at the arch [7]. Adventitial rupture represents the final and most fatal stage of PAU and occurs in 14-40% [7]. Progression to aortic dissection is possible but quite rare (less than 4%) [1]. The clinical context leading to the discovery of PAU is chest pain, which may be difficult to

distinguish from that of an AD. Our patient is not only hypertensive, but was admitted with a chest pain picture that led to the diagnosis of PAU. The absence of an intimal flap ruled out an AD.

However, the per operative discovery of an AD shows the evolutionary continuum that can exist between these different entities of AAS. This is reminiscent of the early theories making PAU the cause of AD. From Virchow (1851) to Möller (1906) via Girode (1887), Ewald (1890), and Rolleston (1893), all were proponents of this theory, before Shennan in 1934 provided evidence for the inadequacy of this theory [8]. Indeed, only 6 of 218 patients in his study had PAU as a cause of AD [8]. However, an advance in the knowledge of the evolutionary mechanisms of UPA has led to the understanding that the resulting IMH can evolve into a "double gun barrel" or thrombosed dissection [9]. The "double gun barrel" dissection presents a communication between the true and the false channel as in the classical dissection, but conversely it is described that the diameter of the true channel is greater or equal to that of the false channel [1]. As for thrombosed dissection, it is more frequent because atheromatous plaques limit the extension of the IMH and prevent the creation of re-entry circuit, appearing on imaging without intimal flap [9]. In 1999, Svensson and colleagues proposed a pathophysiological classification of AD into five subtypes that has been adopted in medical practice [10]. In this classification, PAU represents a type IV AD.

PAU can be described as a localized AD with the ulcer itself as the portal of entry. It is recognized as the initial lesion in about 5% of all AD [11]. Our patient has the particularity to present a penetrating ulcer of the ascending aorta which is an unusual localization, but also to present a rather rare evolution towards AD. The main risk to which she is exposed is the rupture of the aorta which is frequently observed in PAU.

Conclusion

PAU is a well individualized entity of AAS but its evolutionary profile can sometimes cross nosological barriers and cause diagnostic confusion. The location in the ascending aorta deserves special attention. The treatment is well codified and there should be no delay in management.

Data Availability

Figures 1- 5, 7- 9 are data from patient's file while (Figures 6 & 10) are accessible via references.

Conflicts of Interest

None.

Author Contributions

Idrissa Abdel Malick wrote the initial manuscript which was read and validated by all the authors. Pr Laaroussi M is the surgeon who operated on the patient.

Funding

None.

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