

# Revisiting the Pathological Evaluation of the Thoracic Aortic Aneurysm

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**Abstract**

**Objective:** The most recent molecular acquisitions about the pathogenesis of the thoracic aortic aneurysm permit to better understand also the morphological counterpart of this disease, where atherosclerosis is not the only factor involved. The cascade of pathological processes leading to the development of the thoracic aortic aneurysms is multifactorial.

**Methods:** We have investigated five surgical specimens of aneurysms of the thoracic aorta, collected from patients without Marfan's disease or other related genetic syndromes. Histochemistry for elastic fibers and mucosubstances has been performed, following the standard staining protocols.

**Results:** The elastolysis, which can be considered a starting process, involves all the tunica media and it is followed by the mucoid degeneration characterized by the deposition of basophilic mucopolysaccharidic substance. Finally, the overlap of classic atherosclerotic lesions is observed, inside the whole aortic wall.

**Conclusions:** The development of a thoracic aortic aneurysm is a complex event, where different factors and processes are involved, other than the well-known atherosclerosis, which can finally appear the most evident aspect of the overt disease.

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## INTRODUCTION

The classical histopathologic findings of the thoracic aortic aneurysm (TAA) are well known, in fact they are usually interpreted in the light of atherosclerosis, which is considered the most important pathogenetic factor. Today, a lot of ultra-structural, especially molecular and biochemical studies, recall attention to other factors [1-5]. These new acquisitions permit to better understand also the morphological counterpart of TAA, which cannot be merely reported only to a unique atherosclerotic process.

## MATERIALS AND METHODS

We have examined the surgical specimens of five TAA, collected from patients aged between 67 and 74 years, excluding patients affected by Marfan's disease and related genetic syndromes or by autoimmune

diseases. All the patients showed risk factors for atherosclerosis, as arterial hypertension, smoking habitude, and dyslipidemia.

The surgical specimens were fixed in 10% neutral buffered formalin and then paraffin embedded. In addition to Haematoxylin & Eosin (H&E), histochemistry for elastic fibers (Weigert's staining, Van Gieson's staining, Verhoeff's staining) and for mucins (Alcian blue/PAS staining) has been performed, following the standard staining protocols.

## RESULTS

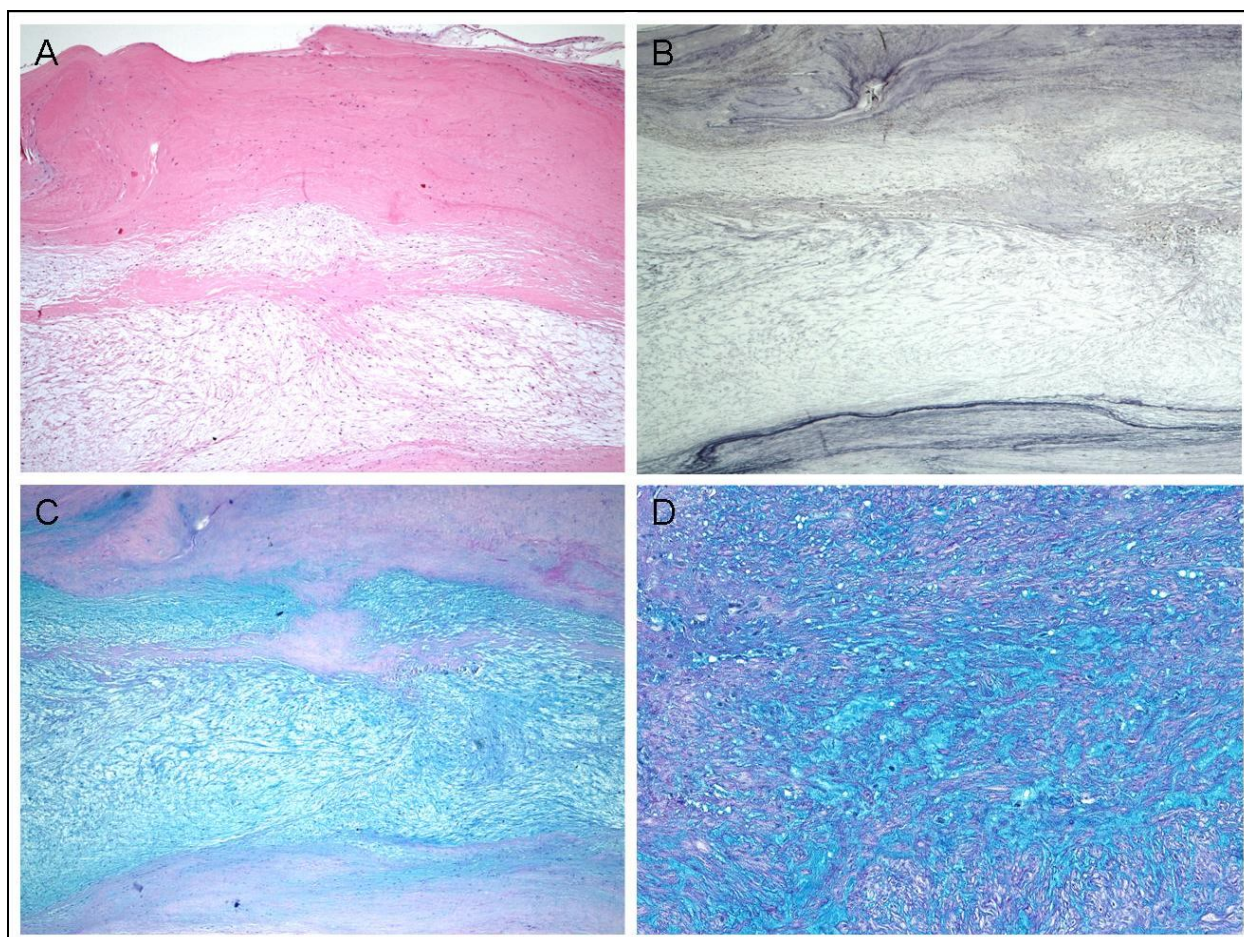
In all examined cases of TAA the disappearance of the elastic component of the tunica media has been observed. This event is secondary to a process of elastolysis, histochemically proved by the fragmentation of the elastic fibers, interspersed with

some areas of their complete destruction (Figure 1). The elastic membranes of the tunica media are subjected to the same degenerative process, appearing largely disrupted.

The pseudocystic mucoid degeneration can be considered a subsequent step, involving the tunica media (Figure 1). It is the result of a deposition of basophilic mucopolysaccharidic substance, not provided

of elastic properties and with a low strength resistance.

The classical atherosclerotic lesions, as lipid accumulations and calcifications, are evident, but they appear an overlapping phenomenon, not so important in relation to the severity of the disease. They are the same alterations observable in other vascular districts, not involved by aneurysmatic disease.



**Fig. 1** Pseudo-cystic mucoid degeneration of aneurysmatic thoracic aorta (A; H&E, x40). The loss of elastic fibers (B; Weigert staining, x40) and the subsequent accumulation of mucins (C; Alcian Blue/PAS staining, x40) are evident in the areas of mucoid degeneration. The mucosubstances fill the spaces left open by the disappearance of the elastic fibers (D; Alcian blue/PAS staining, x200).

## DISCUSSION

The most important morphological aspects of TAA can be revisited and better appreciated, in consideration of the modern contributions of vascular biology.

The elastic component of the ascending thoracic aorta is particularly relevant from an anatomic and functional point of view [6]. The elastolysis can be considered the ending point of genetic and biochemical alterations, and, at the same time, the starting point of the

histopathological lesions. A subsequent and correlated pathological process is the mucoid degeneration, largely involving the tunica media [7-9].

We underline the absence of a smooth muscle-like cell proliferation, which could be expected after the elastolytic process, as observed in other elastic arteries, typically in the internal thoracic artery [10]. This particular attitude of some arterial wall, interpreted as a spontaneous remodeling process, has no equivalent in the ascending thoracic aorta, where, on the contrary, a

mucoid degeneration takes place [11,12].

The classical atherosclerotic lesions appear in the aortic wall, but their appearance does not correspond to the severity of the disease. Moreover, they are less evident than in other vascular districts, as in the aneurysms of the abdominal aorta; it is recalled that the thoracic aorta and the abdominal aorta have a different embryological development. Atherosclerosis can so be interpreted as a concomitant or an overlapping phenomenon, important for the evolution of the disease, but not the unique pathological process involved.

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