Type A aortic dissection

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Figure 1. A – On opening the pericardium, after removing a large blood clot, perforation of the intrapericardial aorta is identified; B – After removing the heart, the sloughed aortic wall is identified. At the opening of the aorta’s emergence, the blood sequestration is found within the dissection plane (arrows); C – The first tracts of the supra-aortic arterial vessels also show wall dissection; D – Photomicrograph of the aortic wall dissection (black arrow), with blood interposition between the two layers (white arrow) (H&E, 2x).

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Acute aortic dissection (AAD) is one of the acute aortic syndromes, along with intramural hematoma, penetrating ulcer, and aortic rupture. AAD is a relatively uncommon condition (the incidence is approximately 2.6 to 3.5 per 100,000 population per year), but often fatal if not timely treated with reconstructive surgery.1,2

Etiologically, AADs can result from congenital causes (structural defect present at birth), genetically determined with onset during childhood or even later, or acquired (inflammatory, degenerative, neoplastic, traumatic).3,4

From a morphological point of view, the AAD consists of a breach in the thickness of the aorta wall, between the tunica media and the intima, leading to the creation of a ‘false lumen’ between these two layers, into which blood infiltrates. It rarely remains localized and often progresses by slimming the aortic wall and, in some cases, extending outside the vessel. AAD involving the aorta from its ascending intrapericardial portion (as in our case) and/or the arch of the aorta is termed acute type A aortic dissection (ATAAAD) according to the Stanford classification.5

ATAAAD is associated with a high mortality rate, near 50% at 48 hours without surgical intervention;6 in particular external rupture of the intrapericardial aortic tract leads to hemopericardium and cardiac tamponade: the latter event is the most common cause of death from AATAAD.

Figure 1 belongs to a 58-year-old man who presented with an acute confusional state and hyperpyrexia. On admission, he was diagnosed with atrial fibrillation of unknown cause, right lung nodule, and diffusion/FLAIR signal changes on brain MRI. The clinical suspicion was encephalitis or endocarditis. The microscopic examination of the heart valves and endocardium lacked inflammatory infiltrate. Notwithstanding, fever is a sign often related to AAD, which could be caused by thrombi formation, necrotic tissue, cytokines, free radicals and oxygen radicals that are associated with aortic dissection.7

Also, no microscopic findings associated with hypertension in either the lungs or the kidneys, and no signs of atherosclerosis or arteritis were found.

In conclusion, the most consistent etiological hypothesis is therefore that of an abnormality of the connective tissue, not further specified.

The final autopsy diagnosis was, therefore, death due to dissection of the intrapericardial aorta (aortic dissection type A), associated with intrapericardial aortic rupture, massive hemopericardium, and cardiac tamponade.

Keywords
Aneurysm, Dissecting, Aortic Arch Syndromes, Aortic Diseases, Aortic Rupture, Cardiac Tamponade

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This study was carried out at San Martino Policlinico Hospital, IRCCS, Genoa, Italy.

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