

*Case Report***An Unsuspected Case of Aortic Dissection**Rossana Bussani<sup>1</sup>, Aneta Aleksova<sup>2</sup>, Salam Kassem<sup>2</sup>, Zandonà Lorenzo<sup>1</sup>, Presello Barbara<sup>3</sup>, Gianfranco Sinagra<sup>2</sup><sup>1</sup>Institute of Pathological Anatomy and Histology, “Ospedali Riuniti di Trieste” and University of Trieste, Trieste, Italy<sup>2</sup>Cardiovascular Department, “Ospedali Riuniti” and University of Trieste, Trieste, Italy<sup>3</sup>Department of Anesthesia and Intensive Care, “Ospedali Riuniti” and University of Trieste, Trieste, Italy

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*Received: 23/04/2015**Revised: 15/05/2015**Accepted: 25/05/2015***ABSTRACT**

Aortic dissection is a rare life-threatening condition that typically presents with acute onset of severe chest, abdominal, or back pain. Some patients might present with atypical symptoms and findings, such as neurological syndromes, making it difficult to make the diagnosis.

Here, we report a case of a painless acute aortic rupture over an already existing Stanford type A chronic aortic dissection in a 59-year-old patient, with prevalence of neurological symptoms. The aortic dissection presented as recurrent episodes of dizziness/syncope and transient amnesia.

We believe it is important to report this case because of the rare clinical presentation of aortic dissection, raising the awareness and diagnosing level of atypical aortic dissections.

**Key words:** Painless aortic dissection, syncope, dizziness, amnesia.

**Introduction**

Aortic dissection is a rare life-threatening condition with an incidence estimated at 6 per 100000 persons per year. <sup>[1]</sup>

Complications often develop rapidly and usually the outcome is fatal. Aortic dissection can produce not only a wide range of symptoms, but also extremely variable, thus making the correct diagnosis difficult and challenging. Typically it presents with acute onset of severe chest, abdominal, or back pain. It is often misdiagnosed in the event of an atypical presentation, especially in pain-free dissections with predominant neurological symptoms. We report a case of a painless acute aortic rupture over an already existing Stanford type A chronic aortic dissection,

presenting with recurrent neurological symptoms.

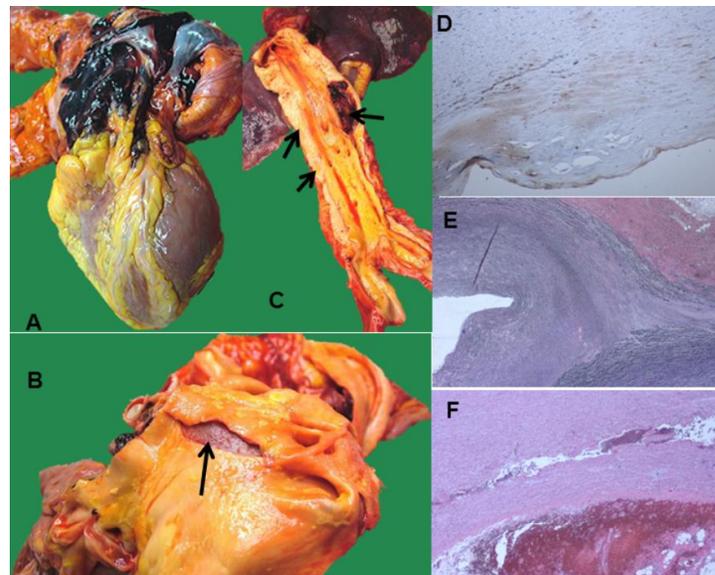
**CASE PRESENTATION**

A 59-year-old woman presented to the emergency department because of an acute episode of syncope at work followed by vomiting at arrival. On examination, she was alert but not collaborative. Upon arrival her heart rate was 66 beats/min, blood pressure 100/60 mmHg, temperature 37 °C and pulse oximetry 99%. Pulses were palpable symmetrically and showed no deficits. The remainder of her physical examination was normal. Her colleague referred that before falling down she accused a strong headache. Medical history of the patient was unremarkable. Her family

reported that during the past months she suffered from memory loss, and frequent dizziness episodes. They also reported that she became weird ultimately, with mood and personality changes. On neurological examination, besides being disoriented to place and time, she was unable to respond to comments or questions (Glasgow coma scale score 11). She had no loss of motor or sensory function, any neck stiffness or previous head trauma. The syncope and confusional state of the patient were suggestive to cerebrovascular disease and a cranial computed tomography, without contrast injection, was performed. It showed no abnormality. An EKG showed sinus rhythm with no evidence of ischemia or infarction. Results of usual laboratory studies including serum creatinine, CRP (C-reactive protein), ionogram were normal. Two hours later, her temperature increased (38.2°C), she closed her eyes and assumed a constant lateral decubitus with all limbs in

semi-flexion. The neurologist, suspecting encephalitis or meningitis, performed lumbar puncture, but examination of the cerebrospinal fluid showed normal concentration of glucose, protein, and lymphocytes. Suddenly, the patient had a cardiac arrest and died within four hours of admission.

Post-mortem examination revealed a Stanford type A chronic aortic dissection of the thoracic aorta with an acute rupture 4 cm above the aortic valve (Figure 1B) with consequent hemothorax and hemopericardium (Figure 1A). The dissection extended from the ascending aorta including the bases of the left subclavian and left common carotid arteries, and continued to the iliac arteries. The aortic neo-intima showed severe arterIALIZATION with fatty streaks, fibrous plaques, calcific foci and deposition of thrombotic material (Figure 1C).



**Figure 1.**

A. Dissection of the thoracic aorta with hemopericardium

B. Macroscopic finding showing acute aortic rupture

C. Thoracic aorta cross The cross shows chronic dissection of the thoracic aorta and the presence of a false lumen. The false lumen shows severe sclerosis and calcification and a large mural thrombus.

D. Histological finding showing chronic aortic dissection with endothelialization of the false lumen [CD31 stain, X10]

E. Histological finding showing fragmentation of elastic fibers of the middle layer of the aorta and severe thickening and fibrosis of the false lumen [Weigert-van Gieson stain, x2.5]

F. Histological finding showing acute aortic dissection [H. & E. stain, x 2.5]

Histopathological examination of the aorta indicated aortic dissection (Figure 1 F); the dissection involved the tunica media, which appeared heavily fibrotized and depleted of elastic fibers (Figure 1 D,E). Cerebral autopsy showed light hypotrophy of left cerebral hemisphere with cortical neuronal loss and reactive gliosis, ectasia of the lateral and third ventricles probably related to the chronicity of the dissection, which also included left sided arch vessels, that compromised blood flow to the left cerebral hemisphere. Final diagnosis: Stanford type A chronic aortic dissection with acute rupture in pericardium.

## DISCUSSION

This case describes a painless aortic dissection resulting in a neurologic syndrome, this time as combination of dizziness/syncope and transient amnesia episodes. It is an example of an atypical presentation of aortic dissection. Several case reports described atypical presentations of aortic dissection, presenting mainly with neurological symptoms [2-5] but only Gaul et al [6] described a similar combination of symptoms in patients with an underlying aortic dissection.

Aortic dissection is a rare condition, but complications develop rapidly and usually the outcome is fatal. [7] Aortic dissection is missed in approximately 40% of patients on initial evaluation, and in up to 28% of patients the diagnosis is made post-mortem. [2,8]

Neurological involvement in aortic dissection has been reported to occur in nearly in 17% to 40%. [9,10] Since pain is by far the most characteristic symptom of aortic dissection, its absence may divert attention from the correct diagnosis. In fact pain-free dissections range from 5% and 15%. [8,10] Neurological symptoms are often fluctuating, transient and fully remitted [6]

probably resulting from transient arterial occlusion with consequent transient cerebral hypoperfusion. This could explain the frequent dizziness episodes from which the patient suffered.

Our patient also suffered from recurrent transient amnesia episodes. We could not define the nature of the amnesia since at the moment of the episodes the patient did not present to the hospital. Several case reports described patients with aortic dissection presenting with transient global amnesia without focal neurological defects [3,11-15] and three of which without pain on initial presentation. [12,13,15] Transient global amnesia (TGA) is a clinical syndrome of reversible anterograde amnesia accompanied by repetitive questioning without impairment of consciousness, focal neurologic deficits, recent head trauma, epileptic features, or duration longer than 24 hours. [16]

The mental status impairment, personality and behavior changes, which accompanied our patient during the last few months could be related to the hypoxic encephalopathy, proved in post-mortem examination, that usually follows chronic hypoperfusion of the brain. It is probably due to extending of the dissection into the left carotid and subclavian arteries, which caused a chronic hypoperfusion of the left cerebral hemisphere.

Therefore, although we have been taught to suspect aortic dissection in the presence of the classical chest pain syndrome, in realty clinical presentations of aortic dissection can be so varied and atypical.

## CONCLUSION

Physicians, particularly emergency doctors, must be alert and maintain a high index of clinical suspicion considering the differential diagnosis of a painless chronic aortic dissection in patients presenting with

syncope alone or in combination with other neurological symptoms considering also AD in the presence of amnesia attack.

**Conflict of Interest:** The authors declare that there are no conflicts of interest.

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