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Before a Syncope in an Aortic Dissection is Attributed to a Direct Affection of the Vagal Nerve, All Other Causes Must be Thoroughly Excluded

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LETTER TO THE EDITOR

We read with interest the article by Donner, *et al*., about an 82-year-old woman with a type A aortic dissection that was diagnosed with a 24-hour delay and initially manifested with vagovasal syncope [Donner, V. *et al*., 2024]. It was concluded that vasovagal activation without pain may be the underlying mechanism of syncope in acute type A aortic dissection [Donner, V. *et al*., 2024]. The study is excellent, but some points should be discussed.

The first point is the discrepancy between the case description, which speaks of collapse, and the title, which speaks of syncope [Donner, V. *et al*., 2024]. These two conditions have a different phenomenology and should be clearly distinguished. Syncope is characterized by a loss of consciousness. During a collapse, the patient remains awake.

The second point is that, in view of the fact that the fall was a syncope, the clarification of the underlying cause was inadequate. Syncope always requires not only a cardiologic evaluation, but also neurologic examinations. No mention was made of cerebral magnetic resonance imaging (MRI), electroencephalography (EEG) or carotid ultrasound. In patients with a history of syncope, ischemic stroke, cerebral hemorrhage, epileptic seizures, encephalitis and carotid artery stenosis or occlusion must be thoroughly ruled out.

The third point is that the parasympathetic overactivity may not only be due to a direct impairment of the vagal nerve by the dissection, but also to an impairment of the central nervous system (CNS) due to transient ischemia of the brain stem. Since the patient had arterial hypotension and bradycardia, intracardiac thrombus formation causing cardioembolism in the brain is also conceivable. Cardioembolism could also have been caused by an episode of atrial fibrillation. Was there any evidence of enlargement of the left atrium on echocardiography?

The fourth point is that various other causes of syncope have not been adequately ruled out. These include hypovolemia, stress, high temperature, vitamin deficiency, strong emotions, profuse sweating or exhaustion.

The fifth point is that Takotsubo syndrome (TTS) has not been ruled out as a cause of syncope. Since acute aortic dissection can be complicated by TTS [Shimizu, K. *et al*., 2022], it would have been imperative to consider short-lasting TTS as the cause of syncope. TTS can be complicated by heart failure, ventricular arrhythmias and cardioembolism.

The sixth point is that the current medication was not stated. As the patient had a history of arterial hypertension and was 82 years old, it is conceivable that the patient was taking medication regularly. These must also be ruled out as a cause of the syncope before it can be attributed to the aortic dissection.

In summary, it can be said that this interesting study has limitations that relativize the results and their interpretation. Addressing these limitations could strengthen the conclusions and corroborate the study's message. Before attributing syncope in an elderly patient to aortic dissection, all other etiologies must be thoroughly ruled out.

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