



Swallowing-induced atrioventricular block and syncope in a patient with achalasia

Achalasia is a motor dysfunction of the oesophagus characterized by increased pressure in the lower sphincter and incomplete relaxation after swallowing (1). Classical symptoms include dysphagia, regurgitation, chest pain and weight loss. Cardiovascular complications of achalasia are very rare (2). We report a patient with achalasia in whom swallowing induced bradycardia and atrioventricular (AV) complete block during ingestion of food with patient's consent. The patient was a 47-year-old man, with an 18-year history of dysphagia and odynophagia. He was diagnosed with achalasia, and pneumatic dilatation was performed once; he had not been undergoing any medical treatment. He had three syncope attacks in the previous month, and the aetiology of his syncope was investigated by cardiologists. He was normotensive and cardiac examination yielded normal results. His resting electrocardiography (ECG) results were also normal. This was followed by transthoracic echocardiography (Philips Envisor C HD Andover, MA, ABD). No significant wall motion disorder or valve dysfunction was reported. Thoracic computed tomography (CT) revealed a noticeably dilated oesoph-

agus (Figure 1a). We performed 24-h ambulatory ECG monitoring (SpiderView®; ELA Medical, Sorin Group, Paris, France) and, at the time of breakfast and dinner, noticed that the patient was bradycardic: there were 2:1 and 3:1 AV blocks and pauses (the longest pause was of 4.3 s; Figure 1b). During the pauses, the patient experienced dizziness. Pneumatic dilatation was performed again and pharmacotherapy [nifedipine 60 mg/day (Bayer medical, Turkey) and isosorbide dinitrate 40 mg/day (Adeka medical, Turkey)] was initiated. The patient complained of mild dysphagia after the treatment, but he did not experience any syncope attacks again. Next, 24-h ambulatory ECG monitoring was repeated. According to his ECG holter, the patient had bradycardia, but no AV blocks were observed. Swallowing-induced syncope occurs because of AV blocks and bradycardia associated with overstimulation by vagal stimuli (1). Mechanoreceptors of the lower oesophagus play an important role in the pathophysiology of swallowing-associated cardiac arrhythmias (3), and hypersensitivity is the key feature of vagotonic reflex syncope (4). The nerves innervating the lower oesophagus can be

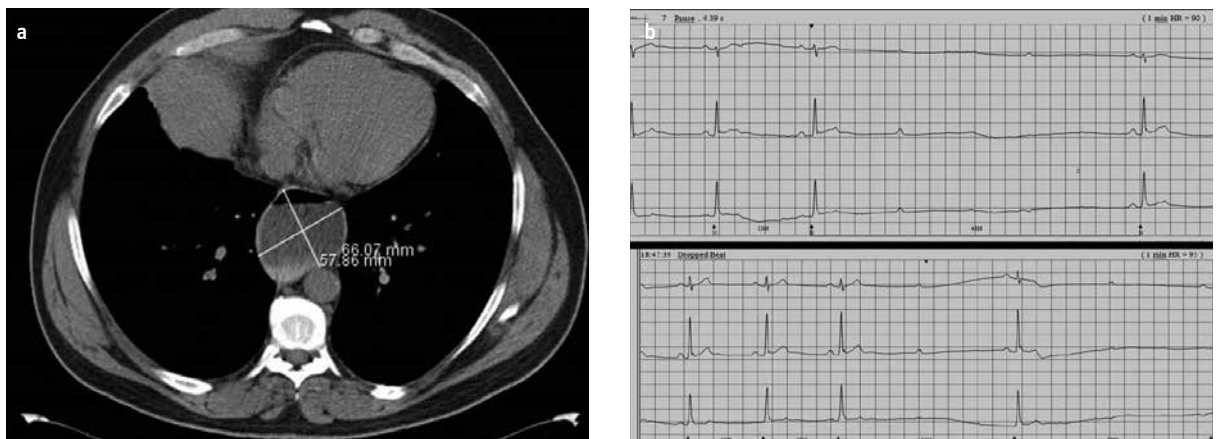


Figure 1. a, b. Thoracic computed tomography showing the dilated oesophagus and food scraps in the distal oesophagus (a). 24-h ambulatory electrocardiography monitoring showing 2:1 and 3:1 atrioventricular blocks and pauses (the longest pause, 4.3 s) at the time of breakfast and dinner (b).

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traced to the nucleus tractus solitarius in the brain, and the nerves originating from there form the parasympathetic nervous system, which affects the heart. Impulses from vagal fibres affect the sinoatrial node, atrial musculature and AV node and can cause clinically important arrhythmias (4). These types of arrhythmias usually disappear after pneumatic dilatation and surgical myomectomy. In rare cases, a patient needs a pacemaker (5). In our case, aetiological treatment reduced the signs and symptoms; therefore, a permanent pacemaker may not be required. We believe that clinicians should be careful when prescribing drugs affecting the AV node such as β -blockers, calcium channel blockers or digoxin in this group of patients.

Informed Consent: Written informed consent was obtained from patient who participated in this study.

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