Pseudoachalasia as a paraneoplastic event in a patient with Hodgkin's disease

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Dear Editor,

62-years-old male with history of arterial hypertension, diabetes, chronic ischemic heart disease, and in 2011 Hodgkin's lymphoma treated with 6 cycles of ABVD with relapse in September 2021 treated with 4 cycles of ABVD in December 2021, and with radiotherapy between January and February 2021.

In November 2021 he was admitted due to dysphagia and regurgitation with rapid progression in two months (Eckardt Score of 9 points). Gastroscopy: dilation of the esophageal lumen and decreased esophageal peristalsis (figure 1a). High-resolution esophageal manometry (HRM): no relaxation of the lower esophageal sphincter (LES) with integrated relaxation pressure at 4 seconds (IRP 4s) of 34 mmHg and failed contractions in all swallows with panpressurization compatible with type II achalasia.
according to the Chicago V4 classification (Figure 1b). Given the active oncological process, conservative treatment was performed with botulinum toxin injection (100 UI) with low efficacy. The option of performing a surgical Heller myotomy or a peroral endoscopic myotomy (POEM) was rejected given the oncological process. Endoscopic pneumatic dilation with a 30 mm (310 kPa) balloon was performed in February 2023 achieving complete deletion of the LES print (Figure 1c-d). After three months, the patient reports complete resolution of symptoms from the first week of dilation.

Pseudoachalasia or secondary achalasia (5% of achalasias that are deemed primary achalasias) is an esophageal motor disorder with manometric criteria for achalasia, but it appears in the context of an underlying pathology that can be attributed to its origin. Usually appears in >60 years with rapid evolution of symptoms (<1 year). The main cause of pseudoachalasia is neoformative etiology, but there are others\textsuperscript{1,2}. Our patient started with rapid progression dysphagia and was diagnosed with type II achalasia within a Hodgkin’s lymphoma. In the radiological-metabolic studies, disease involvement was ruled out as an extrinsic compression of the esophagogastric junction as well as signs of its activity at this level. Chemotherapy has not been shown to play a role in the development of this pathology. On the other hand, radiotherapy has been associated with an esophageal motor disorder\textsuperscript{4}, but, in our case, it was after its onset. Therefore, we propose that the mechanism of pseudoachalasia in our case is a paraneoplastic event. This hypothesis is related to other similar cases reported\textsuperscript{3,5}, and it reflects the importance of continuing to investigate this clinical condition that is indistinguishable by manometry from primary achalasia. In addition, it usually presents differential clinical characteristics whose early recognition has implications for the diagnostic, therapeutic, and prognostic management of the patient.
REFERENCES


Figure 1: Pseudoachalasia in patient with Hodgkin’s disease. 1a: dilation of the esophageal lumen, food debris, and decreased esophageal peristalsis. 1b: no relaxation of the LES, PRI 4s of 34 mmHg and failed contractions with panpressurization. 1c: endoscopic pneumatic dilation with a 30 mm (310 kPa) balloon. 1d: complete deletion of the LES print.