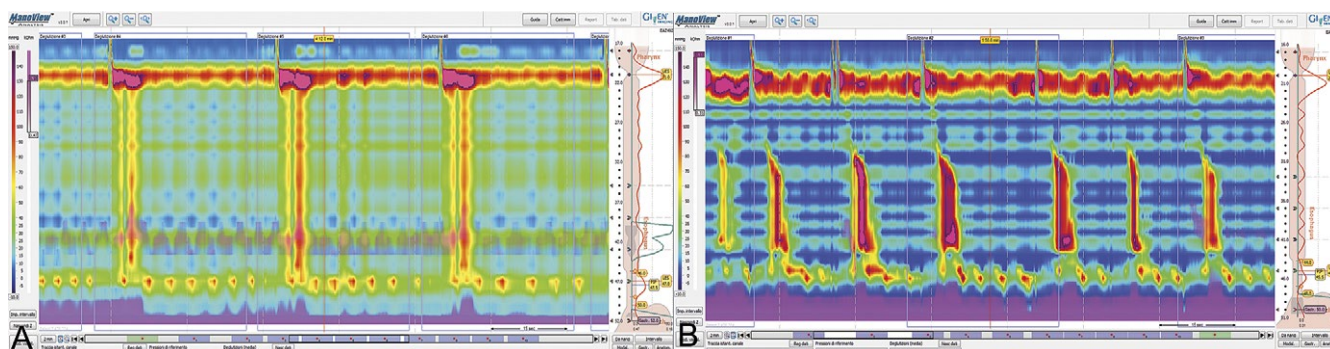


Unexpected Appearance of Esophageal Spastic Activity after Pneumatic Dilatation in a Patient with Type II Achalasia

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A 40-year old man was referred to our clinic due to dysphagia, regurgitation, retrosternal pain and weight loss of 5 kg during the last year (Eckardt score 8). An upper endoscopy did not detect any mucosal lesions. The high resolution manometry (HRIM) detected integrated relaxation pressure (IRP) 26.4 mmHg, basal lower esophageal sphincter (LES) pressure 43.9 mmHg and 100% pressurization, corresponding to esophageal achalasia type II (Fig. A) in accordance with the Chicago Classification 3.0 [1]. A pneumatic dilatation with Rigiflex balloon 30 mm, 15 PSI for 1 min, was performed without any intra- or post-operative complications. At the one-month follow-up visit, the patient reported complete regression of regurgitation and retrosternal pain but persistent dysphagia (Eckardt score 3). The HRIM showed the normalization of the integrated relaxation pressure (IRP) (12.8 mmHg) and the appearance of spastic contractions in the esophageal body [60% premature contractions, average distal latency of 3.1 sec and average distal contractile integral (DCI) of 3192.6 mmHg-cm-s] corresponding to distal esophageal spasm (DES) (Fig. B).

There are several theories stipulated for the evolution of achalasia disease [2-4]. Recently, the continuum of achalasia types, in both patients treated and untreated, hypothesizes that achalasia evolves from type III to type II and then to type I because of the progressive preferential loss of major inhibitory neurons [3]. The appearance of spastic activity on

the esophageal body in our patient after pneumatic dilatation suggests that there are still several things to clarify and understand about achalasia evolution.

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Conflicts of interest: None to declare.

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