EDITORIAL

Achalasia subtype differences based on clinical symptoms, radiographic findings, and stasis scores

Diferencias en los subtipos de acalasia basadas en síntomas clínicos, hallazgos radiológicos y puntajes de estasis

The article by Meillier et al.¹ included in this issue evaluated the clinical differences between achalasia subtypes based on symptoms, esophagogram, and a new esophageal stasis score measured with high-resolution impedance manometry (HRIM). The findings of interest in that study on 108 patients are: 1) clinical symptoms did not aid in distinguishing the different achalasia subtypes, with the exception of vomiting, which was more common in subtype I; 2) the degree of esophageal dilation, obtained through measuring the largest diameter of the distal esophagus in the esophagram, was significantly less in subtype III, but no difference was found in subtypes I and II; and 3) the stasis scores of the three subtypes showed no differences.

Despite the limitations inherent in the retrospective design of the study and the small number of patients with subtype I, it demonstrated the impossibility of classifying achalasia subtypes through symptom questionnaires or parameters of esophageal stasis measurement. High resolution manometry is essential for establishing achalasia classification, whose prediction of treatment response results in its well-known prognostic importance.²⁻⁷

A striking aspect of the case series by Meillier et al.¹ was the lower mean age of patients with subtype I, compared with those with subtypes II and III. Even more intriguing was the fact that the esophageal diameter was greater in subtype II achalasia than in subtype I. Previous studies led us to infer that subtype II corresponded to initial stages of achalasia and that subtype I occurred in advanced disease stages, with the characteristic important dilation of the esophageal body and retention of food remnants.⁸ The findings by Meillier et al.¹ do not concur with that hypothesis. In addition, studies that evaluated the histologic alterations and the type of inflammatory infiltrate of the lower esophageal sphincter obtained through biopsy of complete muscle thickness during surgical myotomy have posited that type III achalasia appears to behave differently from types I and II, suggesting a different pathophysiologic mechanism in terms of neuromuscular lesion.⁹ Further studies are needed to clarify our knowledge of the natural history of achalasia and the factors that determine its different manometric types.

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Conflict of interest

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References


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