Oesophageal physiology abnormalities in patients with joint hypermobility syndrome

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Abstract

Introduction EDS-hypermobility type/joint hypermobility syndrome (JHS) is frequently associated with functional gut symptoms. Altered pain processing and visceral hypersensitivity have been proposed to play a role in this association. However, co-existent gut motor dysfunction secondary to underlying abnormal connective tissue might also play a role. The aim of this study is to investigate the clinical presentation and oesophageal physiology abnormalities in a large cohort of patients with JHS referred for investigation of symptoms of reflux and dysphagia.

Method Forty-nine consecutive patients with JHS, previously diagnosed by a rheumatology consultant, were referred to the gastrointestinal physiology department for further investigation of upper GI symptoms. Twenty-five consecutive patients without JHS, referred over a period of one month, were used as controls. Patients underwent high resolution oesophageal manometry (HRM) and combined multichannel impedance-pH monitoring (MII-pH). Oesophageal physiology data and clinical presentation were reviewed retrospectively. Manometry analysis was done according to the Chicago classification. MII-pH recordings were analysed for total acid time exposure and for reflux-symptom association. Results are reported in percentages and associations were examined with chi-square and fisher tests in SPSS 22.

Results The majority of patients were females (45 F, 4 M, mean age 40 years for JHS vs 16 F, 9 M, mean age 48 years for controls). Dysphagia was the most prevalent symptom in JHS (70% vs 40% in controls, $p<0.005$), followed by reflux (50% vs 80% in controls, $p<0.01$) and epigastric pain (41% vs 60%, $p>0.05$). Oesophageal hypomotility was present in 41% of patients with JHS and 48% of controls ($p>0.05$). A hypotensive lower oesophageal sphincter (LOS) was described in 21% of patients with JHS vs 44% of controls ($p<0.05$). MII-pH recordings showed that 18 patients with JHS (40%) had excessive acid reflux, 9 (20%) had a hypersensitive oesophagus, and 18 (40%) had functional heartburn vs 13 control patients (52%) had excessive acid reflux, 3 (12%) had a hypersensitive oesophagus and 9 (36%) had functional heartburn ($p>0.05$). Dysphagia, heartburn, and excessive acid reflux exposure were not more frequent in patients with JHS and oesophageal hypomotility ($p>0.05$).

Conclusion This is the largest cohort of patients with JHS investigated with oesophageal physiology techniques for assessment of upper GI symptoms. Dysphagia was disproportionately reported by patients with JHS. However, this was not associated with an increased prevalence of oesophageal hypomotility. Correlation between symptoms and oesophageal physiology abnormalities is poor and suggest visceral hypersensitivity or hypervigilance may play a role in symptom generation.

Disclosure of Interest None Declared