advantageous in infected SSc patients, but further controlled studies are necessary for final conclusion.

**Comments** In this study which I would like to present, we evaluated the prevalence of *H pylori* infection in a population of SSc patients, in order to determine whether there is a possible link between this bacterium and disease severity and activity in SSc patients. This study is a part of my Ph.D. thesis 'Correlation between *Helicobacter pylori* infection and systemic sclerosis activity and severity'.

## 8 ERADICATION OF *HELICOBACTER PYLORI* REDUCE DISEASE ACTIVITY AND SEVERITY IN SYSTEMIC SCLEROSIS

Mislav Radić,<sup>1</sup> Dušanka Martinović Kaliterna,<sup>1</sup> Damir Bonacin,<sup>2</sup> Josipa Radić,<sup>3</sup> Damir Fabijanić<sup>3 4</sup> <sup>1</sup>Department of Rheumatology and Clinical Immulogy, University Hospital Split, Split, Croatia; <sup>2</sup>Department of Gastroenterology, University Hospital Split, Split, Croatia; <sup>3</sup>Department of Nephrology, University Hospital Split, Split, Croatia; <sup>4</sup>Department of Cardiology, University Hospital Split, Split, Croatia

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**Background** *Helicobacter pylori* (*H pylori*) is suspected to be one of the factors triggering systemic sclerosis (SSc). Many reports have emerged describing the pathogenetic potential of *H pylori*. Data on the possible role of *H pylori* are lacking.

**Objective** The aim of this study was to assess the effect of *H pylori* eradication in SSc patients.

Methods Forty-two SSc patients without dyspeptic symptoms were recruited (38 women and 4 men, mean age 54.3±13.6 years, median disease duration 6 years with minimum-maximum range 1–43 years) – 26 were *H pylori*-positive and 16 were *H pylori*-negative on the basis of invasive test. The same operator, blind to clinical features, performed upper gastrointestinal endoscopy in all patients. All infected patients were treated successfully. The authors evaluated the disease severity using clinical and laboratory parameters according to a modified Medsger severity scale at baseline and every 6 months during 18 months, and compared the variations in the two subgroups. The level of SSc activity was evaluated before eradication and during a 18 months follow-up period in the similar time intervals. This study was conducted in accordance with the Declaration of Helsinki and its revisions, and the protocol was approved by the Ethics Committee of University Hospital Split. All patients gave informed consent to participate in the study.

**Results:** After 18 months, *H pylori*-eradicated SSc patients differed significantly (p<0.05-0.001) from patients without *H pylori* infection in terms of improvement of skin, muscle and peripheral vascular involvement. At the same time point several laboratory findings (erythrocyte sedimentation rate, antitopoisomerase I antibodies) ahowed significantly lower values (p<0.05) in the *H pylori*-eradicated subgroup compared to the *H pylori*-negative subgroup. *H pylori*-eradicated SSc patients showed progressive improvement over time (p<0.01) of disease severity and activity compared with baseline, whereas *H pylori*-negative SSc patients remained substantially unchanged.

**Conclusion** Our data suggest that H pylori infection is implicated in the SSc pathogenesis and that its eradication may induce a significant impovement of disease severity and activity over 18 months. H pylori eradication seems to be