Dyspepsia Refractory to Conventional Measures in Patients with Rheumatoid Arthritis: Don’t forget Helicobacter Pylori

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Sir,

Gastrointestinal tract (GIT) manifestations may be the initial or late presentation of systemic autoimmune disorders, but they may also be the complication of treatment. Rheumatoid arthritis (RA) does not affect the GIT directly but decreased lower oesophageal sphincter tone, hiatus hernia, chronic atrophic gastritis, collagenous colitis, amyloidosis (secondary) of the GIT, and mesenteric vasculitis were reported.1

I would like to share my experience of three patients with RA encountered in recent past. The patients developed gradual onset of dyspepsia, leading to self-reduction of treatment in two and discontinuation of treatment in one of them. The parameters of patients are mentioned in Table 1.

All possible conventional measures like proton pump inhibitor (PPI) in maximum dose, change of methotrexate (MTX) to subcutaneous route, increase in dose of folic acid, trial of glucocorticoids before MTX, splitting the dose of MTX and use of cox-2 selective molecule were tried.[2] However, during 6 weeks period even after all measures patients’ dyspeptic symptoms were not improved. So, patients were advised upper GIT endoscopy, which showed severe pan-gastritis in all 3 with Los Angeles grade-A esophagitis in 1 patient. All patients were rapid urease test (RUT) positive for Helicobacter pylori (HP) and given HP kit for 14 days. They were improved dramatically and dyspeptic symptoms almost subsided. Now they were able to tolerate oral treatment. All patients showed improvement in disease activity from moderate to mild after 12 weeks.

HP colonizes the stomachs of over half of the world’s human population throughout their lifetimes and is the main risk factor for various gastrointestinal diseases, ranging from chronic active gastritis without clinical symptoms to peptic ulcer disease (PUD), gastric adenocarcinoma, and gastric mucosa-associated lymphoid tissue lymphoma. Disease outcome is the result of the complex interplay between the bacterium, host and environment.3 The pattern of gastric inflammation is associated with disease risk: antral-predominant gastritis is linked with duodenal ulcer, whereas pan-gastritis is linked with gastric ulcer and adenocarcinoma.4 The RUT has a low sensitivity but high specificity and therefore, it can be used as the first choice test for the diagnosis of HP. Additional biopsies and histological examination should be done during endoscopy when RUT is negative.3

Although RA patients have an increased risk of developing PUD, it is not clear that this is directly related to an increased prevalence of HP infection.6 However, few small studies suggested even clinical improvement in RA symptoms after eradication of HP; many others have been unable to corroborate these findings.7

Overall, the data regarding the association of HP infection with the onset or severity of RA and increased risk of dyspepsia or PUD remains unclear.7 However, the experience of just three patients does not provide any conclusion but the observation which I experienced showed that dyspepsia refractory to conventional measures in patients with RA, the possibility of HP infection and its eradication should also be kept in mind. This observation also suggests that well-designed trials with large number of patients may be helpful to determine that whether this particular RA and HP correlation has any clinical significance or not, which may help in routine clinical practice.

References