HH.MED.14

PREVALENCE OF HELICOBACTER PYLORI INFECTION IN A SAMPLE OF PATIENTS WITH CHRONIC GASTRIC ANTRAL INFLAMMATION

S. M. Buharideen[1], K. B. Galketiya[2], H. M. S. R. B. Kotakadeniya[2], A. K. B. B. T. B. Samarasinghe[2], S. P. M. Peiris[2], A. Dharmapala[2], F. Noordeen[3], T. R. D. S. K. Tennakoon[1], S. M. Wickramasingha[1], S. Wijetunge[1]

[1]Department of Pathology, [2]Department of Surgery, [3]Department of Microbiology, Faculty of Medicine, University of Peradeniya

Gastric Helicobacter pylori (H. pylori) infection is almost always associated with chronic inflammation. However, other causes of chronic gastritis, apart from autoimmune gastritis confined to the body and rare causes such as gastric Crohn's, are not well documented. Nevertheless, chronic antral inflammation not associated with H. pylori organisms is not an uncommon finding. The following study was conducted to determine the prevalence of H. pylori infection in gastric antral biopsies with chronic inflammation in a sample of patients presented with dyspeptic symptoms.

This is a cross sectional study of 103 patients referred for upper gastrointestinal endoscopy due to dyspeptic symptoms and detected to have endoscopically visible gastric antral inflammation during March to December 2012. All patients underwent endoscopic gastric biopsy according to a protocol, that included four biopsies from the antrum and one each from incisura angularis and the body. The one minute rapid urease test was performed on an antral biopsy. The other biopsies were processed for routine histology, toluidine blue stain and immunohistochemistry. The following pathological changes were assessed: chronic inflammation, acute inflammation, atrophy, intestinal metaplasia, dysplasia and H. pylori organisms. The severity of each change was graded according to the Sydney grading system.

There were 30 (29.1%) cases with histologically confirmed chronic inflammation; 20, 9 and 1 cases with the Sydney grade 1, 2 and 3 inflammation respectively. None had intestinal metaplasia, atrophy or dysplasia. H. pylori infection was present in 4/30 (13.3%) cases. The prevalence of H. pylori infection in Sydney grade 1, 2 and 3 chronic inflammation were 0%, 33.3% and 100% respectively. The 4 cases with H. pylori infection showed positivity with routine histology, Toluidine blue stain and the one minute rapid urease test and only two of them showed positivity with immunohistochemistry. There were 5 additional cases that showed positive results with the one minute rapid urease test only. In 73 (70.9%) no inflammation was present and none of these cases had H. pylori organisms by routine histology, special stains or immunohistochemistry.

Sydney grade 1 chronic inflammation does not appear to be associated with H. pylori infection. Only 40% of the cases with Sydney grade 2 or 3 chronic inflammation were associated with H. pylori infection and the likelihood of detection of H. pylori appears to increase with increasing severity of chronic inflammation. Causes for H. pylori negative chronic inflammation were not clear and need further investigation.

Funding: National Science Foundation, grant RG/2011/HS/11.