Prevalence of Helicobacter pylori infection in central serous choroidoretinopathy

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Abstract

Purpose: To assess prevalence of Helicobacter pylori (H. pylori) infection in patients of central serous chorioretinopathy (CSCR) along with the effect of anti H. pylori pharmacotherapy on the course of CSCR. Materials and methods: Fifty consecutive cases of CSCR (diagnosis based on clinical examination and digital fundus angiogram). All of them along with 50 age and sex matched volunteers (as control) underwent upper gastrointestinal endoscopy followed by antral biopsy. The biopsy samples were stained to detect H. pylori urease. Urease positive cases were divided into two subgroups. One group received three drug anti H. pylori pharmacotherapy (lansoprazole 30mg twice a day, clarithromycin 250mg twice a day and tinidazole 500mg twice a day daily for ten days) while the other subgroup was placed on placebo therapy. Both the groups were followed up weekly for two months then two weeks thereafter till complete resolution of ocular condition. The patients who receive triple drug therapy for H. pylori were also subjected to repeat upper gastrointestinal endoscopic biopsy followed by rapid urease test to confirm eradication of the organism one month after completion of therapy. Results: Out of 35 patients (31 male and 4 female) who underwent upper gastrointestinal endoscopy with antral biopsy with rapid urease test for H. pylori, 26 tested positive (74.28%, 60% in control group) for H. pylori and 9 (25.71%, 40% in control cohort) were negative. Of the 43 male patients, 12 did not allow endoscopy. Of the remaining 31 male, 23 (74.19%; 95% confidence interval: 59%-90%) were positive for H. pylori. Of the 7 female patients, 3 did not allow endoscopy and out of remaining 4 cases 3 (75%) tested positive for H. pylori. Conclusion: Infection of H. pylori in gastric mucosa is a risk factor of central serous chorioretinopathy.

Keywords: Helicobacter pylori; Gastritis; Central serous chorioretinopathy.

Central serous choroidoretinopathy (CSCR) is an important cause of central vision loss specially in young male patients¹². Though the exact cause of hyper permeability of choroidal vasculature resulting in fluid exudation and subsequent serous detachment of retina remains largely unknown³. Many theoretical risk factors like type A personality, gastritis and gastric ulcer had postulated over the years⁴⁵. Helicobacter pylori is a proven pathogen causing gastritis and gastric ulcer⁶. Against this background, this study had been undertaken to find out prevalence of H. pylori in patients of CSCR along with the effect of anti-Helicobacter pylori treatment on the course of CSCR.

Materials and methods:

Fifty consecutive patients of CSCR attending Institutional retina clinic were selected for the study. Diagnosis of CSCR was based on history and complete ocular examination including examination of macula by 78D condensing lens, indirect ophthalmoscopy, digital fundus angiogram (DFA) and fundus photography. Written informed consent was taken from each of the participants of the study with permission from Institutional Ethics Committee. All the recruited patients along with healthy age and sex-matched control cases (volunteers from hospital staff and relatives of patients) were subjected to upper gastrointestinal endoscopy with Olympus endoscope with gastric antral biopsy by a single surgeon. The biopsy samples were subjected to rapid urease test with in-house standardized reagent containing 10% urea and phenol red indicator at pH 6.8. A change of color from yellow to red within one hour was taken to be positive signifying presence of H. pylori urease within the biopsied tissue.

The biopsy positive cases were randomly divided into two groups. The first group was treated with standard three drug regimen of lansoprazole 30mg twice a day, clarithromycin 250mg twice a day and tinidazole 500mg twice a day daily for ten days. The other group was placed...
on placebo therapy. Both the groups were followed up weekly for two months, two weekly thereafter till complete resolution of ocular condition. The patients who receive triple drug therapy for H. pylori were also subjected to repeat upper gastrointestinal endoscopic biopsy followed by rapid urease test to confirm eradication of the organism one month after completion of therapy.

Results:

Out of 50 consecutive patients of CSCR, 43 were male and 7 were female (M: F=6.14:1). Age of the patients ranged from 24 years to 58 years for male (mean age 38.7yrs, SD=8.351) and the same in female was from 31yrs to 50yrs (mean 43yrs, SD=7.438). Two had smoketrack pattern of leakage in DFA(4%) and 48 had inkblot pattern of leakage(96%). Out of these 48 patients, 2 had multifocal CSCR(4.17%) and the remaining 46 had a single area of dye leakage in DFA(95.83%). Out of these 46 patients, 2 had multifocal CSCR(4.17%) and the remaining 46 had a single area of dye leakage in DFA(95.83%). Out of these 48 patients, 2 had multifocal CSCR(4.17%) and the remaining 46 had a single area of dye leakage in DFA(95.83%). 15 patients(12 male and 3 female) declined to undergo upper gastrointestinal endoscopy. Out of 35 patients(31 male and 4 female) who underwent upper gastrointestinal endoscopy with antral biopsy with rapid urease test for H. pylori, 26 tested positive(74.28%) for H. pylori and 9(25.71%) were negative. The 95% confidence intervals for H. pylori positivity were found to be 60% to 89%. Out of 43 male patients, 12 did not allow endoscopy. Of the remaining 31 male, 23(74.19%; 95% confidence interval: 59%-90%) were positive for H. pylori. Of the 7 female patients, 3 did not allow endoscopy and out of remaining 4 cases 3(75%) tested positive for H. pylori. Out of 35 controls who underwent endoscopy, 21 tested positive rapid urease test (60%; 95% confidence interval: 44%-76%) and 14(40%) tested negative. Chi-square test was performed and a value of 1.62 was obtained which was less than 3.841, the critical value of chi-square at 5% level of significance. Of the 47 subjects who tested positive for H. pylori, 26(55.32%) had CSCR, whereas in the 23 subjects tested negative for the organism 9 had CSCR(39.13%). When the prevalence of H. pylori infection was compared to the prevalence in an age-matched control population south-eastern France, It was found to be significantly higher(p<0.05%). Possible role of H. pylori in CSCR had been supported by Ahnoux-Zabsonre et al 11. This was later supported by Giusti C who had postulated a Helicobacter pylori-dependent immune mechanism based on a ‘molecular mimicry’ between pathogenic antigens expressed on the bacterium and homologous host protein in endothelial vascular wall12,13.

Discussion:

Helicobacter pylori had been implicated as a causative factor for gastric ulcer. Katelaris et al however, did not find any relationship between dyspepsia and H. pylori induced gastritis7. Mishra et al found a high prevalence of H. pylori amongst asymptomatic healthy Indians8. Alaganantham et al reported a high seroprevalence of H. pylori in urban (Chennai), educated and upper class population similar to Western studies9.

The H. pylori positivity among male in our study was 74.19%. This was similar to the study conducted by Mauget-Faysse et al (70%). Similarly the H. pylori positivity among female in our study (75%) was higher than this study (33%)10.

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