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Helicobacter Pylori Colonization in Patients with Chronic Renal Failure

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Abstract

Chronic Renal Failure – End Stage Renal Disease is a multisystem disorder and affects all the systems in the body. 50 consecutive patients attending nephrology department of King George hospital with CRF were studied. All the patients CRF or those patients who attended the predialysis clinic were recruited. The patients were judged infection with helicobacter pylori if the urease slide test was positive or histopathological examinations showing helicobacter pylori bacilli. Out the total 23 helicobacter pylori positive patients only 18 patients 78% were culture positive. ESRD gastric colonization of helicobacter pylori is not more frequent than usual. Helicobacter pylori correlate positively with duodenal ulcer, gastric erosions. We recommend helicobacter pylori treatment before renal transplantation only in those patients with endoscopic duodenal ulcer or erosions but not as a routine. Helicobacter pylori colonization in duodenal ulcer patients with chronic renal failure is high when compared to non-ulcer chronic renal failure. Prevalence of helicobacter pylori is similar in chronic renal failure patients with duodenal ulcer to control group of duodenal ulcer patients. Helicobacter pylori prevalence in non ulcer dyspeptic chronic renal failure patients is higher than Non-ulcer dyspeptic controls.

Key words: Chronic Renal Failure, End Stage Renal Disease and Helicobacter pylori

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Introduction

Chronic renal failure is a clinical syndrome due to persistent dysfunction leading to excretory, metabolic and synthetic failure, culminating in accumulation of non-protein nitrogenous substances which results from progressive and irreversible distribution of nephrons regardless of the cause. [1] The diagnosis of end stage renal diseases (ESRD) implies that glomerular filtration rate is known to have been reduced to at least 5ml/min over a period of 3-6 months.[2] With recent advances in imagiology, biochemistry and pathology, the diagnosis, estimation of severity and prediction of prognosis of a ESRD has become better and easier.[3] Chronic glomerular nephritis in its several forms is the commonest cause in the past.[4] In western countries, Diabetes mellitus and systemic hypertension are the new leading causes of CRF unfortunately in India with the absence of national statistics, the magnitude of CRF and the causation factor is difficult to establish.[5] However, the available data suggest Chronic glomerular- nephritis is the commonest cause followed by diabetes mellitus and systemic hypertension. CRF – ESRD is a multisystem disorder and effects all the systems in the body such as fluid and electrolyte imbalance, endocrinal and metabolic disorders, gastrointestinal disorders, cardio-vascular and pulmonary disorders, skin manifestation, neurovascular changes, haematological and immunological disorders. In the

absence of national registry, the magnitude of CRF and its etiology is difficult to determine. Recently 2 studies from India have addressed this issue.[6]

Helicobacter pylori is a spiral shaped bacterium which measures 2.5 cm in length and 0.5 in diameter. It has a smooth coat and has 1-5, usually 3 unipolar sheath flagellae with terminal bulbs. Helicobacter pylori is a strong producer of the enzymes, oxidase, catalase and urease. The high urease activity forms the basis of a test for rapid identifications of the organism. Helicobacter pylori as a gastric pathogen is dependent on virulence (maintenance) factors and pathogenic mechanism virulence factors are those that allow helicobacter pylori to survive in the hostile environment of gastric lumen. Helicobacter pylori was isolated form antral mucosal biopsy specimen in 41% patients, 82 % out of 50 patients of coastal Andhra Pradesh.

Materials and Methods

50 consecutive patients attending nephrology department of king George hospital with CRF were studied. All the patients CRF or those patients who attended the predialysis clinic were recruited. A detailed history was recorded and a thorough clinical examination performed. No patients had ever been treated with bismuth and none had received antibiotics during the 4 weeks prior to investigations. Serum samples were analyzed for CRF and all efforts were made to establish the etiology of CRF. Serum creatinine was measured using standard method and all patients were asked to complete a standard dyspepsia questionnaire and all medications given were noted. These subjects were admitted to the hospital for investigations and upper GI endoscopy (with biopsy) was performed after informed consent.

All endoscopies were performed by one investigator in the morning following an overnight fast. An Olympus GIF 20 gastro duodenoscope was used and three biopsies each were obtained from the antrum. The endoscopist was unaware of the subject's symptoms. One biopsy specimen from antrum was immediately inoculated in the biopsy urease test medium and other specimen was sent in isotonic saline to which trimethoprim in the concentration of 5mg/lt was added for histologic examination. The biopsy forceps was cleaned with glutaraldehyde solution (cidex) followed by rinsing with water, between biopsies in the same subject and the same procedure was adopted between the patients. The biopsy specimens were then immediately processed within 2 hours in the laboratory. The specimens were subjected to rapid urease test, direct microscopic examination and culture.

The biopsy specimen was added to the glucose broth. 48 hours later, the culture was tested to detect the presence of helicobacter pylori. Patients were judged to be infected with helicobacter pylori if the organism was demonstrated in the antral biopsy by either microscopy or rapid urease positive. Comparative analysis was done on patients without peptic ulcer who had dyspeptic symptoms and with non ulcer dyspepsia controls who were investigated for Helicobacter pylori infection in the same institute. We have included both the sexes above 12 years of age, chronic renal failure of various etiologies and Indication for dialysis. Patients with gastric outlet obstruction, history of previous ulcer surgery and history of intake of drugs which are known to effect helicobacter pylori were excluded from this study. Also post renal transplant patients and acute renal failure patients were excluded from this study.

Result

50 consecutive patients with the CRF (Male 29 and Female 21) who attended nephrology unit king George hospital were studied. All the patients were subjected to complete dyspepsia questionnaire. All the subjects were submitted for upper gastrointestinal endoscopy. Antral biopsies were taken irrespective of their GI symptoms. All the biopsies were sent to microbiology department immediately for identification of helicobacter pylori infection.

The patients were judged infection with helicobacter pylori if the urease slide test was positive or histopathological examinations showing helicobacter pylori bacilli. Out of the total 23 helicobacter pylori positive patients only 18 patients 78% were culture positive. The age of the patients' range from 17 to 70 years. Renal failure was more predominant in the middle years between 3rd and 5th decade. There was an increased prevalence of helicobacter pylori as the age advances in CRF patients. There was neither significant difference in the distribution of the sexes, nor in the type and duration of CRF. Upper GI symptoms were found in 29 of the total 50 consecutive CRF patients 58%. Helicobacter pylori was found in 19 of the 29 patients who had upper GI symptoms giving a positivity rate of 65%. Asymtomatic CRF patients (UGI symptoms) had very low positivity for helicobacter pylori infection (n=21, 19%).

Endoscopic appearance of duodenal ulcer was present in 6 CRF patients' 12% erosion in 8 patients 16% and non-erosive antral gastritis in 7 patients 14 %. Endoscopic evidence of mucosal lesions was found in 21 patients 42% out of the 50 consecutive CRF patients. Duodenal ulcer was found in 6 patients and 5 of them were found to have helicobacter pylori positivity giving a prevalence of 83%.

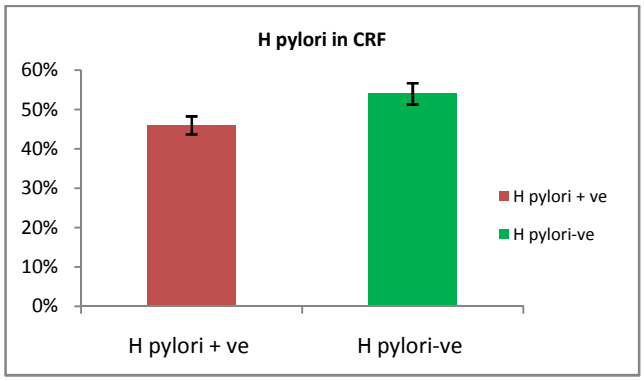

Figure 1: Helicobacter pylori in Chronic renal failure.

There was no significant difference of helicobacter pylori infection in both pre-dialysis and dialysis group of patients. Only 10 subjects of CRF were subjected for dialysis. Male patients needed dialysis treatment more often than their female counterparts. The mean serum creatinine in helicobacter pylori positive patients was 7 mg/dl (4-13) in conservative treatment group of CRF patients. The mean serum creatinine value in helicobacter pylori negative conservative treatment group of CRF is 7.3 mg/dl (3-15). This is in contrast to dialysis group of patients who were helicobacter pylori positive the mean serum creatinine value was 10.5mg/dl (8-16) and in helicobacter pylori negative patients the mean serum creatinine was 8 mg/dl (4-11). There is no association whatsoever between etiological factor of CRF and prevalence of helicobacter pylori. The present study has shown helicobacter pylori prevalence 46%in 50 consecutive non-transplant CRF patients.

Discussion

There has been little research directed towards helicobacter pylori CRF in India. Helicobacter pylori infection in India, as in the under developed countries, is widespread, probably is a consequence of poor hygiene. All the three methods are employed in the present study to establish the diagnosis of helicobacter pylori infection. However because of the low (72- 92%) sensitivity of culture described in the literature we have considered helicobacter pylori positivity based on histopathologic examination and rapid urease test. In the present study 78% of helicobacter pylori positive chronic renal failure patients were culture positive. This was similar to the reports available in the world literature.

Despite the high gastric juice urea levels in patients with chronic uremia diagnostic test based on helicobacter pylori high urease activity remain reliable. In the present study an effort was made to determine whether urea influence the presence of helicobacter pylori in the stomach and the incidence of this infection was determined in patients with CRF who received regular dialysis. There was no significant difference among the dialysis patients, conservative management group of patients and also historical controls. Though urea was considered as a preferred metabolite but there was no proof that high levels of urea influence the occurrence of the organism in the stomach. A higher urea level in the blood in patients with CRF does not seem to be associated with Helicobacter pylori infection. This is in concordance with Gladziwa, U et al, [7] who has shown that urea levels in the blood and gastric juice of patients with renal failure do not seem to be a risk factor for infection with helicobacter pyloril. Tokushima et al. observed helicobacter pylori positivity in 66% of dialysis group patients i.e. in accordance with present study (50% helicobacter pylori positivity)

Upper gastro intestinal mucosal lesions have been one of the most common complications in patients with CRF. Quite few reports are available regarding the prevalence of helicobacter pylori and its influence on the upper GIT. A high frequency of duodenal ulcer up to 56% has been reported in patients with CRF undergoing dialysis. However, an increased frequency of peptic ulcer CRF patients and patients undergoing hemodialysis was not confirmed in studies from Sugimoto, Mitsushige, et al.. [8] In the present study GI symptoms were elicited in 29 out of 50 CRF patients giving a prevalence of 58%. This was in contrast to Devenport et al, who reported symptomatic dyspepsia in only 18% of the CRF group. Similar results 22% were reported by Offerhaus et al for chronic dialysis patients, reported higher incidence of dyspeptic symptoms similar to the present study. Tseng, GY., et al. [9]The major reason for higher of prevalence of symptomatic dyspepsia was due to higher positivity rate of

helicobacter pylori in general studies showing high infection rate in general population in India.

The prevalence of Helicobacter pylori in this study was increased in symptomatic dyspeptic subjects and low in those CRF patients who have no upper GI symptoms. This study is correlation with Malfertheiner et al, [10] who has shown that symptomatic dyspeptic patients have high prevalence Helicobacter pylori antibodies. Helicobacter pylori positive patients had significantly more frequent upper GI symptoms than Helicobacter pylori negative individuals. Presence of helicobacter pylori correlates positively with duodenal ulcer, upper GI symptoms and gastritis but negatively with dialysis, aetiology of CRF and sex of the CRF patients. There is a considerable disagreement concerning prevalence of peptic ulcer of CRF compared to the normal population. Duodenal ulcer described association of peptic ulcer with an advanced kidney disease. However recent studies have shown incidence of ulcer disease to range from 10 to 60 %. It is difficult to compare these studies with the present study because of the method of investigation and definition of ulcer disease. We prefer fiber optic upper GI endoscopy as investigation of choice.

In the present study duodenal ulcer was found in 12% of patients this is in concordance with world literature. Helicobacter pylorus was found in 5 out of these 6 cases. The high incidence of peptic ulcer in CRF patients compared to general population is probably because of the higher gastrin levels and high serum PGI levels. The presence of Helicobacter pylori infection in 46% of our CRF patients was similar to the prevalence observed 46% also the prevalence observed 52% in healthy subjects without dyspepsia and similar to the reported prevalence in NUD patients without CRF from our centre 40%. The prevalence of 44% reported by offerhouse etal but is higher than the 34% described by Reicher, Sofiya, et al. [11] This studies relied on serological test for helicobacter pylori antibodies a method of diagnosis slightly less sensitive than urease based tests.

In this study there was no statistical difference in prevalence of helicobacter pylori those patients with dialysis dependent CRF 50% and the conservative group of CRF 45%. The bottom line, of course, is a debate on whether eradication of helicobacter pylori in CRF is desirable in India. Many studies have shown high infection rates in general population in India.

Conclusion

We conclude that in ESRD gastric colonization of helicobacter pylori is not more frequent than usual. Helicobacter pylori correlate positively with duodenal ulcer, gastric erosions. We recommend helicobacter pylori treatment before renal transplantation only in those patients with endoscopic duodenal ulcer or erosions but not as a routine. In ESRD the gastric colonization of heilicobater pylori is not more frequent than usual. The prevalence of helicobacter pylori in 50 consecutive non-transplant chronic renal failure patients is 46%. CRF patients who are dialysis dependent showed a similar prevalence of helicobacter pylori is with conservative group of patients. The prevalence of helicobacter pylori is high in symptomatic dyspeptic End Stage Renal Failure patients when compared to non symptomatic chronic renal failure patients. The point prevalence of duodenal ulcer is high in chronic renal failure patients. Helicobacter pylori colonization in duodenal ulcer patients with chronic renal failure is high when compared to non-ulcer chronic renal failure. Prevalence of helicobacter pylori is similar in chronic renal failure patients with duodenal ulcer to control group of duodenal ulcer patients. Helicobacter pylori prevalence in non ulcer dyspeptic chronic renal failure patients is higher than Non-ulcer dyspeptic controls.

Conflict of interest: None declared

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