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A Study of Uppergastrointestinal Mucosal Abnormalities in Chronic Renal Failure

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Abstract: *Chronic Renal Failure(CRF) is said to be a risk factor for peptic ulceration. CRF patients do get anorexia, nausea, vomiting and abdominal discomfort. Helicobacter Pylori(H.Pylori) has a close association with development of peptic ulcer disease and could be one of the risk factors for Upper Gastrointestinal (UGI) disturbances in CRF patients.Aim of this study was to assess the UGI mucosal changes in CRF patients and to examine whether H. Pylori infection was involved in the pathogenesis. The study was conducted in 30 CRF patients of both genders, in the age group ranging from 20 – 70 years. After overnight fasting, UGI Endoscopic evaluationand antral biopsy was done. Tissue samples were examined for H. Pylori infection by Rapid Urease Test(RUT) and also stained for HistopathologicalExamination (HPE) using Giemsastaining.Statistical analysis of the data was done using student t test and Chi square test. In this study, 42% of CRF patients had dyspepsia and its occurence increased with degree of uraemia. 57% ofCRF patients had abnormal endoscopy findings,77% had histological abnormality and 43% had H.Pylori infection.UGI mucosal abnormality increased with increasing age and degree of uraemia. Presence or absence of dyspepsia had no significant association with the UGI mucosal abnormalities. The increased risk of post-transplant ulceration and bleeding, justifies detailed pre-transplant UGI endoscopic evaluation in all CRF patients. Appropriate management thereafterwill prevent post-transplant complications and hence reduce the morbidity and improve their quality of life.*

Keywords*: Chronic Renal Failure, Helicobacter Pylori infection, Upper Gastro Intestinal mucosal changes.*

I. Introduction

Chronic Renal Failure (CRF) or End Stage Renal Disease (ESRD) is the final stage of slowdeterioration of kidneys.Renal failure is said to be a risk factor for peptic ulceration[1]. Nausea, vomiting, persistent anorexia and dull gnawing stomachache are some of the symptoms pertaining to Upper Gastro Intestinal (UGI) tract, apart from other symptoms in CRF patients[2]. Peptic ulceration and gastrointestinal tract bleeding are responsible for significant morbidity and mortality in CRF patients.Maliket.al,had observed no association between the severity of CRF &UGI lesions[3].Few other studies also had reported similar findings[4,5,6.]

After the discovery of Helicobacter Pylori (H.Pylori) by Dr.Barry Marshall and Dr.Robin Warren in 1980s, peptic ulcers are believed to be related more to the presence of H.Pylori infection rather than to stress and stomach acidity. H.Pyloriare passed in the stool and the infection spreads via contaminated food and water.Its mechanism of action is by production of ammonia by urease enzyme,which causes ionic changes in themucosal layer with a consequent back leak ofhydrogen ions in the mucosa.H.Pylori also leads to degradation of mucus by protease, activates inflammatory cells by producing toxins and triggers an autoimmune response by producing antigens that cross react with antral gastric antigens.Endoscopic biopsy and histological examination is the "gold standard "for diagnosis of H.Pyloriinfection.Few studies had reportedH.Pylori infection in CRF patients[7].

Aim &Objectives

To assess the Upper gastrointestinal mucosal abnormalities in Chronic Renal Failure patients and to examine whether Helicobacter Pylori is involved in the pathogenesis.

To analyse their association with age, dyspepsia and degree of uraemia.

II. Materials And Methods

This study was conducted in Government Kilpauk Medical College, Chennai.30 CRF patients inclusive of both genders in the age group ranging from 20 – 70 years were included in the study.Institutional Ethical Clearance was obtained.After getting written informed consent from all the subjects, detailed history was elicited and clinical examination was done. Subjects with history of chronic drug intake such as Non-SteroidalAnti Inflammatory drugs (NSAID) and steroids, with history of smoking, alcoholism, previous gastric surgery and other chronic diseases were excluded. Complete Blood Count, Blood Urea & Serum Creatinine estimation were done.

After overnight fasting, Upper gastrointestinal endoscopy using Pentax EPM 3300,flexible video oesophago gastro duodenoscope, was done.The mucosal lining of oesophagus,stomach and duodenum were studied. Antral biopsy was taken in all the subjects. Tissues were examined for the presence of Helicobacter Pylori by Rapid Urease test(RUT) and also stained for histopathological examination (HPE). RUT detects the presence of urease in the biopsy tissue,an enzyme produced abundantly by H. pylori. 1 ml of 10% urea broth in distilled water plus one drop of 1% phenol red as an indicator was prepared and used to detect the presence of H Pylori infection in the tissue specimen.A portion of the tissue specimen was put into the prepared Urea solution with the indicator, that detects the pH change.If H Pylori is present in the biopsy tissue, urea will be broken down to ammonia and carbon dioxide , leading to an increase in pH and a change in colour due to the pH indicator in the test. Colour change in the medium to pink within few minutes to 2 hours is taken as positive result. Rapid Urease tests are used for presumptive identification of H. Pylori in tissue specimens.HPE of the tissue specimen using Giemsa staining was done to identify the actual organism, H. Pyloriand to study the histopathological changes[7].

III. Results

The statistical analysis of the data was done using student t test and Chi square test.

Table 1: Summary statistics of the study subjects

CRF (n=30)		
Dyspepsia	Present (n=14)	42%
	Absent (n=16)	68%
UGI scopy	Normal (n=13)	43%
	Abnormal(n=17)	57%
Histopathology	Normal (n=7)	23%
	Abnormal(n=23)	77%
H.Pylori	Positive (n=13)	43%
	Negative(n=17)	57%

Table 2: Association of dyspepsiaand serum creatininein CRF

CRF (n=30)		Creatinine(mg %) Mean ±SD	T test
Dyspepsia	Present (n= 14)	6.48±3.38	0.020*
	Absent (n=16)	4.17 ±1.53	

*P value < 0.05 statistically significant

The Mean Creatinine level was significantly higher in dyspeptic CRF patients.

Table 3:Associationof histopathological abnormalitywith age in CRF

	Findings	Age(yrs)Mean ±SD	T test
Histopathological Examination (HPE)	Normal (n=7)	32.57±10.61	0.006*
	Abnormal(n=23)	47.70±12.08	

*P value < 0.05 statistically significant

Mean age of CRF patients with histopathological abnormality was significantly higher than those with normal findings.

Table 4:Associationof blood Urea &Creatininewith UGI scopy abnormality in CRF.

CRF (n=30)		Normal UGI scopy	Abnormal UGI scopy	Chi square test
Urea	<75 mgs% (n=16)	63%	37%	0.03*
	≥75mgs%(n=14)	21%	79%	
Creatinine	<5 mgs% (n= 14)	64%	36%	0.03*
	≥5mgs%(n=16)	25%	75%	

* P value < 0.05 statistically significant

Endoscopic abnormality showed significant increase with increased blood urea and creatininelevels .

IV. Discussion

In this study,57% of CRF patients had abnormal UGI endoscopy, 77% had histopathological abnormalities&43% had Helicobacter Pylori infection (Table1). 42% of subjects had dyspeptic symptoms like anorexia, nausea,vomiting and epigastric pain, either separately or in various combinations.This wasconsistent with previous studies[2,4].Commonest GI symptoms reported among CRF patients were anorexia & vomiting[3].Dyspeptic CRF patients showed a significant increase in the creatinine level when compared with non-dyspeptic CRF patients (Table 2).

The pathophysiology behind the UGI disease in CRF patients could be hyperacidity, hypergastrinemia and altered mucosal defence. Hypergastrinemia may be due to decreasedrenal clearance and loss of feedback

inhibition of gastrin release[8]. In addition, serum levels of various other GI hormones involved in modulation of GI motility, GI secretion and regulation of hunger were found to be elevated as a consequence of renal insufficiency.Gastric dysrhythmias and delayed gastric emptying hadbeen reported in CRF patients.Thus, a complex state of GI dysmotilityoccur in CRF[9].These may probably be responsible for the dyspepsia and UGI mucosal changes either by directly affecting the GI smooth muscle or stimulating certain areas of Central Nervous System.

There was no significant association between dyspepsia and UGI mucosal abnormalities in this study.A higher incidence of mucosal inflammation rather than the ulcer disease was observed.Similar observation was reported in earlier studies[4]. Few studies had reported ahigher frequency of peptic ulcer in CRF patients[10].
Mean age of the CRF patients with UGIhistological abnormality was significantly higher in this study(Table3). Similar finding was reported in the study by Verma.et.al[11]. Blood urea &creatinine levels are considered as an index of severity of CRF. This study had observed significant increase in UGI endoscopic mucosal abnormalities with increasing degree of uremia(Table 4).This was consistent with the earlier findings by Ahmed et al[12.]

Limitations of the study
Sample size is small to arrive at a definite conclusion.
Subjects with different duration of illness have to be included in the study.

V. Conclusion

From this study, it is evident that the UGI mucosal inflammatory lesions and H pylori infection are common in CRF patients. Dyspepsia and UGI mucosallesions show positiveassociation with severity of CRF.The UGI lesions in CRF are of crucial importance for the patient's destiny during the course of treatment. There was no significant association between dyspepsia and UGI mucosal abnormalities in this study.Hence , all CRF patients, irrespective of whether they have dyspeptic symptoms or not, need to have a detailed pretransplant UGI endoscopic evaluation &Histopathological examination, including H pylori detection.This will enable appropriate management of the same. To a large extent, this will prevent post-transplant complications and hence reduce the morbidity and improve their quality of life.

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