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Original Research Article

Endoscopic study of upper gastrointestinal mucosal lesion in chronic renal failure

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ABSTRACT

Background: Chronic renal failure is a syndrome complex results from progressive and irreversible destruction of nephrons regardless of cause. This diagnosis implies that GFR is known to have been reduced for atleast 3 to 6 months. To study the clinical manifestations and assess the type and prevalence of upper gastrointestinal lesions and to correlate the clinical and biochemical parameters with upper GI mucosal lesion in chronic renal failure patients.
Methods: Fifty CRF patients from nephrology and Medicine OPD, admitted in the department of Medicine, M.Y. Hospital, Indore, during January 2002 to April 2003 were studied. A detailed History, clinical examination, Urine Examination, renal Function test, U.S.G. was done and then upper GI Endoscopy was performed.
Results: Thirty-six males and fourteen females (n=50) were studied. Most common age group was 41-50 years; commonest GI symptom was Anorexia (100%) and Nausea was present in (94%) of patients and GI bleed was seen in (8%). The common upper GI lesion were gastritis (28%), Oesophagitis (16%) and Duodenitis (12%). No patients had Gastric or Duodenal Ulcers. Majority of patients had creatinine clearance between 5-10 ml /min and most of the patients had duration of disease between 11-20 months.
Conclusions: Gastrointestinal symptoms are very frequent in CRF patients. There is a high incidence of inflammatory charges of gastrointestinal mucosa in patients of CRF and chronic uremic patients are not at high risk of developing ulcer disease. There is no correlation of these gastrointestinal symptoms and inflammatory changes with age, sex, severity and duration of disease.

Keywords: Chronic renal failure, Endoscopic features

INTRODUCTION

Chronic renal failure is a syndrome complex results from progressive and irreversible destruction of nephrons regardless of cause. This diagnosis implies that GFR is known to have been reduced for at least 3 to 6 months. CRF gradually affects all the systems of the body including GIT.

Uremic syndrome in CRF is known to be associated with gross disturbance in GI system included anorexia, nausea, vomiting, epigastic pain, heartburn, hiccups and GI haemorrhage. Peptic ulcer disease is common in uremic patients. GI bleeding occur with greater frequency and higher mortality in uremic patiens.[1,2]

The most common cause of bleeding is peptic ulcer, haemorrhage, oesophagitis, gastritis, duodenitis and gastric telengiectasia. The study of gastrointestinal changes in patients of chronic renal failure is of paramount importance particularly since these patients are subjected to haemodialysis or transplantation. This is because of potential risk of bleeding from diseased mucosa either as an effect of heparinization during

dialysis or with institution of steroids or other immunosuppressive agents.[3]

METHODS

A total of 50 patients (36 males and 14 females) in the age group of 20-70 years attending the nephrology or medicine OPD or admitted in the department of medicine M.Y. Hospital, Indore, Madhya Pradesh, India from January 2002 to April 2003 were studied. Patients were diagnosed as a CRF based on persistent azotemia and signs and symptoms of uremia for >3 months and bilaterally reduced kidney (<9 cm) on USG.

All patients were subjected to detailed history, clinical examination and investigation as per a pre- designed proforma. Patients having H/o acid peptic disease without CRF, drug ingestion, corrosive poisoning and chronic alcoholic were excluded. Upper gastrointestinal endoscopy was done by the fiberoptic upper GI endoscope in the department of gastroenterology (dept. of medicine), M.Y. Hospital, Indore and the details of oesophagus, stomach and 1st and 2nd part of duodenum were noted.

RESULTS

Out of 50 patients studied between January 2002 to April 2003, 36 were males and 14 were females. The age of patients ranges from 20 years to 70 years. Most common age group was 41-50 years.

Table 1: Age and sex distribution.

	No. of males	No. of females	Total
20-30	10	2	12
31-40	7	0	7
41-50	9	9	18
51-60	6	2	8
61-70	4	1	5
Total	36	14	50

Gastrointestinal symptoms were present in all patients studied. Anorexia was the most frequent symptom (100%). Nausea was the second most common symptom (94%) followed by vomiting (72%), hiccups (26%), pain in abdomen (30%) in decreasing order of frequency. Gastrointestinal bleed was present only in (8%) of patients.

The most common upper GI mucosal lesion was gastritis which was seen in 28% of patients. It was mild in 16%, moderate in 10% and severe in 2% of cases. Oesophagitis was the second most common lesion (16%) mild (10%), moderate (6%) and severe in 4%. Other findings in oesophagus was oesophageal ulcer in 3%, leiomyoma and oesophageal varices in 1 patient each. It is followed by duodenitis in 12% patients. No patient had showed gastric or duodenal ulcer. Most of the patients (72%)were in group of creatinine clearance 5-10 ml/min. Rest of the patients had creatinine clearance between 5 ml/min (14%) and 10-15 ml/min (14%). Predominant GI symptoms in all groups were anorexia, nausea and vomiting but there was no significant difference in the incidence of GI symptoms in 3 groups.

Common findings in these groups on endoscopy included oesophagitis, gastritis and duodenitis with no significant difference in 3 groups. On comparing the endoscopic findings in relation to duration of disease value of p> 0.10 i.e. association is highly insignificant.

Table 2: Gastrointestinal symptoms on presentation.

GI symptoms	No. of patients	Percentage
Anorexia	50	100%
Nausea	47	94%
Vomiting	36	72%
Hiccups	13	26%
Pain in abdomen	15	30%
gastrointestinal bleed	4	8%

Patients who underwent both haemodialysis and conservative treatment shared predominantly gastritis in 54.5%, while duodenitis was seen in 27.2% and no patient had shown oesophagitis. Those patients who were kept on conservative treatment alone showed oesophagitis in 25.6% gastritis in 20.5% and duodenitis in 3.69%.

DISCUSSION

Involvement of gastrointestinal tract in chronic renal failure is poorly understood though studies mentioning the gastrointestinal alteration in CRF have been available for more than 60 years, the results have been conflicting. The male patients predominate over the females in present study and the most common age group is 41-50 years. In the present study, GI symptoms are present in all the 50 patients. Anorexia, nausea and vomiting were the commonest symptoms in our study. Nausea and vomiting have been reported as the commonest symptom by Margolis et al.[1] In the Indian study by Kochar R and Goenka M, anorexia, nausea and vomiting were the predominant symptoms.[1,2]

Gastrointestinal bleeding is known to occur in CRF patients. In the present study, GI bleeding is present in 8% of patients and all patients have erosive gastritis. Boyal et al also reported that gastric bleeding sites rather than duodenal ulceration were the most common source of bleeding.[3] Nippon ratio mentioned the mechanism of onset of gastric hemorrhage, mucosal lesion that focuses on blood flow and oxygen supply. Oxygen radicals produced at haemodialysis may participate in the pathogenesis of mucosal hemorrhage. Upper GI mucosal abnormalities are detected in 62% of patients in present study by endoscopy. Inflammatory mucosal lesions were predominant and gastritis being the most common change present in 28% of patients followed by oesophagitis

(16%) and duodenitis (12%). There was no patient of peptic ulcer. Sunder et al in Indian study also reported low incidence of peptic ulcer. Prakash J and Agrawal BK and Toni N et al found that patients with CRF had a high prevalence of inflammatory mucosal changes.[3-5] The difference in the frequency of inflammatory changes in gastrointestinal tract in CRF may be partly due to difference in gastric acid status of different social groups and partly due to subjective variation in the interpretation of various inflammatory changes.

Aetiology of gastrointestinal lesion in CRF may be of multifactorial origin. It included breakage of mucosal defense barrier with back diffusion of hydrogen ion. Mc Leod et al noticed hyperacidity in two out of seven patients Venkateshwaran et al documented increased incidence of hyperacidity and peptic ulceration in patients who were an haemodialysis.[6,7] Shepard et al suggested that hyper gastrinemia was the result of the inability of the diseased kidney to eliminate gastrin from circulation.[10] Tylor et al also showed higher basal gastrin level in CRF patients. Increased calcium has also been implicated in the causation of hyper secretion of gastric acid.[1] Techan considered the role of uremic toxins in GI mucosal changes in CRF.[9]

There is no influence of age and sex on upper GI lesions in patients of CRF. Kochar R et al also observed the similar findings while Benzo et al showed that age was the only predictive variable significant for gastric lesions which were most frequent in older patients.[2,11] In the present study we found that the severity of real disease as determined by creatinine clearance does not affect the GI symptom and upper GI mucosal lesions in CRF. There is no correlation of blood urea and serum creatinine level with the upper GI changes. There is also no correlation of endoscopic changes with the duration of disease. This may be due to the multifactorial etiology of upper GI lesions. Lieber et al observed that decrease in gastric acidity was due to higher ammonia level.[12] Mc Convel et al demonstrated that gastir acid secretion increases progressively during haemodialysis.[13] There is not much difference in the incidence of abnormal findings in dialysis (82and) and non- dialysis (55%). Patients of uraemia had improved capacity to secrete acid due to certain factors leading to gastritis. Haemodialysis by removing these factors allow the manifestation of undergoing hypersecretory state. Gordan et al found no significant change in gastric acid secretion before or after dialysis.[14] Margolis et al and Andrivilli et al also found no relationship between duration of dialysis with the presence or absence of gastrointestinal lesion.[1,15]

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REFERENCES

1. Margolis DM, Saylor JL, Geisse G, DeSchryver-Kecskemeti K, Harter HR, Zuckerman GR. Upper Gastrointestinal disease in Chronic renal failure. A prospective evaluation. Arch Int. Med. 1978;138:1214.
2. Kochar R, Gupta KL, Malik AK, Pereira BJ, Sakhuja V, Mehta SK. Morphological alterations of Upper Gastrointestinal tract in uremic patients Indian J. of Gastroenterology. 1987;7(3):149-50.
3. Boyal JM, Johnston B. Acute Upper Gastrointestinal Hemorrhage in Chronic renal failure. Am J Med. 1983;75(3):409-12.
4. Prakash J, Agrawal BK. Upper Gastrointestinal mucosal lesion in Chronic renal failure. J. Gastroenterology. Indian J Gastroenterol. 1991;10(4):131-2.
5. Tani N, Harasawa S, Suzuki S, Miwa M, Sakita R, Nomiyama T, et al. Lesion of Upper Gastrointestinal tract in patients with Chronic renal failure - Gastroenterologic Japonica. 1980;15(15):480.
6. McLeod LE, Mandin H, Davidman M, Ulan R, Lakey WH. Intermittent Haemodialysis in terminal Chronic renal failure Canan – Med. Assoc. J. 1966;24:318.
7. Venkatesawaran PS, Jeffer N, Hocken AG. Gastric acid secretion in Chronic Renal Failure. Br Med J. 1972;4(5831):22-3.
8. Taylor JL, Sects RA Serum gastrin in patients with Chronic Renal Failure GUT. 1980;21:1062.
9. Techan PE. on the genesis of uremia. American J Med. 1970;48:471.
10. Shepherd AMM, Stewart WK, Wormsley KG. Peptic ulceration in chronic renal failure. Lancet. 1973;13:57.
11. Benzo J, Matos M, Milanes C, Arminio A, Stempel CA. Endoscopic findingsin the upper digestive tracts in patients with terminal chronic kidney failure. G E N. 1994;48(1):34-8.
12. Lieber CS, Lefèvre A. Ammonia as a source of gastric hypoacidity in uremia. J Clin Invest. 1959;38(8):1271-7.
13. Mitchell CJ, Jewell DP, Lewin MR, McLaughlin JE, Moorhead JF. Gastric function and histology in chronic renal failure. J Clin Pathol. 1979;32(3):208-13.
14. Gordon EM, Johnson AG, Williams G. Gastric assessment of prospective renal transplant patient. Lancet. 1972;1(7744):226-9.
15. Andrivilli A, Malfi B. Patients of chronic renal failure are not at risk of developing chronic peptic ulcer clinic nephrology. 1981;23(5):1245.

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