

# Gastric Secretion and Upper Gastrointestinal Endoscopic Findings in End Stage Renal Disease Patients



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## Abstract

**Background:** Impaired bicarbonate secretion is one of the proposed mechanisms for uremic gastrointestinal symptoms. This prospective case-control study was aimed to determine the effects of uremia on gastric acid and bicarbonate secretion in end stage renal disease (ESRD) patients on maintenance hemodialysis (MHD) and to correlate the bicarbonate secretion with morphological changes in the upper gastrointestinal tract and with the presence of *H. pylori*.

**Methods and Materials:** Gastric secretion analysis and upper gastrointestinal endoscopy (UGIE) were performed in 28 patients with ESRD on MHD with symptoms of acid peptic disease and 20 controls with similar symptoms but without evidence of kidney disease. Statistical analysis was done by student t-test and paired t-test.

**Results:** Rate of gastric juice secretion, acid output and volume of parietal cell secretion were significantly less in CKD patients as compared to patients without kidney disease. The non-parietal cell secretion, bicarbonate output and total acid secretion (acid output + bicarbonate output) were also significantly less in CKD patients as compared to controls. The acid bicarbonate output was not significantly different in CKD patients with endoscopic changes on UGIE as compared to CKD patients without significant endoscopic abnormalities.

**Conclusion:** The study indicates that there is a generalized secretory defect in ESRD patients leading to decrease in both acid and bicarbonate secretion. Symptoms of dyspepsia possibly occur because of an imbalance between acid and bicarbonate secretion rather than an absolute change in the amount of either acid or bicarbonate secretion.

## Introduction

Gastrointestinal manifestations of uremia are non-specific and include nausea, vomiting, anorexia, epigastric pain, heart burn, hiccups and gastrointestinal hemorrhage. The pathogenesis of these abnormalities is poorly understood. Proposed mechanisms include increased back diffusion of hydrogen ions across the gastric mucosal barrier, hypergastrinemia, hyperacidity, hyperparathyroidism, *H. pylori* infection and impaired defensive mechanisms like gastric bicarbonate secretion. The present study was undertaken to study the effects uremia on gastric acid and bicarbonate secretion in end stage renal disease (ESRD) patients on maintenance hemodialysis (MHD) and to correlate the bicarbonate secretion with morphological changes in the upper gastrointestinal tract and with the presence of *H. pylori*.

## Patients and Methods

Twenty eight adults with ESRD on MHD with symptoms of acid-peptic disease were included as cases. Control group

comprised of 20 patients with similar symptoms without evidence of kidney disease. After obtaining informed consent, all patients subsequently underwent estimation of gastric secretion and upper gastrointestinal endoscopy (UGIE).

For the purpose of estimating gastric bicarbonate secretion, patients were asked to report empty stomach. Use of antacids or anti-cholinergic drugs was prohibited. Ryle's tube was introduced and positioned fluoroscopically in the gastric antrum. The overnight gastric juice was aspirated and discarded. The secretion in the next one hour was aspirated continuously by using a slow suction pump which works at a suction pressure of 3-5mmHg. Subjects were asked to spit out the saliva during the procedure so as to minimize the interference by salivary bicarbonate. If aspirate was bile stained then the entire sample was discarded and fresh one hour collection was done using after repositioning the Ryle's tube. Venous blood was drawn

for plasma osmolality at 30 minutes of the start of aspiration. Measurements included

- i. Total volume of gastric juice to the nearest 0.5ml
- ii. Osmolality of plasma and gastric juice and
- iii. H<sup>+</sup> ion concentration of gastric juice.

Osmolality was measured by freezing point depression method with the help of advanced digametic osmometer. Readings were taken in triplicate and the mean (expressed as milliosmoles per kilogram) taken for calculation H<sup>+</sup> ion concentration was measured *in-vitro* by titration of gastric juice to pH 7.0 using 0.1N NaOH with the help of El top digital pH meter. All measurements were taken at room temperature.

Calculations were done by the method described by Feldman et al., two basic equations were used:

- I. Measured acid output = Acid secreted - Acid neutralized
- II. Measured osmolar output = Osmoles secreted - Osmoles neutralized
- III. Bicarbonate secretion = Acid neutralized

UGIE was then performed on all CKD patients for visualization of lesions and taking biopsy (2cm) for rapid urease test (RUT) and histopathological examination for *H. pylori*.

**Statistical analysis**

The results were expressed as mean±SD. Statistical analysis was done by Student t-test and paired t-test.

**Results**

Amongst the case 21 (75%, n=28) were males where as all (100%, n=20) the patients in the control group were males. The mean age of cases was 36.3 years whereas it was 35.2 years for controls.

The rate of gastric juice secretion in CKD patients was 58.7±3.2ml/hr as compared to 83.8±6.1ml/hr in controls (p<0.05). Plasma osmolality in CKD patients was significantly higher (330.4±9.0mOsm/kg) in comparison to controls (294.4±3.4mOsm/k; p<0.01). Gastric juice osmolality followed the same pattern as that of plasma osmolality (CKD 218.2±8.1mOsm/kg, controls 156.7±10.6mOsm/kg; p<0.05). There was no difference in the gastric juice acidity, but the acid output and volume of parietal cell secretion were significantly less in gCKD patients as compared to controls (p<0.05) (Table 1). Five patients of CKD had achlorhydria.

The non-parietal cell secretion, bicarbonate output and total acid secretion (acid output + bicarbonate output) were significantly less in CKD patients as compared to controls. The bicarbonate concentration of the non-parietal juice was also less in CKD patients as compared to controls although the difference was not statistically significant (Table 1).

**Table 1:** Comparison of secretory functions of the stomach in CKD patients and controls.

	CKD patients (n=28)	Controls (n=20)	P value
Acidity (mEq/L)	33.2±4.4	37.3±4.6	NS
Acid output (mEq/hr)	1.94±0.27	3.42±0.58	<0.05
Volume of parietal cell secretion (ml/hr)	12.2±1.7	21.4±3.4	<0.05
Volume of non-parietal cell secretion (ml/hr)	46.5±3.0	62.5±3.9	<0.01
Bicarbonate output (mmol/hr)	3.7±0.41	6.26±0.77	<0.01
Bicarbonate output of non-parietal juice (mmol/L)	77.9±6.4	102.3±11.3	NS
Total acid secreted (mmol/hr)	5.64±0.52	9.66±1.32	<0.01

UGIE in CKD patients revealed no abnormality (n=14, 50%), esophagitis (n=10, 35.7%), gastritis (n=9, 32.1%), duodenitis (n=3, 10.7%) and duodenal ulcers (n=2, 7.1%). The acid output, bicarbonate output and total acid output were not significantly different in CKD patients with endoscopic changes on UGIE (n=14, 50%) as compared to those who had normal endoscopic findings (Table 2). Five patients (18.5%) of CKD had a positive rapid urease test and antral biopsies also revealed *H. pylori*. In patients with dyspepsia but no renal failure, *H. pylori* positivity was 45%.

**Table 2:** Relation of UGIE findings in CKD patients with acid and bicarbonate secretion.

Parameter	CKD Patients with Abnormalities on UGIE (n=14)	CKD Patients without Abnormalities on UGIE (n=14)	P value
Acid output (mEq/hr)	2.02±0.26	1.87±0.29	NS
Bicarbonate output (mmol/hr)	3.8±0.42	3.6±0.41	NS
Total acid output (mmol/hr)	5.82±0.43	5.46±0.61	NS

**Discussion**

Although involvement of the gastrointestinal tract in CKD occurs frequently [1], its pathogenesis is poorly understood. Although gastric acid secretion and endoscopic studies have been done in uremic patients, data is scant on the correlation of gastric secretory function with endoscopic findings. Therefore, the present study was carried out to quantify both gastric acid and bicarbonate secretion and study their relation to gastric morphology.

In the present study, the basal acid output in ESRD patients was found to be significantly low as compared to subjects with dyspepsia and normal renal function. Earlier studies on acid output in kidney disease patients have shown conflicting results with some studies reporting hyperchlorhydria [2,3] while other showing normal or low acid output [4-6]. We also found that the

bicarbonate output and bicarbonate concentration in the non-parietal cell secretion were low in CKD patients as compared to controls. These findings point towards a generalized secretory defect in ESRD patients leading to decrease in both acid and bicarbonate secretion. Symptoms of dyspepsia possibly occur because of an imbalance between acid and bicarbonate secretion rather than an absolute change in the amount of either acid secretion [7].

Abnormal findings on UGIE were observed in 50% of ESRD patients in this study. Previously, incidence of endoscopic abnormalities in CKD patients has been reported to be between 41.5-90.7% [8,9]. The reason for this wide variation in the incidence is not apparent but has been proposed to be due to differences in gastric acid status of different racial groups and partly due to subjective variation in interpretation of various inflammatory changes on endoscopy. Notwithstanding, the incidence of peptic ulcers was low (7.1%) in CKD patients, consistent with findings of other series (0-3.3%) [9,10].

The prevalence of *H. pylori* infection in CKD population reported in literature is quite varied (24%-88%) [10,11], with studies reporting both significantly higher [12] and lower [13] prevalence of *H. pylori* infection in uremic patients as compared to those with normal renal function. This discrepancy may be partly explained by the use of less sensitive and specific (techniques serologic or breath analysis) for the detection of *H. pylori* in a number of these studies. Using rapid urease test and antral biopsies for the detection of *H. pylori*, we found that the prevalence of *H. pylori* was less in CKD patients as compared to subjects with normal renal function [14-20].

### Conclusion

The findings of the present study point towards a generalized secretory defect in ESRD patients leading to decrease in both acid and bicarbonate secretion. Symptoms of dyspepsia possibly occur because of an imbalance between acid and bicarbonate secretion rather than an absolute change in the amount of either acid secretion.

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