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Orocecal Transit Time in Patients in the Chronic Phase of Corrosive Injury

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Abstract Accidental/suicidal ingestion of corrosive substances is common in North India. Decreased gastric secretion and delayed gastric emptying in the chronic phase of corrosive injury has been documented at our center. We hypothesize that patients in the chronic phase of corrosive injury may have delayed orocecal transit time (OCTT). *Objective* To measure the orocecal transit time (using the noninvasive method of lactulose hydrogen breath test) in patients in the chronic phase of corrosive injury. *Methods* Thirty patients with corrosive injury to their gastrointestinal tract with its sequelae and attending the gastroenterology services of PGIMER, Chandigarh for endoscopic dilatation of strictures were enrolled in this study. Patients with age >60 years, vagotomy, prior gastric surgery, peptic ulcer disease, systemic sclerosis, history of diabetes, hypothyroidism or intestinal pseudo-obstruction were excluded. Orocecal transit time was measured by using a 15 mL lactulose hydrogen breath test. End expiratory breath was taken every 10 min until there was a rise >10 ppm over the fasting value in two consecutive readings. *Results* Thirty patients (11 females and 19 males) with a median age of 32 years, 27 with acid ingestion and 3 with alkali ingestion, were studied. None had symptoms of gastric outlet obstruction or gastroparesis. OCTT was significantly prolonged in the study group as compared to the control group (135.4 ± 15.8 versus 90.6 ± 10.4 min). No significant difference was observed between different age groups, gender, and type of caustic agent consumed. OCTT was maximally prolonged in patients with involvement of lower oesophagus, whereas patients without lower oesophagus involvement did not show significantly altered OCTT. *Conclusion* Our results show that patients with corrosive injury have prolonged OCTT even in the absence of any gastric symptoms. OCTT was prolonged maximally in patients with lower-third oesophageal cicatrization. This may a result of autovagotomy due to vagal entrapment in the cicatrization process involving the lower third of oesophagus.

Keywords Corrosive injury · Orocecal transit time · Hydrogen breath test

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Introduction

Ingestion of corrosive substances either accidentally or with a suicidal intention is a common problem in India [1]. Corrosive injury to upper GI tract (GIT) is usually caused by strong alkaline or acidic agents. Acids like H_2SO_4 or HCl are easily available to the general population as cheap toilet cleaners. Moreover, industrial and laboratory workers have free access to acids at their work places [2]. The extent of injury to the GIT depends on the type of the agent, its concentration, quality, physical state, and the duration of exposure. In stomach it can cause direct damage to acid-secreting mucosa, decreased capacity of stomach, antral stenosis, and/or pyloric stenosis [1–3]. Functionally, it may cause decreased gastric acid secretion [4], reduced capacity of stomach, delayed gastric emptying [5], and stasis. It can also cause duodenal scarring and narrowing [1, 3, 4]. The major long-term mortality after caustic ingestion is due to cicatrization of the oesophageal strictures [4] and subsequent dysphasia [7–11]. In grade III corrosive injury

involving the lower end of oesophagus, the inflammation and subsequent fibrosis may entrap and compress the anterior and posterior vagal nerves, which is seen in close proximity to the lower end of oesophagus. This possible vagal damage may also lead to delayed gastric emptying and altered small-intestinal motility. Therefore, the present study was planned to measure the orocecal transit time in patients with chronic-phase corrosive injury.

Methods

Thirty consecutive patients who had presented to the gastro clinic of PGIMER, Chandigarh with symptomatic oesophageal cicatrization were enrolled in this study. These patients were successfully managed with endoscopic dilatation to >15 mm. The patients were on regular follow-up for at least for 3 months before inclusion in the study. Patients aged <15 years or >60 years, pregnancy showing evidence of organic gastric outlet obstruction, history of prior oesophageal gastric surgery or vagotomy, peptic ulcer, systemic sclerosis or diabetes, and those who have received board-spectrum antibiotics in the month proceeding the test were excluded from this study. Informed consent was given by all patients and the hospital ethics committee approved the study protocol.

Symptoms such as dyspepsia, regurgitation, vomiting, and early satiety were recorded. Details regarding the nature of the caustic agents ingested and time since consumption were also recorded. The initial barium study films (prior to initiation of dilatation) of all patients were reviewed. These patients also underwent upper gastrointestinal endoscopy, during which a detailed evaluation of oesophagus, stomach, and duodenum was done. In all subjects, endoscopic assessment of upper GIT was done for the presence of any sequelae following corrosive injury. Thirty age- and sex-matched patients who were referred to GE services for routine upper GI endoscopy and had normal endoscopic findings were taken as controls. Thirty such controls were selected on the basis of the following criteria:

1. No history of corrosive ingestion.
2. The same exclusion criteria as used for the study group.

Hydrogen breath test

All 30 patients from the study group and 30 from the control group underwent the lactulose hydrogen breath test according to the method of Bond and Levitt [12], i.e., after an overnight fast. All subjects were asked to avoid high-fibre foods the previous evening because they may cause high fasting values. End expiratory breath was taken for basal hydrogen concentration. Then the subjects were given 15 mL lactulose to drink and hydrogen concentration was measured after every 15 min by using the end expiratory breath test until there was a >10 ppm rise in two consecutive readings over the baseline value. The samples were analyzed by a Model SC Microlyser from Quintron USA for the hydrogen concentration, which was expressed in parts per million (ppm).

Statistical analysis

All the results were expressed as mean ± standard error (SE). The data of orocecal transit time in the study and control groups were analyzed by using Student's unpaired *t*-test. A P value <0.05 was taken as significant.

Results

The study population consisted of 30 patients (11 females and 19 males) with a mean ± standard deviation (SD) age of 31.85 ± 8.46 years and range of 17–45 years, whereas the control group consisted of 9 females and 21 males with a mean ± SD age of 33.48 ± 6.75 years and range 22–49 years. Twenty-seven patients consumed acid and three an alkali. All the patients had undergone successful boogie dilation to 15 mm using Savary Gilliard dilators (Wilson Cook, Winston-Salem, USA) for oesophageal strictures with significant relief of dysphasia. All of them could consume and tolerate normal diet. None of the patients complained of vomiting or early satiety.

Review of the barium studies of 30 patients carried out before initiation of the dilatation programme and endoscopy findings revealed that 14 patients had a lower-third oesophageal stricture, 12 had middle-third stricture, and 4 had upper-third stricture. Twenty-one patients had only oesophagus injury, two had oesophagus plus stomach injury, and seven had only stomach injury. Endoscopy at the start of the study in all 30 patients failed to show any abnormality or narrowing in the stomach. There was easy passage of the endoscope until the second part of duodenum.

The mean orocecal transit time (135.4 ± 15.8 min) was significantly prolonged in the study group, when compared to the values (90.6 ± 10.36 min) in the control group (Table 1). There was no significant difference in OCTT between different age groups (<40 and >40 years), gender (M:F), or the nature of corrosive injury (acid and alkali). However, OCTT differed in patients with stricture

Table 1 Orocecal transit time (min) in patients in chronic phase of corrosive injury

Groups	OCTT (min)
1. Control group ($n = 30$)	90.6 ± 10.36
2. Study group ($n = 30$)	135.4 ± 15.8*
(a) Oesophageal strictures lower one third	170.6 ± 11.9**
(i) Normal OCTT	105.7 ± 106
(ii) Delayed OCTT	185.5 ± 14.8***
(b) Oesophageal strictures upper or mid third	94.5 ± 11.2

* $P << 0.05$ (1 vs. 2)

** $P < 0.001$ (a vs. b)

*** $P < 0.001$ (i vs. ii)

involving the lower third of oesophagus (170.6 ± 11.9 min), when compared to those who had a stricture involving the upper or middle third of oesophagus (94.5 ± 11.2 min) (Table 1).

Discussion

The major morbidity from corrosive ingestion arises from its sequelae in the chronic phase due to cicatrization of the oesophagus and the stomach. Less frequent sequelae include achlorhydria [13], atomicity [14], and gastro paresis [15]. However, as they may not be symptomatic they may not get proper attention from the treating physician. In grade III injury, the inflammation and subsequent fibrosis may entrap and compress the anterior and posterior vagal nerves, which are in close proximity to the lower oesophagus, leading to possible functional damage to vagus nerve [16]. This may lead to hypochlorhydria, delayed gastric emptying, and altered gastrointestinal motility [17]. Orocecal transit time was prolonged in the study group as compared to the control group in the present study. Patients with stricture of the lower third of oesophagus had more delayed of orocecal transit time as compared to those who had stricture at the upper or middle third of oesophagus.

Delayed orocecal transit time in our patients with chronic corrosive injury may be explained by the following factors: gastric musculature injury with subsequent fibrosis resulting in rigidity, or entrapment of vagus nerve in the cicatrization process following transmural injury to lower oesophagus. Our data showing maximal prolongation of OCTT in patients having stricture of the lower third of oesophagus supports the second explanation. It has been shown recently that patients with caustic-induced oesophageal strictures, especially those with distal-third strictures, had higher mean fasting gallbladder volume and lower ejection fraction, pointing to impaired vagal cholinergic transmission, possibly due to vagal entrapment in the cicatrization process [18]. Thus delayed OCTT may be a reflection of vagal dysfunction affecting the gastrointestinal tract on a larger scale. Prolongation of OCTT in patients with chronic corrosive injury may cause symptoms of early satiety, postprandial vomiting, and poor weight gain. In the presence of hypochlorhydria, which has been documented in these patients [4], gastro paresis may be predisposed to bacterial overgrowth and malabsorption. Decreased motility due to autovagotomy may also affect other segments of the gastrointestinal track akin to gallbladder [16]. The prolongation of OCTT in these patients may be due to delayed gastric emptying or, in addition, this may be due to the effect of autovagotomy on other segments of the small intestine. This can be studied by measuring the pylorocecal transit time in these patients and comparing this with the pysocecal transit time in other patients with normal endoscopy. Thus, further studies are required to verify the pathophysiology and the clinical relevance of delayed OCTT after corrosive injury of the upper gastrointestinal tract. If vagal denervation can be determined as the cause of OCTT in corrosive injury, improvement may be expected with cholinergic agonists.

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