Original article

A study of clinical and endoscopic findings in benign strictures of middle and lower thirds of esophagus

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Abstract

Introduction: The aim of this study was to assess the clinical profile of patients with benign strictures of middle and lower thirds of esophagus, to analyse the grade of dysphagia and site of stricture by barium swallow and to describe the endoscopic and histopathological findings in them

Methods: It was a prospective, cross sectional, descriptive study where thirty patients with benign structural esophageal dysphagia of middle and lower thirds were studied. Clinical features and etiology was assessed. Barium swallow and endoscopy were carried out to delineate the stricture. Then histopathological specimens taken from site of stricture were analysed.

Observations and Results: The commonest etiology of benign esophageal dysphagia was found to be peptic stricture (43.33%). Corrosive (23.33%) and post sclerotherapy strictures (20%) followed suit. 53.85% patients with peptic strictures had dysphagia grade II. Patients with corrosive strictures had dysphagia grade III-V. Most patients were poorly built with 56.67% patients having a weight of less than 40 kg. The mean weight of patients in this study was 42.1 + 10.6. On barium swallow, most of the patients had strictures in lower end of esophagus (68%). 19 patients had short strictures (63.33%) while only 9 had long ones. On endoscopy, 5 strictures were very tight (16.67%) (less than 6 mm) and 12 were tight (40%) (7 to 10 mm) and the corrosive strictures were long and tortuous. Fibrosis was the common histopathological finding.

Conclusion: Peptic strictures were mostly smooth and short while corrosive ones were narrow, long and tortuous. Histopathology also showed mostly fibrosis and non specific infiltrate.

Keywords: benign, esophageal, dysphagia, strictures, barium, endoscopy

Introduction:

Dysphagia, defined as difficulty with swallowing refers to problems with the transit of food or liquid from the mouth to the hypopharynx or through the esophagus. There are two types of esophageal dysphagia: structural, which is due to the luminal narrowing or large bolus; and motor, which is due to in coordination or weakness of peristalsis. The normal diameter of the esophagus is 40 mm when distended. The classical structural dysphagia occurs when the lumen of esophagus narrows to 13 mm, but slight dysphagia can be experienced by some patients even when lumen is narrowed to 25 mm. The causes of structural dysphagia can be further grouped into intrinsic and extrinsic. The intrinsic ones are either benign or malignant. Malignancies of esophagus are probably commoner but benign strictures, although rarer, as very troublesome to the patient. They can lead to poor health and vitamin deficiencies. There are several etiologies of benign strictures such as peptic, corrosive, post sclerotherapy, post-radiation, post
operative rings, webs, benign tumours, inflammatory, pill induced and congenital etc.[1,2,3] Peptic strictures are usually due to chronic or severe esophagitis from a variety of causes which lead to scarring and fibrosis.[4] Injury to the esophagus due to ingestion of strong acids (eg, hydrochloric acid) or strong bases (eg, lye) leads to stricture formation 1–3 months after the initial injury (corrosive strictures).[5] Patients who undergo irradiation of the mediastinum (usually 5,000 cGy or more) may develop progressive dysphagia 4–8 months after completion of radiation therapy because of the development of radiation-induced strictures. [6] Drugs such as alendronate may lead to pill induced esophagitis and hence strictures.[7] Many studies have described endoscopic view of various types of strictures regarding their length, lumen and site.[8,9] This study was conducted on thirty patients with structural, intrinsic, benign esophageal dysphagia in middle and lower one thirds of esophagus, in whom radiological, endoscopic and histological findings were studied.

The aims were to study the etiology and clinical profile of these patients and to assess the grade of dysphagia in each. Also, the aim was to describe findings on Barium swallow and study the gross features seen by endoscopy and finally by histopathology of tissue.

**Materials and Methods:**

It was a prospective, cross sectional, descriptive study. Institute ethics committee approval was taken and informed consent was obtained from every patient.

Thirty patients who presented with history of dysphagia and other related complaints and who had endoscopic evidence of narrowing which was benign in nature (benign strictures) were included. These patients were selected from the Medicine OPDs and wards of a tertiary care teaching hospital in Western India over a period of 2 years.

All adult patients above 18 years of age, complaining of dysphagia, who had pathology in middle and lower thirds of the oesophagus, due to any one of peptic stricture, corrosive stricture, post radiotherapy stricture, post operative (usually anastomotic), post sclerotherapy or benign tumors were included in the study. Patients with dysphagia due to motility disorders like achalasia cardia, dysphagia due to pathology in oropharyngal region and upper thirds of oesophagus, oesophageal and tracheoesophageal fistulae and dysphagia due to malignancy of esophagus were excluded from the study. Detailed history of all patients was recorded. A pre-dilation grading of dysphagia was done as follows: Grade 0: No dysphagia, Grade I: Dysphagia intermittently occasionally to solids, Grade II: Dysphagia to solids (at all times), Grade III: Dysphagia to semisolids, Grade IV: Dysphagia to liquidized/pureed food and Grade V: Inability to swallow saliva. A detailed causative history was asked as to corrosive ingestion in the past, exposure to radiation, chronic heartburn, operations on oesophagus, sclerotherapy done, history of vomiting/regurgitation, site of obstruction defined by complaint as upper/middle/lower sternum. The type of dysphagia was assessed – whether intermittent/progressive and the duration was recorded. Thorough clinical examination of the patient was carried out. Basic investigations such as hemogram, blood sugars, renal and liver function tests were done. X-Ray (chest) was done in all cases and ECG wherever indicated. Barium swallow was done to confirm the presence of stricture. It gave information about site, approximate length and severity of stricture, mucosal pattern, tightness of stricture and benign or malignant nature. Patients were then subjected to endoscopy. They were
asked to remain nil by mouth for twelve hours before procedure. The throat was anaesthetized using lignocaine spray and sedation in the form of intravenous midazolam was given in a dose of 0.05mg/kg weight to selected patients with anticipated difficult strictures. Upper gastrointestinal diagnostic endoscopy was now carried out by upper gastrointestinal, forward viewing; PENTAXFG 29V fibreoptic endoscope, to note and confirm the site, approximate length of stricture and associated findings pointing out to the cause such as coexisting gastroesophageal reflux disease (GERD), corrosive ulcerations, sclerotherapy changes in mucosa with white sheathing of varices/ active varices and operative site. Endoscopic biopsy was taken from the site of stricture for histopathology and to rule out malignancy.

Data was collected and compiled in the SPSS software and relevant percentages were calculated.

**Results:**

Most of the patients were between 20 to 60 years of age and the mean was 41.3 ± 18.2 years. There were 19 females (63.33%) and 11 males (36.67%). At presentation most patients had dysphagia grade II to IV with Grade II being commonest (43.33%) (Table 1). Maximum number of patients had complaint of dysphagia in lower sternal region. Most patients (90%) had progressive dysphagia. 80% patients presented with duration of dysphagia between 2 to 6 months. In this study, maximum cases were of peptic strictures (43.33%). Corrosive (23.33%) and post sclerotherapy strictures (20%) followed suit. (Table 2)

53.85% patients with peptic strictures had dysphagia grade II. Patients with corrosive strictures had dysphagia grade III-V, commonest being grade IV (57.14%). Grade II was the commonest grade of dysphagia in post operative and post radiotherapy strictures. (Table 3)

Vomiting was a common symptom (19 patients) when peptic esophagitis and sclerotherapy was cause of stricture. Most patients were poorly built with 56.67% patients having a weight of less than 40 kg. The mean weight of patients in this study was 42.1 ± 10.6. Anemia with haemoglobin less than 9 gm % was found in 66.67% patients.

On barium swallow, most of the patients had strictures in lower end of esophagus (68%) (Table 4) 19 patients had short strictures (63.33%) while only 9 had long ones. (Table 5) 10 patients (35.7%) had dilatation of esophagus proximal to the stricture while 2 patients, both with corrosive poisoning, had diverticula. Six patients (21.43%), all having corrosive strictures had severe degree of narrowing with only a thin streak of dye passing beyond the stricture.

On endoscopy, 10 patients (33.33%) had stricture length more than 5 cm and all of them were corrosive strictures. Maximum patients had stricture in lower third of esophagus, 6 had mid esophageal and two had multiple strictures. 19 (67.86%) patients had smooth mucosa while 28.57% (mostly corrosive & post operative strictures) had irregularity of mucosa. (Table 6) 1 patient had post sclerotherapy sheathing of varices.

5 strictures were very tight (16.67%) (less than 6 mm) and 12 were tight (40%) (7 to 10mm). (Table 7) Fibrosis and non specific inflammatory infiltrate were the commonest findings on histopathology, occurring in 15 (50%) and 16 patients (53.33%) respectively. Dysplasia was found in 4 while Barrett’s esophagus was found in 2 patients. (Table 8)

Pain was the most common complication which occurred in 63.33% patients. No life threatening major bleed occurred during the procedure.
Discussion

In the present study, the age range of patients was 16-91 years with mean age of 41.33±18.3 years. Females outnumbered males with male to female ratio of 0.58:1. Nanda V, Kochhar R et al reported a mean age of 35 yrs. In their study the male: female ratio was 1.6:1.[10] In a study by Desai et al the mean age was 55 yrs. with a male : female of 2.2:1. [8]In this study, most patients had dysphagia ranging between Grade II to IV. In a comparable study by Desai et al, most patients had dysphagia ranging between Grade II to IV. [8] In present study, most patients with peptic strictures had dysphagia Grade II- IV. Those with corrosive strictures had grades III-V dysphagia. The commonest grade of dysphagia in the patients with post sclerotherapy and post radiation strictures was grade II. In patients with post operative strictures, commonest grade of dysphagia was grade II. In a study by Lahoti D et al, on benign strictures, commonest grade of dysphagia at presentation was grade III in corrosive strictures and grade, II in the other causes.[9] Most patients presented within 2-6 months of symptoms in present study. In a study by Desai et al, too similar timeline was observed. In present study, vomiting was a common symptom, present in 90% cases (mostly peptic). In a study by Williamson et al, vomiting was present in 96.4% cases.56.67% patients had weight loss, much higher than some studies. [11] In the present study, only cases with benign strictures were studied. Maximum out of these were peptic strictures(43.33%) while post corrosive(23.3%) and post sclerotherapy(20%) strictures followed closely. Many studies found comparable causative profile, notably, a study by Nanda V and Kocchar R, where 30% were peptic strictures; 25% corrosive; 13.3% post sclerotherapy, post radiation and postoperative strictures being 3.3% each.[10]

On barium swallow, most patients had stricture in lower 1/3rd of esophagus. Studies which only included benign strictures, like Ogilvie et al, Lanza et al showed a preponderance of lower 1/3rd stricture as in present study. [12,13] Most of the patients in present study had short strictures, comparable to study by Poddar U et al. [14]

On endoscopy, in present study, stricture length was short in maximum patients. Strictures were long and tortuous in corrosive etiology. In study by Lahoti D, Broor SL, the mean stricture length was 11.8±2.89 cms with a range of 1.5-25cm.[9] 5 strictures were very tight (16.67%) (less than 6 mm) and 12 were tight (40%) (7 to 10mm) in present study. In a study by Ogilvie et al, range of diameter was 7-11cm.[13] 19 (67.86%) patients had smooth mucosa while 28.57% (mostly corrosive & post operative strictures) had irregularity of mucosa. 1 patient had post sclerotherapy sheathing of varices comparable to other studies. [13]

In a study by Patterson et al the most common histopathological finding was non specific inflammatory infiltrate- which was found in 88%. [15] In a study done by Lahoti et al, which was on 21 patients with benign strictures, all the patients of corrosive ingestion had evidence of fibrosis on histopathology.[9]

Conclusion:

Hence this study described the etiological profile of benign esophageal strictures and noted the Barium and endoscopic findings which easily differentiated from malignant disease. Peptic strictures were mostly smooth and short while corrosive ones were narrow, long and tortuous. Histopathology also showed mostly fibrosis and non specific infiltrate.
References:

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