International Surgery Journal
Varudkar AS et al. Int Surg J. 2017 Mar;4(3):1044-1048
http://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: http://dx.doi.org/10.18203/2349-2902.isj20170859

Original Research Article

Management of benign oesophageal strictures: our experience at tertiary care centre

Anagha S. Varudkar, Santoshkumar N. Deshmukh*, Dyanesh D. Vitekar

Department of General Surgery, Dr. Vaishampayan Memorial Government Medical College, Solapur, Maharashtra, India

Received: 01 January 2017
Accepted: 30 January 2017

***Correspondence:**
Dr. Santoshkumar N. Deshmukh,
E-mail: santoshkumarndeshmukh@gmail.com

ABSTRACT

Background: Oesophageal stricture is a common problem in general surgical practice. It can be benign or malignant, simple or complex. Benign oesophageal strictures include peptic, corrosive, post-surgical anastomotic strictures, post radiotherapy and drug induced strictures. The aetiology of this condition varies in developed and developing countries. Aim of present study was to determine the aetiology of benign oesophageal stricture, to evaluate the role of endoscopic dilatation of stricture and final outcome in these patients in our region.
Methods: This prospective descriptive study was conducted in department of surgery at a tertiary care teaching hospital at Aurangabad from December 2009 to November 2013. All the patients, regardless of age and gender, admitted with diagnosis of benign oesophageal stricture were included in the study. Depending upon the type of stricture treatment was carried out. Postoperatively patients were followed up for detection of possible complications and their treatment.
Results: Total 50 patients of oesophageal stricture were studied. Mean age was 33.65 years with male to female ratio 2.8:1. Ingestion of corrosive substance was the commonest etiology noted followed by peptic strictures.
Conclusions: Corrosive ingestion was the commonest cause of benign oesophageal stricture in our region followed by peptic strictures. Endoscopic dilatation is safe and effective in treating benign oesophageal strictures.

Keywords: Benign stricture, Dilatation, Etiology, Endoscope, Oesophagus

INTRODUCTION

Oesophageal strictures are caused by a diversity of oesophageal disorders or injuries. It can be benign or malignant. Depending on the length, shape and diameter, oesophageal strictures are categorized into simple or complex.[1] Simple oesophageal strictures are defined as those that are focal and straight, with most having a diameter that allows the passage of an endoscope of normal diameter.[2]

Complex oesophageal strictures are defined as those that are long (> 2 cm), tortuous or that have a diameter that prevents passage of an endoscope of normal diameter.[2] Simple strictures are usually caused by peptic ulcer disease, Schatzki's ring or a web.[2,3] Complex stricture are the sequelae of corrosive injury, radiation, anastomotic stricture and severe peptic injury.[4-6]

Peptic strictures are the most common cause of benign oesophageal strictures in the West whereas corrosive strictures are the most common form of benign strictures in developing countries like India.[8-10]

Present study was undertaken to determine the etiology of benign oesophageal strictures in our region, to evaluate

the role of endoscopic dilatation of stricture and to study the final outcome in these patients.

METHODS

After obtaining the institutional ethics committee approval, present prospective descriptive study was carried out in the department of surgery at a tertiary care teaching hospital at Aurangabad, Maharashtra, India. Present study was carried out for a period of 5 years (January 2009 to December 2013) on 50 patients.

Inclusion criteria

All the patients with radiological and endoscopic diagnosis of benign oesophageal stricture, regardless of age and gender admitted and treated were included in the study.

Exclusion criteria

Patients with malignant oesophageal stricture, Achalasia cardia and those not willing to participate in the study were excluded.

On admission, detailed history, regarding onset and duration of dysphagia was taken and grade of dysphagia was determined as per the score suggested by Richard E et al.[11] History of corrosive substance intake and other symptoms like regurgitation, acidity, throat pain and dyspnoea were noted in the proforma. History regarding ingestion of non-steroidal anti-inflammatory drugs (NSAIDS) and associated diseases were documented. A thorough clinical examination was made with particular attention to oral cavity and throat. IDL examination was also carried out. The oesophageal strictures were characterized by barium contrast radiography and upper GI endoscopy. The etiology of the oesophageal stricture was determined by the combination of clinical, endoscopic, radiological and histopathological evidence. Necessary laboratory investigations (like complete blood count, blood sugar, HIV and Hepatitis B status, urine analysis) were performed. Depending on the type of stricture treatment was carried out. Due informed written consent of the patient and relatives was taken before procedure. Prior to dilation, patients were kept nil per mouth overnight to assure a clear view of the oesophageal lumen and to avoid aspiration. Patients taking antiplatelet agents and anticoagulants were instructed to avoid medication seven days prior to the procedure. Dilatation was done under fluoroscopic guidance using Savary Gilliard bougies over the guide wire. Dilatation was typically performed as an ambulatory procedure using conscious sedation and topical pharyngeal anaesthesia. A standard guide wire was positioned with the help of endoscope and fluoroscopy up to the antrum. After the guide wire was positioned the scope was withdrawn and dilatation was performed using Savary Gilliard bougies of increasing size. We followed the 'rule of 3' (no more than three dilators of progressively increasing diameter should be passed in a single session) during dilatation in one session. Procedure details like type of stricture, length of stricture, simple/complex, and procedure performed, number of settings required were recorded. Patients were allowed liquids orally 6 hours after the procedure and were encouraged to resume intake of semisolid one day after the procedure except for those which developed complications during procedure. After procedure all the patient were kept under close observation for detection of signs and symptoms of oesophageal perforation like chest pain, tachycardia, back pain, shoulder pain, epigastric tenderness. After discharge they were followed up for first 3 months for detection of late complications like recurrence. Treatment success was gauged according to the improvement of dysphagia using dysphagia score. Following oesophageal dilatation for peptic strictures, patients were treated with proton pump inhibitors to promote healing and reduce the risk of stricture recurrence. The data collected were entered into MS-Excel sheets and analysis was carried out using statistical package for social sciences (SPSS-version 16.) On the basis of analysis and observation, results were drawn and discussed and compared with other relevant literatures.

RESULTS

During the study period, consecutive 50 patients of benign oesophageal strictures undergoing endoscopic dilatation were included.

Table 1: Age and sex incidence.

Age group (years)	No. of cases		Total	Percentage
	Male	Female		
0- 10	4	1	5	10%
11-20	4	1	5	10%
21-30	12	5	17	34%
31-40	7	3	10	20%
41-50	1	1	2	4%
51-60	3	1	4	8%
61-70	5	1	6	12%
71-80	1	0	1	2%
Total	**37**	**13**	**50**	**100%**

Table 2: Clinical presentation of patient.

Signs and symptoms	No. of cases	Percentage
Dysphagia for solids	38	76%
Absolute dysphagia (Dysphagia to both solids and liquids)	6	12%
Vomiting	2	4%
Cough on swallowing	4	8%

The most vulnerable age group in this study was 21to 30 years (34%). The next most common age group affected was 31 to 40 years (20%). Out of 50 cases studied, 37

were male and 13 were females with male to female ratio of 2.8:1. Thus males clearly outnumbered the females in present study. Mean age of the patient in our study was 33.65

Most of the patients presented with dysphagia to the solids 38 (76%) followed by absolute dyphagia (12%) cough on swallowing was noted in 4 (8%) patients and 2(4%) had vomiting at the time of admission.

Table 3: Distribution of patients according to grade of dysphagia.

Grade of dysphagia	Complaint	No. of cases	Percentage
I	Able to eat everything but with difficulty	2	4%
II	Dysphasia to solids	30	60%
III	Dysphasia to semisolids	10	20%
IV	Dysphasia even to liquids	5	10%
V	Absolute dysphasia	3	6%

Table 4: Distribution of patients according to stricture characteristics.

Etiology of strictures	No. of cases	Percentage
Peptic stricture	19	38%
Corrosive ingestion	27	54%
Postoperative (anastomotic)	3	6%
Drug induced	1	2%
Nature of injury in corrosive strictures (n=27)	**No. of cases**	**Percentage**
Accidental	5	18.5%
Suicidal	19	70.37%
Homicidal	3	11.11%
Location of strictures	**No. of cases**	**Percentage**
Upper one third	12	24%
Middle one third	15	30%
Lower one third	13	26%
Multiple segments involvement	10	20%
Length of stricture	**No. of cases**	**Percentage**
Less than 2 cm	32	64%
More than 2 cm	8	16%
Extensive or multiple or long	10	20%
Number of strictures	**No. of cases**	**Percentage**
Single	35	70%
Multiple	15	30%

Majority of patients 30 (60%) have grade II in our study followed by grade III dyaphagia noted in 10 (20%) patients.

Majority of the patients 27 (54%) in the present study have corrosive strictures followed by peptic stricture. Out of 27 cases of corrosive poisoning, 5 (18.5%) cases were due to accidental ingestion of corrosive substance, 19 (70.37%) cases were due to suicidal intention, and 3 (11.11%) cases had history of homicidal ingestion of corrosive substance. Ingestion of corrosive substance with suicidal intension was the most common nature of corrosive injury. The commonest site for oesophageal stricture noted (30%) in our study was the middle third of the oesophagus, followed by involvement of lower third of oesophagus in 13 (26%) patients. In our study most of the strictures had length less than 2 cm and found in 32 (64 %) patients. 10 (20 %) patients had having multiple strictures. Majority of patients 35 (70%) in our study have single stricture.

Table 5: Post dilatation complications.

Post dilatation complications	No. of cases	Percentage
Perforation	7	14 %
Bleeding	4	8 %
Procedure related death	0	0.00%

In our study 4 patients had small perforations during dilation of stricture of oesophagus, which were identified immediately and treated conservatively. 2 patients with homicidal corrosive ingestion had perforation secondary to dilatation of stricture succumbed to death due to septicaemia. 1 patient with suicidal corrosive ingestion required dilations for recurrent dyphagia. She was advised surgery but patient was not willing hence she was repeatedly underwent dilatation. But ultimately she expired at home possibly due to the cause of malnutrition. Bleeding was noted in 4 patients and treated conservatively. Procedure related death was not observed in any patient.

Table 6: Outcome of patients.

Outcome	No. of cases	Percentage
Successful dilatation (15mm)	37	74%
Unsuccessful dilatation	8	16%
Expired	4	8%
Lost to follow up	1	2%

In our study majority of patients had successful dilation of oesophageal stricture 37 (74%). Four patients expired and one patient lost to follow up. 8 patients have unsuccessful dilation.

DISCUSSION

Corrosive injury to the oesophagus may be caused by ingestion of strong acids or a strong base either with suicidal intent or accidental ingestion. It causes caustic oesophagitis which ultimately leads to formation of oesophageal stricture within 1 to 2 months after initial injury. Depending upon the degree of injury and scar formation, affected patients may develop one or more segmental strictures that have unpredictable location or diffuse oesophageal stricture that reduces the entire oesophagus to filliform strictures. Caustic injury is therefore associated with more extensive stricture formation. In present study corrosive strictures were seen in 54% cases and peptic strictures in 38% patients. Thus corrosive injury is the commonest etiology of oesophageal stricture in our study. Similar observations were also reported in Lahoti et al, Broor SL et al and Zargar SA et al.[12,13]

Male to female ratio in our study was 2.8:1. In the study conducted by Qureshi S et al the male to female ratio was 2.3:1 which is comparable with present study. Mean age of the patient in our study was 33.6.[14] Broor SL et al reported a mean age of 25.5 years in a study of 47 patients with corrosive oesophageal strictures. In the present study mean age of patients with corrosives stricture was 26.4 years as compared to 42.5 years in peptic strictures group showing significant association with age ($p < 0.015$).[10] This could be related to immaturity and emotional instability seen in younger age.

Progressive dysphagia for solids was the commonest presenting symptom in our study followed by cough on swallowing. Similar observations were also reported in Zargar et al study.[13] Atypical presentations included chronic cough and asthma secondary to aspiration of food or acid. In our study corrosive strictures were located in the upper and mid oesophagus in 27 (54%) patients while peptic strictures were observed in 13 (26%) patients in lower oesophagus. Thus corrosive strictures were more common in upper and mid oesophagus while peptic strictures were common in lower oesophagus. Similar observed were also reported in Ilkin NM et al study.[15] In our study majority of patients (74%) had successful dilation of oesophageal stricture. Adequacy of dilatation or its success was gauged in terms of major improvement or relief of dysphagia.

Perforation is the commonest and most serious complication of oesophageal stricture dilatation and usually occurs at the site of the stricture. The perforation rate for oesophageal strictures after dilation has been reported to range from 0.1% to 0.4% in various studies.[16-19]

Perforation rate in our study was 8% which is higher as compared to other studies, this could be due to more patients of corrosive strictures and also small sample size in our study. Incidence of perforation was seen exclusively in corrosive strictures, which were long, eccentric and tight, thus technically more difficult to dilate.

The overall mortality rate in this study was 8% and related to sepsis. In the study conducted by Qureshi S et al on 27 patients mortality reported was 7.4%.[14] This figure is comparable with our study. The mortality related to oesophageal perforation depends on time of recognize. Patients in whom perforation is recognized immediately have a good prognosis whereas a delay of beyond 24 hours leads to a high mortality of 25-50%.[20,21]

CONCLUSION

Ingestion of corrosive agents was the commonest etiology of benign oesophageal strictures in our region, followed by peptic strictures. Corrosive strictures were more frequently seen in younger age group while peptic stricture were common in older age group. Endoscopic dilatation is a safe and effective mode of treatment of benign oesophageal strictures with a low recurrence rate and acceptable morbidity and mortality.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the institutional ethics committee

REFERENCES

1. Hyoung JK, Hoon JS, Young S. Benign strictures of the esophagus and gastric outlet: Interventional Management. Korean J Radiol. 2010;11:497-506.
2. Lew RJ, Kochman ML. A review of endoscopic methods of esophageal dilation. J Clin Gastroenterol 2002;35:117-26.
3. Lima JC, Ramires RP, Zamin I, Cassal AP, Marroni CA, Mattos AA. Endoscopic dilation of benign esophageal strictures: report on 1043 procedures. Am J Gastroenterol. 1999;94:1497-501.
4. Kim JH, Song HY, Kim HC, Shin JH, Kim KR, Park SW, et al. Corrosive esophageal strictures: long-term effectiveness of balloon dilation in 117 patients. J Vasc Interv Radiol. 2008;19:736-41.
5. Siersema PD. Treatment options for esophageal strictures. Nat Clin Pract Gastroenterol Hepatol. 2008;5:142-52.
6. Choi GB, Shin JH, Song HY, Lee YS, Cho YK, Bae JI, et al. Fluoroscopically guided balloon dilation for patients with esophageal stricture after radiation treatment. J Vasc Interv Radiol. 2005;16:1705-10.
7. Kim JH, Song HY, Park SW, Yoon CJ, Shin JH, Yook JH, et al. Early symptomatic strictures after gastric surgery: palliation with balloon dilation and stent placement. J Vasc Interv Radiol. 2008;19:565-70.
8. Patterson DJ, Graham DY, Smith JL. Natural history of benign esophageal stricture treated by dilation. Gastroenterology. 1983;89:346-50.

9. Williamson RCN. The management of peptic esophageal stricture. Br J Surg. 1975;62:448-54.
10. Broor SL, Kumar A, Chari ST, et al. Corrosive esophageal strictures following acid ingestion: clinical profile results of endoscopic dilatation. J Gastroenterol Hepatol. 1989;4:56-61.
11. Earlam R, Cunha JR. Benign esophageal strictures: historical and technical aspects of dilation. Br J Surg. 1981;68:829-36.
12. Lahoti D, Broor SL. Corrosive injury to upper gastrointestinal tract. Indian J Gastroenterol. 1993;12:135-41.
13. Zargar SA, Kocher R, Nagi B, Mehta S, Mehta SK. Ingestion of corrosive acids. Spectrum of injury in upper gastrointestinal tract and natural history. Gastroenterolgy. 1989;97:702-7.
14. Qureshi S, Ghazanfar S, Leghari A, Tariq F. Benign esophageal strictures: Behaviour, pattern and response to dilatation. J Pak Med Assoc. 2010;60(8):656-60.
15. Naharci M, Tuzun A, Erdil A, Ates Y, Bagci S, Yamanel L, et al. Effectiveness of bougie dilation for the management of corrosive esophageal strictures. Acta Gastroenterol Belg. 2006;69:372-6.
16. Hernandez LV, Jacobson JW, Harris MS. Comparison among the perforation rates of Maloney, balloon, and savary dilation of esophageal strictures. Gastrointest Endosc. 2000;51:460-2.
17. Karnak I, Tanyel FC, Büyükpamukçu N, Hiçsönmez A. Esophageal perforations encountered during the dilation of caustic esophageal strictures. J Cardiovasc Surg. 1998;39:373-7.
18. Silvis SE, Nebel O, Rogers G. Endoscopic complications: results of the 1974 American society of gastrointestinal endoscopy survey. JAMA. 1976;235:928-30.
19. Mandelstam P, Sugawa C, Silvis SE. Complications associated with Esophago-gastroduodenoscopy and with esophagaeal dilation. Gastrointest Endosc. 1976;23:16-29.
20. Shaffer HA, Valenzvela G, Mittal RK. Esophageal perforation a reassessment of the literature for choosing medical or surgical therapy. Arch Intern Med. 1992;152:757-61.
21. Andreollo NA, Lopes LR, Inogutti R, Brandalise NA, Leonardi LS. Conservative management of benign esophageal strictures using dilation. Analysis of 500 cases. Rev Assoc Med Bras. 2001;47:236-43.

Cite this article as: Varudkar AS, Deshmukh SN, Vitekar DD. Management of benign oesophageal strictures: our experience at tertiary care centre. Int Surg J 2017;4:1044-8.