Introduction
Corrosive injury related esophageal stricture has been cited as a risk factor for squamous cell carcinoma esophagus. There are no data from prospective surveillance programs. This study was planned to study histopathological changes and safety of chronic dilatation (mean duration 10.1 year) in reference to occurrence of dysplastic changes.

Materials and Methods: This study was conducted in the Department of gastroenterology SMS medical college Jaipur. Patients with caustic esophageal strictures, above 03 years of age and more than 10 year duration of dilatation were included. Wire guided dilatation with increasing size of plastic dilator was done. Various aspects of study group, type of stricture, and histopathological changes were studied in reference to occurrence of dysplastic changes. Throughout the study patient were given standard of care treatment.

Results: Out of 42 patients, 32 patients (77%) were more than 12 years of age. Mean age was 22.9 ranging from 3 years to 50 years. There were twenty six males (62%) and sixteen females (38%). Ten patients (24%) had multiple strictures. twelve patients (28.5%) had long stricture (>3cm). Epithelial hyperplasia, focal hyperkeratosis, and mixed inflammatory exudates in sub epithelium were predominant histologic findings. No dysplasia was reported on histopathological examination.

Conclusion: Caustic Stricture is more common in adolescent and adults in our population. There is high incidence of acid injury as compared to alkali injury. Dysplastic changes are not seen in patients on chronic dilatation at mean age of 10.1 year.

Keywords
Caustic; Esophageal; Strictures; Dilatation; Dysplasia
of structured segment revealed epithelial hyperplasia, focal hyperkeratosis, and sub epithelial mixed inflammatory exudates in majority of samples (Figure 2 & 3). No dysplastic changes were seen on histopathological examination in any of the patient.

**Discussion**

Extent of injury following caustic ingestion depends on amount, concentration and pH of substance and tissue contact time. The most frequent complication of corrosive substance ingestion is esophageal stricture. The characteristic lesions in man, usually limited to the esophagus, consist of acute necrosis, ulceration, and later varying degrees of fibrosis, often with the development of stricture. The subsequent appearance of carcinoma at the site of such a stricture, however, is a less well-recognized complication that may develop years after the original injury. The rate of stricture formation is reported to be between 2% and 63%. There appears to be an increased risk of developing squamous cell carcinoma of the esophagus after severe caustic injury to the esophagus, most commonly after lye ingestion [3].

Scar tissue is probably a premalignant condition, the danger increases if there is chronic irritation at the site of the scar. Of the 10 patients with malignancy in a fibrotic oesophagus, only five had a respectable growth, and in two cases the fact that the dense scar tissue included a malignant growth became evident only after surgery and histological examination of the resected oesophagus [4]. The incidence of cancer in corrosive strictures has been estimated to be 2.3% to 6.2%, and a history of caustic ingestion was present in 1% to 4% of patients with esophageal cancer [5]. A single series from Finland found the magnitude of risk was approximately 1000-fold increased compared with a similar population during 30 yr follow up [6]. The association between corrosion and carcinoma was probably causal in 5 women who developed oesophageal cancer 25-50 years after corrosive injury [7]. Clinical characteristics of patients who developed esophageal cancer after caustic injury included, mean age 35 to 51 years; average interval between caustic injury and development of esophageal cancer approximately 40 years; and cancers were located in the mid-esophagus [8].

There are no data from prospective surveillance programs. Although endoscopic dilatation effectively relieves dysphagia in benign esophageal stricture, there is little information on its efficacy and safety in caustic esophageal stricture. This study is first prospective study of large cohort to evaluate the safety of chronic dilatation in reference to dysplastic changes. No dysplasia was evident in any of patients in this study. Further long term follow up study of this group and more studies will answer the safe period and surveillance protocols in this group of patients.

**Conclusion**

Most common histopathologic changes were epithelial hyperplasia, focal hyperkeratosis, and mixed inflammatory exudates in sub epithelium. Dysplastic changes were not seen in patients on chronic dilatation at mean duration of 10.1 years. This group of patients requires a further follow up.

**References**

1. Mamede RC, DeMello Filho PV (2002) Treatment of caustic ingestion: An analysis of 239 cases. Dis Esophagus 15(3): 210-213.
2. Marshall F (1979) Caustic burns of the esophagus: ten-year results of aggressive care. South Med J 72(10): 1236-1237.
3. Duncan M, Won RK (2003) Esophageal emergencies: things that will wake you from a sound sleep. Gastroenterol Clin North Am 32(4): 1035-1052.
4. Imre J, Kopp M (1972) Arguments against long-term conservative treatment of oesophageal strictures due to corrosive burns. Thorax 27(5): 594-598.

5. Isolauri J, Markkula H (1989) Lye ingestion and carcinoma of the esophagus. Acta Chir Scand 155(4-5): 269-271.

6. Kiviranta UK (1952) Corrosion carcinoma of the esophagus: 381 cases of corrosion and nine cases of corrosion carcinoma. Acta Otolaryngol 42(1-2): 89-95.

7. Ti TK (1983) Oesophageal carcinoma associated with corrosive injury-prevention and treatment by oesophageal resection. Br J Surg 70(4): 223-225.

8. Appelqvist P, Salmo M (1980) Lye corrosion carcinoma of the esophagus. A review of 63 cases. Cancer 45(10): 2655-2658.