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publication. Some minor issues:

1. The authors included children 6 months to at most 2.6 years post corrosive esophageal stricture. However cancer to develop needs long-term process. So the authors should comment on this. It is too difficult to conclude from this study that cancer is possible on the ground of corrosive esophagitis in short term.

Answer: Yes, of course. We did not only perform this study to screen the oesophageal cancer on the ground of corrosive oesophagitis but also to screen the precancerous lesions as squamous dysplasia. We also desired to know to what extent dysplasia occurred in that young age of paediatric patients.

2. There is plagiarism in the discussion. Although interesting to discuss about dilatation on the ground of esophageal stenosis it is not the aim of this study. The authors should concentrate only to the issue which is histopathological findings in esophageal corrosive stricture.

Answer: We edited the discussion to concentrate on histopathological findings in esophageal corrosive stricture

As written in the manuscript's discussion

3. The initial biopsies were blind from the stricture while second biopsies on the dysplastic initials were done after lugol staining. What about virtual chromoendoscopy such as NBI in these cases, which does not need biopsy. At least the authors should commented on the discussion on the possibility of NBI magnification for endoscopic real time tissue visualization of esophageal mucosa in case of corrosive esophageal stricture. Of course target biopsies on this case should confirm the existence of dysplasia or not.

   Answer: We added this important point and highlighted in the manuscript's discussion

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4. The authors stated in the discussion：<<the trauma of repeated bougie dilatation may be a promoter in the ultimate development of dysplasia>>However this conclusion cannot be drained from this study. Actually repeated dilatation is needed in severe corrosive strictures and dysplasia may be due to severe stricture and not due to trauma.

Answer: In our study's conclusion, the development of dysplasia was associated with several risk factors, but the number of dilatation sessions was not significantly associated with the risk of dysplasia development.

In comparison to literatures, **Kavin** and his colleagues who suggested that<<the trauma of repeated bougie dilatation may be a promoter in the ultimate development of dysplasia>>[18] Kavin H, Yaremko L, Valaitis J, Chowdhury L. ChronicOesophagitis evolving to verrucous squamous cell carcinoma:possible role of exogenouschemicalcarcinogens.Gastroenterology. 1996;110: 904-914 PMID: 8608902

Nagaich and his colleagues studied the histopathological changes and safety of chronic dilatation (mean duration of 10 years) in reference to the occurrence of dysplastic changes and reported no risk from chronic dilatation [1]. Nagaich N, Sharma R, Nijhawan S, Nijhawan M, Nepalia S, Rathore M. Histopathological Profile of Caustic Oesophageal Strictures on Chronic Endoscopic Dilatation: What is the Safe Limit??. J Cancer PrevCurr Res 2015; 2: 23. [DOI: 10.15406/jcpcr.2015.02.00023]

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children, but on the other I believe that some sort of preventive program has to be implemented, which, of course does not influence the quality of the paper but it would be appropriate to include in the discussion or future plans.

Answer: We would refer to preventive program in future plan of this study which needs multidisciplinary approach. As highlighted in manuscript

I have some questions and proposals how to improve the paper:

• Methods:o Did the patients also have an oesophagoscopy soon after the ingestion to assess the mucosa in the first 24 h after ingestion with the grading of caustic injury? If they did, than please, add these data to the protocol and results sections.

Answer: No, the patients did not have an oesophagoscopy soon after the ingestion to assess the mucosa in the first 24 h after ingestion. As the majority of patients were referred to our endoscopy unit after one month from ingestion of corrosion.

o Did both pathologists review all the histological samples and did you compare the reproducibility of the histopathological evaluation? If they just evaluated a part of histology specimens, than just clarify, in not, please, add the data on repeatability to the results section.o

Answer: No, the pathologist no. 2 had just evaluated a part of histology specimens, and all results were the same except for minor differences in few patients as described below

Patient no. 9, *pathologist no. 1* described his biopsy as chronic oesophagitis indefinite for early dysplastic changes, while*pathologist no. 2* described it as low grade dysplasia with evidence of epithelial cell disorganization, nuclear pleomorphism, hyperchromasia and cellular crowding.

Chromoendoscopy was decided for patient no. 9 to obtain targeted biopsy from the dysplastic oesophageal mucosa, both pathologists examined the specimen blindly. The final result was the same for both pathologists as low grade dysplasia.

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Patient no. 33 and 45, *pathologist no. 1* described her biopsy as chronic oesophagitis, while *pathologist no. 2* described her biopsy as reactive epithelial atypia / indefinite for dysplasia due to heavy intraepithelial neutrophilic infiltration.

It would be clarified in manuscript's results.

Was statistical software version 20 or 23?

Answer: version 23

It would be clarified in manuscript's methods.

• Results: Many children after caustic ingestion and oesophageal stenosis in your study were underweight or stunted. Beside the bouginage of stenosis, were there any other actions taken to restore their weight gain and growth? For e.g. nasogastric tube feeding, enteral nutrition support or surgery (interposition of large or small bowel) to restore oesophageal function?

Answer: no one had a nasogasrtric tube placed for tube feeding. But patients were allowed enteral nutrition as tolerated. We exclude the post corrosive oesophageal stricture patients who did surgery from the start (as highlighted in methodology)

• Discussion: The age of patients at the time of ingestion was very low. Are there any plans for preventive actions to educate parents to lower the incidence of ingestions in children?

Answer: Yes, of course. There are plans for prevention but it takes time to be implemented.

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o The rate of dysplasia was low. How do you propose to approach these patients, when to do the first biopsy or chromoendoscopy and how to follow-up the patients with dysplasia during the childhood and afterwards when they reach the adult age?

Answer: All patients were enrolled in the study after informed consent was obtained from their parent/guardian. These patients followed in our endoscopy unit for dilatation. We informed the patients with dysplasia about the pathological finding and importance of follow up. They already came in regular basis in our unit to perform every year a new biopsy till the age of 18. After this age, they will follow in adult endoscopy unit in our university hospital.

• Suggestion for further researcho I hope that you will be able to follow this cohort of patients further (e.g. every 5 years) and to publish the results.

Answer: It is our hope also. We are working on it.

• References:o Have to be written according to the WJG instructions and uniformly for all references.

Answer: We edited it according to the WJG instructions and uniformly for all references.

• Others:o Some punctuation corrections throughout the text have to be made.

Answer: We edited it

The paper is very interesting for paediatric and also adult gastroenterologists, therefore I believe that the revised version will be suitable for the publication in WJG.

Yours sincerely,

Reviewer

INITIAL REVIEW OF THEMANUSCRIPT

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