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CASE REPORT

양잿물에 의한 식도협착 환자에서 편평세포암종으로 오인된 거짓상피종 증식

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Pseudoepitheliomatous Hyperplasia Mimicking Esophageal Squamous Cell Carcinoma in a Patient with Lye-induced Esophageal Stricture

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Pseudoepitheliomatous hyperplasia is a benign condition that may be caused by prolonged inflammation, chronic infection, and/or neoplastic conditions of the mucous membranes or skin. Due to its histological resemblance to well-differentiated squamous cell carcinoma, pseudoepitheliomatous hyperplasia may occasionally be misdiagnosed as squamous cell carcinoma. The importance of pseudoepitheliomatous hyperplasia is that it is a self-limited condition that must be distinguished from squamous cell carcinoma before invasive treatment. We report here on a rare case of esophageal pseudoepitheliomatous hyperplasia in a 67-year-old Korean woman with a lye-induced esophageal stricture. Although esophageal pseudoepitheliomatous hyperplasia is infrequently encountered, pseudoepitheliomatous hyperplasia should be considered in the differential diagnosis of esophageal lesions. **(Korean J Gastroenterol 2014;63:366-368)**

Key Words: Pseudoepitheliomatous hyperplasia; Squamous cell carcinoma; Esophagus

INTRODUCTION

Pseudoepitheliomatous hyperplasia (PEH) is a benign condition that may be caused by prolonged inflammation, chronic infection, and/or neoplastic conditions of the mucous membranes or skin.[1,2] Due to its histological resemblance to well-differentiated squamous cell carcinoma (SCC), PEH may occasionally be misdiagnosed as SCC.[1,2] PEH generally involves the skin or mucous membranes.[1,2]

However, no case of an esophageal lesion associated with PEH has been reported. We report here on a rare case of esophageal PEH in a 67-year-old Korean woman with a lye-induced esophageal stricture.

CASE REPORT

A 67-year-old woman presented to our hospital with dysphagia that had developed approximately one month previously. She had a history of an esophageal stricture caused by lye ingestion approximately 50 years previously and had undergone esophageal resection and esophagogastrostomy seven years previously. Her medical history included hypertension and diabetes mellitus, and her family history was unremarkable. She was a non-smoker and social

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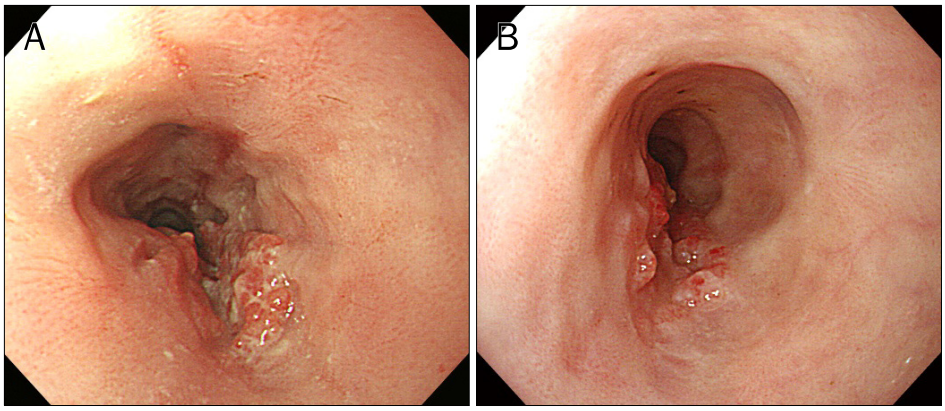

Fig. 1. Endoscopic view of the esophagus showing an intraluminal protruding mass 25 cm from the incisor teeth during: (A) an initial esophagogastroduodenoscopy; (B) a surveillance esophagogastroduodenoscopy performed after two months.

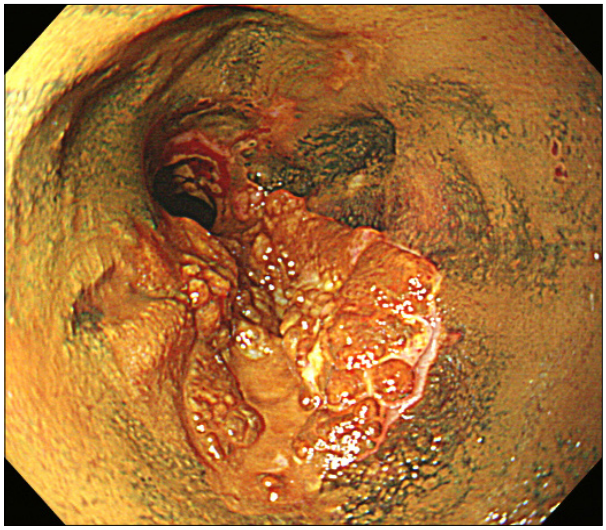

Fig. 2. Lugol chromoscopy showing the mucosal lesion stained with Lugol's solution.

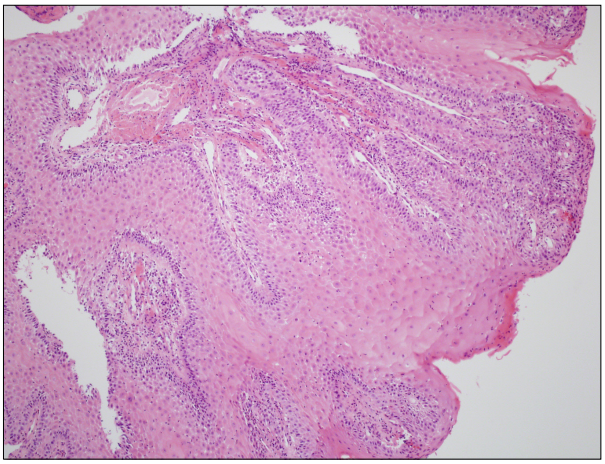

Fig. 3. Histological view of the esophageal mucosal lesion showing prominent hyperplasia of the epithelium (H&E, ×100).

drinker. She had recently reported a weight loss of 18 kg. An initial esophagogastroduodenoscopy showed an intraluminal protruding lesion 25 cm from the incisor teeth (Fig. 1A). The endoscope could not pass through the lesion due to the esophageal stricture. Lugol chromoscopy and biopsy were performed in order to exclude esophageal cancer. The mucosal lesion was stained with Lugol's solution (Fig. 2), and histological examination showed prominent irregular hyperplasia of the epithelium (Fig. 3). On immunohistochemical staining, Ki67 and p53 expression was observed only in the basal layer of the epithelium (Fig. 4). After two months, the lesion showed significant improvement without specific treatment (Fig. 1B).

DISCUSSION

The histological findings of PEH, characterized by the prominent proliferation of epithelium, are similar to those of well-differentiated SCC; therefore, PEH may occasionally be misdiagnosed as SCC.[1,2] The pathogenesis of PEH remains unclear and there is no standard treatment. Although distinguishing PEH from SCC can be difficult, the lack of atypical features, such as nuclear atypia and mitosis, and recognition of the associated underlying entities, are usually helpful in excluding SCC.[3] In addition, immunohistological staining methods, including staining for Ki67 and p53 expression, have been used for differentiation of PEH from SCC. SCC shows nuclear reactivity for Ki67 and p53 with staining of nuclei throughout the entire thickness of the epithelium, whereas PEH usually shows increased nuclear staining only in the basal layer of the epithelium.[4,5] In our case, the esophageal le-

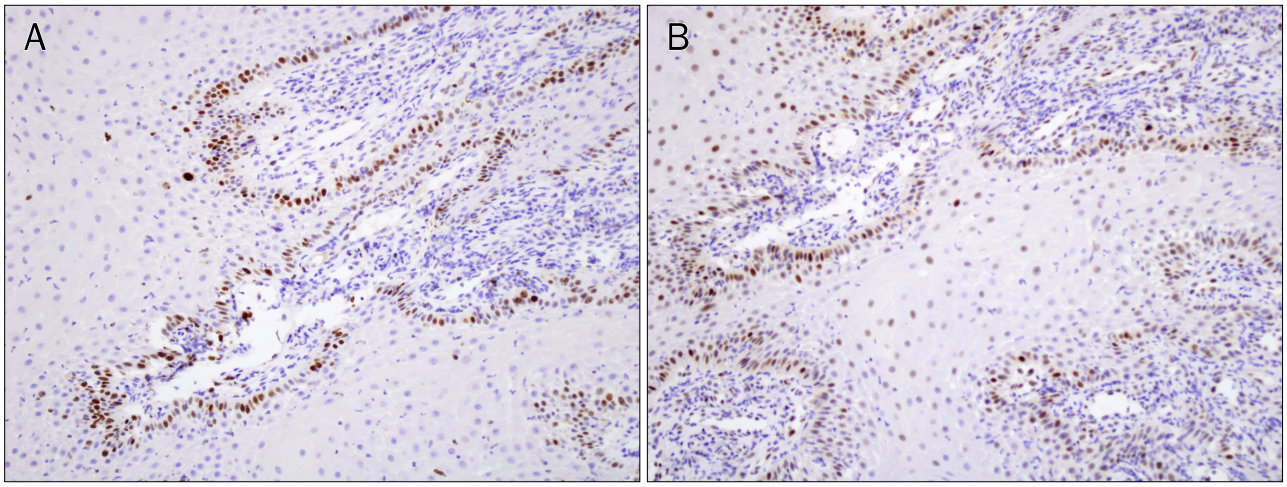

Fig. 4. Immunohistochemical view of the esophageal mucosal lesion showing staining limited to the basal layer of benign epithelium with: (A) Ki67 immunostain (×200); (B) p53 immunostain (×200).

sion was stained with Lugol's solution, and p53 and Ki67 expression was observed only in the basal layer. The importance of PEH is that it is a self-limited condition that must be distinguished from SCC before invasive treatment. PEH usually involves the skin or mucous membranes.[1,2] However, no case of an esophageal lesion associated with PEH has been reported. To the best of our knowledge, this is the first case report of esophageal PEH in a female patient with a history of lye-induced esophageal stricture. Although esophageal PEH is infrequently encountered, PEH should be considered in the differential diagnosis of esophageal lesions.

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