Mucosal bridge formation in a patient with esophageal epidermoid metaplasia

Esophageal mucosal bridge formation is an uncommon condition that is believed to result from mucosa injury. Ingestion of corrosive agents, mediastinal radiation therapy, and variceal sclerosis therapy have been identified as possible causes, but mucosal bridges within the esophagus have also been described in chronic inflammatory conditions such as Crohn’s disease [1,2].

A 53-year-old man with a long history of smoking and chronic obstructive pulmonary disease presented with dysphagia and bolus obstruction. Endoscopy revealed multiple coalescent white patches and plaque-like lesions that covered large parts of the esophageal mucosa (Fig. 1a). In the middle portion of the esophagus, large irregular mucosal bridges were found that linked the anterior and posterior walls transversely across the lumen (Fig. 1b), ultimately leading to double-lumen formation (Video 1). Biopsies obtained from the white patches proved epidermoid metaplasia of the esophagus, characterized by squamous epithelium with a well-developed granular cell layer, hyperorthokeratosis with focal parakeratosis, and mild peripapillary lymphocytic infiltrate (Fig. 2).

Endoscopic view. a Esophageal epidermoid metaplasia characterized by multiple white patches and plaque-like lesions covering large parts of the mucosa. b Irregular mucosal bridges were found in the mid-portion of the esophagus linking the anterior and posterior walls transversely across the lumen.

Video 1

Mucosal bridging leads to double-lumen formation. The tunnel-like lumina can be intubated separately, and coalesce distally.

Histology showed epidermoid metaplasia of the esophagus, characterized by squamous epithelium with well-developed granular cell layer, hyperorthokeratosis with focal parakeratosis, and mild peripapillary lymphocytic infiltrate (original × 100).

In conclusion, mucosal bridges in the esophagus are rare. The association with epidermoid metaplasia, which is reported here for the first time, suggests common pathogenetic pathways related to injury and repair.

Competing interests: None

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