A 50-year-old woman presented with dysphagia since 1 week. She did not drink alcohol and there was no history of systemic disease, including dermatologic, immunologic, or genetic disease. The initial laboratory findings, complete blood count, and serum biochemistries were normal, except the serum vitamin B12 level, which was 52 pg/mL (normal range: 126–505 pg/mL). Upper endoscopy revealed pangastritis with whitish strips and pseudomembranes on the esophageal mucosa, which peed off similarly to eosinophilic esophagitis or a lesion of dermatologic origin (Fig. 1). The lesions, which presented as discrete patches starting in the upper esophagus, extended diffusely through the entire esophagus. While the gastric biopsy samples showed features of atrophic gastritis, the esophageal biopsy samples were interpreted as parakeratosis (Fig. 2). Serum antiparietal antibodies were also positive. The patient was diagnosed as having early-stage pernicious anemia and the control biopsy samples showed only minimal parakeratosis despite the short-term therapy (Fig. 4).

Diffuse esophageal parakeratosis is a rare endoscopic diagnosis and is associated with conditions such as tylosis, mucosal hyperkeratosis syndrome, pachyonychia congenita, ethanol exposure, duodenal reflux, riboflavin deficiency, and zinc deficiency [1–6]. Following this first report of pernicious anemia due to vitamin B12 deficiency leading to esophageal parakeratosis, we recommend adding it to the long list of etiologic factors of this condition. Our patient presented with the sole symptom of dysphagia, that is without the established findings of pernicious anemia, such as low hemoglobin, other cytopenias, neurologic findings, and hemolysis. Like the majority of reported cases of esophageal parakeratosis due to nutritional deficiencies, our patient’s symptoms and signs also resolved after replacement of the specific deficiency [7].

Dysphagia resolved with vitamin B12 therapy: a case of esophageal parakeratosis

Competing interests: None

References

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