Esophageal lesions in myeloproliferative neoplasms

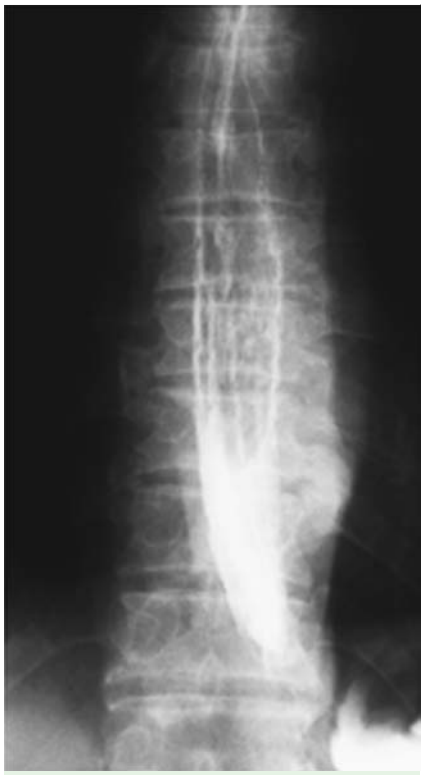

Fig. 1 Upper gastrointestinal barium study of a 67-year-old man with worsening odynophagia showing several longitudinal ulcers in the middle to lower esophagus.

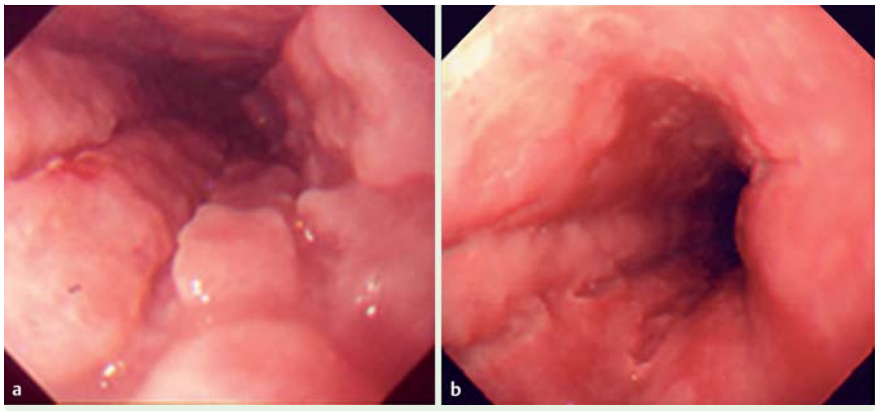

Fig. 2 Esophagogastroduodenoscopy showed multiple (**a**) longitudinal and (**b**) aphthoid ulcers, but no surrounding redness, white coat, or raised plaques in relation to the ulcers.

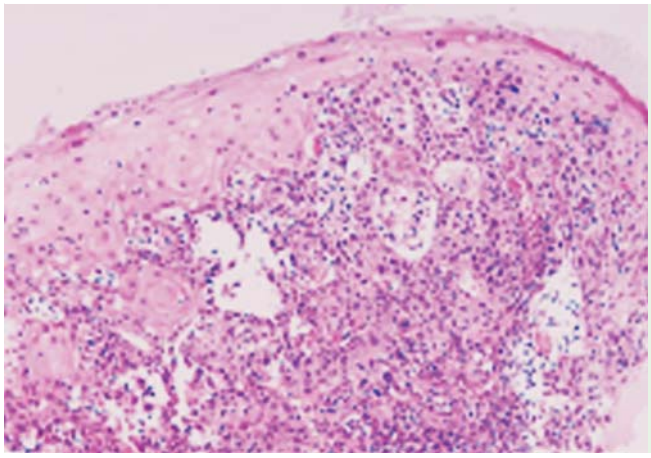

Fig. 3 Histological section (hematoxylin and eosin) showing intense infiltration of polymorphic neutrophils in the mucosa as well as the vascular wall. The biopsy specimen did not contain any microorganisms, inclusion bodies, gigantic cells, or granulomas.

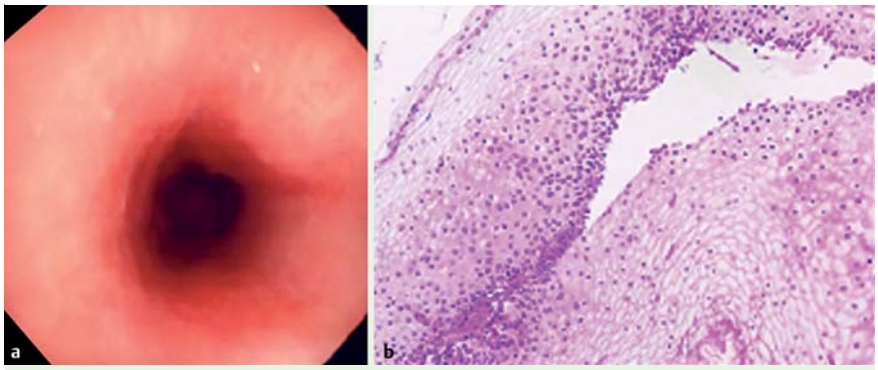

Fig. 4 Both the (**a**) endoscopic appearance and (**b**) histopathological picture were improved after chemotherapy.

A 67-year-old man was admitted to our hospital with worsening odynophagia. He had been diagnosed as having chronic neutrophilic leukemia based on excessive neutrophilia without blasts, anemia, hyperplastic bone marrow with normal neutrophilic maturation, and hepatosplenomegaly, without bcr/abl rearrangement [1]. Finally, he was rediagnosed as having myeloproliferative neoplasm (MPN), unclassifiable, according to the 2008 World Health Organization (WHO) classification [2]. In the 2 years after the diagnosis was made he was treated with hydroxyurea, interferon-alpha, Ara-C, and then VP-16.

Laboratory studies revealed leukocytosis (15.4×10^9/L, 69% mature neutrophils) without blasts, anemia (hemoglobin 10.2 g/dL), and thrombocytosis (569×10^9/L). Fluoroscopy revealed several longitudinal ulcers in the middle to lower esophagus (▶ **Fig. 1**). Esophagogastroduodenoscopy showed multiple longitudinal and aphthoid ulcers (▶ **Fig. 2**). There were not specific lesions in the stomach, duodenum, and colorectum. Histological examination revealed an intense infiltration of polymorphic neutrophils (mimicking the neoplastic cells detected in the bone marrow) in the mucosa as well as the vascular wall (▶ **Fig. 3**), indicating neoplastic cell infiltration to esophagus. Prednisolone 20 mg daily was prescribed in addition to VP-16, resulting in improvement of the esophageal lesions (▶ **Fig. 4**).

Esophageal manifestations in leukemic patients include hemorrhagic lesions, leukemic infiltrates, and pseudomembranous and fungal esophagitis [3,4]. Although esophageal involvement was reported in 7.2% of 207 autopsied cases

with leukemia, only a few cases have been diagnosed antemortem [5]. Endoscopically, the lesions include shallow circular ulcers and erosive esophagitis. In addition, the autopsy review reported that esophageal leukemia was related to a high initial leukocyte count and usually associated with leukemic infiltration into other soft tissue/organs. Odynophagia and dysphagia in patients with leukemia are possibly caused by chemotherapy toxicity, infection, reflux, and benign strictures, however, esophageal leukemic infiltration should also be considered. Besides the longitudinal and aphthoid ulcers present in our case, endoscopic appearances may vary depending on the characteristics of the infiltrating neoplastic cells.

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Competing interests: None

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