Inflammatory myofibroblastic tumor: an unusual submucosal lesion of the stomach

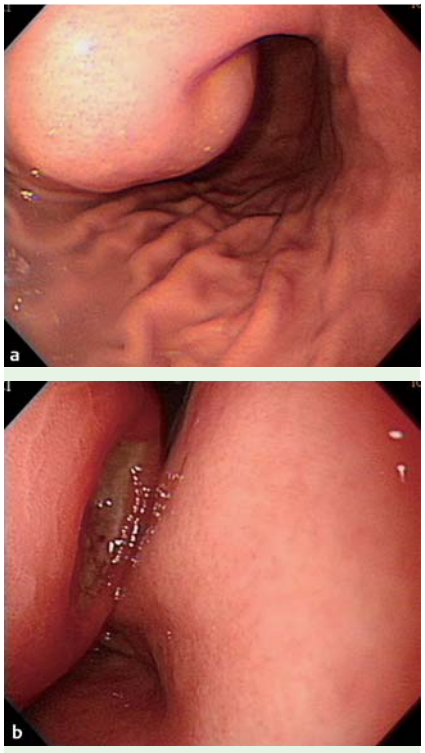

Fig. 1 **a, b** Upper endoscopy showing a broad-based, protruding mass, approximately 5.5 cm in size, in the anterior wall of lower gastric body. The tumor is accompanied by bridging folds and two deep ulcerations on the surface.

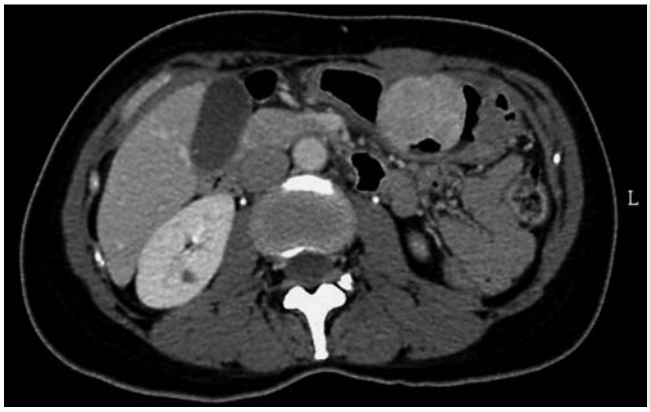

Fig. 2 Abdominal computed tomography (CT) scan demonstrating a strongly enhancing mass, approximately 5.5 cm in size, with surface ulceration, arising from the submucosal layer of the anterior wall of the lower gastric body.

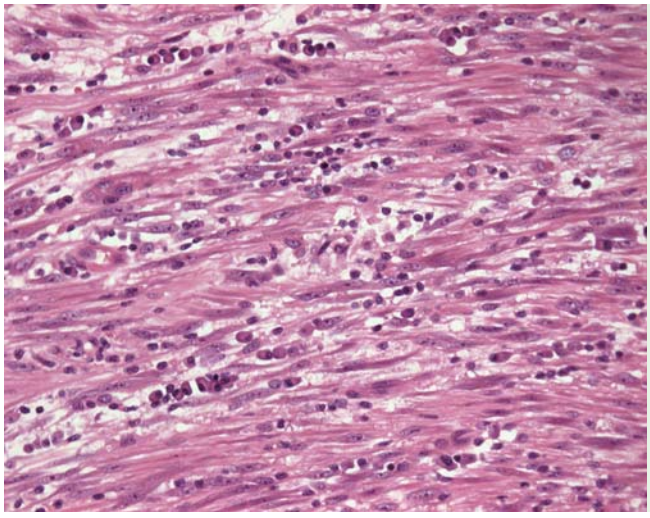

Fig. 3 Microscopic section showing the tumor composed of spindle cells with massive, predominantly inflammatory, infiltration of plasma cells (hematoxylin and eosin, magnification × 40).

Inflammatory myofibroblastic tumor (IMT) is a mesenchymal tumor that occurs preferentially in children and young adults. IMTs were considered arise as a result of a reactive inflammatory or post surgery process [1]. However, they are thought to have low-grade malignant potential, based on the recent molecular finding of rearrangement at chromosome band 2p23, the site of the anaplastic lymphoma kinase (*ALK*) gene in the tyrosine kinase locus [2]. They are most commonly found in the lung but may arise in extrapulmonary sites [3].

A 42-year-old woman presented with intermittent dull epigastric pain since 1 month and tarry stool passage since 1 week. The laboratory findings were unremarkable except for a normocytic anemia (hemoglobin 7.7 g/dL). Upper endoscopy revealed a broad-based, protruding mass of approximately 5.5 cm, located in the anterior wall of lower gastric body. The tumor was accompanied by bridging folds and two deep ulcerations on its surface (▶ **Fig. 1**). Abdominal computed tomography (CT) demonstrated a strongly enhancing mass with surface ulceration, arising from the submucosal layer (▶ **Fig. 2**), which was in keeping with a submucosal lesion such as a gastrointestinal stromal tumor (GIST). The patient underwent local tumor excision. Microscopically, the tumor was composed of spindle cells with massive infiltration of plasma cells (▶ **Fig. 3**). IMT was diagnosed by immunohistochemistry (IHC), which showed positive staining for desmin and smooth muscle actin and was negative for GIST markers including CD117, DOG1, CD34, and S100. Kit-negative GIST was further excluded as there no mutations in the c-KIT and PDGFRA genes.

Gastric IMT is very rare and may be confused with other submucosal lesions, especially GIST, and IHC studies are the only conclusive diagnostic modality [4]. When investigating a gastric submucosal lesion, IMT should be taken into consideration particularly if the patient is young or the pathology shows massive plasma cell infiltration admixed with spindle cells.

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Competing interests: None

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Bibliography

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