Inflammatory myofibroblastic tumor (IMT) is a mesenchymal tumor that occurs preferentially in children and young adults. IMTs were considered to 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 in size, located in the anterior wall of lower gastric body. The tumor was accompanied by bridging folds and two deep ulcerations on the surface. Abdominal computed tomography (CT) demonstrated 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.

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 were 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.

Competing interests: None
References


