A 73-year-old man with esophageal varices due to hepatitis C virus-induced cirrhosis has been followed by endoscopy since 1994. When a smooth, flat elevation first appeared in the gastric antrum in 2004 (Fig. 1a), our provisional diagnosis was gastric verrucosa. Follow-up endoscopy over the next 2 years showed no remarkable change, but in 2007 the lesion grew bigger (Fig. 1b) and a small ulcer appeared on the apex in March 2008 (Fig. 1c). Mesenchymal tumors such as gastrointestinal stromal tumor or malignant lymphoma were suspected but biopsy was inconclusive. After another 6 months the ulcer had extended downward (Fig. 1d) and exhibited the characteristic gross appearance of an inflammatory fibroid polyp (IFP). The polyp, removed endoscopically, was composed of spindle cells arranged in an onionskin-like concentric formation in the submucosal layer, accompanied by inflammatory cell infiltration, which predominantly consisted of eosinophils (Fig. 2).

On immunohistochemistry, the spindle cells were diffusely positive for PDGFRA (platelet-derived growth factor receptor α) (Fig. 3), focally positive for CD34, and negative for KIT. These findings were consistent with those of the classical IFP with concentric formation [1].

IFP is a rare mesenchymal tumor of the gastrointestinal tract and its natural history is unknown [2]. Our report demonstrates that it might take several years for gastric IFP to grow from an endoscopically discernible elevation into the characteristic submucosal tumor. Follow-up endoscopy showed no remarkable change.
Copy enabled not only the establishment of the correct diagnosis before resection but also endoscopic treatment before surgical intervention became inevitable. Since the first description by Vanek [3] in 1949, IFP has been regarded as a reactive polyp rather than a neoplastic lesion. In 2008, however, a seminal study by Schildhaus et al. first demonstrated ubiquitous PDGFRA overexpression and frequent gain-of-functional PDGFRA mutation in IFPs [1]. These findings, supported by subsequent studies [4,5], point to the neoplastic nature of IFP and the current case supports a crucial role for PDGFRA activation in gastric IFP.

**Competing interests:** None

**References**


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**Bibliography**

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