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 (\(\text{Fig. 1a}\)), 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 (\(\text{Fig. 1b}\)) and a small ulcer appeared on the apex in March 2008 (\(\text{Fig. 1c}\)). 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 (\(\text{Fig. 1d}\)) 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 (\(\text{Fig. 2}\)). On immunohistochemistry, the spindle cells were diffusely positive for PDGFRA (platelet-derived growth factor receptor \(\alpha\)) (\(\text{Fig. 3}\)), 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...
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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