

Plexiform fibromyxoma of the lesser curvature of the stomach in a 16-year-old adolescent: A case report and literature review

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Abstract. Gastric plexiform fibromyxoma (PF) is a rare mesenchymal tumor, with ~150 cases reported worldwide, predominantly occurring in the gastric antrum of adults. The present study describes a case of isolated PF in the lesser curvature of the stomach in a 16-year-old male patient. As confirmed by various multi-dimensional analyses of clinical, imaging and pathological data, the unique features of the case included its adolescent onset (only 12 cases of patients ≤18 years old have been reported globally) and the solitary localization in the lesser curvature of the stomach, with no previously documented cases. The patient presented with abdominal pain. A computed tomography scan revealed a solid intramural gastric mass with progressive enhancement. Postoperative pathology combined with immunohistochemistry (vimentin⁺, smooth muscle actin partially⁺, CD10⁺ and CD117/discovered on GIST-1) confirmed the diagnosis. No recurrence was observed during the 12-month follow-up. The present case expands the clinical spectrum of PF and emphasizes the value of multimodal diagnosis for tumors in rare locations.

Introduction

Gastric plexiform fibromyxoma (PF) is a rare mesenchymal neoplasm. Since its initial description in 2007 by Takahashi *et al* (1), ~150 cases have been documented in PubMed (<https://pubmed.ncbi.nlm.nih.gov>), predominantly affecting the gastric antrum of patients aged 40-50 years (2,3). Diagnosing PF poses notable challenges due to non-specific clinical manifestations and imaging features that are insufficient for standalone confirmation (4). To the best of our

knowledge, to date, the lesser curvature of the stomach as an independent site for PF has not been reported, and only ~12 cases of adolescent-onset PF (<18 years) have been described globally. The clinicopathological and immunophenotypic characteristics of these cases require further validation through additional studies (5). Additionally, the differential diagnosis of gastric submucosal tumors is complex, with PF needing distinction from gastrointestinal stromal tumors (GISTs), schwannomas and inflammatory myofibroblastic tumors, necessitating multi-dimensional evaluation. To the best of our knowledge, the present case, integrating multi-level diagnostic evidence, represents the first report of adolescent PF in the gastric lesser curvature, contributing to the clinical spectrum of this disease.

Case report

Clinical data. A 16-year-old male patient was admitted to Changsha Hospital of Traditional Chinese Medicine (The Eighth Hospital of Changsha; Changsha, China) in January 2025 presenting with recurrent epigastric pain that had persisted for 1 month accompanied by retching and acid regurgitation. A physical examination revealed mild epigastric tenderness. In addition, laboratory tests showed an elevated carbohydrate antigen 72-4 (CA72-4) level of 10.70 U/ml (reference range, 0-6.9 U/ml), measured by chemiluminescent immunoassay, with other tumor markers and biochemical indices within normal ranges. The patient had no notable medical history, and there was no family history of hereditary diseases or tumors. Gastroscopy identified a broad-based protrusion on the lesser curvature of the gastric body with congested but intact and smooth mucosa, and no abnormalities were found in the gastric antrum or duodenal bulb (Fig. 1A-C). Endoscopic ultrasonography demonstrated vascularity within the mass (Fig. 1D-F).

Imaging features. A computed tomography (CT) plain scan showed a 4.0x3.5-cm soft-tissue density mass in the gastric wall of the lesser curvature, presenting as a long ellipse with intraluminal growth, uniform density (CT value, 50 HU), intact overlying mucosa and no surrounding invasion (Fig. 2A). Enhanced CT demonstrated mild, relatively uniform enhancement in the arterial phase (CT value, 67

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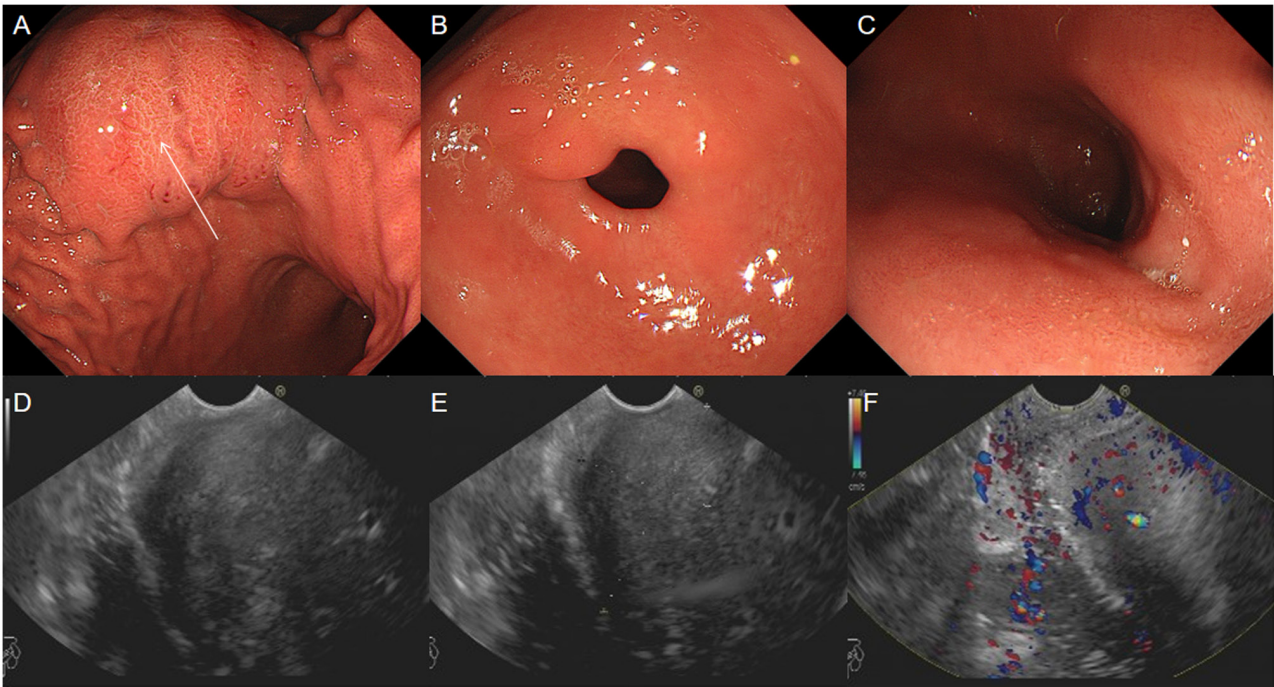


Figure 1. Gastroscopic findings of the gastric submucosal mass. (A) Large broad-based protruding lesion on the lesser curvature of the stomach (white arrow) with congested, smooth and intact mucosa. (B) Congested mucosa in the gastric antrum without erosion, ulceration or mass lesions. (C) Normal duodenal bulb with smooth mucosa and no abnormal protrusions or defects. (D-F) Endoscopic ultrasound performed 7 days after the above gastroscopy revealed a hypoechoic mass originating from the same gastric lesser curvature site as the lesion in (A); color Doppler imaging demonstrated visible vascular signals within the mass, indicating the presence of blood flow.

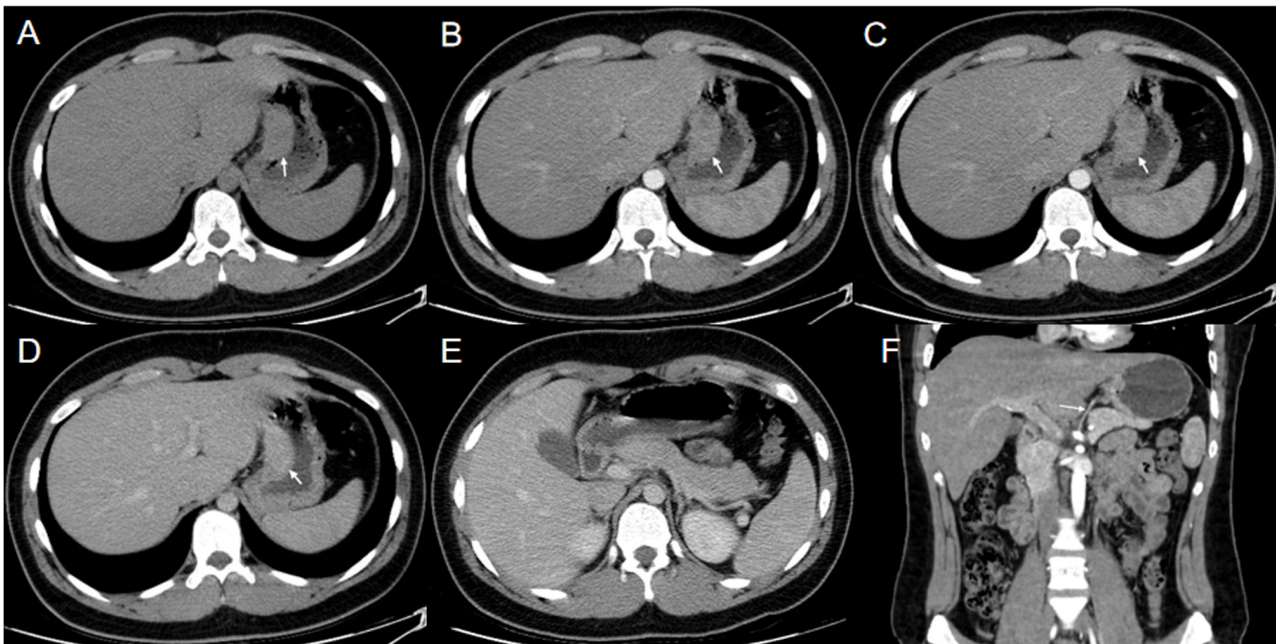


Figure 2. Computed tomography plain and enhanced imaging findings of the gastric lesser curvature mass. (A) Plain scan showing a soft-tissue mass in the gastric lesser curvature. (B) Mild enhancement in the arterial phase. (C) Enhancement in the portal venous phase. (D) Sustained enhancement in the delayed phase. (E) No mass in the gastric antrum level. (F) Coronal reconstruction showing the blood supply of the mass (white arrow).

HU; Fig. 2B), with progressive uniform enhancement in the portal venous phase (CT value, 96 HU; Fig. 2C) and delayed phase (CT value, 90 HU; Fig. 2D), exhibiting a 'progressive enhancement' pattern. No mass was observed in the gastric antrum (Fig. 2E). Finally, 3D reconstructed coronal

images showed a vascular supply from the left gastric artery (Fig. 2F).

Surgical and pathological results. The patient underwent a partial gastrectomy. Intraoperatively, a 5x4-cm hard mass was

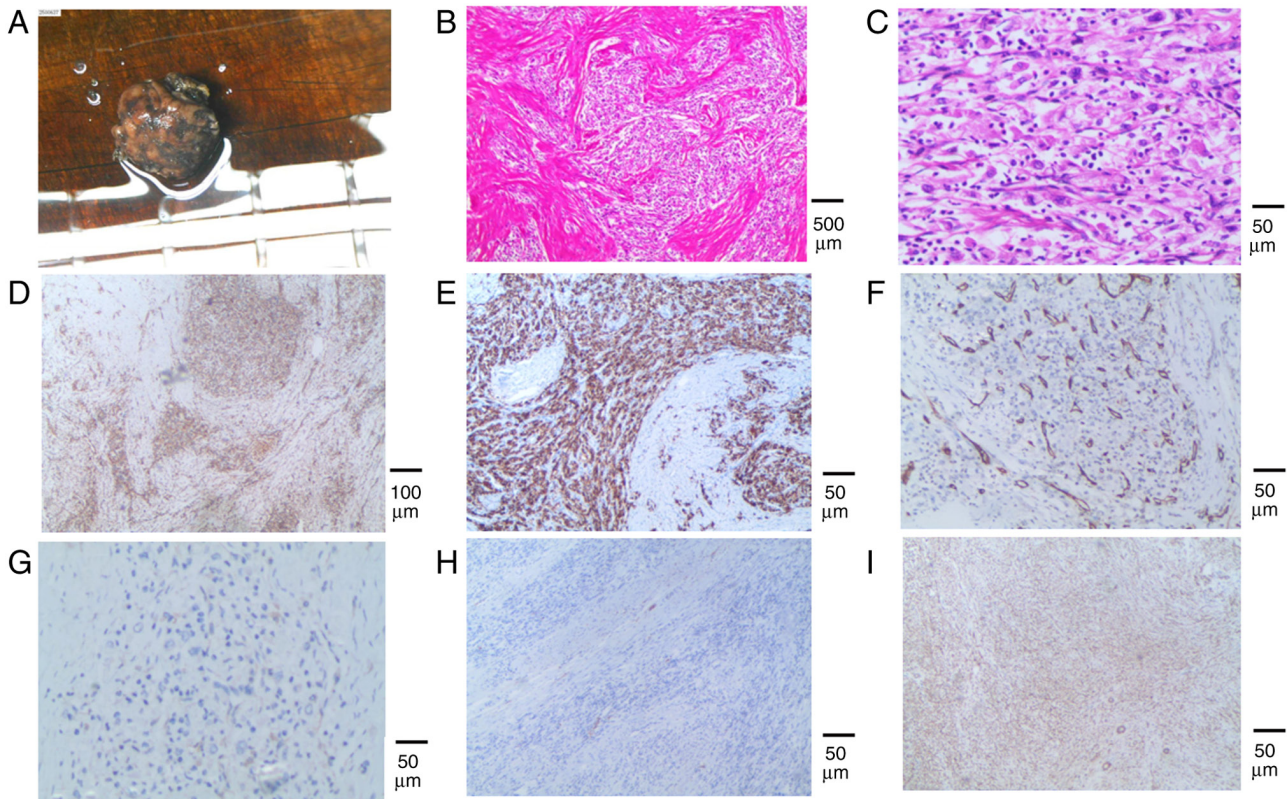


Figure 3. Gross specimen, pathological and immunohistochemical findings of the tumor. (A) Postoperative gross specimen showing a nodular mass in the submucosa of the gastric lesser curvature. The cut surface is gray-white with focal myxoid degeneration (consistent with the gross features of PF). (B) HE staining (magnification, x40). The tumor exhibits characteristic plexiform/multinodular growth, with myxoid components in the stroma (pathognomonic pathological morphology of PF). (C) HE staining (magnification, x400). The tumor is composed of spindle cells with bland morphology and no nuclear atypia. No mitotic figures are observed in 50 high-power fields, indicating low proliferative activity. (D) Vimentin staining (magnification, x200). Diffuse positive expression is noted in tumor cells, supporting a mesenchymal origin of the tumor. (E) Smooth muscle actin staining (magnification, x200). Focal positive expression is seen in tumor cells, consistent with the myofibroblastic differentiation property. (F) CD10 staining (magnification, x200). Partial positive expression is detected in tumor cells, a characteristic immunophenotype of PF, which helps in differentiating PF from a GIST (typically CD10⁻). (G) CD34 staining (magnification, x200). Tumor cells show negative expression, excluding the diagnosis of a GIST. (H) Discovered on GIST-1 staining (magnification, x200). Negative expression is observed in tumor cells, further excluding the diagnosis of a GIST. (I) S-100 staining (magnification, x200). Tumor cells exhibit negative expression, excluding the diagnosis of schwannoma. PF, plexiform fibromyxoma; HE, hematoxylin and eosin; GIST, gastrointestinal stromal tumor.

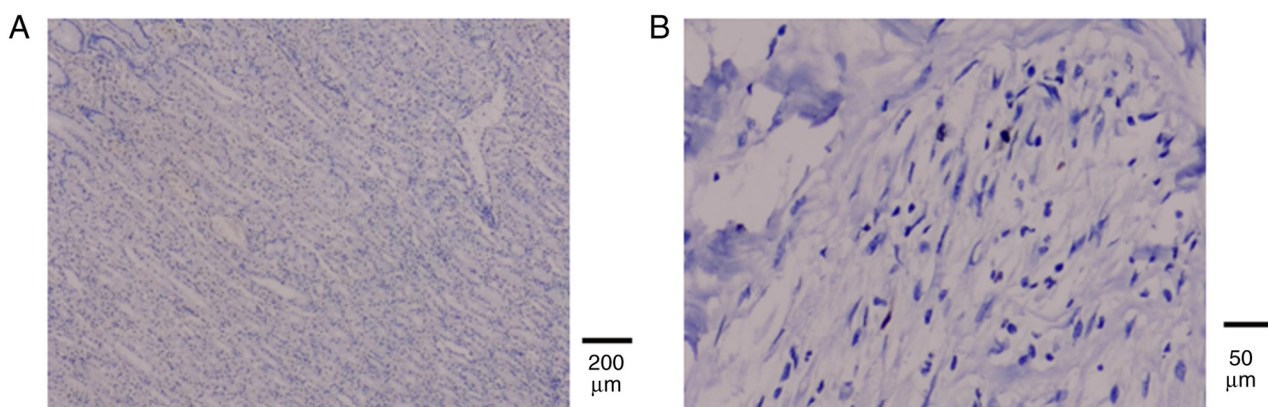


Figure 4. Additional immunohistochemical findings of the gastric plexiform fibromyxoma. (A) Anaplastic lymphoma kinase staining (magnification, x100). Tumor cells show negative expression, excluding the diagnosis of inflammatory myofibroblastic tumor. (B) Ki-67 staining (magnification, x400). The proliferative index in the hot spot area is ~10%, indicating low proliferative activity of the tumor.

identified in the lesser curvature of the gastric wall. No obvious metastatic lesions were found in the peritoneum or liver, and no firm lymph nodes were detected. Surgical resection specimens revealed a submucosal nodular mass with a gray-white cut surface

and focal myxoid degeneration (Fig. 3A). The specimens were fixed in 10% neutral buffered formalin at room temperature for 24 h, followed by routine dehydration at 35°C and embedding in paraffin at 60°C. Sections (3- to 5- μ m thick) were cut and

Table I. Primary and secondary antibodies used in this study.

Target antigen	Catalog number	Manufacturer	Dilution
S100	0925241009	Maxim Biotech, Inc.	Undiluted
CD34	110824111	Maxim Biotech, Inc.	Undiluted
Ki-67	0211250305	Maxim Biotech, Inc.	Undiluted
CD117	2503050632b	Maxim Biotech, Inc.	Undiluted
CD10	1218241225	Maxim Biotech, Inc.	Undiluted
Vimentin	0207230209	Maxim Biotech, Inc.	Undiluted
SMA	0120250211	Maxim Biotech, Inc.	Undiluted
DOG-1	0120250213	Maxim Biotech, Inc.	Undiluted
Secondary antibody detection system	2511125441as	Maxim Biotech, Inc.	1:10

SMA, smooth muscle actin; DOG-1, discovered on GIST-1.

subjected to standard hematoxylin and eosin staining (hematoxylin for 5 min and eosin for 2 min, both at room temperature), followed by dehydration and mounting for observation under a light microscope. For immunohistochemical staining, the paraffin-embedded tissue sections were used for subsequent procedures as follows: Deparaffinization and rehydration, followed by antigen retrieval in EDTA buffer (pH 9.0) for 6 min and PBS washing. Endogenous peroxidase activity was blocked with 3% H₂O₂ at room temperature for 15 min, then washing was performed with PBS. Specific undiluted primary antibody (Maxim Biotech, Inc.) (Table I) was added and incubated at 37°C for 1 h, followed by washing with PBS. Signal amplification reagent was added and incubated at room temperature for 15 min, followed by washing with PBS. Secondary antibody detection system (cat. no. 2511125441as; diluted 1:10; Maxim Biotech, Inc.) was added and incubated at room temperature for 30 min, followed by PBS washing. Staining was visualized with DAB chromogen, followed by washing with water, hematoxylin counterstaining and coverslipping.

Microscopically, tumor cells were arranged in a plexiform/nodular pattern, with spindle to oval cells infiltrating the gastric wall smooth muscle, accompanied by myxoid stroma and small vessel proliferation, with 0 mitoses per 50 high-power fields (Fig. 3B and C). The results of the immunohistochemistry were as follows: Vimentin⁺ (Fig. 3D), smooth muscle actin (SMA) focally⁺ (Fig. 3E), CD10⁺ (Fig. 3F), CD34⁺ (Fig. 3G), discovered on GIST-1 (DOG-1)⁺ (Fig. 3H), S-100⁺ (Fig. 3I), anaplastic lymphoma kinase (ALK)⁺ (Fig. 4A) and a Ki-67 index of 10% found in hotspots (Fig. 4B).

Treatment and follow-up. A laparoscopic gastric mass resection (partial gastrectomy) was performed under general anesthesia several days after admission. A 5x4-cm firm mass, located 3 cm from the cardia on the lesser curvature of the gastric body, was completely resected, and the resulting gastric wall defect was sutured and reinforced. Postoperatively, supportive interventions, including acid suppression, hemostasis, fluid replacement, nutritional support and symptomatic management, were administered. Postoperative hematemeses was resolved following targeted hemostatic treatments, and the patient was discharged after an uneventful recovery. The final diagnosis of gastric plexiform fibromyxoma was

confirmed by histopathological examination of the resected specimen (Figs. 3A-I). No tumor recurrence was observed during the 12-month follow-up, which included clinical and imaging assessments every 6 months; the patient maintained a favorable quality of life.

Discussion

Gastric PF primarily affects patients aged 40-50 years old with no notable sex predilection, presenting with non-specific gastrointestinal symptoms and with a predominant location in the gastric antrum, occasionally involving the esophagus and duodenum (4-6). The case described in the present study is notable for the following two rare features: i) Adolescent onset, with only 12 adolescent PF cases cumulatively reported in the literature (7); and ii) solitary localization in the gastric lesser curvature without involvement of other gastric wall regions, a distribution not previously reported to the best of our knowledge. An inflammatory myofibroblastic tumor (IMT) was initially suspected in the present case, primarily due to the rarity of PF in adolescents and the marked clinical overlap between the presenting symptoms of the patient (recurrent epigastric pain accompanied by nausea, retching and acid regurgitation) and those commonly associated with submucosal gastric tumors such as IMT and GIST. Notably, these entities lack pathognomonic clinical or imaging features that would allow reliable differentiation at initial presentation. Following endoscopic and imaging identification of a submucosal mass located along the lesser curvature of the stomach, a mild elevation in serum CA72-4 was observed (10.70 U/ml; reference range, 0-6.9 U/ml) in the present case. Although non-specific, this finding supported a higher likelihood of a benign neoplasm and informed the clinical decision to proceed with comprehensive histopathological analyses for a definitive diagnosis.

Gastric PF has characteristic imaging features. CT plain scans typically show a solid intramural mass with relatively uniform density, occasionally heterogeneous due to myxoid degeneration or hemorrhage. Enhanced CT demonstrates mild to moderate arterial phase enhancement, with persistent uniform progressive enhancement in portal and delayed phases, attributed to rich thin-walled vessels and fibromyxoid

Table II. Comparison of differential diagnostic points between PF, GIST, IMT and schwannoma.

Disease	Age of onset	Immunohistochemical features	Imaging differences	(Refs.)
PF	Mainly adults; mean, 43.1±18.0 years; median, 46 years; rare in adolescents	Vimentin 100%+, SMA+, MSA+, CD10 focal+, CD117/DOG-1-, S-100-	Predominantly intraluminal, progressive enhancement, mostly intact mucosa, occasional bleeding-related ulceration/erosion	(1,12-14,16-20,25)
GIST	Middle-aged and elderly; 82% ≥50 years; 0.44% <20 years	CD117+, DOG-1+, CD34+	Often extraluminal, heterogeneous density, intense arterial enhancement, ulceration, bleeding, cystic change; potential malignancy with liver/spleen metastasis	(9,15,21-23,28,29)
IMT	Children/adolescents; adult:child ratio ~5:1	ALK+ subset, SMA+, vimentin+	Heterogeneous density/signal, variable margins, diverse enhancement, possible cystic change/necrosis	(1,14,24,25)
Schwannoma	Adults; 40-60 years; female predilection	S-100+, SOX10+, GFAP+, CD117/DOG-1-	Mostly extraluminal, uniform density, progressive enhancement, intact capsule, rare cystic change	(1,16,18,19,27,30)

PF, plexiform fibromyxoma; GIST, gastrointestinal stromal tumor; IMT, inflammatory myofibroblastic tumor; DOG-1, discovered on GIST-1; GFAP, glial fibrillary acidic protein; MSA, muscle-specific actin.

stroma causing slow contrast agent diffusion and clearance (3). The tumor is predominantly intraluminal with intact overlying mucosa, rarely penetrating the serosa or mucosa, and is typically non-invasive (8). The CT findings in the present case, including a solid intramural mass, progressive enhancement and left gastric artery supply, align with the literature. Notably, a clear association exists between the imaging features and histopathological findings in the present case, including: i) The homogeneous attenuation of the mass on unenhanced CT (50 HU) corresponding to the histologically regular arrangement of tumor cells, limited myxoid stromal change and absence of hemorrhage or necrosis; ii) the ‘progressive enhancement’ pattern observed on contrast-enhanced CT reflecting the rich vascular network of the tumor, characterized by proliferating small vessels with thin walls and its fibromyxoid stroma. These histological features facilitate slow diffusion and prolonged retention of contrast medium, resulting in greater enhancement during the portal venous and delayed phases compared with the arterial phase; and iii) the intraluminal growth of the mass with an intact overlying mucosa is consistent with the pathological finding that the tumor is confined to the submucosa, without invasion of either the mucosal or serosal layers. This imaging-pathology association provides a valuable basis for a preoperative presumptive diagnosis, particularly in cases where preoperative biopsy specimens are unavailable or non-diagnostic. By contrast, GIST typically shows intense arterial enhancement, ulceration, extraluminal growth and heterogeneous enhancement (9). The left gastric artery supply aids in distinguishing non-gastric tumors and

guides surgical planning. In addition, ~15% of PF cases may have cystic changes (10), which were absent in the present case, potentially related to tumor differentiation. Initial consideration of a GIST reflected its predominance in adult gastric wall masses, occasional uniform enhancement in low-risk GISTs and limited familiarity with PF. However, key differentiators were not initially considered in the differential diagnosis, including low adolescent GIST incidence and intact mucosa without ulceration, which are uncommon in GIST. Magnetic resonance imaging outperforms CT in delineating tumor details and invasion, serving as a valuable adjunct in complex cases (6).

Gastric PF is characterized by plexiform and nodular arrangements of spindle to oval cells in a fibromyxoid stroma, with minimal atypia, rare mitoses and abundant thin-walled vessels (2,5). Immunohistochemically, tumor cells typically express vimentin and SMA, with focal CD10 and H-caldesmon, and are negative for CD117, DOG-1, CD34 and S-100 (2,3). The plexiform growth pattern is pathognomonic, and tumors lacking this pattern are unlikely to be PF (1). In the present study, the initial misdiagnosis as IMT resulted from insufficient recognition of the characteristic plexiform/nodular architecture of PF under low magnification, focusing instead on spindle cells and lymphocytic infiltration. Additionally, CD10, a key PF marker, was not initially tested for, with diagnosis confirmed through multidisciplinary consultation and supplementary CD10 staining. Unlike IMT, PF lacks ALK positivity and marked inflammatory infiltration (4,11). Unlike GIST, PF is negative for CD117/DOG-1 and lacks KIT proto-oncogene

receptor tyrosine kinase/platelet-derived growth factor receptor α (C-KIT/PDGFR α) mutations. Schwannomas, positive for neural markers (S-100/SOX-10), differ from the vimentin⁺/SMA⁺/CD10⁺ immunophenotype of PF (5).

To accurately differentiate PF from other common gastric mesenchymal tumors (GIST, IMT and schwannoma), the differential diagnostic points from four dimensions are summarized in Table II as follows: i) Age of onset; ii) immunohistochemical features; iii) genetic characteristics; and iv) imaging findings, with further analysis combined with the features of the present case.

PF predominantly affects adults (mean age, 43.1 years; median age, 46 years) and is rare in adolescents (12-14). By contrast, GIST is more common in middle-aged and elderly individuals (82% aged \geq 50 years; only 0.44% aged <20 years) (15), IMT is frequently seen in children/adolescents (1,14) and schwannoma mainly occurs in women aged 40-60 years (16). The present case involved a 16-year-old male patient, which is outside the typical age range for PF. However, sporadic cases of adolescent PF have been reported, indicating that PF should be considered in the differential diagnosis of gastric tumors in adolescents.

The core immunophenotype of PF is characterized by vimentin (100%⁺) (1,17,18), SMA⁺ and muscle-specific actin⁺ expression (1,15,19), focal CD10 positivity in some cases (1,20), and negativity for CD117/DOG-1 (21) and S-100 (18,19). GIST is marked by CD117⁺, DOG-1⁺ and CD34⁺ expression (21-23). IMT is often ALK(focal)⁺, SMA⁺ (1,24) and vimentin⁺ (1,25). Schwannoma is characterized by S-100⁺, SOX10⁺ and GFAP⁺ expression (26,27), and negativity for CD117/DOG-1 (1,19). The immunohistochemical results of the present case showed vimentin⁺, focal SMA⁺, partial CD10⁺ and CD117/DOG-1⁻ expression, which was fully consistent with the immunophenotype of PF. Meanwhile, GIST was excluded due to negative CD117/DOG-1 expression, schwannoma was ruled out due to negative S-100 expression and IMT was eliminated as there was no evidence of ALK positivity.

PF mainly grows intraluminally, showing 'progressive enhancement' on enhanced scanning, with mostly intact mucosa (occasional ulceration/erosion related to bleeding) (18,19). However, GIST is more prone to extraluminal growth, typically presenting with heterogeneous density and obvious, heterogeneous enhancement in the arterial phase, and frequent complications such as ulceration, bleeding and cystic change (9), which may present with malignant features such as liver/spleen metastasis (15,28,29). IMT exhibits heterogeneous density, clear or infiltrative margins, diverse enhancement patterns and possible cystic change/necrosis (24). Schwannoma mainly grows extraluminally, with relatively uniform density, progressive enhancement and an intact capsule (rare cystic change) (27,30). The CT findings of the present case, including a submucosal mass growing intraluminally in the gastric lesser curvature with progressive enhancement, were highly consistent with the imaging pattern of PF, further differentiating it from other tumors (such as the tendency of GISTs to grow extraluminally and the cystic change/necrosis features of IMTs). It is worth emphasizing that several key pathological features possess exclusive diagnostic value in the differential diagnosis of PF, including: i) The plexiform or nodular growth pattern constituting the morphological 'gold standard' for

PF. This architecture is distinctly different from the diffuse spindle cell infiltration observed in IMT and the fascicular arrangement of spindle or epithelioid cells characteristic of GIST, enabling reliable preliminary distinction even at low magnification; ii) partial immunohistochemical positivity for CD10 serving as a valuable ancillary marker for PF. By contrast, neither GISTs nor schwannomas express CD10, allowing these two common submucosal tumors to be effectively excluded based on this single marker. The integrated use of these multidimensional diagnostic criteria notably enhances the accuracy of differential diagnosis for rare gastric mesenchymal neoplasms.

In conclusion, PF can be effectively distinguished from GISTs, IMTs and schwannomas through multi-dimensional integrated analysis of age, immunohistochemistry and imaging. The unique features of the present case (adolescent onset and rare location in the gastric lesser curvature) not only enrich the clinical phenotype spectrum of PF but also highlight the key role of multimodal diagnosis in the differential diagnosis of rare tumors.

The current literature reports no recurrence or metastasis after PF resection (3,7,8). However, the present adolescent case with a Ki-67 index of 10% in hotspots carries a potential recurrence risk, highlighting the need for awareness of clinical outcome heterogeneity. Surgical resection (including endoscopic resection with negative margins) is the only established effective treatment (3). Endoscopic resection feasibility depends on tumor size, location and operator expertise, requiring multimodal imaging assessment. Postoperative endoscopic and imaging surveillance at 6-12 months is recommended for monitoring for recurrence or metastasis.

In conclusion, to the best of our knowledge, the present study is the first report of isolated adolescent PF in the gastric lesser curvature, expanding understanding of its clinicopathological features. PF should be considered in adolescent patients with lesser curvature submucosal masses, with multimodal diagnosis (morphology and immunohistochemistry) to avoid misdiagnosis. Further cases are needed to clarify the biological behavior and optimal management of adolescent PF.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

DS was responsible for the final review of pathological data, revision and finalization of the manuscript, and acting as the

corresponding author. ZF was responsible for the collection and analysis of CT images, and participating in the drafting of the initial manuscript. PT was responsible for the collection, collation and verification of clinical case data, as well as the systematic literature search and summary. XC was responsible for the collation and preliminary interpretation of pathological data. DS and ZF confirm the authenticity of all the raw data. All authors have read and approved the manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Written informed consent was obtained from the guardian of the patient for publication of the present case and any accompanying images.

Competing interests

The authors declare that they have no competing interests.

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