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Case Report

Primary intraperitoneal solitary fibrous tumor in mesentery: How does it present?☆

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ABSTRACT

Intra-abdominal solitary fibrous tumor (SFT), also known as hemangiopericytoma, is rare, especially for those with a mesenteric location, and only a few cases have been reported. Distinguishing a hemangiopericytoma from other intra-abdominal benign or malignant tumors can be difficult, as they have similar presentations on both computed tomography and magnetic resonance imaging. In the present study, the records for a 31-year-old Vietnamese woman who underwent abdominal surgery for greater omental tumor resection and received histopathological results revealing SFT are retrospectively reviewed. The case is discussed and similar reported cases are reviewed. Due to the aggressive behavior and high rate of postoperative recurrence associated with SFT, a thorough understanding of the radiologic and histopathological features of the disease is necessary to achieve an appropriate diagnosis and treatment.

Introduction

Hemangiopericytoma, also known as a solitary fibrous tumor (SFT), is an uncommon mesenchymal tumor, which accounts for <2% of soft tissue tumors [1]. In most cases, SFT derives from the fibroblastic or myofibroblastic cells found on capillary walls. This neoplasm can present anywhere throughout the human body and has a high risk of malignant transformation [2,3]. Zimmermann pericytes, which regulate the blood

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flow, are currently believed to serve as the origins for SFT, as these tumors are always found in the perivascular space. SFT is most commonly found in the pleura, meningeal membrane, retroperitoneal cavity, and intramuscular areas of the lower limbs, head, and neck [2]. Although SFTs can appear anywhere throughout the body, an SFT typically appears as a hypervascular tumor, with a high risk of intra-operative hemorrhage [2]. On clinical examination, symptoms often vary depending on the size, location and malignant features. To the best of our knowledge, only 21 cases of primary intraperitoneal SFTs have been reported with an associated histopathological diagnosis [4–17]. Therefore, the present study aimed to report the clinical presentation, computed tomography (CT) findings, and histopathological features of a case of intraperitoneal SFT.

Case report

A 31-year-old female patient was referred to Viet Duc Hospital (Hanoi, Vietnam) on May 2021 with the chief complaint of progressive abdominal distention lasting 2 months. No marked medical history was identified for either the patient or their family. On clinical examination, the patient presented with moderate ascites, no pain, and no palpable mass. The abdominal shape was within the normal range, and no changes in biochemical reports were documented. The patient underwent an abdominal ultrasound, which revealed a well-defined hyperechoic mass in the left lumbar region, with intratumoral cysts and dilation of the surrounding blood vessels. Additionally, large volumes of free abdominal fluid and a left pleural effusion were detected.

On abdominal CT, a 34 × 76 mm mass was detected on the superior left side of the abdomen, corresponding with the cystic mass identified on ultrasound. After contrast injection, the mass presented with vividly homogenous enhancing features in both the arterial and venous phases. The tumor received a large blood supply from the right gastro-omental artery, which is a branch of the gastroduodenal artery, with serpiginous dilation observed around the periphery of the mass. The well-defined appearance of this tumor indicated benign behavior with no invasive signs (Figs. 1 and 2).

The patient then underwent abdominal endoscopic surgery for tumor resection. The surgeon operated reported a mass located inside the greater omentum in the left lumbar region, with an abundant blood supply, and without any peripherally invasive signs. The postoperative histopathological diagnosis () revealed that the tumor was composed entirely of spindle cells, with a low mitotic count, a large volume of eosinophilic cytoplasm and no necrosis. For histopathology, a tumor sample was fixed in 10% formalin solution for 24 hour at room temperature, sliced into 5-μm sections, paraffin embedded, and stained with hematoxylin and eosin for 4 minute at room temperature; images were captured using a light microscope (magnification, x40-400). The tumor cells were arranged in the perivascular space. A dilated, branching, hyalinized, staghorn-like (hemangiopericytoma-like) vasculature was observed. Immunohistochemical features aligned with those described for primary omental hemangiopericytoma, including positive staining for signal transducer and activator of transcription 6 (STAT6), B-cell lymphoma 2 (BCL2), hematopoietic progenitor cell antigen CD34 (CD34), and Ki-67 (3%), and negative staining for discovered on GIST 1 (also known as anoctamin 1), CD117, CD10, S-100 protein, estrogen and progesterone receptors, transducin-like enhancer 1 and smooth muscle actin (Fig. 3). Immunohistochemical assays were performed on formalin-fixed paraffin-embedded 5-μm tissue sections, which were blocked with Background Sniper (cat. no. BS966G; Biocare Medical) at room temperature for 20 minute. Subsequently, tissues were incubated with a primary STAT6 antibody (1:100 dilution; rabbit monoclonal; cat. no. EP325; Cell Marque; MilliporeSigma) at room temperature for 12 hour. Tissues were then incubated with a secondary antibody (OmniMap anti-Rabbit HRP; Ventana Medical Systems, Inc.; Roche Diagnostics) for 32 minute at 37°C. CD34 (MAB-0034, QBEnd/10, cat. no. MS-363; Fuzhou Maixin Biotech Co., Ltd.), CD99 (MAB-0059, O13, cat. no. MS-294; Fuzhou Maixin Biotech Co. Ltd.) and Bcl-2 (RMA-0660, SP66, cat. no. sc-8543; Fuzhou Maixin Biotech Co., Ltd.) antibodies. After more than 9 months of postoperative observation and follow-up, the patient remained free of recurrence. Written informed consent was obtained from the patient for publication of this study.

Discussion

Hemangiopericytomas, also known as STFs, are infrequent, fibroblastic, mesenchymal neoplasms that derive from Zimmerman pericytes, which were first characterized by Zimmerman in 1923 [18] as pericytic, modified, smooth muscle cells that line the blood vessels and are responsible for vessel spasms. In 1942, Stout and Murray [19] named the pericyte neoplasms hemangiopericytoma to better describe the fact that the tumors were comprised of dilated vessels and associated Zimmerman pericytes. SFTs are more common with increasing age, particularly between 50 and 60 years, with no sex-based predilection [20]. The 2013 World Health Organization (WHO) classification of tumors of soft tissue and bone [21] combined hemangiopericytoma and SFT into a single entity, as they share immunohistochemical nuclear staining characteristics, including positivity for STAT6. During long-term follow-up, the risk of secondary lesions can be as high as 35%-45% [22]. The 2020 WHO classification of tumors of soft tissue and bone [23] indicated that the terms ‘typical’ or ‘atypical’ should not be used to describe SFTs with unpredictable or aggressive behavior, as typical SFTs are not always benign lesions. Recently, risk stratification models have been developed to assess the possibility of metastasis development [23]. SFTs have been described in various sites, most frequently in the pleura, pelvis, and retroperitoneum, followed by the pericardium, mediastinum, and facial sinuses [21,24].

Clinically, most patients present with asymptomatic SFTs; however, certain patients experience non-specific symptoms, such as mild to moderate ascites, the presence of a palpable mass or pain due to tumor compression of adjacent structures [21]. In total, <10% of cases are diagnosed with paraneoplastic syndromes, such as hypoglycemia (Doege-Potte syndrome

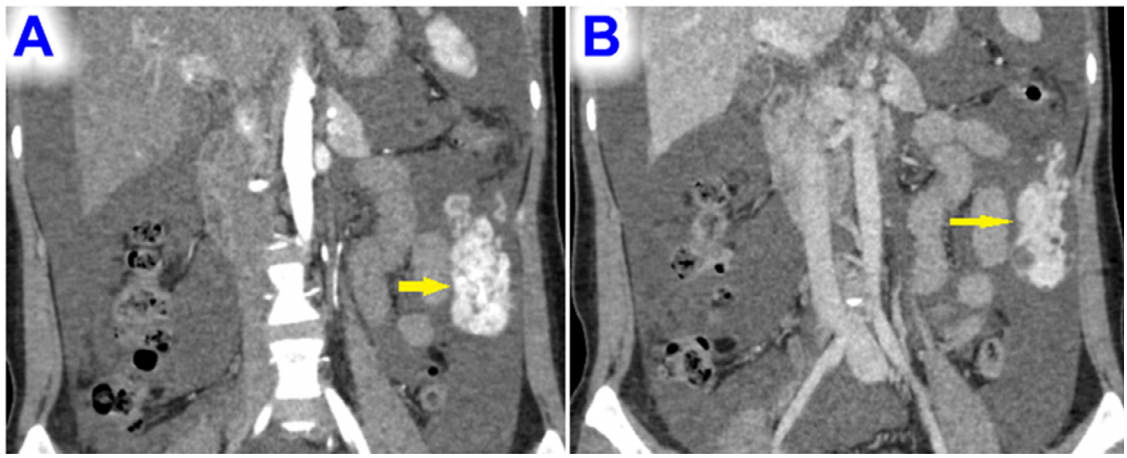

Fig. 1 – Retroperitoneal STF in the CT scan. Axial and coronal contrast-enhanced computed tomography images showing a well-defined mass (long arrows) in the left lumbar region, with vivid and homogenous enhancement in both the (A) arterial phase and the (B) venous phase. Large volumes of free intra-abdominal fluid were also observed.

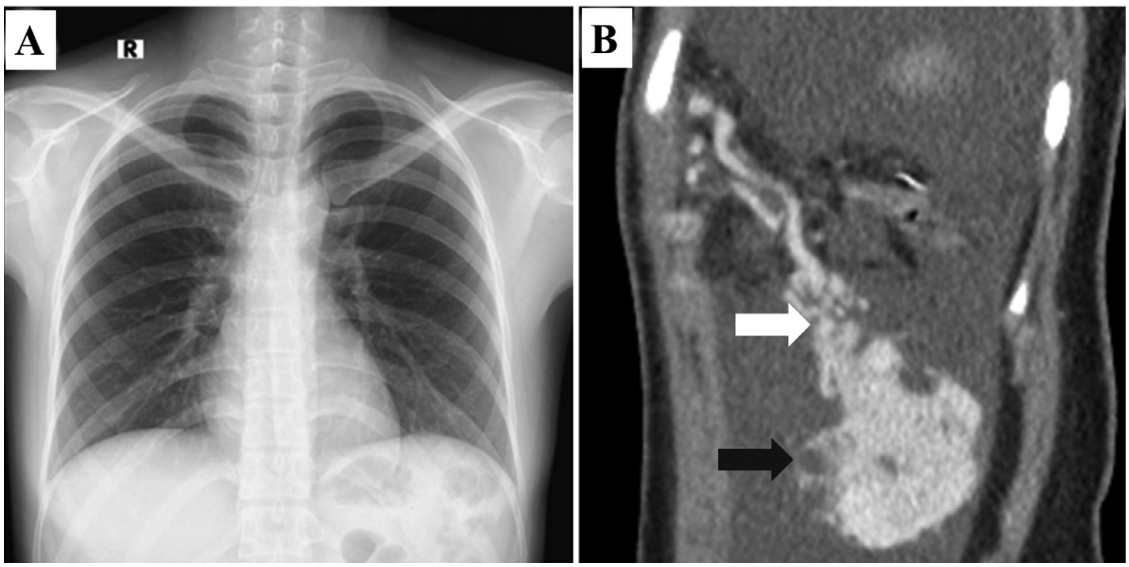

Fig. 2 – Chest X-ray and detection of a retroperitoneal solitary fibrous tumor via CT. (A) Anteroposterior chest X-ray showing no abnormal findings. (B) Sagittal contrast-enhanced computed tomography showing a serpiginous dilation of the right gastro-omental artery, which is the feeding artery (white arrow). Intratumoral cystic components are also present (black arrow).

due to the excessive production of insulin-like growth factor II from non-islet cell tumors), hypertrophic osteoarthropathy (with symptoms such as finger clubbing, hypertrophic skin changes, arthralgia and increased periosteal activity) or low concentrations of growth hormones and insulin-like growth factor I or III [19,21,25]. The patient in the current study presented with an SFT in the greater omentum, associated with ascites, no pain and no palpable mass. No other changes in either paraneoplastic syndromes or biochemical measures were recorded. This case was similar to those in a previous study conducted in 2018 by Fernandez *et al* [2], which described 7 SFT cases (3 patients with liver SFTs and 4 with SFTs in the pelvis) and suggested that abdominopelvic STFs may be less likely to present with paraneoplastic syndromes than pleural SFTs.

SFTs can appear as a well-defined, solid mass in all locations, with globular or elliptical shapes and colors ranging from white to yellow tan. SFTs are hypervascular tumors characterized by numerous peripheral blood supplies, associated with varying levels of interior necrosis, and rarely present with calcification components [2,21]. Histopathological imaging analysis demonstrates a tumor with randomly organized cells, spindled or oval in shape, with mixed hypercellular and hypocellular regions within a predominantly collagenous stroma, associated with plentiful thin-walled branching blood vessels that form staghorn shapes [21,25]. Intra-

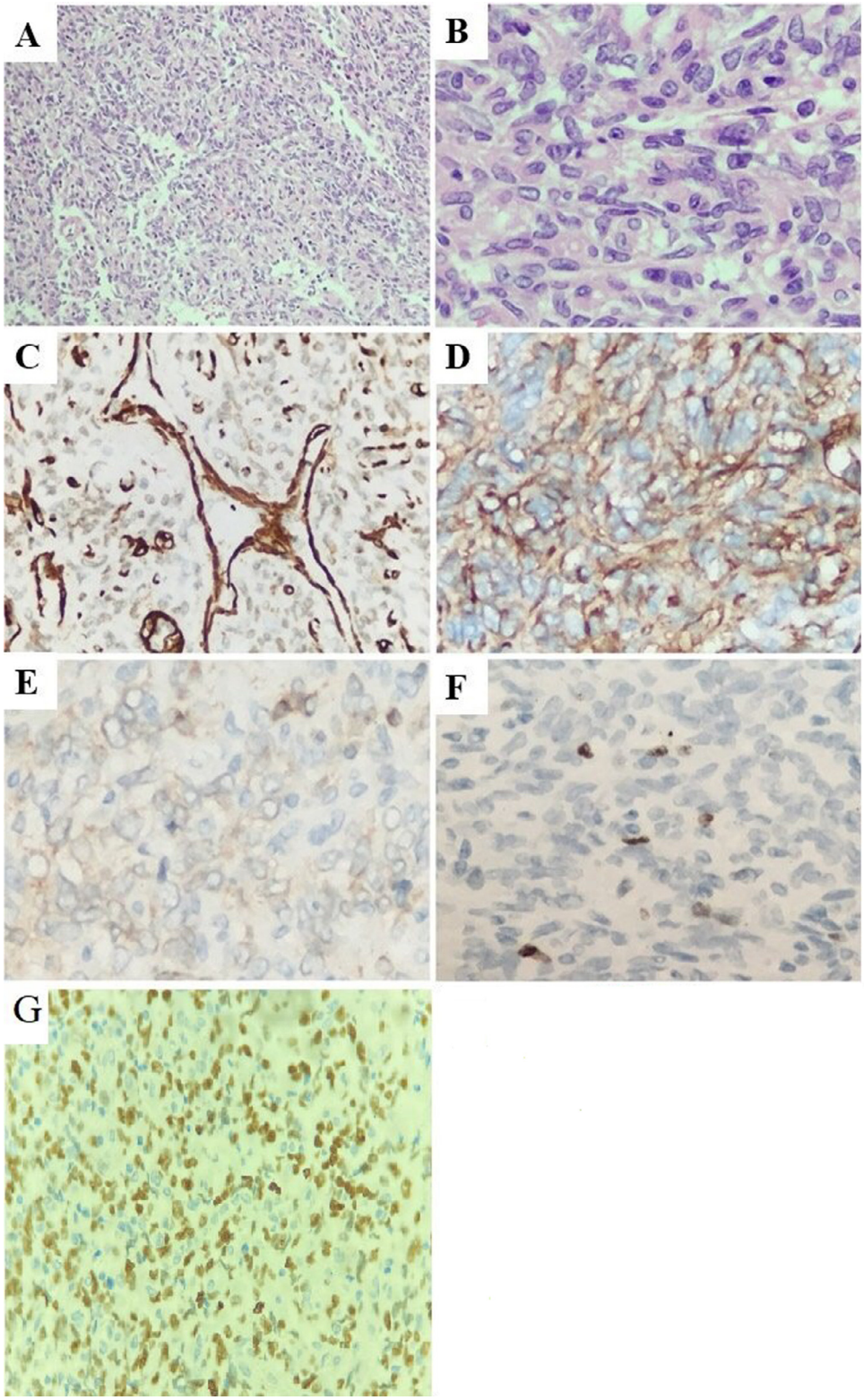

Fig. 3 – Pathological features of the SFT in the mesentery under a light microscope. (A and B) Hematoxylin and eosin staining at (A) x100 and (B) x400 magnification. Microscopic findings indicate hypercellular SFT with randomly organized, spindle-shaped cells, including elongated, ovoid to spindle nuclei, and scant indiscernible cytoplasm, within a predominantly collagenous stroma and thin-walled "staghorn" vessels. (C-F) Immunohistochemical staining at x400 magnification. (C) Immunohistochemical assay showing the negative reactivity of the tumor cells for SMA compared with the intense cytoplasmic immunostaining for SMA in adjacent vascular smooth muscle cells. (D) The tumor demonstrates diffuse and strong positive cytoplasmic staining for CD34. (E) The tumor cells exhibit weak and scattered cytoplasmic staining for Bcl2. (F) Ki-67-positive cells (3%). (G) Immunohistochemical assay showing the positive reactivity of the tumor cells for STAT6. SFT, solitary fibrous tumor; SMA, smooth muscle actin.

tumoral necrosis or hemorrhage may also be detected. Malignant SFTs are often larger than benign SFTs, with infiltrative borders, necrosis and hemorrhage commonly observed. Malignant SFTs consist of atypical cells with nuclear polymorphism and pleomorphism, a high number of mitotic counts (>4 mitoses/10 high-powered fields [HPF]). Preadipocytes and well-differentiated liposarcoma may be found within the tumor tissue, particularly in lipomatous SFTs [21,24,26]. Immunohistochemical studies have detected the presence of excessive expression of STAT6, which serves as a highly sensitive and specific marker for SFTs. SFTs are also reactive for CD34, CD99, vimentin and BCL-2, with negative nuclear immunostaining for S100, keratin and actin [21,27]. Malignant SFTs tend to lose CD34 nuclear staining and overexpress p53 and S100. Additionally, a recent study suggested that a Ki-67 (MIB-1) proliferation index of >5% that is expressed during G_1, S and G_2, which are the active mitosis phases of the cell cycle, is substantially correlated with recurrence [28].

After long-term follow-up, Demicco *et al* [29] concluded that the aggressive behaviors of SFTs could not be predicted based solely on histological features. Therefore, risk stratification models were generated to estimate the risks of secondary metastatic SFTs [28]. The model combined age (<55 years or ≥55 years), largest tumor dimension (<5, 5 to <15 and >15 cm), mitosis (0, 1 to 3 or ≥4/10 HPF) and necrosis (<10 or ≥10%). Scores range from 0 to 3 for each risk factor. Patients with total scores ranging from 0 to 3 were considered at low risk of recurrence, total scores of 4 or 5 points were viewed as an intermediate risk of recurrence, and total scores of 6 or 7 were considered high recurrence risk. The 31-year-old patient in the present study harbored a tumor in the gastrocolic omentum, with the largest diameter of 76 mm, a mitotic count of <3 mitoses/10 HPF, and no evidence of intratumoral necrosis, cystic degeneration or hemorrhage. The patient scored a total of 2 points and was considered at low risk for the development of SFT metastasis.

Radiologically, histopathological images are associated with CT imaging manifestation. On non-enhanced CT, an SFT appears as a mass with solid density in regions with cellularity and fibrous stroma regions, whereas lower attenuation areas represent necrotic or cystic regions, and tumoral calcification is rare [2,3,24,26,27]. SFTs typically exhibit vividly heterogeneous enhancement in the hypervascular region during the early arterial phase of dynamic contrast-enhanced CT due to the presence of dilated peripheral, staghorn-shaped vessels. The hypervascular region often displays progressive enhancement in the latter phase, which corresponds with hypercellular areas on histopathology. However, areas featuring necrotic or cystic degeneration do not show enhancement following the administration of contrast agents on CT. Collagenous or fibrotic components gradually accumulate contrast, demonstrating enhancement during the delayed phase [2,3,24,26,27]. The imaging features observed on plain and dynamic CT in the present case were similar to those described in the literature. The primary greater omental STFs often receive a large blood supply (most of the cases are from the right gastro-omental artery) with serpiginous dilation observed around the periphery of the mass [4–17]. The most common locations for secondary metastatic lesions derived from SFT include the lungs and pleura, followed by the liver, bone, peritoneum, abdominal wall and brain [22]. Lung metastasis may appear as singular or numerous solid nodules, unilaterally or bilaterally, without ground-glass opacities. Pleural effusion is often detected during cases of secondary pleural lesions. Liver SFT metastases are hypervascular and generally well defined. Lytic metastases of bone are frequently observed and might be due to the destruction of the bony trabecula; sclerotic metastasis patterns are rarely reported. Ascites may be present in patients with peritoneal, retroperitoneal or omental metastases [22].

The differential diagnosis of SFTs varies and depends vastly on location. A pre-operative imaging diagnosis is not possible due to the relative rarity of SFTs, and other more common neoplasms should be considered first, such as desmoid tumors, malignant fibrous histiocytoma, sarcomas or neurogenic tumors, when SFTs present in the abdomen and pelvis [21]. Meningioma should be considered for SFTs in the brain, carcinoma should be considered in the lung, and malignant pleural mesothelioma or mediastinal germ cell tumors should also be considered [29]. The pre-operative differential diagnosis in the present case included primary tumors of the mesentery and omentum, such as desmoid tumors and malignant mesothelioma, secondary metastatic lesions such as carcinoid tumors and mesenteric fibromatosis, and inflammatory pseudotumors [30,31]. Desmoid tumors often occur in patients who have undergone prior surgery or trauma, exhibit a well-delineated mass, and show weak and heterogeneous enhancement in the portal venous phase [31]. Metastatic lymphadenopathy from neuroendocrine tumors often derives from small intestine carcinoid tumors with associated carcinoid syndrome. Mesenteric carcinoid tumors appear as a poorly delineated, late enhancement, causing mesenteric retraction and occasional bowel obstruction as a result of the desmoplastic reaction [31]. Tumoral calcification is quite common. An inflammatory pseudotumor is generally related to systemic inflammatory disease, minor trauma or following surgery in adults, appearing as a well-delineated tumor, often showing central necrosis and calcifications, with variable enhancement [31].

Complete surgical resection is a potential treatment choice for most SFTs, and typically results in a better postoperative prognosis than a subtotal resection. Surgical complications, such as hemorrhage, may occur due to the hypervascularity of SFTs. Thus, preoperative or prebiopsy embolization of the supply vessels should be considered to reduce intra-operative bleeding [23]. A number of promising treatment methods have recently been introduced for SFTs. For example, chemotherapy, radiation therapy and early growth response 1-targeted anti-angiogenic therapy, which disrupts a crucial signaling pathway necessary for NAB2-STAT6 transcription [25]. The patient in the present study underwent total excision of the SFT in the larger omentum. After more than 9 months of postoperative observation and follow-up, the patient remains free of recurrence at the time of writing this study.

Conclusion

In conclusion, primary SFTs, also known as hemangiopericytoma, in the larger omentum are extremely rare. Clini-

cal symptoms and imaging manifestations are typically nonspecific. Therefore, the differential diagnosis between SFTs and hyperplasias, dysplasias or neoplasias can be exceptionally challenging. Diagnostic confirmation relies on histological and immunohistochemical analysis, including the detection of positivity against STAT6, CD34, CD99, BCL2 and vimentin serving as a diagnostic indicator. Treatment includes surgical resection, embolization therapy, radiation therapy, chemotherapy and anti-angiogenic agents.

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Patient Consent

Written and Informed consent for patient information to be published in this article was obtained.

Availability of data and materials

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Authors' contributions

HXT and NDH contributed equally to this article as co-first authors. Study concept and design: HXT and NDH; acquisition of data: NDH and NMD; analysis and interpretation of data: NDH and NMD; drafting of the manuscript: NDH and NMD; critical revision of the manuscript: NDH, NHK, NQD, NDH, and NMD; study supervision: NHK, NQD, and NDH. NDH and NMD confirm the authenticity of all the raw data. All authors read and approved final version of this manuscript.

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