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Clinicopathological analysis of sclerosing haemangiomatoid nodular transformation of the spleen: analysis of three cases and a literature review

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Abstract

Objective To examine the clinicopathological features, immunohistochemical profiles, and differential diagnosis of sclerosing angiomatiod nodular transformation (SANT).

Methods Three cases of SANT of the spleen, diagnosed between 2014 and 2023 at the Affiliated Hospital of Zunyi Medical University, were analysed. Pathological features were assessed using haematoxylin and eosin staining, followed by immunohistochemistry with the EnVision system. Additionally, a review of relevant literature was conducted.

Results The study included one male and two female patients aged 40–55 years, with a median age of 47.5 years. All lesions were solitary, with tumour diameters ranging from 4 to 7.4 cm (mean 5.7 cm). Gross examination demonstrated that the masses were well-demarcated from the surrounding splenic tissue, with no evident capsule. The cut surfaces of the masses exhibited irregular, porcelain-white nodules that were tough in consistency, with some areas intermingling with splenic tissue. Microscopic examination revealed round or circular nodules comprising multiple slit-like or sinusoidal capillaries, separated by concentric collagen fibres. The nodules exhibited chronic inflammatory cell infiltration, calcification, haemosiderin deposition, and fibrous connective tissue with hyaline or mucoid changes. Immunohistochemical analysis demonstrated differential expression of markers, including cluster of differentiation (CD) 34, CD31, and CD8, within the sinusoidal nodule areas. Periodic acid-Schiff staining was positive for perinodular collagen deposits, while reticulin staining highlighted nodule profiles and intranodular vessels. None of the patients experienced postoperative recurrence or metastasis, and one patient was on aspirin for thrombocytosis.

Conclusion SANT of the spleen is generally considered a rare, benign lesion with angioma-like characteristics. It exhibits exhibiting distinctive histomorphological features within the red pulp. Understanding the differential diagnosis is crucial to prevent missed or incorrect diagnoses.

Keywords Splenic tumour, SANT, Clinical pathology, Differential diagnosis, Literature review

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Background

Sclerotic nodular transformation, also known as sclerosing angiomatiod nodular transformation (SANT), is a rare benign spleen lesion with angioma-like characteristics and distinctive histomorphological features [1]. It was first described in 2004 by Martel et al. [2], who detailed its unique pathological and immunohistochemical features, establishing it as an independent disease entity. Due to its rarity, the pathogenesis of SANT remains unclear, and it often presents without clinical symptoms. Its distinctive histomorphology and non-specific imaging characteristics contribute to frequent missed or incorrect diagnoses during clinicopathological evaluations. This study reports and analyses three cases of splenic SANT, focusing on their clinical pathology, immunohistochemistry, and imaging characteristics, alongside a review of relevant literature to enhance awareness of this disease.

Materials and methods

Clinical information and diagnosis

From 2014 to 2023, three cases of SANT were confirmed in the Department of Pathology. The immunohistochemical analysis employed antibodies against cluster of differentiation (CD) 34, CD31, CD21, CD23, CD68, factor VIII (F8), anaplastic lymphoma kinase (ALK), leukocyte common antigen (LCA), smooth muscle actin (SMA), vimentin, podoplanin, CD8+, S100, human melanoma black (HMB)-45, and Ki67, all procured from Fuzhou Maixin Co., Ltd. Additionally, periodic acid-Schiff (PAS) and reticulin fibre staining were performed for Case 2. All pathological sections were reviewed and confirmed by two independent pathologists.

Methods

All specimens underwent fixation in a 4% neutral formaldehyde solution, followed by routine dehydration, paraffin embedding, and sectioning at 3–4 μm thickness. Haematoxylin and eosin staining was applied for histopathological examination. Immunohistochemical staining used the EnVision two-step method, with antibodies applied strictly following the manufacturer's instructions. A phosphate-buffered saline buffer substituted the primary antibody for the blank control, while known positive tissues served as positive controls.

Results

Clinical features

The clinical data of the three SANT cases are presented in Table 1. The study included two females and one male, aged 40–55 years, with a median age of 47.5 years. Cases 2 and 3 were incidentally found to have splenic lesions during physical examinations without apparent clinical symptoms. Case 2 had concurrent renal cysts, while Case 3 presented with multiple liver cysts. One patient experienced spleen-related abdominal pain due to chronic calculous cholecystitis. Imaging examinations of all three patients revealed splenic space-occupying lesions, initially suspected to be lymphangioma or lymphoma (Fig. 1). Case 3 involved laparoscopic splenectomy. In Case 2, due to dense adhesions between the lower pole of the spleen and adjacent structures, open total splenectomy with partial resection of the descending colon was performed to ensure complete tumour removal and rule out malignancy. During postoperative follow-up, Case 1 was lost to follow-up, while Cases 2 and 3 were followed up for 11 and 9 months, respectively. Case 2 was clinically managed for primary thrombocytosis and treated with aspirin, with no recurrence or metastasis observed in either case.

Pathological diagnosis

The isolated mass within the splenic parenchyma, measuring between 4 and 7.4 cm in diameter, was distinctly demarcated from the surrounding splenic tissue, lacking

Table 1 Clinical data of the three cases of splenic sclerosing angiomatiod nodular transformation

Number	Sex	Age (years)	Clinical symptoms	Tumour diameter (cm)	Diagnostic imaging	Surgical method	Concomitant diseases	Follow-up
1	F	55	Repeated abdominal pain for 2 months, worsened over 3 days	6.2	MRI revealed a splenic lesion, suggestive of haemangioma or lymphoma	Laparoscopic splenectomy	Chronic calculous cholecystitis	Lost to follow-up
2	M	40	Incidental finding of splenic space-occupying lesion during a physical examination lasting > 10 days	7.4	MRI indicated an angiogenic tumour or haemangioma with calcification, fibrosis, and thrombosis	Open abdominal splenectomy + partial resection of the descending colon	Small left renal cyst	Follow-up for 11 months, primary thrombocytosis treated with aspirin
3	F	51	Incidental finding of splenic lesion during a physical examination over 6 months	4	CT revealed an anterior spleen space-occupying lesion, likely a haemangioma.	Laparoscopic total splenectomy	Multiple small liver cysts	Follow-up for 9 months without recurrence

F, female; M, male; MRI, magnetic resonance imaging; CT, computed tomography

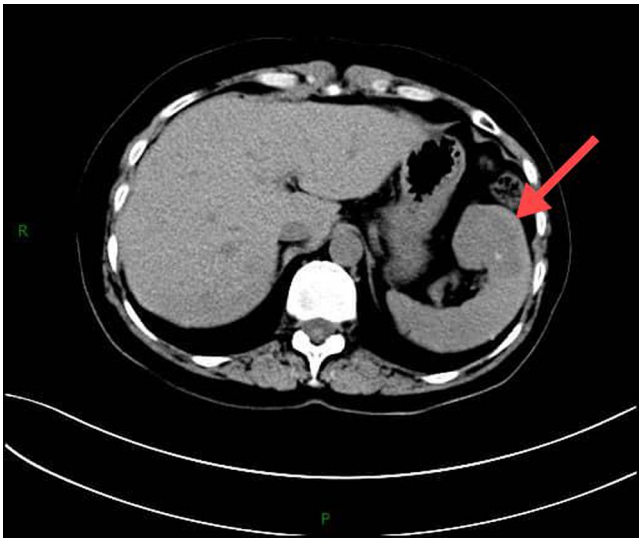

Fig. 1 Computed tomography showing isolated, well-demarcated, low-density, oval foci with occasional calcification (red arrow)

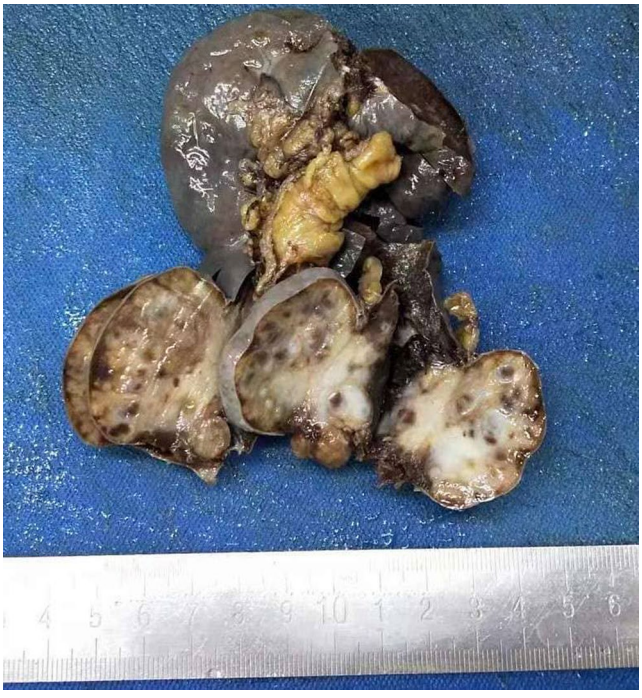

Fig. 2 The boundary between the mass and the surrounding splenic tissue appears clear, without an obvious capsule. The cut surface of the mass shows an irregular, porcelain-white, tough, nodular area in the centre, interspersed with splenic tissue and surrounded by greyish tissue

an obvious capsule. The cut surface of the mass exhibited irregular, porcelain-white nodules that were tough in texture and partially interspersed with the splenic tissue (Fig. 2).

Under microscopic examination, all three cases exhibited masses of varying sizes, characterised by round or oval angioma-like or granuloma-like nodules with partial fusion. Collagen fibres arranged concentrically around the nodules separated them (Fig. 3A). In Case 2, internodular connective tissue showed hyaline or myxoid degeneration, while Case 3 revealed calcification and haemosiderin deposition. Slit-like or sinusoidal capillaries with plump endothelial cells were observed in the nodes using high-power microscopy. These cells appeared spindle-shaped or oval with normal morphology, rare mitotic figures, and abundant light-staining cytoplasm with vacuolation, resembling foam cells or granulomatous lesions (Fig. 3B). No multinucleated giant cells were observed. A small number of red blood cells were present in the central vascular lumen of the nodule,

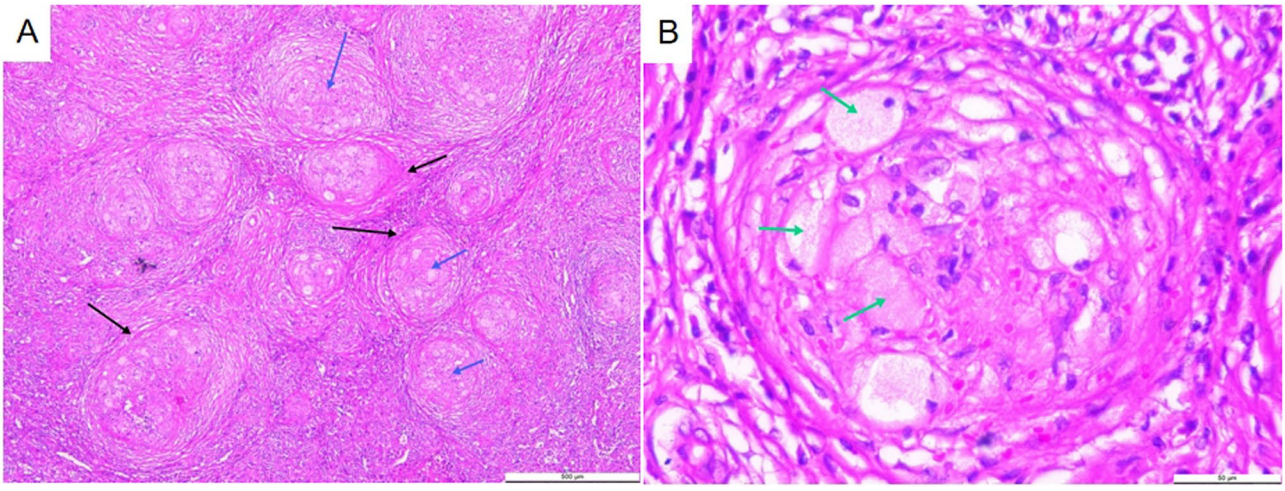

Fig. 3 **A** Low-magnification (50×) view showing the mass, which comprises round or oval angioma-like or granuloma-like nodules (blue arrow) with partial fusion. Numerous concentric collagen fibres (black arrow) surround and separate the nodules. **B** High magnification (400×) view of the nodules comprising slit or sinusoidal capillaries. The endothelial cells in the vascular spaces are plump, spindle-shaped, oval, and atypical, with rare nuclear division. They exhibit rich cytoplasm, light staining, and vacuolation, resembling foam cells (green arrow) and granulomatous lesions

with some extravasation. The margins of the nodules contained scattered neutrophils, lymphocytes, plasma cells, and histiocytes.

Immunohistochemical analysis of the three cases revealed specific staining patterns. In the sinusoidal spaces, both round or irregular vascular endothelial cells and some vascular spindle cells exhibited diffuse and strong positivity for CD34 and CD31 (Fig. 4A and B), as well as positivity for F8. Ovoid cells, spindle-shaped foam cells, and tissue cells outside the nodules expressed CD68 (Fig. 4C) and CD163. Fibrocytes surrounding the nodules and vascular smooth muscle cells within the nodules were positive for SMA (Fig. 4D). Vimentin expression was observed in foam cells and endothelial cells within the nodules, as well as in endothelial cells, fibroblasts, and other mesenchymal cells outside the nodules. Inflammatory cells inside and outside the nodules expressed LCA. Additionally, some regions showed CD8 expression. The Ki67 proliferation index ranged from 2 to 5%. However, no expression of CD21, CD23, ALK, S100, or HMB-45 was observed.

Special staining techniques revealed distinct characteristics in the tissue samples. PAS staining demonstrated positive collagen deposits surrounding the nodules. Reticular fibre staining highlighted the positive nodule contours and also marked the blood vessels with them.

Discussion

In 1993, Krishnan proposed that splenic SANT is a peculiar form of haemangioma or hamartoma [3]. In 2004, Martel et al. [2] documented 25 cases and were the first time to describe the unique pathohistological features of SANT, including a solid mass with clear delineation, a multinodular haemangioma-like structure, and differential expression of vascular immunolabelling in haemangioma-like areas, subsequently naming it SANT. While some scholars consider splenic SANT a neoplastic lesion, others view it as a benign angioma-like non-neoplastic lesion of the splenic red pulp. Therefore, the aetiology and pathogenesis of splenic SANT remain subjects of ongoing debate.

Clinical features of SANT

SANT predominantly affects middle-aged and elderly women, with ages ranging from 9 weeks to 85 years and a median age of 46 years [4]. Most patients are asymptomatic, and the condition is often discovered incidentally during physical examinations or evaluations for other abdominal discomforts. Some patients might present with anaemia, thrombocytopaenia, or other reactive changes. In this study, Case 2 was diagnosed with primary thrombocytosis postoperatively, indicating that splenic SANT can affect spleen red pulp function [5]. Patients with SANT might also have benign or malignant lesions in other organs, such as liver and kidney cysts, chronic cholecystitis, gastrointestinal malignancies, lung cancer, kidney cancer, breast cancer, uterine clear cell cancer, and sarcoma [4, 6–9]. In the reported cases, all three patients had accompanying diseases. Cases 2 and 3 were asymptomatic and discovered incidentally during physical examinations, with kidney and liver cysts, respectively. Case 1 was diagnosed with a splenic lesion during treatment for chronic calculous cholecystitis. Hanwen et al. [10] identified an association between SANT with malignant tumours, coining the term "tumour-splenic SANT syndrome", with its pathogenesis and prognosis related to the nature and American Joint Committee on Cancer stage of the tumour. This association warrants

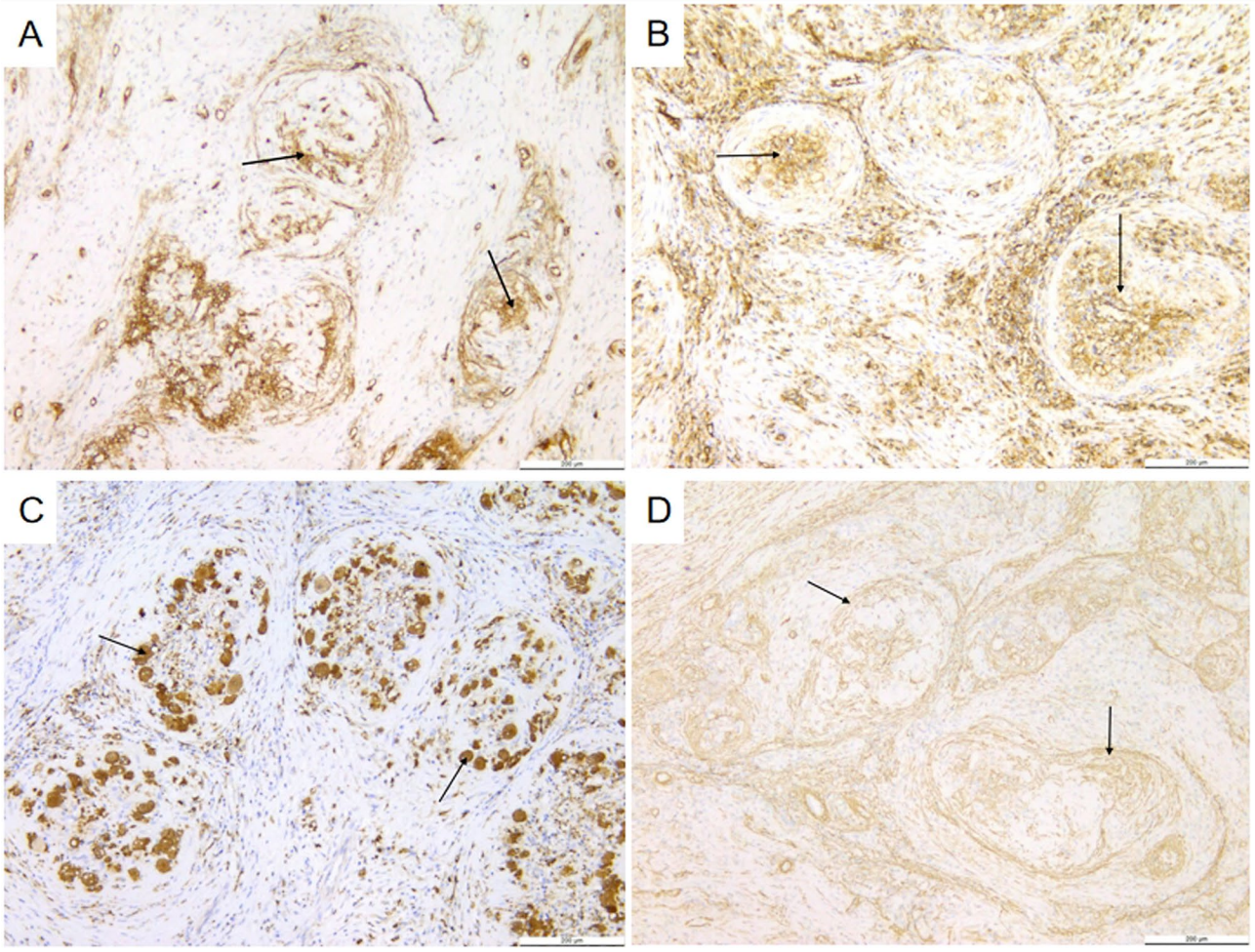

Fig. 4 **A** Sinusoidal, round, or irregular vascular lumen endothelial cells, along with some vascular spindle cells, show diffuse and strong positivity for cluster of differentiation 34 (black arrow). **B** Sinusoidal, round, or irregular vascular lumen endothelial cells, as well as some vascular spindle cells, exhibit diffuse and strong positivity for cluster of differentiation 31 (black arrow). **C** Oval cells, adipose spindle-shaped cells (foam cells), and stromal cells outside the nodules exhibit cluster of differentiation 68 expression (black arrow). **D** Fibrocytes surrounding nodules and vascular smooth muscle cells show smooth muscle actin expression (black arrow)

further attention from clinicians and pathologists. Additionally, some reports describe SANT co-occurring with calcified fibrotic tumours [11, 12], suggesting a possible link to non-specific inflammation and sclerosis in SANT [13]. However, due to the limited number of such reports, further case accumulation and analysis are necessary to provide stronger theoretical support. SANT is an exceptionally rare condition, primarily found in the spleen, with only one reported case occurring in the adrenal gland [14].

Pathological features of SANT

SANT exhibits benign biological behaviour. Typically, spleen masses range from 68 to 2720 g, with lesion diameters spanning from 1.0 to 17.0 cm [15]. Single lesions are more common, although multiple foci have been reported [12, 16]. Lesions are characterised by a clear boundary, often lacking an obvious capsule, although some literature mentions the formation of an enveloping structure around the lesion [17]. On sectioning, the mass appears solid with a grey-brown colouration, displaying an uneven surface and irregular central stellate scar. Some cases might exhibit haemorrhage and alternation with surrounding normal spleen tissue while maintaining a clear boundary. Microscopically, lesions consist of round or oval nodules comprising multiple slit or sinusoidal capillaries. Collagen fibres surround these nodules in concentric patterns, separating them. Chronic inflammatory cell infiltration, fibrous connective tissue changes resembling glass or mucoid material, calcification, and haemosiderin deposition are commonly observed. Immunohistochemical analysis reveals heterogeneous expression in haemangioma-like areas, with reported vessel types including capillary (CD34+, CD31+, and CD8-), sinusoid space (CD31 +, CD8 +, and CD34-), and venular vessels (CD31+, CD34-, and CD8-). Special stains

such as PAS staining highlight positive collagen deposits surrounding the nodules, while reticulin fibre staining delineates the nodular outline and blood vessels.

Imaging examinations of SANT

On computed tomography (CT) scans, splenic SANT typically presents as isolated, well-defined oval lesions with slightly lower density, occasionally showing calcification. Magnetic resonance imaging (MRI) findings include equal or slightly low T1-weighted signal, slightly uneven low T2-weighted signal, low signal on diffusion-weighted imaging, and non-specific apparent diffusion coefficient signal. Enhanced scans reveal gradual, concentric, nodule-like enhancement with central delayed reinforcement, sometimes exhibiting a "star sign" or "spoke wheel sign" [18]. The characteristic "spoke wheel" pattern observed in some CT/MRI scans of SANT is not specific and is believed to correlate with the degree of fibrous connective tissue segmentation within the lesions [19]. Positron emission tomography/CT scans often show high metabolic activity, possibly due to the presence of granuloma-like nodular tissue rich in haemosiderin-laden macrophages, fibrocytes, and chronic inflammatory cells associated with SANT [19]. Given the low incidence and non-specific imaging features of this condition, achieving a definitive preoperative diagnosis remains challenging. Accurate diagnosis continues to rely on pathological histology and immunohistochemistry. In the three reported cases of SANT, early imaging often led to misdiagnoses such as haemangioma, lymphangioma, or lymphoma. Given its rarity, more experience with imaging is needed to distinguish SANT from other diseases.

Aetiology and pathogenesis

Martel et al. [2] proposed that SANT develops through a maturation process from the red pulp structure of the spleen to slit-like vessels, eventually forming granuloma-like nodules surrounded by fibrous connective tissue, culminating in non-specific inflammation and sclerosis. Jun et al. [20] suggested a pathological progression for SANT, involving stages from haemangioma-congestion, haemorrhage, and necrosis to granulation tissue formation, nodular inflammatory pseudotumour-like areas, and finally collagenisation and fibrotic hyperplasia in nodular angioma-like areas, closely tied to haemangioma development. Han et al. [17] investigated the role of CD30 in SANT pathology, proposing its involvement in pulmonary blood vessel remodelling [21], and highlighted differential immunoglobulin (Ig) G4 distribution between lesions and normal spleen, potentially linking IgG4 to SANT development. The association between SANT and Epstein–Barr virus infection and IgG4-related diseases remains debated, necessitating further research for confirmation. Chang et al. [22] in 2006 supported the theory that SANT is a polyclonal reactive lesion rather than a true tumour, suggesting it arises from vascular damage or dysfunction with neoplastic vascular proliferation in the red pulp of the spleen. In 2021, Uzun et al. [23] conducted molecular studies indicating that catenin beta-1 exon 3 deletions, leading to oncogenic β-catenin formation and Wnt pathway hyperactivation, might play a fundamental role in SANT pathogenesis. This evidence supports SANT as a genuine vascular tumour of the spleen, reinforcing its neoplastic nature. The pathogenesis of splenic SANT remains controversial, predominantly viewed as a reactive benign lesion associated with haemangioma, necessitating further investigation for clarification.

Differential diagnosis of SANT

Due to its rarity, limited clinical symptoms, distinctive histomorphology, and poor imaging specificity, splenic SANT can often be mistaken for other splenic diseases. The main differential diagnoses include the following: ① Inflammatory pseudotumour/inflammatory myofibroblastic tumour: These tumours consist primarily of inflammatory and spindle-shaped fibroblastic/myofibroblastic-like cells. They exhibit collagen and mucus changes in the stroma against a background of chronic inflammation, resembling SANT. However, they lack the characteristic nodular angioma-like structure of SANT and typically express ALK by immunohistochemistry, while they do not express CD8, CD31, or CD34 markers. ② Splenic hamartoma: This comprises vessels of varying thickness and irregular arrangement, lined with endothelial cells. Splenic hamartomas express markers such as F8, CD8, CD8, and CD31 similar to SANT, but do not express CD34, CD21, or CD68. Unlike SANT, they do not exhibit nodular angioma-like structures or collagen fibre hyperplasia. ③ Sinusoidal haemangioma: This is characterised by anastomotic irregular vascular spaces lined with a single layer of endothelial cells that can form papillary structures. They express markers such as F8, CD31, CD68, and CD8 but do not express CD34, and they lack surrounding collagen fibres. ④ Granulomatous lesions: Splenic SANT with granuloma-like nodules should be differentiated from various granulomatous lesions, which often include tissue cells and multinucleated giant cells. Granulomas typically exhibit necrosis and more prominent chronic inflammatory cells, with vascular proliferation being less pronounced and lacking collagen fibres around the nodules. ⑤ Niemann–Pick disease: This is typically seen in children, characterised by hepatomegaly and deposition of lipid-laden cells (Niemann–Pick cells) in the spleen. These cells, resembling foam cells found in SANT granuloma nodules, stain positively with lipid stains (Sudan B and oil red O) due to their neutral fat content. Unlike SANT, they do not show evident collagen fibres around the nodules.

Treatment and prognosis of SANT

Splenic SANT is regarded as a biologically benign angioma-like lesion of the spleen, with its pathogenesis still requiring further investigation. Currently, splenectomy remains the preferred and most effective treatment option. Given the low incidence of the disease, the absence of obvious clinical symptoms, and the lack of specificity in preoperative imaging, the decision to perform total splenectomy might be challenging. Since the spleen is a critical lymphoid organ, its removal can significantly impact the patient's systemic immune function. Therefore, regular postoperative monitoring is essential. Literature [4, 24] suggests that splenic core needle biopsy is minimally invasive and can reduce the risk of postoperative bleeding. With experienced operators and appropriate safety measures, it might replace total splenectomy as a feasible and safe diagnostic method. In this study, three patients were lost to follow-up after splenectomy. Two patients were followed up for 9 and 11 months, respectively. One patient was clinically diagnosed with primary thrombocytosis and was treated with aspirin, while the others showed no sign of disease recurrence or metastasis. Although the follow-up period in this study is relatively short, when combined with other reports in the literature, the prognosis for SANT appears to be favourable. Nonetheless, continued monitoring is necessary to ensure long-term positive outcomes.

Conclusion

SANT of the spleen is generally considered a rare, benign lesion with angioma-like characteristics, exhibiting distinctive histomorphological features within the red pulp of the spleen. Understanding the differential diagnosis is crucial to avoid missed or incorrect diagnoses.

Abbreviations

SANT	Sclerosing angiomatiod nodular transformation
CD	Cluster of differentiation
F8	Factor VIII
ALK	Anaplastic lymphoma kinase
LCA	Leukocyte common antigen
SMA	Smooth muscle actin
HMB	Human melanoma black
PAS	Periodic acid-Schiff
CT	Computed tomography
MRI	Magnetic resonance imaging
Ig	Immunoglobulin

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Author contributions

Writing–original draft: JF Z and J L, Writing & editing: JF Z, JJ W. J L and S L prepared all figures. All the authors have read & approved the final manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

This case report was approved by the Ethics Committee of the Affiliated Hospital of Zunyi Medical University. Written informed consent was obtained from the patients and their families for the publication of this clinical case report.

Consent for publication

Written informed consent was obtained from the patients and their families for the publication of this case report and any accompanying images.

Competing interests

The authors declare no competing interests.

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References

1. Tseng H, Ho CM, Tien YW. Reappraisal of surgical decision-making in patients with splenic sclerosing angiomatoid nodular transformation: Case series and literature review. World J Gastrointest Surg. 2021;13(8):848–58. https://doi.org/10.4240/wjgs.v13.i8.848.
2. Maritza Martel M, Wah Cheuk M, Luciano Lombardi M, et al. Sclerosing Angiomatoid Nodular Transformation (SANT) Report of25 cases ofa distinctive Benign Splenic lesion [J]. Am J Surg Pathol. 2004;28(10):1268–79.
3. Krishnan J, Frizzera G. Two splenic lesions in need of clarification: hamartoma and inflammatory pseudotumor [. J] Semin Diagn Pathol. 2003;20(2):94–104.
4. Aziret M, Yilmaz F, Kalpakci Y, et al. Sclerosing angiomatoid nodular transformation presenting with thrombocytopenia after laparoscopic splenectomy - case report and systematic review of 230 patients [J]. Ann Med Surg (Lond). 2020;60:201–10.
5. Pelizzo G, Villanacci V, Lorenzi L, et al. Sclerosing angiomatoid nodular transformation presenting with abdominal hemorrhage: first report in infancy [. J] Pediatr Rep. 2019;11(2):7848.
6. Efared B, Sidibe IS, Erregad F, et al. Sclerosing angiomatoid nodular transformation of the spleen (SANT) in a patient with clear cell carcinoma of the uterus: a case report [J]. J Med Case Rep. 2018;12(1):377.
7. Sitaraman LM, Linn JG, Matkowskyj KA, et al. Sclerosing angiomatoid nodular transformation of the spleen masquerading as a sarcoma metastasis [. J] Rare Tumors. 2010;2(4):e45.
8. Li SX, Fan YH, Wu H, et al. Sclerosing angiomatoid nodular transformation of the spleen: a case report and literature review [. J] World J Clin Cases. 2021;9(1):211–7.
9. Wang H, Hu B, Chen W, et al. Clinicopathological features of sclerosing angiomatoid nodular transformation of the spleen [J]. Pathol Res Pract. 2021;224:153490.
10. Jiang Hanwen W, Huan F, Lina, et al. Analysis of the pathology and pathogenic mechanisms of tumor-splenosclerotic angioma-like nodular transformation syndrome [J]. Lingnan Mod Clin Surg. 2022;22(6):531–6.
11. Xu Yaru H, Lili W. Children with scleroclerotic angioma-like nodular transformation with calcific fibrotic tumor of the pancreas [J]. J Clin Experimental Pathol. 2021;37(10):1269–70.
12. Kuo TT, Chen TC, Lee LY. Sclerosing angiomatoid nodular transformation of the spleen (SANT): clinicopathological study of 10 cases with or without abdominal disseminated calcifying fibrous tumors, and the presence of a significant number of IgG4 + plasma cells [. J] Pathol Int. 2009;59(12):844–50.
13. Turbiville D, Zhang X. Calcifying fibrous tumor of the gastrointestinal tract: a clinicopathologic review and update [. J] World J Gastroenterol. 2020;26(37):5597–605.

14. Zavatta G, De Leo A, Bacci F, et al. Sclerosing Angiomatoid Nodular Transformation of the adrenal gland: a Case Report of a Novel Histopathological Entity. [J] J Endocr Soc. 2019;3(6):1207–13.
15. Danli Y, Yingwen L, Wen LX et al. Clinicopathological analysis of splenosclerotic angioma-like nodular transformation in 6 cases [J]. J Clin Experimental Pathol, 2020, (011): 036.
16. Cao Z, Wang Q, Li J, et al. Multifocal sclerosing angiomatoid nodular transformation of the spleen: a case report and review of literature [. J] Diagn Pathol. 2015;10:95.
17. Wang Han Z, Yingying D, Wei et al. Clinicopathological analysis of 10 cases of nodular transformation [J]. J Clin Experimental Pathol, 2023,39 (6): 724-6,9.
18. Liao J, Wang Z, Li Q, et al. CT and MRI features of sclerosing angiomatoid nodular transformation of the spleen: a report of 18 patients with pathologic correlation [J]. Diagn Interv Imaging. 2021;102(6):389–96.
19. Thacker C, Korn R, Millstine J, et al. Sclerosing angiomatoid nodular transformation of the spleen: CT, MR, PET, and (9)(9)(m)Tc-sulfur colloid SPECT CT findings with gross and histopathological correlation [. J] Abdom Imaging. 2010;35(6):683–9.
20. Hou Jun, Era T, Yunshan, et al. Clinicopathological analysis of the sclerosing angioma-like nodular transformation of the spleen [J]. Chin J Pathol. 2010;39(2):84–7.
21. Luo L, Liu Y, Chen D et al. CD30 Is Highly Expressed in Chronic Obstructive Pulmonary Disease and Induces the Pulmonary Vascular Remodeling [J]. Biomed Res Int, 2018, 2018: 3261436.
22. Chang KC, Lee JC, Wang YC, et al. Polyclonality in Sclerosing Angiomatoid Nodular Transformation of the spleen [J]. Am J Surg Pathol. 2016;40(10):1343–51.
23. Uzun S, Ozcan O, Isik A, et al. Loss of CTNNB1 exon 3 in sclerosing angiomatoid nodular transformation of the spleen [. J] Virchows Arch. 2021;479(4):747–54.
24. Hanlon K, Wilson MR, Kay D et al. Safety and diagnostic yield of splenic core biopsy; a methodical approach using combined Haematology/Radiology assessment in a tertiary referral centre[J]. Br J Haematol, 2019, 186(2).

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