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From Hamartoma to Splenic Hemangioma

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Vascular benign lesions are the most common non-hematological splenic primary tumors. Although rare they may sometimes pose problems in differential diagnosis, because of their morphologic heterogeneity.

We present vascular lesions of the spleen, which were found in archive of the Chair of Pathomorphology. Immunohistochemistry including CD34, CD31, factor VIII, CD8, CD21, CD68, lysosyme, GLUT-1, D2-40, VEGFR3, SMA, Ki67 were performed. 8 benign vascular lesions were identified , including two hamartomas, two lesions representing sclerosing angiomatoid nodular transformation (SANT) and four hemangiomas.

We present briefly the spectrum of vascular lesions occurring in the spleen and discuss differential diagnosis and nosological status of selected lesions.

Introduction

Primary non-hematological tumors of the spleen constitute a group of non-uniform lesions, most frequently of a vascular-proliferative character. The majority of such tumors are relatively rare [1]. Vascular lesions secondary to systemic diseases are much more common than neoplastic lesions [2]. Benign lesions are usually detected incidentally, but at times, they may be manifested as hypersplenism [1]. In contrast to such lesions, angiosarcomas are manifested in an acute and dramatic manner and characterized by an aggressive clinical course. Splenic hemangiomas may differentiate into common endothelium, specialized sinus endothelium and lymphatic vessel lineage. In the currently acknowledged diagnostic categories, the histology of some lesions is overlapping. The lesions are classified as benign, with intermediate degree of malignancy, and malignant [2]. Splenic vascular lesions include entities presented in Table 1.

At present, hamartoma is defined as a lesion composed of normal elements of splenic red pulp, which are characterized by an abnormal quantitative ratio and abnormal distribution. Macroscopically, hamartomas present as round, well-lineated tumors, most often single and rarely multifocal, with a dark red or gray-white cross section and the diameter of 1-15 cm.

TABLE 1
Splenic vascular lesions

Developmental malformations and non-neoplastic lesions	Vascular tumors
Hamartoma Sclerosing angiomatoid nodular transformation (SANT)	**Benign:** • Hemangioma – localized/diffuse • Lymphangioma • Littoral-cell angioma **Malignant:** • Angiosarcoma • Littoral cell angiosarcoma **Intermediate malignancy:** • The so-called hemangiondothelioma • Hemangiopericytoma*

* in spite of the discussion of the hemangiopericytoma in the literature several cases of such lesions in the spleen were described (8, 9).

Histologically, in spite of the macroscopically sharp delineation, the structure of the lesion is merged with the surrounding splenic red pulp. The tumor is composed of irregular, twisted vascular canals that vary in size and are lined with endothelium resembling the splenic sinus lining. The vessels are surrounded by aggregates of lymphocytes and macrophages, but show no organized lymph follicles. In addition, some cases demonstrate focal fibrosis, aggregates of plasmocytes, macrophages, mastocytes and eosinofiles, as well as foci of extramedullary hematopoiesis [3].

SANT (sclerosing angiomatoid nodular transformation) is a vascular lesion with a characteristic morphological appearance and immunophenotype, which has a benign clinical course. Macroscopically, a single, well-encapsulated tumor is seen, 3-17 cm in diameter, with cross sections revealing the lesion to be composed of brownish-red nodules separated by fibrous strands. The histological picture is also characteristic – numerous vascular nodules are noted embedded in a fibrous stroma. In the majority of cases, the nodules are round, with a variable size, at times with irregular indentations and are either separated by broad bands of connective tissue or blended together. Some nodules, especially the small ones, are surrounded by concentrically oriented collagen fibers. The nodules are composed of slit-like, rounded or irregular epithelium-lined vessels; scattered spindle or oval cells are seen among them. Dispersed inflammatory cells (mainly lymphocytes and plasmocytes) are also observed. The stroma between the nodules is myxoid or dense and fibrous, with scattered myofibroblasts, plasmocytes, lymphocytes and siderophages. Atypia is minimal, mitotic figures are very rare and there is no necrosis. Immunohistochemically, three types of vessels are observed within the nodules: CD34+/CD8-/CD31+ capillary vessels, CD34-/CD8+/CD31+ sinusoid vessels and CD34-/CD8-/CD31+ small veins, the three vessel types reflect the splenic red pulp composition [4].

Hemangiomas are the most common benign primary splenic tumors, although they do occur rarely. Macroscopically, they are delineated, non-encapsulated, have the appearance of a honeycomb, are red in color, but often surreptitiously blend with the surrounding pulp. They are most often single, but in a good portion of cases, a thorough examination allows for detection of multifocal lesions. Histologically, the majority of hemangiomas are cavernous in character (large, connected, distended and engorged blood vessels lined with a single layer of endothelial cells with benign appearance, separated by thin fibrous septae or red pulp); mixed forms are encountered less commonly, and capillary forms are the least frequent [2, 3].

Lymphangiomas are less common than hemangiomas. Macroscopically, lymphangiomas are represented by small, subcapsular or peritrabecular multicystic or solid lesions, sometimes with a central scar. Microscopically, the lesion demonstrates cystic spaces with thin walls, predominantly lined with flattened endothelial cells with benign appearance, filled with pink protein fluid devoid of erythrocytes. At times, foamy macrophages or slit-like empty spaces left after leached out cholesterol crystals are noted. Focally, larger endothelial cells may be seen forming papilliform structures [2, 3].

Littoral cell angioma is a unique splenic lesion that does not have any equivalent in tumors of soft tissue or other organs. Macroscopically, it is seen as numerous spongiform, cystic, engorged and delineated nodules, 0.2-9 cm in size; less frequently, the lesions are single or completely replace the splenic pulp. Microscopically, a complex network of distended or narrowed vascular spaces of varying sizes is seen. In the majority of cases, the vessels are lined with a single endothelial cell layer. Such endothelial cells are predominantly tall, with large vesicular nuclei and small nucleoli, but without any atypia and mitotic figures. Some cells have small nuclei with irregular outlines, dense chromatin and scant cytoplasm. In the majority of cases, papillar structures with a fibrous core are noted. Oftentimes, desquamated endothelial cells are visible, the morphology of which resembles that of a macrophage; they frequently show hemophagocytosis, hemosiderin deposits and foamy cytoplasm. Occasionally, solid proliferations of endothelial cells are seen within the vessels. A characteristic observation is focal aggregates of acidophilic 0.5-2 μm spheres, which may fill entire cytoplasm (ultrastructurally, they are lysosomes and residual bodies) [2, 3].

Angiosarcomas are rare (more than 100 described cases). Macroscopically, their cross sections show multinodular hemorrhagic 1-18 cm lesions or a diffuse infiltration. The nodules may be well or poorly delineated. Necrotic areas are frequently present, along with cystic spaces filled with bloody contents. Microscopically, the picture is highly variable, even within the same tumor. The most frequent observation is a vascular architecture composed of an irregular network of narrow or capillary-like vessels; cavernous spaces with papilliform architecture are commonly seen. Although focally it may be poorly expressed, yet atypia of cells lining the vascular spaces is usually a distinct phenomenon. In addition, mitotic figures and multinucleated giant cells are often detected. At times, intranuclear or extranuclear hyaline spheres and hemophagocytosis are noted. Solid areas with spindle, polyhedral, epithelioid or small primitive cells are observed. Immunohistochemically, the majority of these cells are characterized by expression of two or more vascular markers [2].

Littoral cell angiosarcoma is less common than angiosarcoma. Its morphology is that of littoral cell angioma, but it is characterized by atypia and an infiltrating or solid

growth pattern. Although such lesions are regarded a malignant variant of littoral cell angioma, yet, since in angiosarcomas expression of markers of splenic sinus vessels may be also detected, a precise discrimination between the two types of lesions may be at times difficult. Positive reactions to factor VIII, CD31, CD68, CD21, and negative to CD34 and CD8 may indicate a biologically separate entity with a less aggressive course than angiosarcoma.

Splenic vascular tumors are so uncommon that they pose numerous diagnostic problems. Immunohistochemistry allows for confirming the vascular character of poorly differentiated neoplasms and for differentiation of benign lesions. Nevertheless, apart from conventional morphological features, there are no reliable indications that would determine the potential for malignancy. For this reason, in cases with atypical morphology, follow-up – to say the least – is indicated [2].

Material and Methods

The investigation included archival cases from the Chair of Pathomorphology from the years 1992-2007. In all instances, formalin-fixed, paraffin-embedded and hematoxilin and eosin stained materials were reassessed. Additionally, immunohistochemical reactions listed in Table 2 were done.

Results

The search of the archival material revealed eight vascular lesions, including two hamartomas, two lesions representing SANT and four hemangiomas. The clinical data, macroscopic findings, original diagnosis and the diagnosis determined following re-evaluation of specimens are presented in Table 3.

TABLE 2
Panel of antibodies

Antibody specificity	Clonality	Supplier	Titer	Antigen retrieval	Time	Detection system
CD31	M	DAKO	1:20	Microwave, EDTA buffer pH=8.0	60 min	En Vision
CD34	M	DAKO	1:25	Microwave, citrate buffer pH=6.0	30 min	En Vision
Factor VIII	M	DAKO	1:50	Microwave, citrate buffer pH=6.0	60 min	En Vision
CD68	M	DAKO	1:50	Microwave, EDTA buffer pH=8.0	30 min	En Vision
Lysosyme	P	DAKO	-	-	30 min	En Vision
CD8	M	DAKO	1:50	Microwave, EDTA buffer pH=8.0	60 min	En Vision
CD21	M	DAKO	1:50	Microwave, citrate buffer pH=6.0	30 min	En Vision
GLUT-1	P	DAKO	1:200	Microwave, citrate buffer pH=6.0	30 min	En Vision
D2-40	M	Abcam	-	Microwave, citrate buffer pH=6.0	30 min	En Vision
VEGFR3		Novocastra	1:50	Microwave, EDTA buffer pH=8.0	60 min	Lab Vision
CD20	M	DAKO	1:50	Microwave, citrate buffer pH=6.0	30 min	En Vision
CD3	P	Novocastra	1:100	Microwave, citrate buffer pH=6.0	60 min	Lab Vision
SMA	M	DAKO	1:50	Microwave, citrate buffer pH=6.0	30 min	En Vision
Ki-67	M	DAKO	1:50	Microwave, citrate buffer pH=6.0	Over night	En Vision
CD61	M	Novocastra	1:50	Microwave, citrate buffer pH=6.0	60 min	En Vision
MPO	P	DAKO	1:200	-	30 min	En Vision

M – monoclonal antibody, P – polyclonal antibody

TABLE 3
Clinical data, macroscopic findings and histopathological diagnosis

No.	Sex	Age	Macroscopic findings	Clinical diagnosis	Original diagnosis	Diagnosis after re-evaluation
1	M	73	Single tumor, 4 cm in diameter	Tumor lienis	Hamartoma	Hamartoma
2	M	30	Splenic fragment with a tumor		Haemangioma lienis	Hamartoma
3	M	29	Grey-white, partially visible hemorrhagic spherical tumor, 8x6.5x8 cm in size	Tumor lienis	Hamartoma	SANT
4	K	34	Lobular, sharply delineated tumor, 12.0x10x5cm in size	Tumor lienis Lymphoma suspitio. Anaemia sideropenica secundaria	Haemangioma capillare lienis ("Splenic cord hemangioma")	SANT
5	K	76	Numerous scattered hemorrhagic foci measuring from several mm to 2 cm		Lymphohaemangioma cavernosum multiplices lienis	Haemangioma cavernosum diffusum
6	M	60	Fragmented spleen with numerous small cysts, 520 g in weight	Multiple splenic cysts	Angiomatosis lienis	Haemangioma cavernosum diffusum
7	K	63	Fragmented spleen, 127 g in weight		Haemangioma cavernosum	Haemangioma cavernosum
8	K	67	Fragmented material – spleen with the total weight of 276 g	Tumor lienis.	Haemangioma cavernosum	Hemangioma cavernosum

In both hamartoma cases, single, well-delineated tumors were seen macroscopically. Histologically, however, the borderline between the lesion and the surrounding splenic pulp was blurred. The tumors were composed of irregular, twisted vascular canals of varying size lined with epithelium resembling the lining of the splenic sinuses. The vessels were surrounded by macrophages and lymphocytes, both single and forming small aggregates, but without forming proliferation centers or a marginal zone. In Case 1, the predominant feature was vascular structures with a small number of stromal elements (Fig. 1), while Case 2 focally demonstrated the architecture with predominance of stromal elements over the vascular component (Fig. 2). In addition, in Case 1, small, scattered foci of extramedullary hematopoiesis were noted. Immunohistochemically, the vessels demonstrated expression of vascular antigens CD31(Fig. 3), CD34 and factor VII, as well as CD8 (Fig. 4), but in Case 2, CD8-positive vessels were less numerous than in Case 1.

The macroscopic and histological findings in both cases of SANT were strikingly similar. Macroscopically, both lesions formed sharply delineated, single tumors. Histologically, the lesions were composed of numerous vascular nodules embedded in the fibrous stroma (Fig. 5). In their majority, the nodules were round in shape, at times with irregular indentations, and varied in size. The nodules were widely separated by fibrous tissue or blended. Some nodules, especially the small ones, were surrounded by concentrically arranged collagen fibers. The nodules themselves were composed of slit-like, round or irregular vessels lined with endothelium, with scattered spindle or oval cells visible among the vessels. In addition, among the nodules, in the fibrous stroma, scattered inflammatory cells (mainly lymphocytes and plasmocytes) and hemosiderin-laden macrophages were seen. No cellular atypia was noted or mitotic figures or necrosis. Immunohistochemically, the majority of vessels demonstrated CD31 antigen expression (Fig. 6), while reaction to the presence of CD34 antigen

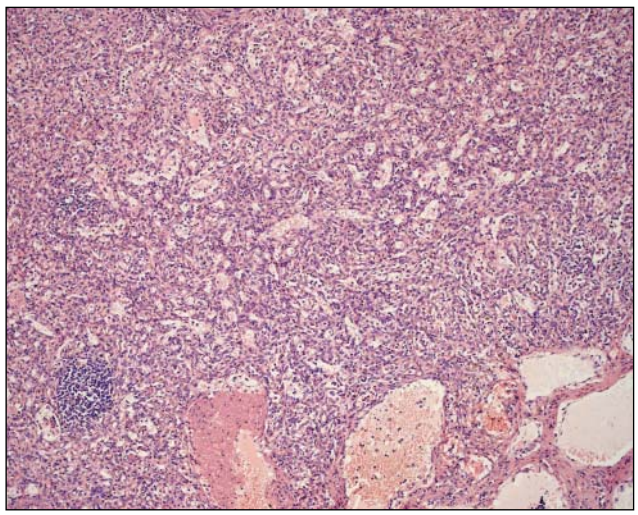

Fig.1. Hamartoma, Case 1 (hematoxylin and eosin, original magnification x 50).

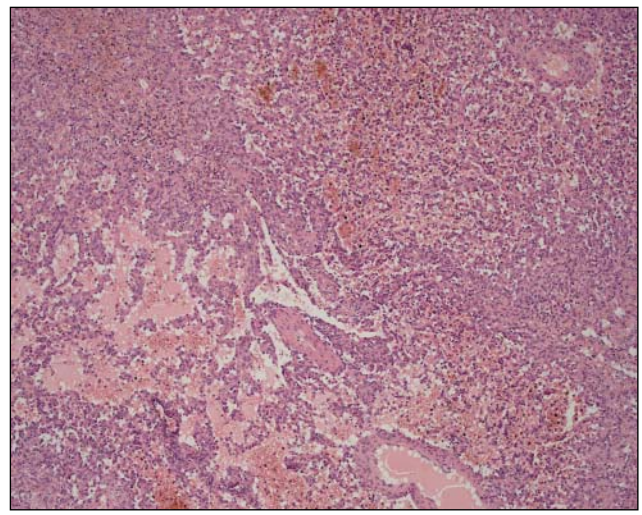

Fig. 2. Hamartoma, Case 2 (hematoxylin and eosin, original magnification x 100).

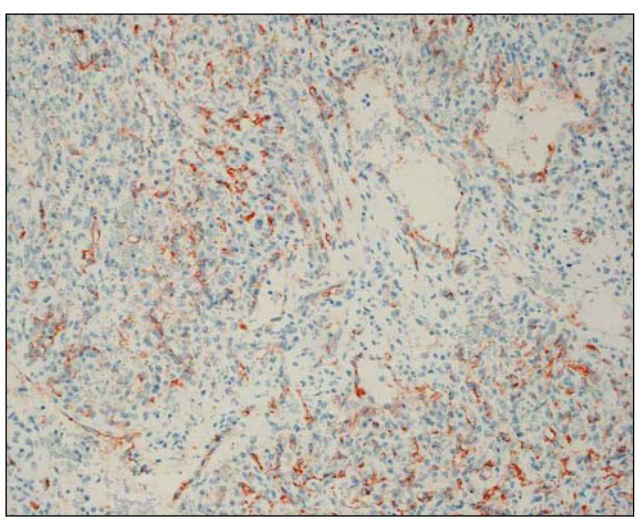

Fig. 3. Hamartoma, Case 2. The majority of vessels show CD31 antigen expression (original magnification x 200).

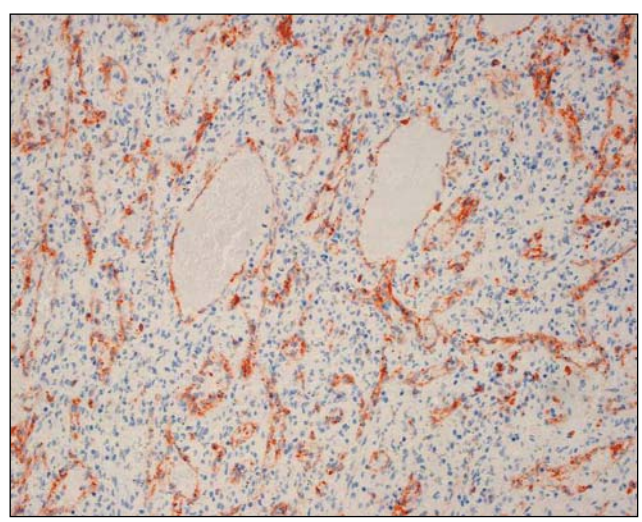

Fig. 4. Hamartoma, Case 1. The majority of vessels show CD8 antigen expression (original magnification x 200).

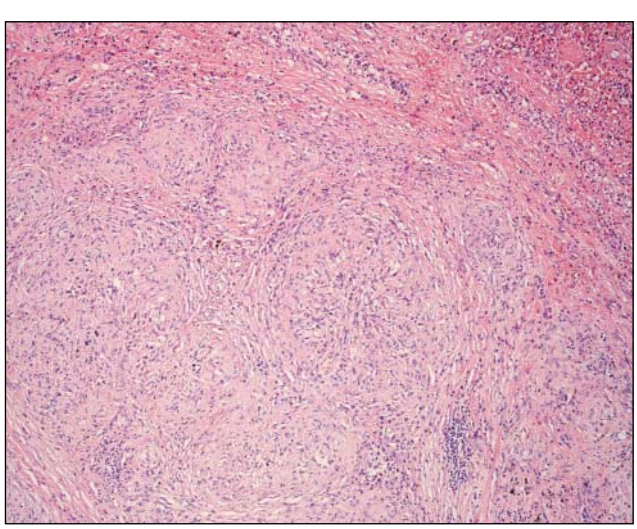

Fig. 5. SANT, Case 4 (hematoxylin and eosin, original magnification x 100).

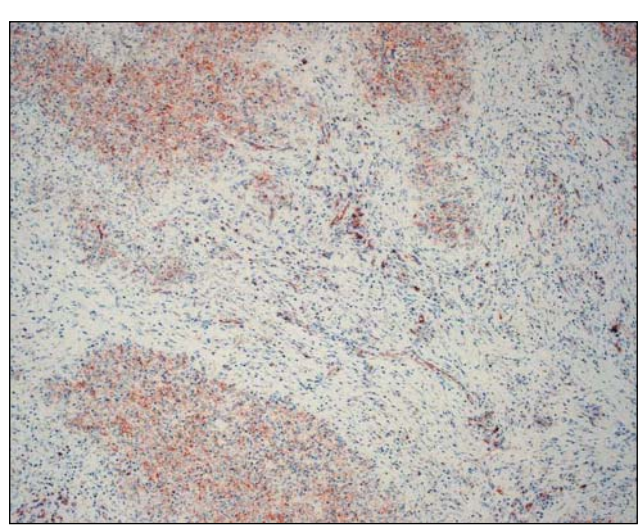

Fig. 6. SANT, Case 4. CD31 antigen expression (original magnification x100).

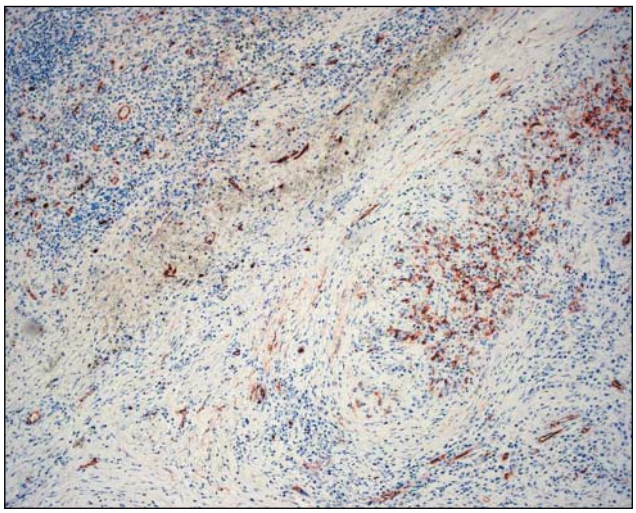

Fig. 7. SANT, Case 4. CD34 antigen expression (original magnification x 100).

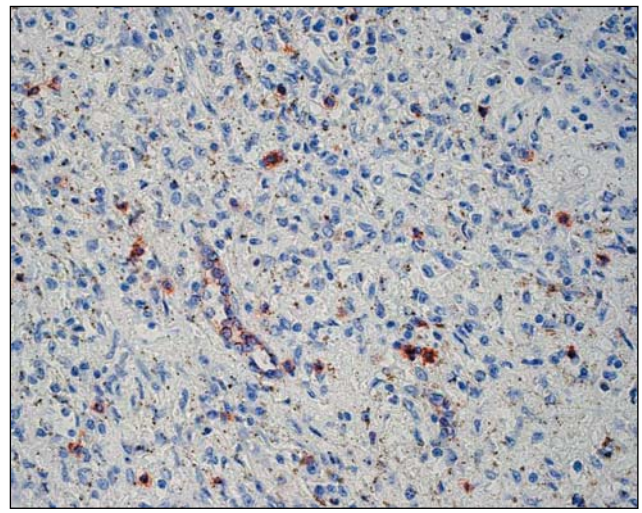

Fig. 8. SANT, Case 4. CD8 antigen expression (original magnification x 200).

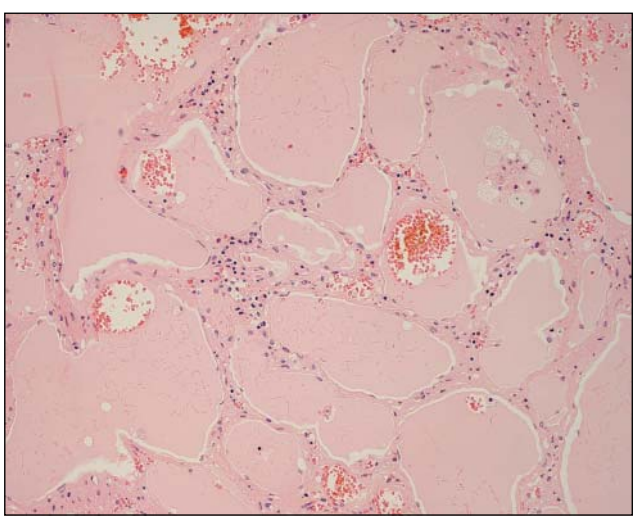

Fig. 9. Hemangioma, Case 5 (hematoxylin and eosin, original magnification x 200).

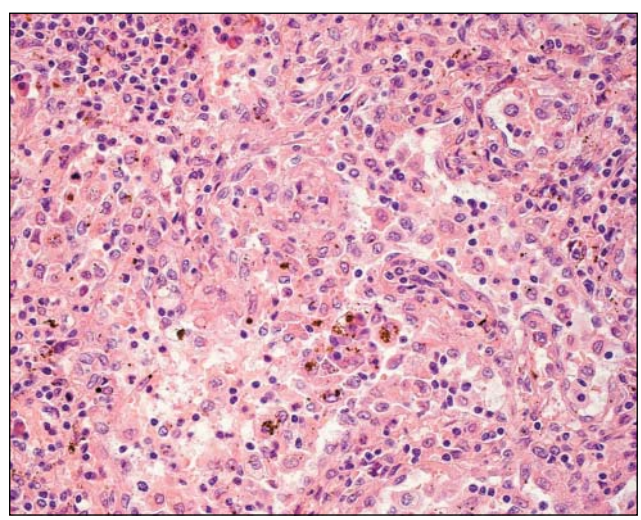

Fig. 10. Hemangioma, Case 7 (hematoxylin and eosin, original magnification x 400).

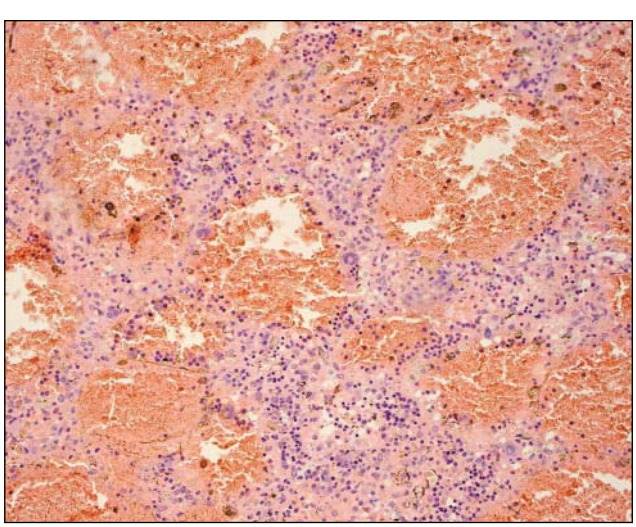

Fig. 11. Hemangioma, Case 8 (hematoxylin and eosin, original magnification x 200).

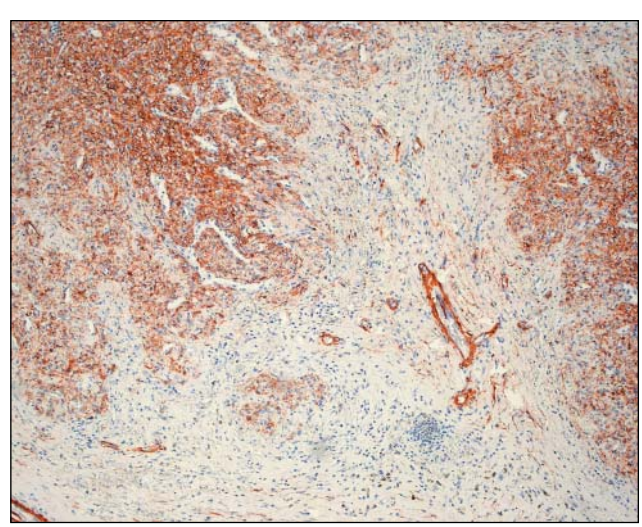

Fig. 12. SANT, Case 4. SMA antigen expression (original magnification x 100).

(Fig. 7) was present in small capillary vessels within the nodules and in larger vessels seen in the fibrous septae between the nodules. Single vessels with CD8 antigen expression were scattered throughout the lesion (Fig.8).

In the case of hemangiomas, the findings were diversified.

Case 5 represented a diffuse cavernous hemangioma composed of distended spaces lined with flattened epithelium and filled mainly with acidophilic protein contents, or, in some spaces, with bloody contents. Single vessels demonstrated aggregates of foamy macrophages (Fig. 9). The endothelial cells revealed expression of CD31 and CD34 antigens, but did not show expression of CD8 and CD68 antigens or lymphatic vessel markers: D2-40 and VEGFR3.

In Case 6, multifocal lesions were apparent, with the architecture of the classic cavernous hemangioma with focal aggregates of megakaryocytes between the vessels. The endothelial cells were characterized by expression of CD31, CD34 and CD68 antigens, but showed no expression of CD8 and CD21.

In Case 7, a single lesion, 5 mm in diameter, was seen; it was composed of irregular vascular spaces lined chiefly with flattened endothelial cells, which formed papilliform indentations protruding into the vessels. The vessels contained numerous foamy and hemosiderin-laden macrophages (Fig. 10). The endothelial cells demonstrated expression of CD31 and CD34 antigens, but no expression of CD21, CD8 and CD68.

In Case 8, among numerous distended vascular spaces, foci of extramedullary hematopoiesis were observed, as well as numerous hemosiderin-laden macrophages (Fig.11). The endothelial cells showed expression of CD31, CD68 and CD8 antigens, but did not demonstrate CD34 and CD21 expression.

The results of immunohistochemical reactions of the greatest importance in differential diagnosis are presented in Table 4.

In all the cases, reactions to GLUT-1, D2-40 and VEGFR3 antigens were also performed, but they all yielded negative results. Reactions to SMA were seen in all the cases – but especially in hamartomas and SANT (Fig. 12) in numerous myoid cells surrounding the vessels. Reactions to MPO and lysosyme were demonstrated by numerous macrophages in the stroma of the tumors. In all the lesions, proliferative activity measured by Ki67 expression did not exceed 1%.

Discussion

Differential diagnosis of benign vascular lesions of the spleen may be at times difficult in view of the diversified appearance of such lesions and their low incidence.

Some investigators consider hamartomas a variant of hemangioma, yet in hamartomas, both splenic sinus struc-

TABLE 4
Results of immunohistochemical reactions

Antigen \ Case No	1	2	3	4	5	6	7	8
	Hamartoma		**SANT**		**Hemangioma**			
CD31	++	++	++	++	++	++	++	++
CD34	++	++	+ +*	++*	++	+	++	-
Factor VIII	++	+	+	++	++	-	-	-
CD21	-	-	-	-	-	-	-	-
CD8	++	+	+	+	-	-	-	+
CD68	+	+	+	+	-	++	-	+

++ positive reaction in the majority of vessels
+ positive reaction in some vessels
* in numerous small vessels within tumors and in vessels of the septae

TABLE 5
Immunohistochemical differential diagnosis in benign vascular lesions of the spleen

	Splenic red pulp sinuses	Hamartoma	Hemangioma	Lymphangioma	Littoral-cell angioma
CD31	+	+	+	+	+
CD34	-	+/-	+	+	-/+
Factor VIII	+	+	+	+	+
CD68	+	-	+/-	+/-	+
CD8	+/-	+	-/+	-	-
CD21		-	-		+

tures and other red pulp elements are encountered. In the architecture of hemangiomas, well-organized lymphoid tissue is often observed, but no such finding is noted in hamartomas. Although fibrosis may be present in hamartomas, intensified fibrosis rather points to a hemangioma. Moreover, in immunohistochemistry, in hamartomas, the endothelium demonstrates CD8 expression, whereas hemangiomas rarely do so [3]. In the presented above Case 2, vessels with CD8 expression were less numerous than in Case 1, yet the presence of two types of vessels and stromal elements of the red pulp favored the diagnosis of a hamartoma rather than a hemangioma; in the latter, one type of vessels usually predominates.

In contrast to hemangioma, lymphangioma is often found in a subcapsular or peritrabecular location, while hemangiomas are situated mostly anywhere and the vessels are filled with protein contents rather than blood [2, 3]. Our Case 5 representing a cavernous hemangioma was recognized as such because - although the major part of vessels were filled with acidophilic protein contents - some vessels also showed bloody contents and, moreover, reactions to lymphatic vessel antigens (D2-40 and VEGFR3) were negative.

In addition, while performing differential diagnosis in Case 7, littoral cell angioma was taken into consideration in view of irregular vascular spaces with papilliform indentations protruding into the vessels and the vessels themselves being predominantly filled with foamy macrophages and hemosiderin-laden macrophages. However, the two populations of endothelial cells typical for littoral cell angioma were not observed, and the phenotype corresponded to a common hemangioma.

In addition to morphological properties, immunohistochemistry is of considerable help in differential diagnosis of benign vascular lesions of the spleen. Table 5 presents immunohistochemical reactions.

Histologically, hamartomas may manifest a varying picture, depending on the quantitative ratio of its components. Krishnan and Frizzera proposed distinguishing several histological variants of hamartoma, including in this group lesions that were classified as separate entities by other authors. Thus, the classification encompasses:

1. Classic hamartoma, which contains numerous CD8+ splenic sinus vessels with stromal elements (histiocytes and CD34+ capillary vessels),
2. Cord capillary hemangioma (SANT), where the predominating elements are represented by capillary vessels and fibrosis, while splenic sinus vessels appear in a small number,
3. Myoid angioendothelioma (benign vascular neoplasms of the spleen with myoid and angioendotheliomatous features), where myoid cells and capillary vessels predominate, while splenic sinuses are scarce,
4. Histiocyte-rich hamartoma, with predominance of histiocytes and CD34+ capillary vessels, and scattered CD8+ splenic sinuses [5].

Inasmuch as the classic variant of hamartoma and the histocyte-rich variant do not raise any major doubt, the status of the other two lesions is less clear.

The lesion first described as a cord capillary hemangioma , later reclassified by the same author as a variant of hamartoma [5], was described as SANT in 2004. In the same year, in the textbook by Rosai, a lesion with an identical histological picture was called “multinodular hemangioma” [6]. The authors that coined the term “SANT” postulate that the lesion may reflect changes occurring within the splenic red pulp in response to excessive proliferation of the stroma, but do not provide any explanation whether such a lesion develops de novo or is a terminal stage of other benign lesions (inflammatory pseudotumor, hamartoma or hematoma). Nevertheless, they do not rule out a possibility that this is indeed a variant of hamartoma. In their opinion, an argument against such a classification of the lesion might be found in a clear delineation of the lesion observed both macroscopically and microscopically and its distinct vascular character. According to the authors, the characteristic histological pattern seen in the lesion justifies

its recognition as a separate entity. On the other hand, the three separate types of vessels in SANT reflect the composition of the red pulp of the spleen, what might speak in favor of regarding the lesion as a variant of hamartoma [5].

Four cases of a lesion termed "benign vascular neoplasms of the spleen with myoid and angioendotheliomatous features" have been described. Macroscopically, these were single, well-delineated, solid tumors. Microscopically, the tumors were also well delineated, composed of small, medium-sized or, less frequently, large vessels lined with flattened endothelium. Their architecture showed a predominance of large polyhedral cells with blurred borders and abundant fibrillar or dense acidophilic cytoplasm. The nuclei of these cells varied in shape, from oval to elongated, were hyperchromatic, contained acidophilic nucleoli and demonstrated single intranuclear pseudoinclusions. In two cases, the architecture of the tumor was separated by small arterioles surrounded by basophilic fibromyxoid stroma containing stellate cells, as well as lymphocytes, plasmocytes and eosinophils (resembling an inflammatory pseudotumor). Immunohistochemically, the phenotype of endothelial cells was CD34+/CD8-/CD21-/CD31+/-/SMA-. The phenotype of the polyhedral cells was: SMA+/MSA+/vimentin+/desmin-/CD68+/-/S100-/CD8-/CD21-/EBV-/LMP [7].

Although the authors did not demonstrate expression of CD8 antigen in the endothelial cells, yet, similarly as in the case of SANT, such vessels may occur only singly in this type of lesion.

Our Case 2 of hamartoma presented an intermediate picture situated between the classic variant of hamartoma, SANT and myoid angioendothelioma. In this case, apart from a vascular component, areas were observed with stromal elements predominating, what at times gave an impression of the nodules becoming separate. Moreover, both in the two cases of hamartoma and in SANT, a large number of SMA+ cells was noted surrounding the vessels, what suggests that the lesions may constitute a spectrum of the histological picture of hamartoma.

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