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Hepatic pseudolymphoma: a clinicopathological study of five cases and review of the literature

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Hepatic pseudolymphoma is a rare and controversial condition, the clinicopathological characteristics of which have not been well documented. In this study, we retrospectively examined clinical and pathological features of five patients (two males and three females, 40–81 years old) with hepatic pseudolymphoma. Two patients had multiple lesions (two lesions each). Three patients had histories of chronic liver disorders, including primary biliary cirrhosis, nonalcoholic steatohepatitis, and chronic viral hepatitis B. Tumor sizes ranged from 0.5 to 5.5 cm in diameter (average, 2.1 cm). Histologically, hepatic pseudolymphoma consisted of tumorous infiltrates of mature lymphocytes with multiple lymph follicles or clusters of epithelioid histiocytes. Lymphocytes characteristically extended into nearby portal tracts. Ductal structures positive for cytokeratin 7 were entrapped in the peripheral parts of nodules. *In situ* hybridization of immunoglobulin light chains revealed B lymphocytes and plasma cells to be polyclonal. In addition, clonal rearrangements of immunoglobulin heavy chains could not be shown in any cases using PCR. Two patients were diagnosed by needle biopsy. Interestingly, their nodules spontaneously diminished in size without any treatment. Malignant transformation was not observed in any cases during the follow-up periods. In conclusion, this study revealed that hepatic pseudolymphoma had benign behavior. The diagnosis of hepatic pseudolymphoma can be challenging especially with biopsied specimens, but could be aided by a characteristic growth pattern, *in situ* hybridization, analyses of gene rearrangements, or a follow-up based on images.
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Nodular reactive lymphoid proliferation has been called pseudolymphoma or reactive lymphoid hyperplasia.[1] New techniques such as *in situ* hybridization and the analysis of gene rearrangements have revealed some lesions previously diagnosed as pseudolymphomas to be clonal proliferations of lymphocytes.[2] The concept of a marginal-zone B-cell lymphoma of the MALT type is now being considered for such lesions.[3,4] Moreover, pseudolymphoma is still an established disease entity.[5] Hepatic pseudolymphoma is an extremely rare condition, with most reports to date dealing with single cases.[6–24] Therefore, the pathogenesis and clinical implications of hepatic pseudolymphoma have not been well documented.

We studied five patients with hepatic pseudolymphoma to reveal the clinicopathological characteristics of this controversial disease entity.

Patients and methods

Patients

Five patients originally diagnosed with hepatic pseudolymphoma were selected from the histopathology files of Kanazawa University Hospital in Japan and consultation files of the authors. All patients were diagnosed from 1992 to 2009. Average age at the time of diagnosis was 63 years (range 40–81). There were three women and two men. Three patients underwent surgical resection of tumor masses and in two instances material was obtained

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with liver biopsy. One case (patient 1) was previously reported.[24]

Histological Examinations

After a macroscopic examination of the explanted livers, tissue blocks were sampled from nodular lesions and background livers. The sampled tissue and needle biopsied tissue were fixed in neutral formalin, and embedded in paraffin. Sections (4 μm) were cut from each paraffin block, and stained with hematoxylin and eosin or reticulin. Additional sections were saved for immunohistochemistry.

Immunohistochemistry

Representative blocks were chosen for immunohistochemistry in each case. Immunostaining of CD3, CD20, CD79α, cytokeratin 7 (CK7), or immunoglobulin G4 (IgG4) was performed by an autostainer (HX System Benchmark, Ventana Medical Systems, Tucson, AZ, USA). Primary antibodies used were a rabbit monoclonal antibody for human CD3 (LAB VISION corporation, Fremont, CA, USA), a rabbit monoclonal antibody for human CD20 (Abcam, Tokyo, Japan), a mouse monoclonal antibody for CD79α (Dako Cytomation, Glostrup, Denmark), a mouse monoclonal antibody for CK7 (Dako Cytomation), and a mouse monoclonal antibody for human IgG4 (ZYMED Laboratory, San Francisco, CA, USA). Sections were pretreated with a heated plate (for CD3, CD20, and CD79α) or proteinase (for IgG4). Negative controls were evaluated by substituting the primary antibody with similarly diluted nonimmunized mouse serum.

In situ Hybridization

The *in situ* hybridization of κ- and λ-chains was performed by an autostainer (HX System Benchmark, Ventana Medical Systems) as per the manufacturer's instructions. Specific probes for κ-chain and λ-chain were obtained from Ventana Medical Systems. We used specimens of gastric marginal-zone B-cell lymphoma (MALToma) for a positive control and those of tonsillitis for a negative control.

Gene Rearrangement Analysis

PCR was used to investigate the arrangement of the immunoglobulin heavy chain gene with DNA templates obtained from the paraffin-embedded specimens. Gene rearrangements were examined by semi-nested PCR using five primers: FR3A 5′-CT GTCGACACGGC(T/C)(G/C)TGTATTACTG-3′; LJH, 5′-AACTGCAGAGGAGACGGTGACC-3′; VLJH, 5′-G TGACCAGGGT(A/G/C/T)CCTTGGCCCCAG-3′; VH_{1-5}, 5′-AAGCTTGTGACCAGGGT(G/T/C)CC(T/C)TGGCC CCAG-3′; VH_6, 5′-AAGCTTGTGACCGTGGTCCCTT GCCCCCAG-3′. Two sets of semi-nested PCR were performed as follows: Set 1, outer nest FR3A-LJH, inner nest FR3A-VLJH; Set 2, outer nest FR3A-VJH, inner nest FR3A-mixed VH_{1-5}/VH_6. Tissue samples of B-cell lymphoma were used as positive controls and those without lymphomatous lesions as negative controls. After PCR, the amplified products were analyzed on 4.5% agarose gels.

Results

Clinical Features

Clinical features of five patients are listed in Table 1. Two patients were incidentally found to have nodular lesions in the liver during a work-up for diabetes mellitus (patient 1) and Takayasu's arteritis or anti-phospholipid syndrome (patient 5). Patient 2 had been followed up for primary biliary cirrhosis, diagnosed on the basis of a needle biopsy 9 years before. Similarly, patient 3 had a history of chronic viral hepatitis B. Patient 4 was referred to hospital

Table 1 Clinical characteristics of patients examined in this study

Patient	*Age (years)*	*Gender*	*Past history*	*No. of lesions*	*Size (in cm)*	*Location*	*Operation/ biopsy*
1	66	F	Nonalcoholic steatohepatitis, diabetes mellitus	1	1.5	Right lobe	Partial resection
2	63	F	Primary biliary cirrhosis, chronic thyroiditis, adrenocortical adenoma	2	0.9, 0.5	S7, S6	Partial resection
3	40	M	Chronic viral hepatitis B	1	2.0	S6	Partial resection
4	81	M	Cholecystolithiasis, no liver disease	1	5.5	S3	Needle biopsy
5	64	F	Takayasu aortitis, antiphospholipid syndrome, no liver disease	2	3.5, 1.0	S5, S7	Needle biopsy

F, female; M, male; S, segment.

with abdominal pain, where ultrasonography and computed tomography revealed a hepatic nodular lesion, as well as cholecystolithiasis. Patients 2 and 5 had two lesions. The remaining three patients had a single nodular lesion. Interestingly, all lesions except the one in patient 4 were in the right lobe. Tumor sizes ranged from 0.5 to 5.5 cm in diameter (average 2.1 cm). A total of four lesions were surgically resected from patients 1–3. Needle biopsies were performed in cases 4 and 5 (the larger lesion of patient 5). Those liver specimens from six lesions were used in the following analyses.

Pathological Features

Macroscopically, surgically resected specimens showed vaguely nodular, homogeneous, gray lesions. Histologically, all six lesions showed a nodular proliferation of mature small lymphocytes. Lymph follicles with germinal centers were well formed in all cases (Figure 1). In interfollicular areas, several histiocytes and immunoblasts were also identified. Clusters of epithelioid histiocytes without necrosis were observed in the lesions of patients 1 and 3. Multinucleated giant cells in a granulomatous reaction contained cholesterin clefts (patient 1). Small calcifications were also identified in patient 3. No atypical cells suggestive of neoplasia were identified in any lesions examined (Figure 2). Bile ducts were identified within the nodules, especially at the periphery. Ductal structures were angulated and surrounded by lymphocytes; however, characteristic lymphoepithelial lesions could not be identified. Interestingly, lymphocytic infiltration extended along portal tracts around all lesions (Figure 3). In addition, a capsule-like fibrous structure was partially identified around the lesion in patient 3. The two lesions in patient 2 showed similar histological features. Needle biopsies of cases 4 and 5 revealed the characteristic feature of a hepatic parenchyma divided by broad areas of lymphocytic infiltration, in which small bile ducts were entrapped (Figure 4). These features seemed to correspond to the edge of nodular lesions in surgical cases.

In the background liver, patient 1 showed steatosis, hepatocellular ballooning, and some Mallory bodies. These histological features were consistent with nonalcoholic steatohepatitis. In patient 2, the background liver showed portal fibrosis with bile duct damage and cholangitis, all of which corresponded to primary biliary cirrhosis, stage 2. Patient 3 showed chronic hepatitis B with focal interface hepatitis. Patients 4 and 5 showed only focal steatosis in the background livers.

Immunohistochemical Analysis and *In Situ* Hybridization

Lymphocytes within all lesions were composed of CD3-positive T cells and CD20/CD79α-positive B cells (Figure 5). T and B cells were regularly distributed. IgG4-positive cells were scarce in all lesions. Immunostaining of CK7 revealed irregularly distributed CK7-positive ductal structures especially at the periphery of each nodule (Figure 5). *In situ* hybridization of immunoglobulin light chains revealed the polyclonal nature of plasma cells in all lesions (Figure 6).

Analysis of Gene Rearrangements

A PCR analysis of the immunoglobulin heavy chain gene did not show clonal rearrangements in any liver specimens (Figure 7).

Figure 1 Histopathology of hepatic pseudolymphoma. Nodular infiltration by lymphocytes with the formation of lymphoid follicles is observed. Epithelioid cells cluster in the interfollicular area. H&E, original magnification: ×100.

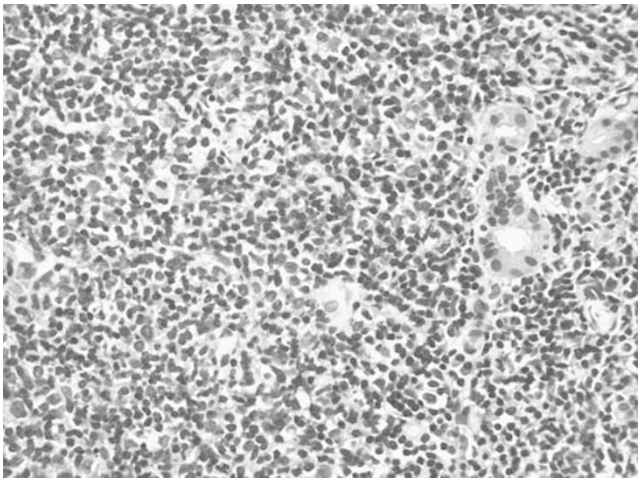

Figure 2 Histopathology of hepatic pseudolymphoma. Lymphocytes within a lesion are mainly small and appear mature with scattered medium and large cells. They have round nuclei with scant cytoplasm and do not show any atypical features. H&E, original magnification: ×400.

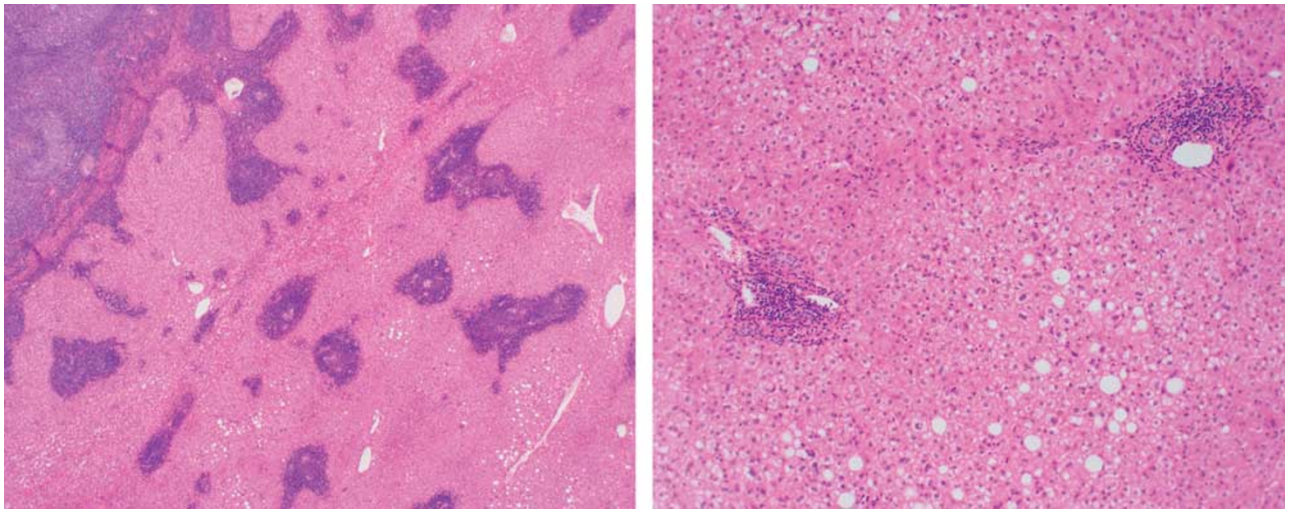

Figure 3 Histopathology of hepatic pseudolymphoma. At the edge of the nodule, lymphocytic infiltration extends into perinodular portal tracts (left). Compared with the background liver with chronic hepatitis B (right), lymphocytic infiltration is more pronounced in the portal tracts around the nodule (left). H&E, original magnification: left ×20; right ×100.

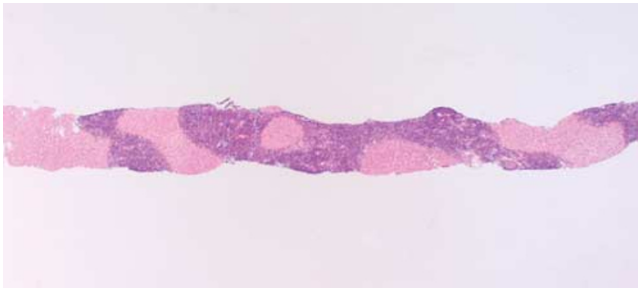

Figure 4 Histopathology of hepatic pseudolymphoma (a needle biopsy case). Hepatic parenchyma is divided by broad lymphocytic infiltration, in which bile ducts can be identified. H&E, original magnification: ×20.

Follow-Up Data

Patient 1 was found to have three nodular lesions 6 years after a surgical resection of pseudolymphoma. The three lesions were diagnosed as hepatocellular carcinomas (two lesions) and a dysplastic lesion by needle biopsies. There was no recurrence of the pseudolymphoma during the follow-up for 10 years. Similarly, no recurrence of lymphoproliferative lesions was identified in the other two surgical cases during the follow-up periods after surgical resection (15 months for patient 2 and 30 months for patients 3). After the diagnosis by needle biopsy, two patients were followed up for 6 months (patient 4) and 36 months (patient 5) without any treatments. Interestingly, nodules in those two cases gradually decreased in size on radiological images. In patient 4, the nodule decreased in diameter from 5.5 to 3.5 cm. The larger nodule in patient 5 decreased from 3.5 to 1.5 cm. Similarly, the smaller nodule, which was not biopsied, became inconspicuous on follow-up images. No patients were found to have malignant lymphomas at any sites, including the extrahepatic organs and lymph nodes during the follow-up periods.

Discussion

Hepatic pseudolymphoma is an extremely rare condition and most reports on it have been case reports. In 1999, Sharifi *et al*[13] reported three cases of pseudolymphoma, the largest series until now. All cases reported in English are reviewed in Table 2.[6–24] The total number of cases was 26, including 5 patients in the current study. Five patients (19%) had multiple (two or three) lesions. In total, 32 lesions were summarized. The youngest patient is a 15-year-old female, who had primary immunodeficiency. Except for that case, all patients were adults (36–85 years old). Interestingly, 82% of patients were female. In all, 60% of the lesions developed in the right hepatic lobe. The average diameter was 1.7 cm. Regarding background liver disease, 27% had chronic liver disorders. In addition, 23% of patients had autoimmune disorders in extrahepatic organs. Interestingly, 23% of patients had histories of malignancy, including gastric, colon, and renal cell cancers. Malignant transformation was not observed in any cases.

The pathogenesis of hepatic pseudolymphoma is still unknown, though it might involve infectious or autoimmune reactions.[25,26] The relatively high prevalence of autoimmune disorders suggests a possible involvement of autoimmunity to hepatic pseudolymphoma. In addition, the association with gastrointestinal malignancies suggests that antigens coming from the gastrointestinal tract through portal veins participate in the pathogenesis. However, the association with autoimmune or neoplastic

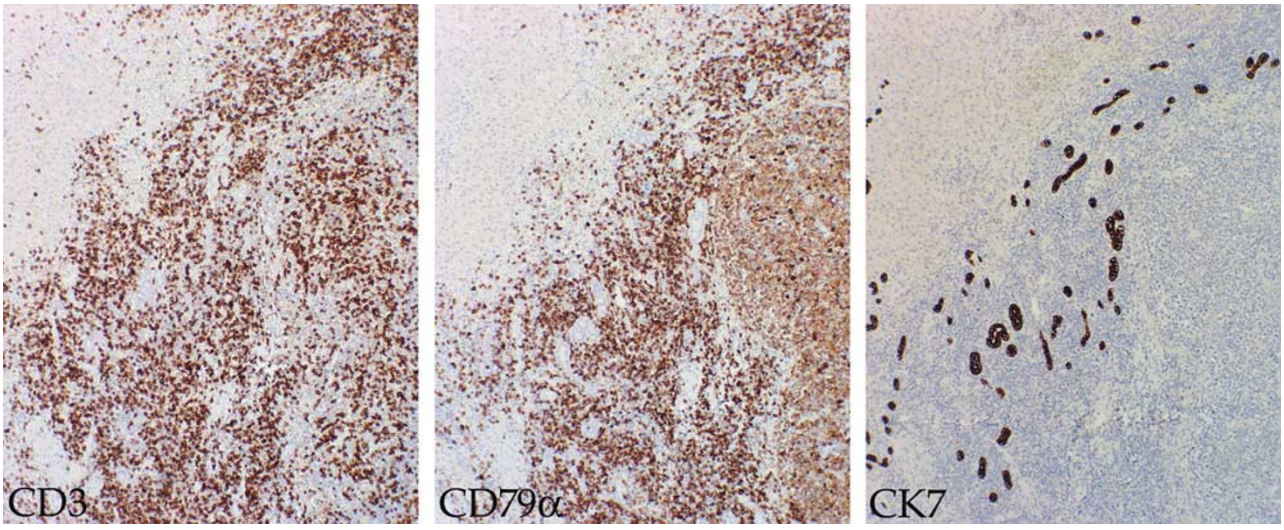

Figure 5 Immunostaining of CK7 in hepatic pseudolymphoma. Lymphocytes within the nodule consist of T cells (positive for CD3) and B cells (positive for CD79α). Ductal structures positive for CK7 are observed at the periphery of the nodule. All, original magnification: × 100.

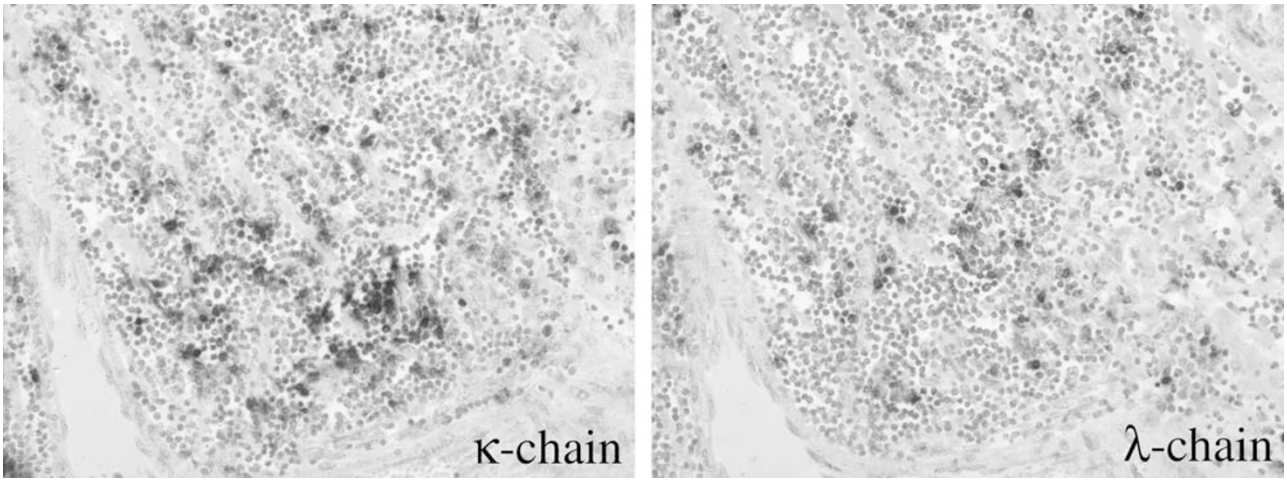

Figure 6 *In situ* hybridization of immunoglobulin light chains. The κ-positive and λ-positive cells are intermixed in this field. Original magnification: × 400.

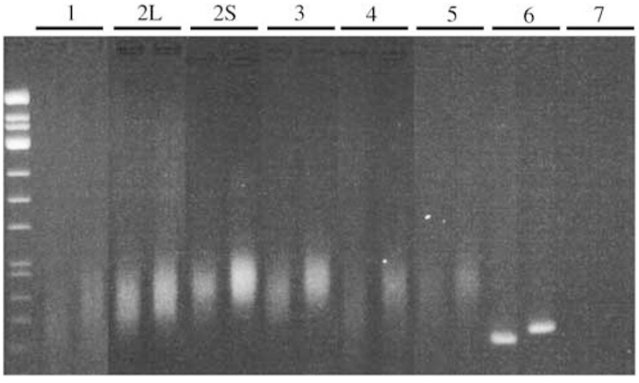

Figure 7 Immunoglobulin heavy chain gene rearrangements. Clonal rearrangement was not observed in any case. Lane 1, patient 1; lane 2L, the larger lesion in patient 2; lane 2S, the smaller lesion in patient 2; lane 3, patient 3; lane 4, patient 4; lane 5, patient 5; lane 6, positive control; and lane 7, negative control. Each case was examined with two sets of primers (left, FR3A-LJH, FR3A-VLJH; right, FR3A-LJH, FR3A-VH1-5/VH6).

conditions might merely reflect the fact that patients frequently undergo abdominal examinations using radiological images. Half of the previous reports were published after 2006. This is probably related to recent advances in imaging devices. Therefore, hepatic pseudolymphoma might be detected more in the future.

Making a diagnosis of pseudolymphoma by needle biopsy can be challenging. *In situ* hybridization of light-chain immunoglobulins and analyses of gene rearrangements can also be useful. In addition, two needle biopsy cases examined in this study revealed a characteristic pattern of infiltration by lymphocytes at the edge of nodules. Broad areas of infiltration divided the hepatic parenchyma. The hepatocytes formed hepatocellular islands within the lymphoid stroma. This might be another feature useful for the diagnosis. Immunochemistry of CK7 showed many entrapped bile ducts within and at

Table 2 Review of the literature regarding hepatic pseudolymphoma

Total number of cases	26
Total number of lesions	32
Age (average and range)	58 years (15–81)
Gender (male, female)	18, 82%
Location (right; left; caudate lobes)	15; 9; 1[a]
Size (average and range)	1.7 cm (0.5–5.5)
Multiple lesions	19% (2 or 3 lesions)
Chronic liver diseases	27%
Primary biliary cirrhosis[13]	Patient 2
Chronic viral hepatitis[9,11]	Patient 3
Nonalcoholic steatohepatitis	Patient 1
Nodular and neoplastic liver lesions	4%
Focal nodular hyperplasia[16]	
Cavernous hemangioma[16]	
Extrahepatic autoimmune disorders	23%
Sjögren syndrome[14]	
Hashimoto's thyroiditis[12,21]	Patient 2
Takayasu aortitis	Patient 5
Antiphospholipid syndrome	Patient 5
CREST syndrome[13]	
Carcinoma	23%
Gastric cancer[7,17]	
Colorectal cancer[17,18,23]	
Renal cell carcinoma[22]	

CREST, calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia.
[a]Data available in only 25 lesions.

the edge of nodules. This result suggests that pseudolymphoma is derived from lymphoid tissue related to a portal tract, and could be enlarged by involving nearby portal tracts.

It is important to distinguish hepatic pseudolymphoma from low-grade lymphoma.[27] Lymphoepithelial lesions and cellular atypia are important to be a differential diagnosis. It is rare to diagnose low-grade B-cell lymphoma by needle liver biopsy because of the infrequent hepatic involvement. Until now, we diagnosed only two cases of B-cell chronic lymphocytic leukemia (B-CLL) on a needle biopsy. Both showed portal distribution of atypical lymphoid cells without nodule formation. Two previous studies examined patterns of hepatic infiltration in malignant lymphoma.[28,29] The studies examined 294 cases in total, including 76 cases of low-grade B-cell lymphomas (marginal-zone B-cell lymphoma, B-CLL, mantle cell lymphoma, and follicular lymphoma).[28,29] The patterns of hepatic infiltration were classified into nodular, portal, and sinusoidal. Interestingly, combined results of both studies, most of low-grade B-cell lymphomas showed a predominantly portal infiltration pattern with or without sinusoidal infiltration. A nodular infiltration pattern like hepatic pseudolymphoma was identified in 15 of 76 cases (20%).[28,29] Interestingly, marginal-zone B-cell lymphoma, the most important differential diagnosis of hepatic pseudolymphoma, showed this pattern in only one case (6% of 16 cases).[28,29] It is still ambiguous whether or not a characteristic pattern of infiltration at the edge of hepatic pseudolymphoma can be observed in low-grade B-cell lymphoma. But, unique features observed in needle biopsies (Figure 4) was not described in those two studies. These data suggest that infiltration patterns could be helpful to discriminate hepatic pseudolymphoma from low-grade B-cell lymphoma.

Most hepatic pseudolymphomas have been treated surgically because of difficulties with the diagnosis by needle biopsy. In 2006, Ota *et al*[15] reported a patient with hepatic pseudolymphoma diagnosed by needle biopsy and followed up without surgical resection. In that report, the nodular size decreased from 1.6 to 0.8 cm during the follow-up for 18 months. Similarly, patients 4 and 5 in the current study also showed spontaneous regression. We first detected a decrease in tumor size at 2 and 4 months.

In conclusion, this study revealed that hepatic pseudolymphoma shows benign behavior. The diagnosis of hepatic pseudolymphoma can be challenging but might be aided by *in situ* hybridization, analyses of gene rearrangements, or follow-up based on images.

Disclosure/conflict of interest

The authors declare no conflict of interest.

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