Int J Hematol (2008) 88:418–423
DOI 10.1007/s12185-008-0153-9

CASE REPORT

Primary hepatic marginal zone B cell lymphoma of mucosa-associated lymphoid tissue type: case report and review of the literature

Hirokazu Doi · Norio Horiike · Atsushi Hiraoka · Yohei Koizumi · Yasunori Yamamoto · Aki Hasebe · Soichi Ichikawa · Makoto Yano · Yasunao Miyamoto · Tomoyuki Ninomiya · Yoshihiro Ishimaru · Masao Miyagawa · Kazuhito Takamura · Hideki Kawasaki · Teruhiko Kozuka · Toshiharu Maeda · Tadashi Yoshino

Received: 19 January 2008 / Revised: 22 June 2008 / Accepted: 4 July 2008 / Published online: 23 September 2008

Abstract A primary hepatic marginal zone B cell lymphoma of mucosa-associated lymphoid tissue (MALT) is very rare. We found a solitary mass 27 mm in size in the left lobe of the liver of a 58-year-old Japanese man with a history of hepatitis-C infection. Based on the results of imaging studies, the tumor was diagnosed as a hepatocellular carcinoma (HCC). The left lobe of the liver was lobectomized and microscopic findings showed that the tumor was a hepatic MALT lymphoma, while immunohistochemistry showed it to be positive for CD20 and CD79a. In a fluorodeoxyglucose-positron emission tomography examination integrated with computed tomography scanning (FDG-PET CT) before surgery, the tumor was revealed to have a high standardized uptake value (SUV) for FDG. The patient received chemotherapy after surgery. To the best of our knowledge, 45 cases had been reported with a mean age for all patients of 61.4 years. The pathogenesis remains unclear, although half of the patients had a past history of chronic inflammatory liver disease. Surgical resection was performed in most cases and some patients received postoperative chemotherapy or radiotherapy. The clinicopathologic characteristics and management of this extremely rare disease are also discussed.

Keywords MALT lymphoma · Hepatic malignant lymphoma · FDG-PET

H. Doi (✉) · N. Horiike · A. Hiraoka · Y. Koizumi · Y. Yamamoto · A. Hasebe · S. Ichikawa · M. Yano · Y. Miyamoto · T. Ninomiya
Department of Gastroenterology, Ehime Prefectural Central Hospital, Ehime 790-0024, Japan
e-mail: hirokazud@me.pikara.ne.jp

Y. Ishimaru · M. Miyagawa
Department of Radiology, Ehime Prefectural Central Hospital, Ehime, Japan

K. Takamura · H. Kawasaki
Department of Surgery, Ehime Prefectural Central Hospital, Ehime, Japan

T. Kozuka
Department of Hematology, Ehime Prefectural Central Hospital, Ehime, Japan

T. Maeda
Department of Medical Laboratory, Ehime Prefectural Central Hospital, Ehime, Japan

T. Yoshino
Department of Pathology, Okayama University Graduate School of Medicine and Dentistry, Okayama, Japan

1 Introduction

Primary hepatic malignant lymphomas are rare, with the most common histological type reported to be a diffuse large B cell lymphoma [1]. We treated an extremely rare case of mucosa-associated lymphoid tissue (MALT) type that originated in the liver [2]. To the best of our knowledge, this is the only report of primary hepatic MALT lymphoma that was examined with fluorodeoxyglucose-positron emission tomography integrated with CT scanning (FDG-PET CT). Herein, we compare our findings to those of previously reported cases with respect to clinical presentation, pathologic features, therapy, and clinical outcome.

2 Case report

A 58-year-old Japanese male was referred by his physician for an abdominal ultrasonography (US) examination in

June 2007. The patient had a history of hepatitis C virus (HCV) infection discovered 15 years ago and was curatively treated with polyethylene glycol interferon (PEG-IFN)-α-2b and ribavirin in August 2005. Thereafter, HCV-RNA was negative in follow-up examinations and there was no evidence of hepatocellular carcinoma (HCC) in imaging studies. He also showed no abnormal physical findings, including lymphadenopathy and hepatosplenomegaly. Blood cell counts and serochemical findings including liver enzymes, concentrations of serum α-fetoprotein (AFP), fucosylated AFP (L3-AFP), protein induced by vitamin K absence or antagonist II (PIVKA-II), and soluble Interleukin 2 (s-IL2) receptor were within normal limits and anti-Helicobacter pyloric antibody was positive (Table 1). Our abdominal US examination showed a solitary and hypoechoic homogeneous mass with a maximum size of 27 mm in the second segment (S2) of the left lobe of the liver. There were no masses in the spleen, splenomegaly, lymphoadenopathy, ascites, or other abnormal findings.

The liver tumor was enhanced peripherally in the early phase and detected in the delayed phase by dynamic CT scanning (Fig. 1). Using FDG-PET CT imaging, the tumor was revealed by a high standardized uptake value (SUV) for FDG, while there was no other areas with high levels of SUV (Fig. 2). CT angiography (CTA) and CTA portography

Table 1 Laboratory data on admission

WBC	3,620/μl	*Blood urea nitrogen*	*21.4 mg/dl*
RBC	520 × 10^4/μl	Creatinine	0.84 mg/dl
Hb	14.7 g/dl	Sodium	142 mEq/l
Hematocrit	44.7%	Potassium	3.8 mEq/l
MCV	86.0 fl	Chloride	106 mEq/l
MCH	28.3 pg	Glucose	109 mg/dl
MCHC	32.9%		
Platelet count	11.5 × 10^4/μl	Prothrombin time	83.5%
		Activated partial thromboplastin time	39.5 s
Total-bilirubin	*1.4 mg/dl*		
Direct-bilirubin	*0.5 mg/dl*	HBs-Ag	(−)
Aspartate aminotransferase	22 IU/I	*Anti-HCV*	(+)
Alanine aminotransferase	16 IU/I	HCV-RNA	(−)
Alkaline phosphatase	182 IU/I		
Lactate dehydrogenase	172 IU/I	AFP	3.7 ng/ml
γ-Glutamyltranspeptidase	35 IU/I	L3-AFP	0.0%
Total protein	8.0 g/dl	PIVKA-II	35 mAU/ml
Albumin	4.6 g/dl	*sIL2-R*	*677 U/ml*
		H. pylori-Ab	(+)

The significance of *italics* shows abnormals of laboratory data

HBs-Ag hepatitis B virus surface antigen, *Anti-HCV* hepatitis C virus antibody, *AFP* α-Fetoprotein, *PIVKA-II* protein induced by vitamin K absence or antagonist II, *sIL2-R* soluble interleukin 2 receptor, *H. pylori-Ab* helicobacter pylori antibody

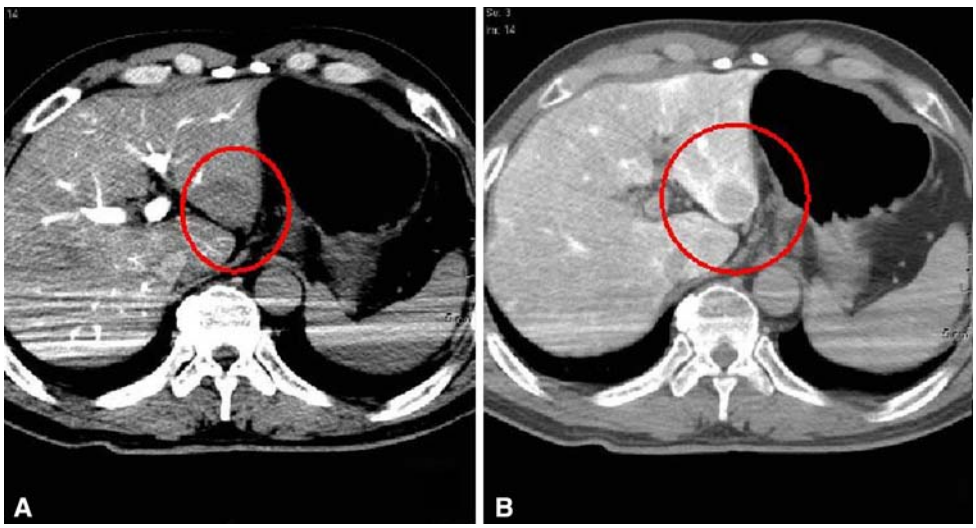

Fig. 1 **a** Tumor in the second segment of the left lobe of the liver enhanced peripherally in the early phase by dynamic computed tomography (CT). **b** The tumor was not enhanced in the delayed phase by dynamic CT. **b** The tumor was not enhanced in the delayed phase by dynamic CT

(CTAP) findings were similar to those of dynamic CT. Due to the results of the imaging studies and his past history of infection with HCV, we diagnosed the tumor as an HCC and performed a hepatic resection in July 2007.

Macroscopic findings of the resected specimen revealed that the tumor measured 25 × 20 mm and had no capsular formation. Microphotography showed heterogeneous lymphocytes forming reactive follicles. Plasma cells and giant cells proliferating in a medullary manner and clearly nodular pattern were observed in the tumor. Immunohistochemistry findings were positive for CD20 and CD79a, while they were negative for CD3, CD5, and CD10 (Fig. 3). As a result, we diagnosed the tumor as a primary hepatic marginal zone B cell lymphoma of MALT type. t(11;18)(q21;q21) that is most popular fusion signal in MALT lymphoma could not be proven by a two-color fluorescence in situ hybridization (FISH) assay after resection.

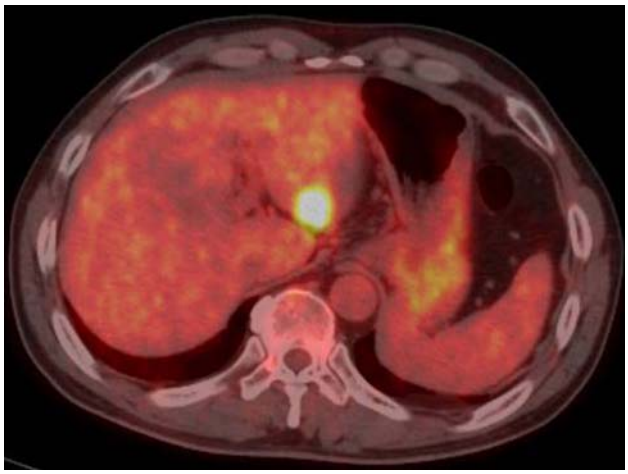

Fig. 2 The tumor showed a high standardized uptake value (SUV) for fluorodeoxyglucose (FDG). No other areas with high levels of SUV were found in FDG-positron emission tomography integrated with computed tomography imaging

FDG-PET CT, and upper endoscope and colonscope examinations found no other MALT lymphoma in any other organ. The patient received three courses of chemotherapy (R-CHOP consisting of rituximab at 375 mg/m^2 on day 1, cyclophosphamide at 750 mg/m^2 on day 2, doxorubicin at 50 mg/m^2 on day 2, vincristine at 1.4 mg/m^2 on day 2, and prednisolone at 100 mg/kg of body weight on days 2–6) after resection and has not shown relapse up to the time of writing.

3 Discussion

Jaffe et al. [3] reported that hepatic malignant lymphomas comprised less than 1% of all malignant lymphomas, while hepatic MALT lymphomas are reported to occur in only 3% of cases of hepatic malignant lymphoma [1]. In the present patient, the levels of s-IL2 receptor were not high, and he had a past history of hepatitis with HCV and HCV-RNA that was eliminated by IFN therapy (PEG-IFN-α-2b), therefore, we treated the tumor as an HCC, based on the results of imaging studies. A retrospective analysis of the ultrasonographic findings at admission showed that the tumor appeared as a cystic lesion. Although there were no findings to indicate a malignant lymphoma in systemic screening, it is important to distinguish not only HCC but also hepatic malignant lymphoma by considering the criteria for each [1].

The etiology of hepatic malignant lymphomas, especially MALT lymphomas, has not been elucidated. Although a past report [4] noted a relationship between HCV and hepatic malignant lymphoma, there are no reports that concluded a relationship between HCV and MALT lymphoma. In an interesting study of gastric MALT lymphomas in patients infected with HCV, Tursi [5] reported the disappearance of a gastric MALT lymphoma after successful treatment with IFN-α-2b and ribavirin. However, although a relationship between HCV-infection and MALT lymphoma is speculated, the MALT lymphoma

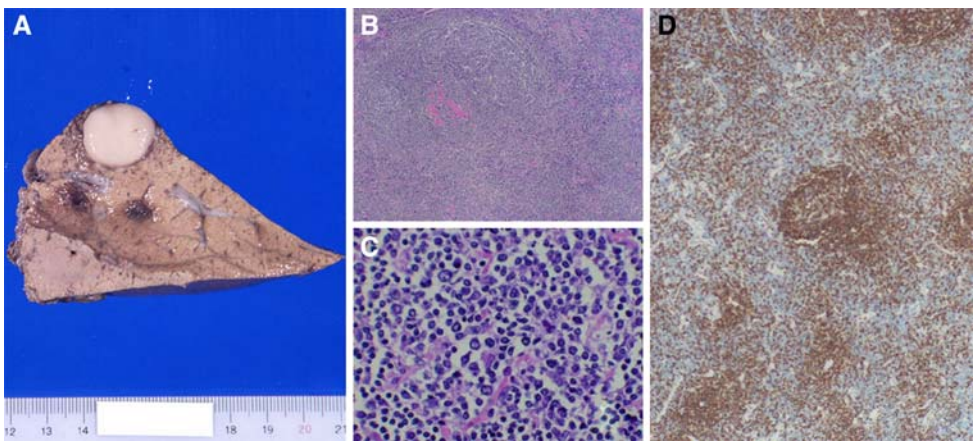

Fig. 3 **a** Gross appearance of the confined hepatic tumor. The cut surface of the tumor showed a *yellow-white* medullary pattern. **b** A vague nodular pattern of lymphoid cells was found to have infiltrated along Gleason's sheath (H&E stain, ×100). **c** Higher magnification showing proliferation of lymphoid cells (H&E stain, ×400). **d** Lymphoid cells diffusely positive for CD20 antibody (×100)

Table 2 Summary of clinical features of reported cases of primary hepatic low-grade MALT lymphoma

Case no.	Age/sex [ref]	Liver mass	Associated disease	HBs antigen	Anti-HCV	Treatment	Outcome
1	64/M [6]	Solitary	Gastric cancer	(−)	(−)	Resection	Alive/2 years
2	66/M [9]	Solitary	Ureteral carcinoma	NA	NA	Resection	Alive/1 year
3	73/F [9]	Solitary	Cholecystectomy	NA	NA	Resection	Unknown
4	85/M [9]	Dual	Prostate cancer, Abdominal aortic aneurysm	NA	NA	Liver biopsy	Death/unknown
5	60/F [9]	Multiple	IgM paraproteinemia, liver cirrhosis	(+)	NA	Liver transplantation	Alive/1 year
6	48/M [10]	Solitary	Chronic hepatitis B	(+)	NA	Resection + chemotherapy	Alive/3 years
7	47/F [11]	Solitary	Multiple biliary unilocular cysts	(−)	(−)	Resection + radiotherapy	Alive/2.5 years
8	64/M [11]	Solitary	Colon cancer	NA	NA	Resection	Unknown
9	62/F [12]	Solitary	Primary biliary cirrhosis	(−)	(−)	Resection	Unknown
10	64/F [13]	Solitary	Liver cirrhosis (C)	NA	(+)	CVP	Alive/2 years
11	65/F [13]	Solitary	Liver cirrhosis (C)	NA	(+)	Vinblastine	Alive/2.5 years
12	69/F [14]	Solitary	None	NA	NA	Resection	Unknown
13	57/F [15]	Solitary	Primary biliary cirrhosis	(−)	(−)	Liver transplantation	Alive/9 months
14	64/F [16]	Solitary	Ascariasis	NA	NA	Resection	Pulmonary MALT lymphoma after 8 years
15	61/M [17]	Solitary	Gastric cancer, history of liver injury	(−)	(−)	Resection	Alive/1.5 years
16	73/M [18]	Solitary	Liver cirrhosis (C)	(−)	(+)	Resection	Alive/34 months
17	72/F [19]	Solitary	Colon cancer	(−)	(−)	Resection	Alive/2 years
18	65/F [20]	Multiple	Chronic hepatitis B	(+)	(−)	Resection	Alive/10 months
19	72/F [21]	Solitary	Colorectal adenocarcinoma	NA	NA	Liver biopsy	Died 1 month after liver biopsy
20	61/F [21]	Solitary	Rheumatoid arthritis	NA	NA	None	Died of pulmonary embolism
21	58/F [21]	Multiple	None	NA	NA	Chemotherapy	Alive/37 months
22	62/F [21]	Solitary	Breast cancer	NA	NA	Resection + tamoxifen	Alive/9 months
23	59/M [22]	Solitary	Liver cirrhosis (C)	NA	(+)	Resection	Alive/30 months
24	64/M [23]	Solitary	Primary biliary cirrhosis	(−)	(−)	Liver transplantation	Alive/11 months
25	57/F [24]	Solitary	Ascariasis	NA	NA	Resection	Alive/4.6 years
26	66/M [24]	Solitary	Unknown	NA	(−)	Spontaneous remission	Alive/3 years
27	69/M [24]	Solitary	Unknown	NA	(−)	Unknown	Unknown
28	50/M [24]	Solitary	Unknown	NA	(−)	Unknown	Unknown
29	50/F [25]	Solitary	None	(−)	(−)	Resection + rituximab	Alive/2.5 years
30	69/M [26]	Dual	None	(−)	(−)	Radiofrequency Ablation therapy + rituximab	Alive/2 years
31	67/M [27]	Solitary	Drug hepatitis	(−)	(−)	Radiotherapy	Alive/6 years
32	41/F [28]	Solitary	Primary biliary cirrhosis	(−)	(−)	Followup carefully	Alive/1 year
33	64/F [29]	Solitary	None	(−)	(−)	Resection	Alive/6 years
34	59/F [30]	Multiple	Unknown	NA	NA	Resection + 2-CdA	Unknown
35	53/M [31]	Multiple	Liver cirrhosis (C)	NA	(+)	Liver transplantation + rituximab	Alive/1 month
36	36/M [32]	Solitary	History of hepatitis	(+)	NA	Resection + rituximab	Alive/26 months
37	59/M [33]	Dual	Liver cirrhosis (B)	(+)	NA	Liver transplantation	Alive/6 months
38	59/M [34]	Solitary	Burger disease	(−)	(−)	Resection	Alive/5 months
39-45	[35–38]	Not available					

Table 2 continued

Case no.	Age/sex [ref]	Liver mass	Associated disease	HBs antigen	Anti-HCV	Treatment	Outcome
46	Present case	Solitary	Chronic hepatitis C after IFN therapy	(−)	(+)	Resection + R-CHOP	Alive/6 months

MALT mucosa-associated lymphoid tissue, *HBs antigen* hepatitis B virus surface antigen, *HCV* hepatitis C virus, *M* male, *F* female, *NA* not available, *CVP* cyclophosphamide + vincristine + prednisolone, *R-CHOP* rituximab + cyclophosphamide + doxorubicin + vincristine + prednisone, *2-CdA* 2-Chlorodeoxyadenosine

in the present patient occurred following successful treatment with IFN. Although, infection of *Helicobacter pylori* (*H. pylori*) have influence for gastric MALT lymphoma, we cannot deny the relationship between infection of *H. pylori* and the pathogenesis of primary hepatic MALT lymphoma. Iida [6] described that there was a strong association between the presence of *H. pylori* in the stomach and in the bile juice. More accumulation of cases will be needed for certain conclusion.

The molecular genetics underlying the pathogenesis of MALT lymphomas are only recently beginning to be understood. At least three recurrent chromosomal translocations, t(11;18)(q21;q21), t(1;14)(p22;q32), t(14;18)(q32;q21) have been implicated in the pathogenesis and t(11;18)(q21;q21) is the most frequent translocation depending on the site of tumor origin; lung (38%), stomach (24%), conjunctiva (19%), and orbit (14%) [7]. In the present case, we have examined the t(11;18)(q21;q21) by FISH analysis and no fusion signal has been detected.

In regard to imaging studies, Perry et al. [8] reported that FDG-PET CT was useful for initial staging in patients with an extranodal MALT lymphoma, although the intensity of FDG uptake seemed to be lower in indolent non-Hodgkin's lymphoma when compared with aggressive non-Hodgkin's lymphoma. Since the sensitivity of FDG-PET CT depends on disease location and stage at initial diagnosis, the present primary hepatic MALT lymphoma was revealed with a high uptake of FDG. To the best of our knowledge, there are no other reports of patients with a hepatic MALT lymphoma that underwent an FDG-PET CT examination. Since the present case did not show high levels of tumor markers (AFP, L3-AFP, PIVKA-II), we considered other types of liver tumors beside an HCC. In these cases, FDG-PET CT may reveal the liver tumor as a primary hepatic MALT lymphoma, although a greater accumulation of case reports is needed.

A primary hepatic MALT lymphoma is an extremely rare disorder, with only 45 cases reported [6, 9–38] (Table 2), mainly elderly patients (mean age, 61.4 years old). In most patients, the liver tumor was identified incidentally by surgical resection or imaging studies during examinations for liver or other diseases. In 16 cases, pre-existing liver disease, including primary biliary cirrhosis and infectious liver diseases (e.g. hepatitis B virus, HCV, ascariasis), was present and chronic liver inflammation is assumed to be a factor in the development of MALT lymphomas. The present case had a past history of hepatitis with HCV and HCV-RNA that was eliminated by IFN therapy (PEG-IFN-α-2b), and we consider that the participation of HCV in the development of a hepatic MALT lymphoma was not denied by our findings.

Because of the small number of patients with a primary hepatic MALT lymphoma, therapeutic methods have not been established. Surgical resection was performed in most of the reported patients with good liver function. Recently, one case underwent radiofrequency ablation therapy (RFA) [26] and another radiotherapy alone [27], while other patients received chemotherapy or postoperative radiotherapy. Nasr [39] reported that therapeutic methods for primary hepatic non-Hodgkin lymphoma have not been established (e.g. surgery, radiation, chemotherapy, or combinations). Further, Raderer [40] found that rituximab was effective for 50% of cases with an advanced MALT lymphoma and could be used safety without a reactivation of HCV. We selected chemotherapy after surgical resection, because an extra-gastric MALT lymphoma, not only hepatic but also extra-hepatic, is reported to be relapse more frequently than a gastric MALT lymphoma (gastric MALT lymphoma vs. extra-gastric MALT lymphoma: 22.2 vs. 48.0%) [38]. To date, no recurrence of the MALT lymphoma has occurred after the surgical resection and chemotherapy treatments. Establishment of therapeutic methods for MALT lymphoma is expected, although additional accumulation of cases is needed to establish effective diagnostic methods (e.g. FDG-PET CT) and treatments.

References

1. Lei KI. Primary non-Hodgkin's lymphoma of the liver. Leuk Lymphoma. 1998;29:293–9.
2. Zucca E, Conconi A, Pedrinis E, et al. Nongastric marginal zone B-cell lymphoma of mucosa associated lymphoid tissue. Blood. 2003;101:2489–95.
3. Jaffe ES, et al. Malignant lymphomas: pathology of hepatic involvement. Semin Liver Dis. 1987;7:257–68.

4. Izumi T, Sasaki R, Miura Y, et al. Primary hepatosplenic lymphoma: association with hepatic C virus infection. Blood. 1996;87:5380–1.
5. Tursi A, et al. Disappearance of gastric mucosa-associated lymphoid tissue in hepatitis C virus-positive patients after anti-hepatitis C virus therapy. J Clin Gastroenterol. 2004;38:360–3.
6. Iida T, Iwahashi M, Nakamura M, et al. Primary hepatic low-grade B-cell lymphoma of MALT-type associated with *Helicobacter pylori* infection. Hepatogastroenterology. 2007;54:1898–901.
7. Ye H, Liu H, Attygalle A, et al. Variable frequencies of t(11;18)(q21;q21) in MALT lymphomas of different sites: significant association with CagA strains of *H. pylori* in gastric lymphoma. Blood. 2003;102:1012–8.
8. Perry C, Herishanu Y, Metzer U, et al. Diagnostic accuracy of PET/CT in patients with extranodal marginal zone MALT lymphoma. Eur J Hematol. 2007;79:205–9.
9. Isaacson PG, Banks PM, et al. Primary low-grade B-cell lymphoma of mucosa-associated-lymphoid tissue (MALT)-type. Am J Surg Pathol. 1995;19:571–5.
10. Ueda G, Oka K, Matsumoto T, et al. Primary hepatic marginal zone B-cell lymphoma with mantle cell lymphoma phenotype. Virchows Arch. 1996;428:311–4.
11. Maes M, Depardieu C, et al. Primary low-grade B-cell lymphoma of MALT-type occurring in the liver: a study of two cases. J Hepathol. 1997;27:922–7.
12. Prabhu RM, Medeiros LJ, et al. Primary hepatic low-grade lymphoma of mucosa-associated lymphoid tissue (MALT) associated with primary biliary cirrhosis. Mod Pathol. 1998;11:404–10.
13. Ascoli V, Lo Coco F, et al. Extranodal lymphoma associated with hepatitis C virus infection. Am J Clin Pathol. 1998;109:600–9.
14. Kirk CM, Levin D, et al. Primary hepatic B-cell lymphoma of mucosa-associated lymphoid tissue. Arch Pathol Lab Med. 1999;123:716–9.
15. Ye MQ, Suriawinata A, et al. Primary hepatic marginal zone B-cell lymphoma of mucosal associated lymphoid tissue type in a patient with primary biliary cirrhosis. Arch Pathol Lab Med. 2000;124:604–8.
16. Chen F, Ike O, et al. Pulmonary mucosa-associated lymphoid tissue lymphoma 8 years after resection of the same type of lymphoma of the liver. Jpn J Thorac Cardiovasc Surg. 2000;48:233–5.
17. Murakami J, Fukusima N, et al. Primary hepatic low-grade B-cell lymphoma of the mucosa-associated lymphoid tissue type: a case report and review of the literature. Int J Hematol. 2002;75:85–90.
18. Yago K, Shimada H, et al. Primary low-grade B-cell lymphoma of mucosa-associated lymphoid tissue (MALT)-type of the liver in a patient with hepatitis C virus infection. Leuk Lymphoma. 2002;43:1497–500.
19. Chatelain D, Maes C, et al. Primary hepatic lymphoma of MALT-type: a tumor that can simulate a liver metastasis. Ann Chir. 2006;131:121–4.
20. Takeshima F, Kunisaki M, et al. Hepatic mucosa-associated lymphoid tissue lymphoma and hepatocellular carcinoma in a patient with hepatitis B virus infection. J Clin Gastroenterol. 2004;28:823–6.
21. Streubel B, Lamprecht A, et al. T(14;18) (q23;q21) involving IGH and MALT1 is a frequent chromosomal aberration in MALT lymphoma. Blood. 2003;101:2335–9.
22. Mizuno S, Isaji S, et al. Hepatic mucosa-associated lymphoid tissue (MALT) lymphoma associated with hepatitis C. J Hepatol. 2002;37:872–3.
23. Golli L, Taieb J, et al. Lymphome du MALT hepatique de bas grade associe a une cirrhose biliaire primitive. Gastroenterol Clin Biol. 2003;27:127–9.
24. Arai O, Wani Y, et al. A case of primary hepatic low-grade B-cell lymphoma of mucosa-associated lymphoid tissue (MALT). Liver Cancer. 2003;9:144–9.
25. Ramos-Casals M, Trejo O, et al. Triple association between hepatitis C virus infection, systemic autoimmune diseases, and B-cell lymphoma. J Rheumatol. 2004;31:495–9.
26. Hamada M, Tanaka Y, et al. A case of MALT lymphoma of the liver treated by RFA and Rituximab. Nippon Shokakibyo Gakkai Zasshi. 2006;103:655–60.
27. Shin SY, Kim JS, et al. Longlasting remission of primary hepatic mucosa-associated lymphoid tissue (MALT) lymphoma achieved by radiotherapy alone. Korean J Intern Med. 2006;21:7127–31.
28. Sato S, Masuda T, Oikawa H, et al. Primary hepatic lymphoma associated with primary biliary cirrhosis. Am J Gastroenterol. 1999;94:1669–73.
29. Bouron D, Leger-Ravet MB, Gaulard P, et al. Unusual hepatic tumor. Ann Pathol. 1999;19:547–8.
30. Raderer M, Traub T, Formanek M, et al. Somatostatin-reseptor scintigraphy for staging and follow-up of patients with extraintestinal marginal zone B-cell lymphoma of the mucosa associated lymphoid tissue(MALT)-type. Br J Cancer. 2001;85:1462–6.
31. Orrego M, Guo L, Reeder C, et al. Hepatic B-cell non-Hodgkin's lymphoma of MALT type in the liver explant of a patient with chronic hepatitis C infection. Liver Transpl. 2005;11:796–9.
32. Gockel HR, Heidemann J, Lugering A, et al. Stable remission after administration of rituximab in a patient with primary hepatic marginal zone B-cell lymphoma. Eur J Haematol. 2005;74:445–7.
33. Nart D, Ertan Y, Yilmaz F, et al. Primary hepatic marginal zone B-cell lymphoma of mucosa-associated lymphoid tissue type in a liver transplant patient with hepatitis B cirrhosis. Transplant Proc. 2005;37:4408–12.
34. Mahjoub WK, Chaumette-Planckaert MT, Murga Penas EM, et al. Primary hepatic lymphoma of mucosa-associated lymphoid tissue type: a case report with cytogenetic study. Int J Surg Patol. 2008.
35. Page RD, Romaguera JE, Osborne B, et al. Primary hepatic lymphoma: favorable outcome after combination chemotherapy. Cancer. 2001;92:2023–9.
36. Bronowichi JP, Bineau C, Feugier P, et al. Primary lymphoma of the liver: clinical–pathological features and relationship with HCV infection in French patients. Hepatology. 2003;37:781–7.
37. Conconi A, Martinelli G, Thieblemont C, et al. Clinical activity of rituximab in extranodal marginal zone B-cell lymphoma of MALT type. Blood. 2003;102:2741–5.
38. Raderer M, Streubel B, Woehrer S, et al. High relapse rate in patients with MALT lymphoma warrants lifelong follow-up. Clin Cancer Res. 2005;11:3349–52.
39. Nasr Ben Ammar C, Chaari N, Kochbati L, et al. Primary non-Hodgkin lymphoma of the liver: case report and review of the literature. Cancer Radiother. 2006;10:595–601.
40. Raderer M, Jager G, et al. Rituximab for treatment of advanced extranodal marginal zone B cell lymphoma of the mucosa-associated lymphoid tissue lymphoma. Oncology. 2003;65:306–10.