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Case Report

Primary hepatic angiosarcoma with spleen metastases in an adult woman: a case report and literature review

Ziyue Li[1], Hui Sun[1], Chuan Shen[1], Cuili Yang[3], Yadong Wang[1], Wei Wang[1], Yanhong Jia[1], Qian Zhao[2], Caiyan Zhao[1]

Departments of [1]Infectious Disease, [2]Public Health, The Third Affiliated Hospital of Hebei Medical University, Shijiazhuang, China; [3]Department of Obstetrics, The Sixth Hospital of Shijiazhuang, Shijiazhuang, China

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Abstract: Primary hepatic angiosarcoma (PHA) is a rare malignancy that carries a poor prognosis, accounting for less than 2% of all primary hepatic tumors. It is reported to be associated with chronic exposure to environmental carcinogens, but the majority of patients were still with unknown etiology. For patients with PHA often present with nonspecific symptoms and its rapid progression, accurate and early diagnosis is difficult and necessary. We described a 41-year old woman with no history of exposure to toxic chemicals having intermittent abdominal distention for 1 month. Imaging examinations showed multiple nodules with different sizes throughout markedly enlarged liver and spleen. Liver histology showed majority of necrotic lesions with foci of atypical cells, which displayed immunoreactivity for endothelial markers CD31, CD34 and FLi-1, supporting the diagnosis of angiosarcoma. She was finally diagnosed as PHA concomitant with spleen metastases through imaging technology combined with the histopathologically results. Then the patient showed a rapidly worsening clinical course. Finally the patient received liver transplantation and splenectomy. Unfortunately, the patient died of infection in 35 days after liver transplantation.

Keywords: Hepatic angiosarcoma, FDG-PET, treatment, outcome

Introduction

Primary hepatic angiosarcoma (PHA) is a rare form of malignancy. It accounts for up to 2% of all primary liver tumors [1, 2], but it is still the third most common primary liver malignancy [1]. PHA originates from endothelial cells and usually presents with non-specific symptoms and signs, including abdominal pain, weakness, fatigue, and weight loss. The majority of the patients with PHA have metastatic lesions in the lung or spleen at the time of presentation [3]. The prognosis of patients with PHA is poor and the median survival of the patients is about 6 months without treatment. However, a few of them survived for more than 2 years with treatment [4]. Patients with PHA often present with nonspecific symptoms and its rapid progression and usually fatal outcome [5], therefore accurate and early diagnosis is critical and complete surgical resection with adjuvant chemotherapy is the key to improve prognosis [6]. There are multiple treatment options for PHA patients. Complete excision with adjuvant chemotherapy appears to be the optimal way of prolonging survival time [6, 7]. For the reasons of a high recurrence rate and poor posttransplantation survival, liver transplantation still needs further investigations. In the present report, the patient was diagnosed as PHA concomitant with spleen metastases finally through imaging technology and percutaneous needle liver biopsy. At the same time, we present these additional 21 articles published from 2006 to 2016 including 39 cases as the results of a review in the literature.

Case description

A 41-year-old woman was admitted to our hospital with a one-month history of intermittent abdominal distension. She reported a previous history of anemia and iron containing medicine had been given to treat anemia. The patient did not present a history of diabetes, heart disease, kidney disease, hepatitis and tuberculo-

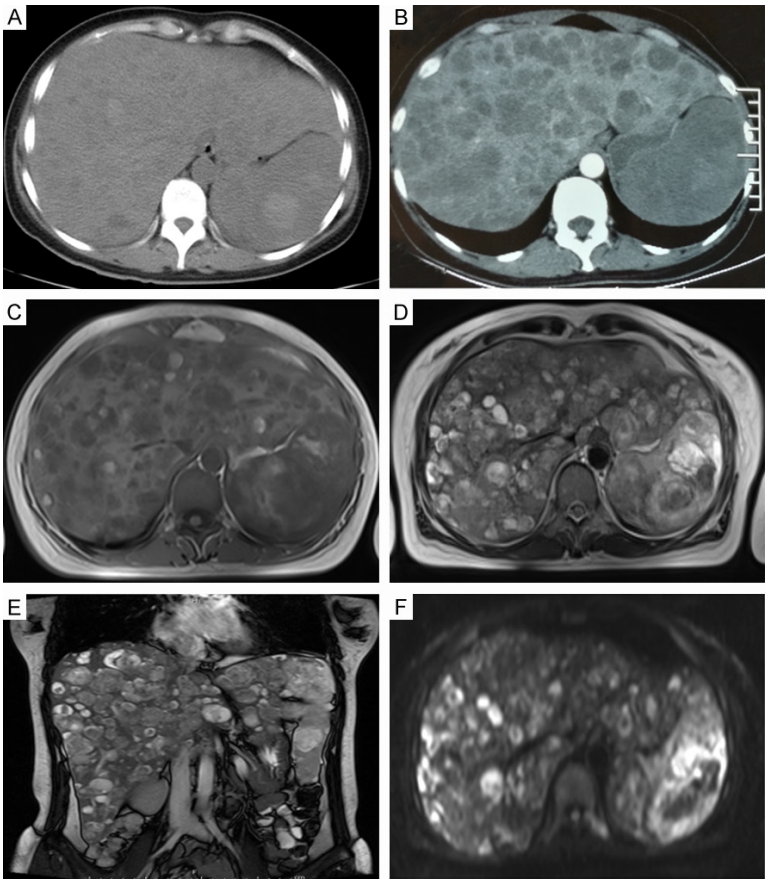

Figure 1. Abdominal plain CT scan, contrast-enhanced CT and MRI images. Abdominal plain CT scan and contrast-enhanced CT showed hepatomegaly and splenomegaly. They showed multiple kind of circular low-density shadows distributed in the liver and spleen in our case. The contrast-enhanced CT showed multiple liver nodules with slight reinforcement (A and B). MRI revealed multiple space-occupying lesions in the liver and the spleen. On MR images, there were multiple sizes long T1 and T2 signal that scattering within the liver parenchyma, and the lesions signal is uneven. Multiple nodular shadows were visible within enlarged spleen and the signal was similar to liver nodules (C-E). The lesions were characterized by a high intensity signal on DWI (F).

sis disease. In addition, she didn't have a habitual drinker and any significant history of exposure to carcinogenic chemicals such as vinyl chloride monomer, thorium dioxide (thorotrast), or arsenic. She had never used any hepatotoxic or herbal medications. There was no significant family history of biliary or liver diseases. On physical examination, the patient had anemic appearance. The abdomen was soft. No tenderness and rebound tenderness or palpable mass in the abdomen were detected. She exhibited hepatomegaly and splenomegaly. The liver and spleen edges descended 5 cm and 3 cm below the costal margin, which were no painful to touch. She presented with abdominal discomfort, but she had no other symptoms such as fatigue, weight loss and followed by ascites, icterus. The results of blood count were: white blood cell count, 9.981×10^9 cells/L (normal range, $3.5\text{-}9.5\times10^9$ cells/L), red blood cell count, 2.331×10^{12} cells/L (normal range, $4.3\text{-}5.8\times10^{12}$ cells/L) and platelet count, 178×10^9 cells/L (normal range, $125\text{-}350\times10^9$ cells/L). Hematological tests revealed that she was anemic (64.9 g/L). The erythrocyte sedimentation rate was 33 mm/h (normal range, 0-20 mm/h) and D-dimer was 14.5 mg/L (normal range, 0-0.3 mg/L). Laboratory results also demonstrated alterations in liver function: serum albumin was 30 g/L (normal range, 40-55 g/L), prothrombin time 13 seconds (normal range, 9.0-12.5 seconds), and prothrombin time activity (PTA) 69% (normal range, 80-160%). The serum levels of tumor markers, e.g. alpha-fetoprotein, carcinoembryonic antigen and carbohydrate antigen 19-9 were within normal values. Autoantibodies and viral hepatitis test results were negative. The result of bone marrow puncture was that the red blood cells were proliferous with anemia. Abdominal ultrasound examinations showed diffuse enlargement and solid or solid-cystic hepatic lesions with heterogeneous echo and irregular border in the liver and the spleen. No lung metastasis was found by Chest computed tomography (CT) scan. Abdominal plain CT scan and contrast-enhanced CT images showed hepatomegaly and splenomegaly. They showed multiple kind of circular low-density shadows distributed in the liver and spleen in our case. The contrast-enhanced CT showed multiple liver nodules with slight reinforcement (**Figure 1A** and **1B**). Next, the patient underwent an abdominal magnetic resonance imaging (MRI) (**Figure 1C-E**). MRI revealed mul-

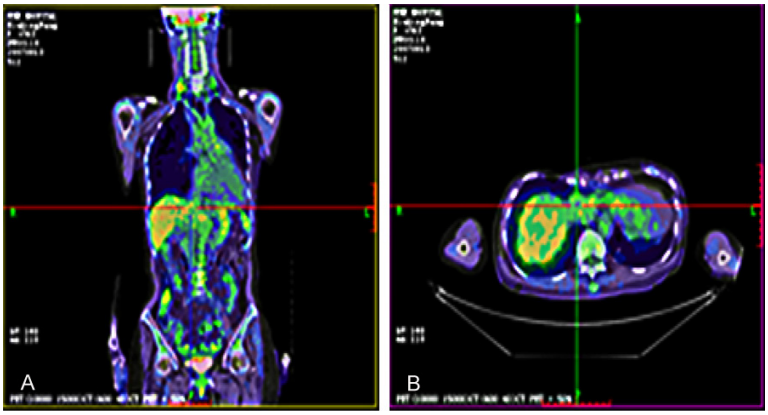

Figure 2. ^{18}F-FDP PET/CT images. ^{18}F-FDP PET/CT showed multiple hypoattenuating nodules with different sizes throughout markedly enlarged liver and spleen (A and B).

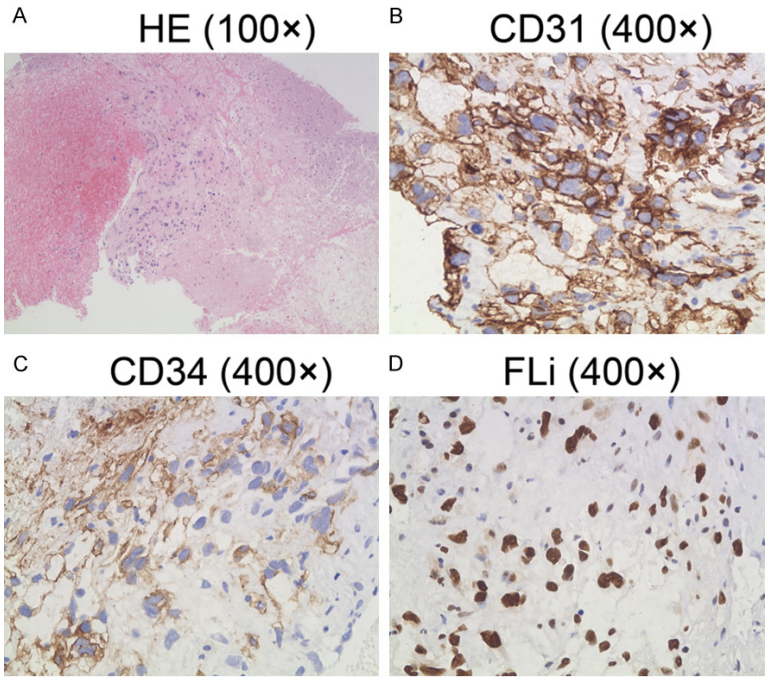

Figure 3. Liver histopathologic staining.Hematoxylin and eosin (HE) staining showed majority of necrotic lesions with foci of atypical cells (A). The tumor cells exhibited strong immunoreactivity for the spe cific vascular endothelial markers CD31, CD34 and FLi-1 (B-D). Original magnification: ×400.

tiple space-occupying lesions in the liver and the spleen. On MR images, there were multiple sizes long T1 and T2 signal that scattering within the liver parenchyma, and the lesions signal is uneven. Multiple nodular shadows were visible within enlarged spleen and the signal was similar to liver nodules. The lesions were characterized by a high intensity signal on diffusion weighted imaging (DWI) (**Figure 1F**). Whole-body fluorine-18 fluorodeoxy glucose positron emission tomography (^{18}F-FDP PET/CT) fusion scanning was performed for staging purposes to identify metastatic sites. ^{18}F-FDP PET/CT showed multiple hypoattenuating nodules with different sizes throughout markedly enlarged liver and spleen, which exhibited increased metabolism with the maximum standard up-take values (SUV) were 4.3 and 8.7, respectively (**Figure 2A** and **2B**). In order to further diagnosis, the patient was performed percutaneous biopsy ultrasonographic guidance. Histopathological examination (HE) showed majority of necrotic lesions with foci of atypical cells (**Figure 3A**). On immunohistochemistry, the malignant endothelial cells showed strong CD31, CD34 and FLi-1 immunostaining consistent with hepatic angiosarcoma (**Figure 3B-D**). On the basis of these histologic findings, the diagnosis of PHA was confirmed.

After the liver biopsy was performed, the patient's clinical course worsened. What's more, the patient was diagnosed at advanced stage, so it was not suitable for surgical resection. Finally the patient received liver transplantation and splenectomy. Unfortunately, the patient died of infection in 35 days after liver transplantation.

Discussion

PHA is a rare malignantmesenchymal neoplasm of endothelial cells, accounting for less than 2% of primary hepatic tumors [8]. It has a male preponderance and most commonly presents in the fifth or sixth decade of life and shows an obvious male predominance with a male to female ratio of 3:1 [9]. Hepatic angio-

Table 1. Case reports of hepatic angiosarcoma from 2006 to 2016

References	Report Year	n	Age	Sex	Etiology	Clinical manifestations	Extrahepatic metastasis	Pathological Diagnosis	Treatment	Prognosis
[22]	2006	1	76	M	None	Abdominal pain;	None	CD31, CD34, MIB-1	Surgery and chemotherapy	Several days
[23]	2007	1	65	F	Primary biliary cirrhosis	None	None	None	Systemic chemotherapy using paclitaxel	More than years
[24]	2007	1	62	M	None	No systems	None	Fc(+)	Liver resection	More than 16 months
[25]	2008	1	70	M	None	Jaundice and weight loss	NA	CD31, CD34, Vimentin	Conservative therapy	Several days
[26]	2009	2	65, 73	M, M	None	Jaundice, abdominal distension, and malaise, abnormally high bleeding	None	CD31, CD34,	Conservative therapy	36 hours, <7 days
[27]	2011	9	56-83	6 M, 3 F	1 infected with HCV and exposure to PVC, 1 infected with HCV	Abdominal pain, right subcostal. Pain, general. Weakness, general. Edema, epigastric. Mass, liver tumor	3 (brain, lung, colon, respectively)	CD31, CD34, D2-40, c-kit, Factor VIII, FLI-1, vimentin,	4 Conservative therapy 3 Operation + chemotherapy 1 TAE+TACE 1 TAE+operation	4 months (Median)
[28]	2011	7	33-74	5 M, 2 F	3 exposure to Vinyl chloride, 1 steroid hormone	Abdominal pain, Abdominal distension, fever, Tumor rupture, Weight loss,	None	CD31, CD34, Vimentin	3 Operation±target drugs, 2 liver transplantation, 2 Conservative therapy	MS: 6.5 months
[29]	2011	1	60	F	None	Lower-limb edema	Spleen, lungs, bones	CD31	Recombinant interleukin-2	2 months
[30]	2011	1	7	Childhood (boy)	None	Abdominal pain, loss of appetite	None	CD31, Ki-67	Liver resection+ target drugs	More than 3 months
[31]	2012	1	76	M	None	Swelling of inferior limbs, asthenia, adinamia and hiporexia as well as jaundice, coluria, nausea and disorientation. Weakness,	None	CD31	Complaint management	No more than one month
[32]	2013	2	31, 20	M, M	1 exposure to vinyl chloride	1 asthenia, anorexia, progressive jaundice, and abdominal distention; som-nolence, disorientation, and disturbances of the sleep-wake	None; NA	CD31, CD34, factor VIII, and CD10; CD31 and Factor VIII; Ki67	Liver transplantation and chemotherapy; palliative therapy	2 months; 17 days
[33]	2013	1	64	M	Exposure to carcinogens	Melena	Gastrointestinal tract	CD31, CD34	Liver resection	7 months
[34]	2014	2	38, 43	M, M	1 hepatitis B viral infection	Hepatosplenomegaly, ascites, and jaundice. abdominal pain and jaundice	None	None	Liver resection, Liver transplantation	Lost to follow-up, 18 months
[35]	2014	1	26	M	With HBV infection	Short breath, fever	Bilateral lungs, right atrium and spine	CD31 and CD34	Without treatment	1 week
[36]	2014	1	71	M	None	Feeling of lump and heaviness in the right hypochondrium.	None	CD34	Without treatment	6 months
[37]	2015	1	81	F	None	Pruritus and jaundice, anemia, thrombocytopenia, and jaundice	None	Factor VIII, FLI-1, and CD34	Complaint management	Within 24 hours
[9]	2015	1	75	M	None	Anorexia, abdominal fullness, chills, and jaundice	None	None	Without treatment	One month
[38]	2015	2	58, 58	M, F	Possible occupational exposure to vinyl chloride	Abdominal pain; fever; abdominal pain; fatigue and weight loss.	None; None	CD31, Ki-67; CD31 and CD34	Liver resection+TACE; hemihepatectomy	8 months; 16 months
[39]	2015	1	41	M	None	Abdominal pain	Spleen, suprarenal glands and pancreas	CD31, CD34	Liver transplant of the right lobe	5 months
[40]	2016	1	72	F	NA	Right upper quadrant pain, nausea, and dizziness	None	CD31, CD34, and ERG,	Liver resection	NA
[8]	2016	1	78	M	None	Hematemesis, melena	None	CD31, CD34	Palliative care	45 days

M, man; F, femal; NA, not available; TACE, transarterial chemoembolization; TAE, transarterial embolization.

sarcoma is found throughout the world. Majority of patients have symptoms such as abdominal pain, weakness, fatigue, and weight loss. On physical examination, they also present hepatosplenomegaly, ascites, jaundice and anemia [10]. The etiology of PHA remains unclear and it is reported that about 1/3 of all cases of PHA appear to be associated with chronic exposure to environmental carcinogens such as polyvinyl chloride, thoriumdioxide (thorothrast), arsenic and inorganic copper [11]. But in most of cases, there are no obvious risk factors as shown in **Table 1** and these angiosarcomas are considered idiopathic [12]. The clinical diagnosis of PHA is complicated and often delayed due to the majority of cases of PHA do not exhibit any relative signs or symptoms [1]. Most patients are diagnosed at an advanced stage because of the nonspecific symptoms. In our case report, the patient only presented with abdominal discomfort. In addition, it is difficult to differentiate from the diseases such as hepatocellular cancer (HCC), metastases, primary hepatic lymphoma, hepatic cystadenocarcinoma, hemangioblastoma and hepatic c (HEHE) via imaging examinations. What is worse that PHA progress rapidly and most patients are diagnosed at advanced stage. To improve the long-term outcomes of hepatic angiosarcoma, early detection and diagnosis are of great importance. Morphologically, patients with PHA often appear as multiple nodules, dominant masses, or a diffusely infiltrating lesion, and its appearance may vary slightly in CT and MRI [3]. The most common site of metastases is the lung and spleen. Pathological analysis diagnosis is very necessary. Epithelioid hemangioendothelioma (EHE), a rare and usually low-grade malignant tumor, should be considered in the differential diagnosis of PHA [6]. CD31, CD34, D2-40 and factor VIII -related antigen are often used in combination for the immunohistochemical diagnosis of angiosarcomas. In our case report, immunohistochemical analysis demonstrated the tumor to be CD31+, CD34+, FLi-1+.

Primary hepatic angiosarcomas progress very rapidly. Most patients are diagnosed at late stage of malignant tumor. The median survival time was about 6 months. The majority of patients die within a year after diagnosis and only 3% survive more than 2 years [1, 13]. In our literature review, the patients survived no more than 6 months without treatment.

Extrahepatic metastatic disease may be seen in approximately 60% of patients at presentation. The most common site of metastases is the lung, followed by the spleen [9]. In our literature review, PHA was documented to metastasize to the lungs, spleen, bone, gastrointestinal tract, colon, brain.

The treatment of PHA has not been standardized due to the rarity of the disease. To date, the treatment of the malignant tumor is empirical. There are several choices of treatment for PHA patients. Complete surgical resection with adjuvant chemotherapy appears to be the most promising treatment with the possibility of cure if the tumor is solitary and is diagnosed early [6, 7]. As shown in the **Table 1**, patients receiving surgical resection had longer survival time compared with those who did not have their tumor resected in general. Duan et al. concluded that complete surgical resection is the key to improve prognosis through a retrospective study of 6 patients diagnosed with primary hepatic angiosarcoma [14]. Arima-Iwasa et al. concluded that a long-term survival is possible after complete tumor resection in a preselected population with early-stage disease by a retrospective and prospective cohort study [15]. Liver transplantation has showed survival benefit in the management of other primary liver cancers such as HCC and HEHE. However, it has been abandoned because of a high recurrence rate and poor posttransplantation survival in HAS [16, 17] when compared with surgical resection in reported studies [14-16, 18]. In our case report, the patient survived for only 35 days after liver transplantation. Hepatic angiosarcoma is a malignant vascular neoplasia. Liver transplantation remains controversial because of its poor prognosis in the short term [19].

Transarterial chemoembolization (TACE) has been used as palliative therapy to for deadly intra-abdominal bleeding in patients with hepatic angiosarcoma. TACE and chemotherapy may be helpful in improving survival. There has been no established chemotherapy scheme, or few clinical trials of chemotherapy in PHA. There are some clinical studies of angiosarcomas recently, which show single-agent doxorubicin, and weekly paclitaxel-, gemcitabine-, and doxorubicin based schemes deserve consideration [20, 21], while bevacizumab,

sorafenib, and pazopanib are potential target drugs.

Conclusion

PHA is a rare malignant that carries a poor prognosis. Currently, the best treatment option for PHA is surgical resection of the liver to remove the tumor. Liver transplantation remains controversial because of its poor prognosis in the short term. Research on early diagnostic strategies and newer therapies are needed to improve prognosis. However,the rarity of this disease inhibits progress.

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Disclosure of conflict of interest

None.

Address correspondence to: Dr. Caiyan Zhao, Department of Infectious Disease, The Third Affiliated Hospital of Hebei Medical University, 139 Ziqiang Road, Shijiazhuang 050051, China. Tel: +86-311-6677-6874; Fax: +86-311-6677-6875; E-mail: zhaocy2005@163.com

References

[1] Locker GY, Doroshow JH, Zwelling LA and Chabner BA. The clinical features of hepatic angiosarcoma: a report of four cases and a review of the english literature. Medicine (Baltimore) 1979; 58: 48-64.

[2] Kang TW, Lee MW, Choi D, An C, Kim MJ, Joo I, Lee SJ, Lim S, Park JG, Seo JW and Jung SH. Safety of percutaneous biopsy for hepatic angiosarcoma: results of a multicenter Korean survey. J Vasc Interv Radiol 2016; 27: 846-851.

[3] Park YS, Kim JH, Kim KW, Lee IS, Yoon HK, Ko GY and Sung KB. Primary hepatic angiosarcoma: imaging findings and palliative treatment with transcatheter arterial chemoembolization or embolization. Clin Radiol 2009; 64: 779-785.

[4] Falk H, Herbert JT, Edmonds L, Heath CW Jr, Thomas LB and Popper H. Review of four cases of childhood hepatic angiosarcoma--elevated environmental arsenic exposure in one case. Cancer 1981; 47: 382-391.

[5] Molina E and Hernandez A. Clinical manifestations of primary hepatic angiosarcoma. Dig Dis Sci 2003; 48: 677-682.

[6] Zheng YW, Zhang XW, Zhang JL, Hui ZZ, Du WJ, Li RM and Ren XB. Primary hepatic angiosarcoma and potential treatment options. J Gastroenterol Hepatol 2014; 29: 906-911.

[7] Weitz J, Klimstra DS, Cymes K, Jarnagin WR, D'Angelica M, La Quaglia MP, Fong Y, Brennan MF, Blumgart LH and Dematteo RP. Management of primary liver sarcomas. Cancer 2007; 109: 1391-1396.

[8] Ito Z, Kajihara M, Kobayashi Y, Kanai T, Matsumoto Y, Takakura K, Yukawa T, Ohkusa T, Koyama S, Imazu H, Arakawa H, Ohata M and Koido S. Hepatic angiosarcoma associated with esophageal variceal hemorrhage. Case Rep Gastroenterol 2016; 10: 440-445.

[9] Abegunde AT, Aisien E, Mba B, Chennuri R and Sekosan M. Fulminant hepatic failure secondary to primary hepatic angiosarcoma. Case Rep Gastrointest Med 2015; 2015: 869746.

[10] Chen G, Li J, Wan R, Wang G and Shi J. Primary hepatic angiosarcoma in a 64-year-old man: a case report. Oncol Lett 2016; 11: 2445-2448.

[11] Thomas LB and Popper H. Pathology of angiosarcoma of the liver among vinyl chloride-polyvinyl chloride workers. Ann N Y Acad Sci 1975; 246: 268-277.

[12] Cano-Garcia F, Athie-Athie Ade J, Garcia-Gomez JI, Chable-Montero F and Albores-Saavedra J. Cystic angiosarcoma of the liver. A previously undescribed neoplasm. Ann Hepatol 2016; 15: 283-286.

[13] Fayette J, Martin E, Piperno-Neumann S, Le Cesne A, Robert C, Bonvalot S, Ranchere D, Pouillart P, Coindre JM and Blay JY. Angiosarcomas, a heterogeneous group of sarcomas with specific behavior depending on primary site: a retrospective study of 161 cases. Ann Oncol 2007; 18: 2030-2036.

[14] Duan XF and Li Q. Primary hepatic angiosarcoma: a retrospective analysis of 6 cases. J Dig Dis 2012; 13: 381-385.

[15] Matthaei H, Krieg A, Schmelzle M, Boelke E, Poremba C, Rogiers X, Knoefel WT and Peiper M. Long-term survival after surgery for primary hepatic sarcoma in adults. Arch Surg 2009; 144: 339-344; discussion 344.

[16] Orlando G, Adam R, Mirza D, Soderdahl G, Porte RJ, Paul A, Burroughs AK, Seiler CA, Colledan M, Graziadei I, Garcia Valdecasas JC, Pruvot FR, Karam V and Lerut J. Hepatic hemangiosarcoma: an absolute contraindication to liver transplantation--the European liver tra-

nsplant registry experience. Transplantation 2013; 95: 872-877.
[17] Yoshida Y, Yoshizumi T, Wang H, Sakata K, Shimokawa M, Kurihara T, Motomura T, Itoh S, Harada N, Harimoto N, Ikegami T, Uchiyama H, Soejima Y and Maehara Y. Liver transplantation for cryptogenic liver failure caused by diffuse hepatic angiosarcoma: case report. Surg Case Rep 2017; 3: 21.
[18] Timaran CH, Grandas OH and Bell JL. Hepatic angiosarcoma: long-term survival after complete surgical removal. Am Surg 2000; 66: 1153-1157.
[19] Tran Minh M, Mazzola A, Perdigao F, Charlotte F, Rousseau G and Conti F. Primary hepatic angiosarcoma and liver transplantation: radiological, surgical, histological findings and clinical outcome. Clin Res Hepatol Gastroenterol 2017.
[20] Italiano A, Cioffi A, Penel N, Levra MG, Delcambre C, Kalbacher E, Chevreau C, Bertucci F, Isambert N, Blay JY, Bui B, Antonescu C, D'Adamo DR, Maki RG and Keohan ML. Comparison of doxorubicin and weekly paclitaxel efficacy in metastatic angiosarcomas. Cancer 2012; 118: 3330-3336.
[21] Stacchiotti S, Palassini E, Sanfilippo R, Vincenzi B, Arena MG, Bochicchio AM, De Rosa P, Nuzzo A, Turano S, Morosi C, Dei Tos AP, Pilotti S and Casali PG. Gemcitabine in advanced angiosarcoma: a retrospective case series analysis from the Italian rare cancer network. Ann Oncol 2012; 23: 501-508.
[22] Leowardi C, Hormann Y, Hinz U, Wente MN, Hallscheidt P, Flechtenmacher C, Buchler MW, Friess H and Schwarzbach MH. Ruptured angiosarcoma of the liver treated by emergency catheter-directed embolization. World J Gastroenterol 2006; 12: 804-808.
[23] Maeda T, Tateishi U, Hasegawa T, Ojima H, Arai Y and Sugimura K. Primary hepatic angiosarcoma on coregistered FDG PET and CT images. AJR Am J Roentgenol 2007; 188: 1615-1617.
[24] Arima-Iwasa S, Chijiiwa K, Makino I, Tanabe R, Ohuchida J and Kondo K. A case of hepatic angiosarcoma surviving for more than 16 months after hepatic resection. Hepatogastroenterology 2007; 54: 533-535.
[25] Bhati CS, Bhatt AN, Starkey G, Hubscher SG and Bramhall SR. Acute liver failure due to primary angiosarcoma: a case report and review of literature. World J Surg Oncol 2008; 6: 104.
[26] Egea Valenzuela J, Lopez Poveda MJ, Perez Fuenzalida FJ, Garre Sanchez C, Martinez Barba E and Carballo Alvarez F. Hepatic angiosarcoma. Presentation of two cases. Rev Esp Enferm Dig 2009; 101: 430-434, 434-437.
[27] Huang NC, Wann SR, Chang HT, Lin SL, Wang JS and Guo HR. Arsenic, vinyl chloride, viral hepatitis, and hepatic angiosarcoma: a hospital-based study and review of literature in Taiwan. BMC Gastroenterol 2011; 11: 142.
[28] Chi TY, Yang ZY, Xue HD, Lü K, Feng RE, Xu HF, Yang XB, Sang XT, Lu X, Mao YL, Zhong SX, Huang JF. Diagnosis and treatment of primary hepatic angiosarcoma: a report of 7 cases with a literature review. Zhonghua Yi Xue Za Zhi 2011; 91: 1694-1697.
[29] Mitsui F, Aikata H, Hashimoto Y, Nagaoki Y, Kimura Y, Katamura Y, Kawaoka T, Takaki S, Hiraga N, Tsuge M, Waki K, Hiramatsu A, Imamura M, Kawakami Y, Takahashi S, Arihiro K and Chayama K. A first case of hepatic angiosarcoma treated with recombinant interleukin-2. Hiroshima J Med Sci 2011; 60: 91-96.
[30] Geramizadeh B, Safari A, Bahador A, Nikeghbalian S, Salahi H, Kazemi K, Dehghani SM and Malek-Hosseini SA. Hepatic angiosarcoma of childhood: a case report and review of literature. J Pediatr Surg 2011; 46: e9-11.
[31] Montell Garcia M, Romero Cabello R, Romero Feregrino R, Atri Moises M, Trejo Estrada R, Alvaro PR, Moreno Manlio Gerardo G and Feregrino Rodrigo R. Angiosarcoma of the liver as a cause of fulminant liver failure. BMJ Case Rep 2012; 2012.
[32] López R, Castro-Villabón D, Álvarez J, Vera A and Andrade R. Hepatic angiosarcoma presenting as acute liver failure in young adults. Report of two cases and review of literature. Case Reports in Clinical Medicine 2013; 2: 439-444.
[33] Chou JW, Cheng KS and Chen SF. Primary hepatic angiosarcoma with multifocal metastases in the gastrointestinal tract. Endoscopy 2013; 45 Suppl 2 UCTN: E319-320.
[34] Terzi A, Deniz EE, Haberal N, Moray G and Ozdemir BH. Hepatic angiosarcoma and liver transplant: a report of 2 cases with diagnostic difficulties. Exp Clin Transplant 2014; 12 Suppl 1: 126-128.
[35] Huang H, Le XH, Zhou BP and Lu PX. Hepatic angiosarcoma with multiple metastases in a young man. Quant Imaging Med Surg 2014; 4: 291-293.
[36] Thapar S, Rastogi A, Ahuja A and Sarin S. Angiosarcoma of the liver: imaging of a rare salient entity. J Radiol Case Rep 2014; 8: 24-32.
[37] Mazharuddin S, Podduturi V, Guileyardo JM and Cooper B. Hepatic angiosarcoma associated with disseminated intravascular coagulation. Proc (Bayl Univ Med Cent) 2015; 28: 54-56.
[38] Zhu YP, Chen YM, Matro E, Chen RB, Jiang ZN, Mou YP, Hu HJ, Huang CJ and Wang GY. Primary hepatic angiosarcoma: a report of two cases and literature review. World J Gastroenterol 2015; 21: 6088-6096.

[39] Huerta-Orozco LD, Leonher-Ruezga KL, Ramirez-Gonzalez LR, Hermosillo-Sandoval JM, Sandoval-Alvarado Jde J and Moran-Galaviz RE. [Hepatic angiosarcoma and liver transplantation: case report and literature review]. Cir Cir 2015; 83: 510-515.

[40] Ling W, Qiu T, Ma L, Lei C and Luo Y. Contrast-enhanced ultrasound in diagnosis of primary hepatic angiosarcoma. J Med Ultrason (2001) 2017; 44: 267-270.