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A Case of a Spontaneous Disappeared Giant Liver Hemangioma and its Literature Review

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Abstract

Hepatic hemangioma is the most common benign tumor of the liver. It is reported that the change of hemangioma may be related to VEGF. In our case, a 62-year-old male patient, who had a 20 years history of HBV, was diagnosed as giant liver hemangioma (102 mm × 73 mm) 17 years ago and did not receive any review and treatment before admission. After admission, CT showed liver cirrhosis and hepatocellular carcinoma. Compared with the CT of 17 years ago, the giant hemangioma disappeared completely. To date, the giant liver hemangioma, which had spontaneously disappeared without treatment, has not been reported.

Keywords: Giant liver hemangioma; Spontaneous disappear; Hepatocellular carcinoma; Vascular Endothelial Growth Factor (VEGF)

Introduction

Hepatic hemangioma is the most common benign tumor of the liver, accounting for 73 percent of all benign tumors of the liver [1]. The etiology and mechanism of liver hemangioma is not clear. It may be caused by the vascular malformation (anomaly peripheral vascular expansion) which related with congenital vascular dysplasia, estrogen levels, Vascular Endothelial Growth Factor (VEGF) dysregulation, even genetic factors [2]. In addition, hepatic hemangioma, which usually supplied by hepatic artery [3], usually reduce in size only when the VEGF level changes [4] or after selective hepatic artery embolization [5]. To date, the giant liver hemangioma, which had spontaneously disappeared without treatment, has not been reported.

Case Presentation

A 62-year-old male patient, who complained about his abdominal pain and low fever for 1 month, was admitted to our hospital in June 2017. CT showed a 51 mm × 43 mm lesion in S5 (Figure 1). Laboratory examination showed HBSAg (+),

HBeAg (+), HBcAb (+) and AFP 445.50 ng/ml. He was diagnosed with hepatitis B, liver cirrhosis and hepatocellular carcinoma accompanied by portal venous tumor thrombus. The patient suffered from HBV 20 years ago, and the ultrasound showed hepatic hemangioma (30 mm × 25 mm) in the right liver lobe. About 17 years ago, ultrasound showed giant occupying lesion in the right liver lobe. CT showed giant lesion (102 mm × 73 mm) (Figure 2) in the right liver lobe, which was diagnosed as giant liver hemangioma. Laboratory examination showed HBSAg (+), AFP (-). Because of a family history of liver cancer and a significant increase in the lesion within 3 years, biopsy was performed, and the pathological findings were hemangioma of the liver (Figure 3). The patient did not receive any review and treatment before this admission. After admission, we compared with the enhanced CT images of 17 years ago, the giant hemangioma disappeared completely (Figure 4).

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Figure 1 Enhanced CT image of patients with liver cancer. (**a** and **b**) The mass was located in S5 (51 mm × 43 mm), and it enhanced obviously in the arterial phase. In the portal venous phase, the enhancement degree was relatively low, and the portal vein tumor thrombus was found.

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Figure 2 The Enhanced CT image of patients with giant hemangioma. **(a-d)** The mass (102 mm × 73 mm) was located in the right liver lobe. In the arterial phase, the tumor edge demonstrated scattered and patchy enhancements. Then the scattered enhancements merged and expanded gradually to the center of the tumor in portal venous phase. In delayed phase, the tumor revealed further centripetal filling.



Figure 3 Pathological section of hepatic hemangioma. (a and b) HE Stain, 400 times. Immunohistochemical results: CD34 (+), CD31 (+), CR (-), CK19 (-) and P53 (-).



Figure 4 (a and b) The giant hemangioma disappeared completely.

Discussion

Hepatic hemangioma is usually found in abdominal ultrasound examination [6]. Although the mechanism is still unknown, the size of hepatic hemangioma in adults seldom change [7,8]. So far, there is no literature and data showing that hepatic hemangioma has a malignant tendency [9].

High VEGF expression, which plays an important role in tumor progress, leads to increased angiogenic formation in hemangioma endothelial cells [10]. According to such theory, some literatures reported that anti-VEGF treatment has been shown to have a direct and rapid effect in tumors. Mahajan [4] reported a male patient with a colon tumor accompanied by intrahepatic hemangioma, whose diameter of the hepatic hemangioma reduced from 3.6 cm × 2.8 cm to 2.5 cm × 2.5 cm after antiangiogenic therapy. The author considered that antiangiogenesis effect of chemotherapy drugs played a role on liver hemangioma reduction. Ono [11] reported that a patient with a 9-year history of hepatic hemangioma (2.0 cm × 2.5 cm) was treated with the anti-diabetic drug metformin, resulting in complete disappear of the tumor. In his report, the changes of hemangioma were significantly correlated with the dosage of metformin, and the reduction of hemangioma was indicated by the anti-angiogeneic effect of metformin. Hashimoto [12] reported a case of hepatic hemangioma that decreased in size after chemotherapy for an ovarian tumor. The analysis may be related to the obvious decrease of estrogen level after the oophorectomy or the anti-angiogenesis of the chemotherapy drug, or the combination of the two. Guillonnet [13] reported a case of a patient with intrahepatic multiple hemangiomas. The MRI T2 signal and the size of the hemangiomas revealed an obvious change after 2 years, especially the size of one giant liver hemangioma reduced from 104 mm × 94 mm to 75 mm × 72 mm with an unknown reason.

These reports have some similarities, the changes in hemangiomas occur on the basis of no hepatitis and no cirrhosis. Many studies have reported different levels of VEGF in chronic hepatitis, cirrhosis, liver cancer, and even different child-pugh grades and differentiated of liver cancer [14-16].

Conclusion

Some studies suggest that the expression of VEGF in liver cirrhosis and liver cancer is higher than that of chronic hepatitis, and the VEGF in liver cancer is lower than in surrounding cirrhotic liver tissues, and there are significant differences in the expression of VEGF in low-grade and highgrad hepatocellular carcinoma [14,17,18]. However, some studies showed that the level of VEGF in the cirrhotic group was significantly lower than that in the non-cirrhosis group [19].

Although the findings are not identical, it is at least true that VEGF is also in flux during the development of liver cancer from hepatitis, rather than always the tendency to rise or fall. Patients in this case developed from hepatitis to liver cirrhosis and liver cancer in 20 years, during this period, the process of a hepatic hemangioma from small to large and spontaneous disappearance, it may be the process of VEGF decreasing from low to high, and then it goes down but the mechanism cannot be explained in detail due to the lack of exact evidence and similar reports. And the limitation of this article is the long span from 2000 to 2017, we cannot explain the exact time of the disappearance of hemangioma.

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