

Sclerosed hemangioma of the liver

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INTRODUCTION

Cavernous hemangioma of the liver is the most common benign neoplasm, and the liver is its most common visceral site, having been found at that location in about 19% of cases in one series.¹ It is usually found incidentally during a radiologic workup. However, it can result in specific symptoms or manifest with large size or enlargement. Cavernous hemangiomas usually present as solitary well-delineated, subcapsular, discolored nodules that are smaller than 5 cm. They can exhibit various forms of pathology, such as necrosis, scarring, and calcification, some of which mimic the primary or metastatic malignancy.

We present herein a case of sclerosed hemangioma in a 52-year-old man. We discuss the histopathologic features of this case along with another case of giant cavernous hemangioma of the liver.

CASE SUMMARY

A 52-year-old male was transferred to our hospital for surgery of liver mass. He had visited an affiliated hospital of our university and had undergone surgery to repair a C-spine disc herniation 1 month previously. A liver mass was found during the preoperative workup. The initial hematologic and blood chemistry data were as follows: 5,100/ μ L white blood cell count, 12.8 g/dL hemoglobin, 175,000/ μ L platelet count, 21 mg/dL blood urea nitrogen, 0.7 mg/dL creatinine, 4.2 g/dL albumin, 33 IU/L aspartate aminotransferase, 40 IU/L alanine aminotransferase, and 0.7 mg/dL total bilirubin. He was positive for hepatitis B

(HB) surface antigen (HBsAg) and anti-HBe, and negative for anti-HBs and HBeAg. The alpha-fetoprotein level was 1148.2 ng/mL, and carcinoembryonic antigen and cancer antigen 19-9 levels were in the normal ranges.

Dynamic computed tomography (CT) disclosed a 3.8 \times 3.2 \times 3.1 cm-sized mass with a bulging contour in the lateral segment of the liver that was well enhanced in the arterial phase and showed a subtle low density with focal capsular enhancement in the delayed phase. This mass demonstrated a low signal intensity on T1-weighted image and a high signal intensity on T2-weighted image. It was enhanced and washed out with low signal intensity in the hepatobiliary phase, making it compatible with typical hepatocellular carcinoma. Another enhanced mass was noted in segment 6, with marginal globular and gradual internal enhancement on dynamic CT. Magnetic resonance image (MRI) demonstrated a low signal intensity on T1-weighted image and a slightly high signal intensity on T2-weighted image. It exhibited subtle marginal enhancement on delayed phase and well-demarcated low signal intensity on hepatobiliary phase. Based on the radiologic findings, hemangioma or well-differentiated hepatocellular carcinoma in a dysplastic nodule were suspected. Given the impression of two hepatocellular carcinomas, or else one hepatocellular carcinoma and another lesion, a left lateral segmentectomy and wedge resection of segment 6 were performed.

PATHOLOGIC FINDINGS

The resected left liver weighed 155 g and measured 12 \times 8 \times 4

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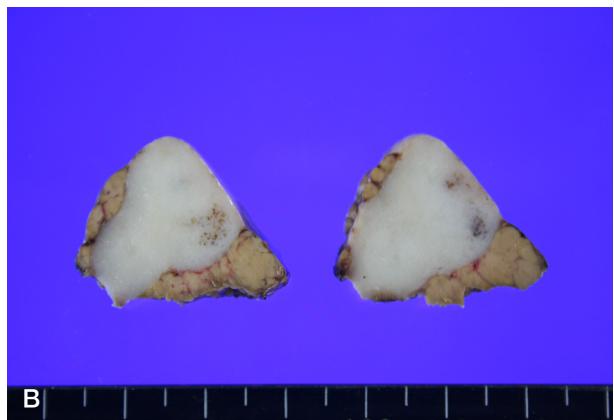
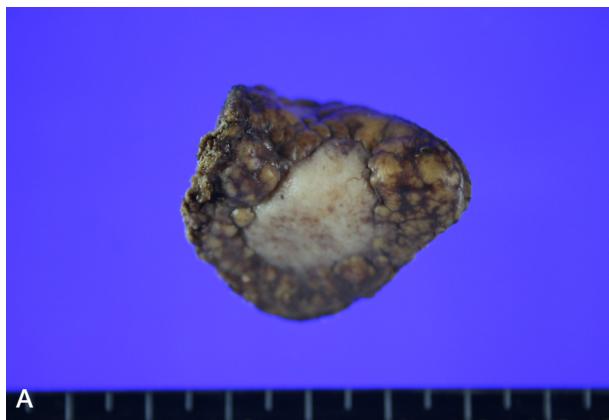


Figure 1. Gross appearance of the sclerosed hemangioma. A gray-white sunken nodule is noted in the subcapsular area (A). The cut surface discloses a well-demarcated homogenous gray-white solid nodule, measuring 2.1 cm at its greatest dimension, with tiny red spots (B).

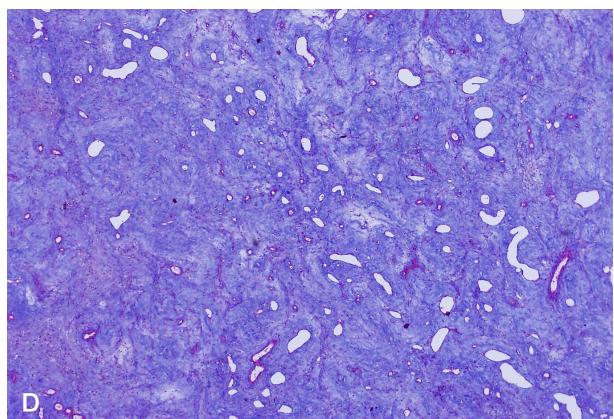
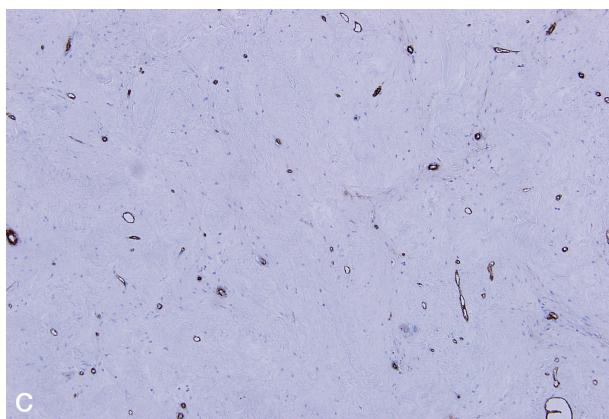
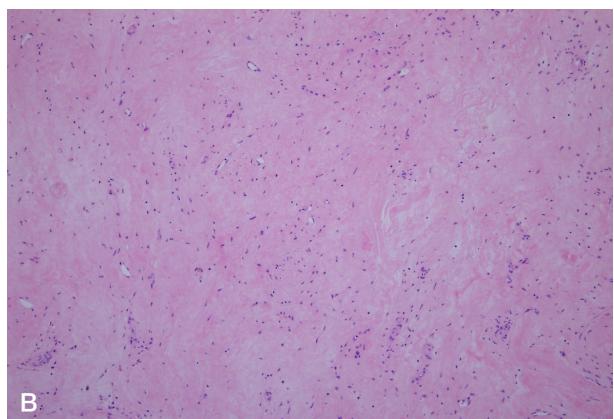
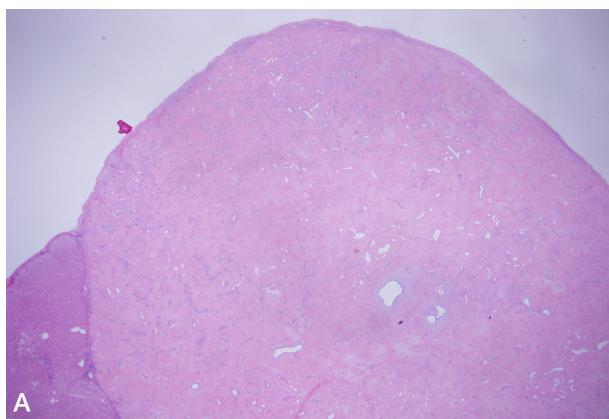


Figure 2. (A) Histologic features of the sclerosed hemangioma nodule in the right lobe. A hyalinized mass is identified from the surrounding liver. (B) This mass consists of collapsed vascular spaces on the rich paucicellular fibrous stroma. (C) The vascular spaces are easily identified on immunohistochemistry for an endothelial marker. (D) Sclerosis is highlighted with a special stain, Masson's trichrome (A: H-E, $\times 12.5$; B: H-E, $\times 200$; C: factor-VIII-related antigen, $\times 100$, D: Masson's trichrome, $\times 40$).

cm. A bulging mass lesion was present that measured 3.5×2.7 cm. On sectioning, the mass was well circumscribed from the surrounding noncirrhotic liver and its cut surface was yellowish/greenish-gray in color. Microscopic examination disclosed a

hepatocellular carcinoma of Edmondson-Steiner grade II with foci of portal vein invasion.

The wedge-resected liver from segment 6 weighed 8 g and measured $3.8 \times 2.8 \times 2.0$ cm. The external Glisson's capsule

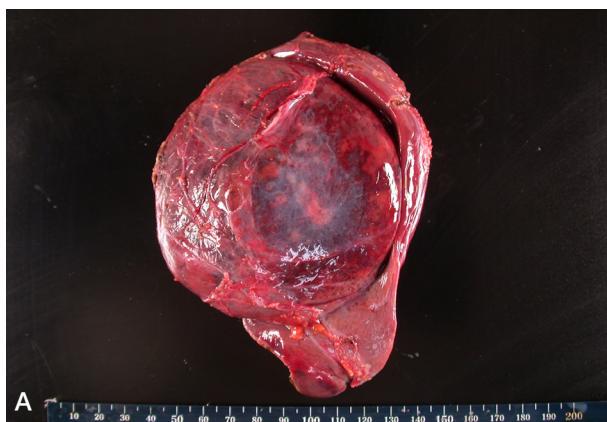


Figure 3. Macroscopic finding of the giant hemangioma. An externally bulging, huge, blue-red mass is noted (A). The cut section shows a discrete dark red, sponge-like nodule, measuring 10 cm at its greatest dimension, in the noncirrhotic liver (B).

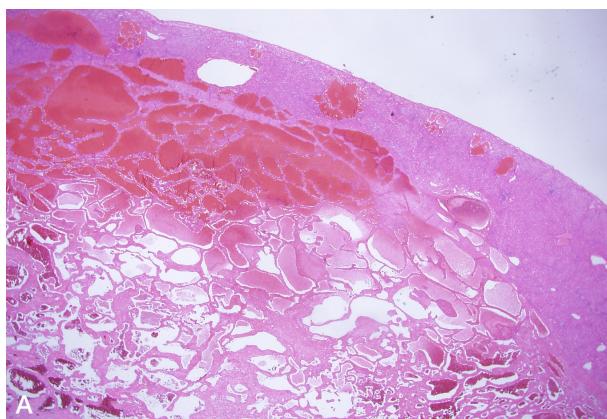
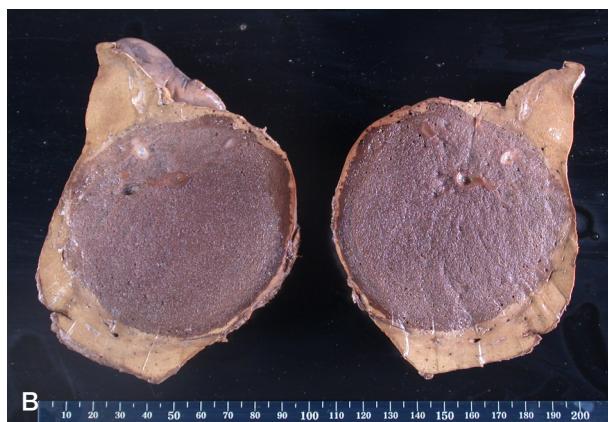
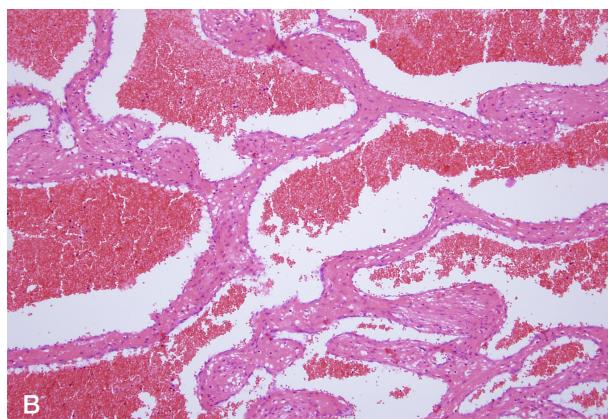


Figure 4. Microscopic finding of the giant hemangioma. (A) Low-power view disclosing proliferation of the cavernous vascular structures. (B) Each vascular space is lined by flat endothelial cells and filled with fresh red blood cells. (A: H-E, $\times 12.5$; B: H-E, $\times 100$).



was discolored and sunken by an underlying whitish, firm nodule (Fig. 1A). Sectioning revealed a relatively homogenous, well-circumscribed gray-white solid nodule with several dark-red, pin-point spots measuring 2.1×1.6 cm (Fig. 1B). Microscopic evaluation revealed that most areas comprised sclerotic hyalinized collagenous tissue with scattered tiny-to-small, thin-walled vascular spaces (Fig. 2A and 2B). The vascular spaces were frequently collapsed and lined by flat endothelial cells. These cells were positive on immunohistochemistry for an endothelial marker (Fig. 2C). There was a focal marginal zone of identifiable vascular spaces. Some vascular channels were surrounded by a loose myxoid matrix and a concentric cuff of stellate cells. A special stain highlighted the sclerotic condition of the mass (Fig. 2D), and the surrounding nonneoplastic liver showed periportal fibrosis. Areas of stromal calcification were focally present.

DISCUSSION

Cavernous hemangiomas of the liver can be found incidentally and may be readily diagnosed due to their characteristic homogeneous hyperechogenicity and posterior acoustic enhancement on ultrasound examination. They can sometimes present different stages of involution. In these cases, radiological findings show atypical features,² occasionally mimicking malignant lesions.³ The giant size of the tumor and specific symptoms including Kasabach-Merritt syndrome make the diagnosis difficult. In such cases, pathologic confirmation is needed to accurately characterize the lesion.

The giant cavernous hemangioma shown in Figure 3 and 4 was resected from a 36-year-old woman due to progressive enlargement and multiplicity. Macroscopically, cavernous hemangioma presents as a subcapsular blue-red flat mass (Fig. 3A), with cut sections showing a homogeneous spongiform surface

(Fig. 3B). Areas of gray-white scarring can be present. Microscopically, cavernous hemangiomas comprise cavernous vascular spaces lined by flattened endothelium with fibrous septa (Fig. 4). Thrombosis and phleboliths are evident, and variable proportions of scarring and calcification complicate the histopathologic features.

The hemangioma is basically a type of hamartoma. Female sex hormones may play some role in its development and progression. Makhlof and Ishak⁴ compared the findings between sclerosed hemangioma and sclerosing cavernous hemangioma. According to their theory, recent hemorrhages and hemosiderin deposits, rich with mast cells are present in the sclerosing hemangioma. Moreover, fibrosis, increased elastic fibers, and dystrophic or psammomatous calcifications with a decreased number of mast cells can be observed in the sclerosed hemangioma. Based on these findings, they suggest the involvement of the mast cells in angiogenesis, regression, and fibrosis.

The principal management of cavernous hemangioma of the liver is conservative. It will be helpful to understand the natural involution stages of this lesion. However, pathologic confirmation of the mass is mandatory if the possibility of malignancy cannot be ruled out.

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