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Case report

Histologically apparent hepatic hemangioma mimicking fibrolamellar hepatocellular carcinoma: Rare case report

Bikash Parida*, Sherry Garg, Somadatta Das

Department of Radiodiagnosis, Institute of Medical Sciences and SUM Hospital, Siksha 'O' Anusandhan Deemed to be University, Bhubaneswar, India

Hepatic hemangioma is a frequently occurring benign liver lesion detected by imaging. Making a definite diagnosis is difficult because its radiological characteristics can mimic those of hepatic malignancies such as metastatic liver cancer. A 65-year-old female patient complained of a month-long reduction in appetite and abdominal discomfort/distension in the right hypochondriac area. A B-mode ultrasound was suggested for the patient. Ultrasonography revealed a large, poorly defined heterogeneous lesion that occupied the right lobe of the liver entirely. The lesion featured a hyperechoic periphery, an irregular hypoechoic patch in the center, necrosis, and several central coarse calcifications. Vigorous imaging workup was performed using cross-sectional imaging modalities such as computed tomography, magnetic resonance imaging, and positron emission tomography to distinguish hemangioma from other liver lesions.

Keywords: Benign liver lesion, fibrolamellar hepatocellular carcinoma, giant hepatic hemangioma.

Hepatic hemangioma is the most common benign vascular lesion of the liver. A hemangioma larger than 5 cm is called giant hemangioma.[1] This lesion is more common in females than in males (5 : 1). Its prevalence ranges from 1.0% to 20.0%.[2] Most hemangiomas present a characteristic hemodynamic pattern on contrast-enhanced magnetic resonance imaging (MRI) and computed tomography (CT), hence labeled as typical. By contrast, atypical hemangiomas present in different ways, posing difficulty in distinguishing these benign lesions from hepatocellular carcinoma (HCC), intrahepatic cholangiocarcinoma, and fibrolamellar hepatocellular carcinoma (FL HCC).[3] Therefore, differentiating atypical hemangiomas from their mimickers is important to guide the treatment strategy. Here, we describe an atypical hemangioma mimicking FL HCC.[4]

***Correspondence to: Bikash Parida,** Department of Radiology, Institute of Medical Sciences and SUM Hospital, Siksha 'O' Anusandhan Deemed to be University, Bhubaneswar, Indian.
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Case report

A 65-year-old female presented with abdominal distention and pain at the right hypochondriac region with decreased appetite for a month. The patient was advised to undergo a B-mode ultrasound. Ultrasonography (USG) revealed a very large ill-defined heterogeneous lesion with hyperechoic periphery and central irregular hypoechoic area. Multiple coarse calcifications appeared in the central part, completely occupying the right lobe of the liver **(Figure 1).** The lesion showed vascularity on Doppler. Another similar morphology lesion was seen in the left lobe segment II. The residual liver parenchyma in the left lobe had a normal echo texture with a regular surface. The portal vein was compressed with normal flow on Doppler. The spleen had normal size and echo texture. No evidence of ascites was present. Therefore, the differential diagnosis of atypical giant hemangioma or FL HCC was made.

A triple phase contrast-enhanced CT of abdomen pelvis and other biochemical tests were advised. For the CT, 100 ml of nonionic contrast was administered intravenously. The large lesion measuring approximately 20.5 × 20.1 × 16.8 cm (ap × ml × cc) completely occupied the right lobe (segment V, VI, VII, and VIII) segment IVa, with peripheral nodular puddling of contrast on the arterial phase and gradual centripetal filling in the portal venous phase.

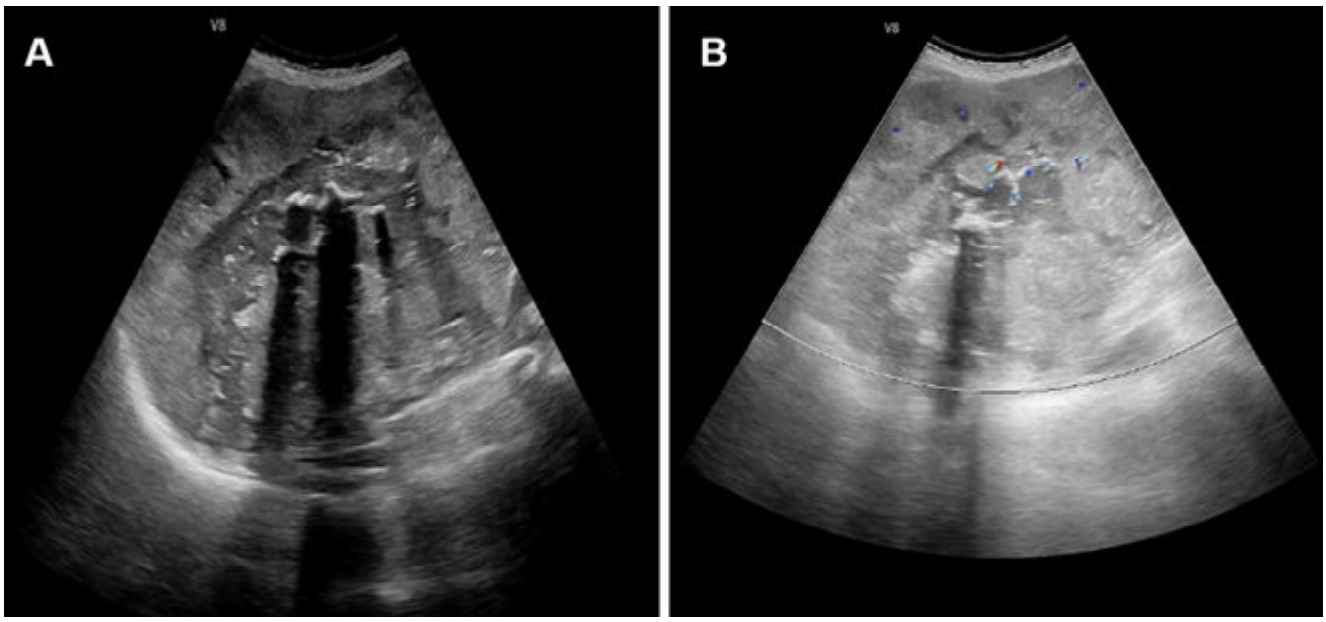

Figure 1. (A) large ill-defined heterogeneous lesion with the hyperechoic periphery and central hypoechoic area with multiple coarse calcifications completely occupying the right lobe of the liver; **(B)** mild vascularity on colour Doppler.

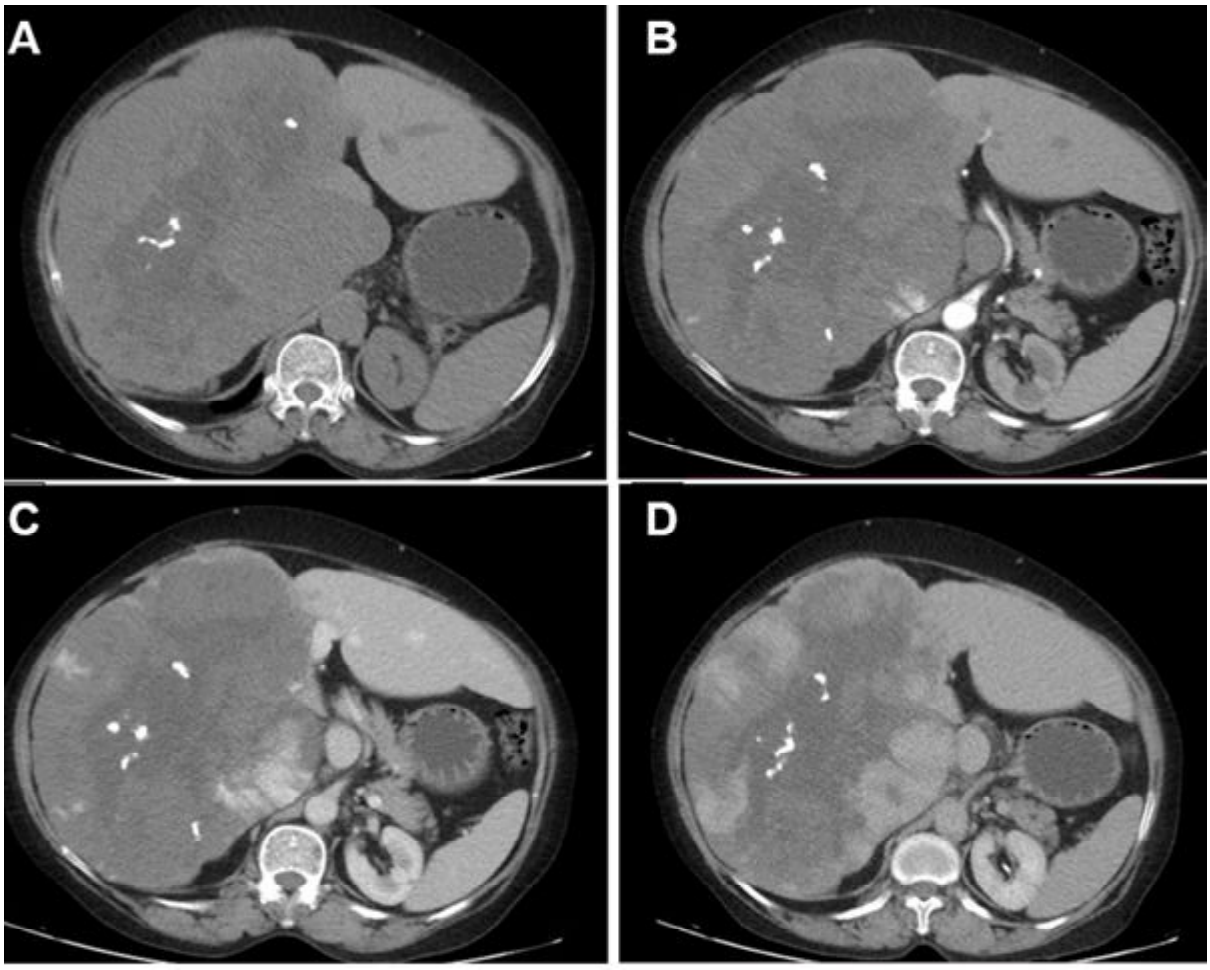

Figure 2. (A) large lesion completely occupying the right lobe of liver with coarse calcification and hypoattenuation. On triple-phase imaging; **(B)** peripheral nodular puddling of contrast on arterial phase; **(C)** gradual centripetal filling on portal venous phase; and **(D)** delayed phase.

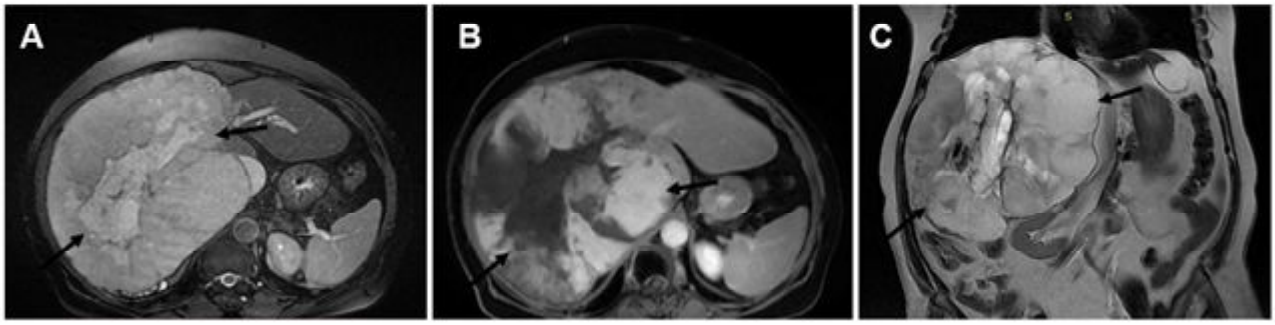

Figure 3. **(A)** hyperintense signal on Fast imaging employing steady-state acquisition (FIESTA) in the periphery; **(B)** on triple-phase imaging, peripheral nodular puddling of contrast with gradual centripetal filling on portal venous phase and isointense on delayed phase; **(C)** coronal T2WI showing peripheral hyperintense and irregular central hypointense signal.

In the delayed phase, the peripheral part showed isoattenuation with liver parenchyma, and the central part exhibited hypoattenuation **(Figure 2).** Extensive coarse calcifications were found in the central hypodense part. Another small hypodense lesion with a similar enhancement pattern was observed in the segment II. No signs of intrahepatic biliary radicle dilatation were noted. Mass effect in the form of inferomedial displacement of the right kidney, compression of an intrahepatic segment of the inferior vena cava, and obliteration of right and middle hepatic veins were noted. Compression of portal vein without any intraluminal invasion or thrombosis was noted. No evidence of retroperitoneal nodes or ascites was noted. Intrahepatic biliary radicles were also normal. These findings point toward diagnosing giant hemangioma rather than FL HCC. The biochemical profiles (coagulation profile, liver function tests, alpha-fetoprotein, and complete blood count) were also within normal limits. Carcinoembryonic antigen was 4.6 ng/ml, and alpha-fetoprotein was 2.3 ng/ml (normal < 5.8). Complete blood counts such as hemoglobin, platelet count, and red blood cells were also within normal limits.

Further triple phase contrast-enhanced MRI of the abdomen was performed to characterize the lesion and determine its enhancement pattern. The lesion showed a T2 hyperintense signal in the periphery, a central irregular hypointense signal, a hyperintense signal on diffusion-weighted imaging, and a iso to hypointense signal on apparent diffusion coefficient sequence **(Figure 3).** On the post-contrast sequences of liver, the lesion showed peripheral nodular puddling of contrast on the arterial phase with gradual centripetal filling on the portal venous phase and isointense on the delayed phase. No evidence of portal vein invasion or thrombosis was noted. The lesion compressed the hepatic veins. A similarly enhancing lesion was also found in the left lobe segment II.

Owing to the risk of hemorrhage, a biopsy was avoided. Positron emission tomography (PET) scan was performed to rule out any possibility of HCC or metastasis. The non-FDG avid Ill-defined, enlarged, nodular, irregular hypodense mass lesion occupied the entire right lobe with central scarring and calcification, with a standardized uptake value of 2.8 g/ml (no significant hypermetabolism) **(Figure 4)**. No abnormal hypermetabolic abdominal pelvic lymph nodes or macroscopic peritoneal disease were observed, and no evidence of abnormal metabolic activity was found in the rest of the body. On the basis of the above imaging findings, a diagnosis of atypical giant hepatic hemangioma with central scar and calcification was made.

Discussion

Hepatic hemangioma is the most frequent benign vascular liver lesion with a prevalence of 20.0%.[5] It has been observed in all age groups and has a female preponderance. Most lesions tend to be smaller than 5 cm and asymptomatic. However, large hemangiomas can become symptomatic. Giant hemangiomas pose a diagnostic challenge for radiologists because they mimic serious malignant liver pathologies such as HCC.[6] They also have a high risk of causing complications, including rupture with hemoperitoneum, mass effect on the adjacent structures such as a biliary tree and vessels, and Kasabach-Merritt syndrome-a rare and life-threatening complication involving thrombocytopenia, microangiopathic hemolytic anemia, and consumptive coagulopathy. Fortunately, our case did not have any of these complications.

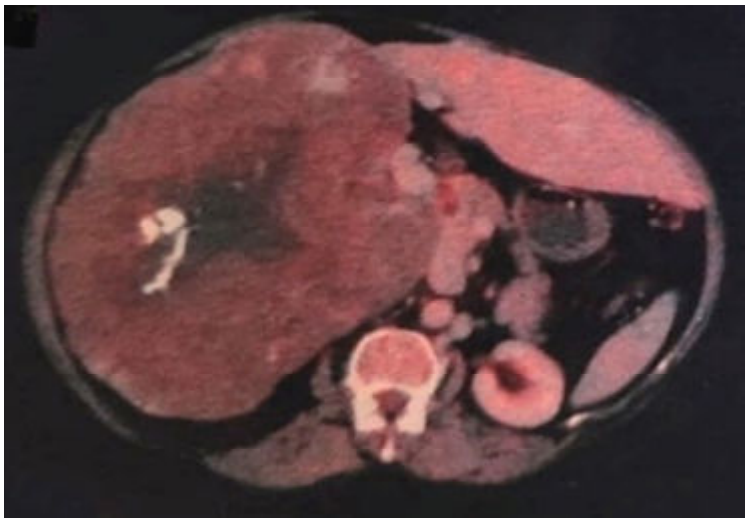

Figure 4. Non-FDG avid Ill-defined enlarged nodular irregular hypo dense mass lesion entirely occupying right lobe with central scarring and calcification, SUV max 2.8 g/ml.

For typical hemangiomas, imaging modalities are highly reliable for diagnosis. In USG, they appear as a well-defined homogenous echogenic mass. These lesions show hypoattenuation in noncontrast CT. After contrast administration, typical hemangiomas show peripheral nodular discontinuous enhancement on the arterial phase, and this peripheral attenuation is equal to the density of the contrast of the aorta. They have progressive centripetal enhancement in the portal vein and further centripetal filling on the delayed phase, making them iso-hypoattenuating to liver parenchyma. On MRI, these lesions are hypointense to the liver parenchyma on T1 and hyperintense to the liver parenchyma on T2.[7] Upon contrast administration, peripheral nodular discontinuous contrast enhancement appears on the arterial phase with progressive centripetal filling until the delayed phase. Post-contrast images using hepatobiliary contrast are less advantageous because they show a wide range of appearances.

Among the atypical hemangiomas reported in literature, the following have been described: large size, irregular hypodense area in the center due to thrombus, necrosis, and scar formation. Hemangiomas can also show calcifications, cystic degeneration, and fluid–fluid levels.

This case presented as a diagnostic challenge because of its vast size and central scar with coarse calcifications evident on ultrasound. Hepatic hemangiomas rarely show calcification, only found in 10.0% of USG images and 20.0% of CT scans.[2]

On the basis of the above features, FL HCC was one of the differentials. This malignancy is commonly found in young patients. Chronic liver disease and cirrhosis are not considered risk factors. On USG, FL HCC appears as well-defined masses with variable echogenicity. On CT, it is hypoattenuating on unenhanced scans and commonly shows calcification (40.0% - 68.0%); central stellar scar is present in 65.0% - 70.0% of cases.[8] A large scar (width > 2 cm) with radiating bands is characteristic of FL HCC. In contrast-enhanced CT, it is heterogeneously hyperattenuating in the arterial phase, and the enhancement pattern on portal venous and delayed phases is variable.[9] Delayed scar enhancement can be observed in 25.0% - 65.0% of cases. In the present case, AFP level was normal, which did not help in differentiating between FL HCC and giant hemangioma as its level is normal in both conditions. Less than 10.0% of FL HCC cases show an increased AFP level of more than 200 ng/ml. Meanwhile, the PET scan was normal and did not show any uptake, which helped to rule out any serious malignant condition like FL HCC and metastasis **(Table 1).**[10] Tc99RBC radionuclide is the confirmatory test that will demonstrate the focal increased accumulation of the tagged red blood cells corresponding to the hepatic lesion, confirming it to be a hemangioma.[11, 12]

Table 1. Characteristic imaging features of different hepatic lesions mimicking hemangioma.

Liver lesions	USG	CT	MRI	PET
Hemangioma	Well-defined homogenous echogenic mass	Arterial phase- peripheral nodular discontinuous enhancement Portal-venous phase- progressive enhancement with centripetal fill-in following blood pool Delayed phase- further fill-in	Arterial phase- peripheral nodular discontinuous enhancement Portal-venous phase- progressive enhancement with centripetal fill-in following blood pool Delayed phase- further fill-in	No uptake
HCC	Heterogenous lesions due to fibrosis, fatty change, necrosis and calcification	Late arterial phase- enhances vividly Portal-venous phase- becomes indistinct or hypoattenuating due to rapid washout Delayed phase-pseudo-capsular enhancement	Late arterial phase- enhances vividly Portal-venous phase- becomes indistinct or hypoattenuating due to rapid washout Delayed phase-pseudo-capsular enhancement	Uptake is present
FL HCC	Heterogenous lesion with central scar, can have calcification	Arterial phase enhancement Central scar can show enhancement in delayed phase	T1- Iso to hypointense T2- hypo to hyperintense T1C+ - arterial phase – heterogeneous enhancement Portal-venous/delayed- iso to hypointense Central scar – T1/T2 hypointense	Minimal uptake as compared to liver parenchyma
FNH	Well-defined mass with variable echogenicity and central scar	Arterial phase- bright homogenous enhancement Portal-venous phase- slightly hyperintense or iso attenuating Not associated with calcification	T1- Iso to hypointense T2- Iso to hyperintense T1C+ - bright homogenous enhancement on arterial phase Isointense on portal venous phase Central scar-T1- hypointense, T2 - hyperintense	No uptake
Sclerosed Hemangioma	A lesion with geographic pattern and variable echogenicity	Arterial phase-nodular foci or rim enhancement Portal-venous/ delayed phase - some new irregular enhancing areas may appear	T1- hypointense T2- variable signal T1C+ absent or mild enhancement Thin rim of Peripheral enhancement on arterial phase	No uptake

In patients who are symptomatic because of extrinsic compression, hemangiomas have been managed with surgical resection. Nonsurgical treatments include hepatic artery embolization and radiotherapy; however, these are rarely selected as the first choice.

Another benign hepatic tumor, fibronodular hyperplasia (FNH), is a differential in our case because it also has a central scar and occurs in a noncirrhotic liver background. The scar may show enhancement on the delayed scan. FNH can be differentiated from FL HCC on MRI: the central scar in FNH is hyperintense on T2 but hypointense in FL HCC.[13] Another type is sclerosed hemangioma, a hemangioma that has undergone degeneration and fibrous replacement **(Table 1)** and exhibits a geographic pattern with volume loss and capsular retraction.[14]

Conclusion

Hepatic hemangioma is a benign liver lesion. In terms of management, it is difficult to differentiate from other liver lesions with malignant potential such as HCC and metastasis. In these cases, aggressive imaging workout with the help of cross-sectional imaging modalities such as CT, MRI, or PET scan should be performed to differentiate hemangioma from other liver lesions.

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Conflict of interest statement

Each of the authors has completed an ICMJE disclosure form. None of the authors declare any potential or actual relationship, activity, or interest related to the content of this article.

Data sharing statement

Data generated or analyzed for the present report are included in this published article. Further details are available from the corresponding author on reasonable request after deidentification of the patient whose data are included in the report.

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