

Giant Hepatic Hemangioma: A Case Report

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Introduction:

Hepatic hemangiomas are the most common benign liver tumors,^{1,2} representing benign vascular malformations composed of dilated blood-filled spaces lined by endothelium and supported by fibrous stroma. Prevalence in the general population varies widely, with estimates ranging from 0.4% to 20% in autopsy series³⁻⁵ (some imaging-based studies report 2–7% in clinical populations, reflecting improved detection with modern modalities). Giant hepatic hemangiomas (GHH) represent a rarer and more clinically significant subtype. The definition of a GHH varies in the literature: it is commonly defined as a lesion greater than 4cm, 5cm, or 10cm in diameter, with >10 cm often used for very large or symptomatic cases in surgical and complication-focused reports.⁶⁻⁹ These lesions are

Abstract

Hepatic hemangiomas are the most common benign liver tumors, typically asymptomatic and discovered incidentally. Giant hepatic hemangiomas (>10 cm) are rare and may present with symptoms due to mass effect. This report describes a 45-year-old woman presenting with abdominal fullness due to a massive hepatic hemangioma measuring approximately 20.5×17.5×23 cm in the left lobe, with a smaller lesion in the right lobe. Diagnosis was confirmed via ultrasonography, contrast-enhanced computed tomography (CECT), and fine-needle aspiration cytology (FNAC). Laboratory findings included mild normocytic normochromic anemia and leucopenia, with normal liver function tests and tumor markers. Management was conservative, with recommendations for open biopsy. This case underscores the diagnostic utility of multiphasic imaging and the benign nature of these lesions, supported by a review of similar cases in the literature.

Keywords: Giant hepatic hemangioma, Liver tumor, Contrast-enhanced CT, Fine-needle aspiration cytology

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predominantly found in women between 20 and 60 years of age and are often located in the right hepatic lobe.^{6,10}

Hepatic hemangiomas are typically asymptomatic and discovered incidentally during imaging studies performed for unrelated conditions.^{2,3} However, GHHs can become symptomatic due to mass effect on adjacent organs or structures, leading to abdominal distension, pain (including epigastric pain¹¹), loss of appetite, early satiety, or compressive symptoms.^{10,11} In rare cases, GHHs are associated with severe complications, including spontaneous rupture, intratumoral hemorrhage, torsion (especially in pedunculated variants), Kasabach-Merritt syndrome (consumptive coagulopathy and thrombocytopenia), sclerosing variants presenting as Bornman-Terblanche-Blumgart syndrome,¹² and high-output cardiac

failure.^{4,5,10,13} For example, one reported case involved a GHH complicated by bilateral pulmonary embolism and portal hypertension,¹⁴ while another described a 23 cm GHH with severe malnutrition and blood coagulopathy.¹⁵ Giant abdominal extension originating from the liver has also been documented.¹⁶ Unusual presentations, such as fetal hepatic masses, have been noted in other contexts.¹⁷ A rapidly enlarging 25-cm GHH in a Bangladeshi patient required surgical resection due to Kasabach-Merritt syndrome,¹⁸ while an earlier Bangladeshi report described conservative management of multiple lesions (largest ~11.5 cm) in a 28-year-old woman diagnosed via characteristic MRI findings.¹⁹ The risk of complications generally correlates with tumor size, with higher risks observed for lesions exceeding 5cm, consistent with Laplace's law (where wall tension increases with radius).⁶

Diagnosis of GHH relies on characteristic imaging features observed in ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI).^{2,20} The most common type, cavernous hemangioma, shows discontinuous peripheral nodular enhancement in the arterial phase, with progressive centripetal fill-in in the portal venous and delayed phases on dynamic contrast-enhanced studies.²¹ This pattern reflects the low-flow hemodynamic state ("slow river" flow). On T2-weighted MRI, lesions often appear markedly hyperintense ("light-bulb bright") due to the long T2 relaxation time of stagnant blood.

Management of GHH depends on symptoms, size, growth rate, and presence of complications. Asymptomatic or minimally symptomatic GHHs are usually managed conservatively with observation.^{2,3} Symptomatic or complicated cases may require intervention, including surgical resection or enucleation, transarterial embolization (TAE), radiofrequency ablation, or in rare, unresectable cases with severe symptoms liver transplantation.^{10,20,22,23} Transarterial embolization using agents such as bleomycin-lipiodol emulsion has shown efficacy in reducing tumor size and alleviating symptoms.^{24,25} Surgical resection guided by three-dimensional visualization is effective for GHHs with hemorrhagic necrosis.²¹ The overall prognosis for GHH is excellent with appropriate individualized management.²⁰

Case Presentation

A 45-year-old woman presented on January 25, 2026, with progressive abdominal fullness over several months. She had no history of jaundice, fever, weight loss, gastrointestinal bleeding, or oral contraceptive use. Physical examination revealed a large, soft, non-tender abdominal mass without signs of anemia, jaundice, edema, or organomegaly. Vital signs were normal. Initial ultrasonography (USG) of the whole abdomen revealed a large heterogeneous soft tissue mass measuring more than 34×22cm, displacing abdominal organs, with a suspected retroperitoneal origin. The liver's right lobe was partially visualized without focal lesions in the visible portions; gallbladder, biliary tree, pancreas, spleen, kidneys, urinary bladder, uterus, and adnexa appeared normal where visualized.

Subsequent spiral contrast-enhanced CT (CECT) of the whole abdomen demonstrated an enlarged liver spanning 28cm. A large hypodense lesion measuring approximately 20.5×17.5×23cm was identified in segments II, III, and IV of the left lobe. Triphasic imaging showed peripheral discontinuous nodular enhancement in the arterial phase, gradual centripetal filling in the portal venous phase, and prolonged enhancement in the delayed phase, characteristic of hemangioma. A smaller similar lesion was noted in the right lobe. The right kidney and pancreas were compressed and displaced inferiorly, with normal contrast excretion and no dilatation of the pelvicalyceal system. No lymphadenopathy, ascites, or other abnormalities were seen. Impression: Classical findings consistent with giant hepatic hemangioma involving both lobes.

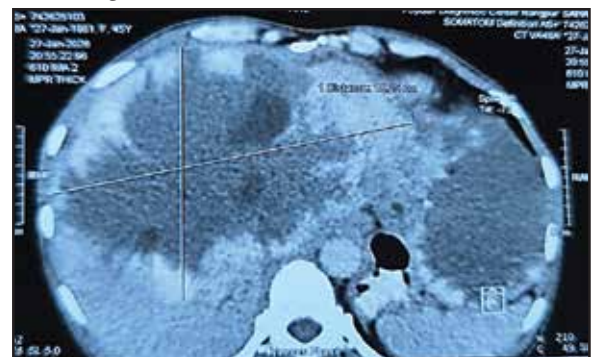


Figure-1: Representative axial slices from plain, arterial, portal venous, and delayed phases of CECT showing the giant hemangioma with characteristic enhancement patterns

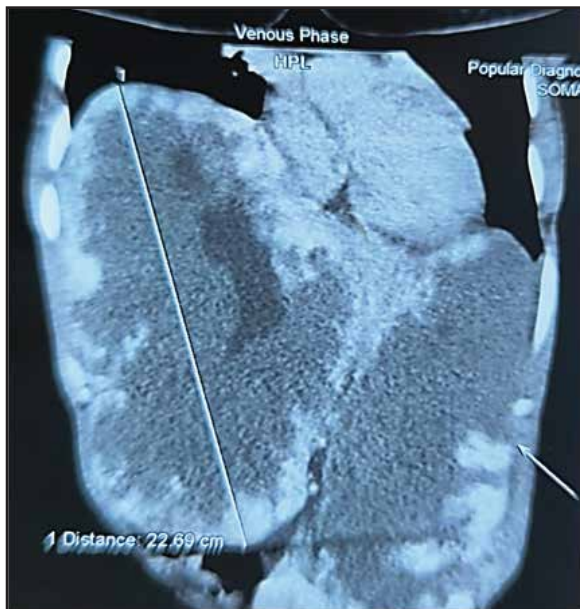


Figure-2: Coronal and sagittal reformatted images demonstrating mass effect on adjacent structures

Hematology showed hemoglobin 9.3 g/dL, erythrocyte sedimentation rate 37mm/1st hour, red blood cell counts $3.0 \times 10^{12}/L$, white blood cell count $3.8 \times 10^9/L$ (with normal differential), and platelet count $190 \times 10^9/L$. Peripheral blood film indicated normocytic normochromic anemia with leucopenia. Biochemistry revealed random blood sugar 6.8mmol/L, serum creatinine 1.03mg/dL, and ALT 38 U/L, all within normal limits. Immunology: HBsAg negative, CA-125 17.96 U/mL (normal). Blood group: A positive.

USG-guided fine-needle aspiration cytology (FNAC) from the abdominal mass yielded fresh blood spontaneously. Microscopic examination revealed formed elements of blood without malignant cells. Diagnosis: Compatible with giant hemangioma. Remark: Open biopsy recommended for further evaluation.

The patient was admitted to the gastroenterology ward and managed conservatively with soft liquid diet, blood transfusion (2 units), intravenous fluids, iron supplementation (Ferisen 500mg IV daily for 5 days), and analgesia (Disopan 1mg IV as needed). Follow-up was advised in 2 weeks, with CECT and FNAC already performed.

Discussion:

Giant hepatic hemangiomas (GHHs) are benign vascular tumors and the most common benign liver neoplasms, with prevalence ranging from

1.4% to 20% in autopsy series.¹⁻⁵ In this report, a 45-year-old woman presented with progressive abdominal fullness attributable to a giant hepatic hemangioma measuring approximately 20.5×17.5×23cm in the left lobe (segments II, III, and IV), fulfilling the criteria for GHH commonly defined as >10 cm.⁵⁻⁸ This size places it among the largest reported lesions, comparable to the 23cm case complicated by severe malnutrition and coagulopathy and the 25cm GHH from Bangladesh that required surgical resection.⁹

Although GHHs are predominantly located in the right hepatic lobe,⁹ this lesion originated primarily from the left lobe with an additional smaller lesion in the right lobe. The patient experienced symptoms secondary to mass effect, including displacement of adjacent structures (right kidney and pancreas), which aligns with the typical presentation of symptomatic GHH causing abdominal distension, fullness, and early satiety.^{9,10} There was no history of oral contraceptive use (a factor noted in some pedunculated variants)⁷ and, importantly, no severe complications such as spontaneous rupture, intratumoral hemorrhage, Kasabach-Merritt syndrome, pulmonary embolism, or high-output cardiac failure.

Diagnosis was established through a stepwise imaging approach. Initial ultrasonography suggested a large heterogeneous mass (>34×22cm) possibly of retroperitoneal origin, illustrating the limitations of ultrasound when lesions reach massive proportions. Subsequent multiphase contrast-enhanced computed tomography (CECT) provided the definitive diagnosis, demonstrating the classic pattern of discontinuous peripheral nodular enhancement in the arterial phase, gradual centripetal filling in the portal venous phase, and prolonged enhancement in the delayed phase.^{12,15} These features reflect the low-flow hemodynamic state ("slow river" flow) characteristic of cavernous hemangiomas.¹⁵

USG-guided fine-needle aspiration cytology (FNAC) yielded fresh blood without malignant cells, supporting the benign vascular nature; however, open biopsy was prudently recommended for further histological confirmation, reflecting the cautious diagnostic approach advocated for giant lesions. Laboratory investigations revealed mild normocytic normochromic anemia and leucopenia (hemoglobin 9.3g/dL, WBC $3.8 \times 10^9/L$)

with normal platelet count, liver function tests, and tumor markers. These mild hematological changes did not evolve into consumptive coagulopathy or thrombocytopenia, distinguishing this case from complicated GHHs associated with Kasabach-Merritt syndrome.¹⁹

In the absence of life-threatening complications or rapid enlargement, management remained conservative with supportive care (blood transfusion, intravenous iron supplementation, and symptomatic relief), consistent with recommendations for uncomplicated or mildly symptomatic GHH. This approach contrasts with the interventions required in more aggressive cases surgical resection or enucleation,¹⁹ transarterial embolization,²¹ or liver transplantation²² typically reserved for symptomatic, rapidly growing, or hematologically complicated lesions. The present case adds to the limited Bangladeshi experience with GHH, demonstrating that even very large lesions can be safely observed when multiphasic imaging confirms the classic benign pattern.

This case reinforces the diagnostic utility of multiphasic CECT and the generally favorable prognosis of GHH with individualized management. Early recognition through characteristic imaging features avoids unnecessary aggressive interventions, while regular follow-up is essential to monitor for potential growth or rare complications, whose risk increases with tumor size according to Laplace's law.

Conclusion:

This case highlights a symptomatic giant hepatic hemangioma diagnosed through characteristic imaging and cytology, managed conservatively. Early recognition avoids unnecessary interventions, with biopsy recommended for confirmation. Further studies on regression factors could inform non-surgical approaches.

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