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HEPATIC HEMANGIOMA IN INFANCY: A RARE CASE REPORT

Radiology

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ABSTRACT

Background: Infantile hepatic hemangioma (IHH) is the most frequently occurring benign vascular tumor in infants, typically presenting within the first six months of life. It exists in focal, multifocal, or diffuse forms and can range from asymptomatic cases to severe complications, including high-output cardiac failure and consumptive hypothyroidism. **Case Report:** We describe a case of a 1-month-old male infant who presented with respiratory distress, abdominal distension, and irritability. Clinical examination revealed hepatomegaly, and laboratory tests indicated an elevated albumin-to-globulin (A/G) ratio, increased direct bilirubin, and significantly high alpha-fetoprotein levels. Imaging via contrast-enhanced computed tomography (CECT), ultrasonography (USG), and magnetic resonance imaging (MRI) identified a well-defined hepatic lesion with peripheral enhancement and central necrosis, consistent with IHH. **Discussion:** IHH is often identified through imaging, with USG and MRI being key diagnostic tools. Its pathogenesis is linked to endothelial cell proliferation stimulated by vascular endothelial growth factor (VEGF), and histopathological analysis typically reveals GLUT-1 positivity. While many cases regress spontaneously, symptomatic or extensive lesions may require medical intervention. Propranolol has become the preferred treatment, promoting tumor regression through VEGF suppression and vasoconstriction. Other therapeutic approaches include corticosteroids, sirolimus, embolization, or surgical removal in unresponsive cases. **Conclusion:** Timely recognition of IHH is vital to avoid complications and guide appropriate management. Imaging plays a critical role in differentiating it from malignant hepatic tumors. While spontaneous resolution is common, propranolol therapy has significantly improved patient outcomes, reducing the necessity for invasive interventions. A multidisciplinary approach is essential for ensuring optimal care and prognosis.

KEYWORDS

INTRODUCTION

Infantile hepatic hemangioma is Benign vascular tumor of the liver that is lined by endothelium and is the most common benign hepatic tumor in infants.

It is divided into focal, multifocal, and diffuse subtypes.

Focal infantile hepatic hemangioma is thought to be the hepatic form of cutaneous rapidly involuting congenital hemangioma (RICH).

There is typically no association with cutaneous hemangiomas and no known sex predilection.

Histopathologic antibody staining of the endothelial cells in RICH is negative for glucose transporter protein-1 (GLUT1). RICH is usually symptomatic because of arteriovenous shunting.

Multifocal subtype of infantile hepatic hemangioma is hepatic form of simple cutaneous infantile hemangioma and is often associated with multiple cutaneous hemangiomas which commonly present before 6 months of age.

It is positive for GLUT1 and involute spontaneously by 12-15 months of age

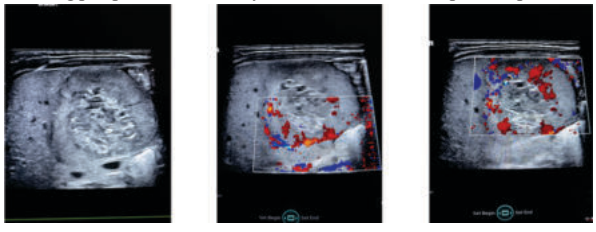
Case Report

1 month old male child came with complaints of breathlessness, abdominal tightness and irritability for 2 days. No complaints of fever, vomiting, altered bowel or bladder habits. On examination, the child had palpable liver (indicating hepatomegaly) with liver function test showing increased A/G ratio (2.9) and increased direct bilirubin levels & increased Alfa Feto Protein levels: 1210 ng/dl.

The child had gotten CECT abdomen pelvis outside which showed well defined hypodense lesion with exophytic component arising from segment V of liver with focal faint calcifications showing moderate peripheral enhancement & predominant central necrosis/ cystic change.

The child was referred for USG & MRI Abdomen Pelvis (Plain + Contrast)

USG showed heteroechoic lesion involving segment V of liver with central cystic spaces and multiple hyperechoic strands within it showing peripheral vascularity and mass effect on the porta hepatis.

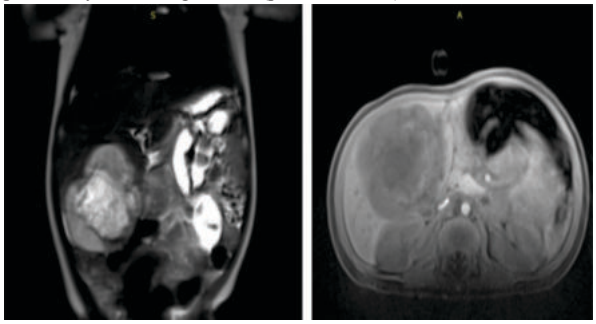


MRI Abdomen Pelvis with contrast was done for clear characterization of the lesion.

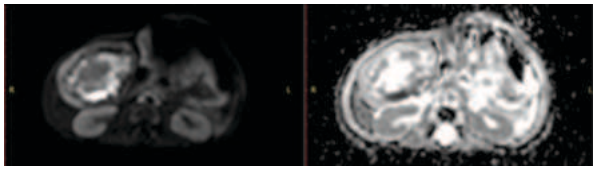
Findings Included: well-defined T2 heterointense (predominantly hyperintense) and T1 hypointense mass lesion involving segment V of right lobe of liver with few areas of diffusion restriction on DWI sequence.

On contrast study, the lesion shows irregular wall enhancement with central non enhancing areas.

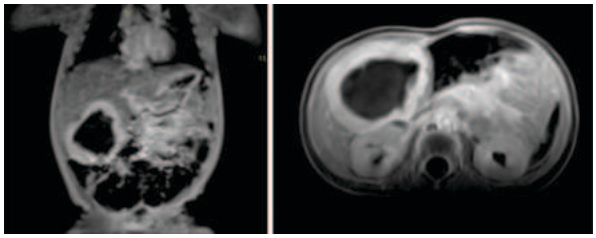
On delayed phases, the lesion shows intense wall enhancement with mass effect on the bowel loops and upper pole of right kidney and posteriorly, narrowing of aorta (post celiac axis) noted



T2 T1



DWI



Post Contrast

DISCUSSION

Infantile hepatic hemangioma (IHH), previously termed hepatic infantile hemangioendothelioma, is the most common benign vascular liver tumor in infants. It is typically identified within the first six months of life and presents with a broad clinical spectrum, ranging from asymptomatic cases to severe complications such as high-output cardiac failure, abdominal distension, and consumptive hypothyroidism due to increased thyroid hormone metabolism within the tumor (1,2).

The exact etiology of IHH remains unclear, but it is believed to result from endothelial proliferation driven by vascular endothelial growth factor (VEGF) (3). These tumors may be solitary or multifocal, with the latter often associated with cutaneous hemangiomas and systemic manifestations. Imaging plays a crucial role in diagnosis, with ultrasonography (USG) and Doppler studies revealing well-defined hypoechoic or hyperechoic lesions with increased vascularity (4). Further characterization with contrast-enhanced computed tomography (CT) and magnetic resonance imaging (MRI) typically demonstrates early arterial enhancement with delayed venous washout, a hallmark of hepatic hemangiomas (5).

Histologically, IHH closely resembles cutaneous infantile hemangiomas and is GLUT-1 positive, a feature distinguishing it from other vascular tumors (6). The clinical course of IHH is variable; while many lesions resolve spontaneously, large or symptomatic cases require intervention. Propranolol, a non-selective beta-blocker, is now the first-line treatment due to its effectiveness in reducing VEGF levels and inducing regression by promoting vasoconstriction (7). Other treatment options include corticosteroids, sirolimus, and in resistant cases, embolization or surgical resection (8).

Complications such as Kasabach-Merritt syndrome (thrombocytopenia and coagulopathy) and cardiac failure necessitate close monitoring. In the present case, early initiation of propranolol therapy led to significant tumor regression, emphasizing the importance of timely diagnosis and management.

CONCLUSION

IHH is a rare but significant cause of hepatic tumors in neonates and infants. Its diverse clinical spectrum necessitates a thorough evaluation using imaging and, in selected cases, histopathology. While many cases resolve spontaneously, medical management with propranolol has revolutionized treatment, reducing the need for invasive procedures. Multidisciplinary care involving neonatologists, pediatric hepatologists, and radiologists is crucial for optimal outcomes.

This case report highlights few radiological findings in such case.

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