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REVIEW ARTICLE

Space Occupying Lesions in the Liver

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Abstract Space occupying liver lesions usually present with abdominal pain or abnormal physical findings, such as a palpable abdominal mass or distention. Liver lesions identified in children include benign and malignant neoplasms, inflammatory masses, cysts and metastatic lesions. Two-thirds of liver lesions in children are malignant. Hepatoblastoma accounts for two-thirds of malignant liver tumors in children. Benign lesions of the liver in children include vascular lesions, hamartomas, adenomas, and focal nodular hyperplasia. Although benign and malignant liver masses share some clinical manifestations, however treatment and prognosis differ. Evaluation involves physical examination, imaging evaluation and laboratory investigations such as serological markers [alpha-fetoprotein (AFP)] for malignant liver lesions. Ultrasound is the initial imaging modality of choice because it can detect, characterize, and provide the extent of liver lesions. However, CT or MRI are often subsequently performed for further characterization, assessment of precise extent, and detection of associated metastatic disease in cases of malignant hepatic neoplasm. Serological markers (such as alpha fetoprotein) can be useful in narrowing the differential diagnosis when they are markedly elevated but a substantial number of patients unfortunately do not have high levels of these markers at the time of presentation or cautious interpretation is warranted as AFP level is frequently elevated in infants up to 6 mo of age and may be slightly elevated with benign tumors and with hepatic insult or regeneration. Therefore, a tissue diagnosis is often required to guide subsequent management. The histology and anatomy of a pediatric liver tumor guides the treatment and prognosis.

Keywords Focal nodular hyperplasia · Hepatoblastoma · Hepatocellular adenoma · Hepatocellular carcinoma · Infantile hemangioendothelioma · Nodular regenerative hyperplasia

Introduction

Space occupying lesions in the liver present a relatively common clinical dilemma, particularly with the increasing use of various imaging modalities in the initial assessment of children presenting clinically with deranged liver function tests or non specific abdominal symptoms [1, 2]. The management of pediatric liver lesions may be challenging and it may require a complete work-up because of symptoms or concern about malignancy. Initial evaluation should be focused on patient history, gestational history and age, weight and findings on physical exam. Diagnostic imaging modalities may facilitate the identification of benign and malignant liver tumors, however biopsy or resection for histological diagnosis sometimes becomes necessary [3]. Standard

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Table 1 Common tumor markers elevated in pediatric liver malignancies

Tumor marker	Malignancy
Beta HCG	Hepatoblastoma, Malignant germ cell tumors
Testosterone	Hepatoblastoma
Ferritin	HCC
CEA	HCC
LDH	Most malignant tumors
Catecholamine	Infiltrative neuroblastoma

HCG Human chorionic gonadotropin; *CEA* Carcino embryonic antigen; *LDH* Lactate dehydrogenase; *HCC* Hepatocellular carcinoma

histologic examination usually is complemented by immunohistochemical analysis of protein biomarkers. Some of the infantile hepatic neoplasms are highly vascularized and surgical interventions are at high risk of bleeding. Certain tumor markers may be helpful in the initial work-up and evaluation of response to therapy. Alpha-fetoprotein (AFP) level may be elevated in children with malignant lesions such as hepatoblastoma and hepatocellular carcinoma, but cautious interpretation is warranted as AFP level is frequently elevated in infants up to 6 mo of age and may be slightly elevated with benign tumors and with hepatic insult or regeneration [3, 4]. Therapy must be tailored according to the nature of the lesion. Observation is recommended for asymptomatic hepatic hemangioma, whereas complete surgical resection is the mainstay of treatment in hepatoblastoma [4]. Unresectable metastatic masses require oncologic consultation and therapy. The efficient characterization and management of liver lesions therefore requires a multidisciplinary collaboration between the hepatologist, radiologist, pathologist, hepato-biliary or transplant surgeon, and pediatric oncologist.

Initial Clinical Evaluation

A careful review of the personal history and physical examination findings often helps in narrowing the differential diagnoses of liver lesions. A history of constitutional symptoms such as fever may be useful in the diagnosis of hepatic abscesses; although fever can also be associated with malignancy.

The physical examination may show features of chronic liver disease such as spider angiomas, periumbilical caput medusa indicative of portal hypertension, hepatomegaly, or splenomegaly.

Presence of skin hemangiomas raises the possibility of liver lesions likely to be vascular lesions. The family history is also of value in the initial clinical evaluation. A family history of young-onset diabetes mellitus, for example, may predispose to hepatic adenomatosis.

Most children with a primary liver tumor present with abdominal distention and a palpable mass in the upper abdomen often without other signs of severe disease. Anemia is often present [1]. Only in advanced stage of disease, overall status deteriorates and the children develop abdominal pain, weight loss, nausea, vomiting, and ascites. Jaundice, signs of hepatic insufficiency or an incidental rupture of the tumor with intraabdominal bleeding are very rarely observed. Many tumors may have reached a considerable size before they are noticed and treatment initiated [5]. However, some specific symptoms are associated with the different tumors, such as fever and thrombocytosis with hepatoblastoma and precocious puberty secondary

Table 2 Common space occupying lesions of liver [1, 11, 12]

Benign	Malignant	Benign/Malignant
Infantile hemangioendothelioma	Hepatoblastoma	Primary hepatic teratoma
Hemangioma	Hepatocellular carcinoma	
Focal nodular hyperplasia	Undifferentiated embryonal sarcoma	
Mesenchymal hamartomas	Rhabdoid tumor	
Hepatic adenoma	Metastasis	
Biliary and simple hepatic cysts	Embryonal rhabdomyosarcoma	
Pyogenic and amebic abscess	Angiosarcoma	
Hematoma		
Parasitic cysts		

to human chorionic gonadotropin or rarely testosterone production in hepatoblastoma or germ cell tumors. High output cardiac insufficiency due to arterio venous shunting in the lesion and platelet sequestration and consumptive coagulopathy (Kasabach-Meritt syndrome) can be encountered in young infants with a hemangioma or hemangioendothelioma of the liver, who often show hemangiomas of the skin and organs [5].

Differential diagnosis of liver lesions in neonates seems to be particularly difficult because diagnostic criteria and tumor markers do not apply and imaging is nonspecific. Rarely, hepatic choriocarcinoma can occur in neonates, clinically resembling infantile hemangioendothelioma but secreting beta HCG. Malignant Rhabdoid tumors can become a differential diagnostic problem to hepatoblastoma in infants and young children and benign teratomas have to be differentiated from mesenchymal hamartomas in the same age group.

Children with background genetic or metabolic disorders or infectious disease can develop liver lesions [4]. Most important are hepatoblastoma in children with a Beckwith-Wiedmann syndrome or other hemihypertrophy syndromes, familial polyposis coli and children with very low birth weight. Hepatocellular carcinoma can occur in endemic areas with perinatal hepatitis B or C infection or chronic liver diseases.

Laboratory Investigations

The history and physical examination are complemented by laboratory tests that may show active hepatitis, a low platelet count caused by chronic liver disease with cirrhosis, portal hypertension and hypersplenism, or hyperbilirubinemia.

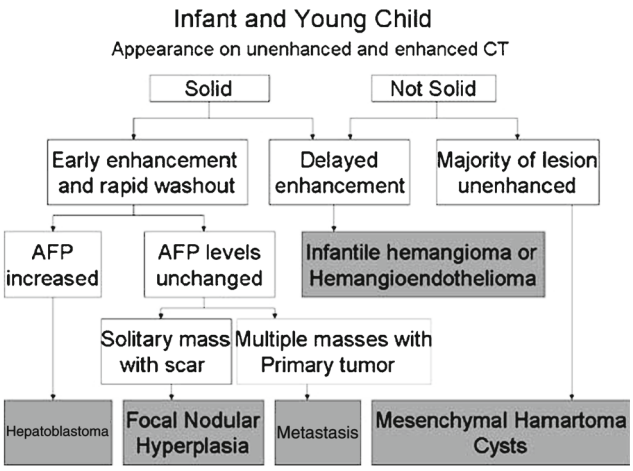
The most important tumor marker is alpha-fetoprotein (AFP), which is highly elevated in 80 to 90 % of all hepatoblastomas and moderately elevated in 50 % of hepatocellular carcinoma (HCC) patients. It can also be highly elevated in malignant teratoma and yolk sac tumors of liver. AFP normal levels can exceed 500,000 ng/ml in neonates and 300 ng/ml in 3-y-old children. Neonatal hepatoblastomas do not produce enough AFP to produce serum levels markedly above the normal range. A slightly elevated level of AFP is found in other tumors, after damage or during regeneration of liver parenchyma [4]. Other markers elevated are listed in Table 1.

Virological titers should be investigated for hepatitis B, and C (HCC) [6], HIV-1 (Fibrosarcoma), cytomegaly and EBV (Lymphoma).

Role of Imaging

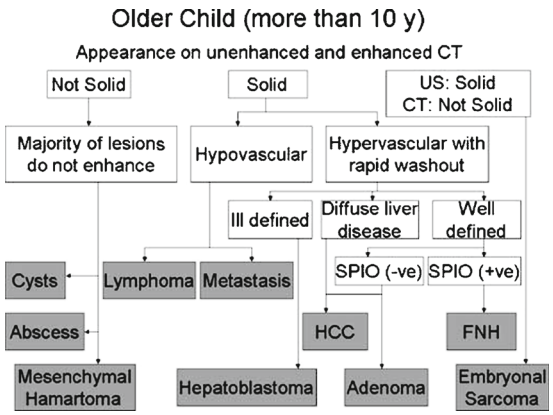
The initial modality of choice in imaging space occupying lesion in the liver is ultrasound (US). The lesion can be localized to the liver, characterized as solid or cystic on grey scale and vascularity within and around the lesion can be gauged with color/power Doppler [3, 7]. Doppler also helps to determine vascular invasion or thrombosis. It can be performed without anesthesia and repeated often for follow up, as it does not involve ionizing radiation. US is the recommended technique for follow-up [8]. Contrast enhanced ultrasound (CEUS) is increasingly promising in demonstrating the benign nature of focal liver lesions that are indeterminate on grey-scale sonography in children, potentially reducing the use of CT [1, 9]. US-guided liver biopsy in children is a procedure with a low rate of major complications and a high rate of minor bleeding that does not require intervention [9].

The role of advanced imaging tests such as CEUS and Promovist enhanced magnetic resonance imaging, which allow for non-invasive assessment of liver tumors, is of utmost importance in pediatric patients, especially when repeated imaging tests are needed and radiation exposure should be avoided.



AFP Alpha feto protein

Fig. 1 Imaging algorithm useful to diagnose hepatic lesions in infants and young children



SPIO Super paramagnetic iron oxide; *FNH* Focal nodular hyperplasia; *HCC* Hepatocellular carcinoma

Fig. 2 Imaging algorithm useful to diagnose hepatic lesions in older children (more than 10 y)

CT and MRI help in identifying the lesion as either solitary or multifocal and best delineate the size and margins of the liver lesion [1]. MRI with contrast enhancement may provide the best identification of flow characteristics and surrounding vascular structures without ionizing radiation risk of CT [8].

Accurate characterization of liver masses by cross-sectional imaging is particularly dependent on an understanding of the unique phasic vascular perfusion of the liver and the characteristic behaviors of different lesions during multi- phasic contrast imaging. Cross-sectional imaging with CT or MRI is enhanced by the use of intravenous contrast agents and dynamic multiphasic examination techniques. The liver has 3 distinct phases after intravascular contrast agent is injected *via* a peripheral vein. The arterial phase occurs 10 to 15 s after peripheral contrast injection and is caused by the direct infusion of arterial blood with a high concentration of contrast from the heart through the hepatic artery into the liver. Next, the portal venous phase occurs 60 to 75 s after contrast injection as blood from the gastrointestinal tract is collected in the portal vein for processing in the liver. Finally, in the venous phase, blood from the liver is collected into the hepatic veins, which converge to the inferior vena cava for return to the right atrium. The intravascular contrast leaks through the liver sinusoids into the extracellular space and about 3 to 5 min after injection, the extracellular contrast reaches equilibrium with the

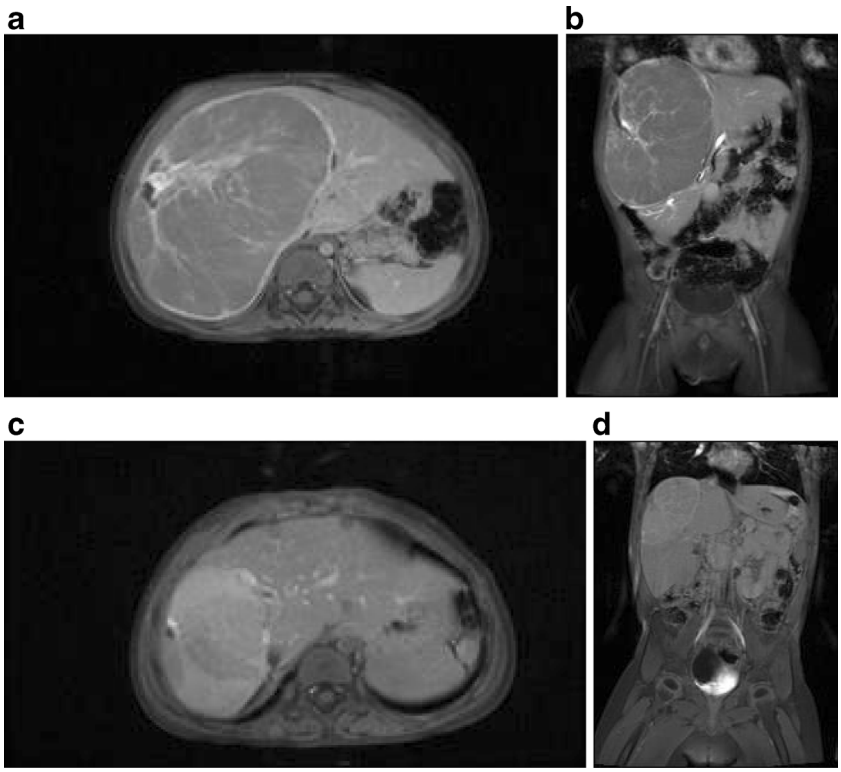


Fig. 3 Hepatoblastoma: **a** & **b** Pre treatment T1 fat sat post intravenous gadolinium axial and coronal sequence of the liver show a large predominant non enhancing mass occupying the right lobe of liver and involving the right hepatic vein and right branch of portal vein. **c** & **d** Post treatment T1 fat sat post intravenous gadolinium axial and coronal sequences of the liver show more than 50 % reduction in the size of mass in the right lobe of liver and retraction from right hepatic vein and right branch of portal vein

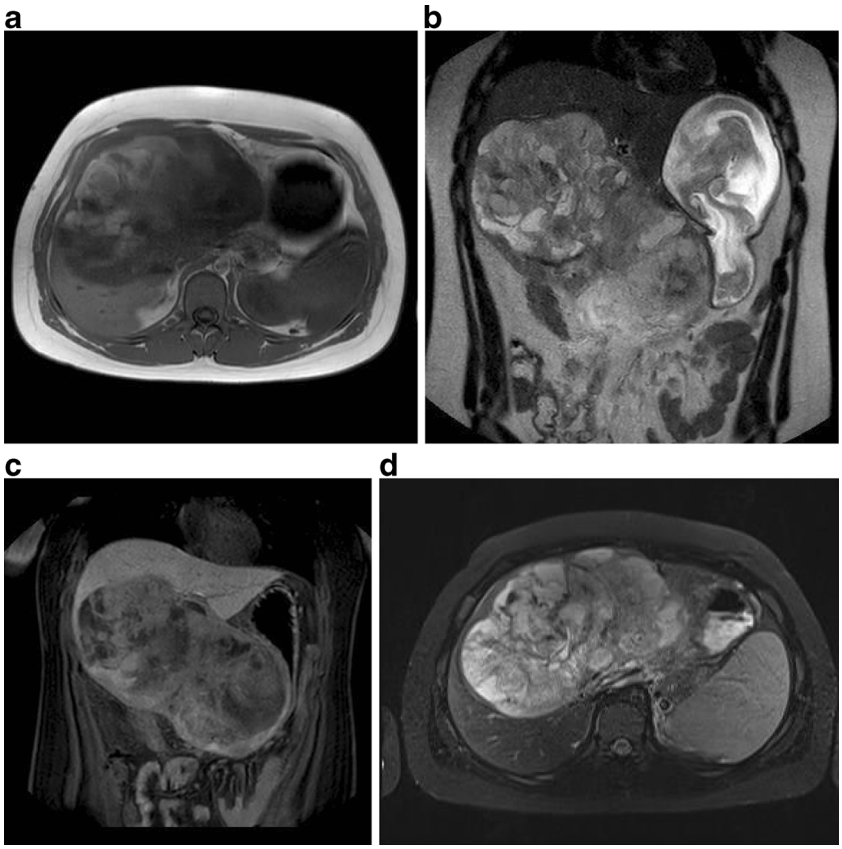


Fig. 4 Undifferentiated embryo sarcoma: **a, b** & **c** T1 axial, T2 and STIR coronal sequences shows a large predominant solid mixed intensity lesion arising and involving the entire right lobe of liver. **d** T1 fat sat post intravenous gadolinium axial sequence shows predominantly enhancing solid mass

concentration in the vascular system. This is known as the equilibrium phase. This unique blood supply to the liver is exploited by contrast imaging techniques because many mass lesions have characteristic patterns of appearance in the arterial, portal venous, and equilibrium phases. Newer contrast agents that are taken up by functioning hepatocytes and excreted into bile, such as disodium gadoxetate (Gd-EOB- DTPA; Eovist; Bayer Corporation, Pittsburgh, PA) and gadobenate dimeglumine (Gd-BOPTA; MultiHance; Bracco Diagnostics Inc., Princeton, NJ), provide further phenotypic characterization of liver masses and are particularly useful in the differentiation of adenomas from focal nodular hyperplasias (FNHs) and the diagnosis of HCC and metastases. The enhancement of hepatocytes with these hepatobiliary contrast agents in the hepatocyte or parenchymal phase typically peaks between 20 and 60 min after intravenous injection. Uptake of gadoxetate and gadobenate is believed to occur mainly through cell membrane proteins in the bile canaliculi and ducts, including organic anion transporting polypeptides and multidrug resistance protein. The expression of these proteins is usually suppressed in adenomas and HCCs and lack of the hepatocyte phase enhancement is useful in differentiating them from FNH.

Magnetic resonance elastography and acoustic radiation force impulse imaging are currently under investigation and may potentially be useful techniques in the characterization of liver masses.

Radiological findings may not always reflect true liver pathology. CT and MRI are instrumental in delineating intra- and extra-hepatic extent of disease [4, 7]. MRI can further improve diagnosis by defining features such as signal intensity characteristics, vascularity, stromal component and intra-lesional necrosis and hemorrhage [1]. Positron-emission tomography (PET) CT offers a greater sensitivity for residual and relapsed disease and may facilitate surgery. Radiological findings alone are useful in diagnosis in 58 % of cases, while in remainder histology is necessary [1].

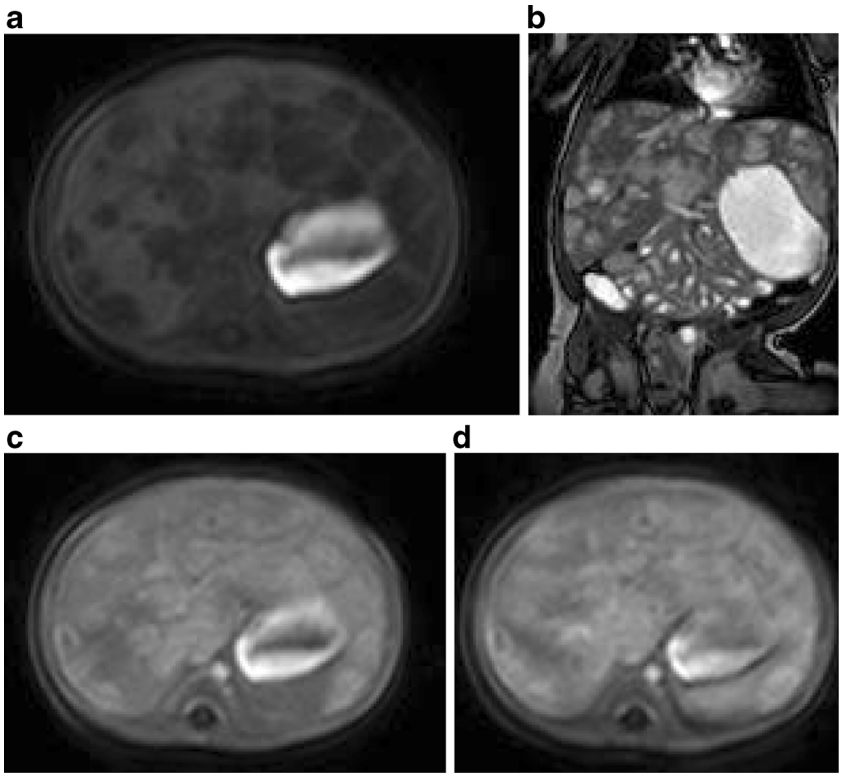


Fig. 5 Hemangioendothelioma: **a** T1 axial sequence shows multiple rounded and oval hypointense lesions in both lobes of liver. **b** STIR coronal sequence shows multiple iso - hypoechoic lesions in both lobes of liver. **c** T1 vibe fat sat axial sequence in arterial phase shows peripheral intense ring enhancement of the liver lesions. **d** T1 vibe fat sat axial sequence in portal venous phase shows variable fill in of contrast in the liver lesions

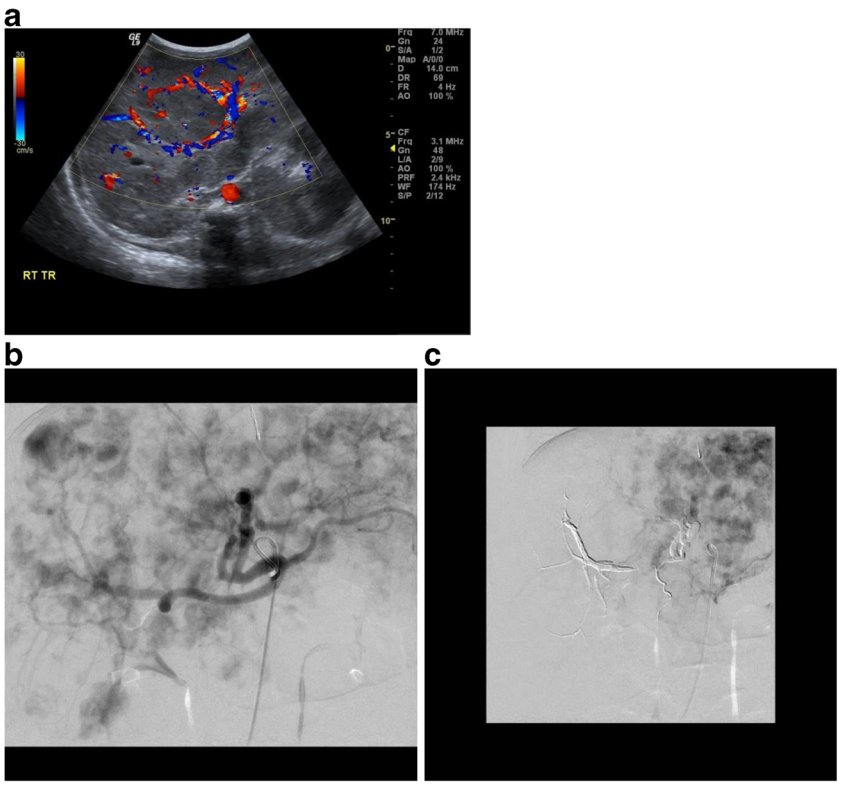


Fig. 6 Hemangioendothelioma: **a** Colour Doppler ultrasound of the liver shows multiple oval iso to hyper echoic lesions with increased vascularity. **b** Angiogram of hepatic artery reveals increased vascular recruitment by the liver lesions. **c** Angiogram of hepatic artery following coil embolization reveals reduced vascularity by the liver lesions

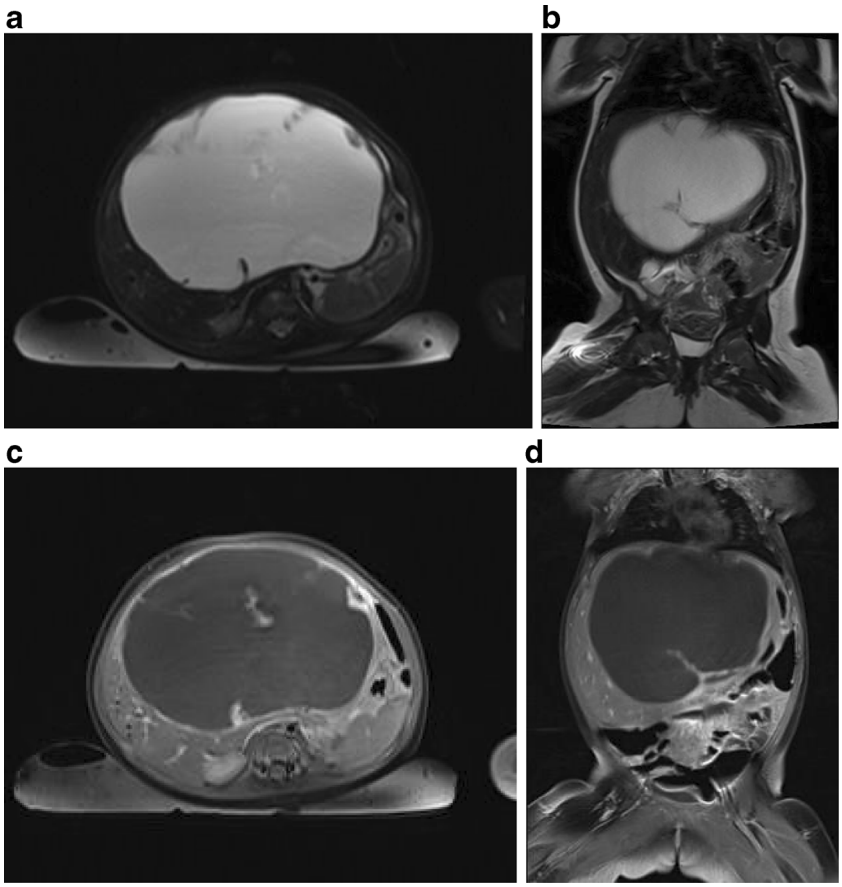


Fig. 7 Mesenchymal Hamartoma: **a** & **b** STIR axial and coronal sequences of liver demonstrate a large cyst occupying the right lobe of liver with incomplete internal septae. **c** & **d** T1 fat sat post intravenous gadolinium axial and coronal sequence of the liver show non-enhancing centre, enhancing peripheral rim and enhancing incomplete internal septae

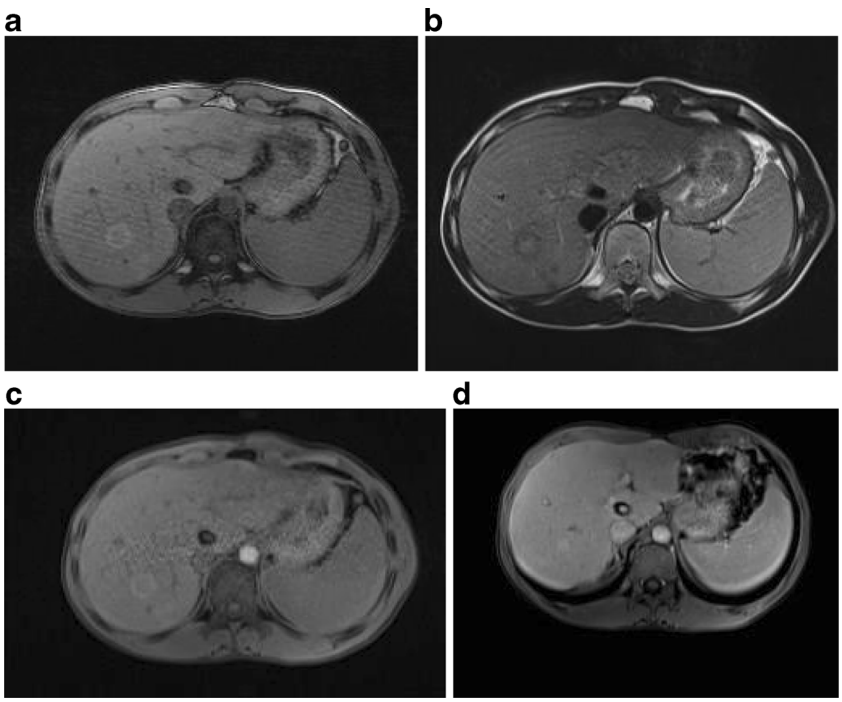


Fig. 8 Nodular regenerative hyperplasia: **a** T1 axial sequence shows round lesion with iso to hyper intense rim with hypo intense centre. **b** T2 axial sequence shows round lesion with iso to hypo intense rim with mildly hyperintense centre. **c** T1 VIBE axial fat sat post intravenous Gadolinium sequence arterial phase shows round lesion with enhancing rim and non-enhancing centre. **d** T1 VIBE axial fat sat post intravenous Gadolinium sequence late venous phase shows round lesion iso to hyper to the rest of liver due to complete fill in of contrast

Table 3 Malignant liver lesions [1, 8, 11–19]

	Age at presentation	Gender M: F	Symptoms	US	CT	MRI
Hepatoblastoma [8] (Fig. 3)	0–3 y	1.4–2:1	Abdominal mass, hepatomegaly, abdominal swelling, acute abdomen, fever, hirsutism, thrombocytosis	Well-delineated, septa, heterogeneous, hypoechoic (cysts, necrosis), calcification	Epithelial type: homogenous; mixed type: heterogeneous, peripheral rim enhancement, septa, calcification	Epithelial type: T1 hypointense, T2 hyperintense; mixed type: heterogeneous intensity on T1/T2. (Fig. 3)
Hepatocellular carcinoma [8]	10–12 y	M = F	Tender hepatomegaly, pain, anorexia, jaundice, fever, hemoperitoneum (rupture)	Hypoechoic, halo sign, diffuse: disorganization of normal pattern	Hypodense, calcification, hemorrhage, fatty metamorphosis, peripheral enhancement	T1: hypo- to hyperintense (due to necrosis, hemorrhage, septa, fat); T2: hyperintense
Undifferentiated embryonal sarcoma [18] (Fig. 4)	6–12 y	M = F	Abdominal mass, hepatomegaly, pain, fever, jaundice, rupture	Cystic + multiseptated heterogenous calcification	Hypodense septa, calcification, peripheral rim enhancement	T1: hypointense T2: hyperintense, hemorrhage, peripheral rim, septa hypointense on T1/T2. (Fig. 4)
Rhabdoid tumor	0–3 y	M > F	Abdominal mass, thrombocytosis, anemia or spontaneous rupture	Single or multiple hypoechoic lesions	Single or multiple hypodense lesions on CECT	Single or multiple T2 heterointense lesions
Metastatic neuroblastoma [8]	0–8 y	M = F	Abdominal mass, respiratory compromise, abdominal compartment syndrome	Multiple hypoechoic lesions	Multiple hypodense lesions on CECT	Single or multiple T2 heterointense lesions
Embryonal rhabdomyosarcoma [8]	3–5 y	Not known	Jaundice, abdominal distension or occasionally pain, nausea, vomiting, and fever	Biliary duct dilatation with an echogenic intraductal mass	Intraductal mass with variable contrast enhancement	T1: hypointense, T2: hyperintense with intense, heterogeneous contrast enhancement
Angiosarcoma	Mean age 3.7 y	F > M	Rapid abdominal enlargement and jaundice	Heterogenic echotexture regions	CECT: peripheral filling followed by centripetal opacification of the lesion and subsequent puddling of the contrast unevenly throughout the tumor	Not described in literature

	Angiography	Tumor marker	Histo-pathology/Cytology/ Immunology	Treatment	Prognosis
Hepatoblastoma [8] (Fig. 3)	Malignant neovascularization, spoke-wheel pattern, vascular invasion	Raised AFP, βHCG, testosterone	Epithelial type or mixed epithelial and mesenchymal type or anaplastic	Combination of conventional surgical lobar resection, neo adjuvant chemotherapy, and liver transplantation, depending on whether the tumor can be excised or not	Poor in extended cases; better if chemotherapy and surgery is applicable
Hepatocellular carcinoma [8]	Malignant neovascularization, vascular invasion	Raised AFP, ferritin, CEA	Expansive, encapsulated with collapsed portal vein branches or infiltrative, non encapsulated with invasion of portal and hepatic veins	Complete surgical resection or transplantation of localized disease	Poor in extended cases; better if chemotherapy and surgery is applicable
Undifferentiated embryonal sarcoma [18] (Fig. 4)	Nonspecific, mostly hypovascular mass effect	None	Primitive undifferentiated stellate cells in whorls/sheets with foci of hematopoiesis	Adjuvant chemotherapy, radiotherapy and surgical resection	Poor
Rhabdoid tumor	Not described in literature	Raised LDH	Round cells with abundant eosinophilic cytoplasm. Immunohistochemistry studies show abnormally low INI1/BAF 47 protein	Multi drug chemotherapy	Very poor

Table 3 (continued)

	Angiography	Tumor marker	Histo-pathology/Cytology/ Immunology	Treatment	Prognosis
Metastatic neuroblastoma [8]	Not described in literature	Raised catecholamines	Small round cells with scant cytoplasm. Horner-wright rosettes	Chemotherapy and liver radiation. Surgical abdominal decompression for abdominal compartment syndrome	Good
Embryonal rhabdomyosarcoma [8]	Not described in literature	Elevated conjugated bilirubin and alkaline phosphatase	Embryonal subtype with characteristic cross striations	Multiagent neoadjuvant chemotherapy with surgical resection and radiotherapy	Good for localized disease
Angiosarcoma	Not described in literature	None	Vessels lined with malignant endothelial cells	Multiagent neoadjuvant chemotherapy with surgical resection and radiotherapy, liver transplant	Poor

US Ultrasound; *CT* Computerized axial tomography; *MRI* Magnetic resonance imaging; *AFP* Alpha feto protein; *CEA* Carcino embryonic antigen; *βHCG* Beta human chorionic gonadotropin; *CECT* Contrast enhanced computerized axial tomography; *LDH* Lactate dehydrogenase

Histological Diagnosis

Needle biopsies combined with histopathology and immunohistochemistry can be definitive for evaluating patients with discrete hepatic masses. However, liver biopsy poses a number of diagnostic challenges [3]. The incidence of complications after percutaneous liver biopsy reported in pediatric patients is 6.83 %, of which 2.4 % were major complications. Correlation with clinical, radiological, and cytological findings is helpful in arriving at the correct diagnosis and therefore increases overall accuracy and cost effectiveness of the procedure [10]. Common space occupying lesions of liver are tabulated in Table 2.

The imaging algorithms useful to diagnose hepatic lesions in children are given as Figs. 1 and 2 [7].

The malignant liver lesions are enlisted in Table 3. Table 4 enlists classifications of the clinical categories of infantile hemangiomas of the liver. Table 5 provides list of benign lesions of the liver.

Conclusions

The diagnosis of pediatric liver lesions is made on the basis of clinical features, serum α-fetoprotein (AFP) level, age of the child, and imaging characteristics. The role of imaging is to determine the organ of origin and the character and extent of the lesion. Knowledge of pediatric liver diseases and their imaging appearances is essential in order to make an appropriate differential diagnosis. Challenges exist for the non-invasive detection and characterization of focal liver lesions (FLLs) in the pediatric population. The selection of the appropriate imaging test depends on a number of factors, such as:

(1) Children require imaging strategies with a higher resolution due to smaller anatomic structures [9].
(2) Children may be unable to tolerate or hold still for an imaging test, so they may require sedation or anesthesia [9].
(3) The use of imaging tests with ionizing radiation should be minimized given that children are more sensitive to the long-term effects of radiation exposure than adults [9].

Differentiation of masses is still complex, and biopsy or resection for histological diagnosis sometimes becomes necessary. The incidence of complications after percutaneous liver biopsy in pediatric patients was 6.83 %, of which 2.4 % were major complications.

Acknowledgments Education Centre, Birmingham Children's Hospital NHS Foundation Trust.

Table 4 Classifications of the clinical categories of infantile hemangiomas of liver [2, 3, 7–9, 11, 12, 14, 17, 19–22] (Figs. 5 and 6)

Type of malformation	Age at presentation	Gender	Clinical presentation	US	CT	MRI	Angiography	GLUT1 reactivity	Pathologic features	Outcome
Multifocal	0–6 mo	F > M	Patients are usually asymptomatic, but some have CHF; manifests in first few months of life	Multiple well demarcated hypoechoic lesions. Enlarged hepatic artery and veins with multiple vascular channels noted on color doppler	NCCT – Multiple hypodense lesions with calcification CECT – Lesions enhance intensely and uniformly	T1: Hypointense T2: Hyperintense Dynamic contrast enhanced scan: Intense peripheral nodular enhancement with centripetal fill-in.	Dilated tortuous feeding arteries. Early draining veins.	GLUT1 positive	Small with no central necrosis	Proliferation followed by involution
Focal			Patients are usually symptomatic and may have CHF; manifests in perinatal period	Well-demarcated heteroechoic lesion. Enlarged hepatic artery and veins with multiple vascular channels noted on color doppler	NCCT – Heterodense lesion with calcification CECT – Intense peripheral nodular enhancement in arterial phase and centripetal enhancement in portal venous phase	T1: Hypo or heterointense T2: Heterointense Dynamic contrast enhanced scan: Intense peripheral nodular enhancement with centripetal fill-in	Dilated tortuous feeding arteries. Early draining veins. Large venous varices with anomalous draining veins.	GLUT1 negative	Large with central necrosis, hemor-rhage, or fibrosis	Involute by age 12–14 mo
Diffuse			Manifests with mass effect; patients may develop abdominal compartment syndrome or severe hypothyroidism; no CHF	Diffuse enlarged hypoechoic liver or multiple hypoechoic lesions	NCCT – Multiple hypodense lesions CECT – Multiple lesions with centripetal enhancement	T1: Hypointense T2: Hyperintense Dynamic contrast enhanced scan: Intense peripheral nodular enhancement with centripetal fill-in	Dilated tortuous feeding arteries. Early draining veins	Not yet established	Liver enlarged and replaced by masses	Complicated clinical course

US Ultrasound; *CT* Computerized axial tomography; *MRI* Magnetic resonance imaging; *NCCT* Non contrast enhanced computerized axial tomography; *CHF* Chronic heart failure; *CECT* Contrast enhanced computerized axial tomography

Table 5 Benign lesions of the liver (Other than above vascular) [1–3, 10–12, 16–19]

	Age at presentation	Gender M:F	Symptoms	US	CT	MRI	Angiography	Histopathology/ Cytology/ Immunology	Treatment	Prognosis
Mesenchymal hamartoma [8, 20] (Fig. 7)	0–10 y	M > F	Painless abdominal mass	Cystic type: hypo- or anechoic, septa; Solid type: heterogeneous	Cystic type: hypodense, septa; solid type: iso- or hyperdense, small cysts	Cystic type: T1, hetero- or hypointense; T2, hyperintense (Fig. 7)	Non specific hypo- or avascular, mass effect, stretching of vessels	Disordered arrangement of primitive fluid filled mesenchyme, bile ducts with cysts upto 14 cm.	Surgical resection, enucleation or marsupialization	Survival rate is high (90 %)
Focal nodular hyperplasia [8]	2–10 y	F > M	Usually asymptomatic, rarely abdominal mass/pain	Well-circumscribed, variable echogenicity, Stellate hyperechoic central scar. Spoke-wheel pattern on doppler	NCCT - Homogenous mass of slightly decreased density CECT - Transient hyperdensity on bolus injection followed rapidly by iso density. Delayed enhancement of central scar	T1-weighted images: Isointense/ slightly hypointense to the liver. Hypointense central scar. T2-weighted images: Isointense/ slightly hyperintense. Hyperintense central scar. Arterial phase Hyperintense lesion. Non-enhanced central scar. Portal venous phase: Slightly hyperintense/isointense Delayed phase: Enhancement of the central scar	Discretely marginated hypervascular mass. Decreased vascularity in central stellate scar.	Abnormally arranged hepatocytes, bile ducts, vascularised fibrous septa Kupffer cells. Portal triads, central vein absent	Resection of peduculated mass. Biopsy for extensive mass	Spontaneous involution to ongoing growth.
Hepatic adenoma [8]	10–16 y	F > M	Asymptomatic or present with abdominal pain or an acute large hemorrhage	Hypo-, iso- or hyperechoic. Color Doppler may demonstrate central vessels with a triphasic pattern or a continuous flat venous waveform with no central arterial flow	NCCT - discrete, hypodense lesion with a well-defined border. CECT - early arterial-phase enhancement and may become iso attenuating on the delayed images.	T1: hypo to hyperintense with uniform signal loss on out-of-phase T1 WI. T2: isointense to slightly hyperintense. IV Contrast - Gadolinium enhancement is maximal during the arterial phase with rapid fading in the venous phase. Most of the lesions show no uptake of super magnetic iron oxide (SPIO) due to inactive Kupffer cells within the lesions. Hypointense compared with liver parenchyma on delayed MR images after injection of the hepatobiliary contrast agent Gd-BOPTA (gadobenate dimeglumine Multihance).	Hypervascular mass with centripetal flow	Benign hepatocytes with increased glycogen and fat	< 4 cm - Conservative > 4 cm. Surgical resection	Good
Nodular regenerative hyperplasia (NRH)(Fig. 8)	0–16 y	Not available	Asymptomatic or portal hypertension	Inapparent or heterogeneous echotexture or distortion of normal architecture. If visible, nodules are hypoechoic but may be hyperechoic compared with normal liver	NCCT – hypo or isodense CECT - Hypodense Occasionally, they may enhance diffusely or demonstrate peripheral rim-like enhancement	T1: slightly hyperintense to uninvolved liver T2: variable Decreased signal intensity on fat-suppressed T1-weighted images may be observed Post IV gadolinium the nodules may enhance preferentially in the portal venous phase like normal liver parenchyma. (Fig. 8)	Not available in literature	Hyperplastic hepatocytes	Treatment of portal hypertension	Good

US Ultrasound; *CT* Computerized axial tomography; *MRI* Magnetic resonance imaging; *NCCT* Non contrast enhanced computerized axial tomography; *CECT* Contrast enhanced computerized axial tomography

Compliance with Ethical Standards

Conflict of Interest None.

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