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ANAESTHETIC MANAGEMENT OF SURGICAL RESECTION OF GIANT HEPATIC HEMANGIOMA IN 12 YEAR OLD CHILD WITH LITERATURE REVIEW

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Abstract

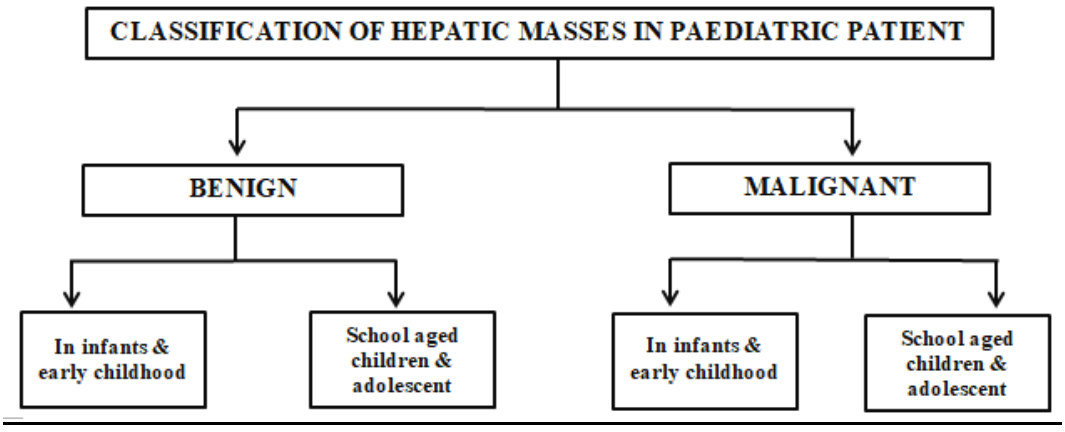
Hepatic Hemangioma are most common benign vascular hepatic tumor which accounts for 5-6% of all intra abdominal tumor.Most of the patients with H.H. are asymptomatic and frequently diagnosed as an incidental finding on imaging,shows female preponderance M:F=1:5.They range from small hemangioma to large cavernous hemangioma that involve the entire liver.Malignant transformation has not been described.Most common reason for patient with H.H. to undergo surgical resection is progressive abdominal pain due to increasing size of tumor & intratumoral bleeding.Its surgical resection is a complex procedure involving vascular structure & physiological derangement.Hepatic resection requires meticulous anaesthetic & surgical management with proper blood & its products replacement therapy.Coagulopathy,hepatic & renal failure & bile leak are some of the important complications of hepatic resection. We present a case of H.H. with successful surgical management.

Key words :- Hepatic hemangioma, Consumptive coagulopathy, Hepatectomy, Blood replacement, Progressive Intra-abdominal pain

INTRODUCTION

The liver accounts for 5-6% of all intra abdominal masses detected in children. Out of them 1/3 of hepatic masses are benign & 2/3 are malignant.

Clinical presentation - majority of hepatic masses present in a similar way with progressive abdominal distention, palpable abdominal mass, abdominal pain, hepatomegaly (liver enlargement).



Hepatic Hemangioma (HH)

It is also known as hepatic venous malformation or hepatic hemangio endothelioma, congenital in origin, almost associated with cavernous in type, non-neoplastic (benign) & vascular in nature.

Malignant transformation has not been reported in hepatic hemangioma.

Introduction-Over all most common benign solid tumor of the liver as well as most common benign vascular liver tumor in neonatal & early childhood period which is manifested in the 1st six month of life with rate of 85%.Its blood supply is predominantly hepatic arterial. Frequently diagnosed as an incidental finding on imaging.these are result of haematomatous proliferation of vascular endothelial cells. Hepatic hemangioma, cavernous hemangioma, angiosarcoma are the main vascular hepatic tumor which have been reported in infancy & young children.

Epidemiology

Much more common in female child (female predominance) M:F=1:5. Prevalence - 1-5% of all tumors in term neonates. Incidence- 0.5-1.5 case per million.

Clinical features of HH

Many HH are asymptomatic & remain undetected. Most of the HH manifested with palpable abdominal mass with progressive abdominal pain in the 1st six month of life and may accompany with cutaneous hemangioma.

Progressive Abdominal Pain	Palpable Abdominal Mass	Abdominal Distension
Hepatomegaly	Abdominal Compartment Syndrome	Vomiting
GI Tract Obstruction	Congenital Heart Failure Due To High Volume Arteriovenous Shunt	Intra Abdominal Bleeding
Cholelithiasis	Kasabach Merritt Syndrome	Anemia, Restlessness, Respiratory Distress
Consumptive Coagulopathy	Thrombocytopenia	

CLASSIFICATION OF HEPATIC HEMANGIOMA	
Congenital HH	**Infantile HH**
1. Rapidly Involuting Congenital Hemangioma (RICH) 2. Non Involuting Congenital Hemangioma (NICH) 3. Partially Involuting Congenital Hemangioma (PICH)	1. Focal HH (FHH) 2. Multi Focal HH (MHH) 3. Diffuse HH (DHH)

Congenital HH

Develops prenatally & fully grown at birth. It comprises of 3% of all infantile tumors & much more rare than I.H.H.

Infantile Hepatic Hemangioma (IHH)

Most of the IHH are clinically silent & eventually resolve spontaneously without any incident which is most commonly found in peripheral location within the liver. All subtypes of IHH may present asymptomatically or abdominal distension with progressive abdominal pain. Other features are hepatomegaly, self limiting anemia and thrombocytopenia. FHH (like CHH) may develop prenatally & may cause fetal cardiomegaly, fetal cardiac failure, hydrops fetails & cardiac insufficiency.

Conditions Associated with IHH

- Cutaneous hemangioma
- Extrahepatic hemangiomata
- Hereditary haemorrhagic telangiectacia (osler weber rendu disease)
- Kasabach merritt syndrome with giant hemangioma
- Hepatic arterio portal shunts

Multifocal and diffuse HH

Both subtypes of IHH is a part of spectrum of disease.lateraly MHH usually progress into DH. DHH usually present near complete displacement of liver parenchyma & associated with high mortality & morbidity.

Complications Associated with IHH (Mainly DHH)

- Massive hepatomegaly with fulminant hepatic failure.
- Compression of surrounding organs & vasculature.
- Abdominal compartment syndrome and Life threatening complications.
- High output congestive heart failure
- Profound consumptive hypothyroidism, secondary to over production of type-3 iodothyronine deiodinase which deactivates thyroid hormones (T3 & T4).
- Multi system organ failure (in late stage)

DIFFERENTIAL DIAGNOSIS OF HH

1) Focal Hepatic Steatosis
2) **Hepatic Metastasis** :- Hypervascular hepatic metastasis shows marked early enhancement with continuous ring & on delayed phases it shows progressive centripetal filling.
3) Hepatocellular Carcinoma (HCC)
4) Hepatic Abscess
5) Regenerative Hepatic Nodule/Dysplastic Hepatic Nodule
6) Cystic Hepatic or Biliary Neoplasm
7) Hemangio Endothelioma
8) Hepatic Cyst

Diagnosis of HH

Radiological Imaging :- USG,CEUS,CT SCAN & MRI are commonly used to diagnose.

A) Ultrasound (USG with doppler) :-

- Most commonly used screening test for initial work up of hepatic masses(HH) and to characterize the consistency of hepatic mass as cystic or solid.

USG findings with doppler:-

It shows typical well defined hyperechoic lesions which demonstrate high flow signals & peripheral arterial feeding vessels & direct shunts to the hepatic veins (specially in CHH). USG is also used prenataly to detect fetal abdominal mass during pregnancy.

B) Contrast Enhanced Ultra Sonography (CEUS) :-

HH shows characteristic rapid filling phase of hemangioma which is typically completed at the end of arterial phase or by the beginning of the venous phase.IHH classically appears as an isoenhanced or mildly hyperenhanced image in comparison to normal surrounding liver parenchyma.

Appearance of IHH in different phases in CEUS :-

1. **Arterial Phase** :- Peripheral nodular discontinuous enhancement.
2. **Portal Venous & delayed phases :-** Continued "filling in " of the lesion until the entire hemangioma is hyperechoic , relative to surrounding liver parenchyma.
3. Non enhancing central haemorrhagic portion seen in case of cavernous hemangioma.

CT Scan (CECT) :-

Most of the HH are relatively well defined & its typical features are

1. **Non Contrast Phase :-** Appears as a homogenous hypoattenuating (hypoenhancing) (< 20 Hounsfield units) area relative to surrounding liver parenchyma.
2. **Late Arterial Phase : -** Typically shows dis continuous nodular peripheral enhancement
3. **Porto Venous Phase:-** Progressive peripheral enhancement with more centripetal fill in.
4. **Delayed Phase :-** Further irregular fill in, therefore iso or hyper attenuating (hyper enhancing) lesion relative to surrounding liver parenchyma.
5. **Bright Dot Sign**

MRI :-

It accurately demonstrates a lesion because of high spatial resolution. Typical findings of HH on MRI includes clear round mass which appear as a

T1 weighted Image :- Hypo intense lesion relative to surrounding liver parenchyma.

T2 Weighted Image :- Hyper intense lesion relative to surrounding hepatic parenchyma, but less intense than that of intensity of CSF or hepatic cyst.

T1C + (Gd) :- Peripheral nodular discontinuous enhancement which progress centripetally (inwardly) on delayed images.

T1C + Hepatobilliary Contrast :- Hyperintense on diffusion weighted imaging even with high b value due to slow blood flow.

If diagnostic finding and imaging picture of HH are not typical then following further examination should be considered-

Type of HH	Finding on U.S.G.	Finding on C.E.U.S.	Finding on C.E.C.T.	Finding on M.R.I.
I.H.H.	Typical well defined hyperechoic lesions which demonstrate high flow signals & peripheral arterial feeding vessels & direct shunts to the hepatic veins	Classically appears as an isoenhanced or mildly hyperenhanced image in comparison to normal surrounding liver parenchyma.	Appears as a solitary spherical tumor with robust & rapid enhancement with presence of calcification & central cystic changes	Appear as a solitary spherical tumor with robust & rapid enhancement with presence of calcification & central cystic changes
Multifocal H.H.		Appears as a multiple discrete masses that as hypoechoic or mixed echogenicity	Appears as a hypodense lesion on CT that has uniform or centripetal enhancement	Appear as a homogenously enhancing spherical mass that are hypo intense on T1 imaging & hyper intense on T2 imaging
Diffuse H.H.		Appears as a innumerable centripetally enhancing lesions.	Appears as a innumerable centripetally enhancing lesions that almost completely replace the normal hepatic parenchyma.	Appear as a hypo intense on T1 weighted imaging & have a prominent flow void on T2 imaging.

Spect :- Tc-99m RBC's labelled SPECT can be sensitive for larger size hemangioma & typically demonstrate decreased activity on delayed blood pool changes.

LAPAROSCOPIC EXPLORATION

Biopsy :-

Gold standard method to confirm the diagnosis of H.H. or any hepatic mass but liver biopsy is generally not recommended because it may lead to life threatening haemorrhage or massive bleeding.

Gross appearance of IHH :-

- FHH- Large tumor with presence of central necrosis, haemorrhage or fibrosis.
- MHH- Small tumor with no central necrosis
- DHH - characteristically replace the normal liver parenchyma.

Histopathology :-

- IHH are histologically similar to CHH, with fibrous stroma interposed by thin vascular channels lined with endothelial cells.

Management for HH :-

The general natural history of HH shows that small and asymptomatic lesion in children may heal spontaneously, while large and symptomatic lesion requires active treatment to prevent serious complications.

1) **Drugs used in management of HH :-**
   Propranolol (non selective beta blocker & first line treatment for both cutaneous & hepatic hemangioma), **Corticosteroids,** drugs with anti angiogenic effects (**IFN Alpha, Cyclophosphamide**, **Vincristine, Actinomycin-D) Bevacizumab** (anti VEGF monoclonal antibody) **Sorafenib** (multi kinase inhibitor). Last two drugs has been recently used to shrink the size of tumor.
2) **Radiotherapy – Hepatic Irradiation :-** Dose 15-30 Gy in 15-22 fractions over several weeks
3) **Arterial Embolization/Hepatic Artery Embolization -**
4) **Radio Frequency Ablation :-** Both percutaneous and laparoscopic RFA have been used to improve abdominal pain in patients with symptomatic hepatic hemangiomas.
5) **Surgical Ligation of feeding vessels :-** Performed by selective location of large feeding vessels via transhepatic compression sutures using poly tetra floro ethylene (PTFE) pledgets. This technique reduces the intratumoral shunting.
6) **Surgical Resection/Enucleation/Hepatic Lobectomy :-** Most confirmatory treatment of giant H.H.

Indication :-

1) Spontaneously ruptured hemangioma.
2) Large symptomatic hemangioma
3) Rapidly growing hepatic hemangioma
4) When HH cannot be differentiated from hepatic malignancy on imaging studies.

Types of Surgery:- Surgical resection, Enucleation, Hepatic Lobectomy (in large reasons)

7) Orthotopic Liver Transplantation :-

Anaesthetic Management of Giant HH :-

Large hemangioma will develop symptoms & complications that require prompt Surgical intervention. Hepatic resection is a complex procedure which involves vascular structures & physiological derangements. It requires meticulous anaesthetic & surgical management & proper blood replacement therapy. Surgical resection (Hepatectomy) was planned under general anaesthesia with controlled ventilation.

Major anaesthetic concerns during surgical resection of HH (Specially during partial hepatectomy) include & its management

Anaesthetic Considerations	Management & Preventive Measures
1. • Hemodynamic Instability • Hemorrhage/massive bleeding/increase risk of bleeding • Consumptive thrombocytopenia • Coagulopathy/Coagulation dysfunction	• Preoperative arrangement of blood & blood products, 3 units PCV/PRBC, 3 units of FFP, 3 units of RDP/2 units of SDP, 1 unit of cryoprecipitate. • Establishment of IV access with two 22 G cannula on B/L forearm Establishment of right femoral artery cannulation for continuous BP monitoring • Establishment of right IJV cannulation for continuous CVP monitoring & to deliver inotropes if required.CVP pressure was kept < 5 cm H2O to reduce the risk of bleeding. • Epidural was deferred. Due to risk of the coagulopathy. • Avoid hypothermia, acidosis, hypocalcemia - because these conditions increases the risk of bleeding • To maintain BP , U/O - by crystalloids, colloids , blood and blood products
2. Increased risk of consumptive hypothyroidism	• IV administration of triiodothyroine as a therapeutic modality to stabilize a hypothyroid children prior to surgery of HH under GA.
3. Increase risk of myocardial depression secondary to hypothyroidism	• Avoid Cardiac Depressant Drugs
4. Increase risk of Sepsis/septicaemia	• Administration of antibiotic prophylaxis
5. Increase risk of hypothermia	• Use of in line fluid warmers & forced air warmers.
6. Increase risk of metabolic acidosis, prolonged duration	•

DISCUSSION

Hepatic resection is indicated in several benign & malignant liver masses. The **Surgical Aim** is to excise the diseased area of liver with minimal blood loss and preservation of adequate healthy liver & **Anaesthetic Consideration** is to maintain patients hemodynamic stability by maintaining the BP & urine output & to prevent metabolic acidosis, Intraoperative hypotension, hypothermia, septicemia, bleeding.

During the initial step of mobilizing the liver, there may be sudden fall in cardiac output & CVP.

During isolation of portal vein and Hepatic artery there may be massive hemorrhage & Use of vascular occlusion techniques will minimize the intraoperative bleeding during liver resection & providing hemodynamic stability which will allow optimal surgical results such as.

Pringle Maneuver provides total occlusion of inflow of the portal vein & Hepatic artery which decreases the cardiac output by 10% & increases the left ventricular afterload by 20-30 % & **Low CVP (0-5% H_2O)** reduces the Hepatic venous congestion but low CVP during Hepatic parenchymal resection may predispose to venous air embolism.

Meticulous plan of Anaesthesia, fluid administration, good knowledge of surgical techniques & anticipated complications with vast discussions with the surgeon are essential during surgical resection of any large H.H. Any intraoperative fall in BP must be rapidly treated to preserve blood flow of liver and minimize the postoperative

hepatic dysfunction. **N_2O should be avoided** to prevent gut distension & risk of air embolism. **Invasive Hemodynamic Monitoring** should be instituted to continuous real time BP & ABG monitoring. **Noninvasive cardiac output Monitoring & Coagulation Monitoring** would be beneficial in case of dynamic hemodynamic changes & requirement of massive blood transfusion. Complications of acidosis, hypothermia, hypocalcemia should be monitored, prevented & meticulously treated if present. The common postoperative complications are **coagulopathy, renal dysfunction, liver dysfunction, intra abdominal infection, sepsis, pulmonary complication.** Epidural analgesia usually **avoided** due to increased risk of spinal and epidural hematoma as a result of post operative Hepatic dysfunction induce coagulopathy.

CONCLUSION

Appropriate imaging strategies are vital for the diagnosis of H.H. Adequate treatment time is necessary to cure the disease such as. Drugs, embolization & surgical resection are therapeutic options for H.H. Partial hepatectomy due to surgical resection of giant H.H causes significant morbidity and mortality due to physiological & biochemical changes that occur in the perioperative period. The post operative management should be started with detailed preoperative assessment which involve both the physical & physiological aspects of the patient till the post operative rehabilitation and pain management.

CASE REPORT

A 12 year old male child presented to the paediatric department with complain of progressive abdominal pain & palpable mass in right upper quadrant of abdomen since 20-25 days. CECT showed left hepatic lobe occupancy, he was diagnosed to have a huge left hepatic hemangioma. He was planned for **left hepatectomy for removal of hemangioma under G.A. with controlled ventilation. During pre anesthetic work up** he had no known comorbidities except hypothyroidism & was on tablet thyroxine 25 mcg per day. He was average built, weight 32kg, on examination. Pre-operative thyroid function test, ECG, CHEST X-RAY, 2D ECHO were within normal limits. He belonged to ASA Physical status -2 & MET's > 4. A written informed high risk consent was obtained from his parents. Pre operatively 3 units of PCV, 3 UNITS OF FFP, 3 Units of platelets & 1 unit of cryoprecipitate were arranged. **Epidural was deferred due to fear of coagulopathy.**

Intravenous access was established with two 22 G cannula on right and left forearm. Antibiotic prophylaxis was administered. Trachea was intubated with 6.0 mm ID cuffed endotracheal tube after induction by using **Inj Midazolam** 0.05mg/kg, **Fentanyl** 2mcg/kg, **Glycopyrrolate** 5mcg/kg, **Ketamine** 1mg/kg, **Propofol** 1.5mg/kg, **Atracurium** 0.5mg/kg with **Sevoflurane & O_2** Anaesthesia was maintained with **Oxygen & Air (50:50) Mixture, Sevoflurane, Atracurium Boluses** (0.1mg/kg), **Inj Fentanyl** was supplemented 0.5mcg/kg every 45 minutes. **Right Femoral Artery Cannulation** for continuous BP monitoring & **Right Internal Jugular Vein Cannulation** for continuous CVP monitoring & to deliver inotropes were established. Plan is to maintain **CVP < 5cm H_2O** throughout the surgery, **Urine Output of 1mg/kg/hour** & to do serial blood sugar and arterial blood gas (ABG) monitoring.

When resection of the left lobe started, surgeon requested to decrease BP & CVP to reduce bleeding. In order to reduce BP & CVP we started **Propofol Infusion** at the rate **8-12 mg/hour** and sevoflurane titration, BP was kept around 90-100/50-60 mm Hg and CVP pressure was kept below 5cm of H_2O. During the period of hepatic resection, there was excessive bleeding. The BP and urine output were maintained with crystalloids, colloids and blood products. Intra operatively, 3 serial ABG were done to look for metabolic acidosis and coagulation profile. IV line warmers and forced air warmers were used to prevent hypothermia. During intra operative period SBP remained between 92-116mm Hg & DBP remained between 64-86mm Hg with adequate volume infusion, phenylephrine boluses, inotropes (inj noradrenaline infusion 0.15mcg/kg/min & inj dopamine at 2.5mcg/kg/min infusion.

After resection of liver parenchyma, intrahepatic bile ducts & oozing vessels were ligated. During the 5 hours of surgery the patient received 1500 ml crystalloids, 3 units of PCV, 4 units of FFP, 3 units of RDP, 350 ml of colloid, 1 unit of cryoprecipitate. **Sodium Bicarbonate & Calcium Gluconate** were given as a divided boluses to correct metabolic acidosis and to increase ionic calcium. Total blood loss was estimated to be 1200 ml and urine output of 500 ml measured. Abdomen was closed in layers after placing portex drains.

BP remained at 94-116/62-88 mm Hg and heart rate 100-130/ min till patient was shifted to post anesthesia care unit. Post operatively the patient was electively ventilated overnight in view of metabolic acidosis, prolonged duration of surgery and excessive volume shifts. **Fentanyl Infusion** was started at the rate of 10 mcg/hour for post-operative pain relief. On first post- operative day patient was extubated after thoroughly suctioning done. Nebulization, spirometry & VTE prophylaxis (mechanical) were started. He required **Dopamine** 2.5mcg/kg/min and **Noradrenaline** 0.1mcg/kg for first 24 hours. Inotropic support were tappered and discontinued on post- operative day 2. He maintained urine output at the rate of **1ml/kg/hr.** His liver enzymes, platelet count and coagulation profile returned to normal by post-operative day 5. The patient was mobilized, started on oral feeds and shifted to ward on post-operative day 5. He was discharged on post-operative day 8 uneventfully.

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