


# Hepatocellular Carcinoma with Inferior Vena Cava Thrombus in a Child

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## ABSTRACT

Hepatocellular carcinoma (HCC) in children is the second common malignant liver tumor after hepatoblastoma. Chronic hepatitis B virus (HBV) infection causes most hepatocellular cancer worldwide. Metastases to the inferior vena cava (IVC) and right atrium (RA) tumor thrombi are even less common. We reported a case of a 13-year-8-month-old girl with HCC and IVC involvement. Vascular invasion predicts poor overall survival in HCC patients. Patients with HCC involving the IVC had a higher risk of sudden mortality and a poor treatment outcome. It is difficult to treat, and no standard therapy has been established. This case report aimed to describe HCC with IVC involvement. This is a rare disease with a poor prognosis, therefore it is important to detect and manage early.

**Keywords:** Hepatocellular carcinoma; inferior vena cava thrombus; pediatric

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## INTRODUCTION

Hepatic tumours account for about 1% of child malignancies, with hepatoblastoma (HB) and hepatocellular carcinoma (HCC) constituting the majority of the cases (Kelly et al., 2015). Paediatric HCC is the second common malignant liver tumour in children after HB (Khanna and Verma, 2018). The extension of the tumor into a blood vessel is defined as an intravascular tumor thrombus (Quencer et al., 2017). Inferior vena cava (IVC) and right atrium (RA) tumor thrombi arising from HCC are rare, occurring in approximately 3-4% of HCC patients, including adult patients. (Luo et al., 2015; Aly et al., 2020). Data on tumour thrombus in paediatric HCC are minimal. We presented a child with HCC and IVC thrombus

## CASE REPORT

A 13-year-8-month-old girl was admitted to the paediatric ward with an abdominal mass (Figure 1) with a plan to do TACE therapy for a hepatoblastoma which had been diagnosed previously by abdominal ultrasonography. The child had a history of weight loss for one year and a rapidly enlarging abdominal mass over 6 months, accompanied by abdominal pain, recurrent vomiting, and lethargy. Family history of the child showed that her mother once developed icterus, abdominal pain, vomiting, and fever for about 5 days when the child was 4 years old.

Anthropometric examination on the first day of admission revealed that the patient's weight was 41 kg, height 156 cm and upper arm circumference (UAC) 16 cm. Physical examination revealed an ill-looking child with a blood pressure of 110/60 mmHg, a regular heart rate of 108 beats per minute and a respiratory rate of 28 times per

minute. The axillary temperature was 38.8 0C. The axillary temperature was 38.8 0C. There was no jaundice, no pale conjunctiva, no cyanosis and oxygen saturation was 98%. On abdominal examination, the liver was palpable under the costal margin 5cm x 4cm x 3cm with multiple palpable nodules; spleen was not palpable. There was ascites. The extremities were warm and there was no oedema. The capillary refill time (CRT) was <2 seconds. There were venae ectasia over the legs and back of the feet.

Laboratory finding revealed a haemoglobin (Hb) level of 10.7g/dl, a haematocrit (HCT) of 33.4%, a mean corpuscular volume (MCV) of 76.3fl, a mean corpuscular haemoglobin (MCH) of 27pg, a mean corpuscular haemoglobin concentration (MCHC) of 35.4g/dl, a total white blood cell (WBC) count of 14.640/cu mm, a differential count of 1% eosinophils, 0.03% basophils, 76.6% neutrophils, 15.6% lymphocytes and 6.4% monocytes and a platelet count of 676.000/cu mm. The prothrombin time (PT) was 12.2 seconds (normal range 9-12 seconds) and the activated partial thromboplastin time (APTT) was 42.4 seconds (normal range 23-33 seconds).

The serum sodium (Na) was 132 mmol/l, serum potassium (K) was 4.4 mmol/l, serum calcium (Ca) was 7.2 mg/dl and the serum chloride (Cl) was 85 mg/dl. The serum aspartate transaminase (AST) was 45 U/L (normal range 0-35 U/L) and the serum alanine transaminase (ALT) was 28 U/L (normal range 0-35 U/L). The serum albumin was 3.6 g/dl, blood urea nitrogen (BUN) was 5mg/dl and the serum creatinine was 0.44mg/dl. The hepatitis B surface antigen (HbsAg) was reactive. The lactate dehydrogenase (LDH) was 338 U/L, serum alkaline phosphatase (ALP) was 94IU/L and alpha fetoprotein (AFP) was 1439ng/ml.



Figure 1. Child with abdominal mass indicated by arrow.



Figure 2. Chest x-ray on child showing elevated right diaphragm. Heart and lung within normal limits.

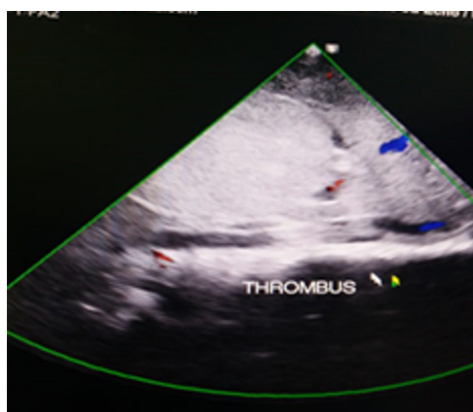


Figure 3. Echocardiography showing inferior vena cava thrombus.

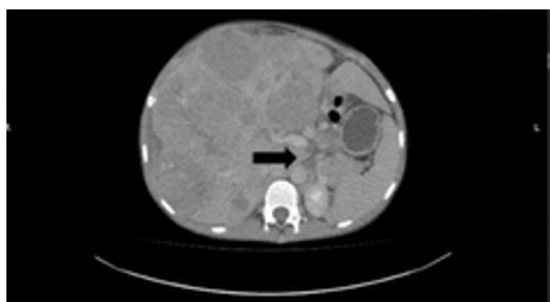


Figure 4. CT angiography showing inferior vena cava thrombus shown by arrow.

The chest x-ray of the child showed an elevated right diaphragm (Figure 2). Echocardiography showed the inferior vena cava thrombus (Figure 3). This was confirmed by computed tomography (CT) angiography (Figure 4).

## DISCUSSION

In this case, the history revealed that the child had lost body weight over the past year and that during the past 6 months the stomach mass had rapidly become larger and the child had complained of abdominal pain, nausea, vomiting, and lethargy. The history also revealed that the patient's mother had icterus, abdominal pain, vomiting, and fever for approximately 5 days when the child was 4 years old. However, the mother did not check her hepatitis B virus level. From the laboratory findings in the child there was positive HbsAg.

The level AFP of this patient was 1,439 ng/ml. In pediatric patients, HCC levels of AFP are high in 50-92% of cases (Czuderna et al., 2002; Głowska-Ciemny et al., 2023). In this case, the diagnosis of HCC was established based on history of chronic hepatitis B with HbsAg positive. Chronic hepatitis B virus (HBV) infection is a major global cause of hepatocellular carcinoma (HCC) (Takano et al., 2017). In this case, the IVC thrombus was found by abdominal CT angiography. Ultrasonography is the best initial imaging technique because it allows assessment of an intrahepatic mass, usually a large solitary mass. Vascular invasion can also be assessed by this modality. Computed tomographic scans define the extent of the tumour involvement throughout liver parenchyma and vessels as well as more distant metastatic disease (McAree et al., 2013).

A previous study stated that HCC in children tends to be more advanced, leading to poorer survival than adults (Wang et al., 2017). HCC with IVC involvement is a rare disease with poor prognosis. Vascular invasion is a prognostic factor for poor overall survival in patients with HCC. Patients with HCC involving IVC had an increased risk of sudden death and dismal treatment outcome, difficult to treat and a standard therapy has not been established. (Quencer et al., 2017). Surgery, TACE, and systemic treatment were adopted in the management of these cases (Huang et al., 2019; Zhang et al., 2021).

## SUMMARY

This case was confirmed as a HCC with IVC involvement based on clinical findings, laboratory, USG, and abdominal CT angiography. HCC with IVC involvement is an uncommon disease with a poor prognosis. The treatment is challenging, and a standard therapy has not yet been established. In the management of these cases, surgery, TACE, and systemic treatment were all opted for as treatment methods. The follow-up for this patient is still being carried out .

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None

## CONFLICT OF INTEREST

The authors declare there is no conflict of interest.

## PATIENT CONSENT FOR PUBLICATION

Case reports must include a letter of approval for publication from the patient and his/her guardian.

## FUNDING DISCLOSURE

None

## AUTHOR CONTRIBUTION

All authors have contributed to all process in this research, including preparation, data gathering and analysis, drafting and approval for publication of this manuscript.

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