A Unique Case of Cardiac *Echinococcus multilocularis*

Human alveolar echinococcosis (AE) or alveolar hydatid disease is extremely rare in children due to the prolonged incubation period of 5-15 years [1]. A tumor-like infiltrative growth characterizes it. Metacestodes of AE can infiltrate into adjacent areas resulting in its spread to different organs, primarily liver and lungs [1].

We report the case of a 7-year-old child from Iraq who presented with the complaints of cough and breathing difficulty, with progressive worsening over two months before presentation. Parents had also noticed increasing yellowish discolouration of eyes and skin, loss of appetite and weight in the previous month. The patient had a low-grade, intermittent fever for the past 20 days. The child had been diagnosed to be a case of hepatic failure and had been referred for liver transplant. On examination, the child had tachycardia, tachypnea and mild subcostal, intercostal retractions. Breath sounds were absent on the right side. There was non-tender hepatomegaly, with the liver span of 16 cm and smooth surface. Minimal ascites was present. Liver functions were deranged (serum glutamic oxaloacetic transaminase or SGOT/ serum glutamic pyruvic transaminase or SGPT 145 / 345 U/L, gamma-glutamyl transferase or GGT 528 U/L, total bilirubin and direct 6.4/2 mg/dL, total protein 8.1 g/dL, serum albumin 2.8 g/dL). The international normalized ratio (INR) was 1.37. He also had severe anemia (hemoglobin – 5.8 g/dL), with absolute eosinophil count of 2.45×10^9/L and high pro-inflammatory markers. Chest radiograph revealed right-sided pleural effusion with underlying collapse and consolidation. Pleural tap revealed almost bile-like pleural fluid with high bilirubin level suggestive of a trans-diaphragmatic extension of the hepatic disease. Evaluation of the fluid for infection was negative. Contrast-enhanced, multiphasic, multi-detector computed tomographic (MDCT) scan of abdomen revealed hepatomegaly with a large hypodense lesion in the liver, invading the inferior vena cava and serosa of the oesophagus with cystic changes, and was reported by the radiologist to possibly be mitotic etiology of the biliary tract or *Echinococcus alveolaris*. Qualitative Echinococcus (E) IgG was positive. Endoscopy revealed normal esophageal and gastric mucosa. Echocardiography demonstrated inferior vena cava infiltration by a mass extending into the right atrium. Ultrasound-guided liver biopsy revealed an inflammatory pathology with the possibility of mass forming *E. multilocularis*. The child was treated with 15 mg/kg/day divided in two doses of continuous albendazole therapy and other supportive treatment, and was under regular follow up. Unfortunately, the child died 2 months later.

Alveolar echinococcosis, due to *E. multilocularis* is extremely unusual, accounting for < 5% of all cases of hydatid liver disease and, less frequently, lung disease. The mean age of presentation is 55 years [1,2], with children being rarely affected. Liver is the primary site of cyst development in almost all patients. The characteristic feature of *E. multilocularis* is that they behave just like malignant tumors with invasion and destruction of surrounding tissue, spread into contiguous areas and metastasis to distant organs, with the most common organ being lung [3]. Lung manifestations always appear after the involvement of the liver [3]. Cardiac echinococcosis is very rare (0.03%-1% of all cases) [2], with the left ventricle being most frequently affected (55–60%).

We diagnosed our patient to be a confirmed case of alveolar echinococcosis based on clinical findings, contrast-enhanced MDCT, histopathology and serology [4]. We further classified the case as per the WHO-IWGE (WHO-Informal Working Group on Echinococcosis) PNM classification as P4N1M1 [5].

The focus of management in these patients is early diagnosis and radical (tumour-like) surgery, which is followed by anti-infective prophylaxis with benzimidazoles [1,3,4]. However, as in our case, most patients are diagnosed at an advanced stage, when radical surgery (a distance of larval to

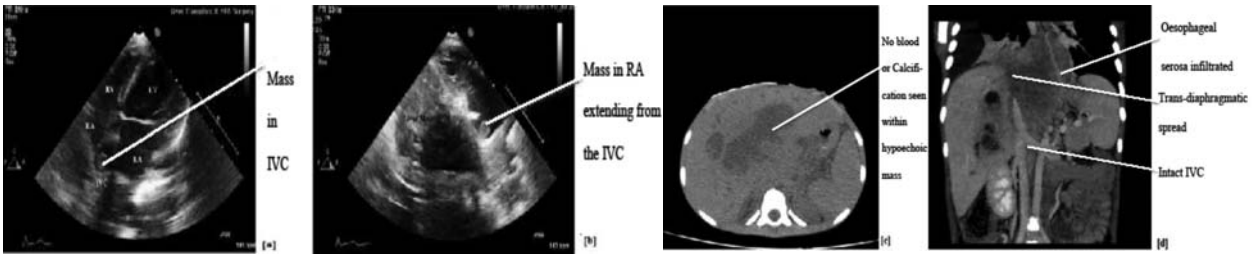

Fig. 1 *(a) 2-D Echocardiography (modified 4 chamber view) showing mass in IVC (b) Modified subcostal view with mass clearly seen in RA (c) CT: Nature of disease seen here – hypoechoic lesion with no blood/calcification within, unlikely to be carcinoma (d) Venous Phase of Triphasic CT: Trans diaphragmatic spread seen into the adjoining tissue, oesophageal serosa infiltrated. IVC, inferior vena cava; RA, right atrium; CT, computed tomography.*

liver tissue of >2 cm) cannot be achieved. Hence, as per current recommendations, the cornerstone of treatment remains the continuous medical treatment with albendazole, with individualized interventional measures at the appropriate time [1,4]. Radical surgery could not be done in our patient as R0 (no residue) resection was not possible. Palliative surgery was not possible as the lesion was unresectable due to invasion into the oesophagus, as well as into a blood vessel, leading to its spread to distant organs (both lungs and heart) [1]. Liver transplant was contraindicated due to the presence of extra-hepatic locations [1].

The first reported case of cardiac alveolar echinococcosis in adults, has been recently published [6]. In another interesting recent case report, E. granulosus causing cystic echinococcosis (CE) in left ventricle has been described in an 8-year-old child [2]. Yet another publication reports a giant hydatid cyst of the left ventricle in an 11-year-old child, also reviewing the 18 cases of cardiac echinococcosis reported thus far, all of which were due to cystic echinococcosis (CE) [7]. This is the first reported case of cardiac AE in children and highlights the need to consider this rare entity in patients with extensive liver disease extending into heart and lungs.

NEETU TALWAR,* NIKITA AGARWAL AND KRISHAN CHUGH
Division of Pediatric Pulmonology,
Fortis Memorial Research Institute,
Sector 44, Gurugram 122 002, Haryana, India.
**neetu.talwar1306@gmail.com*

REFERENCES

1. Bulakci M, Kartal MG, Yýlmaz S, *et al*. Multimodality imaging in diagnosis and management of alveolar echinococcosis. Diagn Interv Radiol. 2016; 22:247-56.
2. Su L, Yu J, Dai C, Liu Y, Peng L. Echinococcosis in left ventricle: a case report. Medicine (Baltimore). 2019;98:e15267.
3. Morar R, Feldman C. Pulmonary echinococcosis. Eur Respir J. 2003;21:1069-77.
4. Brunetti E, Kern P, Vuitton DA. Writing panel for the WHO-IWGE. Expert Consensus for the Diagnosis and Treatment of Cystic and Alveolar Echinococcosis in Humans. Acta Trop. 2010;114:1-16.
5. Kern P, Wen H, Sato N, *et al*. WHO classification of alveolar echinococcosis: Principles and application. Parasitol Int. 2006;55:S283-87.
6. Zhang X, Wei X, Ran L, Tang H. A rare case of cardiac alveolar echinococcosis. Eur Heart J. 2020;41:2698.
7. Fiengo L, Bucci F, Giannotti D, Patrizi G, Redler A, Kucukaksu DS. Giant cardiac hydatid cyst in children: Case report and review of the literature. Clin Med Insights Case Rep. 2014;7:111-16.

Deep Vein Thrombosis After Trivial Blunt Trauma at High Altitude in a SARS-CoV-2 Positive Child: Complication of the Hypercoagulable State

Deep venous thrombosis and spontaneous thrombosis have previously been reported among patients infected with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) as a sequelae of hypercoagulable state [1,2]. We report the clinical course of coronavirus disease 2019 (COVID-19) in a 14-year-old boy living at high altitude whose manifestations could primarily be attributed to this hypercoagulable state.

A 14-year-old previously healthy boy, native of high altitude, presented with left thigh swelling for 1 week and breathlessness, chest pain, cough, fever and poor urine output for 5 days following trivial blunt trauma. The thigh trauma had occurred after jumping from a height of around three meters. This child belonged to a COVID-19 containment zone which was located at an altitude of 8000 feet above sea level. He had no significant past or family history suggestive of thrombo-embolism or bleeding disorders. He had no external injury or bleeding after the trauma but had tenderness at the thigh and difficulty in walking. On examination he was sick, lethargic, and febrile with PR=120/min with low volume pulse, respiratory rate of 32/minute, SpO_2 at room air of 78%, blood pressure of 80/60 mm Hg. Chest auscultation revealed bilateral crackles. There was left thigh swelling with tenderness and restriction of movement at the knee and rest of the clinical examination was normal.

Initial *X*-ray thigh was normal and did not reveal any fracture. Doppler ultrasound thigh revealed left common femoral vein thrombus measuring 12.56 cm × 0.79 cm, which was non-compressible with no Doppler flow. The thrombus extended into the left saphenous vein. Chest *X*-ray showed bilateral fluffy shadows. Treatment for suspected SARS-CoV-2 infection was immediately started. High flow oxygen via nasal cannula at 8 liters per minute was initiated. Fluid bolus with normal saline at 20 mL/kg once was given over one hour followed by maintenance intravenous fluid. Intravenous broad spectrum antibiotics and injection dexamethasone 6 mg once daily were started. In view of suspicion of COVID-19 with a differential diagnosis of traumatic deep vein thrombosis with pulmonary thromboembolism, initial treatment comprised of oral hydroxychloroquine, acetylsalicylic acid (anti-platelet dose), and injection low molecular weight heparin (LMWH) 40 mg subcutaneous twice daily. His hemodynamic status improved with fluid resuscitation and he did not require inotropic support. Preliminary investigations showed hemoglobin of 13.3 g/dL, total leucocyte count of 11×10^9/L (polymorphs 84%, lymphocytes 12%), and platelet count of 398×10^9/L. CRP was positive. Blood urea (279 mg/dL) and