

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.

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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, SpO₂ 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 11×10⁹/L (polymorphs 84%, lymphocytes 12%), and platelet count of 398×10⁹/L. CRP was positive. Blood urea (279 mg/dL) and

serum creatinine (4.7 mg/dL) were raised, with normal serum electrolytes. Prothrombin time was 16 sec with INR 1.6, and activated partial thromboplastin time (APTT) was 17 second. Liver function test was normal. His nasopharyngeal swab RT-PCR for SARS-CoV-2 was positive on day two of admission and he was shifted to the district Covid-hospital. Over the next few days, his respiratory status initially improved and oxygen flow was gradually reduced.

From the second week of illness, the patient developed repeated episodes of hemoptysis and occasional epistaxis and required blood transfusion for symptomatic anemia with hemoglobin dropping to 7.5 g/dL. His PT/INR and aPTT remained normal during this period, and anti-factor Xa was not done. Pulmonary thromboembolism was clinically suspected as the etiology of hemoptysis in the setting of the COVID-19 and DVT. Patient's repeat nasopharyngeal RT-PCR sample tested negative for SARS-CoV-2 on day 10 and rapid antigen test was also negative. Hence, he was shifted back to our center.

High-resolution computed tomography (HRCT) scan of chest could only be done on day 11 of the hospitalization and revealed multiple bilateral nodular parenchymal opacities with areas of cavitation seen in bilateral lung fields (suggestive of septic emboli) with bilateral pleural effusion (left more than right). Repeat HRCT chest after four days reported bilateral nodular shadowing with multiple cystic bronchiectasis changes in both lung fields, more in upper lobes. Echocardiogram was reported normal. The patient's renal function recovered after the initial fluid resuscitation and did not require dialysis. Other investigations like blood culture, D-dimer, ferritin, IL-6, protein C and S, Factor V Leiden *etc.* could not be done due to non-availability at the facility. From day 20 of admission, his oxygen saturation remained greater than 90% at room air. Repeat USG thigh showed resolution of DVT. Both dexamethasone and LMWH were given for 10 days each. Oral warfarin was started after ceasing heparin but was stopped after onset of repeated hemoptysis. From the third week, he again developed high fever and the thigh swelling worsened. X-ray left femur demonstrated signs of acute osteomyelitis of the left femur. Antibiotics were upgraded and pus was drained from the thigh. Pus culture was sterile, as the patient was already on antibiotics. After two

weeks of surgical drainage, he became afebrile and was discharged after 40 days of total hospitalization.

In addition to primary lung involvement due to COVID-19, this patient developed a hypercoagulable state with consequent DVT and suspected pulmonary thromboembolism, which greatly increased the comorbidity and duration of hospital stay. Although rarely reported in children [3,4], the hypercoagulable state can result in significant clinical sequelae. High altitude is also a predisposing factor for thromboembolic phenomenon [5].

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Virus-Induced Wheezing With COVID-19

Pediatric coronavirus disease 2019 (COVID-19) has now been documented to be a milder illness worldwide except for the few presenting with pediatric multi-system hyperinflammatory syndrome (PIMS). Viral respiratory tract infections are the most common triggers of wheezing illnesses in children. With the ongoing pandemic, a rapid increase in wheezing-related illnesses may be theoretically anticipated. However, COVID-19 induced wheezing is currently thought to be rare. On a related note, a

recently published online survey of members of the Pediatric Asthma in Real Life think tank and the World Allergy Organization Pediatric Asthma Committee [1] also suggested that COVID-19 is not associated with acute onset wheezing in children with underlying asthma. We report our experience with COVID-19 induced wheezing in three children (**Table 1**), who presented to our emergency room with respiratory distress.

COVID-19 associated asthma exacerbation [2] is rare; although there is a theoretical risk of COVID-19 causing a virus triggered asthma exacerbation. Previous epidemics of the coronavirus also did not report significant numbers of asthma exacerbations [3].