

Spontaneous hemothorax in a 10-year-old boy with COVID-19.

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Abstract

Spontaneous hemothorax occurs in the absence of trauma or iatrogenic causes. Etiologies of spontaneous hemothorax in children include connective tissue disease, neoplasia and coagulopathy which is associated with thromboembolic events. We present the case of a 10-year-old chronic hemodialysis patient with spontaneous hemothorax with a concurrent COVID 19 infection.

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Contribution:

- Abir boussetta wrote the article, managed the patient
- Nesrine Abida contributed in the writing of this article
- Manel jellouli and Tahar gargah revised the article and contributed in the management of the patient

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ABSTRACT:

Spontaneous hemothorax occurs in the absence of trauma or iatrogenic causes. Etiologies of spontaneous hemothorax in children include connective tissue disease, neoplasia and coagulopathy which is associated with thromboembolic events. We present the case of a 10-year-old chronic hemodialysis patient with spontaneous hemothorax with a concurrent COVID 19 infection.

KEY WORDS:

SARS-COV-2 - chronic kidney disease - pediatric - Spontaneous hemothorax

BACKGROUND:

The severe acute respiratory syndrome coronavirus 2 (SARS-COV-2) has emerged as a novel human pathogen in December 2019 and was declared a pandemic by the World Health Organization (WHO) on March 11, 2020 (1). Since its discovery, this once thought to be a respiratory illness is now considered as a multisystemic disease with multiple organ involvement including clotting disorders (2). Coagulopathy in Covid 19 infection is associated rather with thromboembolic events than bleeding tendency (1). Hemorrhagic events related to the Covid 19 disease are at the moment strictly limited to case reports.

We here by present the case of a 10-year-old chronic hemodialysis patient with spontaneous hemothorax with a concurrent COVID 19 infection.

OBSERVATION:

A 10-year-old boy with poor vascular access, had been on renal replacement therapy for the past 6 years. The native kidney disease was reflux nephropathy progressing to end-stage renal failure. Peritoneal dialysis was initially started but had to be switched to hemodialysis due to recurrent peritonitis with peritoneal catheter dysfunction.

The Patient was admitted to our emergency department for a sudden-onset of a respiratory distress with chest pain. There was no history of trauma. The patient had no contact with suspected or diagnosed COVID-19 patients.

On admission, his weight and height were 23 kg and 107 cm, respectively. The patient's heart rate was 137 bpm. His respiratory rate was 54 breaths per minute. He was grunting and had intercostal recession. blood pressure was 110/50 mmHg and oxygen saturation in room air was 95%. Dullness to percussion with decreased breath sounds on the left lung field were also noted without crackles nor wheeze.

Blood analysis revealed a hemoglobin level of 64 g/L, a platelet counts of 113.10^3 per mm^3 with a lymphopenia of $590/\text{mm}^3$

Prothrombin time (PT) was found to be normal. Partial thromboplastin time (PTT) was high with a value of 46.5 seconds and a ratio of 1.55. Liver function blood tests were normal and C reactive protein was found to be normal.

Chest X-ray noted left sided pleural effusion with shift of the mediastinum to the right (Figure 1). Thoracentesis was performed with ultrasound guidance allowing the drainage of only few milliliters (ml) of blood suggesting a clotted Hemothorax. Bacterial cultures of the pleural fluid were negative for both non-specific organisms and tuberculosis. Polymerase Chain Reaction (PCR) of the COVID 19 virus was performed on both nasopharyngeal swab and on the removed fluid were found to be positive.

The patient was transfused with packed red blood cells to achieve a hemoglobin concentration of 92 g/L.

A thoracic CT scan was performed showing a massive and compressive right-sided pleural effusion without extravasation of contrast agent (Figure 2).

Thoracoscopy allowed the unclotting of the hemothorax and the evacuation of 700 ml of blood. The pleural cavity per-procedure's investigation showed no active bleeding. A chest tube was inserted at the end of

procedure and was removed 5 days later.

The patient was discharged after 10 days and was clinically asymptomatic.

DISCUSSION:

Hemothorax is defined as a pleural fluid with a hematocrit level greater than 50% of the patient's blood. It is a clinical entity caused in most cases by trauma or iatrogenic causes through procedures such as pleural biopsies or central line insertion (3).

Spontaneous hemothorax (SH) involves the accumulation of blood within the pleural space in the absence of trauma or iatrogenic causes(3). It is extremely rare in the pediatric population with data limited to few case reports (4).

Spontaneous hemothorax etiologies in children include connective tissue disease, neoplasia, arteriovenous malformations, multiple exostosis and coagulopathy (3).

Approximately 20%-50% of hospitalized patients with COVID-19 have hematologic changes in coagulation tests (1).

Coagulopathy in Covid 19 infection is associated rather with thromboembolic events than bleeding tendency.

The pathogenesis of SARS-CoV-2-induced coagulopathy is not yet fully understood. It has been proven that the SARS-CoV-2 enters the host via the angiotensin converting enzyme receptor 2, which is expressed in cells including endothelial cells (5).

Recent evidence showed that the virus can induce endothelial activation directly or by an immune-mediated mechanism in the presence of a locally dysregulated inflammation. Activated endothelial cells release high numbers of ultra large von Willebrand factor (vWF) multimers. Under normal circumstances, vWF flows in intact vessels together with platelets and erythrocytes without binding to any blood element. Elevated microcirculatory shear stress due to endothelial cell dysfunction leads the unfolding of vWF from a globular structure to an extended chain structure exposing its binding sites. Long strands of vWF multimers becoming functionally active. It has been proven that a functional imbalance in vWF multimer sizes can lead to either microcirculation bleeding or thrombosis (5).

While a meta-analysis comprising 28,173 patients with COVID-19 estimated an in-hospital prevalence of venous thromboembolism of 14.1% (6), Researchers reported a case of spontaneous retroperitoneal hematoma in a 47-year-old woman with a COVID-19 concurrent infection. She had no medical priors and was not taking any medication(7).

Others reported 6 cases of abdominal bleeding associated with COVID-19 infection. Five of them used low/medium dosage heparin while the sixth took a high dose for pulmonary embolism. Laboratory investigations showed a normal PTT and PT, excluding a possible adverse effect from anticoagulant therapy (8).

In addition, 2 cases of massive intracranial hemorrhage have been reported in patients being treated with VV-ECMO for COVID-19, with normal PTT and in the absence of other risk factors that could predict such complication(9). Patients with chronic kidney disease (CKD) have a paradoxical hemostatic potential with increased rates of bleeding associated with a higher risk of thromboembolic events. Bleeding events have been reported in 24–50% of patients on HD (10). Increased bleeding is essentially due to platelet dysfunction and alterations in the coagulation cascade with deranged vWF and platelet interactions (10).

CKD was the major risk factor of bleeding in our patient. In the absence other relevant findings, we concluded that the spontaneous hemothorax was due to the covid 19 vasculopathy probably by rupture of small chest wall vessels in a predisposed pediatric patient.

CONCLUSION:

The relationship between the SARS COV2 virus infection and bleeding events are still at this point hypothetical. We report this case to emphasize on the need of further research focusing on the pathogenesis of such complication. While screening covid 19 patients for thromboembolic events is common practice, clinicians should keep in mind the bleeding risk especially in predisposed individuals.

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All co-authors contributed to patient management.

CONFLICT OF INTEREST

Authors do not declare any conflict of interests.

AUTHORS CONTRIBUTION

AB: conceived the study and helping in writing the paper. NA: wrote the paper. MJ: contributed to the management of the patient. TG: supervised and aprobated the project.

ETHICAL APPROVAL

Mother's consent has been obtained before submission.

CONSENT

Written informed consent was obtained from the patient's mother to publish this report in accordance with the journal's patient consent policy.

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