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CASE REPORT

Cardiac Tamponade, Sever Hypothyroidism and Acute Respiratory Distress Syndrome (ARDS) with COVID-19 Infection

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Abstract

A 21-years-old with Down syndrome presented with respiratory distress. Initial investigations revealed a cardiac tamponade. On further evaluation, he had positive coronavirus disease-2019 (COVID-19), severe chest infection and severe hypothyroidism. He responded well to urgent pericardiocentesis, levothyroxine, hydrocortisone and tocilizumab.

Keywords: COVID-19, Hypothyroidism, Tamponade, ARDS, Tocilizumab, Pericarditis

History of presentation

A 21-year-old with Down syndrome was brought into the emergency department (ED) by his mother with a complaint of shortness of breath for two days. His presentation preceded by three days of flu-like symptoms, including runny nose, cough and reduced oral intake and vomiting. On examination, he was in distress, respiratory rate of 28 breaths/min, oxygen saturation 95% on 15 L non-rebreathing mask, blood pressure (BP) of 85/60 mm Hg, heart rate of 68 beats/min, temperature of 36.4C, raised JVP, distant heart sounds and diminished breath sounds over the lower left lung field.

Past medical history

The patient had a past medical history of congenital tracheobronchomalacia, which was treated successfully with surgical intervention during neonatal age. He had no past medical history of weight gain, weakness, cold intolerance, hoarseness, or changes in his mental status or bowel habits.

LEARNING OBJECTIVES

1 To learn physical examination and early investigations finding of critical rare extra-pulmonary manifestations of COVID-19 for early diagnosis and management.
2 To get attention to cardiac tamponade and severe hypothyroidism as a rare presentation of COVID-19.

Differential diagnosis

The differential diagnosis includes COVID-19 pneumonia, viral pericarditis and cardiac tamponade. In the absence of expected compensatory tachycardia during cardiac tamponade, we include hypothyroidism as one of the differential diagnosis.

Investigations

Chest x-ray (CXR) (Figure 1) demonstrated left lung consolidation and enlarged cardiac silhouette. Electrocardiography (ECG) (Figure 2) showed sinus rhythm and diffuse low QRS voltage. Transthoracic

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echocardiography (TTE) (Figure 3) (Videos 1) revealed large circumferential pericardial effusion, swinging heart, early diastolic right ventricular (RV) collapse and normal left ventricular ejection fraction (LVEF) of 60%. Nasopharyngeal swab (NPS) revealed a positive SARS-CoV-2 virus and negative for other viral respiratory pathogens (including influenza A virus, H1N1 virus and Middle East respiratory syndrome-related coronavirus (MERS-CoV). Thyroid function tests showed severe hypothyroidism, thyroid-stimulating hormone (TSH) level of 163 uIU/mL, T3 level of <0.4 pmol/L and T4 level of <0.3 pmol/L. Positive anti-thyroid antibodies, thyroglobulin antibody of 170 IU/mL and thyroid peroxidase antibody of 88 Iu/mL. QuantiFERON-TB Gold blood test was negative for tuberculosis and no blood, trachea aspiration or urine cultures growth. Other laboratory investigations revealed a white blood cell count (WBC) of 9300 cells/pL, hematocrit of 30% and platelet of 180,000. High serum level of N-terminal pro-B-type natriuretic peptide (NT-PROBNP) 2444 pg/mL, D-Dimer of 2.0 ug/mL and C-reactive protein 52 mg/L, and no significant increase of high-sensitivity cardiac troponin T level (0.043 ng/mL; AMI cutoff is 0.1).

Abbreviations

COVID-19	Coronavirus disease-2019
SARS-CoV-2	Severe acute respiratory syndrome coronavirus-2
CXR	Chest X-ray
ECG	Electrocardiography
TTE	Transthoracic echocardiography
TSH	Thyroid-stimulating hormone
NPS	Nasopharyngeal swab
ARDS	Acute respiratory distress syndrome
LVEF	Left ventricular ejection fraction
WBC	White blood cell count

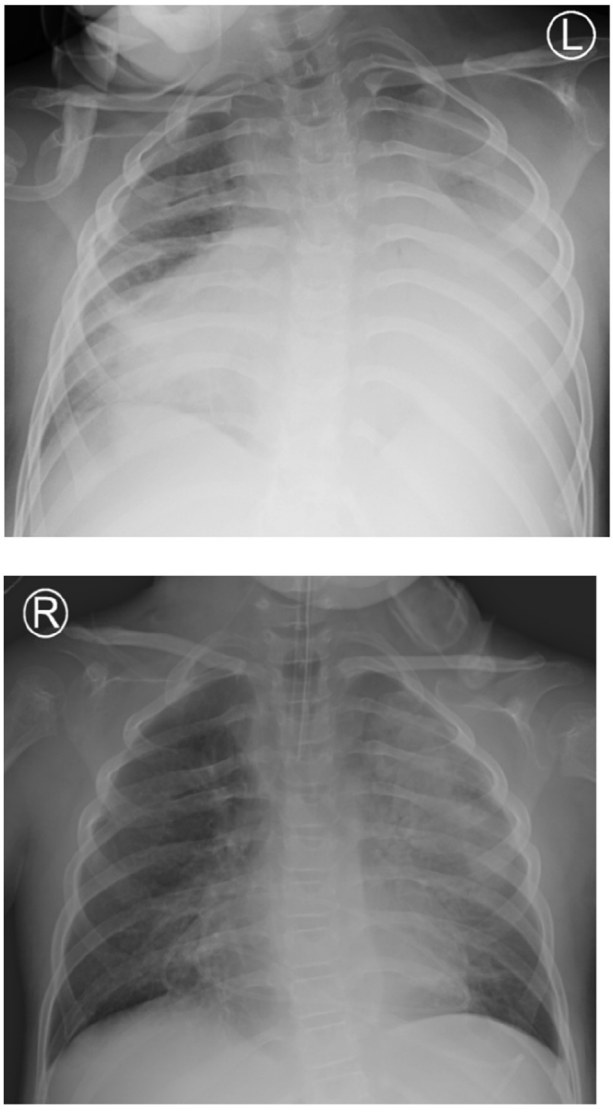

*Figure 1. initial Chest x-ray. **(Left)** Chest x-ray before pericardiocentesis: left lung consolidation and enlarged cardiac silhouette. **(Right)** Chest x-ray after pericardiocentesis: normal cardiac silhouette size and left lung consolidation.*

Management

An urgent echo-guided pericardiocentesis from an apical approach was performed, draining 950 cc of an exudative straw color fluid. The blood pressure was recovered immediately following pericardiocentesis (BP = 102/71). However, the patient was in respiratory distress, not responded to initial non-invasive positive pressure ventilation (NIPPV), and underwent elective intubation. The pericardial fluid analysis revealed exudative results of total protein (TP) of 85 g/L (pericardial fluid TP/serum TP > 0.5), total fluid WBC of 253 cells/microL with a predominance of polymorphonuclear leukocytes 62.3%, mononuclear cells of 37.6%, and no fluid cultures growth. Pericardial fluid cytology showed atypical cell appearance, and no malignant cells could be seen. On the night of admission day, severe hypothyroidism managed with intravenous levothyroxine and hydrocortisone for few days then changed to oral form through a nasogastric tube. In the hope of growing reported evidence of anticoagulation to reduce mortality in patients with severe COVID-19 with coagulopathy, therapeutic heparin infusion started.[1] However, over the initial five days of admission, the patient was critically ill and clinically deteriorated. He developed acute respiratory distress syndrome (ARDS) with increased of bilateral lung

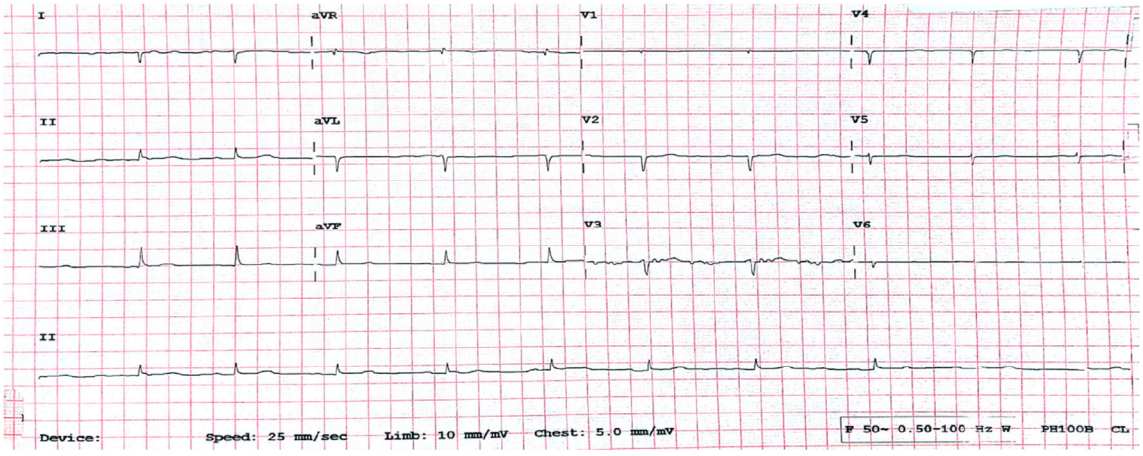

Figure 2. Initial Electrocardiogram. Electrocardiogram: sinus rhythm and diffuse low QRS voltage.

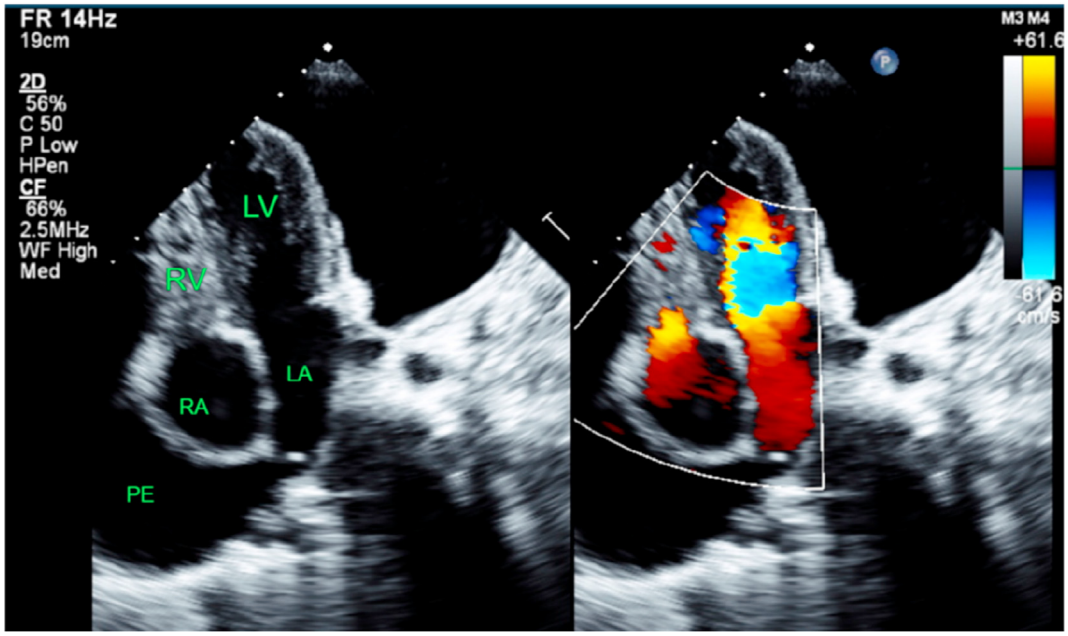

Figure 3. Initial Transthoracic echocardiography. Transthoracic echocardiography: large circumferential pericardial effusion and early diastolic right ventricular collapse. LA = left atrium; LV = left ventricle; PE = pericardial effusion; RA = right atrium; RV = right ventricle.

infiltrations (Figure 4), the partial pressure of arterial oxygen (PaO2) to the fraction of inspired oxygen (FiO2) of 133 mm Hg (<300 mm Hg) and positive end-expiratory pressure (PEEP) of 10 cm H2O. Moreover, he developed pancytopenia with a WBC of 400 cells/pL, a hematocrit of 29% and platelets of 68,000 cells/pL. Heparin-induced thrombocytopenia antibody test was negative. Repeated TTE (Figure 5) (Videos 2) revealed small pericardial effusion without features of cardiac tamponade. At this stage, COVID-19 related Cytokine release syndrome (or cytokine storm) had been suspected. An interleukin-6 level test revealed a high-level result of 130 pg/mL. Therefore, interleukin-6 receptor antagonist tocilizumab started. In the subsequent days, the global clinical condition improved, recovered pancytopenia, and reduced CXR lung infiltration. On account to prolong intubation, tracheostomy performed to facilitate gradually weaning from mechanical ventilation.

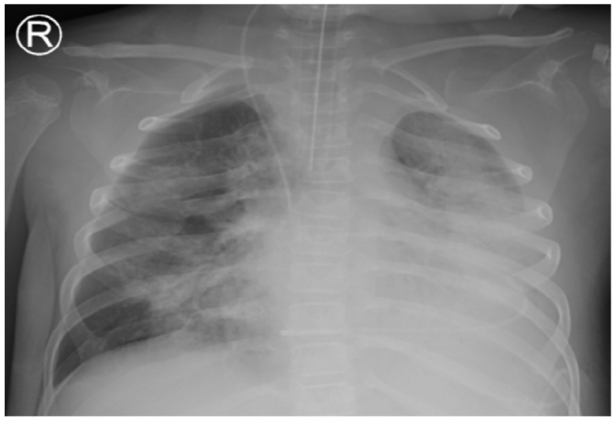

Figure 4. Chest x-ray on day 5 of admission. Chest x-ray: bilateral increase in lung consolidation.

Discussion

Coronavirus disease-2019 (COVID-19) represents a potentially fatal disease that causes massive destruction in the health care system. Herein, we report a case of Down syndrome who developed COVID-19 infection, presented with cardiac tamponade, chest infection and severe hypothyroidism. Among all COVID-19 previous reported cases, the present case is the first suchlike clinical presentation. COVID-19 clinical manifestations are not limited to the respiratory system, and other organs can be affected.[2] It is hypothesized that COVID-19 virus can cause systemic dysfunction by direct cytotoxic effects or immune-mediated excessive cytokines release termed cytokines storm.[3] Although viral pericarditis is the most common cause of pericardial effusion,[4] there are few reported cases of COVID-19 who developed cardiac tamponade.[5,6,7] Tocilizumab is an IL-6 receptor (IL-6R) blocker that had a promised initial efficacy data in cytokines storm management.[3] In the present case general condition, respiratory illness and pancytopenia respond well to tocilizumab treatment. On the other hand, hypothyroidism is a common cause of pericardial effusion.[8] However, owing to a slow accumulation of pericardial fluid, hypothyroidism rarely causes cardiac tamponade.[8] Provocative factors (for example, viral infection) usually precede most of hypothyroidism related cardiac tamponade.[8] Thyroid dysfunctions, particularly overt and subclinical hypothyroidism, are common in Down syndrome, with a prevalence varies between 4% and 18%, and autoimmune antibodies are one of the hypothesized causes.[9,10] The early recognition of hypothyroidism in Down syndrome can be difficult and annual screening of thyroid function tests are recommended.[10] In the present case, positive anti-thyroid antibodies raise a possibility of previous subclinical or overt hypothyroidism, which rapidly deteriorated to severe form and cardiac tamponade provoked by COVID-19 infection. Medical treatment of hypothyroidism and large pericardial effusion with levothyroxine demonstrate a good effusion resolution effect.[8] However, pericardiocentesis indicated for diagnostic sample or therapeutic medical emergency in hypothyroidism patients with cardiac tamponade.[8]

Follow-up

The first negative NPS of COVID-19 recorded on the 25th day of admission. After a total of 31 days of mechanical ventilation support, the patient successfully weaned off and planned for tracheostomy closure. CXR and ECG changes were almost resolved (Figure 6,7).

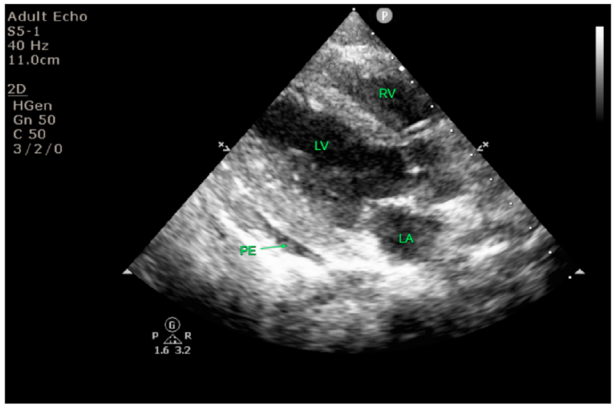

Figure 5. follow up transthoracic echocardiography during admission. Transthoracic echocardiography: small pericardial effusion. Abbreviations as in Figure 3.

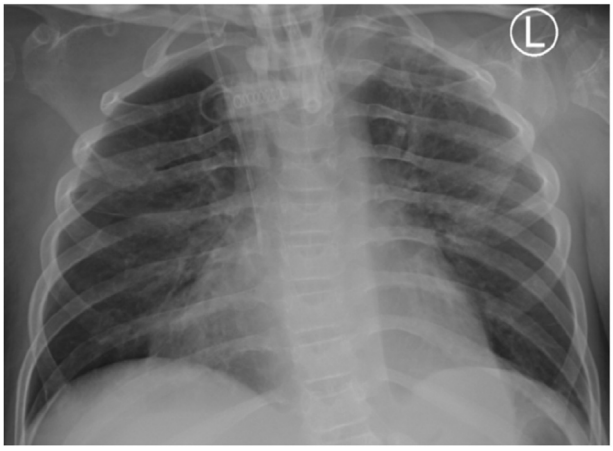

Figure 6. Chest x-ray on day 25th of admission. Chest x-ray: resolved lung consolidation.

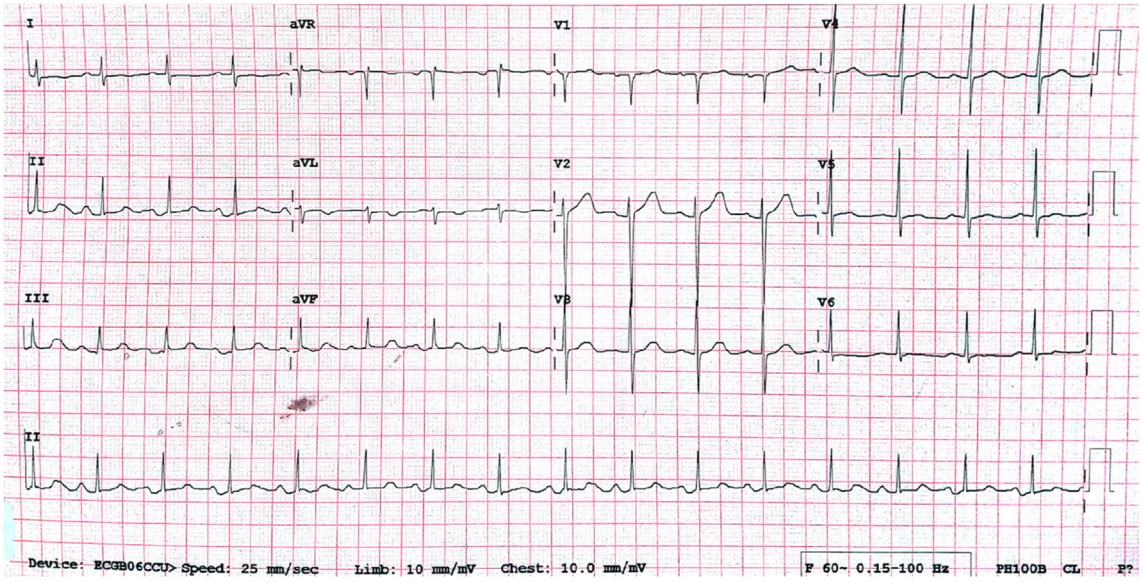

Figure 7. Electrocardiogram. Electrocardiogram: sinus rhythm with resolved low voltage QRS.

Conclusions

We report a rare critical presentation of COVID-19 in Down syndrome patient with cardiac tamponade, ARDS and severe hypothyroidism. The patient responds well to pericardiocentesis, levothyroxine, hydrocortisone and tocilizumab. This case highlights the early recognition of a rare extra-pulmonary manifestation of COVID-19 can save patients life with proper management.

Author contributions

Conception and design of Study: Fahmi A. AL-Kaf, Turki A. Al Garni. Literature review: Turki A. Al Garni, Nahes AL-Harbi. Acquisition of data: Fahmi A. AL-Kaf, Turki A. Al Garni, Nahes AL-Harbi. Analysis and interpretation of data: Turki A. AL Garni, Nahes AL-Harbi, Hassan Sandokji, Sondos Samargandy. Research investigation and analysis: Nahes AL-Harbi, Hassan Sandokji, Sondos Samargandy. Data collection: Fahmi A. AL-Kaf, Turki A. AL Garni, Nahes AL-Harbi, Hassan Sandokji, Sondos Samargandy. Drafting of manuscript: Fahmi A. AL-Kaf, Turki A. AL Garni, Nahes AL-Harbi, Hassan Sandokji, Sondos Samargandy. Revising and editing the manuscript critically for important intellectual contents: Fahmi A. AL-Kaf, Turki A. AL Garni, Nahes AL-Harbi, Hassan Sandokji, Sondos Samargandy. Data preparation and presentation: Fahmi A. AL-Kaf, Turki A. AL Garni, Nahes AL-Harbi, Hassan Sandokji, Sondos Samargandy. Supervision of the research: Fahmi A. AL-Kaf, Turki A. AL Garni. Research coordination and management: Hassan Sandokji, Sondos Samargandy.

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Conflicts of interest

All authors have none to declare.

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