Necrotizing Pneumonia Caused by H1N1 Virus in a Child with Total Anomalous Pulmonary Venous Connection after Cardiac Surgery

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Total anomalous pulmonary venous connection is a rare form of congenital heart disease, occurring in only 1.5% of children with congenital heart disease. Although the mortality and morbidity of total anomalous pulmonary venous connection have decreased dramatically due to improvements in surgery, postoperative pulmonary venous obstruction is still a cause of late mortality in patients with corrected total anomalous pulmonary venous connection. Influenza A H1N1, the most common cause of human influenza in 2009, may cause pneumonia presenting with increased disease severity. Herein we have presented a well-documented case of necrotizing H1N1 pneumonia mimicking postoperative pulmonary venous obstruction in a 4-month-old patient with surgically corrected total anomalous pulmonary venous connection.

Key Words: Influenza • Pneumonia • Pulmonary venous stenosis • Total anomalous pulmonary venous connection

INTRODUCTION

Total anomalous pulmonary venous connection (TAPVC) is a rare form of congenital heart disease in which all pulmonary veins connect to the systemic veins, right atrium, or the coronary sinus.¹ Although the mortality and morbidity of TAPVC have decreased due to improvements in surgical management, pulmonary venous stenosis is still a cause of postoperative and late mortality.²,³ Various infectious agents including influenza A may cause pneumonia, which is one of the leading causes of death in children.⁴ A well-documented case of surgically corrected total anomalous pulmonary venous connection and H1N1 necrotizing pneumonia is described here.

CASE REPORT

A 4-month-old male patient with no significant past medical history was admitted to a local hospital with complaints of perioral cyanosis and poor feeding. He was referred to our hospital with the diagnosis of total anomalous pulmonary venous connection, ostium secundum-type atrial septal defect (ASD) and hypoplasia of the aortic arch. His vital signs were as follows: heart rate 130 beats/min, respiratory rate: 25/min, blood pressure 85/40 mmHg, axillary temperature 36.9 °C and pulse oximetry was 88-90%. Upon examination, S2 was fixed split, 3/6 systolic murmur was auscultated in all areas, being more prominent in the pulmonary area and other physical examination findings were normal. The patient’s electrocardiogram showed right ventricular hypertrophy, and chest radiography revealed an enlarged cardiac silhouette. Echocardiographic imaging indicated
an enlarged right atrium and ventricle; an interventricular septum deviated to the left side, 13 mm ostium secondum type ASD and hypoplastic aortic arch. Ascending aorta, transverse aorta, aorta distal to left subclavian artery and abdominal aorta were measured as 7 mm, 4.2 mm, 4 mm and 4.9 mm, respectively. A sac of anomalous return of pulmonary veins was noted behind the left atrium, and the sac was draining to the innominate vein. The patient was hospitalized with the diagnosis of supracardiac TAPVC. Thoracoabdominal computed tomography (CT) confirmed the diagnosis of TAPVC, enlargement of right cardiac chambers and prominent thinning of the aorta distal to the aortic arch. Initially, the patient was treated medically for heart failure, whereafter the TAPVC was repaired surgically. He was extubated on the third day of surgery, and on the fifth day he was transferred from intensive care unit to the ward. During follow-up, tachypnea and dyspnea gradually developed. Preliminarily, his chest radiography was interpreted as pulmonary congestion; to rule out pulmonary vein obstruction, echocardiography was performed instantly. This revealed normal pulmonary sac-left atrium connection, no obstruction of pulmonary flow and normal systolic-diastolic function. Thereafter, the patient had a fever of 38 °C and laboratory tests revealed normal findings except for the following: erythrocyte sedimentation rate was 12 mm/h, and C-reactive protein was 14 mg/L. After providing throat, blood and urine cultures, his antibiotic treatment (cefazolin) was changed to cefoperazone and amikacin IV. Due to respiratory insufficiency, the patient was intubated; although mechanical ventilation and inotrope support were utilized, he had signs of cardiac failure, pulmonary edema, and acute respiratory distress syndrome. Due to the patient’s clinical deterioration, chest radiography (Figure 1) and then thorax CT scan (Figure 2) were performed. Chest x-ray showed necrotic cavitations in the lower lobes, most prominent in the right lung. Thorax CT scan revealed bilateral diffuse consolidation most prominent in the lower lobes, ground-glass opacity most prominent in the upper lobes, with soft tissue density in the left main bronchus and necrotic cavities. The patient was diagnosed with necrotizing pneumonia and his antibiotic regimen was shifted from IV cefoperazone + amikacin to IV colistin + teicoplanin + levofloxacin. Tracheal aspirate culture was taken, and viral and bacterial polymerase chain reaction (PCR) analyses were performed as well. Despite antibiotic treatment for five days, the patient’s condition did not ameliorate. H1N1 was the only agent identified, discovered by real time PCR analysis of tracheal aspirate. Oseltamivir was co-administered and the patient was treated successfully. He was extubated on the 40th day and discharged on the 76th day after surgery. Recent chest x-ray revealed sequels of pneumonia and no necrotic cavity. Ultimately, we believe this was a case of necrotizing pneumonia caused

![Figure 1. Chest x-ray with necrotic cavities prominent in right lower lobe.](image1)

![Figure 2. Thorax computed tomography scan of the patient showing necrotic cavities.](image2)
by H1N1 virus because of the patient’s clinical deterioration despite proper antibiotic therapy, and the identification of H1N1 by PCR analysis of trachea aspirate and the recovery of the patient after administration of oseltamivir as a combination therapy.

DISCUSSION

Total anomalous pulmonary venous connection is a rare form of congenital heart disease occurring in only 1.5% of children with congenital heart disease, in which all pulmonary veins connect to the systemic veins, right atrium, or coronary sinus. Although most patients develop symptoms in the first year of life, the onset of symptoms may be in the first weeks of life in patients with pulmonary venous obstruction. Patients commonly present with tachypnea, cyanosis, poor feeding, failure to thrive and frequent respiratory infections. With the improvements in surgical management, the mortality and morbidity of TAPVC have decreased dramatically in the last several decades. However, the incidences of postoperative and later occurring mortality are still high due to postoperative pulmonary venous stenosis (PVS) and increased pulmonary vascular resistance. Incidence and mortality rate of PVS are reported to be 5-18% and 37-100%, respectively. Pulmonary edema, enlarged liver, and normal sized heart are generally suggestive of PVS. However, diagnosis can be confirmed by echocardiography, and urgent surgical repair is typically warranted if treatment is required.

It is a well-known fact that children with congenital heart disease are more susceptible to respiratory infections, and the H1N1 virus can mimic the clinical findings. Various infectious agents may cause pneumonia, which is one of the leading causes of mortality in children. Necrotizing changes may be observed in up to around 7% of bacterial pneumonia cases, and necrotizing pneumonia is an increasingly diagnosed complication in children. Most common pathogens are *Staphylococcus aureus*, *Klebsiella pneumonia*, *Haemophilus influenzae*, *enterobacter*, *Nocardia*, *actinomyces*, and *pseudomonas* and *pseudomonas* species. Influenza-associated pneumonia constitutes 4-17% of all pneumonia cases. Influenza A H1N1 virus is the subtype that was the most common cause of human influenza in 2009, which can be confirmed by real time polymerase chain reaction assay using nasal swabs. H1N1 virus pneumonia is reported to present with increased disease severity, higher hospitalization and mechanical ventilation rates. The main target of influenza A/H1N1 is type II pneumocytes, and the major histopathological finding is diffuse alveolar damage. A case of necrotizing pneumonia caused by community-acquired staphylococcus aureus and pandemic influenza A (H1N1) co-infection has been earlier reported. Annual influenza vaccination for all persons > 6 months of age is recommended by the Advisory Committee on Immunization Practices, and especially targeting individuals who are at risk of acquiring, transmitting, or developing complications from the disease.

We have presented this case to remind fellow medical practitioners that although pulmonary venous stenosis is a common postoperative complication, necrotizing pneumonia caused by H1N1 virus can mimic the clinical findings.

CONCLUSIONS

Although pulmonary venous stenosis is a common postoperative complication, necrotizing pneumonia caused by H1N1 virus can mimic the clinical findings. Patients with congenital heart problems are more susceptible to respiratory infections, and the H1N1 virus can cause a be a cause of pneumonia presenting with increased disease severity in these patients. Therefore, we would suggest that people who are at risk of transmitting the disease to these patients should be properly vaccinated.

CONFLICTS OF INTEREST

The authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers’ bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.
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