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Brief Report

Pericardial effusion in an infant with severe respiratory syncytial virus bronchiolitis

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Abstract Respiratory syncytial virus is the most common pathogen causing lower respiratory tract infection in infants. Respiratory syncytial virus infection is also associated with a number of extrapulmonary manifestations, including the cardiac system. Pericardial effusion, however, is a very rare occurrence with respiratory syncytial virus infection. We report a very young infant with respiratory syncytial virus bronchiolitis whose clinical course was associated with pericardial effusion, treated conservatively.

Keywords: Extrapulmonary manifestations; cardiovascular system; children

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RESPIRATORY SYNCYTIAL VIRUS IS THE MOST common pathogen causing lower respiratory tract infection in infants and children.[1]

Respiratory syncytial virus infection is also associated with a number of extrapulmonary manifestations, including in the cardiac system.[2] The most commonly reported effects on the heart during respiratory syncytial virus infection include elevated cardiac troponin levels and cardiac arrhythmias. Respiratory syncytial virus infection is also associated with myocarditis and heart block.[3–7] Pericardial effusion, however, is a very rare occurrence with respiratory syncytial virus infection. Searching the medical literature revealed only one case report of cardiac tamponade.[8]

We report an infant with respiratory syncytial virus bronchiolitis whose clinical course was associated with pericardial effusion, which was treated conservatively.

Case

A 1-month-old infant was admitted to our paediatric intensive care unit during the winter of 2010–2011 with a 3-day history of coryza, cough, and increasing respiratory distress with desaturations and apnoeas on the day of admission. Pregnancy was uneventful with a normal antenatal ultrasound; labour was at term, and medical history did not indicate any cardiac or pulmonary pathology. On physical examination, the infant was found to be alert, the anterior fontanel was normal, respiratory rate was 80 per minute, heart rate was 170 per minute, temperature was 38.0°C, systolic blood pressure was 90 millimetres of mercury and diastolic pressure was 40 millimetres of mercury, intercostal and subcostal retractions were noted, good air entry with wheezing and crepitations were heard over both lungs, the heart sounds were normal, and no murmurs or gallops were heard. Chest X-ray showed hyper-inflated lung fields with right upper lobe atelectasis and normal cardiac shape. Nasopharyngeal swabs were sent for detecting respiratory viruses. The real-time polymerase chain reaction was negative for influenza A, B, and H1N1 viruses, as well as for adenovirus. Immunofluorescence studies for adenovirus, parainfluenza viruses 1–3, and influenza A and B viruses yielded a negative result. The swab was positive for respiratory syncytial virus antigen. Blood and urine cultures showed no growth of bacteria. He was treated with inhalation of hypertonic saline combined with salbutamol, and aminophyline was given intravenously because of

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apnoeas, with good effect. No more apnoeas were observed, and aminophyline was discontinued after 24 hours. Tachycardia up to 195 per minute, tachypnoea up to 88 per minute, and increasing respiratory distress with marked inter-costals and subcostal retractions were, however, noted on the second day of hospitalisation, which necessitated respiratory support with a high-flow nasal cannula (Vapotherm, Stevensville, Maryland, USA). Repeat chest X-ray showed bilateral patchy atelectasis and a rounded heart shadow. An echocardiogram performed on the third day of hospitalisation showed a moderate amount of clear pericardial fluid with normal heart size and function. The troponin I level in the blood was normal, as also the serum albumin level and kidney functions, and repeat blood cultures were negative. The clinical status of the infant improved gradually and he was discharged 10 days after admission. At discharge a repeat echocardiogram showed no pericardial fluid, and the heart size and function were normal.

Discussion

Many studies have examined the pulmonary manifestations including complications, treatment, and long-term sequelae of respiratory syncytial virus. There are, however, only a few studies that have examined the extrapulmonary manifestations of the infection.

Previous studies demonstrated direct evidence that respiratory syncytial virus might spread outside the respiratory tract by documenting the presence of respiratory syncytial virus viral messenger ribonucleic acid in arterial blood mononuclear cells, suggesting the manner in which the respiratory syncytial virus may be carried to extrapulmonary sites.[9,10] The extrapulmonary effects of respiratory syncytial virus infection may also be the end result of released inflammatory mediators such as cytokines and chemokines triggered by the respiratory syncytial virus infection of the respiratory tract.[2]

Cardiovascular failure with hypotension, sepsis-like syndrome, elevated cardiac troponin levels, cardiac arrhythmias, myocarditis, central apnoea, seizures, focal neurologic abnormalities, hepatitis, and hyponatremia has been documented in the setting of respiratory syncytial virus infection.[3–7] However, pericardial effusion associated with respiratory syncytial virus infection seems to be very rare. In reviewing the medical literature we could find only one case report of a 9-month-old infant with cardiac tamponade in the setting of respiratory syncytial virus bronchiolitis, which was associated with a severe clinical course including pneumothorax treated with a thoracostomy tube, mechanical ventilation, and signs of cardiogenic shock.[8] An echocardiogram showed a large pericardial effusion and very poor myocardial contractility. Pericardiocentesis was performed, with marked improvement thereafter. The pericardial fluid showed no bacteria on Gram's stain, and no growth of bacteria, *Mycoplasma*, respiratory syncytial virus, cytomegalovirus, echovirus, or enteroviruses on culture.

Our patient adds to this report in being very young, and, although suffering from severe respiratory syncytial virus bronchiolitis with a moderately sized pericardial effusion, he was treated conservatively without a need for intubation and pericardiocentesis.

As pericardiocentesis was not performed, viral analysis of the effusion was not carried out. Considering the information in the published case, it is conceivable that the extrapulmonary manifestations of respiratory syncytial virus infection include the pericard.

Awareness and recognition of the possible extrapulmonary manifestations of respiratory syncytial virus infection are important in improving the management of patients with this infection, particularly those with a severe course.

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