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Brief Report

Streptococcus pneumoniae purulent pericarditis in a neonate

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Abstract Purulent bacterial pericarditis is an uncommon infection that manifests during childhood, and in the post-antibiotic era *Streptococcus pneumoniae* is an unusual cause. We report a case of purulent bacterial pericarditis in a neonate caused by *Streptococcus pneumoniae* serotype 7F. Although cases of bacterial pericarditis caused by *Streptococcus pneumoniae* as a causative agent have been reported, their combination in a neonate is unique and this is, to our knowledge, the first case of this combination in the newborn period.

Keywords: Pericarditis; pneumococcus; pericardiectomy

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Purulent bacterial pericarditis is an uncommon infection in childhood, and in the post-antibiotic era *Streptococcus pneumoniae* is an unusual cause. Since 1980, there have been 13 published cases in the English language literature with patients' ages ranging from 6 months to 13 years.[1] We report here the first case, to our knowledge, of *Streptococcus pneumoniae* pericarditis occurring in the newborn period.

Clinical case

A 7-day-old boy was presented at his paediatrician's office with fever, decreased oral intake, respiratory distress, and lethargy noted over the previous 24 hours. The infant had been born at term through normal spontaneous vaginal delivery; the pregnancy had been complicated only by diet-controlled gestational diabetes. No complications were noted while in the nursery, and the infant was discharged home on the third day of life. At presentation, the infant was found to be lethargic and in respiratory distress with tachycardia, tachypnoea, and hypoxaemia. He was taken to a nearby emergency room, where blood, urine, and cerebrospinal fluid cultures were obtained, and treatment was begun with intravenous ampicillin, cefotaxime, and acyclovir. The peripheral white blood cell count was 26,000/square centimetre, with 53% neutrophils and 24% bands, and the cerebrospinal fluid was normal. In the emergency room, the patient became apnoeic and increasingly tachycardic. He was intubated and given an intravenous fluid bolus. An echocardiogram demonstrated a moderate-sized pericardial effusion. He was assessed to be haemodynamically unstable and was started on infusions of milrinone and dobutamine.

The patient was transferred to the Kravis Children's Hospital at The Mount Sinai Medical Center where an echocardiogram and cardiac catheterisation were performed. The echocardiogram soon after admission (Fig 1) demonstrated a moderate-sized pericardial effusion with numerous fibrinous strands. Ventricular function was assessed to be normal and there was no evidence of tamponade by two-dimensional or Doppler assessment. Cardiac catheterisation revealed mildly elevated right and left heart filling pressure volumes – mean right atrial pressure was 10 millimetres of mercury and capillary wedge pressure was 10 millimetres of mercury. Pericardiocentesis was performed, and 30 millilitres of "milky" fluid was removed from the pericardial space. Post-procedural echocardiogram showed a mild residual pericardial effusion, and repeat

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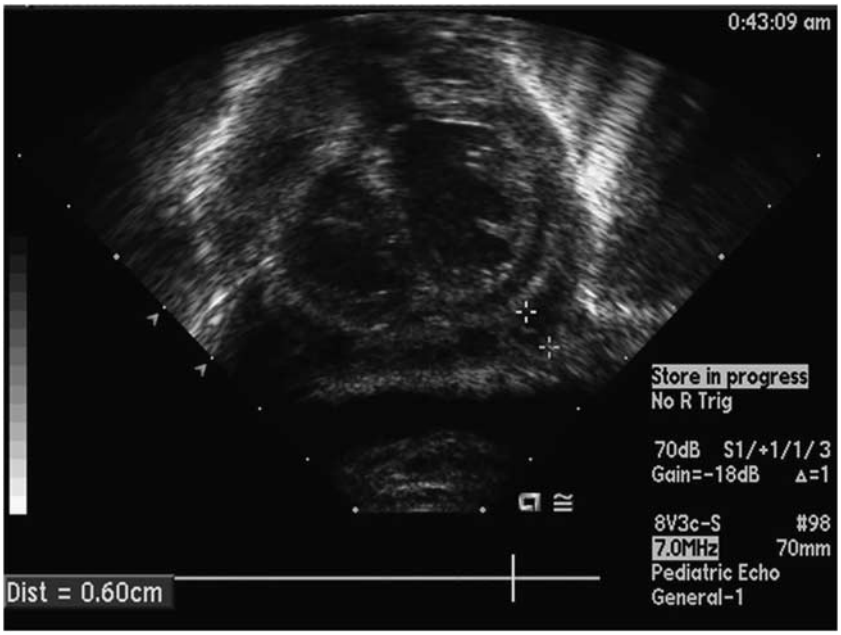

Figure 1.
Sub-xyphoid short-axis view: multi-septated effusion (measuring 6 millimetres) is seen surrounding the posterior aspect of the right and left ventricles.

cardiac catheterisation demonstrated a decreased right atrial pressure up to 8 millimetres of mercury.

The patient was admitted to the paediatric intensive care unit and remained intubated on inotropic support. The pericardial fluid was reported to contain 4000 red blood cells/cubic millimetre and 2000 white blood cells/cubic millimetre with 81% neutrophils. His physical examination was significant for tachycardia, a 2/6 systolic murmur along the lower left sternal border, moderate generalised oedema with abdominal ascites, and extremities that were warm and well perfused. His antibiotic treatment was changed to intravenous ampicillin, cefotaxime, and vancomycin. Both the initial blood culture from the referring hospital and the pericardial fluid culture were positive for *Streptococcus pneumoniae* sensitive to penicillin (minimum inhibitory concentration = 0.032). Urine and cerebrospinal fluid cultures were negative. Treatment was continued with intravenous ampicillin.

The patient's condition improved over the next 72 hours and he was extubated. However, over the following several days he became increasingly irritable and intermittently febrile. Complete blood counts demonstrated a white blood cell count up to 34,800/square centimetre with 60% neutrophils and 12% bands. The C-reactive protein level increased to 143.7 milligrams/litre. An echocardiogram demonstrated a small re-accumulation of pericardial fluid, with increased thickening and loculation of the pericardial space by multiple thick fibrinous strands. There was no evidence of pericardial tamponade; however, a Doppler study revealed a 20% respiratory variation in the mitral valve inflow and increased reversal of flow in the hepatic veins, both of which suggested development of constrictive physiology. On hospital day 7, the patient underwent pericardial debridement. The right ventricle was found to be adherent to the anterior pericardium, and fibrinous material surrounded the right and left atria and the cardiac apex. The pericardial space was debrided and irrigated, and a chest tube with its tip in the pericardium was left in place. The procedure was well tolerated and he remained in the hospital to complete a 4-week course of intravenous ampicillin. An echocardiogram before discharge demonstrated good biventricular function, no residual pericardial effusion, and normal respiratory variation in mitral and tricuspid valve Doppler inflows. Both the blood and pericardial fluid isolates of *Streptococcus pneumoniae* were tested by the New York State Department of Health and were found to be serotype 7F positive.

Discussion

Purulent bacterial pericarditis in the neonate is extremely uncommon. The case presented here is to our knowledge the first reported case of neonatal *Streptocuccus pneumoniae* pericarditis. Similar to cases reported among older infants and children with

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pneumococcal pericarditis, this patient presented with fever and respiratory distress. Early recognition and diagnosis of purulent pericarditis in this patient resulted in a good outcome with no long-term sequelae.

Although mortality due to purulent pericarditis has been greatly reduced with the combination of early initiation of appropriate antibiotics and surgical drainage, 10–30% mortality is still reported.[1–4] As demonstrated in previous case reports and retrospective studies, purulent pericarditis requires both appropriate antibiotic therapy and pericardial drainage.[2–8] Surgical intervention usually includes the creation of a pericardial window with pericardial washout. Total pericardiectomy may be necessary but is usually reserved for those patients, like ours, demonstrating restrictive physiology and impaired diastolic function by echocardiography.[4,6]

In the pre-antibiotic era, the main causative organisms of purulent bacterial pericarditis were *Streptococcus pneumoniae*, followed by *Staphylococcus aureus*, other *Streptococci* species, and Gram-negative organisms.[1,3,8] With the advent of penicillin, both the rates of *Streptococcus pneumoniae* purulent pericarditis and the overall incidence of purulent pericarditis greatly declined.[1,3] Currently, the main causative organisms of purulent pericarditis are *Staphylococcus aureus*, followed by *Neisseria meningitidis*, *Haemophilus influenzae*, other *Streptococci* species, Gram-negative organisms, and much less commonly *Streptococcus pneumoniae*.[1–7,9]

With the introduction of the 7-valent pneumococcal conjugate vaccine (serotypes 4, 6B, 9V, 14, 18C, 19F, 23F), there has been a decline in the prevalence of invasive pneumococcal disease among children. However, the Centers for Disease Control and Prevention and several studies have demonstrated a rise in invasive pneumococcal infections caused by serotypes not found in 7-valent pneumococcal conjugate vaccine, primarily serotype 19A.[1,10] A recent study in the United States by Kaplan et al[10] found that several serotypes have become increasingly common causes of invasive disease, particularly serotypes 19A, 7F, 1, and 3; they reported serotype 7F as the second most common cause of invasive pneumococcal disease, after serotype 19A. In addition, two European studies also reported serotype 7F as a frequent cause of invasive pneumococcal disease in children and found a significant association between serotype 7F and invasive infection in children less than 4 months and 6 months of age.[11,12] The explanation for this observed age-related association is unclear. Despite the relative increase in invasive pneumococcal disease secondary to serotypes 19A and 7F, the decline in invasive disease seen following the introduction of 7-valent pneumococcal conjugate vaccine should continue with the recent introduction of 13-valent pneumococcal conjugate vaccine, which broadens protection against additional *Streptococcus pneumoniae* serotypes, including serotypes 19A and 7F.

Conclusion

Streptococcus pneumoniae is a rare cause of purulent pericarditis in children, and the current report of neonatal infection is unique. In addition, this case emphasises the importance of early diagnosis, initiation of appropriate antibiotic therapy, and surgical intervention in minimising the morbidity and mortality associated with this infection.

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