Finding pneumo: purulent pericarditis presenting with pulsus paradoxus
Christopher C Blyth, Indira Jayakumar, Peter Richmond, Andrew M Bullock and Simon J Erickson

TO THE EDITOR: In the pre-antibiotic era, purulent pericarditis was a disease predominantly affecting children and young adults and was commonly associated with pneumonia or empyema.1 With therapeutic advances, the incidence and mortality of purulent pericarditis have decreased, and it is now most often seen in the elderly, the ill and the immunosuppressed.2

We report a 16-month-old girl with recurrent otitis media and mild pulmonary valve stenosis who presented with a 4-day history of fever, lethargy and anorexia. She had previously received Haemophilus influenzae type b and meningococcus type C vaccines but not pneumococcal vaccines. Oral amoxicillin and clavulanic acid had been commenced to treat acute otitis media 2 days before the current presentation.

On presentation, she had tachycardia and tachypnoea. Blood pressure was 98/65 mmHg, and peripheral perfusion was poor. Examination showed significant hepatomegaly but no abnormality on auscultation. Laboratory tests showed acidemia (pH, 7.06), with a serum lactate concentration of 9.3 mmol/L, neutrophilia (neutrophil count, 28 x 10⁹/L), coagulopathy (international normalised ratio, 3.7; activated partial thromboplastin time, 50 s), transaminitis (serum alanine aminotransferase concentration, 3591 U/L), and normal renal function. Anteroposterior chest x-ray showed a normally sized cardiac silhouette (cardiothoracic ratio, 55%) without focal changes.

Despite endotracheal intubation, ventilation, fluid resuscitation and inotropic support, tachycardia persisted, and hypotension worsened (70/50 mmHg). Pulsus paradoxus was identified on continuous blood pressure recording, with a difference of 15 mmHg between inspiration and expiration. Elevated central venous pressure (23 cmH₂O) was noted following insertion of a right internal jugular central venous catheter. The patient suffered a bradycardic arrest, requiring cardiopulmonary resuscitation for 2 minutes. Following this, electrocardiography demonstrated sinus tachycardia with widespread ST elevation.

A transthoracic echocardiogram showed a large pericardial effusion. There was evidence of tamponade with diastolic right atrial and ventricular collapse and generalised marked systolic dysfunction. Some fibrinous strands were seen within the effusion. Needle pericardiocentesis was performed using the standard approach, and 55 mL of purulent fluid was drained, with marked improvement in haemodynamic parameters. Microscopic examination of the fluid revealed polymorphonuclear leukocytosis and gram-positive diplococci. Streptococcus pneumoniae antigen was demonstrated on latex particle agglutination. However, cultures of blood and pericardial fluid remained negative.

The infant was treated with intravenous antibiotics for 23 days. Her intensive care stay was complicated by acute lung injury, acute renal failure requiring peritoneal dialysis, and hepatic dysfunction. Daily echocardiograms revealed no reaccumulation of pericardial fluid, with no Doppler evidence of constrictive pericarditis. Investigations to identify an underlying immunodeficiency gave negative results. She subsequently made a full recovery.

This case highlights the difficulty of diagnosing purulent pericarditis in young children, who commonly lack the classic symptoms and signs. A high degree of clinical suspicion is required in children with fever and haemodynamic compromise who fail to respond to appropriate therapy, to enable early management.

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References

Correction
Re: “Notable Australian contributions to the management of ventilatory failure of acute poliomyelitis. With special reference to the Both respirator and Dr John A Forbes”, by Ronald V Trubuhovich, in the December issue of the Journal (Crit Care Resusc 2006; 8: 383-393). In the caption to Figure 1 on page 384 and the Acknowledgements section on page 392, the Ny Carlsberg Glyptotek, Kobenhavn, was incorrectly referred to as the Ny Carlsbad Glyptotek.
Negative-pressure pulmonary oedema with normal concentration of B-type natriuretic peptide

David J Sturgess, Bala Venkatesh, Chris Perry and Wayne Kelly

TO THE EDITOR: Negative-pressure pulmonary oedema (NPPO) is an uncommon but life-threatening complication of acute or chronic upper airway obstruction.1

B-type natriuretic peptide (BNP) is a novel biochemical marker for ventricular dysfunction.2 The availability of rapid, bedside assays has stimulated interest in critical care settings. BNP concentration measured early after the onset of acute pulmonary oedema appears to help differentiate acute lung injury from cardiogenic pulmonary oedema.3 Here, we describe what we believe is the first specific documentation of BNP concentrations in NPPO.

A 53-year-old man underwent septoplasty, functional endoscopic sinus surgery and uvulopalatopharyngoplasty. Elective admission to the intensive care unit had been organised for postoperative airway monitoring, given his past history of obstructive sleep apnoea confirmed on diagnostic polysomnography. He was a recent ex-smoker.

Due to restricted laryngoscopic view, endotracheal intubation was performed blindly over a gum-elastic bougie. Surgery was uneventful, but extubation was followed by complete airway obstruction. Following relief of this obstruction, the patient developed frank pulmonary oedema. He was transferred to the ICU after endotracheal reintubation.

On arrival in the ICU, he was afebrile, with a pulse rate of 80 beats per minute in sinus rhythm, and blood pressure of 155/70 mmHg. Heart sounds were normal, and jugular venous pressure was not elevated. Copious frothy pink fluid was present in the endotracheal tube. Auscultation demonstrated generalised coarse inspiratory crackles.

Portable supine chest radiography revealed extensive bilateral air space shadowing consistent with acute pulmonary oedema. Initial arterial blood gas analysis documented a large alveolar–arterial oxygen tension gradient (\(\text{FiO}_2\) of 1.0; pO\(_2\) of 81 mmHg).

Plasma troponin T and creatine kinase concentrations were normal at the time of ICU admission and 12 hours later. Respective B-type natriuretic concentrations were 50 pg/mL and 47 pg/mL (reference range < 100 pg/mL). Serial electrocardiograms appeared normal. Culture of the oedema fluid subsequently showed normal respiratory flora.

Transthoracic echocardiography performed the morning after surgery documented normal systolic function (left ventricular ejection fraction, 65%). Left ventricular filling pressure was also normal, as assessed by Doppler echocardiography (the ratio of early diastolic mitral inflow velocity to mitral annular velocity \([E/E'] = 7\]). Impaired oxygenation, and clinical and radiographic evidence of pulmonary oedema were present at the time of echocardiography.

The patient was successfully extubated and discharged from the ICU on the third postoperative day. He made a full recovery and was discharged from hospital thereafter.

This case of perioperative acute pulmonary oedema occurred in a patient who underwent elective surgery, was previously well and had fasted for longer than 8 hours before surgery. The fluid from the endotracheal tube was not suggestive of gastric fluid, and subsequent culture showed normal respiratory flora. The provisional diagnosis of NPPO was made early. However, as cardiogenic pulmonary oedema was considered a possible cause, measurement of BNP concentrations and echocardiography were requested.

BNP concentrations were normal. Echocardiography was performed before the resolution of pulmonary oedema. Filling pressure was assessed by Doppler echocardiography and was normal. An \(E/E'\) ratio< 8 accurately predicts a normal mean left ventricular diastolic pressure.4 This technique has been validated in a number of clinical settings, including mechanically ventilated ICU patients.5 Normal systolic function further supported the diagnosis of non-cardiogenic acute pulmonary oedema.

Research regarding this specific use of BNP measurement is likely to be hindered by the infrequency of NPPO. However, we propose that measurement of BNP concentrations might be useful in differentiating NPPO from cardiogenic acute pulmonary oedema.

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References
Letters


Leptospirosis: an unusual presentation
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To the Editor: We read with interest the case report on an unusual presentation of leptospirosis by Thomas and Stephens,1 and report a similar case in our hospital.

A 19-year-old Nepalese man with no relevant past medical history presented to hospital in Melbourne with a 4-day history of fever, myalgia, dry cough and headache. He had travelled from Nepal 2 weeks previously. He had no history of animal exposure or water-based activity. On examination, he had a fever and tachycardia. Conjunctival suffusion was noted.

Initial investigations revealed a normal chest x-ray appearance, and normal liver function, and serum urea and electrolyte concentrations. Abnormal results included thrombocytopenia (platelet count, 65 x 10^9/L) and glomerular haematuria. Respiratory swabs were taken for viral testing. As a viral respiratory infection was suspected, the patient was nursed in isolation and managed symptomatically.

Eighteen hours after admission, his condition deteriorated significantly, with a temperature of 40°C and severe hypoxia. Repeat chest x-ray showed diffuse alveolar infiltrates (Figure 1). He was intubated on the ward, and frank blood was aspirated from his airways. A bronchoscopy revealed inflamed airways and diffuse petechial haemorrhage. Other investigations included baseline serological testing for multiple infections, including leptospirosis, and vasculitis screening. The patient was treated with empirical antibiotics, including azithromycin, flucloxacillin and ceftriaxone.

The patient’s course in the intensive care unit was uneventful, and inotropic and ventilatory support were weaned. He was discharged home on Day 16. Serological testing of convalescent blood taken on Day 15 revealed the diagnosis of leptospirosis, with a rise in antibody titre to Leptospirosis copenhageni from < 50 at baseline to 1600.

Flooding in Nicaragua in 1995 was associated with an epidemic of leptospirosis complicated by pulmonary haemorrhage without icterus.2 Another study in Nicaragua illustrated that risk factors for death with pulmonary haemorrhage were elevated serum potassium concentration, haemodynamic disturbance and renal impairment, but not impaired liver function.3 These studies and our case support the concerns raised by Thomas and Stephens that pulmonary haemorrhage is a rare but increasingly recognised complication of leptospirosis, and that it can occur in patients without icterus.

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