Emergency Percutaneous Balloon Mitral Valvotomy in a Patient With Septic Shock*

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We report an 18-year-old patient with severe mitral stenosis complicated by right lower lobe pneumonia, sepsis, and shock. Intractable low cardiac output led to an emergency percutaneous balloon mitral valvotomy in a patient, resulting in immediately improved hemodynamic parameters. We are unaware of another report of percutaneous balloon mitral valvotomy performed in a patient with sepsis and shock. This case supports previous isolated reports of the benefit from emergency percutaneous balloon mitral valvotomy in critical situations where thoracotomy is not possible due to coexisting medical problems.

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Of patients with pneumonia admitted to the ICU, 55% will develop shock requiring inotropic support.1 Mitral stenosis in the presence of sepsis requiring an increased cardiac output can lead to critical decompensation. The patient may become so ill as to present a prohibitive risk for open-heart surgery. The role of percutaneous balloon mitral valvotomy to acutely improve hemodynamic performance in this situation is described.

CASE REPORT

An 18-year-old black man with a known history of mitral stenosis was admitted to the medical ward with a cough productive of purulent sputum, severe dyspnea, and rigors. He had a fever (38.9°C), tachycardia (heart rate, 140 beats per minute and regular), and tachypnea. Blood pressure was 106/70 mm Hg. Jugular venous pressure was raised, and the patient was jaundiced. Bilateral crackles and rales were present and the liver was enlarged.

Electrocardiogram revealed a sinus rhythm, P pulmonale, P mitrale, a right axis, and right ventricular hypertrophy. Chest radiograph showed a large right ventricle and left atrium, enlarged pulmonary arteries, and pulmonary edema. Air bronchograms were present in the right lower lobe. Arterial blood gas value analysis revealed a severe, partially compensated metabolic acidosis (pH, 7.3; P,CO, 13.3 mm Hg; P,0, 78 mm Hg; bicarbonate, 6.3 mmol/L). A prerenal azotemia (urea value, 7.4 mmol/L; creatinine level, 72 mmol/L) was present. Bilirubin level (37 mmol/L) and transaminases (aspartateaminotransferase, 178 U; alanine aminotransferase, 173 U) values were elevated. Albumin level was 3.3 g/L. International Normalized Ratio (INR) was 1.4, and WBC count, 17.2×10⁹/L.

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Hepatitis viral studies were negative.

The patient’s condition deteriorated and he was transferred to our ICU. On admission, the blood pressure was unrecordable and a cardiorespiratory arrest occurred shortly afterwards. Intubation was immediately achieved, and following cardiopulmonary resuscitation, sinus cardiac rhythm was restored. A dobutamine (25 μg/kg/min) and epinephrine (0.3 μg/kg/min) infusion was started (norepinephrine is not available in South Africa), and a blood pressure of 90/30 mm Hg was achieved. Central venous pressure was 19 cm H2O, and the patient was adequately oxygenated on a fraction of inspired oxygen of 0.35. Dopamine was infused at renal doses (2.5 μg/kg/min), and an intravenous dose of cefotaxime, amikacin, and erythromycin was given.

A few hours later, the patient developed acute pyrexia and blood pressure decreased to 70/40 mm Hg. Blood lactate level rose to 4.9 mmol/L and the patient became oliguric. He was placed on

Figure 1. Transthoracic and transesophageal echocardiographic findings.

Figure 2. Balloon catheter valvotomy. LA=left atrium; LV=left ventricle.
continuous venous venous hemodiafiltration. A pulmonary artery balloon catheter was inserted, and a cardiac index of 2.1 L/min/m², a pulmonary capillary wedge pressure of 28 mm Hg, and a systemic vascular resistance of 1,179 dynes•cm⁻⁵•m⁻² was recorded. Epinephrine infusion was increased to 0.5 µg/kg/min and dobutamine to 35 µg/kg/min. A balloon valvotomy was requested as the increased demand on cardiac output caused by the coexistent sepsis could not be met.

**ECHO FINDINGS**

Transthoracic and transesophageal echocardiography revealed a thickened and stenosed mitral valve with a valve area of 0.65 cm² on planimetry and pressure half-time and a mean diastolic gradient of 31 mm Hg (Fig 1). A valve score of seven was assigned to the mitral valve according to previously defined criteria.

**CATHETERIZATION AND VALVOTOMY PROCEDURE**

The patient underwent standard right and left cardiac catheterization. Following transeptal puncture with the Brockenbrough technique, a 30-mm Inoue balloon catheter was used for valvotomy. One inflation was performed at 26 mm (Fig 2).

**RESULTS**

Table 1 shows the hemodynamic results as assessed immediately before and after valvotomy. The patient’s hemodynamic state improved dramatically. Left ventricular angiography done at the end of the procedure showed no mitral regurgitation, and on oxygen saturation there was no detectable atrial shunt. Lactate level decreased from 4 mmol/L to 1.5 mmol/L in 24 h. Inotropic support was weaned rapidly. Renal function improved allowing discontinuation of renal support. Hepatic function improved. Echocardiographic follow-up failed to reveal evidence of endocarditis, and antibiotic therapy was stopped after 1 week. The patient was weaned from the ventilator and was transferred to a high-care area. The patient’s course was uneventful for the following 3 weeks; however, while still hospitalized, he developed nosocomial pneumonia and severe sepsis and subsequently died.

**DISCUSSION**

Mitral stenosis most commonly results from rheumatic fever and is not uncommon in black South Africans. The course is often insidious with gradually worsening symptoms. The hemodynamic consequences are left atrial enlargement, pulmonary venous congestion, reactive pulmonary hypertension, and eventually right ventricular failure, perhaps with tricuspid regurgitation.

Diastolic filling of the left ventricle is achieved by the pressure gradient across the valve and the diastolic time available for left ventricular filling. An increased heart rate caused by exercise, atrial fibrillation, or sepsis will therefore decrease ventricular filling, and cardiac failure may be precipitated.

Severe pneumonia may progress to septic shock. Septic shock is characterized by a decreased systemic vascular resistance, hypovolemia, a compensatory increase in heart rate, and hypotension despite an increased cardiac output. A myocardial depressant factor is also implicated.

Our patient had evidence of both advanced mitral stenosis and right lower lobe pneumonia and showed features of septic shock. His further deterioration was likely due to an inability to compensate with an increased cardiac output as a result of a severely stenosed mitral valve. All attempts at medical management failed and a more aggressive approach was indicated. As the sepsis and hemodynamic instability of the patient precluded surgery, mitral balloon valvotomy, which has been described previously in a similar hemodynamic situation, was considered more appropriate. The use of transvenous mitral valvotomy was first described by Inoue et al in patients who could not undergo thoracotomy due to coexisting medical illness. The effectiveness and safety of this technique is now well recognized.

The result of this intervention was immediate hemodynamic improvement (Table 1) followed by clinical improvement over a period of 10 days. No evidence of bacterial endocarditis or other complications relating to the procedure was noted.

In conclusion, balloon mitral valvotomy resulted in immediate hemodynamic improvement and subsequent clinical improvement in a critically ill, hemodynamically decompensated septic patient. The death of the patient from severe sepsis because of advanced nosocomial pneumonia was unrelated to the mitral stenosis. We suggest its use be considered in similar situations.

**REFERENCES**

Facilitation of Percutaneous Dilational Tracheostomy by Use of a Perforated Endotracheal Tube Exchanger*

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Endoscopic guidance during percutaneous dilational tracheostomy reduces the risk of paratracheal insertion of the tracheostomy tube but may impair ventilation and does not eliminate the possibility of premature endotracheal extubation. Use of a small-caliber, hollow endotracheal tube exchanger during sternal dilation in lieu of a fiberoptic bronchoscope allows for better ventilation and more secure airway control.  

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Key words: bronchoscopy; endotracheal intubation; intraoperative complications; mechanical ventilation; tracheostomy

Percutaneous dilational tracheostomy is an attractive alternative to classic surgical tracheotomy for many critically ill patients. Uncontrolled reports suggest that the postoperative risks of bleeding and infection may be less with the dilational procedure than with surgical tracheotomy. Additionally, the procedure may be safely performed at the bedside, which eliminates the need to transport patients in the ICU to the operating room. Intraoperative complications have primarily been limited to hypoxia, hypotension, and paratracheal insertion of the tracheostomy tube. Direct endoscopic visualization of guide wire, dilator, and tracheostomy tube placement reduces the likelihood of paratracheal insertion; however, endoscopic visualization may precipitate intraoperative hypoxia and does not eliminate the possibility of endotracheal tube dislodgement during rigorous dilation and tracheostomy tube insertion.

There are several technical limitations of fiberoptic endoscopy during percutaneous dilational tracheostomy. First, a large-sized endotracheal tube (≥8 mm inside diameter) is necessary to allow passage of a standard adult 6-mm outside diameter fiberoptic bronchoscope, while still permitting adequate ventilation. Even the use of a pediatric-sized scope may not allow adequate ventilation around the bronchoscope for prolonged periods of time. Also, these smaller scopes do not permit secretions to be adequately cleared from view. Second, a separate operator is required to maintain position of the fiberoptic endoscope, and a very proximal scope position provides little margin of safety against extubation. Finally, a bronchoscope does not allow for tube exchange in the event of endotracheal tube cuff rupture during needle insertion; also, it cannot provide ventilation itself in the event of accidental extubation.

We herein report a modification to the conventional percutaneous dilational tracheostomy procedure that utilizes endoscopic guidance during guidewire placement, but subsequently uses a commercially available endotracheal tube exchanger (Fig 1) to improve airway security and reduce airway resistance during sternal dilation. We have recently performed 17 dilational tracheotomies using either a fiberoptic bronchoscope only, or the modified technique that combines fiberoptic guidance with an endotracheal tube exchanger. All 17 cases were performed using narcotic analgesia, benzodiazepine sedation, local and aerosolized lidocaine anesthesia, neuromuscular blockade, 100% FiO2, and full mechanical ventilatory support. Prior to bronchoscopy, povidone-iodine skin preparation, sterile drapes, local anesthetic, skin incision, and careful blunt dissection to the level of the pretracheal fascia were employed using standard anatomic landmarks. This report focuses on modifications of the currently recommended percutaneous dilational tracheostomy procedure, detailed reports of which can be found elsewhere.

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