tetralogy of Fallot, may be present as well.\textsuperscript{1,2}

Patients with scimitar syndrome are typically asymptomatic.\textsuperscript{3} Because of this, the actual prevalence of this rare syndrome is difficult to determine. The diagnosis is usually suspected as a result of incidental discovery of the scimitar vein on chest radiograph. Symptoms, when present, most often include the following: (1) recurrent pulmonary infection secondary to alteration of the tracheobronchial architecture; (2) congestive heart failure due to left to right shunt from the anomalous draining vein; or (3) associated cardiac malformations.

The PA catheter measurements very effectively monitor left atrial filling pressures allowing safe and adequate fluid resuscitation. The pulmonary artery catheter's value in determining the hemodynamic status of a patient is predicated upon the ability to obtain pressure measurements that can be extrapolated to reflect the volume status of the various heart chambers. Of paramount importance in most cases, and in this patient in particular, is a determination of the left ventricular end-diastolic volume. With the balloon inflated within a segment of the pulmonary artery, the pressure transmitted to the transducer is a reflection of the left atrial pressure. At end-diastole, with the mitral valve open, the transmitted pressure reflects the pressure at end-diastole in the left ventricle. These pressures are converted to volumes and cardiac function based upon the Starling relationship.\textsuperscript{4}

The CVP is theoretically linked to the filling pressures of the ventricles but is removed from it by a significant series of "baffles" such as the right cardiac chambers which interfere with the ability to determine the left ventricular volume and pressure status. In a young healthy adult with a normal cardiopulmonary circuit, the CVP may be an accurate reflection of all central filling pressures. However, with any pathologic or stress state, the baffle effect, as well as the varying compliance of the different conduits between the left ventricle and the central vein (pulmonary artery, right ventricle, right atrium, and vena cava) decrease the ability to determine left-sided cardiac function.

The PA catheter measurements are made with the premise that the catheter is wedged in a branch of the pulmonary artery and reflects left atrial pressures. Due to the unique pathophysiology of the scimitar syndrome, however, there is partial or complete drainage of the right lung into the systemic venous circulation, usually, in this case, into the IVC, or less commonly into the right atrium or a hepatic vein. If, as in this case, the catheter is wedged in a branch of the right lung, the pressure readings erroneously reflect CVP rather than left atrial pressure. Fluid management will then be tailored to the CVP pressures and could result in iatrogenic pulmonary edema. Pressure readings reflecting low or normal "pseudo-left atrial pressures" would lead to a maintained or increased rate of fluid replacement since actual left atrial pressures are not being measured. In the current case, the pressure readings were a reflection of a very compliant right heart and veins and contributed to overly aggressive volume therapy with consequent pulmonary edema.

In patients in whom the presence of scimitar syndrome is known or is ascertained from the chest radiograph, placement of the pulmonary artery catheter into a branch of the left pulmonary artery would obviate these problems because of its normal venous drainage to the left atrium.

This case illustrates that when the pathophysiologic consequences of the abnormal pulmonary venous drainage pattern associated with the scimitar syndrome are not recognized, and pulmonary artery catheter measurements are obtained from the side with anomalous venous drainage, iatrogenic pulmonary edema may result.

\section*{REFERENCES}

\section*{Meliodosis Pneumonia and Blast Injury*}

\textbf{Chew Y. Wang, M.B., Ch.B.; Boon H. Yap, M.B., Ch.B.; and Alexius F. Delikan, M.B., B.S.}

We present the case of a 24-year-old woman with acute septicemic melioidosis resulting from inhaled infective dust during a blast injury. With appropriate antibiotic treatment and supportive therapy in the ICU, the patient made an uneventful recovery.

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Meliodosis, or *Pseudomonas pseudomallei* infection, was first described by Whitmore and Krishnaswami in 1912. It was formerly only recognized in Southeast Asia and North Australia, but also occurs in India, Iran, Central Africa, and Central and South America. Its true incidence and distribution are unknown. It can present in an acute septicemic form, which has a high mortality rate. It is difficult to diagnose, and conventional antimicrobial therapy is ineffective. We present a case of acute septicemic melioidosis resulting from inhaled infective dust during a blast injury. To our knowledge, there is no previous report of such a case.

**CASE REPORT**

A 24-year-old woman who was a factory worker was admitted to our Accident Emergency Department following a fireworks explosion. In this patient, the main concern was her lung injury which was possibly due to inhalational pneumonitis, lung contusion due to the blast wave, and possible aspiration pneumonia because she gave a history of cerebral concussion. She also had full thickness burns of the left leg and a first-degree burn in the back and abdomen. On examination, the patient was fully conscious and slightly dyspneic. Systemic arterial pressure was 120/60 mm Hg, and the heart rate was 96 beats per minute. Auscultation of the lungs revealed bilateral basal crepitations with good air entry. Blood gas levels while breathing 5 L of oxygen via a nasal cannula were as follows: $\text{PaO}_2$, 127.8 mm Hg; $\text{PaCO}_2$, 23.7 mm Hg; base deficit, 12.1 mmol/L; and pH, 7.32. A chest x-ray film showed bilateral alveolar infiltrates in the middle and lower zones (Fig 1). Results of laboratory tests were within normal limits except for a serum K+ value of 1.7 mmol/L. This may have been due to exposure to the chemicals present in the fireworks, mainly barium sulfate. The patient was admitted to the ICU for further observation and management because of her lung injury. The provisional diagnosis at this stage was recorded as lung contusion, inhalational pneumonitis, and aspiration pneumonia.

Four hours later in the ICU, she developed respiratory failure which required intermittent positive pressure ventilation. Arterial blood gas value analysis revealed a $\text{PaO}_2$ of 65.8 mm Hg; $\text{PCO}_2$, 27.7 mm Hg; base deficit, 12.4 mmol/L; and pH, 7.28, while breathing 5 L/min of oxygen via a nasal cannula. Methylprednisolone and antibiotic therapy was instituted: she began receiving crystalline penicillin, cloxacillin, and gentamicin therapy for her burns and lung injuries. Her hypokalemia was treated with K+ supplement. Sputum, urine, and wound cultures were taken.

Her condition was serious for the next 48 h. She developed pyrexia. Her temperature rose to 40°C. Her gas exchange deteriorated which required an increase in inspired oxygen of 60 percent and positive end-expiratory pressure of 10 cm H$_2$O to achieve satisfactory $\text{PaO}_2$. She had developed surgical emphysema in the upper chest and neck. A repeat chest x-ray film (Fig 2) showed increased consolidation of the middle and lower zones. The thoracic surgeon thought that this could be due to a tear in the esophagus as a result of the blast injury. However, a gastrografin study showed no perforation of the esophagus. Blood cultures were taken because of her septic condition. The culture of the sputum that was taken on the day of admission grew *Klebsiella pneumonia* and possibly *Pseudomonas aeruginosa*. Because of her persistent pyrexia and deteriorating condition, we thought that additional antibiotics were required and she began receiving ceftazidime and metronidazole. From days 3 to 7, despite the treatment, there was no improvement.

On day 7, the result of the blood cultures grew *Pseudomallei*, which is sensitive to ceftazidime. And a further review of the sputum culture taken on day 1 confirmed the presence of *P pseudomallei* and not *P aeruginosa*. Because of the acute septicemia, it was believed that the dosage of ceftazidime should be increased to (6 g/d) and combination therapy was essential. She also began receiving high doses of intravenously administered trimethoprim-sulfamethoxazole, 10 ml (Bactrim [trimethoprim, 16 mg/ml;...
sulfamethoxazole, 80 mg/ml, given twice daily.

After aggressive treatment from then on, the patient made a gradual recovery. Ceftazidime therapy was stopped after four weeks, and she was advised to continue taking an oral preparation of the same dose of trimethoprim-sulfamethoxazole (T. Bactrim II, given twice daily; one tablet contains 80 mg of trimethoprim and 400 mg of sulfamethoxazole) for a further six months. She was discharged well. There was no evidence of recurrence after six months of treatment.

DISCUSSION

Melioidosis caused by the Gram-negative bacillus P pseudomallei has a wide spectrum ranging from subclinical infection to acute septicemic infection, often fatal. Commonly, the primary source of infection may not be evident. Clinical diagnosis is difficult as would be expected from the varied manifestations of this disease. In our case, the diagnosis of melioidosis was made only after isolation of the organism from the blood cultures, and the standard initial antimicrobial therapy that was instituted was ineffective against the P pseudomallei organism. Because of the relentless course and high mortality of overwhelming sepsis and the unpredictable effectiveness of single antimicrobial agents, a combination of antibiotics usually is advocated for patients with septicemic manifestations. In addition, a large dose of each antibiotic is suggested in order to achieve adequate serum inhibitory and bactericidal concentrations.4

Recently, ceftazidime and trimethoprim-sulfamethoxazole have been advocated for the treatment of septicemic melioidosis.5 Since relapses and recurrences are common, it is imperative that the patients are treated for at least three to six months.6

In conclusion, when patients present with fulminant respiratory failure following blast injury, especially in endemic areas, a high clinical awareness of this disease is necessary for early diagnosis, and appropriate antibiotics should be prescribed. The possibility of inhaled soil organisms causing aspiration pneumonia following blast injury should be borne in mind.

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Severe Ischemic Injury to the Proximal Airway Following Lung Transplantation*

Immediate and Long-term Effects on Bronchial Cartilage

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Ischemia due to interruption of the bronchial circulation has been recognized as a cause of immediate postoperative anastomotic dehiscence in lung and heart-lung transplant recipients. Since patients do not ordinarily survive such major ischemic insults, the long-term effects of airway ischemia and the differentiation of these effects from those of transplant rejection and infection have not been clearly defined. We describe a patient who suffered extensive airway ischemia, necrosis, and subsequent diffuse airway stenosis. Loss of the bronchial circulation with variable ischemia may be a major cause of late airway abnormality responsible for significant morbidity and mortality in transplant recipients.

(Amer J Med 1993; 103:1899-1901)

A irway complications of lung transplantation are well described, including bronchiolitis obliterans,1 bronchiectasis,2 bronchitis, and bronchiolectasis, and are most commonly attributed to rejection. Other mechanisms postulated for the proximal airway changes following transplantation have included pulmonary infection, altered innervation, and ischemia.

Airway ischemia, with early postoperative dehiscence of the bronchial or tracheal anastomosis resulting in death, had been a major limitation to the development of lung transplantation.3,4 Avoidance of steroids,5 omentopexy,6 and more recently anastomoses using a telescoping procedure7 have been developed to promote healing and revascularization, and to support the anastomosis in the event of dehiscence. Interruption of the bronchial circulation is recognized as a major cause of this dehiscence. Patients have not traditionally survived such major ischemic insults and therefore the long-term effect of such an injury on the airways is uncertain. More subtle consequences of ischemia have only recently been described.

We describe a patient who demonstrated immediate ischemic changes following lung transplantation and who subsequently developed generalized irreversible airway stenoses. The airway obstruction progressed over a 6-month period and ultimately necessitated retransplantation.

CASE REPORT

The patient is a 43-year-old woman with respiratory failure due to lymphangioleiomyomatosis who underwent bilateral (bibranchial)

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