# The tale of two cases of pulmonary oedema

**GORDIAN FULDE**

MB BS, FRACS, FRCS(Ed), FRCS/RCP(A&E)Ed, FACEM

**Presentations and outcomes can vary widely, even in critical conditions, as illustrated by the two cases of pulmonary oedema described below.**

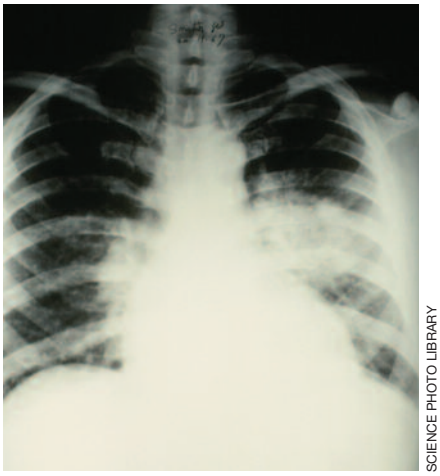

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Figure. Pulmonary oedema, a common critical condition that usually rapidly improves with treatment in the emergency department.

## An unfavourable outcome

You remember well the case of the 80-year-old grandfather who was brought in by ambulance during an evening shift at the emergency department of the local hospital where you work as a GP.

This patient was a visitor from out of town who had been referred by the hotel doctor for vomiting and diarrhoea that had not settled with the injection of prochlorperazine (Stemetil). He had been well until that morning, when he began having severe vomiting and diarrhoea. There was no obvious cause, but he had eaten seafood the day before. His current medications were:

- morphine (MS Contin), piroxicam (Feldene), celecoxib (Celebrex) and gabapentin (Neurontin) for back pain (he had had two lumbar laminectomies)
- irbesartan and hydrochlorthiazide (Avapro HCT) for hypertension
- fluoxetine (Prozac) for depression.

There were no known cardiac or other significant health problems.

It was obvious the patient was quite unwell. His blood pressure was 170/70 mmHg; pulse, 120 beats/min atrial fibrillation; respiratory rate, 28 breaths/min; temperature 37.8°C; and oxygen saturation, 90% on 6 L/min of oxygen. An ECG confirmed atrial fibrillation (new) but showed no acute ischaemic changes, and a chest x-ray showed cardiomegaly and pulmonary oedema. Early blood tests revealed serious abnormalities:

- sodium, 133 mmol/L (normal range, 137 to 146 mmol/L)
- potassium, 2.9 mmol/L (normal range, 3.5 to 5.0 mmol/L)
- white cells, 21.9 x $10^9$/L (normal range, 4.0 to 10.0 x $10^9$/L)
- neutrophils 18 x $10^9$ (normal range, 2 to 7.5 x $10^9$/L).

The blood gases revealed an alkalosis, pH 7.53 (normal range, 7.35 to 7.45).

The provisional diagnosis was pulmonary oedema and atrial fibrillation with possible cardiac ischaemia. the electrolyte disturbance was possibly secondary to the vomiting and diarrhoea.

The following treatment was initiated:

- potassium replacement
- frusemide intravenously, starting with 40 mg
- continuous positive airway pressure ventilation, for the pulmonary oedema, starting at 5 cm $H_2O$ pressure
- high flow oxygen
- amiodarone 300 mg intravenously, to reverse the atrial fibrillation
- morphine 2.5 mg intravenously, for vasodilation, anxiety and back pain
- glyceryl trinitrate infusion, for vasodilation.

In spite of this treatment, the patient required aggressive resuscitation. The emergency, cardiology and intensive care teams were all involved.

The clinical state began to improve but the cardiac marker troponin results came back very raised at 84.4 mg/L, confirming our worst fears that he had suffered a major myocardial infarct. (This serves as a reminder that baseline ECGs are often normal in many patients with infarcts, the changes developing later. In non-ST elevation myocardial infarction [NSTEMI], the baseline ECG may be normal while the cardiac troponin is elevated.)

A femoral intravenous line was inserted and, on the cardiologist's advice, heparin and magnesium were administered.

By now the patient's respiratory rate was 40 breaths/min, his urine output had become poor (less than 30 mL/h), and his serum creatinine had increased

Professor Fulde is Director, Emergency Department, St Vincent's Hospital; Associate Professor in Emergency Medicine at the University of New South Wales, Sydney, NSW.

from 80 to 110 mmol/L (normal range, 40 to 100 mmol/L).

A low dose dopamine infusion was commenced to augment renal blood flow in an effort to preserve renal function and increase urine output.

It was obvious that, in spite of maximal intensive therapy, the patient was dying. Three hours later, even though laboratory blood tests (potassium, sodium and pH) had improved slightly, the patient had a ventricular fibrillation arrest and was not able to be resuscitated.

This was a distressing outcome because almost all patients with pulmonary oedema, a very common presentation, rapidly improve with treatment once in the emergency department – whatever the cause (see Table 1).

## An excellent outcome

The next week you see a 48-year-old woman who was brought in by ambulance with chest discomfort and difficulty breathing.

The history revealed that the symptoms had come on at rest four days ago and had increased since then. The patient denied any chest pain but reported that she had discomfort in both shoulders, and had not been able to lie flat during the previous few nights because of increased dyspnoea and nonproductive cough. She had no medical history apart from a hysterectomy, and was on no medication. She worked full time as a packer at a laundry, smoked eight cigarettes a day and drank alcohol socially on weekends (about 40 g; equivalent to four standard drinks).

On arrival, the woman had classic pulmonary oedema: she was in respiratory distress, unable to speak in sentences and peripherally shut down. Her initial observations were blood pressure, 160/110 mmHg; pulse, 120 beats/min; respiratory rate, 28 breaths/min; oxygen saturation 99% on 10 L/min; and temperature, 36.7°C.

She was immediately given the standard treatment for pulmonary oedema:

- high flow oxygen
- glyceryl trinitrate
- frusemide 40 mg intravenously
- morphine 2.5 mg intravenously
- continuous positive airway pressure ventilation (5 cm $H_2O$ pressure).

She was also given aspirin 300 mg for a possible cardiac ischaemic event.

An ECG showed only sinus tachycardia, and a chest x-ray showed pulmonary oedema and an enlarged heart. All her initial blood tests, including cardiac markers, were normal.

The provisional diagnosis was acute pulmonary oedema, probably secondary to a cardiac event or pulmonary embolus.

The patient responded well to the standard treatment and underwent several investigations. A ventilation–perfusion lung scan (V/Q scan) was reported as medium probability of a pulmonary embolism with matched ventilation–perfusion defects, leg venous doppler studies showed no abnormalities and a pulmonary CT angiogram showed no emboli.

The diagnosis was dilated cardiomyopathy. This was demonstrated by transoesophageal echocardiogram showing global hypokinesis and an ejection fraction of 35%, and coronary angiogram showing patent coronary arteries and an ejection fraction of 35%.

The patient could not understand what all the fuss and tests were for because she felt well and wanted to return home and to work. It seemed difficult for her to realise the significance of having dilated cardiomyopathy, especially as the cause in this case was not known (possible causes include ischaemic, hypertensive or postviral heart disease). The patient was discharged on an ACE inhibitor, and follow up appointments were made, including with the smoking cessation clinic.

## Conclusion

These two patients each had the same common critical condition but their outcomes were at the extremes of the wide range of possible outcomes. However, you remember that the five-year survival of patients with pulmonary oedema is worse than that of acute leukaemia, with a greater than 50% five-year mortality.

One of the advantages of being attached to a hospital is that it helps in keeping up to date with new diagnostic methods. This emergency department was involved in a trial of the usefulness of the blood test utilising brain natriuretic peptide (BNP) that detects cardiac failure and which may, in the future, be used for monitoring patients with known failure. The BNP test is already being used overseas for the diagnosis of pulmonary oedema in emergency and general practice. MT

**Table 1. Common causes of acute cardiogenic pulmonary oedema***

- Inappropriate reduction of therapy or noncompliance
- Arrhythmia – tachycardia or bradycardia
- Pulmonary embolism
- Myocardial ischaemia
- Systemic infection
- High output state – anaemia, thyrotoxicosis, beri beri
- Drugs (nonsteroidal anti-inflammatories)
- Volume overload
- Alcohol excess or withdrawal
- Other heart disease – cardiomyopathy, myocarditis, endocarditis, valvular disease, ruptured valve leaflet
- Hypertension

* Modified from Brown AFT. Acute pulmonary oedema. In: Fulde GWO, ed. Emergency medicine: the principles of practice. 4th ed. Sydney: Elsevier; 2004. p. 81.

**DECLARATION OF INTEREST:** None.