PULMONARY EDEMA

By Bryan E. Bledsoe, DO, FACEP, & Paul Bojan, EMT-P

Early recognition & prompt prehospital therapy improve patient outcomes

Medic 1534 and ALS Quint 1542 are dispatched to a “man down” at a local car dealership. The patient, a 52-year-old, obese man, is seen unconscious for about 10 seconds. He’s alert, and his skin is dry with normal color. Initial vital signs reveal a regular pulse of 100; blood pressure of 200/130 mm Hg; normal, non-laboratory respirations of 18; and a SpO2 of 98% on room air. The ECG reveals a normal rhythm with a heart rate of 100; blood pressure of 200/130; normal color. Initial vital signs reveal a regular rhythm with a heart rate of 100.

Hospital evaluation
On arrival at the hospital, the patient is moved to the emergency department (ED), where the staff is assembled and awaiting his arrival. The patient is deteriorating by the minute. Rales are heard in all lung fields, and he begins coughing up pink foam sputum. Because of the patient’s rapid respiratory failure, the ED physician elects to perform emergent rapid sequence intubation (RSI). He’s given 6 mg midazolam (Versed) and 150 mg succinylcholine (Anectine). Following adequate relaxation, he’s intubated and placed on a mechanical ventilator. Paralysis is maintained with vecuronium (Norcuron), and sedation is maintained with additional doses of midazolam. The ED physician also puts him on IV nitroglycerin and starts diuresis with IV furosemide (Lasix).

Evaluation & treatment
The patient, a 52-year-old, obese (weight 325 lb, height 6 ft) male in no acute distress, had a brief syncopal episode while standing. Bystanders report he was unconscious for about 10 seconds. He’s alert, and his skin is dry with normal color. Initial vital signs reveal a regular pulse of 100; blood pressure of 200/130 mm Hg; normal, non-laboratory respirations of 18; and a SpO2 of 98% on room air. The ECG reveals a paced rhythm.

The patient has a history of congestive heart failure (CHF), hypertension, cardiac dysrhythmias, elevated cholesterol, renal insufficiency, and a below-knee amputation (BKA) of his left leg due to vascular insufficiency. He has an implanted pacemaker and an automated internal cardiac defibrillator (AICD). Current medications include Lasix, Coumadin, Pravachol, Coreg, Cordarone, Vasotec, Viagra and Lanoxin. When questioned, the patient states that he hasn’t taken the Viagra “in several days.”

You provide supportive care, including supplemental oxygen, IV access and cardiac monitoring. The patient initially does well during transport to the hospital of his choice. Suddenly, he develops difficulty breathing, cyanosis and altered mental status. His SpO2 drops into the 50s. You immediately detect the patient’s deterioration, divert to a closer hospital and have your communications center alert hospital staff for rapid transport to the hospital of his choice. The patient initially does well during transport to the hospital of his choice. Suddenly, he develops difficulty breathing, cyanosis and altered mental status. His SpO2 drops into the 50s. You immediately detect the patient’s deterioration, divert to a closer hospital and have your communications center alert hospital staff for rapid transport to the hospital of his choice.

The term flash pulmonary edema describes pulmonary edema that develops rapidly—often over a few minutes. Flash pulmonary edema almost always occurs in patients with a history of CHF and pulmonary edema. These patients normally remain stable on such medications as diuretics, digitalis, nitrates and antihypertensives. However, on occasion, they can rapidly decompensate and develop acute pulmonary edema in a matter of minutes. Causes of this decompensation can include myocardial ischemia, cardiac dysrhythmias, a change in medications or diet (e.g., eating foods high in sodium) or worsening renal insufficiency. Often, the exact cause remains unknown.

Most patients with flash pulmonary edema respond to aggressive diuresis and vasodilator therapy. Emergency treatment of pulmonary edema is directed at correcting the underlying cause, removing excess fluid and providing supplemental oxygenation. However, the first therapy should always be administration of 100% oxygen via the appropriate device.

Case review
Pulmonary edema is an accumulation of fluid within the pulmonary tissues. Although most commonly caused by CHF, it can also result from sepsis, respiratory distress, severe liver disease, exposure to high altitudes and toxic gas inhalation. Pulmonary edema most frequently occurs when the left ventricle begins to fail as an effective forward pump. Causes of left ventricular failure include acute myocardial ischemia or infarction, dysrhythmias and exacerbation of chronic CHF. Vascular pressures in the pulmonary tissues begin to increase as blood to the left side of the heart is supplied from the pulmonary circulation. This results in an increase in pulmonary hydrostatic pressure.

When the hydrostatic pressure exceeds the pressure that normally holds fluid within the vascular system (oncotic pressure), fluid moves into the spaces between the cells of the pulmonary system (interstitial space).

Initially, the lymphatic system removes the fluid between the cells. But the capacity of the lymphatic system is limited, and, eventually, the fluid is forced into the alveoli, where gas exchange takes place. The presence of fluid in the interstitial space and alveoli significantly affects respiratory gas exchange, leading to hypoxia and dyspnea.

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Facial continuous positive airway pressure (CPAP) devices can help increase oxygen tension and airway pressures, which can decrease the amount of fluid that enters the alveoli. If this fails, emergent intubation and mechanical ventilation are indicated. If the cause is myocardial ischemia or infarction, therapy, such as thrombolytics, should be provided. If the patient is suffering an exacerbation of chronic CHF efforts should be made to eliminate excess fluid through the use of potent diuretics, such as furosemide (Lasix).

If the patient’s blood pressure is normal or elevated, vasodilators, such as nitroglycerin, can be used. They decrease cardiac preload and, thus, cardiac work. In severe cases, nitroglycerin should be administered intravenously, if allowed by local protocols. Alternatively, and in patients who are in less severe distress, nitroglycerin paste can be applied to the patient’s chest.

Morphine can also be used in the treatment of acute pulmonary edema. Morphine sulfate acts as a venous vasodilator and can decrease the amount of blood being delivered to the heart (preload), which can decrease pulmonary pressures. In addition, some patients may benefit from the use of agents that increase the force of the cardiac contraction, thus increasing the heart’s cardiac output. Such agents include dobutamine (Dobutrex), dopamine (Intropin) and amrinone (Inocor).

Conclusion
Patients with advanced heart disease and CHF often develop flash pulmonary edema. EMS personnel must always keep this in mind and be alert for early indicators of respiratory distress, such as increased respiratory rate, heart rate, cyanosis and a fall in oxygen saturation. Often, intubation and mechanical ventilation can be avoided by early recognition and prompt prehospital therapy. By Bryan E. Bledsoe, DO, FACEP, & Paul Bojan, EMT-P

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