CASE STUDY PNEUMOCONIOSIS

ADMITTING HISTORY

This 72-year-old black man was well known to the treating hospital, having received care there for over 12 years. While in the U.S. Navy during WWII, he worked on the East Coast in the ship construction industry. After his discharge in 1945 he returned to his home in Mississippi for about 6 months; he then moved to Detroit, Michigan, and began to work for an automobile manufacturer. His primary job for the twenty years was undercoating cars. In early 1970's the patient was transferred to a nearby automotive plant, where he worked the assembly line, fastening bumpers and chrome trim to cars. He was a well liked by his fellow workers and was considered a hard worker by the administration. When he retired in 1980, he was one of four supervisors in charge of the chrome assembly line.

Although the patient had been a two-pack-a-day smoker for more than 40 years, his health was essentially unremarkable until about 4 years before he retired. At that time he started to experience periods of coughing, dyspnea, and weakness. A complete examination provided by the company concluded that the patient had a moderate pneumoconiosis.

On the basis of the patient’s work history, the doctor speculated that the pneumoconiosis was caused by asbestos fibers. This was later confirmed with a Perl stain of sputum, and the diagnosis of asbestosis was put in the patient’s chart. Just before the patient retired, his pulmonary function studies showed mild-to-moderate combined restrictive and obstructive disorder.

Although the patient was able to enjoy a couple of relatively good years of retirement with his wife, after that his health rapidly declined. His cough and dyspnea quickly became a daily problem. Despite his deteriorating health, the patient continued to smoke. When he was 68 years old, he was hospitalized for 8 days for pneumonia and severe respiratory distress. When he was discharged at that time, his pulmonary function studies still showed a moderate-to-severe restrictive and obstructive disorder. He started using oxygen at home on a regular basis.

Ten months before the current admission the patient was hospitalized for congestive heart failure. He was treated aggressively and sent home within 5 days. At discharge his pulmonary function study showed that he had a severe restrictive and obstructive respiratory disorder. His arterial blood gases (ABG’s) on 1 lpm oxygen were pH 7.35, PaCO2 86 mmHg, HCO3- 46 mEq/L, B.E. +18.1 mEq/L, and PaO2 63 mmHg.

Three hours before this admission, the patient woke up from his afternoon nap extremely short of breath. His wife stated that he coughed almost continuously and that he had difficulty speaking. She took his temperature and found it to be 38°C (100.4°F). Concerned, the patient’s wife drove him to the hospital’s emergency room.

PHYSICAL EXAMINATION

As the patient was wheeled into the emergency room, he appeared nervous, weak, and in obvious respiratory distress. His breathing pattern was classified as tachypnea by the emergency room doctor. He was on 1.5 lpm oxygen cannula, which was connected to an “E” cylinder attached to the wheelchair. His skin felt damp and clammy. He appeared pale and cyanotic. His neck veins were distended, and his fingers and toes were clubbed. He had a frequent but wet cough productive of a moderate amount of thick, yellow secretions. He had a 3+ peripheral edema of the ankles and feet. He said this was the worst his breathing had ever been.

The patient’s vital signs were blood pressure 182/106, heart rate of 108 bpm, respiratory rate 32/min, and an oral temperature 38.3°C (100.8°F). Palpation of the chest was negative. Percussion produced bilateral dull notes in the lung bases. Wheezing, rhonchi, and crackles were heard throughout both lung fields. A pleural friction rub could be heard over the right middle lobe between the sixth and seventh ribs and between the anterior axillary line and the mid-axillary line.

The patient’s lower lobes had a diffuse ground-glass appearance on the CXR. Also seen were irregularly shaped opacities in the right and left lower pleural spaces that were identified by the radiologist as calcified pleural plaques. A possible infiltrate consistent with pneumonia also was seen in the right middle lobe. The CXR also disclosed that the right side of the heart was moderately enlarged. His ABG’s on 1.5 lpm were pH 7.56, PaCO2 51 mmHg, HCO3- 38 mEq/L, B.E. +15.9 mEq/L, and PaO2 47 mmHg.

The physician started the patient in I.V. furosemide (Lasix) to treat the patient’s cor pulmonale and began an antibiotic for the patient’s pneumonia. Respiratory care was called to perform
respiratory care evaluation, and to outline further care per RC protocols. The physician said she did not want to commit the patient to a ventilator unless it was absolutely necessary.

Complete an assessment using RC ASSESSMENT FLOW CHART

The Next Morning
Throughout the night the patient’s condition remained unstable. He continued to cough frequently but was not able to expectorate secretions very well on his own. When the therapist assisted the patient with his cough effort, a moderate amount of thick, white and yellow sputum was produced. Even though he was conscious, alert, and able to follow simple directions, he did not answer any questions asked by the respiratory care practitioner regarding his breathing.

His skin was cold and damp to the touch, and he appeared short of breath. His color was improved, but he was still pale and cyanotic. His neck veins were distended, although not so severely as when he was first admitted, and edema of his ankles and feet still could be seen. The patient’s vital signs were blood pressure 192/108, heart rate 113 bpm, respiratory rate 34/min, and temperature 38°C (100.4°F). Palpation of the chest was negative.

Dull percussion notes were elicited over the lung bases. Wheezing, rhonchi, and crackles continued to be heard throughout both lung fields. A pleural friction rub was still present and in the same location. No further CXR’s have been performed. His ABG’s were pH 7.57, PaCO2 47 mmHg, HCO3^- 36 mEq/L, B.E. +15.1 mEq/L and PaO2 40 mmHg. His SpO2 was 77%.

Complete a second assessment using RC ASSESSMENT FLOW CHART

20 Hours Later
At 0615 the alarm on the patient's ECG monitor sounded. The ECG strip showed several premature ventricular contractions (PVC’s) followed by ventricular flutter and fibrillation. The nurse on duty called a Code 99. Cardiopulmonary resuscitation was started immediately. Epinephrine and dobutamine were administered through the patient’s IV. Twelve minutes into the code the patient returned to normal sinus rhythm. Spontaneous respirations were absent.

The patient was intubated, transferred to ICU and placed on a mechanical ventilator. The patient' ventilator settings were: VT 800 cc, SIMV – 10 bpm, FIO2 1.0, pressure support +4 cm H2O, PEEP +10 cm H2O. The patient's cardiovascular status continued to be unstable. PVC’s were frequently noted on ECG monitor. A Swan-Ganz catheter and arterial lines were inserted.

The patient's skin was pale, cyanotic, and clammy. His neck veins were still distended, and his ankle and feet were swollen. The patient's vital signs were blood pressure 135/90, heart rate 84 bpm, respiratory rate 16/min, and temperature 38.3°C (100.6°F). Palpation of chest wall was negative. Dull percussion notes were produced over the lung bases. Wheezing, rhonchi, and crackles could be heard in both lungs. Therapist was suctioning greenish yellow sputum from the endotracheal tube.

The plural friction rub was still present with no change in location. CXR had been taken but interpretation was not available. The following hemodynamic indices were elevated: CVP, RAP, PA, RVSWI, PVR. All the remaining hemodynamic parameters were normal. The ABG’s values were pH 7.53, PaCO2 56 mmHg, HCO3^- 38 mEq/L, B.E. +17.3 mEq/L and PaO2 246 mmHg. His SpO2 was 100%.

Complete a third assessment using RC ASSESSMENT FLOW CHART