Acute Ventilatory Failure From Massive Subcutaneous Emphysema*

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A 66-year-old woman developed massive subcutaneous emphysema following intubation. Acute thoracic restriction developed resulting in life-threatening respiratory acidosis. The patient could not be ventilated with conventional means. A tracheostomy was performed to decompress the chest and mediastinum with rapid resolution following. Although ventilatory failure from subcutaneous emphysema is very unusual, decompression with tracheostomy can be life saving.

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Subcutaneous emphysema is a well-known complication of positive pressure ventilation. Usually it is only a cosmetic problem and does not pose a serious threat to the patient. We have observed one case in which extra-alveolar gas accumulated under pressure resulting in ventilatory failure while the patient was on a mechanical ventilator.

CASE REPORT

A 66-year-old woman with a history of seizure disorder was brought to the emergency department after an episode of tonic-clonic seizures at home. At the scene, the emergency medical service found her in a grand mal seizure that responded to 5 mg of intravenous diazepam. She was intubated orally by the ambulance team with an 8-mm endotracheal tube without difficulty. Within 1 h of arrival at the hospital, she became alert and responsive with a blood pressure of 152/106 mm Hg. Bilateral ronchi were noted on chest auscultation. The initial chest radiograph showed the endotracheal tube to be in proper position and was otherwise unremarkable. She was then sent from the emergency department for computed tomography of the head.

When she returned, she was found to be restless and thrashing in the stretcher. The nurse noted subcutaneous emphysema on the left side of her neck, face, and trunk. No breath sounds were auscultated on the left side of the chest. A presumptive diagnosis of pneumothorax was made. A chest tube was then inserted into the left hemithorax by a surgical resident. A large air leak was noted.

Over the ensuing 3 h, the subcutaneous emphysema continued to progress producing swelling of the face, eyelids, and entire torso down to the thighs and calves. The mechanical ventilator began to exceed its set pressure limit on each breath. Although the inspired tidal volume had been set at 650 ml, the exhaled volumes were varying between 150 and 250 ml per breath. Bilateral breath sounds were audible and proper endotracheal tube position was again confirmed by chest radiography. The blood pressure dropped to 91/69 mm Hg.

Because of the rising airway pressures with no other obvious explanation, it was suspected that the subcutaneous emphysema had produced an acute thoracic restriction. In addition, it was believed that high intrapleural pressures may have been impeding venous return causing the fall in blood pressure. Tension pneumomediastinum or pneumopericardium, although not radiographically evident, were other considerations.

REFERENCES


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Multiple manipulations with the mechanical ventilator were made in an attempt to obtain an adequate minute volume with lower airway pressures. The peak inspiratory pressures remained in the range of 80 to 90 cm H₂O despite the adjustments. Exhaled tidal volumes continued to decline to about 100 ml and the blood pressure dropped to 86/47 mm Hg. A severe respiratory acidosis developed (Fig 1). The arterial blood gas at this time showed a pH of 6.79, a PaCO₂ of 165 mm Hg, and a PaO₂ of 128 mm Hg while on an FIO₂ of 1.0. In a desperate attempt to decompress the chest, as well as the mediastinum, an emergency tracheostomy was performed. On incision of the skin, a rush of gas was noted by the operating team. Bronchoscopy was then performed; no lacerations in any of the airways or esophagus were seen. A chest roentgenogram following surgery confirmed proper placement of the tracheostomy tube and persistent massive subcutaneous emphysema.

In the postanesthesia care unit, serial arterial blood gas determinations demonstrated adequate ventilation with a tidal volume of 700 ml (Fig 1). The blood pressure increased to 105/60 mm Hg. The patient was transferred to the medical ICU. Over the next several days, as the subcutaneous emphysema resolved, she was weaned from the ventilator and the chest tube was removed. The trachea was decannulated on the 8th hospital day. She was discharged from the hospital to home, fully ambulatory, after 14 days of hospitalization.

**DISCUSSION**

Subcutaneous emphysema is not an unusual complication of positive pressure ventilation. Most commonly it arises from airway trauma either at the time of intubation or when high airway pressures result in alveolar rupture and escape of gas into the interstitial space. The free gas can travel along fascial planes and enter the soft tissues. Usually, subcutaneous emphysema is only a cosmetic problem and does not have any serious pathophysiologic sequela.¹

This case illustrates a very unusual problem that is rarely seen in patients with subcutaneous emphysema. The continuous accumulation of gas, under pressure, in the soft tissues can result in an acute thoracic restriction. As the chest wall becomes increasingly noncompliant, the mechanical ventilator will require progressively higher airway pressures to deliver a given tidal volume. A vicious cycle is thus set up whereby high airway pressures result in further accumulation of extra-alveolar gas and thus more restriction which in turn requires even greater pressure to deliver the same volume. The standard volume-cycled ventilator will begin to exceed its pressure limit and not deliver the intended minute ventilation.

Intuitively, high frequency jet ventilators or oscillators, which ventilate with low airway pressures, may be ideal for this situation. These devices, however, are not always readily available and furthermore, there is no reported experience with their use for this indication. The physician is thus faced with a life-threatening problem for which there is no form of accepted therapy.

In this patient, tracheostomy was performed to decompress the soft tissues of the chest wall by providing the accumulated gas with an escape route to atmospheric pressure. The added potential benefits of this procedure were to allow for mediastinal decompression and provide a secure airway.

Recently, Eveloff and associates¹ reported a similar experience with two patients who both improved after surgical decompression of their subcutaneous emphysema. In each of these, in contrast to our case, the incisions made for chest tube placement resulted in lower airway pressures and improved parameters of ventilation.

In our patient, improvement was not noted until after the tracheostomy had been performed. With a functioning endotracheal tube already in place, it is clear that it must have been the tracheostomy incision by itself that was therapeutic. Why the chest tube’s incision did not provide this same benefit is speculative. There was no leakage of gas noted at the tube insertion site as reported in the two cases of Eveloff et al. This suggests that the extra-alveolar gas was preferentially accumulating elsewhere, under tension, and was not in direct communication with the chest tube site. Given the gravity of the respiratory and hemodynamic compromise, coupled with the concern for mediastinal compression, immediate tracheostomy was performed. The
Hemoptysis during sexual arousal has been reported in patients with mitral stenosis, coronary artery disease, end-stage left ventricular failure, and pulmonary embolism. This report describes a patient whose presenting complaint was hemoptysis on sexual arousal. Extensive evaluation subsequently disclosed the presence of cardiac (and possibly pulmonary) amyloidosis as the cause of the patient's hemoptysis.

**Case Report**

The patient was a 57-year-old man who noted on approximately four occasions that he coughed up small amounts of blood after being sexually aroused. Because of the hemoptysis, the patient did not go on to sexual intercourse. Concomitant with this, the patient developed progressive dyspnea on exertion and also noted bilateral lower extremity weakness and pain with exercise. He had also lost about 13.5 kg over the past four to five months and complained of intermittent abdominal pain. The patient had been entirely healthy prior to these events. He had undergone a right carpal tunnel release operation at about the same time that the hemoptysis started. On physical examination, the patient was an ill-appearing man in no acute distress. Vital signs were remarkable for a pulse of 120 and a respiratory rate of 32. Findings from the remainder of the examination were within normal limits.

Initial laboratory data showed a hematocrit of 38.7 percent. Prothrombin time, partial thromboplastin time, and platelet count were normal. Serum protein electrophoresis and urine protein electrophoresis were negative for monoclonal proteins. Resting arterial blood gases on room air showed a pH of 7.40, a PCO2 of 37 mm Hg, and a PO2 of 81 mm Hg. Pulmonary function tests showed an FVC that was 77 percent of predicted. The FEV1/FVC ratio was normal. The FRC was normal. The diffusing capacity for carbon monoxide was 47 percent of predicted. A chest radiograph showed a mildly enlarged cardiac silhouette. There was an atelectatic scar in the left mid-lung field.

The patient underwent fiberoptic bronchoscopy that showed old blood present at the entrance of the right lower lobe, but no evidence of any acute bleeding. No other endobronchial abnormalities were noted. No biopsy specimens were obtained. A V/Q scan was performed that showed a matched ventilation/perfusion defect in the left mid-lung field. A chest computed tomographic scan showed a focal, linear density adjacent to the pleural surface in the left mid-chest just below the level of the carina. The patient had a cardiac evaluation that included a thallium stress test, echocardiogram, and cardiac catheterization. The thallium stress test showed some reversible inferior wall ischemia. The patient was exercised on the Bruce protocol, but he was only able to complete 1 min and 43 s of exercise because he developed severe shortness of breath and back pain radiating down the legs. An echocardiogram showed mild concentric left ventricular hypertrophy and low-normal contractility with an estimated ejection fraction of 50 percent. There was mild mitral regurgitation and tricuspid regurgitation. The patient's cardiac catheterization showed normal coronary arteries and a left ventricular ejection fraction of 55 percent. The patient had electromyograms and nerve conduction velocity studies performed that showed carpal tunnel syndrome on the left.

Subsequent diagnostic procedures included a bone marrow biopsy specimen that showed moderate, diffuse plasmacytosis (5 percent to 10 percent), a gastroscopy that showed erythema at the gastroesophageal junction with mild erosive changes, and a colonoscopy that was normal. Bone marrow, antral, and rectal biopsy specimens were all negative for amyloid. Repeated cardiac catheterization was performed with myocardial biopsies, and these biopsy specimens showed myocardial infiltration with amyloid.

**Discussion**

This patient had several features associated with amyloidosis, including carpal tunnel syndrome, left ventricular dysfunction, anemia, abdominal pain, and weight loss. The diagnosis was elusive (rectal biopsy specimens are diagnostic in 73 percent of cases) and finally was made with myocardial biopsy. Although exertional hemoptysis in amyloidosis has been described previously, this case is unusual in that, to my knowledge, it is the first description of hemoptysis associated with sexual arousal in amyloidosis.

Bleeding complications are fairly common in amyloidosis and are thought primarily to be due to amyloid infiltration of blood vessels with a consequent increase in vessel fragility. Hemoptysis has been reported to occur in 2 percent of all cases of amyloidosis.

Pulmonary involvement in amyloidosis is not unusual, with a frequency of 30 percent reported in a large autopsy series. The incidence is closer to 90 percent in primary amyloidosis, which was this patient's most likely diagnosis, given the absence of any underlying disease. Pulmonary amyloidosis has been traditionally classified as tracheobronchial or parenchymal. Tracheobronchial involvement most commonly causes hemoptysis and/or atelectasis, but there was no evidence for this in this patient on bronchoscopy examination. Parenchymal involvement has been described as nodular, which is fairly indolent, or diffuse, in which interstitial and perivascular deposition of amyloid is usually associated with a progressive downhill course. This patient

**References**


**Hemoptysis During Sexual Arousal**

An Unusual Manifestation of Amyloidosis

Robert Hoffman, M.D., F.C.C.P.

Hemoptysis associated with sexual arousal is thought to be secondary to cardiac dysfunction brought on by the physiologic stress of sexual activity. A patient who presented with hemoptysis occurring only during episodes of sexual stimulation was subsequently found to have cardiac amyloidosis.

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