Pneumomediastinum


Pneumomediastinum Caused by Subcutaneous Emphysema in the Shoulder*

A Rare Complication of Arthroscopy

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Severe subcutaneous emphysema following arthroscopy of the shoulder developed in a 62-year-old man. It subsequently evolved into pneumomediastinum with respiratory distress, but the patient recovered spontaneously. To our knowledge, this constitutes the first report of pneumomediastinum caused by subcutaneous emphysema in the shoulder.

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Subcutaneous emphysema in the shoulder causing pneumomediastinum has not been described. I wish to report such a case, which occurred as a complication of arthroscopy of the shoulder.

CASE REPORT

A 62-year-old man underwent arthroscopy of the right shoulder for debridement of the glenoid labral tear. General anesthesia was induced, and the trachea was intubated easily on the first attempt with an 8.0-mm oral endotracheal tube. It was then connected to a volume-cycled ventilator with a tidal volume of 600 ml and a rate of 8/min. Peak inspiratory pressure was around 15 to 16 cm H2O. The shoulder joint was distended with saline solution, of which the continuous inflow and outflow were regulated by an arthroscopy infusion pump. Surgery and anesthesia were uneventful, but toward the end of the 2-hour surgery, swelling and crepitus of the right arm and shoulder were noted. These rapidly spread to the right side of the chest, the neck, and the face and later also involved the left side as well.

A chest radiograph (Fig 1) demonstrated subcutaneous and mediastinal emphysema. Two hours after completion of surgery, the patient developed a fever of 38.4°C. He felt slightly short of breath and complained of sore throat and substernal chest pain. Arterial blood gas measurements, while the patient was breathing oxygen 2 L/min by nasal cannula, revealed that the PaO2 was 57 mm Hg, the PaCO2 was 38 mm Hg, and the pH was 7.43. With increase of Fio2 to 40 percent (by a Ventimask), the PaO2 increased to 81 mm Hg. Fiberoptic bronchoscopy disclosed no evidence of pharyngeal, laryngeal, or tracheal injury.

Imipenem-clavulanate therapy was started intravenously. He continued to run a fever for the next 3 days and then defervesced. Blood and sputum cultures grew no microorganisms. Subsequent chest radiographs showed no development of pneumothorax. Both subcutaneous emphysema and pneumomediastinum started to decrease on the third postoperative day and completely resolved on day 7.

DISCUSSION

Air in the mediastinum can originate from five sites: the neck, the alveoli, the airways, the esophagus, and the abdominal cavity. In the case presented, there was no evidence of airway, esophageal, or abdominal injuries. The patient was not under prolonged or high positive pressure ventilation and therefore unlikely to have development of alveolar rupture. Had alveolar rupture occurred, giving rise to pneumomediastinum and subsequently subcutaneous emphysema, the latter would have been more symmetrical rather than commencing and remaining predominant around the right shoulder as occurred in this case. Thus, it appeared that the subcutaneous air in the neck, which originated from the right shoulder, was the most likely cause of this patient's pneumomediastinum.

Subcutaneous emphysema in the shoulder causing pneumomediastinum is, however, rather unusual. Deep cervical fascia divides the soft-tissue structures of the neck into three distinct compartments: the previsceral space, the visceral

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FIGURE 1. Anteroposterior radiograph of the chest revealing the pneumomediastinum (arrows) and radiolucent streaks of air in the subcutaneous tissue.

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space, and the prevertebral space.2 The visceral space contains the pharynx, the larynx, the trachea, and the esophagus, and provides a conduit for free movement of gas between the mediastinum and the neck. The prevertebral space, which extends laterally to the axillary sheath, is separated from the visceral space by the prevertebral fascia. Thus, there is no direct communication between the prevertebral space and the mediastinum. Subcutaneous air generated around the shoulder joint would travel readily along the axillary sheath to the prevertebral space due to absence of anatomic barrier. It would cause pneumomediastinum only when the subcutaneous air ruptures the prevertebral fascia, allowing air to enter the visceral space of the neck and subsequently the mediastinum. This rare occurrence has been described1 and is probably the pathogenesis of pneumomediastinum in our case.

Subcutaneous emphysema developing as a complication of arthroscopy is rare. Only a few cases3-6 have been reported in the literature and all of them involved arthroscopy of the knee. Three of these patients3-5 had pneumoperitoneum but none had pneumomediastinum. Continuous inflow of saline solution into the joint was maintained by an infusion pump in our patient. As described by Henderson and Hopson,4 a loose junction between the collapsible plastic bags of saline solution and the inflow tubing has been implicated as the portal of entry of the air. The pumps injected the mixture of saline solution and air into the shoulder, and because of the multiple puncture sites in the capsule, both saline solution and air were extravasated, resulting in subcutaneous emphysema.

This case illustrates that arthroscopy of the shoulder, a procedure seemingly unrelated to the respiratory system, may cause potentially serious pulmonary complications. It also alerts us that subcutaneous emphysema may lead to pneumomediastinum.

ADDENDUM
Since acceptance of this manuscript, three similar cases have been reported by Lee et al (Chest 1992; 101:1265-67)

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Myocarditis as a Cause of Primary Right Ventricular Failure*

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Patients with cardiomyopathy secondary to inflammatory myocarditis usually present with biventricular signs and symptoms. On occasion, the disease may progress focally, with left ventricular involvement predominating. This patient had elevated neck veins, edema, and a dilated right ventricle in the absence of left ventricular abnormalities. At autopsy, diffuse, transmural fibrosis of the right ventricle was found, consistent with end-stage myocarditis, with minimal disease of the left ventricle. This case emphasizes that the clinical manifestations of myocarditis can be limited to the right ventricle and should be considered in the differential diagnosis of right ventricular failure.

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Myocarditis accounts for a significant percentage of patients presenting with idiopathic biventricular congestive cardiomyopathy.1 Because it has been assumed that the inflammatory process involves both right and left ventricles to an equal extent, the diagnosis can often be facilitated by right ventricular endomyocardial biopsy specimens.2-3 In a limited number of cases, however, left heart symptoms predominate and the histologic changes can be shown only in the left ventricle.4 This is consistent with some work that the inflammatory process may be focal, particularly in its early course.

Despite its focal nature, myocarditis has rarely been reported as a cause of right ventricular failure in the absence of abnormalities of the left side of the heart. In this case, we describe such a patient who presented with primary right ventricular failure and on autopsy was found to have end-stage fibrosis of the right ventricle and only patchy myocarditis of the left ventricle.

CASE REPORT
A 67-year-old woman was referred to our hospital for increasing abdominal girth, edema, and lethargy over the past 6 months. She was considered to have right ventricular failure with secondary tricuspid insufficiency and had been treated conservatively by her local physician with bed rest, diuretics, and digoxin. Her medical history was significant for placement of a permanent pacemaker 8 years prior to hospital admission, at which time she presented with near syncpe and atrial fibrillation with a "slow ventricular response." The cause of the conduction disease was assumed idiopathic and no further workup was done. Her cardiovascular history was otherwise unremarkable and she denied symptoms of chest pain, shortness of breath, or fevers.

On examination, she was cachectic and afibrile, with a pulse of 76 beats per minute, a blood pressure of 120/60 mm Hg, and a respiratory rate of 16 per minute. Jugular venous distention was noted at the angle of the jaw and her liver was pulsatile and spanned 15 cm by palpation. Shifting dullness and bilateral pretilial edema were also observed. Her lungs were clear to auscultation and her cardiovascular examination showed a precordial lift with a variable S1, a physiological split S2, and a third heart sound. In addition, a 3/6 pansystolic blowing murmur was heard, just left of the sternum.

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