Surgical Decompression of a Tension Pneumomediastinum* 
A Ventilatory Complication of Status Asthmaticus

H. F. Shennib,† A. N. Barkun,‡ E. Matouk∥ and P. E. Blundell¶

A case of status asthmaticus was associated with cardiorespiratory arrest, illustrating rarely reported complications of ventilatory therapy including tension pneumomediastinum and coronary air embolism. Proposed pathophysiologic mechanisms and recommendations for surgical management are discussed.

Mortality from asthma in North America remains significant despite the advent of pharmacologic and ventilatory therapy. In mechanically ventilated patients, barotraumatic complications are well described and thought to be related to high inspiratory pressures.

We report the occurrence of a tension pneumomediastinum in the context of status asthmaticus—an entity which, although experimentally shown over 50 years ago, has not since been described, to our knowledge.

CASE REPORT

A 23-year-old man who had been suffering from asthma for 16 years walked into the emergency department complaining of gradually worsening dyspnea of four hours' duration. He was noncompliant to therapy with both oral oxytriphylline and aerosolized salbutamol.

Laboratory values were as follows: blood pressure, 140/90 mm Hg; pulsus paradoxus, 12 mm Hg; pulse, 108 beats/min; and respirations, 26/min. No visible jugular venous distention was seen, and he was able to talk comfortably. Bilateral expiratory wheezes were noted. The remainder of the physical examination was noncontributory.

 Spirometric tests showed an FEV, of 0.6 L and FVC of 1.9 L (the best results recorded one month previously were 2.25 L and 4.75 L, respectively). The initial chest roentgenogram showed hyperinflation but was otherwise interpreted as normal. Although intravenous aminophylline and methylprednisolone, aerosolized salbutamol, and subcutaneous epinephrine, the patient progressed to respiratory failure. An arterial blood gas analysis showed pH, 7.17; Pco2, 73; and Po2, 54.

The patient underwent an uncomplicated orotracheal intubation and soon after exhibited mediastinal and subcutaneous emphysema in the supraclavicular area; a repeated chest x-ray film showed bilateral small pneumothoraces were also seen and bilateral chest tubes were inserted (Fig 1).

He soon suffered complete cardiovascular collapse, and cardiopulmonary resuscitation was started. A diagnosis of left tension pneumothorax was made clinically. It was immediately decompressed with a 16-gauge needle, and the chest tube was replaced on the left side.

Despite clinical disappearance of the tension pneumothorax, and although both chest tubes were now draining air freely, the patient remained in electromechanical dissociation (EMD). There was no evidence of hypovolemic or cardiogenic shock as shown by bedside assessment and ECG recording, but we could not rule out the possibility of a large pulmonary embolism as being responsible for the hypotension.

He exhibited a superior vena cava (SVC) syndrome, and we questioned the possibility of cardiac tamponade of unclear etiology, the patient was rushed to the operating room.

Insertion of a central line demonstrated a markedly elevated central venous pressure. A transverse suprasternal incision was made, and digital probing extruded a significant amount of mediastinal air as well as a small amount of blood but failed to decompress his mediastinum. It was decided to perform a full sternotomy. This revealed strikingly hyperinflated, noncompliant lungs protruding from the chest cavity, and air bubbles within the coronary vessels.

Table 1—Intraoperative and Postoperative Pharmacotherapy

<table>
<thead>
<tr>
<th>Medication</th>
<th>Route</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aminophylline*</td>
<td>IV</td>
<td>0.5 mg/kg/hr</td>
</tr>
<tr>
<td>Methylprednisolone*</td>
<td>IV</td>
<td>125 mg q 6 hr</td>
</tr>
<tr>
<td>Salbutamol</td>
<td>Aerosol</td>
<td>0.5 ml in normal saline, 2.5 ml, q 4 hr</td>
</tr>
<tr>
<td>Salbutamol*</td>
<td>IV</td>
<td>250 μg bolus × 2 doses followed by 10-15 μg/min drip</td>
</tr>
<tr>
<td>Ipratropium*</td>
<td>Aerosol</td>
<td>80 μg q 6 hr</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>Inhalation</td>
<td>Anesthetic dose</td>
</tr>
<tr>
<td>Ephedrine</td>
<td>IV</td>
<td></td>
</tr>
<tr>
<td>Fentanyl</td>
<td>IV</td>
<td>100 μg—1 dose</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>IV</td>
<td>1 mg—2 doses</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Transbronchial</td>
<td>1 mg—1 dose</td>
</tr>
</tbody>
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*From the Divisions of Internal Medicine, Cardiovascular and Thoracic Surgery, and Respirology, The Montreal General Hospital, McGill University, Montreal, Quebec, Canada.
†Associate Physician, Division of Respirology.
‡Senior Resident in Cardiovascular and Thoracic Surgery.
¶Chief Medical Resident.
∥Director, Cardiovascular and Thoracic Surgery.

*Drugs continued during the postoperative period.

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Immediately after this maneuver, the patient's blood pressure was restored, and all signs of the SVC syndrome disappeared. Attempts at reducing the lungs and closing the mediastinum led to recurrence of hypotension, and it was elected to leave the chest cavity open. A temporary nylon patch was draped over the lungs and sewn onto the edge of the skin incision, thus overlying and protecting the exposed intrathoracic organs.

Intraoperative bronchoscopic examination revealed no mucous plugging in the large airways, and intensive pharmacotherapy was required to diminish the severe bronchospasm (Table 1), as intraoperative inspiratory pressures reached 75 cmH₂O.

Postoperatively, both the patient's blood gas values and inspiratory pressures gradually improved. The lungs were placed back in the chest cavity, and the incision was closed on the fourth day of admission.

Presumably due to prolonged hypotension, although the possibility of cerebral air embolism could not be excluded, the patient exhibited no cerebral activity clinically or on serial EEGs. He was declared dead on the sixth hospital day and autopsy permission was not granted.

**DISCUSSION**

The mortality of status asthmaticus is 1 percent; there is a three- to eight-fold higher incidence of barotrauma in patients given ventilatory support for this condition.

Our patient developed well recognized complications of positive-pressure ventilation, including bilateral pneumothoraces, subcutaneous emphysema, and pneumoperitoneum.

Despite adequate decompression of his left tension pneumothorax, the patient remained in EMD and seemed to respond only to mediastinal air decompression and removal of the lungs from within the chest cavity.

The pathophysiology of pneumomediastinum was described by Macklin and Macklin. To our knowledge, this is the first case of tension pneumomediastinum with severe interstitial pulmonary emphysema described in association with asthma.

We believe that, as Macklin and Macklin postulated, the severe interstitial pulmonary emphysema may have compressed the pulmonary venules and also caused the development of a tension pneumomediastinum. The resulting decreased venous return was probably responsible for the EMD. The SVC syndrome presumably was attributable to its compression by the tension pneumomediastinum and the hyperinflated, stiff lungs.

At surgery, air bubbles were noted in coronary vessels; air embolism has been noted in asthma previously. This complication denotes the presence of bronchovenous or pleurovenous fistulas, which may occur either spontaneously or in association with cardiopulmonary resuscitation as previously reported.

The difficulty in clearly establishing this sequence of events both in vivo and at autopsy may explain the paucity of similar reports in the literature; perhaps a fraction of both out-of-hospital and unexplained in-hospital sudden deaths might be attributable to such complications.

We suggest that in the face of sudden cardiovascular collapse in an intubated asthmatic patient and in the absence of improvement after adequate chest tube drainage, if no other obvious causes of EMD such as hypovolemic or cardiogenic shock are found, more aggressive decompressive therapy such as suprasternal or subxiphoid incisions for release of mediastinal emphysema should be contemplated, and sternotomy with a view to mediastinal decompression should be considered.

As shown in this case, severe bronchospasm with interstitial emphysema may not allow reduction of the lungs in the chest cavity immediately. To circumvent this problem, the sternum should be left open with a sheath of soft, impermeable material applied over the sternotomy site and sewn to the skin edges, permitting delayed closure a few days later.

The causes of sudden death in asthma are controversial, but one of the main contributory factors, as documented in autopsy series, seems to be extensive mucous plugging. In this case its absence is interesting, at least in the large airways as demonstrated by bronchoscopic examination. This may reflect the acute onset of the episode, although we cannot of course rule out the presence of mucus in the smaller airways without an autopsy.

Although pneumomediastinum is almost always a benign condition, this case illustrates the potential lethality of barotrauma in ventilated patients with status asthmaticus. It is only with greater awareness of this complication that early, aggressive intervention may lead to its successful therapy.

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**Pulmonary Hyperinflation following Nd-YAG Laser Resection of an Obstructing Mainstem Tumor**

Walter R. Fairfax, M.D.; and R. James Rollins, M.D.

*From the Division of Respiratory Disease and Critical Care Medicine, Department of Internal Medicine, and The Laser Endoscopy Laboratory, University of Utah College of Medicine, Salt Lake City.
†Assistant Professor of Internal Medicine.
‡Instructor in Internal Medicine.

Reprint requests: Dr. Fairfax, 50 North Medical Drive, Salt Lake City, Utah 84132