Bronchiectasis following Heroin Overdose*

A Report of Two Cases

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Two patients developed respiratory insufficiency following episodes of heroin overdose complicated by aspiration. In both instances pulmonary function testing demonstrated marked obstructive airway disease and arterial hypoxemia. Bronchograms performed on both of these patients demonstrated bronchiectasis. The persistence of respiratory insufficiency into the recovery phase of heroin pulmonary edema may indicate the onset of chronic pulmonary disease. Pathology of the bronchial tree should be looked for in evaluating this syndrome.

The majority of complications that occur in the acute phase of heroin overdose involve the lung; in particular, these complications are pulmonary edema, pneumonia, pulmonary emboli, and aspiration pneumonia.1-5

Recent investigation of heroin addicts following overdose reveals that they may develop chronic pulmonary changes; these changes express themselves as impairment in pulmonary function.6

That chronic forms of pulmonary disease have not been more frequently reported following the acute insults to the pulmonary system associated with heroin may be a function of the familiar difficulties in following heroin addicts beyond the phase of their acute illness. The patients presented in this report demonstrate a pathologic entity, bronchiectasis, which may easily be anticipated as a late sequela following heroin overdose.

CASE REPORTS

Case 1

A 20-year-old man was admitted to the Naval Hospital, St. Albans, New York on November 3, 1971, stuporous and in acute respiratory distress.

Questioning of the patient’s companions revealed that he was a heroin addict, who on the night of admission administered an intravenous dose of heroin to himself and became unresponsive within 30 minutes. In an attempt to revive the patient his friends administered vinegar orally. This resulted in repeated episodes of vomiting.

Physical examination upon admission revealed a stuporous man, tachypneic, with a pulse rate of 120. Auscultation of the chest revealed diffuse wheezes and rhonchi; there was no murmur. The extremities demonstrated cyanotic nail beds, as well as thrombosed veins in the antecubital fossa. The neurologic examination revealed the patient to be stuporous, his pupils were pinpoint and unreactive to light, and his gag reflex was absent.

The initial laboratory examinations revealed a white blood count of 23,000 cells per mm³ with a normal differential. An arterial blood sample revealed a pH of 7.36, an oxygen tension (PaO₂) of 31 mm Hg, and a carbon dioxide tension (PaCO₂) of 30 mm Hg. Chest roentgenogram demonstrated bilateral pulmonary infiltrates (Fig 1A). Electrocardiogram revealed sinus tachycardia.

The patient was admitted to the intensive care unit where 100 percent oxygen was administered under positive pressure through an endotracheal tube. Because of the history of acute aspiration the patient was begun on kanamycin, 1 gm, intramuscularly, daily; cephalothin (Keflin) 2 gm intravenously, every six hours; and hydrocortisone, 1 gm intravenously, daily in divided doses.

By the second hospital day the patient’s PaO₂ rose to 93 mm Hg. On the fourth hospital day the patient was extubated, and thereafter oxygen was delivered by face mask.

The patient’s initial sputum culture grew Staphylococcus aureus, coagulase positive, and antibiotic administration was continued for 18 days. Steroid administration was gradually tapered, and discontinued after three weeks.

Despite the apparent success of the patient’s initial therapy, serial chest roentgenograms continued to demonstrate diffuse parenchymal infiltrates during this phase of his hospitalization (Fig 1B). Pulmonary function testing was performed repeatedly, and demonstrated both marked restrictive and obstructive airway disease, with severe hypoxemia (Table 1).

The patient’s clinical course was characterized by a 30 lb weight loss, and intractable coughing productive of copious

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amounts of yellow phlegm. Therapy consisted of postural drainage, intermittent courses of antibiotics, and oxygen administered by nasal cannula.

Chest roentgenograms, as well as full chest tomograms performed in January 1972, demonstrated peribronchial thickening, and bilateral varicose dilatation of the bronchial tree (Fig 2). These findings were felt to be compatible with bronchiectasis, and this suspicion was confirmed by bronchograms performed on June 14 (Fig 3).

During his recovery it was learned that the patient had no prior history of pulmonary disease. At present, eight months after his initial insult, the patient remains dyspneic at rest, able to ambulate only short distances when unassisted.

Case 2

A 19-year-old youth was transferred to the Naval Hospital, St. Albans on April 27, 1972 complaining of a productive cough and dyspnea at rest.

The patient had initially been admitted to another hospital on April 4, comatose and cyanotic following a heroin overdose complicated by aspiration. An initial chest roentgenogram demonstrated bilateral infiltrates which were interpreted as pulmonary edema. The patient's initial therapy included intubation, oxygen delivered by intermittent positive pressure, and intravenous antibiotics and steroids. Serial roentgenograms demonstrated slow resolution.

The patient improved gradually over a period of three weeks, but was still markedly dyspneic at the time of his transfer. Physical examination at that time was remarkable by the presence of rhonchi throughout both lung fields, and the presence of cyanosis.

Laboratory studies included a normal chest roentgenogram. Pulmonary function studies performed on May 3 demonstrated marked restrictive and obstructive airway disease with severe arterial hypoxemia (Table 1). Bronchograms were performed on May 8 and revealed bronchiectasis involving the right lower lobe (Fig 4).

Table 1—Pulmonary Function and Arterial Blood Gas Determinations

<table>
<thead>
<tr>
<th>Date</th>
<th>Conditions of Study</th>
<th>Vital Capacity</th>
<th>Arterial Blood Gases</th>
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<tr>
<td></td>
<td></td>
<td>Vol, ml  % Predicted FEV1%</td>
<td>pH</td>
</tr>
<tr>
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<td>Room air</td>
<td>2195 39 50</td>
<td>7.58 49 25</td>
</tr>
<tr>
<td>11-11-71</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12-2-71</td>
<td>Room air</td>
<td>1903 51 51</td>
<td>7.51 47 42</td>
</tr>
<tr>
<td>2-17-72</td>
<td>Room air</td>
<td>2592 48 45</td>
<td>7.44 44 33</td>
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<tr>
<td>Case 2</td>
<td>Room air</td>
<td>2270 44 57</td>
<td>7.52 43 30</td>
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<tr>
<td>5-3-72</td>
<td>Room air</td>
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<td>Serial Blood Gas Studies</td>
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</tr>
<tr>
<td>Case 1</td>
<td>Room air</td>
<td>2195 39 50</td>
<td>7.44 56 38</td>
</tr>
<tr>
<td>12-27-71</td>
<td>5 min of forced hyperventilation</td>
<td>7.43 58 31</td>
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<tr>
<td>12-27-71</td>
<td>5 min exercise</td>
<td>7.37 52 39</td>
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</tr>
<tr>
<td>12-27-71</td>
<td>100% oxygen</td>
<td>7.40 480 42</td>
<td></td>
</tr>
<tr>
<td>Case 2</td>
<td>100% oxygen</td>
<td>7.52 520 40</td>
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The patient's dyspnea remained unchanged throughout the remainder of his hospital stay. He was separated from military service because of pulmonary disability two months after his acute episode.

DISCUSSION

Most patients suffering from heroin overdose and associated pulmonary infiltrates recover spontaneously from the acute phase of a given episode. A significant number succumb to relentless pulmonary insufficiency during the same phase.1-5 The patients described in this report differ from both patterns in that their "pulmonary edema" persisted well beyond the acute stage of their overdoses.

FIGURE 3, Case 1. Bronchogram performed June 14, 1972 demonstrating saccular bronchiectasis.

FIGURE 4, Case 1. Bronchogram performed May 8, 1972 demonstrates bronchiectasis in the right lower lobe.
The pathogenesis of pulmonary infiltrates seen with heroin overdose remains unknown for the most part. The frequency of aspiration, resulting either from a depressed gag reflex, or the attempts on the part of the addict's friends to administer an antidote in the form of milk, or as in case 1, vinegar, has been noted often.

The pathologic changes that occur with aspiration have been detailed in animal and clinical studies. In addition to bronchiolitis obliterans that occurs in the acute phase, and may account for the pulmonary fibrosis in the later stages, a direct necrotizing effect of the aspirated material is produced on the bronchial tree. Both obstruction and infection are common sequelae to aspiration, and this combination is well recognized to be the forerunner of bronchiectasis.

Review of the literature reveals only one other case in which bronchiectasis was associated with acute heroin intoxication and aspiration. This report described a patient who died 33 days after acute heroin intoxication. Autopsy findings disclosed diffuse bronchiectasis with an ulcerated bronchial mucosa. That such a sequence of pathologic events can and does occur in patients who survive heroin pulmonary edema and aspiration is illustrated by our patients.

In view of the frequency of pulmonary infiltrates and aspiration associated with acute heroin intoxication, it may be anticipated that a reservoir of chronic pulmonary disease might be forming in that segment of the addict population that has survived acute episodes of drug overdose. Bronchiectasis may be one of the prominent sequelae of this syndrome.

REFERENCES