Massive air embolism is almost uniformly fatal. If recognized, possible therapeutic maneuvers include: stopping the source of air if possible; placing the patient in the head down, left lateral decubitus position; aspirating the air on the right side with a central venous catheter; administering 100 percent oxygen; and transferring the patient to a hyperbaric oxygen chamber. Under special circumstances, emergency thoracotomy, cardiopulmonary bypass and direct cardiac and arterial aspiration may be indicated.

Massive air embolism is a rare complication of positive pressure ventilation and should be considered in the differential diagnosis in patients who sustain sudden circulatory collapse while on positive pressure ventilation.

REFERENCES

1 Kizer CB, Goodman PC. Radiographic manifestations of venous air embolism. Diagnostic Radiol 1982; 144:35-9
2 Hart GB. Treatment of decompression illness and air embolism with hyperbaric oxygen. Aerospace Med 1974; 45:1190-93
6 Albin MS. The paradox of paradoxic air embolism—PEEP, Valsalva, and patent foramen ovale. Should the sitting position be abandoned? Anesthesiology 1984; 61:222-23
10 Kogutt MS. Systemic air embolism secondary to respiratory therapy in the neonate: Six cases including one survivor. AJR 1978; 131:425-29
14 Macklin CC. Transport of air along sheaths of pulmonic blood vessels from alveoli to mediastinum. Arch Intern Med 1939; 64:913-26
15 O’Quin RJ, Lakshminarayan S. Venous air embolism. Arch Intern Med 1982; 142:2173-76

Pulmonary Hypertension in a Patient with Rheumatoid Arthritis*

Junisieiro Morikawa, M.D.; Kazuto Kitamura, M.D.; Yoshitomi Higuchi, M.D.; Yoshitomi Tsujimura, M.D.; Tetsujiro Minamikawa, M.D.; and Tetsuro Takamatsu, M.D.

A 53-year-old woman who had suffered from severe rheumatoid arthritis developed pulmonary hypertension. Her small arteries in the lung showed plexogenic arteriopathy with fibrous intimal hyperplasia. There was also vasculitis of the small arteries in other organs and mural thrombosis in the pulmonary stem and abdominal aorta. The plexogenic arteriopathy which was responsible for pulmonary hypertension appears to be the result of vasculitis in association with rheumatoid arthritis.

It is well known that collagen disease is associated with vasculitis, but pulmonary vasculitis in association with rheumatoid arthritis (RA) is rare. We report a case of pulmonary hypertension which is attributed to pulmonary vasculitis with RA.

CASE REPORT

A 53-year-old woman with 19-year history of RA was admitted to hospital for cough and anasarca. Heart rate was 68 beats per minute and regular. Blood pressure was 124/90 mm Hg. Jugular veins were markedly dilated. Parasternal heave was present and the pulmonary second sound was palpably accentuated. Holosystolic murmur (grade 2/6) was heard on the lower left sternal border. Marked edema of both upper and lower extremities was present. Her hands and feet had the classic, advanced deformities of RA. The chest x-ray films and cardiac series showed cardiomegaly with right ventricular enlargement. The main pulmonary artery and its main branches were enlarged with pronounced peripheral attenuation. The ECG exhibited right ventricular hypertrophy and right atrial enlargement. The echocardiogram also showed marked right ventricular enlargement and paradoxic movement of interventricular septum. At the pulmonic valve, depth of A wave was 1 mm, E-F slope 2 mm/s and maximal opening slope 390 mm/s. Mid-systolic rapid semi-closure followed by reopening was seen. Perfusion lung scan using 99mTc labeled macroaggregated albumin revealed no perfusion defect. The hand x-ray film showed osteoporosis, bony destruction, joint deformity, and ankylosis (Fig 1).

Laboratory tests revealed positive RA test (+2), increased gammaglobulin, mainly of IgG component, positive LE-cell and LE-phenomenon, and positive antinuelear antibody. Arterial gas analysis showed mild respiratory alkalosis with pH of 7.48, Pco2 of 26.7 mm Hg, and Po2 of 79.9 mm Hg, respectively.

According to the diagnostic criteria of the American Rheumatism Association, the case was diagnosed as "classical RA," and therapeutically classified as "stage 4" in progression, "class 4" in functional capacity, because of osteoporosis, destruction of the bone and cartilage, and deformity plus ankylosis of the joint.

Cough and anasarca improved by therapy with a diuretic, but the patient continued to complain of severe dyspnea. Therefore, digitalis and more diuretic medication were administered, but she died suddenly. Cardiac catheterization could not be performed.

*From the Third Department of Internal Medicine, the Department of Laboratory Medicine, and the Second Department of Pathology, Kyoto Prefectural University of Medicine, Kyoto, Japan. Reprint requests: Dr. Morikawa, Third Department of Internal Medicine, Kyoto Prefectural University, Kusaramachi Hirokoi Kamikyo-ku, Kyoto 602, Japan
Now you can achieve superior safety and efficacy in the treatment of chronic bronchitis and emphysema.
Superior efficacy vs. albuterol¹*

For first-line maintenance in newly diagnosed patients³

For first-line maintenance in long-term therapy as demonstrated in multicenter study²
Superior safety and minimal side effects

Not a single incidence of tremor or tachycardia in long-term multicenter studies.

ATROVENT is relatively free of systemic anticholinergic side effects.

ATROVENT is relatively free of many of the side effects common to sympathomimetics and theophyllines.

PERCENT OF PATIENTS (>1%) EXPERIENCING ADVERSE REACTIONS (N = 254)

<table>
<thead>
<tr>
<th>Category</th>
<th>Adverse Effects</th>
<th>%</th>
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<tr>
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<tr>
<td>CNS</td>
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<tr>
<td></td>
<td>Dizziness</td>
<td>2.4</td>
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<tr>
<td></td>
<td>Headache</td>
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<tr>
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<td>Rash</td>
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<tr>
<td>Gastrointestinal</td>
<td>Nausea</td>
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<tr>
<td></td>
<td>G.I. distress</td>
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<tr>
<td>Ophthalmological</td>
<td>Blurred vision</td>
<td>1.2</td>
</tr>
<tr>
<td>Oro-Otaryngeal</td>
<td>Dry mouth</td>
<td>2.4</td>
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<tr>
<td></td>
<td>Irritation from aerosol</td>
<td>1.6</td>
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<tr>
<td>Respiratory</td>
<td>Cough</td>
<td>5.9</td>
</tr>
<tr>
<td></td>
<td>Exacerbation of symptoms</td>
<td>2.4</td>
</tr>
</tbody>
</table>

*As demonstrated in a double-blind triple crossover study of ipratropium bromide 40 mcg, albuterol 180 mcg and placebo.

ATROVENT is not indicated in the initial treatment of acute episodes of bronchospasm where rapid response is required.

ATROVENT should be used with caution in patients with narrow-angle glaucoma, prostatic hypertrophy or bladder-neck obstruction.

Now you can achieve superior safety and efficacy in the treatment of chronic bronchitis and emphysema.

Please see following page for brief summary of prescribing information.
ATROVENT®
(ipratropium bromide)
INHALATION AEROSOL
18 mcg per puff
For superior safety and efficacy in the treatment of chronic bronchitis and emphysema

Ipratropium bromide
Percent of Patients

<table>
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<th>Reaction</th>
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<th>Metaproterenol sulfate</th>
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<td>Tremor</td>
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<td>3.6</td>
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</table>

Additional adverse reactions reported in less than one percent of the patients considered possibly due to Atrovent® (ipratropium bromide) include urinary difficulty, fatigue, insomnia, hoarseness, and difficulty in accommodation.

The large uncontrolled, open-label study included seriously ill patients. About 7% of patients treated discontinued the program because of adverse events. Of the 2301 patients treated in the large uncontrolled study and in clinical trials other than the 90 day studies, the most common adverse reactions reported were: dryness of the oropharynx, about 5 in 100; cough, exacerbation of symptoms and irritation from aerosol, each about 3 in 100; headache, about 2 in 100; nausea, dizziness, blurred vision, difficulty in accommodation, and drying of secretions, each about 1 in 100. Less frequently reported adverse reactions that were possibly due to Atrovent include tachycardia, paresthesias, drowsiness, coordination difficulty, itching, hives, flushing, alopecia, constipation, tremor, mucosal ulcers.

Cases of precipitation or worsening of narrow-angle glaucoma, acute eye pain and hypotension have been reported. A case of giant urticaria with positive rechallenge has been reported from the foreign marketing experience (see CONTRAINDICATIONS).

HOW SUPPLIED Atrovent® (ipratropium bromide) Inhalation Aerosol is supplied as a metered dose inhaler with mouthpiece. Each 14 gram vial provides sufficient medication for 200 inhalations. NDC 0597-0082-14. Each actuation delivers 18 mcg of ipratropium bromide from the mouthpiece.

Consult package insert before prescribing.

FIGURE 1. The hand x-ray film shows osteoporosis, bony destruction, joint deformity, and ankylosis.

At necropsy, pleural spaces were totally obliterated by serofibrinous pleuritis. The lungs were congested and microscopically showed widespread obstructive lesions of the pulmonary vasculature, including intimal fibrous proliferation and plexiform lesions of small muscular arteries and arterioles. The plexiform lesions were associated with fibrinous microthrombi. Lymphocyte infiltration and destruction of the elastic lamina were seen (Fig 2).

Mild fibrosis of the peribronchial region and the alveolar septa was also present. There was no evidence of rheumatoid pneumoconiosis or rheumatoid nodules.

The pericardium was adhesive due to serofibrinous pericarditis without effusion. The heart was enlarged, weighing 460 g. The dilated right ventricle was hypertrophied with a wall thickness of 7 mm. The tricuspid valve ring was also dilated. There was no congenital or valvular disease. At the left main pulmonary artery, there was a large organizing thrombus, measuring 1.5 cm in diameter and 6.5 cm in length. Moreover, other large thrombi were found in the abdominal aorta, and left external and internal iliac arteries. Lymphocyte infiltration was represented around the vasa vasorum of these arteries and aorta (Fig 3, left).

The survey of the deep venous system of the legs revealed no thrombi.

The examination of the salivary glands revealed chronic inflammatory cell infiltration around the small arteries with intimal fibrous proliferation and medial hypertrophy (Fig 3, right).

DISCUSSION

This patient had well documented RA. Pulmonary hyper-
Bilateral Otorrhagia Associated with Continuous Positive Airway Pressure*

Lindell K. Weaver, M.D.,† Walter B. Fairfax, M.D.,‡ and Loren Greenway§

A patient had bilateral tympanic membrane rupture and otorrhagia, an unusual complication of continuous positive airway pressure (CPAP). CPAP, applied by a bag-mask system using disposable spring valves, was used to treat acute pulmonary edema during volume resuscitation and vasopressin therapy for bleeding from esophageal varices.

Continuous positive airway pressure (CPAP) is frequently used to improve arterial oxygenation, to treat adult respiratory distress syndrome and atelectasis, and to avoid or delay endotracheal intubation in spontaneously breathing patients. We present a case of bilateral tympanic membrane rupture associated with CPAP.

CASE REPORT

A 49-year-old man was admitted with recurrent upper gastrointestinal bleeding. The patient was alert, and his examination was unremarkable. The tympanic membranes and auditory canals were normal. He had adequate arterial oxygenation while breathing supplemental oxygen at 4 L/min by nasal cannula.

Shortly after admission, the patient had profuse hematemesis associated with hypotension, which was treated with intravenous (IV) blood and crystalloid administration, as well as intravenous administration of vasopressin. Approximately 30 minutes later, the patient was drowsy, orthopneic, agitated, and vigorously coughing (blood pressure was 220/120 mm Hg). Fine rales were heard in the lung bases and arterial blood gas levels demonstrated moderate hypoxemia. The patient was given furosemide, morphine sulfate, and diazepam intravenously; pitressin was discontinued. The fractional inspired concentration of oxygen (FiO₂) was increased to 0.50 by face mask. The patient experienced progressive dyspnea. A self-inflating adult silicone bag-valve resuscitator with 2,600 ml reservoir (Laerdal) incorporating a 5 cmH₂O Down's CPAP valve (Vital Signs) was connected to the exhalation port of the expiration diverter (Laerdal valve system designed to separate inhalation from exhalation, part number 85-05-00). This was attached to a Downs face mask (Life Designs Systems) with the exhalation port sealed and firmly applied to the patient's face. CPAP was continuously measured with a manometer. The patient was breathing spontaneously with CPAP (5 cm H₂O) with occasional coughing until he was pharmacologically sedated. Assisted ventilation was begun with the CPAP bag-valve-mask prior to oral-tracheal intubation for hypercapnia (pH 7.26; PaCO₂ 45; PaO₂ 61; SaO₂ 98; FiO₂ 0.50 presumed to be 1.00; 15 L/min O₂ flow). A chest roentgenogram showed pulmonary edema. The patient improved. He was extubated the following day, and his pulmonary edema lessened over the next week. Immediately after intubation, the patient was found to have...

REFERENCES


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Bilateral Otorrhagia and CPAP (Weaver, Fairfax, Greenway)