CASE REPORT

Adult Respiratory Distress Syndrome Caused by Inhalation of Oxides of Nitrogen

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Abstract

We experienced a case of permeability pulmonary edema (ARDS) caused by massive inhalation of oxides of nitrogen. Using a flow-directed pulmonary artery catheter (Swan-Ganz), we monitored the systemic and pulmonary circulation and searched for evidence of pulmonary microembolism by carrying out balloon-occluded pulmonary angiography (BOPA) at the bed-side. Then we observed coagulation disorders, which may be found at a high rate of incidence in ARDS, and it is interesting to note that these disorders occurred during the same period as the appearance of microemboli in BOPA. We carried out peep respiratory management at 5–10 cm H₂O with a respirator. Massive administration of methylprednisolone, and heparin administration were carried out. The patient was disconnected from the respirator on the tenth day, and he was discharged about one month after injury.

Key words: ARDS, ballon-occluded pulmonary angiography, coagulation disorder, pulmonary microembolism

Introduction

We treated a sixty year-old man, who was stricken with pulmonary edema after inhaling large amounts of gaseous N₂O, N₂O₃, NO₃, which were released when he acci-
dentally placed an iron plate into concentrated nitric acid. We carried out monitoring of the patient's hemodynamics by inserting a flow-directed pulmonary artery catheter, and considered pulmonary microembolism as a cause of pulmonary hypertension and carried out BOPA and examinations of blood coagulation. The patient was saved from respiratory failure approximately 10 days after the injury.

Case Report

The patient, a sixty year-old male, inhaled gases (N₂O, N₂O₃, NO₃) for about fifteen minutes, which were released when he accidentally placed an iron plate into concentrated nitric acid while working. At that time, the patient did not have any subjective symptoms, but about 5 hours later, he suddenly experienced acute dyspnea and was carried to the Saiseikai Kanagawa-ken Hospital. His consciousness at admission was clear, and his blood pressure was 120/50 mmHg. The pulse rate was 110 beats/

Fig. 1  Chest X-ray on admission (5 hours after injury)
A diffuse, bilateral infiltration was shown, but cardiac enlargement was not evident.
minute, and remarkable cyanosis was observed on the nail beds. Moist rales were heard over both lung fields. Diffuse, bilateral infiltration was observed on the chest X-ray, but cardiac enlargement was not evident (Fig. 1).

The arterial blood gas analysis with room air breathing showed pH 7.349, PaO₂ 37.1 mmHg, PaCO₂ 45.6 mmHg and B.E. -4.7. Tracheostomy was immediately carried out, upon which large amounts of pink, foamy sputum could be suctioned out. One hour after admission (6 hours after injury), the patient was connected to a respirator, and control mandatory ventilation was carried out at FiO₂ 1.0 with peep of 5 cm H₂O. The arterial gas partial pressure values improved to PaO₂ 77.0 mmHg and PaCO₂ 49.8 mmHg. We inserted a flow-directed pulmonary artery catheter (Swan-Ganz) and moni-

Fig. 2 BOPA on the day four of injury
A pulmonary artery filling defect (an arrow) was found.
tored the systemic and pulmonary circulation. Pulmonary arterial pressure was 38/27 mmHg and pulmonary vascular resistance was 409.1 dynes/sec.cm⁻⁵ 9 hours after injury.

We considered the possibility of pulmonary microembolism as the reason for pulmonary hypertension, and pulmonary angiography was carried out at the bedside, using the method which was roughly similar to the BOPA described by Greene, et al.¹ The balloon of the Swan-Ganz catheter was inflated, and while keeping an eye on pulmonary arterial pressure wave form, the tip of the catheter was wedged into the pulmonary artery. Thereafter, 20 ml of 65% amidotrizoate meglumine was manually injected at a speed of approximately 1 ml/sec. via the same catheter and X-ray photographs were taken immediately after, and approximately 2 seconds after injection. Clear pictures were obtained of the pulmonary circulation, from the pulmonary artery until the pulmonary microvasculature in the first X-ray photograph, and from the pulmonary artery until the pulmonary vein in the second one. We carried out this BOPA successively from day two until day seven of injury, and microemboli were observed in the right lower lobe pulmonary artery on day two, four and five of injury (Fig. 2). As the pulmonary artery catheter was wedged into the right upper lobe pulmonary artery at

![Graph showing changes in platelet count, prothrombin time (PT), fibrinogen, and fibrinogen degradation products (FDP) during 9 days after injury. Decreased platelet counts, prolongation of PT, and increased FDP were observed from day two until day five of injury, and pulmonary microemboli were detected by BOPA during the same period.](image-url)
day three of injury, no information could be obtained with respect to the right lower lobe.

During the same period as the appearance of the pulmonary microemboli, the patient was complicated by blood coagulation abnormalities. The platelet count decreased to 43,000/mm³ from the third day of injury, and prolongation of the prothrombin time, decrease in fibrinogen and increased fibrinogen degradation products (FDP) were observed (Fig. 3).

As for the treatment, control mandatory ventilation with a peep of 5–10 cm H₂O was carried out with the respirator. This was switched to intermittent mandatory ventilation on the eighth day of injury, and the patient was weaned from the respirator on the tenth day. Massive administration (4 g/day) of methylprednisolone was commenced immediately after admission. Because the blood coagulation disorders were observed and the microemboli were detected by BOPA, heparin (10,000–20,000 units/day) was given intravenously. On day fourteen of injury the arterial blood gas analysis improved to pH 7.431, PaCO₂ 40.6 mmHg, PaO₂ 70.8 mmHg and B.E. 2.5 on room air. On day twenty-second of injury, the flow-directed pulmonary artery catheter was again inserted and BOPA was carried out. No filling defects were observed and all hemodynamic values were within normal limit.

Discussion

This was a case of permeability pulmonary edema, which occurred by inhalation of oxides of nitrogen.

Greene, et al. have carried out BOPA on 40 cases of acute respiratory failure, and they state that a higher incidence of pulmonary artery filling defect (PAFD) may be observed, the more severe the respiratory failure is, or the higher the pulmonary vascular resistance is. In our case, we observed PAFD on days two, four and five after injury, which roughly coincided with the appearance of blood coagulation abnormalities (Fig. 3). Heparin administration was commenced from day three, and PAFD had disappeared by day six. Hence, the patient may be considered to have responded to heparin administration.

In ARDS, many cases are complicated by blood coagulation abnormalities, and according to Schneider, et al., compared to the control group, the patients with acute respiratory failure have decreased platelet counts which come to about half the values of the control group. Prolongation of the prothrombin time (PT) and partial thromboplastin time (PTT), and increase in FDP may also be observed. In our case, a decrease in fibrinogen and increased FDP were observed (Fig. 3).

Recently, much attention is being paid to high dose corticosteroid therapy in the treatment of ARDS. Sibbald, et al. administered large doses of corticosteroids on ARDS patients, and observed remarkable decrease in alveolo-capillary permeability. Apart from this, the corticosteroids suppress complement-induced neutrophilic aggregation,
and also suppress the damaging of pulmonary vasculature by superoxide radicals.\(^3\)

**References**


