

Multidisciplinary Treatment by Pneumonectomy, PMX and CHDF in a Case of Pulmonary Suppuration Complicated with Septic Shock

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A 68-year-old male, who had suffered from pulmonary tuberculosis with cavities on the right upper lobe, developed breathlessness, bloody sputum, right chest pain and fever. His laboratory data on admission showed severe infection or sepsis (WBC 2,600/mL, CRP 40.2 mg/dL), and his respiratory condition rapidly worsened. In the intensive care unit (ICU) he was given continuous hemodiafiltration (CHDF), but his respiratory condition failed to improve and it was therefore decided to perform a right pneumonectomy. His severe hypoxemia was resolved but because high dose catecholamines medication was still required, polymyxin-B immobilized fiber (PMX) and CHDF were performed. The operation was successful and he was transferred from the ICU to a general ward seven days postoperatively. The vicious circle of septic shock presenting in this case was successfully broken by the pneumonectomy and subsequent treatment by PMX and CHDF, which eliminated the causative factors of sepsis. (Ann Thorac Cardiovasc Surg 2003; 9: 319–22)

Key words: septic shock, pulmonary suppuration, pneumonectomy, blood purification therapy, multidisciplinary treatment

Introduction

Because many aspects of treatment for septic shock¹⁾ are still controversial, it is often very difficult to make a therapeutic strategy. In this paper, we report a case of a 68-year-old male who underwent pneumonectomy and pre- and postoperative blood purification therapy [polymyxin-B immobilized fiber (PMX) and continuous hemodiafiltration (CHDF)] for septic shock due to pulmonary suppuration.

Case Report (Fig. 1)

The patient, who had suffered from pulmonary tuberculosis with cavities on the right upper lobe (Fig. 2) and

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had repeated pneumonia in the same portion, developed breathlessness, bloody sputum, right chest pain and fever two days before admission. The patient's physician pointed out an abnormality on his chest X-ray (Fig. 3) and recommended consulting a respiratory internist in our hospital. Due to diagnosis of pneumonia in the right upper lobe, he was urgently admitted to the Department of Respiratory Medicine of our hospital. His laboratory data on admission indicated severe infection or sepsis, i.e. white blood cell count (WBC) 2,600/mL, CRP (C-reactive protein) 40.2 mg/dL, platelet count 11×10^4 /mL. Despite administration of antibiotic medication, within a few hours his respiratory condition rapidly worsened, blood pressure fell to 86/58 mmHg, pulse rate was 130/min, respiratory rate 30/min and PaO₂ 54.7 torr under 10 L oxygen inhalation. At midnight on the day after admission, he was transferred to the intensive care unit (ICU) in a state of septic shock. Chest radiograph revealed multiple nodules of varying sizes with indistinct margins that were suspected to be septic pulmonary emboli,²⁻⁵⁾ (Fig. 4) but were expanded only on the right side lung field. These abnormal shadows extended rapidly within the right

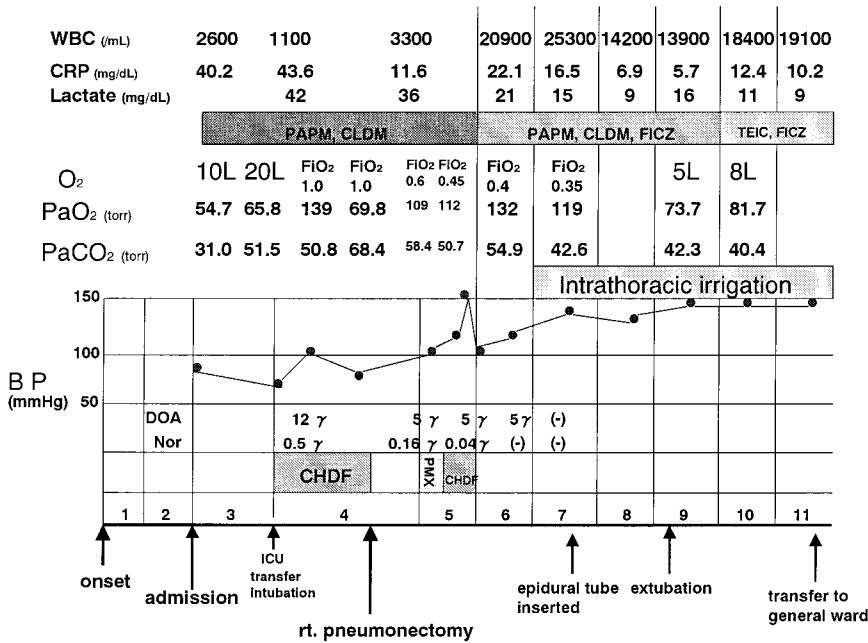


Fig. 1. Clinical course of the patient.

WBC, white blood cell count; CRP, C-reactive protein; BP, blood pressure; DOA, dopamine; Nor, noradrenaline; PAPM, panipenem; CLDM; clindamycin; FICZ, fluconazole; TEIC, teicoplanin; CHDF, continuous hemodiafiltration; PMX, polymyxin-B immobilized fiber.

lung field (Fig. 5). Intratracheal intubation was performed immediately and the patient was put on an artificial respirator and administered catecholamines. These treatments failed to break the vicious circle of sepsis, resulting in renal failure which necessitated CHDF. However, his respiratory condition still did not improve; i.e. PaO₂ 69.8 torr, PaCO₂ 68.4 torr under FiO₂ 1.0, because of a ventilation-perfusion (VA/Q) inequality produced by the intrapulmonary shunt.⁶⁾ In order to break the vicious circle, we decided to perform a right pneumonectomy.

Operation findings

One lung ventilation technique with a double-lumen endotracheal tube was performed and right standard thoracotomy was done. The chest was opened through the fourth intercostal space, however, the entire right lung could not deflate owing to huge retention of pus, which was mixed with blood, and there were severe adhesions between the cavities wall in the upper lobe and parietal pleura. After removal of the adhesions the right main pulmonary artery was stapled and divided with Endo-GIA (United States Surgical Corp., Norwalk, CT, USA) and then the right superior and inferior pulmonary veins were stapled and divided by the same means. As soon as these vessels were separated, PaO₂ jumped from 132 torr to 511 torr under 100% O₂, because of diminishing intrapulmonary shunt. The right main bronchus was stapled and divided with Endo-GIA and the bronchial stump was reinforced with a pericardium flap. Because of severe

adhesions and the inability to deflate the right lung, it took a long time (6 hours) and much bleeding (7,000 mL) to take out the entire right lung.

While his severe hypoxemia dissolved until PaO₂ was 109 torr under FiO₂ 0.6, septic shock condition still continued to the extent that catecholamine medication (noradrenaline 0.16 γ , dopamine 5 γ), was still required. Therefore, PMX (polymyxin-B immobilized fiber⁷⁾) and CHDF were performed. Two days after the operation he could be weaned from catecholamines and two days later from the respirator.

His laboratory data still indicated severe infection or sepsis soon after the operation, i.e. WBC 25,300/mL, CRP 16.5 mg/dL, platelet count 1.3 \times 10⁴/mL, however, four days postoperatively they gradually improved, i.e. WBC 13,900/mL, CRP 5.7 mg/dL, platelet count 5 \times 10⁴/mL. Seven days after the operation he was transferred from the ICU to a general ward.

Histopathological findings (Fig. 6)

The weight of the right lung was 1,800 g, and each lobe increased in solidity.

The cut surface of the lung was reddish brown, grayish white and solid. There were some cavities with necrosis. Microscopic findings showed that in almost the whole lung infiltrations of lymphocytes, neutrophils and macrophages and edema existed in the alveolar cavities. A reddish brown portion involved remarkable congestion. Pathological diagnosis was pneumonia with pulmonary abscess.



Fig. 2. Chest X-ray film about one month before admission. There were large cavities on the right upper lobe due to pulmonary tuberculosis.



Fig. 3. Chest X-ray film one day before admission. Pneumonia shadows with niveau in the cavities were on the right upper lobe.



Fig. 4. Chest X-ray film on admission. Chest radiograph revealed multiple nodules of varying sizes with indistinct margins that were suspected to be septic pulmonary emboli, but the left side lung field was intact.



Fig. 5. Chest X-ray film of the next day of admission in ICU. Abnormal shadows extended rapidly. Intratracheal intubation was performed immediately and the patient was put on an artificial respirator and administered catecholamines.

Discussion

The systemic response to infection has been termed sepsis.⁸⁾ The incidence of sepsis has been increasing gradually because of the use of more potent and broad-spec-

trum antibiotics, immunosuppressive agents, and invasive technology in the treatment of inflammatory, infectious, and neoplastic diseases.¹⁾ Recently, among patients with severe sepsis and septic shock, infections of the lung have been predominant, accounting for 36% of all infections.⁹⁾

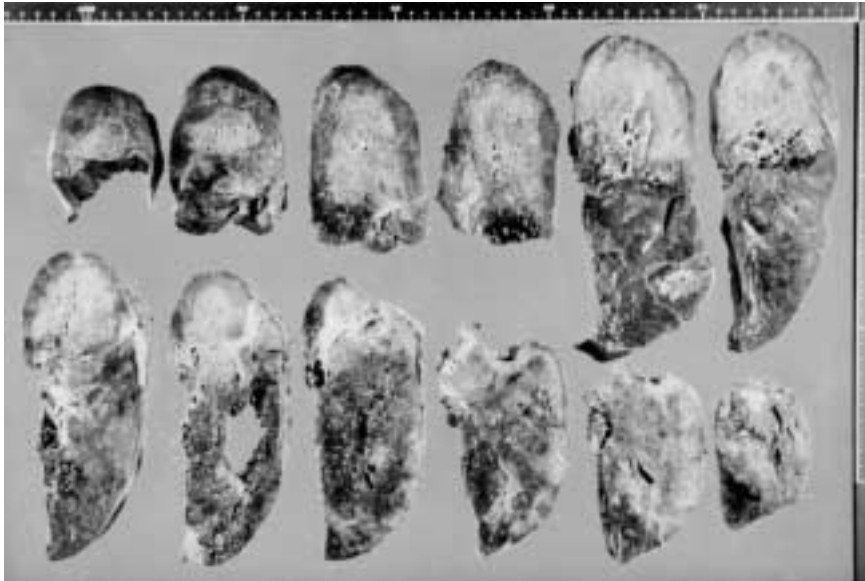


Fig. 6. Cut surfaces of the resected specimen (macroscopic findings). There are large cavities with necrosis. Pathological diagnosis is pneumonia with pulmonary abscess.

In this case, cavities, which were formed by tuberculosis, became focuses of repeated infections and after the operation, candida albicans proved to be the pathogen that provoked the septic shock. Respiratory physicians had used an antibiotic against it, but the antibiotic could not be efficacious against an abnormal multiplication of the pathogen. On the other hand, in cases of huge multiplication like this there is a possibility that the antibiotic might force the pathogen to release endotoxin, cytokine, anandamide and other mediators of septic shock. The best therapies for septic shock are elimination of the causative factors and purification of toxic blood. In this case another cause of the deteriorating condition was an abnormal gas exchange. It might occur as a result of a combination of mechanisms, including VA/Q inequality, intrapulmonary shunt, diffusion impairment, and mixed venous hypoxemia, and it is therefore probable that this patient might have developed hypoxia. For improvement of VA/Q inequality it was necessary to interrupt the pulmonary flow. From this point of view, our decision to perform a right pneumonectomy and postoperative PMX and CHDF seems to have been reasonable.⁷⁾ Krishnadasan et al. reported five lung gangrene cases in which it was possible to perform successful resection, even in the setting of diffuse parenchymal changes and ventilator dependency.¹⁰⁾ We concluded that in this case neither surgical treatment nor purification and medicinal treatment by itself could improve the septic condition, but multidisciplinary treatment by surgical operation, blood purification and medication could provide a synergistic effect against septic shock.

References

1. Bone RC, Fein AM, Balk RA, et al. Definitions for sepsis and organ failure and guideline for the use of innovative therapies in sepsis. The ACCP/SCCM Consensus Conference Committee. *Chest* 1992; **101**: 1644–55.
2. Zelefsky MN, Lutzker LG. The target sign: a new radiologic sign of septic pulmonary emboli. *AJR Am J Roentgenol* 1977; **129**: 453–5.
3. Huang RM, Naidich DP, Lubat E, Schinella R, Garay SM, McCauley DI. Septic pulmonary emboli: CT-radiographic correlation. *AJR Am J Roentgenol* 1989; **153**: 41–5.
4. Kuhlman JE, Fishman EK, Teigen C. Pulmonary septic emboli: diagnosis with CT. *Radiology* 1990; **174**: 211–3.
5. Müller NL, Fraser RS, Corman N, Paré PD. Radiologic diagnosis of diseases of the chest. In: *Embolism Lung Disease*. Philadelphia: W.B. Saunders Company, 2001; pp 399–400.
6. Dantzker DR. Pulmonary embolism. In: Crystal RG, West JB, Weibel ER, Barnes PJ eds.; *The Lung Scientific Foundations*, 2nd ed. Philadelphia: Lippincott-Raven Publishers, 1997; pp 1599–607.
7. Tani T. Review of endotoxin-adsorbing direct hemoperfusion therapy using a column containing polymyxin B immobilized fiber. *Curr Opin Crit Care* 2000; **6**: 416–20.
8. Balk RA, Bone RC. The septic syndrome: definition and clinical implications. *Crit Care Clin* 1989; **5**: 1–8.
9. Bochud PY, Glauser MP, Calandra T. Antibiotics in sepsis. *Intensive Care Med* 2001; **27**: S33–S48.
10. Krishnadasan B, Sherbin VL, Vallieres E, Karmy-Jones R. Surgical management of lung gangrene. *Can Respir J* 2000; **7**: 401–4.