J Anesth (2004) 18:48–50
DOI 10.1007/s00540-003-0207-3

Journal of
Anesthesia

Respiratory failure caused by massive pleural effusion in a patient with deep neck abscess

Keisuke Yamada[1], Kazumi Hashimoto[1], Tsunehisa Tsubokawa[1], Katsumi Tashiro[1], Shigeo Ohmura[1], Takumi Taniguchi[2], and Keizo Shibata[3]

[1] Department of Anesthesiology and Intensive Care Medicine, Graduate School of Medical Science, Kanazawa University, 13-1 Takara-machi, Kanazawa 920-8641, Japan
[2] Department of Emergency Medicine and Critical Care Medicine, Graduate School of Medical Science, Kanazawa University, Kanazawa, Japan
[3] Intensive Care Unit, Tonami General Hospital, Tonami, Japan

Key words Deep neck abscess · Diabetic mellitus · Dorsal lung collapse · Mediastinitis · Pleural effusion

Introduction

Peritonsillar abscess, a complication of acute tonsillitis, can occasionally extend through the cervical anatomic space and cause some complications such as deep neck abscess [1–3]. When deep neck abscess spreads into the mediastinum, the most lethal form of mediastinitis, known as descending necrotizing mediastinitis (DNM), can occur, causing respiratory failure, septic shock, or other complications with high mortality [4–6]. However, severe respiratory complications may occur even when the abscess does not extend into the mediastinum. This report describes a diabetic patient with respiratory failure that developed after cervical drainage for a deep neck abscess. Although the abscess did not extend into the mediastinum, respiratory failure was caused by massive pleural effusion.

Case report

A 61-year-old man, 175 cm in height and 55 kg in weight, was brought to the Kanazawa University Hospital by ambulance because of dyspnea, with redness and swelling of the neck. He had been diagnosed as having diabetes mellitus 3 years previously, but was not given any medication. He had suffered from sore throat and dysphagia for 10 days before being admitted to our hospital. A physician had diagnosed peritonsillar abscess, and he had been given antibiotics, for which he had taken for 2 days, but redness and swelling of the neck had developed gradually, followed by dyspnea.

On admission, physical examination showed he was confused. Redness and swelling were seen on the left side of the neck, and the area around the left tonsil was extremely swollen. Blood pressure was 136/68 mmHg and heart rate 125 bpm. The patient had a slight fever, temperature 37.6°C, his respiratory rate was 24 breaths per min, and stridor could be heard. S_{pO_2} was 99% under oxygen administration (5 l·min^{-1}) with a face mask.

Blood examination showed that C-reactive protein (CRP) had increased to 47 mg·dl^{-1}, and the white blood cell (WBC) count had increased to 12 400 · mm^{-3}. The examination also showed Na, 130 mEq·l^{-1}; K, 3.0 mEq·l^{-1}; Cl, 93 mEq·l^{-1}; blood glucose level, 422 mg·dl^{-1}; hemoglobin A1c, 7.6%; and renal dysfunction (blood urea nitrogen [BUN], 45 mg·dl^{-1}; creatinine [Cr], 1.5 mg·dl^{-1}).

Computed tomography (CT) scan of the neck revealed an abscess extending from the peritonsillar space to the parapharyngeal and retropharyngeal space, and swelling of the area surrounding the glottis (Fig. 1), but the abscess did not extend directly into the mediastinum, and pleural effusion was not detected. Emergency surgical drainage was scheduled.

In the operating room, tracheostomy was performed first, under local anesthesia because the pharynx was extremely edematous. Anesthesia was induced with thiopental (200 mg), and was maintained with sevoflurane (1.0%–1.5%) in 40% oxygen and 60% nitrous oxide under mechanical ventilation. Blood loss during the operation was 50 g, and 2200 ml of acetated Ringer's solution was administered during surgery. Bacterial examination revealed infection with a combination of aerobic (β-streptococcus) and anaerobic gram-negative rod bacteria.

After surgery, Pa_{O_2} was 447 mmHg under 100% oxygen, and X-ray of the chest revealed no abnormalities. The patient was returned to a general ward. He received

Address correspondence to: K. Yamada
Received: February 7, 2003 / Accepted: October 21, 2003

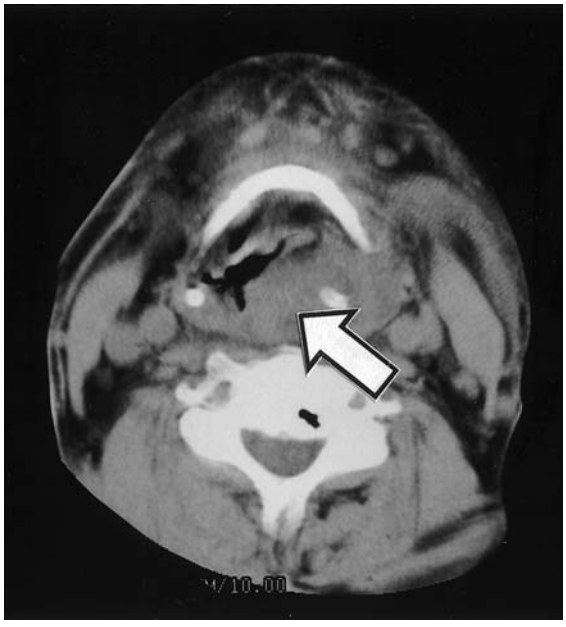

Fig. 1. Preoperative neck computed tomography (CT) scan, showing abscess (*arrow*) on the left side of the glottis and extreme swelling of the area surrounding the epiglottis

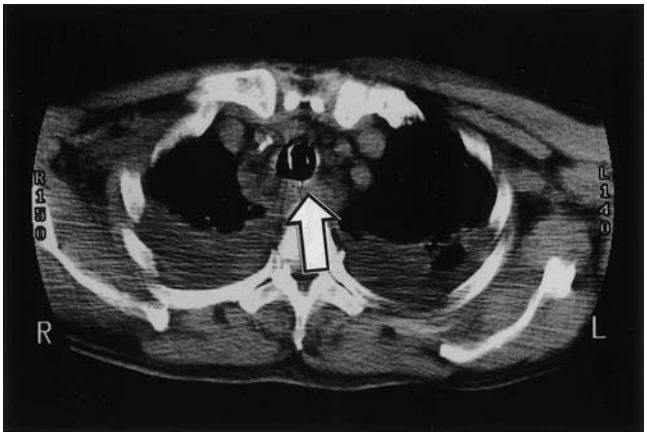

Fig. 2. Chest CT scan obtained 2 days after surgery, showing swelling of the posterior mediastinum (*arrow*) without the abscess extending into the mediastinum

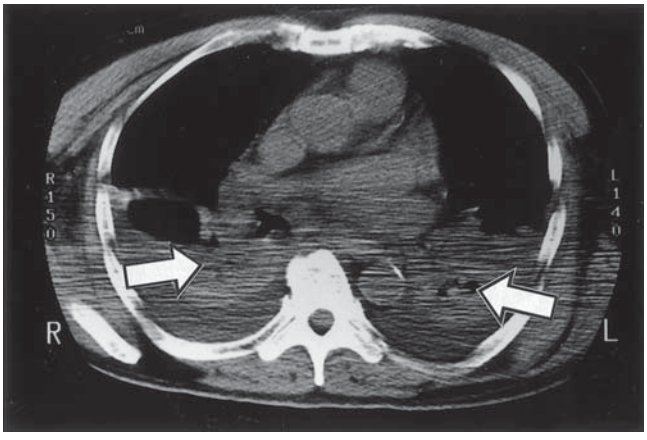

Fig. 3. Chest CT scan obtained 2 days after surgery, showing bilateral pleural effusion and nonsegmental consolidation (*arrow*) in the dorsal lung

cefozopran hydrochloride ($2\,g \cdot day^{-1}$) and clindamycin phosphate ($600\,mg \cdot day^{-1}$) intravenously. Diabetic control was performed with insulin. However, his body position was not changed regularly and his body movements were restricted (an intravenous sedative [midazolam] was used because he was restless and could not obey the staff's instructions). His Pa_{O_2} deteriorated gradually and eventually fell to 41 mmHg under 50% oxygen 2 days after the surgery.

CT scan of the chest revealed bilateral pleural effusion, dorsal lung collapse, and swelling of the posterior mediastinum. However, direct extension of the abscess into the mediastinum was not seen (Figs. 2, 3).

On the first day after surgery, 1900 ml of crystalloid fluid solution was administered intravenously, and the urine volume was 874 ml. Laboratory examination results 2 days after surgery were: WBC, $7300 \cdot mm^{-3}$; CRP, $42\,mg \cdot dl^{-1}$; Na, $142\,mEq \cdot l^{-1}$; K, $4.1\,mEq \cdot l^{-1}$; Cl, $105\,mEq \cdot l^{-1}$; total protein, $4.4\,g \cdot dl^{-1}$; Cr, $1.3\,mg \cdot dl^{-1}$; and BUN, $58\,mg \cdot dl^{-1}$.

Pulmonary edema due to water excess was suspected, and furosemide (60 mg) was administered intravenously. Reactive urine was excreted (1000 ml per 1 h), and then albumin and furosemide administration was continued. Although the urine volume on the second day after surgery was 2400 ml, his blood gas analysis data did not improve (pH, 7.49; Pa_{CO_2}, 36 mmHg; Pa_{O_2}, 51 mmHg [$F_{I_{O_2}}$, 0.5]; $H_{CO_3^-}$, $27\,mmol \cdot l^{-1}$). The patient was transferred to our intensive care unit (ICU) for respiratory treatment on the third postoperative day.

The pleural effusion, examined by thoracentesis in the ICU, was cloudy and yellow, with specific gravity 1.024; protein, $2.9\,g \cdot dl^{-1}$; and lactate dehydrogenase (LDH), $419\,mg \cdot dl^{-1}$, and was considered to be exudate. Neither culture of the effusion nor that of the blood showed any bacteria. Transthoracic echocardiogram revealed that his heart function was good, and central venous pressure was $4\,cmH_2O$. Laboratory examination results were: WBC, $11\,200 \cdot mm^{-3}$, CRP, $32\,mg \cdot dl^{-1}$; Na, $139\,mEq \cdot l^{-1}$; K, $4.3\,mEq \cdot l^{-1}$; Cl, $101\,mEq \cdot l^{-1}$; total protein, $4.8\,g \cdot dl^{-1}$; serum albumin, $2.5\,g \cdot dl^{-1}$; Cr, $1.2\,mg \cdot dl^{-1}$; and BUN, $44\,mg \cdot dl^{-1}$.

The patient received mechanical ventilation with pressure support of $10\,cmH_2O$ and positive end-expiratory pressure of $6\,cmH_2O$. Physiotherapies, such as changing his body position every 2 h, compression of

the thorax in accordance with the expiratory cycle, and intratracheal suction were also applied. Intravenous cefozopran hydrochloride ($3\,g \cdot day^{-1}$) and clindamycin phosphate ($1200\,mg \cdot day^{-1}$) were continued for 10 days until the WBC count had dropped to the normal range. Blood glucose levels were controlled with insulin below $200\,mg \cdot dl^{-1}$. Diuretics were administered intravenously and albumin was given to increase colloid osmotic pressure.

Four days after he was admitted to the ICU, a second neck drainage was performed for the residual abscess. Five days after he was admitted to the ICU, the patient's Pa_{O_2} had improved to 240 mmHg ($F_{I_{O_2}}$, 0.5) and pleural effusion had decreased on chest X-ray, so the ventilatory support was stopped. The patient's subsequent course was uneventful. He was discharged 7 weeks after surgery.

Discussion

Deep neck abscess secondary to oropharyngeal infection is a rare but severe complication. Especially in diabetic patients, deep neck abscess can become more severe because of suppressed immunity and close observation to detect life-threatening complications is crucial [7].

When cervical drainage for the deep neck abscess was performed in this patient, there were no abnormalities in the patient's oxygenation capacity and chest X-ray findings, so the patient was returned to a general ward, but then his Pa_{O_2} decreased critically. Respiratory failure was suspected to have been caused by collapse of the dorsal lung as the result of massive pleural effusion, although the deep neck abscess did not extend into the mediastinum.

We considered the following factors may have caused the pleural effusion. First, the occurrence of secondary mediastinitis or pleuritis caused the pleural effusion because the pleural effusion was exudate, and swelling of the posterior mediastinum was observed on the CT scan. Second, increasing vessel permeability and decreasing colloid osmotic pressure, due to the widespread neck infection may also have caused the pleural effusion and atelectasis. Along with the pleural effusion, the compulsory rest, resulting from the use of a sedative, and insufficient respiratory physiotherapy, must have contributed to the retention of the secretions and the dorsal lung collapse.

When active respiratory physiotherapy and ventilatory support were instituted, together with the administration of antibiotics, diuretics, and albumin, and the diabetic control, the patient rapidly recovered from respiratory failure. Changing the patient's body position, and the physical treatment for the excretion of sputum were especially important in this patient in order to reexpand the atelectatic lung segments.

To summarize, we reported a diabetic patient who developed respiratory failure after cervical drainage for deep neck abscess. Even when a deep neck abscess does not extend into the mediastinum, attention should be paid to newly developed pleural effusion, due to secondary mediastinitis or pleuritis, and hydrostatic changes accompanying the infection.

References

1. Nilesen TR, Clement F, Andreassen UK (1996) Mediastinitis: a rare complication of a peritonsillar abscess. J Laryngol Otol 110:175–176
2. Matsuda A, Tanaka H, Kanaya T, Kamata K, Hasegawa M (2002) Peritonsillar abscess: a study of 724 cases in Japan. Ear Nose Throat J 81:384–389
3. Sakaguchi M, Sato S, Ishiyama T, Katsuno S, Taguchi K (1997) Characterization and management of deep neck infections. Int J Oral Maxillofac Surg 26:131–134
4. Cordero L, Torre W, Freire D (1996) Descending necrotizing mediastinitis and respiratory distress syndrome treated by aggressive surgical treatment. J Cardiovasc Surg (Torino) 37:87–88
5. Sancho LM, Minamoto H, Fernandez A, Sennes LU, Jatene FB (1999) Descending necrotizing mediastinitis: a retrospective surgical experience. Eur J Cardiothorac Surg 16:200–205
6. Papalia E, Rena O, Oliaro A, Cavallo A, Giobbe R, Casadio C, Maggi G, Mancuso M (2001) Descending necrotizing mediastinitis: surgical management. Eur J Cardiothorac Surg 20:739–742
7. Chen MK, Wen YS, Chang CC, Lee HS, Huang MT, Hsiao HC (2000) Deep neck infections in diabetic patients. Am J Otolaryngol 21:169–173