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CLINICAL REPORT

Postoperative respiratory failure caused by acute exacerbation of idiopathic interstitial pneumonia

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Abstract We herein report a case of postoperative respiratory failure caused by acute exacerbation of undiagnosed interstitial pneumonia (IP). A 76-year-old woman underwent two subsequent operations, cholecystectomy and resection of lipoma in the neck, under general anesthesia at an interval of 12 days. Although the postoperative course of the first operation was uneventful, the patient abruptly developed respiratory failure on the 4th postoperative day (POD) of the second operation. Although steroid therapy was transiently effective to improve oxygenation, respiratory failure was gradually deteriorated. She died on the 25th POD. She had a past history of right upper lobectomy for pseudotumor resulting from cryptogenic organizing pneumonia in another hospital 4 years ago. The follow-up CT performed in this hospital demonstrated subtle foci of ground-glass opacities in the left lung; however, no diagnosis of IP had been made. Thus, we concluded that idiopathic IP had gradually advanced preoperatively, and acute exacerbation was triggered by perioperative stress. The present case warned us that acute exacerbation of IP could occur in a patient with mild symptoms. Therefore, preoperative proper diagnosis is thought to be important as acute exacerbation of IP is a highly morbid clinical event.

Keywords Respiratory failure · Idiopathic interstitial pneumonia · Acute exacerbation

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Introduction

Idiopathic interstitial pneumonias (IPs) are a group of diffuse parenchymal lung diseases of unknown etiology with varying degrees of inflammation and fibrosis [1]. The prevalence of IPs is estimated to be 20 per 100,000 in Japan. However, the true prevalence including undiagnosed cases with mild respiratory symptoms is thought to be 10 times higher than the estimate. Acute exacerbation of IPs is a highly morbid clinical event that could be triggered by perioperative stress [2, 3]. Acute postoperative exacerbation is not predictable and not completely avoidable even by the several strategies described in the literature [4]. Therefore, preoperative proper diagnosis for IPs is essential. We report here a patient with postoperative respiratory failure caused by acute exacerbation of IP that was not diagnosed preoperatively.

Case report

A 76-year-old woman diagnosed with cholecystolithiasis was admitted to our hospital to undergo open cholecystectomy. She had undergone right upper lobectomy in another hospital 4 years ago. However, the patient and her family did not understand the details of her lung disease but only told us the tumor was not lung cancer. She was not a smoker but had complained of a mild dry cough persisting for years. Although preoperative pulmonary function test showed mild restrictive ventilatory defect (%VC 57%, $FEV_{1.0}$ 90%), SpO_2 in room air was normal (98%) and inspiratory crackles was not observed on lung auscultation. Although a preoperative chest roentgenogram showed a subtle ground-glass appearance in the left lung (Fig. 1), the surgeons and anesthesiologists did not recognize a

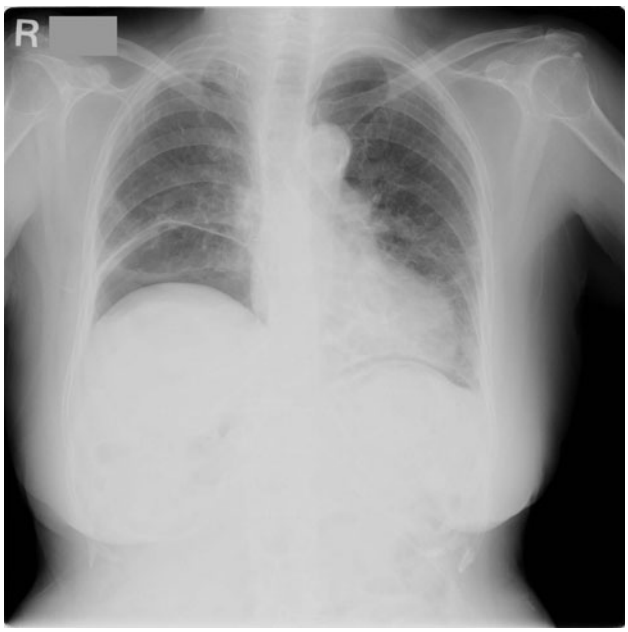

Fig. 1 Preoperative chest X-ray shows subtle ground-glass appearance in the left lung

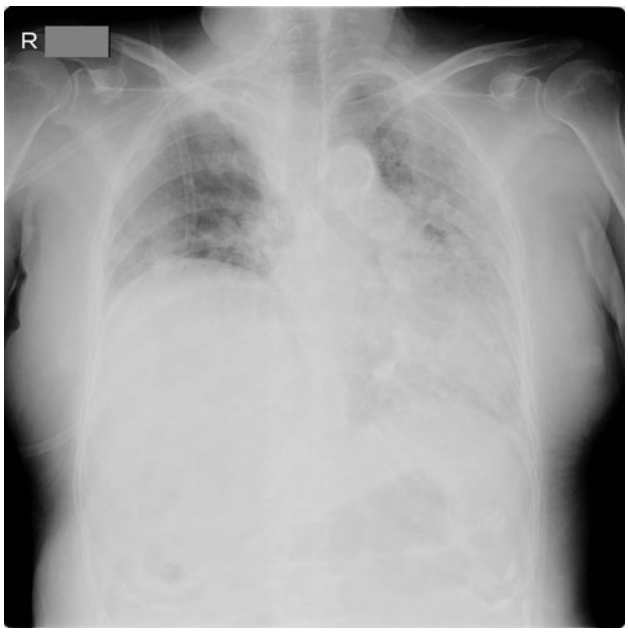

Fig. 2 Chest X-ray on 4th postoperative day (POD) after second operation shows diffuse infiltrates in the bilateral lung

relationship with IP, and thus the restrictive pattern of pulmonary function test was believed to result from right upper lobectomy. Preoperative laboratory data did not suggest inflammatory lung disease [lactate dehydrogenase (LDH), 165 IU/l; C-reactive protein (CRP), 0.25 mg/dl; white blood cell count (WBC), 6,090/μl].

The first operation, cholecystectomy, was performed under general anesthesia and thoracic epidural block. Anesthesia was maintained with continuous intravenous infusion of propofol and remifentanil and with epidural infusion of 0.38% ropivacaine. Intraoperative oxygenation was well maintained with 40% oxygen and intermittent positive pressure ventilation. The patient recovered from anesthesia smoothly 10 min after the operation. Although mild hypercapnia resulting from epidural morphine at 2 mg was observed after extubation, blood gas analysis showed adequate oxygenation with oxygen inhalation at 3 l/min (PaO_2, 176 mmHg; $PaCO_2$, 54 mmHg). Her postoperative course was uneventful.

The patient hoped for a second operation to remove a lipoma in the nucheal region. Because the tumor was large (>10 cm in diameter), we decided to use general anesthesia. This second operation was performed on the 12th postoperative day (POD). Anesthesia was maintained with continuous intravenous infusion of propofol and remifentanil. The operation concluded in 30 min in prone position. The patient recovered from general anesthesia after 15 min. Hypoxemia and respiratory difficulty were not observed after extubation.

On the 4th POD after the second operation, abrupt increase of body temperature (39.4°C) and respiratory difficulty were observed. Wheezing respiration progressively worsened and hypoxemia developed (SpO_2, 60%; face mask, 8 l/min). Chest roentgenogram showed diffuse infiltrate in the bilateral lung (Fig. 2). Pressure support ventilation was initiated by tracheal intubation. Lung compliance was poor; a tidal volume of 150–200 ml was narrowly obtained with support pressure of 20 cm H_2O and positive end-expiratory pressure 5 cm H_2O. Blood gas analysis data demonstrated acute respiratory acidosis and poor oxygenation (pH 7.26; PaO_2, 61.0 mmHg; $PaCO_2$, 64.0 mmHg; FIO_2, 1.0). Laboratory data showed leukocytosis (18,160/μl) and marked increase in serum CRP (22.8 mg/dl). Gram stain of sputum showed many neutrophils but not significant bacteria to cause pneumonitis. Further, serum tests showed no other organisms that could cause pneumonitis. Infusion of piperacillin 13.5 g/day and sivlestat sodium (neutrophil elastase inhibitor) 200 mg/day was started; however, her clinical symptoms did not improve. Serum biomarkers suggesting IP including LDH and sialylated carbohydrate antigen KL-6 had increased by 353 and 570 IU/ml, respectively. Furthermore, computed tomography (CT) of the lungs showed a diffuse reticulonodular interstitial region (Fig. 3). Thus, the patient was diagnosed with severe IP, and steroid therapy was initiated on the 6th POD. Initially, methylprednisolone 500 mg/day was administered for 3 days and then prednisolone 40 mg/day was given for maintenance therapy. CRP and body temperature were transiently improved; however, the patient could not be weaned from the respirator because of her poor lung compliance. Respiratory failure gradually deteriorated, and she died on the 25th POD.

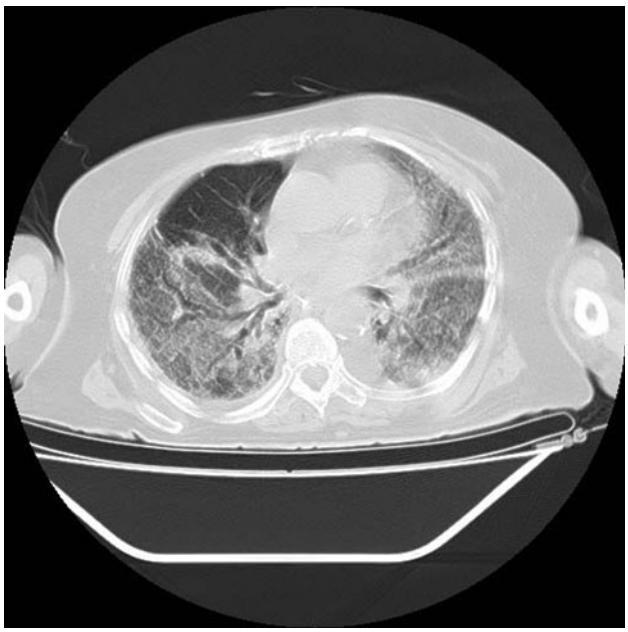

Fig. 3 Lung computed tomography (CT) shows diffuse reticulonodular interstitial lesion suggesting severe interstitial pneumonia

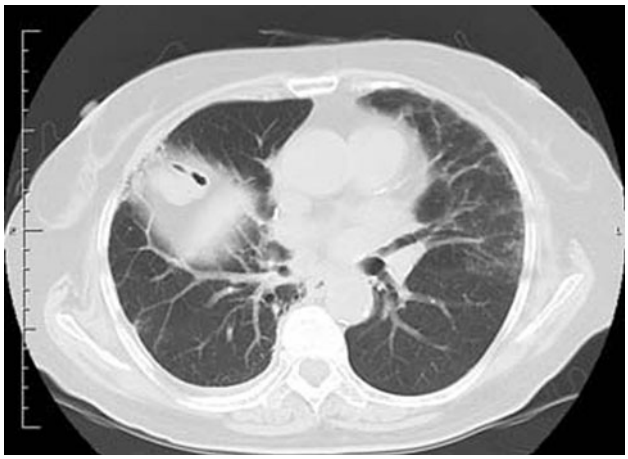

Fig. 4 Chest CT from previous hospital performed 1 year before our operations shows subtle foci of ground-glass opacities in the left lung

We asked the department of thoracic surgery in her previous hospital about the pathology of the lung tumor. The right lung lesion specimen when reevaluated by a pathologist indicated cryptogenic organizing pneumonia (COP). The follow-up lung CT from the previous hospital 1 year before our operations was also reevaluated in detail. We found subtle foci of ground-glass opacities in the left lung (Fig. 4). Because these lesions were not conspicuous and her clinical symptoms were not severe, no diagnosis of IP was determined in the first hospital. Thus, we concluded that IP had been gradually advancing preoperatively, and acute exacerbation was triggered by perioperative stress.

Discussion

In the present case, the patient had a past history of right upper lobectomy 4 years previously caused by COP, one of the seven clinicopathological entities of IP. However, solitary focal organizing pneumonia usually does not relapse after surgical excision [5]. Furthermore, the prognosis of COP is relatively good (5-year mortality is reported be <5%). Thus, the relationship between the present respiratory failure and the past history of COP remains unclear.

Follow-up CT performed 3 years after right upper lobectomy in the previous hospital had revealed slight reticular opacities in the contralateral lung, suggesting that IP has been slowly progressive. There are several reasons why her IP was overlooked preoperatively. First, we did not have enough information about her previous lung disease treatment. Neither the patient nor her family understood the disease pathology, and no treatment for IP had been initiated. Second, her clinical symptoms were not severe; she only manifested a mild dry cough, which did not interfere with her daily life. Thus, both surgeons and the anesthesiologist overlooked the subtle reticular shadows in the preoperative chest roentgenogram. Third, the mild restrictive pattern of spirometry was misunderstood to be a result of the previous right upper lobectomy. Fourth, preoperative laboratory examination showed no inflammatory data, suggesting that IP was in an inactive phase. Although most cases reported previously showed evident symptoms and clinical IP data preoperatively [2–4], the present case has alerted us that acute exacerbation of IP could occur in cases that are almost asymptomatic.

Acute exacerbation of IP is a serious postoperative complication and highly morbid. Inflammatory response to surgical intervention, exposure to high oxygen concentrations, and overdistension of the lung by positive pressure ventilation are possible factors triggering the progression of IP [4]. Thus, it seems important to limit operations with general anesthesia to the treatment of life-threatening disease in such cases. If IP had been diagnosed preoperatively in the present case, the second operation for lipoma removal would have been refused by surgeons and anesthesiologist; alternatively, it could be performed under regional nerve block. We think that the accumulative lung stress in a short time period produced by two subsequent operations under general anesthesia was the crucial determinant that triggered fatal respiratory failure caused by IP.

Corticosteroids have been the mainstay therapy for IP. The treatment dose of corticosteroids is not standardized, but intravenous methylprednisolone 500 mg–1 g for the first 3–5 days is used in severe cases. Further, 0.5–1.5 mg/kg per day of prednisolone is administered for 4–6 weeks [6]. In the present case, transient improvement of oxygenation and inflammatory response was found after methylprednisolone

treatment but the patient failed to wean from mechanical ventilation. Sugino et al. [2] reported that corticosteroid therapy was almost ineffective for postoperative exacerbation of IP. Further, postoperative corticosteroid therapy could cause surgical wound infection, resulting in sepsis. Thus, although immunomodulatory agents such as azathioprine and cyclophosphamide were the alternative choice to treat IP [7], we hesitated to use these agents for fear of surgical wound infection and sepsis.

In conclusion, we report a case of respiratory failure caused by IP. The present case showed that acute exacerbation and severe respiratory failure could occur even in a mild case of IP in inactive phase. Postoperative exacerbation is highly morbid; thus, preoperative diagnosis is important even in asymptomatic cases. This case provides us with sufficient warnings not to overlook undiagnosed cases of IP. Geriatric patients who complain of long-term dry cough accompanied with mild restrictive respiratory impairment are not uncommon. Many anesthesiologists commonly perform general anesthesia for these patients. If we find such a patient preoperatively, it would be preferable to carry out further radiographic examinations such as high-resolution CT of the lungs.

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Conflict of interest None.

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