


A case of negative-pressure pulmonary oedema after first-time electroconvulsive therapy

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Abstract

Electroconvulsive therapy (ECT) has been used for many years as an important treatment modality in patients with schizophrenia. Recently, many new oral medications have become available to treat schizophrenia. However, ECT remains a valuable therapy for patients who are resistant to oral medications. A 16-year-old girl with schizophrenia was admitted to our hospital with hypoxaemia due to negative-pressure pulmonary oedema (NPPE) after her first ECT. NPPE is an exceedingly rare complication after ECT. However, it can result in serious morbidity if not immediately recognized and treated. This case illustrates the importance of recognizing this rare complication.

KEYWORDS

electroconvulsive therapy, laryngospasm, negative-pressure pulmonary oedema, schizophrenia

INTRODUCTION

Electroconvulsive therapy (ECT) was first reported in 1938 as an important and key therapy for patients with schizophrenia and severe depression.¹ Recently, modified ECT has been more commonly performed, and the mortality rate and occurrence of severe complications have decreased.² However, complications still rarely occur; for example, respiratory complications with modified ECT have been reported in some instances. Herein, we describe a rare case of a respiratory complication, negative-pressure pulmonary oedema (NPPE), which occurred in a 16-year-old girl after her first-time modified ECT treatment. We also compare our case to four similar cases that have been previously reported in the literature.

CASE REPORT

A 16-year-old girl was admitted to our hospital due to hypoxaemia after receiving ECT. She was diagnosed with schizophrenia at 15 years of age and was treated with oral medications. However, her symptoms, especially the visual and auditory hallucinations, worsened despite treatment.

Therefore, she was admitted to a psychiatric hospital to receive modified ECT. The patient's medical history was unremarkable except for schizophrenia. Her medications included aripiprazole 30 mg po qam, levomepromazine 50 mg po tid and paliperidone 6 mg po qam. The vital signs before ECT were normal. The patient's nothing by mouth status was confirmed, and anaesthesia was induced with an intravenous injection of thiopental 100 mg and suxamethonium 40 mg. Right unilateral stimulation was performed, resulting in a seizure lasting 96 s. During ECT, the patient's blood pressure increased to 178/88 mmHg, and her pulse rate was 98 beats per minute. The ECT was completed successfully without any complication, but 5 min after the treatment, the patient's oxygen saturation decreased to 85% in ambient air, and she developed difficulty speaking, shortness of breath and a small amount of pink-foamy sputum. Chest auscultation revealed inspiratory stridor. The arterial blood gas values in ambient air were as follows: the pH was 7.46, partial pressure of arterial oxygen was 62 Torr, partial pressure of arterial carbon dioxide was 33.7 Torr and bicarbonate level was 23.4 mg/dl. Based on these values, the patient was diagnosed with hypoxaemia and admitted to our hospital for treatment.

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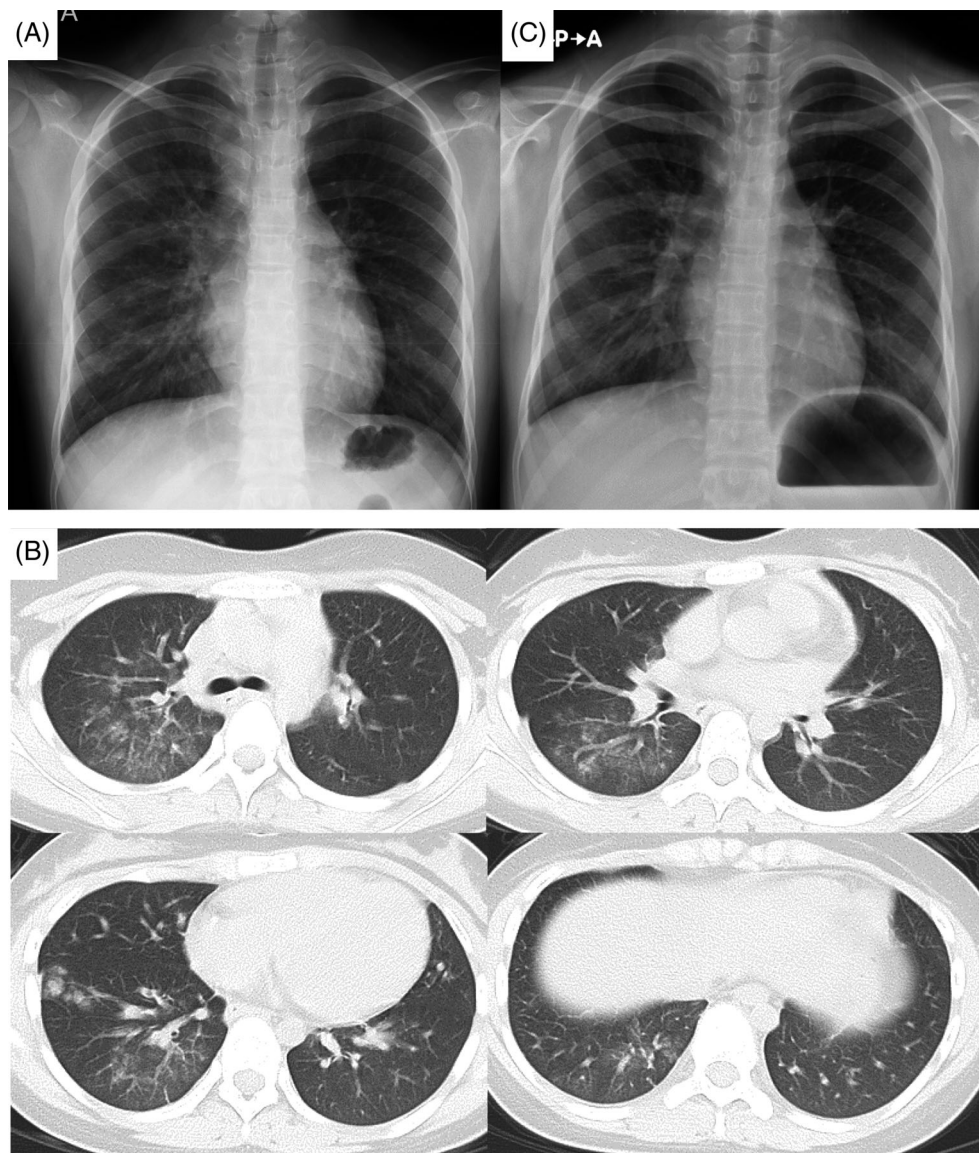


FIGURE 1 Chest radiograph on admission (A) and on the fourth day of hospitalization (C). High-resolution chest computed tomography on admission (B)

Upon hospital admission, the patient still expressed difficulty speaking and had a productive cough. Her oxygen saturation was 90% in ambient air, and her body temperature was 38.8°C. Other vital signs were normal. Physical examination findings were normal despite coarse crackles on chest auscultation. The inspiratory stridor was no longer present. The chest radiograph showed a normal cardiac silhouette and bilateral ground-glass opacities (GGO) distributed mostly on the right lung (Figure 1A). High-resolution computed tomography of the chest showed GGO distributed along the bronchial vascular bundle on both lung fields (Figure 1B). These radiological findings were consistent with non-cardiogenic pulmonary oedema. The electrocardiogram showed sinus rhythm without any ST-T changes. The laboratory test values were as follows: the white blood cell count was 17,000/ μ l, neutrophil count was 15,010/ μ l, serum

albumin concentration was 4.0 g/dl (normal 3.8–5.0 g/dl), serum lactate dehydrogenase concentration was 222 IU/L (normal 119–229 IU/L), serum C-reactive protein concentration was 1.41 mg/dl (normal <0.3 mg/dl), serum procalcitonin concentration was 12.0 ng/ml (normal <0.1 ng/ml), serum brain natriuretic hormone concentration was 13.6 pg/ml (normal <18.4 pg/ml), Krebs von den Lungen 6 concentration was 161 U/ml (normal <500 U/ml) and serum surfactant protein-D concentration was 93.2 ng/ml (normal <110 ng/ml). A Gram stain of the patient's sputum revealed normal bacterial flora.

In this case, the radiological findings, laboratory tests results and clinical course were consistent with non-cardiogenic pulmonary oedema, and the inspiratory stridor as the symptom at onset was more consistent with NPPE than neurogenic pulmonary oedema (NPE). Therefore, the

TABLE 1 Comparison of cases with NPPE after ECT

Reference	Age (years)	Sex	ECT session (number)	Bloody or pink-foamy sputum	Treatment for NPPE ^a
Cochran ⁵	46	F	4	+	Furosemide
Hatta ⁶	69	M	8	+	Hydrocortisone
Myers ⁷	21	M	28	+	Positive-pressure ventilation
Mansoor ⁸	30	M	2	+	Furosemide
Our case	16	F	1	+	Only observation

Abbreviations: ECT, electroconvulsive therapy; NPPE, negative-pressure pulmonary oedema.

^aAll five cases revealed hypoxaemia and received temporary oxygen administration.

patient was diagnosed with NPPE. Her symptoms, including cough and difficulty speaking, improved without any treatment except for temporary oxygen administration. The chest radiograph obtained on the fourth day of hospitalization showed the disappearance of GGO (Figure 1C). She was discharged to the psychiatric hospital on the fifth day of hospitalization.

DISCUSSION

Pulmonary oedema can occur when there is a disruption of the Starling forces that influence the movement of fluid from the pulmonary capillaries to the lung interstitium. It is classified as cardiogenic pulmonary oedema, which results from an increase in hydrostatic pressure in the pulmonary capillaries, and noncardiogenic pulmonary oedema, which is associated with an increase of fluid in the interstitium and alveoli secondary to increased vascular permeability.³ Cardiogenic pulmonary oedema is associated with left-sided heart failure, and noncardiogenic pulmonary oedema is usually associated with other clinical disorders, including pneumonia, sepsis, major trauma, neurological disorders and upper airway obstruction. Previous reports have shown that both NPE and NPPE can be complications associated with ECT.^{4–8} NPE is known to occur in epileptic patients after a seizure associated with markedly elevated vital signs. The mechanism of NPE is thought to be pulmonary vasoconstriction and increased permeability of pulmonary capillaries caused by centrally mediated adrenergic excitation.⁴ On the other hand, NPPE is caused by upper airway obstruction and large inspiratory effort. The various causes of upper airway obstruction have been reported, including laryngospasm, epiglottitis, bilateral vocal cord palsy, acromegaly, goitre and obstruction of the endotracheal tube. However, laryngospasm resulting from stimulation by airway secretions is the most probable cause of NPPE as a complication of anaesthesia, with an incidence rate of approximately 0.1%.⁹ The physical manifestations and clinical course of these two types of pulmonary oedema are similar and difficult to distinguish from each other. However, our patient presented with the classical manifestations of NPPE as a complication of anaesthesia, including inspiratory stridor on chest auscultation, pink-foamy sputum and difficulty speaking. Therefore, the patient was diagnosed with NPPE after ECT.

To our knowledge, there are four previous reports of NPPE as a complication of ECT.^{5–8} The ECT session and treatment for NPPE varied from case to case (Table 1). The most severe case required positive-pressure ventilation. However, in our case, the patient did not need any treatment except for temporary oxygen administration. The ECT session when NPPE occurred ranged from the second to 28th in the four reports. However, in our case, NPPE occurred after the patient's first ECT. The cause of NPPE is thought to be resulting from laryngospasm, which occurs incidentally. This is consistent with the various ECT sessions when NPPE occurred. The age of these reported cases ranged from 21 to 69 years, and our patient was 16 years of age. This suggests that the inspiratory effort in patients with NPPE complicated with ECT is heavy due to their relatively young age.

Interestingly, the serum procalcitonin level was elevated in our case. In general, procalcitonin is recognized as a useful and highly sensitive marker of bacterial infections.¹⁰ However, high procalcitonin levels have been reported in patients with seizures.¹¹ The seizure induced by ECT lasted briefly, but may have been the cause of the increase in serum procalcitonin level in our patient.

Although rare, NPPE can complicate ECT treatment regardless of the ECT session. It can also result in serious morbidity if not immediately recognized and treated. This case illustrates the importance of recognizing this rare complication of ECT.

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CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTION

Yasuhito Sekimoto, Yoshifumi Suzuki, Koichiro Kanamori, Isao Kobayashi and Hiroki Ienaga were the attending doctors who treated the patient on admission. Yasuhito Sekimoto, Hiroki Ienaga and Kazuhisa Takahashi drafted the manuscript. Yasuhito Sekimoto submitted the final manuscript. All authors read and approved the final manuscript.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

Appropriate written informed consent from the patient and parent was obtained for publication of this case report and accompanying images.

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