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Pneumomediastinum after Convulsion and Vomiting

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Abstract A 15-year-old woman had a vomiting following 10 minutes of clonic convulsion after intentional overdose of cold remedy. When emergency medical technicians checked her, convulsion had subsided, however, she had a convulsion again during transportation. On arrival, she was coma state but had clear airway. Her head computed tomography (CT) was negative, however, chest roentgen and CT showed spontaneous pneumomediastinum (SPM). Initially, physician on duty did not know that she had had overdose, she just underwent infusion of evetiracetam and tazobactam / piperacillin. Her consciousness became clear on day 2. Post admission course was no eventful and she discharged without sequlae. We present extremely rare case of SPM immediately after a convulsion. As mechanism of such complication is poorly understood, further analysis by accumulation of such cases is necessary.

Keywords: *convulsion, pneumomediastinum, mechanism*

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1. Introduction

Spontaneous pneumomediastinum (SPM) is classified as free air in the mediastinum without any traumatic or iatrogenic cause. [1] The air that enters the mediastinum can originate from the bronchial tree, the lungs, or the esophagus. SPM often occurs after "triggering events," such as Valsalva maneuvers, exercise, drugs, asthma, or vomiting. Many cases of SPM are benign, but cases of SPM that are associated with esophageal perforation are often accompanied by severe complications such as mediastinitis and sepsis. We present here a case of SPM after convulsion and vomiting induced by overdose of over-the-counter cold remedy.

2. Case

A 15-year-old woman had a vomiting following 10 minutes of clonic convulsion after intentional ingesting approximate 130 tablets of over-the-counter cold remedy to cure cold soon, which contained guayacol potassium sulfonate, 2600 mg; dihydrocodeine phosphate, 346mg; methylephedrine hydrochloride, 866 mg; acetaminophen, 12990 mg; chlorpheniramine maleate, 108 mg and anhydrous caffeine, 1082 mg. Her mother called an ambulance. When emergency medical technicians checked her, convulsion had subsided, however, she had a convulsion again during transportation to our hospital. She had past history of urachal remnant and allergy for melon. Her small sister had febrile convulsion. On arrival, she had Glasgow Coma Scale of 3, a blood pressure of 120/60 mmHg, a heart rate of 120 beats per minute, a respiratory rate of 30 breaths per minute, and an SPO_2 of 99% under room air; and a body temperature of 39.9°C. Air way was clear and a physical examination was negative. A venous gas analysis revealed the following findings: pH, 7.330; $PaCO_2$, 26.9 mmHg; HCO_3^-, 13.8 mmol/L; base excess, -10.4 mmol/L; and lactate, 7.0 mmol/L. An electrocardiogram showed sinus tachycardia. Her head computed tomography (CT) was negative, however, chest roentgen and CT showed SPM (Figure 1). The main results of a blood analysis were as follows: white blood cell count, 13,600/μL; c-reactive protein, 0.02 mg/dL; acetaminophen, 58.2μg/ml, 8 hours after ingestion; aspartate aminotransferase (AST), 23 IU/L; and alanine aminotransferase (ALT), 16 IU/L. Initially, as physician on duty did not know that she had had overdose, she just underwent infusion of 1000 mg per day of evetiracetam for convulsion and 13.5 g per day of tazobactam / piperacillin for prevention of mediastinitis. Her consciousness became clear on day 2. After admission, she was free of convulsion so that infusion of evetiracetam ceased on day 3. She temporally had liver dysfunction (maximum AST, 38 IU/L on day 7 and ALT 99 on day 8) and the dysfunction improved spontaneously. As surgical physicians strongly insisted on possibility of esophageal perforation so that she was managed by inanition and intravenous hyperalimentation until on day 16. She was no eventful and discharge on 27 days without sequlae.

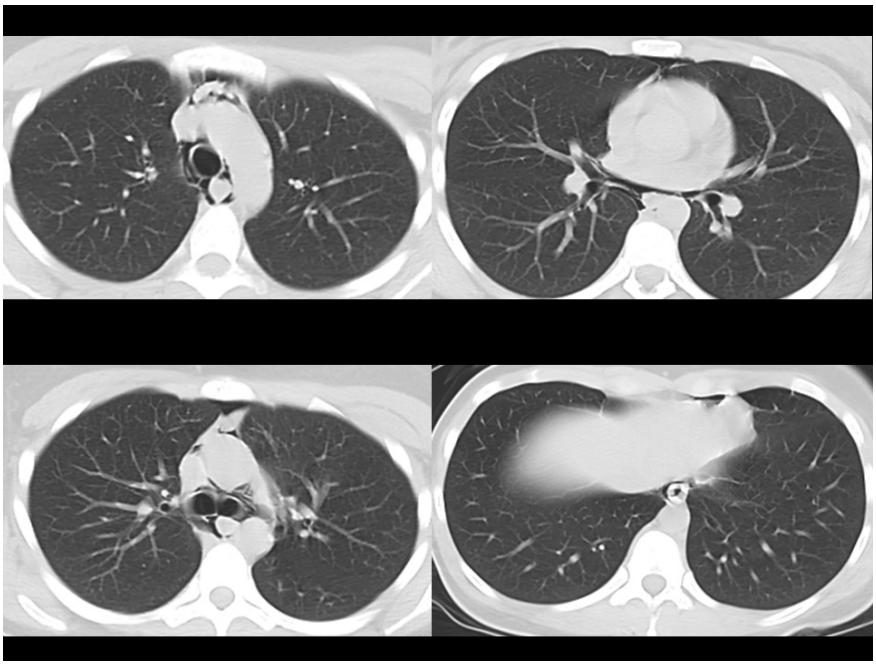

Figure 1. Chest computed tomography (CT) on arrival (The CT showed spontaneous pneumomediastinum, principally located peri-tracheal and main bronchus area)

3. Discussion

This is a rare case of SPM after convulsion and vomiting. As vomiting was only once and SPM located mainly around trachea so that possibility of esophageal rupture (Boerhaave syndrome) was thought to be minimized. A PubMed search to identify any related English articles using the key words “pneumomediastinum” and “convulsion” or “epilepsy” or “eclampsia”. We found three English medical record which occurred SPM after convulsion. [2,3,4] One of the three was that a 37 years old patient at 40 weeks gestation presented with a seizure followed by fetal bradycardia. [2] She had history of pleural endometriosis with multiple pneumothoraces and hydrothoraces. Caesarean section was performed and she was intubated and ventilated. A following chest CT showed SPM and extensive bilateral pnenumothoraces. While, the present case did not have history of pleural endometriosis and tracheal intubation, both of which might induce SPM respectively. Second case of the three was that a CT of an 18-year-old man after a tonic-clonic seizure initially showed bilateral lung lesions suggesting either chemical pneumonitis or pulmonary edema. [3] A repeat portable chest radiograph 9 hours after admission showed bilateral pneumothoraces and SPM even he did not undergo tracheal intubation or positive pressure by bag mask ventilation. Mechanism of the delay between the seizure and the subsequent development of the SPM and pneumothoraces was unknown. The last case of the three reported that occurrence of SPM during labor and delivery had been described well, but simultaneously occurring convulsive eclampsia was extremely rare. Labor associates with Valsalva maneuver which leads to distal alveolar rupture by excessive intrathoracic pressure. Again, the present case did not have labor and delivery. Accordingly, the direct association between pneumothorax and convulsion is poorly described. Only 1 case report in the French literature that describes bilateral pneumothoraces and pneumomediastinum immediately following a convulsion, similar to us. [5] The authors proposed that the likely mechanism related to increased intra-alveolar pressure generated by expiratory effort against a closed glottis during seizure activity. [5] This pressure was hypothesized to cause rupture of alveoli at the lung periphery with a subsequent escape of air into the mediastinum and pleural space. In addition to this hypothesis, we also hypothesize that spontaneous rupture at posterior membraneous wall of tracheal rupture, which is the weakest portion of trachea, as a result of positive pressure of airway during convulsion might be causative for occurrence of SPM in the present case. [6] Because the present case complicated only SPN but not pneumothorax.

4. Conclusion

We present extremely rare case of SPM immediately after a convulsion. As mechanism of such complication is poorly understood, further analysis by accumulation of such cases is necessary.

Conflict of Interest

The authors declare no conflicts of interest in association with the present study.

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List of Abbreviations

ALT, alanine aminotransferase; AST, aspartate aminotransferase; CT, computed tomography; SPM, Spontaneous pneumomediastinum

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