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SEVERE HYPOXAEMIA AND HAEMODYNAMIC INSTABILITY FOLLOWING SALT TABLET BRONCHO-ASPIRATION: IN-HOSPITAL MIMICKING OF SALTWATER DROWNING

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ABSTRACT

A 70-year-old woman, hospitalised for asymptomatic hyponatraemia secondary to syndrome of inappropriate antidiuretic hormone secretion (SIADH), required emergency intubation due to severe acute respiratory failure and haemodynamic instability following broncho-aspiration of a salt tablet. We hypothesise that a pathophysiological mechanism resembling saltwater drowning may have contributed to the clinical presentation.

KEYWORDS

Salt tablet, broncho-aspiration, drowning

LEARNING POINTS

- Salt tablet broncho-aspiration could be associated with severe hypoxaemia and haemodynamic instability.
- The pathophysiology of salt tablet broncho-aspiration may mimic drowning in saltwater.

INTRODUCTION

Hyponatraemia is the most common electrolyte abnormality encountered in clinical practice, and syndrome of inappropriate antidiuretic hormone secretion (SIADH), the nonosmotic release of arginine vasopressin, is the most frequent cause[1]. Certain drugs, including antidepressants, can enhance antidiuretic hormone (ADH) release or effect. Oral sodium chloride tablets are a recognised treatment for hyponatraemia in non-oedematous patients with mild or absent symptoms[2].

We describe a case of a salt tablet broncho-aspiration, resulting in severe acute respiratory failure and haemodynamic instability, requiring intubation and vasopressors.

CASE DESCRIPTION

A 70-year-old woman with a history of active smoking and schizoaffective disorder, currently on trazodone and risperidone therapy, presented to the emergency department with a three-day history of progressively

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worsening dyspnoea. She had exacerbation of a chronic non-productive cough, and mild hypoxaemia evidenced by a saturation of 88–89% on room air and a PaO_2/FiO_2 ratio of 210 mmHg. Two weeks prior to presentation, the patient underwent percutaneous needle biopsy of a 10 cm lump in the left breast, which identified an oestrogen receptor-positive infiltrating carcinoma of no special type. She did not report fever, and inflammatory markers were only mildly elevated (C-reactive-protein at 10 mg/l and leucocytes at 12.8 $\times 10^9$/l). She had no oedema, the NT-ProBNP level was normal (474 ng/l), and auscultation revealed several added sounds, including rhonchi and wheezes. A chest X-ray *(Fig. 1A)* showed a predominantly interstitial involvement, consistent with vascular congestion in the small circulation. In this context, the emergency department colleagues administered bronchodilators, for a possible exacerbation of a previously undiagnosed chronic obstructive pulmonary disease, and diuretics. This treatment resulted in an improvement of symptoms. Laboratory tests subsequently revealed hypotonic hyponatraemia (Na^+ 116 mmol/l, plasma osmolality 242 mmol/kg, urine sodium 10^9 mmol/l, urine osmolality 278 mmol/kg). Given the absence of neurological deficits and the patient's history, these findings were deemed consistent with SIADH induced by antidepressant medications. Although the initial differential diagnosis included a potential paraneoplastic origin, this was considered unlikely, as such syndromes are rarely associated with this type of tumour.

The patient was admitted to our Intensive Care Unit to monitor and correct sodium levels appropriately and for potential non-invasive ventilation, while awaiting further investigations of the clinical picture. Within 24 hours of admission, the patient exhibited normal breathing patterns and maintained peripheral oxygen saturation with 2–3 litres/min of supplemental oxygen with a nasal cannula. However, following the accidental inhalation of a 1-gram sodium chloride tablet, she experienced immediate respiratory distress and rapid significant desaturation. A chest X-ray showed complete opacity of the left hemithorax, parenchymal atelectasis, and the ipsilateral obliteration of the main bronchus *(Fig. 1B)*. Her condition did not improve with non-invasive ventilation, necessitating urgent intubation.

During post-intubation bronchoscopy, performed approximately two hours after the accidental inhalation, no tablet was found in the left bronchial tree. Instead, we observed a clear, greyish liquid, which we hypothesise to be the dissolved tablet. Concurrently, the patient was haemodynamically unstable, requiring up to 0.8 mcg/kg/min of norepinephrine. The patient exhibited mostly the aspects of a distributive shock, as evidenced by warm extremities and preserved urine output, along with some features of relative hypovolaemia. Transpulmonary thermodilution parameters, provided by a PiCCO catheter inserted after intubation, indicated a mildly reduced cardiac index (CI 2.88 l/min/m^2). These findings were observed in the context of a preload (global end-diastolic volume index 669 ml/m^2) and systemic vascular resistance (systemic vascular resistance index 1807 dyn s$\times$cm$^{-5}\times$m^2) at the lower limits of normality, despite high vasopressor use. Additionally, a cardiac ultrasonography showed a preserved biventricular function. Following endotracheal intubation and bronchoscopy, the patient required high-inspired oxygen fractions (FiO_2) of 80–100% for several hours; haemodynamic instability resolved within 36 hours. Two days later, a contrast-enhanced chest CT scan ruled out pulmonary embolism, showed some residual parenchymal consolidations in the left lung and revealed lymphangitis carcinomatosis as the cause of the interstitial involvement. Viral and bacterial cultures were negative.

Once the needs for catecholamines and ventilatory assistance decreased, the patient was successfully extubated. At the time of discharge from the ICU, the patient still required low-flow oxygen at 1–2 litres per minute. Additionally, secretion management was complicated by previously unrecognised dysphagia.

DISCUSSION

We present a case of salt tablet broncho-aspiration with a

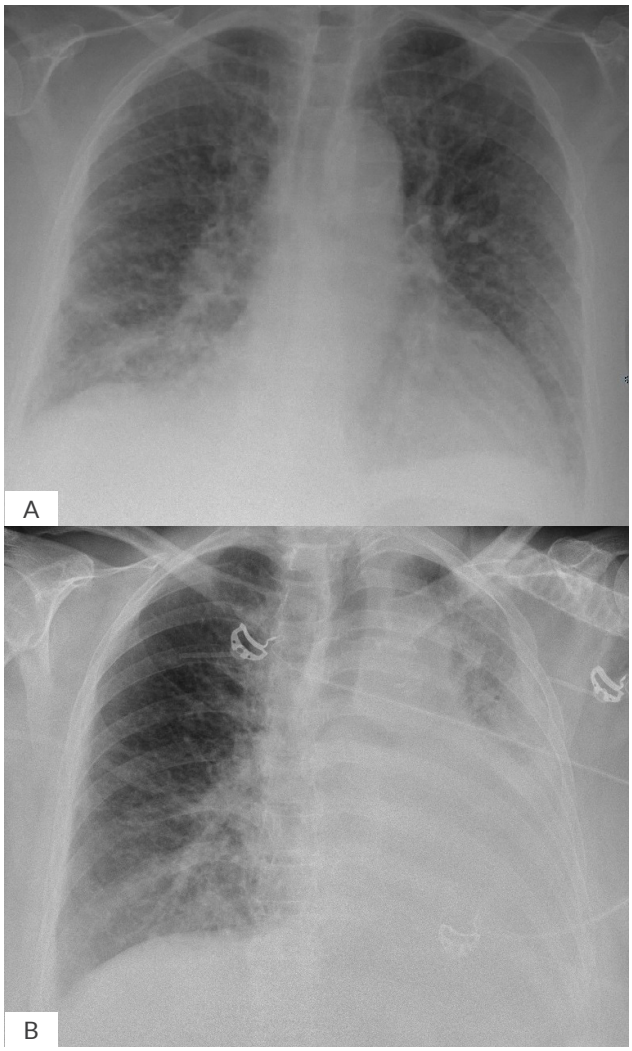


Figure 1. A) Chest X-ray obtained prior to bronchoaspiration of the salt tablet; B) Chest X-ray showing the radiological findings immediately following the procedure.

well-documented timeline, resulting in the sudden onset of severe hypoxaemia and haemodynamic instability, in a previously mildly symptomatic patient. We believe that simple inhalation of a foreign body does not fully account for the high catecholamine requirements and severe hypoxaemia, which persisted well beyond the dissolution of the tablet. Another pathophysiological mechanism may have contributed.

We hypothesise that the hypertonicity of the dissolved salt tablet (NaCl equivalent of about 30 ml of seawater) triggered a rapid influx of fluid from the plasma into the alveoli of the left lung, leading to damage to the surfactant function[3], disruption of the alveolar-capillary membrane and generation of foam. These combined factors significantly impaired gas exchange, mimicking the pathophysiology observed in saltwater drowning[4] and producing a pronounced shunt effect through the affected lung.

The patient developed warm shock without lactate elevation and maintained urine output, which is inconsistent with hypovolaemia or elevated intrathoracic pressure as the sole cause. An echocardiogram performed after intubation showed normal biventricular function. Although inflammatory markers increased 24 hours later, cultures remained negative for bacterial growth, making an infectious cause of the shock unlikely. Instead, a systemic inflammatory response due to the release of mediators in the lung, as described following drowning events, might be the most plausible explanation[5].

CONCLUSION

The case described here highlights a previously unreported complication of salt tablet aspiration. The hypertonic nature of the dissolved substance likely triggered pathophysiological changes analogous to those observed in saltwater drowning, including a systemic inflammatory response. Clinicians should be aware of these mechanisms when assessing sudden respiratory and haemodynamic deterioration following aspiration of hypertonic substances.

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