Review article

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Hyponatraemia associated with the use of quetiapine in ICU: A case report

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Abstract

A 28-year-old adult male with comorbidities developed quetiapine-induced hyponatremia, leading to critical illness and intensive care unit (ICU) admission. Serum sodium levels reached 117 mmol/l, indicating a syndrome of inappropriate antidiuretic hormone (SIADH). Reducing medication dose, restricting water intake, and administering salt supplementation improved sodium levels. The case study suggests that quetiapine-induced hyponatremia should be rapidly diagnosed and treated in younger patients with critical illness and comorbidities.

Key words: ICU, hyponatraemia, quetiapine, SIADH.

Introduction

Hyponatremia, a common electrolyte abnormality, is prevalent in the elderly and can lead to severe neuropsychiatric symptoms and increased morbidity and mortality if not managed properly. Factors contributing to hyponatremia include low salt consumption, chronic diseases, and the syndrome of inappropriate antidiuretic hormone secretion (SIADH). (1) Quetiapine, an atypical antipsychotic, has been linked to hyponatremia, but the frequency of younger patients developing hyponatremia from quetiapine is unknown. The following case study is going to discuss a young patient who has developed hyponatremia while taking quetiapine in the intensive care unit (ICU).

Case presentation

On 29 May 2023, a 28-year-old male patient was admitted to the ICU with a history of performing decompression craniotomy with subdural hematoma evacuation and insertion of an intracranial pressure (ICP) monitor. However, the patient failed extubation and was tracheostomized on 21 June 2023. He was released to ward care on tracheostomy and nasogastric tube (NGT) feeding, and the patient performed intracranial haemorrhage (ICH) evacuation as well as cranioplasty again on 8 August 2023, followed by reinsertion of a bone flap on 11 August 2023. On 21 December 2023, while receiving treatment and replacing the tracheostomy tube, the patient experienced bouts of desaturation. The ICU staff opted to take out the tracheostomy tube and conduct endotracheal intubation. The patient was moved to our hospital, intubated, and placed on ventilatory support to treat tracheal stenosis. Plain radiographic imaging of the chest showed clean scanned lung areas with no consolidation, collapse, or cavitary lesion. The left costophrenic angle is not visible. A clear right costophrenic angle is shown in the

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imaging. There is no indication of hilar or mediastinal lymph node hypertrophy. However, the sixth, eighth, and ninth ribs on the left side have been fractured.

On 22 January 2024, the cardiothoracic consultant conducted the bronchoscopy. This indicated a mid-tracheal stenosis of 1-2 cm long and 4 cm above the carina. The patient was put on quetiapine 25 mg NGT twice a day for sedation. On 29 January 2024, the quetiapine dose was raised to 50 mg NGT twice daily. After being extubated, the patient was moved to intermediate care three days later. Subsequently, he was discharged from the ICU to the long-term care unit. On the same day, the patient had acute respiratory distress and disrupted consciousness. Blood gas analysis (BGA) revealed type 2 respiratory failure, pH 7.0, PaCO2 105 mmHg, and a bilaterally reduced air entry. An endotracheal tube (ETT) size 5 was used for emergency intubation (a greater size could not pass through the tracheal stenosis) by an anesthesia consultant. The patient was attached to mechanical ventilation, sedated, and transferred to the ICU. A plain x-ray of the chest and heart was conducted and revealed prominent broncho-vascular markings, a right basal consolidation patch, and the diaphragm's left copula was raised. Sputum culture revealed Pseudomonas aeruginosa; thus, he was placed on meropenem 500 mg every 6 hours. On 26 February 2024, a tracheotomy was done. The patient experienced hyponatremia, in which serum sodium was 125 (**Table 1**). The patient was weaned from the mechanical ventilator to tracheal mask oxygen. The patient remained hyponatremic with serum sodium (Na) of 122 mmol/l. The quetiapine dosage was reduced by 25%. On 1 March 2024, serum Na was 117 mmol/l. On 2 March 2024, the patient was started on hypertonic saline 3% for three days, followed by hydrocortisone 50 mg IV TID. Serum Na increased to 132 mmol/l, so hypertonic saline 3% was stopped. On 2 March 2024, serum Na was 121 mmol/l. The serum osmolality value was 270 mOsm/kg (typical range 275-295). The sodium concentration in random urine was 174 mmol/l (typical range 30-90) (**Table 2**). The thyroid stimulating hormone (TSH) level was 0.89 IU/ml (typical range 0.27-4.2). On 4 March 2024, serum Na dropped by 126 mmol/l. Salt was supplied to the NGT feed. On 8 March 2024, serum Na was 123 mmol/l. Nephrology advised limiting free water consumption during the next 2-3 days. We also reduced the quetiapine dosage by 25%. On 11 March 2024, normal saline 60-80 ml/h (continuous IV infusion since 15 January 2024) was discontinued following the nephrology consultant's advice to improve hyponatremia. Salt was also increased throughout the NGT diet. On 12 March 2024, the serum osmolality value was 274 mOsm/kg (normal range 275-295). The sodium concentration in random urine was 124 mmol/l (normal range 30-90). The urine osmolality result was 517.89. On 15 March 2024, serum Na rose to 135 mmol/l. Quetiapine (25 mg AM and 50 mg PM) was reduced by another 25%. On 16 March 2024, serum Na rose to 136 mmol/l. On 20 March 2024, the quetiapine dosage was stopped, and the patient was transferred to the long-term care unit. The serum Na level was 139 mmol/l.

Discussion

The case report presents a rare incidence of quetiapine-induced hyponatremia in a young ICU patient, suggesting that even younger individuals can acquire this side effect (**Figure 1**). The serum sodium levels were in the normal range of between 136 and 137 mmol/l up to 25 February 2024. However, on 26 February 2024, the serum sodium levels substantially decreased to 125 mmol/l, meaning hyponatremia had developed. This date aligned with the introduction and subsequent dose increase of quetiapine to the patient, which started on 22 January 2024 and increased on 29 January 2024. Even after administering hypertonic saline and hydrocortisone, the serum sodium levels decreased to as low as 117 mmol/l on 1 March 2024. The gradual trend of the serum sodium levels decrease after the initiation of quetiapine and the subsequent dose escalation is an adequate demonstration that the role of quetiapine in inappropriately releasing antidiuretic hormone (ADH) and causing water retention that quetiapine presenting before hospital admission was sufficient to cause dilutional hyponatremia (**Figure 2**).

The pathophysiology of quetiapine-induced hyponatremia is thought to involve the SIADH. (1-5) In this case, the low serum osmolality of 270 mOsm/kg was one such deviation. Furthermore, the urine sodium levels obtained from the random sample, 174 mmol/l, told us that adequate amounts of sodium and inappropriately excessive amounts of water were being excreted. The combined effect of various factors described above might have led to the patient's increased predisposition to quetiapine-induced hyponatremia. Specifically, the patient's critical illness and polytrauma state, accompanied by significant medical interventions and a prolonged hospital course, altered the pharmacokinetics and pharmacodynamics of quetiapine. (1,4)

Moreover, the concurrent use of other medications and the patient’s comorbidities might have similarly

predisposed them to develop hyponatremia. Many agents, such as antidepressants, diuretics, and antiepileptic drugs, have previously resulted in SIADH and hyponatremia formation. Given that this case report did not enumerate the patient's entire medical history or drug regime, it could not be excluded that the collective effect of quetiapine and other medications taken by the patient might have amplified the risk of having SIADH and hyponatremia.

The patient's neurological condition, including head trauma and brain damage, may increase the possibility of disturbances in ADH secretion. (3,4) According to the literature, some neurological conditions, such as stroke, traumatic brain injury, and brain tumors, are linked with SIADH because of the disorders of regulatory mechanisms underpinning ADH release. Therefore, the patient's neurological state was a contributing factor in the enhancement of the vulnerability to quetiapine-related SIADH and hyponatremia. (6,7)

The management of quetiapine-induced hyponatremia remains a multi-faceted process. Gradual dose reduction or cessation of the quetiapine is essential to reduce its effect on ADH secretion. In this case, a gradual reduction in serum sodium following tapering of quetiapine from the first day would most likely contribute to the gradual improvement observed in the patient. Careful monitoring of blood sodium levels and appropriate fluid and electrolyte management are essential. (1,8)

Conclusion

This case report highlights the importance of ongoing awareness of quetiapine-induced hyponatremia in vulnerable age groups, particularly critically ill and polysymptomatic patients. Healthcare clinicians should assess vigilance by observing drug-induced problems and considering personal factors and individual risk dominance in pharmacologic prescriptions. A rigorous pharmacovigilance evaluation, with a responsive clinical pharmacologist's team, strengthened by polypharmacy potential, is required to ensure patient safety and prevent drug-induced problems.

Table 1. Initial lab results

Blood test	Result values	Reference range
Sodium (mmol/l)	131 (7/2/2024)	133-146
Potassium (mmol/l)	5.89 (7/2/2024)	3.5-5.3
Chloride (mmol/l)	95.4 (7/2/2024)	95-108
Creatinine (μmol/l)	53 (7/2/2024)	59-104
Urea (mmol/l)	8.1 (7/2/2024)	2.5-7.8
Bicarbonate (mmol/l)	27 (7/2/2024)	22-29
Glucose (mmol/l)	98 mg/dl=5.4 mmol/l (7/2/2024)	4-7
Thyroid-stimulating hormone (μU/l)	0.89 (2/3/2024)	0.3-4.2
Calcium (mmol/l)	2.35 (7/2/2024)	2.2-2.6
Serum osmolality (mOsm/kg)	279 (2/3/2024)	275-295
Albumin (g/l)	39 (7/2/2024)	35-50
Early morning cortisol (nmol/l)	Not done	250-600
C-reactive protein (mg/l)	123.9 (9/2/2024)	<10
Hemoglobin (g/l)	128 (7/2/2024)	130-180
White cell count (10^9/l)	26.41 (7/2/2024)	4.0-11.0
Platelet count (10^9/l)	540 (7/2/2024)	150-400

Table 2. Urine test results

Spot urine test	Results
Osmolarity (mOsm/kg) (7/3/2024)	517.89
Sodium (mmol/l) (2/3/2024)	174 (normal 30-90)
Urea (mmol/l) (7/3/2024)	286.2

Figure 1. Sodium levels during intensive care unit admission

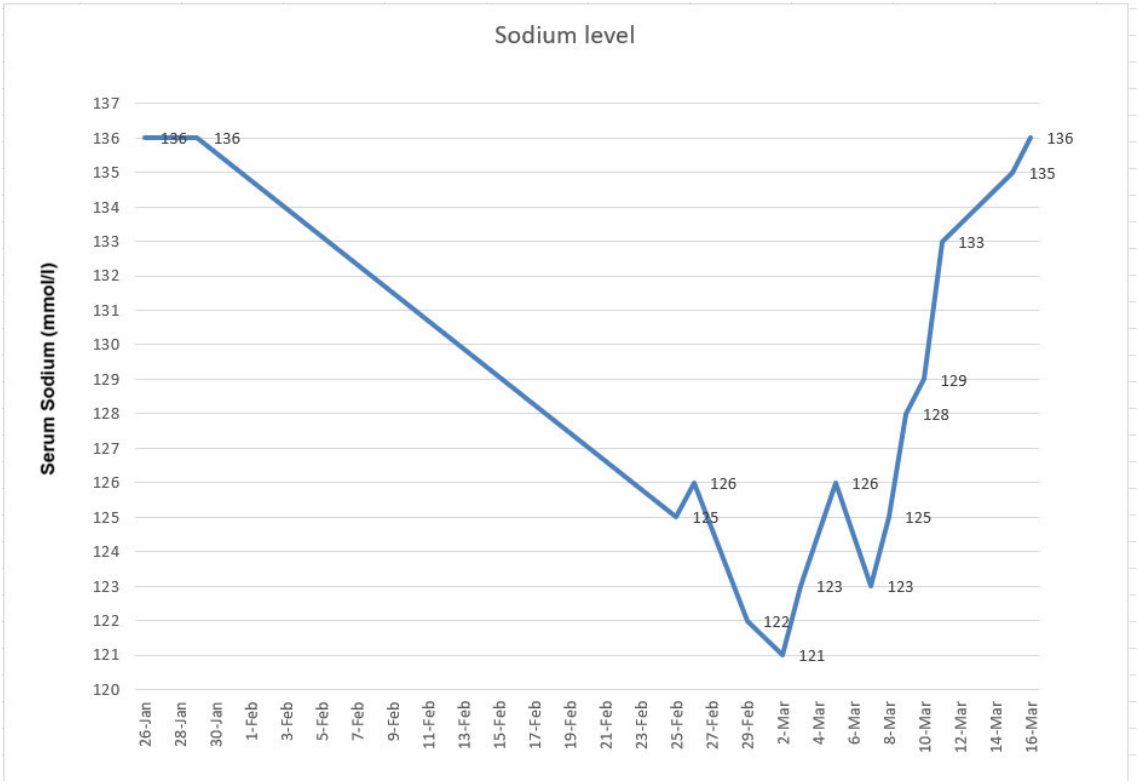
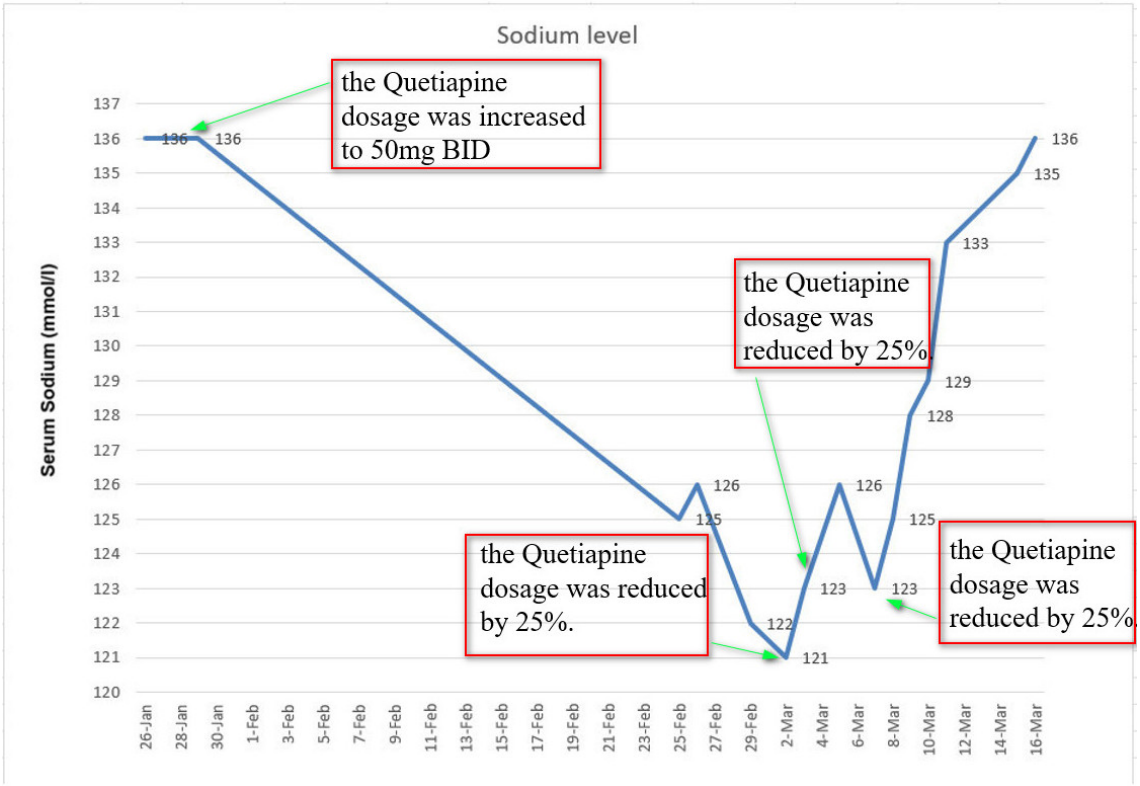


Figure 2. Sodium levels during intensive care unit admission in relation to quetiapine dosage



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