Case Report/*Olgu Sunumu*

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A Case of Severe Hypokalemia Triggered by Thiopental Sodium Infusion

Tiyopental Sodyum İnfüzyonu ile Tetiklenen Şiddetli Hipokalemi Vakası

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ABSTRACT

Dyskalemia is a rare but potentially life-threatening side effect of thiopental sodium, a sedative agent that has been utilized for many years in the treatment of patients with increased intracranial pressure. Severe hypokalemia may occur during the infusion, while rebound hyperkalemia may arise after the infusion is discontinued, posing significant challenges in intensive care settings. This case report examines a patient who developed severe hypokalemia (serum potassium 1.5 mmol L^{-1}) during thiopental infusion and rebound hyperkalemia (serum potassium 5.4 mmol L^{-1}) post-infusion cessation. These findings underscore the critical importance of close monitoring and appropriate management strategies in intensive care settings.

Keywords: Thiopental sodium, hypokalemia, electrolyte imbalance

ÖZ

Uzun yıllardır intrakranial basıncı artmış hastaların tedavisinde kullanılan bir sedatif ajan olan tiyopental sodyumun nadir görülen ancak potansiyel olarak yaşamı tehdit eden bir yan etkisi de diskalemidir. İnfüzyon sırasında şiddetli hipokalemi meydana gelebilirken, infüzyon kesildikten sonra rebound hiperkalemi ortaya çıkabilir ve yoğun bakım ortamlarında önemli zorluklara yol açabilir. Bu vaka raporunda tiyopental infüzyonu sırasında şiddetli hipokalemi (serum potasyumu 1,5 mmol L^{-1}) ve infüzyon kesildikten sonra rebound hiperkalemi (serum potasyumu 5,4 mmol L^{-1}) gelişen bir hasta incelenmektedir. Bu bulgular yoğun bakım ortamlarında yakın izleme ve uygun yönetim stratejilerinin kritik önemini vurgulamaktadır.

Anahtar sözcükler: Tiyopental sodyum, hipokalemi, elektrolit imbalansı

INTRODUCTION

Owing to its neuroprotective properties, thiopental sodium has been widely used as a sedative in patients with elevated intracranial pressure. However, this therapy is associated with several risks. The most common adverse effects of thiopental sodium infusion are hypotension, decreased cardiac motility, systemic dehydration, respiratory depression, and decreased gastrointestinal motility (1). Although these adverse effects are well documented, dyskalemia, which is characterized by severe hypokalemia during treatment and rebound hyperkalemia following discontinuation, is a less common but potentially life-threatening complication. This report highlights the clinical challenges associated with this rare adverse event.

CASE PRESENTATION

Following a pedestrian accident, a 20-year-old male sustained a diffuse traumatic subarachnoid hemorrhage in both convexities, notably in the left frontal region, along with a 3-mm subdural hemorrhage in the left temporal region. The patient was found to have a Glasgow Coma Scale score of 4–5; he was admitted to the intensive care unit for treatment and monitoring after endotracheal intubation to preserve the airway. The patient's cranial computed tomography scan is shown in Figure 1.

The anti-edema measures employed included hypertonic saline; maintenance of normothermia, normovolemia, normoglycemia, and normocarbia; and elevation of the head to 30°. Phenytoin was used as an anti-epileptic drug. Despite the ongoing infusion of noradrenaline, cerebral perfusion pressure

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remained low; therefore, thiopental sodium was initiated at a dosage of 2 mg kg^{-1} h^{-1} (Hour 0 or H0). Following the development of anisocoria 24 hours later (H24), a cranial computed tomography scan revealed a midline shift, prompting the patient to undergo a decompressive craniotomy.

After the initiation of thiopental sodium infusion, sodium levels were 152 mmol L^{-1} at 42nd hour (H42), despite the target sodium level being 145–150 mmol L^{-1}; therefore, the 3% sodium chloride treatment was discontinued. Potassium infusion was initiated at 60th hour (H60) when serum potassium levels were found to be 1.5 mmol L^{-1}. At 68th hour (H68), thiopental sodium infusion was discontinued. Potassium chloride replacement (180 mEq) was then performed; 13 hour after the thiopental infusion was discontinued (H81), the potassium level reached 5.4 mmol L^{-1}. Figure 2 shows an ECG image obtained at H60 when the serum potassium level was 1.5 mmol L^{-1}. Table I presents the sodium and potassium levels from the start of thiopental sodium infusion. Figure 3 illustrates the relationship between serum potassium levels and thiopental infusion.

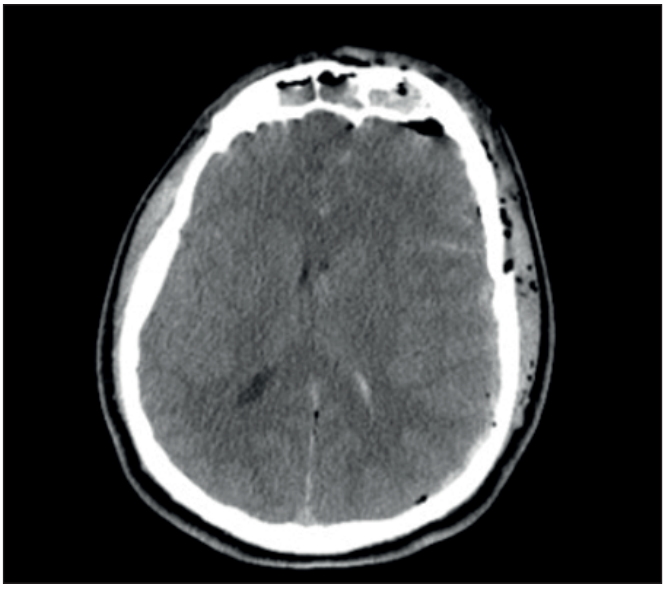

Figure 1. A section from the computed tomography image of the brain taken at the time of the patient's admission to the hospital. Diffuse traumatic subarachnoid hemorrhage in both convexities, along with subdural hemorrhage in the left temporal region.

On the 12th day of follow-up, percutaneous tracheostomy and endoscopic gastrostomy were performed. On day 25, the patient was discharged from the intensive care unit in a vegetative state, with a Glasgow Coma Scale score of 4T.

DISCUSSION

This case highlights the dual challenge of severe hypokalemia during thiopental administration and subsequent rebound hyperkalemia after infusion. In a study investigating the side effects of barbiturates in patients with head trauma, liver dysfunction was observed in 87% of patients, hypokalemia in 82%, respiratory complications in 76%, arterial hypotension in 58%, infection in 55%, and renal dysfunction in 47% (2). This indicates that the prevalence of hypokalemia is not low.

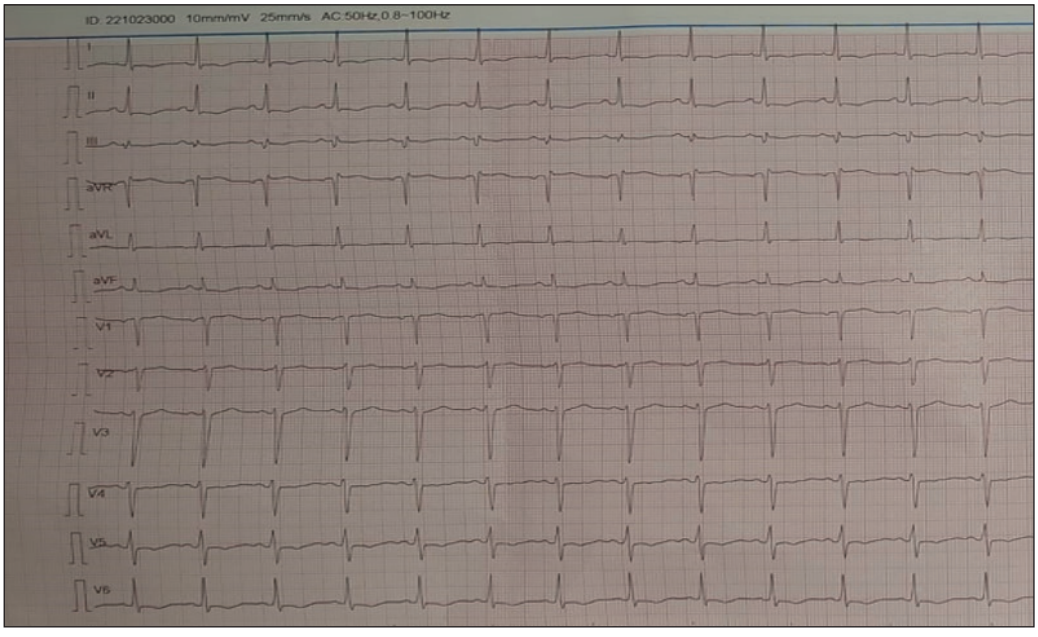

Figure 2. Electrocardiogram taken during the development of profound hypopotassemia. 1-p wave amplitude and width increase 2- t wave flattening and inversion 3- QTc:480 ms prolonged QTc present 4- There is a prominent u wave in leads V5-V6.

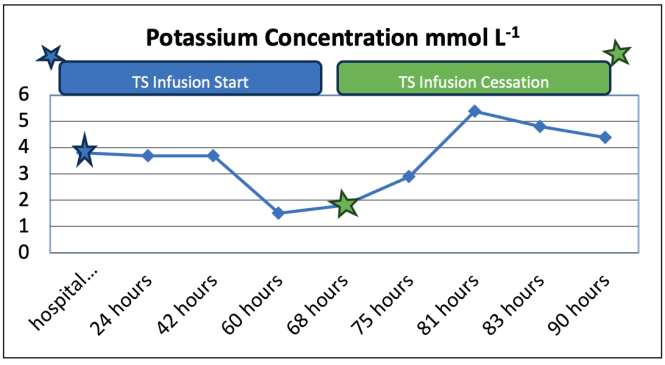

Figure 3. Correlation of patient's serum potassium concentration with thiopental sodium (TS) infusion.

Several mechanisms may account for the occurrence of dyskalemia resulting from thiopental infusion. Thiopental inhibits voltage-dependent potassium channels that sequester potassium within the cells (3). Furthermore, the inhibition of phosphofructokinase leads to a reduction in intracellular pyruvate and lactate production, subsequently increasing intracellular pH and promoting the translocation of potassium into cells (4). Consequently, severe hypokalemia may be encountered in clinical practice.

In another retrospective study involving 47 patients, 89.4% of the patients receiving thiopental treatment for intracranial hypertension developed hypokalemia after treatment initiation. In the same study, rebound hyperkalemia developed in 34% of patients after the discontinuation of thiopental treatment. Additionally, the study found that after thiopental induction, the median time to onset of severe hypokalemia was 11 hour (range 6–23 hours), whereas the median time to reach the lowest serum potassium levels was 25 hour (range 15–41 hours). The median onset time for hyperkalemia after treatment discontinuation was 31 hour (range 25–44 hours), with the median time to peak potassium levels occurring at 31st hour (range 28–56 hours) post-discontinuation. Moreover, the mean peak serum potassium level after cessation of thiopental treatment was 6.2 ± 0.8 mmol L^{-1}. This was the only study to implement a gradual reduction in infusion over a period of 12–24 hours (5). In our case, hypokalemia developed 60 hour after the initiation of the infusion, and the highest potassium level was observed 13 hour after its discontinuation. We observed a later onset of hypokalemia and a more rapid recovery of serum potassium levels after discontinuation of the infusion compared with those reported previously. This rapid recovery may be attributed to the fact that we did not gradually discontinue thiopental infusion for our patient.

In a previously reported case, the lowest potassium level was recorded as 1.6 mmol L^{-1} at 36th hour, with a total potassium replacement of 248.6 mmol. Following the gradual discontinuation of thiopental treatment, the highest recorded potassium level was 5.6 mmol L^{-1}. The authors argued that gradual cessation of thiopental treatment combined with reasonable potassium replacement was a key strategy for preventing dyskalemia. However, gradually tapering thiopental treatment did not completely prevent rebound hyperkalemia (6). In our case, thiopental treatment was not gradually discontinued, and potassium replacement was maintained at a reasonable level, which helped prevent severe rebound hyperkalemia.

CONCLUSION

We aimed to draw attention to hypokalemia, a rare yet serious adverse effect of thiopental sodium, which is an important sedative agent used in the intensive care unit. We emphasize the need for vigilant monitoring of serum potassium and sodium levels during and after treatment, particularly when therapy is discontinued. Proactive interventions are essential to minimize adverse outcomes and ensure patient safety.

AUTHOR CONTRIBUTIONS

Conception or design of the work: YKE
Data collection: GK
Data analysis and interpretation: YKE
Drafting the article: SSI
Critical revision of the article: SSI
Other (study supervision, fundings, materials, etc): GK
The author (SSI, YKE, GK) reviewed the results and approved the final version of the manuscript.

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