CASE REPORT

Refractory hypokalemia during barbiturate coma therapy used for treating refractory intracranial hypertension in traumatic brain injury

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ABSTRACT

Barbiturate coma therapy (BCT) is a choice treatment for refractory intracranial hypertension after all surgical or medical managements have failed to control the intracranial pressure (ICP). It helps to reduce cerebral blood flow, cerebral metabolic rate of oxygen consumption and ICP. However, this therapy can also cause many complications. One of the underreported, but life-threatening complications is refractory hypokalemia, which can lead to subsequent rebound hyperkalemia after sudden cessation. We report our experience of managing unusual complication of refractory hypokalemia during BCT with thiopentone in postdecompressive craniectomy patient.

Key words: Barbiturate coma, hypokalemia, intracranial hypertension, traumatic brain injury

Introduction

Barbiturate coma therapy (BCT) is an effective second-line treatment for refractory intracranial hypertension. However, it is also potential to cause some complications such as arterial hypotension, infections, hepatorenal dysfunction and reduction in cortisol activity.[1] One of the underreported complications is severe fluctuation of potassium level. We present herein a case of severe refractory hypokalemia during BCT with thiopentone.

Case Report

A 46-year-old man, alleged fall from height, presented with transient loss of consciousness with Glasgow coma scale (GCS) of 14/15. His hemodynamic status was stable. Computed tomography (CT) brain showed multiple contusions, right occipital extradural hematoma, right frontal-parietal subdural hematoma and bilateral subarachnoid hemorrhage.

He was initially planned for close observation. After 16 h of observation, GCS was deteriorated, and repeat CT brain showed worsening of right parietal contusion and right hemispheric edema with midline shift to the left. He underwent emergency right decompressive craniectomy, evacuation of clots and insertion of intracranial pressure (ICP) monitoring with baseline potassium of 3.2 mmol/l. The surgery was uneventful and postoperative potassium level was 4.1 mmol/l.

Intracranial pressure was initially maintained ≤ 20 mm Hg but persistently elevated ≥20 mm Hg after 10 h. Repeat CT brain showed an expansion of left frontal contusion, effaced bilateral ventricles and obliterated right prepontine cistern. Therefore, BCT with thiopentone bolus dose of 250 mg and followed by continuous infusion at 2 mg/kg/h were started. The aims were to achieve ICP ≤20 mm Hg and bispectral index ≤20. Baseline serum potassium prior to thiopentone infusion was 3.4 mmol/l, and ongoing 67 mmol of potassium supplement was given over 24 h. 7.4% hypertonic saline (HTS) infusion at the rate of 30 ml/h was also started. Electrolytes and serum osmolarity were monitored every 6 h. HTS infusion was continued at 30 ml/h for 20 h, followed by 15 ml/h for the next 8 h; it was stopped when serum sodium was >155 mmo/l. After 12 h of BCT, potassium level decreased to 3.0 mmol/l and further decreased to 1.7 mmol/l just 2 h after we increased thiopentone infusion to 3 mg/kg/h due to persistent ICP ≥20 mm Hg.

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Subsequent potassium measurement remained at 1.6 mmol/l with thiopentone infusion rate at 3 mg/kg/h. At this moment, there was no administration of inotropic drugs, insulin or β-agonist therapy. Glucose level was also within normal range, and there was no alkalosis. We managed to control ICP ≤20 mm Hg with thiopentone infusion rate of 3 mg/kg/h.

At the end of the first 24 h of BCT, potassium level was markedly dropped to 1.3 mmo/l and electrocardiogram (ECG) showed ST segment depression and bradycardia. BP was also reduced and required noradrenaline infusion. A total of 108 mmol of potassium supplement was given over 24 h since the beginning of BCT. Thiopentone was stopped after 38 h of infusion when ICP was maintained ≤20 mm Hg and potassium level at the moment remained at 1.5 mmol/l. Urinary potassium level was within normal range. He was never started on frusemide or corticosteroids therapy and his renal function was normal. Patient was still on noradrenaline infusion at this moment to maintain mean arterial pressure ≥70 mm Hg and cerebral perfusion pressure ≥60 mm Hg. After 6 h of thiopentone cessation, ICP was again increased ≥30 mm Hg and thiopentone therapy was recommenced at 2 mg/kg/h. At this point, the potassium level remained ≤2.0 mmol/l. Examination showed unequal pupils size and urgent CT brain showed an expansion of left frontal contusion with worsening cerebral edema and midline shift about 2 cm to the right.

Patient required second emergency left decompressive craniectomy and clot evacuation in spite of potassium level of 1.4 mmol/l. ECG showed transient ventricular tachycardia intraoperatively prior to surgical incision. However, it responded well to 26.8 mmol of potassium fast correction. Potassium level was closely monitored intraoperatively, and It ranged between 1.5 mmo/l and 1.9 mmol/l. During the surgery, thiopentone was temporarily withheld and the surgery lasted for 4 h with ECG subsequently remaining in the sinus rhythm.

After 48 h of BCT, potassium level remained between 1.6 mmol/l and 1.8 mmol/l and another total of 120.6 mmol potassium supplement was given. The dose of thiopentone infusion at that moment was 200 mg/h and we decided to reduce the infusion gradually by 20 mg every hour for 10 h. Further potassium correction was temporarily withheld due to concern over transcellular potassium shift back to the extracellular space causing rebound hyperkalemia. Potassium level was monitored every 2 h. After 6 h of stopping thiopentone infusion, he became hypotensive and bradycardic. Patient also developed diabetes insipidus at that moment with evidence of polyuria and persistent hypernatremia with serum sodium between 160 mmo/l and 175 mmo/l. Despite aggressive resuscitation and inotropic supports with noradrenaline, dopamine and dobutamine, the patient developed cardiac arrest and succumbed to death after 30 min of cardiopulmonary resuscitation. The latest potassium level 4 h prior to cardiac arrest was 1.2 mmol/l. There was no hyperkalemic episodes documented up to 4 h before the cardiac arrest. Cumulative dose of the whole potassium correction was 429.4 mmol during 6 days of managing this patient.

Discussion

Barbiturate coma therapy has been known as a method for controlling ICP, which is refractory to medical and surgical treatments. In our case, our first management was always decompressive craniectomy before we decided to start BCT after the ICP was persistently high. We have done decompressive craniectomy twice in this case and subsequently, followed by BCT. We have experienced severe hypokalaemia after 12 h of the first BCT and it remained persistently low until the end.

Hypokalemia is initially thought to be rare during BCT, but it is actually common. It has been reported to occur in 82%,[1] 65%,[2] and 89.4%[3] of the patients during BCT. However, severe hypokalemia <2.0 mmol/l was reported in only 23.4%[3] and 25.8%[1] of the patients.

Hypokalemia is explained by intracellular shift of potassium caused by thiopentone therapy.[4] It leads to inhibition of neuronal voltage-dependent on potassium current and inhibition of phosphofructokinase. This will cause an increase in intracellular pH and promote a shift of potassium into the cells. There is possibility that persistence increase in ICP provoked an intense release of endogenous catecholamine and induced β2 stimulation of Na^+-K^+ pump.[5]

In our case, the other factors that could contribute to hypokalemia were inotropic support and HTS therapy. Hypokalemia has been documented as one of the complications of HTS. During HTS therapy, sodium is reabsorbed in exchange for potassium to maintain osmotic gradients necessary for urinary concentration. This results in a large amount of potassium being lost in the urine.[6,7] However, the urinary potassium level was within normal limit in our case. Other factors that can contribute to hypokalemia in head injury are endogenous catecholamine, insulin infusion and therapeutic cooling.[8]

Severe hypokalemia in our case has been managed aggressively with the potassium supplement in order to prevent from fatal complications. We also monitored electrolytes closely every 6 h and continuous ECG monitoring throughout this therapy. Cumulative dose of the whole potassium correction in our case was 429.4 mmol during 6 days of managing this patient.

After cessation of the therapy, rebound hyperkalemia can occur, and it has been reported in 34% of the patients. All patients who developed hyperkalemia had been previously hypokalemic and had higher total dose replacement of potassium.[3] We have stopped thiopentone twice in our case. The first time was an abrupt cessation, and the second time

was planned for gradual cessation. However, there were no episodes of rebound hyperkalemia documented during both episodes.

Our plan to stop potassium replacement despite persistent severe hypokalemia during gradual cessation of thiopentone infusion required a review. Gradual cessation might help to reduce the potential risk of rebound hyperkalemia, but we could not let the condition of severe hypokalemia without supplementation. This could further weaken the function of the heart, which most likely led to bradycardia and cardiac arrest.

Conclusion

Refractory hypokalaemia is life-threatening complication during BCT. Our case showed that hypokalemia might be also contributed by other factors such as HTS therapy, endogenous catecholamines, diabetes insipidus and inotropic support.

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