Cardiac Arrest due to Severe Hypokalemia during Barbiturate Coma Therapy in a Patient with Severe Acute Head Injury

-A case report-

Departments of 1Anesthesiology and Pain Medicine, 2Neurosurgery, 3Institute of Health Sciences, Gyeongsang National University College of Medicine, Jinju, Korea

Il-Woo Shin, M.D.,1 Ju-Tae Sohn, M.D.,1,3 Ju-Young Choi, M.D.,1 Heon Keun Lee, M.D.,1 Chul-Hee Lee, M.D.,2 and Young-Kyun Chung, M.D.1

An emergency left frontotemporal craniectomy with direct neck clipping and hematoma removal was performed in a 36-year-old man with a ruptured left middle cerebral artery aneurysm and sylvian hematoma. Because of severe brain swelling postoperatively, we induced barbiturate coma therapy to treat his intractable brain swelling. He had an initial loading dose of sodium thiopental (5 mg/kg) followed by continuous infusion of sodium thiopental (5 mg/kg/hour). The lowest potassium concentration recorded during the barbiturate coma therapy was 1.1 mmol/L, necessitating treatment with cardiac massage, epinephrine, and atropine because of asystole and severe bradycardia. However, he did not recover from cardiac arrest. We present here a case of cardiac arrest due to severe life threatening hypokalemia that occurred during barbiturate coma therapy. (Korean J Anesthesiol 2006; 50: S 71–3)

Key Words: barbiturate coma, cardiac arrest, hypokalemia.

Barbiturate coma therapy has been used for several years in the treatment of patients with severe intractable intracranial hypertension. The side effects1 encountered during barbiturate coma therapy include arterial hypotension, electrolyte imbalance,2,3 respiratory complications, hepatic dysfunction and renal dysfunction. Dramatic changes in serum potassium are not common in patients with increased intracranial pressure who receive high-dose barbiturates. We present here a case of cardiac arrest due to severe life threatening hypokalemia that occurred during barbiturate coma therapy in a patient with direct surgical clipping of a ruptured middle cerebral artery (MCA) aneurysm and sylvian hematoma removal.

CASE REPORT

A 36-year-old man was admitted to the intensive care unit at university hospital with a subarachnoid hemorrhage (Hunt Hess grade IV) and acute sylvian hematoma due to a ruptured MCA aneurysm visualized on a brain computerized tomogram. An emergency left frontotemporal craniectomy with direct neck clipping of the left MCA aneurysm and left sylvian hematoma removal was performed on his admission. The intracranial pressure (ICP, Brain-Pressure monitor Model: HDM 13.3, Spiegelberg, Germany) was monitored postoperatively. He had severe brain swelling that was observed on the brain computerized tomogram taken postoperatively, and ICP increased to 37.9 mmHg postoperatively. Therefore, the patient was moderately hyperventilated (PaCO2: 29-32 mmHg) and mannitol (6 × 37.5 g/day) was used to control the ICP. We then started barbiturate coma therapy to treat the intractable brain swelling 7 hours after the operation. He had an initial loading dose of sodium thiopental (5 mg/kg) followed by a continuous infusion of sodium thiopental (5 mg/kg/hour) (Fig. 1). His EEG displayed burst suppression during barbiturate coma therapy. The ICP was controlled after barbiturate coma therapy was started, and the ICP was decreased to 10.5 mm Hg. He received intravenous dopamine (5-20 ug/kg/min) and dobutamine (5-20 ug/kg/min) 4 hour after barbiturate coma therapy was started to maintain

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Corresponding to: Ju-Tae Sohn, Gyeongsang National University College of Medicine, 90 Chilam-dong, Jinju 660-702, Korea. Tel: 82-55-750-8141, Fax: 82-55-750-8142, E-mail: jtsohn@nongae.gsu.ac.kr
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blood pressure. He received a total of 55 mmol of potassium over the postoperative period of 16 hours. The lowest serum potassium concentration recorded during barbiturate coma therapy was 1.1 mmol/L (Fig. 1), which necessitated drastic intervention with cardiac massage, epinephrine (1 mg), and atropine (0.5 mg). We then had to discontinue the barbiturate infusion because of asystole and severe bradycardia. The severe hypokalemia induced cardiac arrest, and cardiopulmonary resuscitation was unsuccessful. His body temperature was stable normothermia throughout treatment. The arterial blood pH was normal (pH 7.35) or moderately acidic (pH 7.15) before and during the barbiturate coma therapy, respectively (Fig. 1).

**DISCUSSION**

High-dose barbiturate coma therapy has been used for hemodynamically stable and salvageable severe head injury patients that suffer from intracranial hypertension that is refractory to the maximal medical and surgical intracranial pressure-lowering therapy. The immediate complications from high-dose barbiturate therapy include tachycardia and hypotension. The delayed complications from high-dose barbiturate therapy include hypokalemia, liver dysfunction, infection, cardiac failure and renal failure.

Several factors, including catecholamine levels, body temperature, acid-base balance, severe acute head injury, barbiturates, and mannitol, can affect serum potassium levels, as was seen in this case. Dopamine infusion (10, 30 μg/kg/min) induces hypokalemia in dogs through beta-2-adrenergic stimulation of the sodium-potassium pump. However, the infusion of dopamine during barbiturate coma therapy probably did not cause hypokalemia (potassium; 2.8 mmol/L), because dopamine infusion was started 4 hours after the barbiturate coma therapy was started. Induced hypothermia (32°C) has been used as a potential treatment for neurologic trauma, and is associated with severe electrolyte disturbances, such as hypokalemia, hypomagnesemia, and hypophosphatemia. As this patient was at stable normothermia during the barbiturate coma therapy, hypothermia-induced polyuria did not induce the hypokalemia observed in this case. For patients with severe head trauma and the potential risk of excessive catecholamine release, special attention must be given to excessive hypokalemia and hyperkalemia following head injury. An intracellular potassium shift due to increased plasma catecholamine (norepinephrine, epinephrine)-induced beta-2-adrenergic stimulation of the sodium-potassium pump may have been partially involved in this hypokalemia.

This patient received 1,000 cc of 15% mannitol before barbiturate coma therapy. However, a previous study reported that intravenous 20% mannitol (1.4 g/kg) administration does not induce hypokalemia in a patient with a brain tumor. Some reports indicate that an increase in plasma osmolality induced by administration of mannitol causes an increase in plasma potassium due to the shift of potassium from cells to the extracellular fluid. In addition, serum potassium increased from 3.7 mmol/L to 4.6 mmol/L (Fig. 1) before barbiturate coma therapy. Taking into consideration the above facts and reports, it seems highly unlikely that polyuria due to mannitol administration alone caused this kind of severe hypokalemia.

Induction of barbiturate coma is accompanied by a physi-