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Case Report Open Access

Hypertonic Mannitol-Induced Hyperkalemia during Craniotomy

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Abstract

We experienced two cases of mannitol-induced hyperkalemia during craniotomy for ruptured aneurysms. Hyperkalemia was first diagnosed by peaked T wave on the ECG. Serum potassium concentration in each patient was approximately 2 mEq/l higher than the baseline value, reaching 6.0 and 5.7 mEq/l, respectively, at 2 hours after completion of infusion of 45 and 30 g mannitol, respectively. Although the underlying mechanism was not elucidated, we recommend that patients with potassium concentration more than 4 mEq/l before infusion, should undergo repeated arterial blood gases analysis until at least 2 hrs after completion of mannitol infusion.

Keywords: Hypertonic mannitol; Hyperkalemia; Craniotomy; ECG abnormality

Introduction

Hypertonic mannitol has been used since 1960s to reduce intracranial pressure during neurosurgery [1]. Electrolyte abnormalities including hyperkalemia are well known complications of mannitol [2,3]. Although a search of the 1980-2000 MEDLINE database showed only one case report of mannitol-induced severe hyperkalemia [4], no less than seven cases of mannitol-induced hyperkalemia during craniotomy have been reported since 2000 [5-10]. Here, we add to the list two more cases of mannitol-induced severe hyperkalemia, with different clinical presentation.

Case Report

Case 1

A 57-year-old man (height 174 cm, weight 67 kg) with subarachnoid hemorrhage (SAH) was admitted to our hospital for clipping of aneurysm of the right internal carotid artery (IC-Anterior choroidal artery). His diffuse SAH was evaluated Fisher grade 3. The patient had been in good health and not on any medications. Preoperative electrocardiography (ECG) and chest radiography showed no abnormalities. Preoperative serum concentrations of sodium, potassium and creatinine (Cre) were 140 mEq/L, 4.0 mEq/L and 0.6 mg/dl, respectively. No pre-medication was provided. General anesthesia was induced with thiopental 300 mg, remifentanil 0.4 μg/kg/min and inhaled sevoflurane 2%. Vecronium bromide (8 mg) was administered for tracheal intubation. The lung was ventilated using oxygen in air (F_IO_2 0.4) and end-tidal carbon dioxide was maintained at 25-30 mmHg. Anesthesia was maintained with remifentanil 0.1 to 0.4 μg/kg/min, inhaled sevoflurane 2-2.5% and vecronium bromide. The patient was hemodynamically stable throughout induction of anesthesia. Acetate Ringer solution 400 ml was infused initially before mannitol and after the start of surgery, mannitol 45 g was infused during a 15-min period before incision of the dura mater. Approximately 110 min after completion of mannitol infusion, the ECG showed peaked T wave without arrhythmia. Arterial blood gases (ABG) showed pH of 7.424, PaO_2 of 190 mmHg, $PaCO_2$ of 32 mmHg, base excess of -2 mmol/l, and blood tests showed sodium concentration of 129 mEq/l and potassium concentration of 6.0 mEq/l. Acetate Ringer solution was discontinued and normal saline was started. Furosemide 5 mg and 7% HCO_3- solution 20 ml were also administered. This resulted in a decrease in the T wave amplitude, and subsequent gradual normalization. At the end of surgery, sodium and potassium concentration and base excess were 134 mEq/l, 4.4 mEq/l and -0.5 mmol/l, respectively. Surgery was completed over 380 min without further incident. Urine output was 700 ml and urine color was clear. The postoperative course was uneventful.

Case 2

A 49-year-old man with SAH was admitted to the Emergency Department of our hospital for clipping of aneurysm of the right internal carotid artery (IC-Anterior choroidal artery). His diffuse SAH was evaluated Fisher grade 3. Neurological evaluation on admission showed Hunt & Kosnik grade 3 and he was intubated at the Emergency Department. Preoperative ECG and chest radiography showed no abnormalities, and serum electrolytes showed sodium 138 mEq/L and potassium 3.7 mEq/L, with serum Cre of 0.6 mg/dl. The patient was not on any medications, as indicated by the accompanying family members. Anesthesia was maintained with remifentanil (0.1-0.3 μg/kg/min), inhaled sevoflurane (1.5-2.0%) and vecronium bromide. The lung was ventilated using oxygen in air (F_IO_2 0.4) and end-tidal carbon dioxide was maintained at 25-30 mmHg. Acetate Ringer solution (250 ml) was infused first, then followed by mannitol infusion (30 g, over a 20-min period before incision of the dura mater). Approximately 150 min after completion of mannitol infusion, the ECG showed peaked T wave without arrhythmia. ABG showed pH of 7.393, PaO_2 of 213 mmHg, $PaCO_2$ of 33 mmHg, base excess of -3.1 mmol/l, and serum electrolytes showed sodium 130 mEq/l and potassium 5.7 mEq/l. Acetate Ringer solution was discontinued and normal saline was started. Furosemide 2.5 mg and 7% HCO_3-solution 60 ml were also administered. These changes resulted in gradual normalization of the T wave amplitude. At the end of surgery, serum electrolyte analysis showed sodium 131 mEq/l, potassium 5.0 mEq/l and base excess -0.6 mmol/l. Surgery was completed over 305 min without further incident. Urine output was 500 ml and urine color was clear. The patient was extubated successfully the day after surgery and had uneventful postoperative course.

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Age (yr)	Gender	Diagnosis	Mannitol (g)	Potassium concentration (mEq/L)			Peak time (min)*	Authors
				Before IV	Peak	End of surgery		
52	Male	SAH	60	4.8	6.8	5.0	40	Seto et al.[5]
34	Male	SAH	60	3.1	5.4	3.5	20	Hirota et al.[6]
68	Male	ICH	100	4.1	6.1	5.4	60	Hirota et al.[6]
15	Male	AVM	120	3.4	6.6	5.1	50	Kimura et al.[7]
31	Female	Tumor	80	4.1	6.1	4.4	20	Hassan et al.[8]
41	Male	SAH	120	NR	7.5	3.9	50¶	Flynn [9]
43	Male	Tumor	60	4.8	6.7	4.0	30¶	Tobita et al.[10]
57	Male	SAH	45	4.0	6	4.4	110	Current report
49	Male	SAH	30	3.7	5.7	5	150	Current report

*Time between completion of mannitol infusion and peak concentration of potassium
¶Time between initiation of mannitol infusion and peak concentration of potassium
NR: not reported, SAH: Subarachnoid hemorrhage, ICH: Intracerebral hemorrhage, AVM: Arteriovenous malformation

Table 1: Characteristics of hypertonic mannitol-induced hyperkalemia in seven patients reported since 2000.

Discussion

We experienced two cases of severe hyperkalemia during craniotomy for ruptured aneurysms, although serum potassium concentration is decreased in a patient with SAH [11]. We consider that hemolysis and acute renal failure during surgery could be completely ruled out because urine color was clear throughout anesthesia and pre- and postoperative renal function was within the normal range in each patient. Mannitol-induced hyperkalemia during craniotomy has been reported since 2000 [5-10] and we therefore confirm that hyperkalemia in our two cases was also induced by hypertonic mannitol. Table 1 summarizes the features of the seven published cases of mannitol-induced hyperkalemia [5-10] and compares them to our two patients. We observed common features in all nine patients. First, the ECG abnormality provided a clue to the development of hyperkalemia. All anesthesiologists in these case reports suspected hyperkalemia and decided to order ABG after observing the peaked T wave on the ECG. ECG in patients with SAH has shown morphological changes including T-wave abnormalities [12]. Theses changes appear within first 48 h following SAH and therefore anesthesiologists should distinguish the cause of elevated T wave on ECG, i.e. hyperkalemia or SAH itself. Second, the increase in serum potassium concentration was approximate 2 mEq/l, relative to the baseline value, after completion of mannitol infusion.

On the other hand, the patients showed the following differences. First, the peak potassium concentrations were noted within 1 hr after completion of mannitol infusion [5-10], whereas maximal hyperkalemia occurred in our patients at approximately 2 hrs after completion of mannitol infusion. This finding emphasizes the need for continuous care of hyperkalemic patients until at least 2 hrs after completion of mannitol infusion. Second, only a small dose of mannitol induced hyperkalemia in our patients, while a larger dose of mannitol was infused in at least three patients [6,7,9]. Manninen et al. [13] reported that infusion of high-dose mannitol (2 g/kg) resulted in significant increase in serum potassium and that severe hyperkalemia developed in some patients. They speculated that hyperosmolality induced by large-dose mannitol moves potassium-rich intracellular fluid into the hypertonic extracellular compartment. However, when intracellular potassium concentration is high, e.g., intracellular fluid is extremely low or intracellular potassium concentration is much higher than extracellular, a slight increase of osmolality in the extracellular fluid by small-dose mannitol further increases the intracellular potassium concentration and massive potassium with low fluid content results in diffusion of potassium from intracellular to the extracellular compartment to maintain steady cell osmolality.

Two patients described in the previously published case reports suffered from life-threatening arrhythmia induced by mannitol [5,9]. This finding emphasizes the need to measure serum potassium concentration before infusion of hypertonic mannitol. Unfortunately, we cannot measure intracellular potassium concentration and cell osmolality. When the concentration of potassium is higher than 4 mEq/l before hypertonic mannitol infusion, ABG should be analyzed repeatedly until at least 2 hrs after completion of infusion. Hypertonic saline was reported to be more efficacious than mannitol in lethal intracranial hypertension in addition to avoidance of electrolyte imbalance [14,15]. Therefore hypertonic mannitol should be alternatively substituted with hypertonic saline when high concentration of potassium before administration of mannitol is observed already.

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