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Case Report Open Access

Acute Hyperkalemia and Hyponatremia Following Intraoperative Mannitol Administration

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Abstract

Intravenous mannitol may lead to electrolyte disturbances involving sodium and potassium and resulting in cardiac dysfunction. This report demonstrates that when mannitol is given during intracranial surgery it can cause large increase potassium ion concentration and decrease in sodium ion concentration in the absence of any other possible causes. Intraoperative checks of serum electrolyte levels, arterial blood gas analysis and electrocardiogram monitoring could be recommended to be done routinely when using mannitol.

Introduction

Mannitol is a commonly used agent employed to decrease intracranial pressure during craniotomy. Its actions as an osmotic diuretic can cause large shifts in ion concentrations of sodium and potassium. In some cases this may cause disturbance of cardiac function. This is another report showing that during intracranial surgery the use of mannitol can cause hyperkalemia in the absence of any other possible causes.

Case

37 year old male with no previous medical, surgical or significant social history presented for orbytozygomatic craniotomy and intracranial dermoid cyst resection. Patient had no prior allergies. He was not taking any medications, vitamins or nutrition supplements preoperatively. His physical and neurological examinations were both unremarkable, and his preoperative serum electrolyte levels, complete blood count and coagulation studies were within normal limits. His preoperative arterial blood pressure was 108/70 mmHg, heart rate was 60 and he was afebrile. His weight was 68 kilograms Midazolam was given on arrival to operating room at 07:35 am. General anesthesia was induced at 08:00 am with Propofol, Lidocaine and Rocuronium. Sevoflurane, oxygen, air and Remifentanyl infusion were used for maintenance. Shortly after general anesthesia was induced a radial artery catheter was placed in the right wrist. Arterial Blood Gas (ABG) sample was sent for analysis at 09:08 am and all data were within normal limits (Table 1). 10 mg of intravenous Dexamethasone were administered and slow intravenous infusion of 1 g of Phosphenytoin was infused slowly prior to the start of the surgery. Surgery then began at 09:32 am. At 09:45 am surgeon requested to give patient 50 g of Mannitol (0.7 g/kg). Mannitol was intravenously infused in a period of 45 minutes. Infusion was completed by 10:30 am. All vital signs remained stable. At 10:45 peaked T-waves were noted on EKG monitor. ABG sample was immediately sent and revealed potassium level of 7.2 mmol/l and sodium level of 126 mmol/ml. Another ABG sample was sent for confirmation and revealed same deranged electrolyte levels. One gram of Calcium Chloride, 10 units of regular insulin and 25 ml of 50% Dextrose were immediately administered and moderate hyperventilation was started to treat hyperkalemia. No cardiac dysrhythmias followed. Next ABG sample was sent at 11:57 am and showed potassium level of 4.2 mmol/l. EKG data shortly returned to the baseline. No further treatment was administered. Adequate urine output and balanced intravenous fluid administration (lactated Ringer's solution) were maintained throughout the whole case (Table 1). Follow up ABG sample was sent at 12:30 and revealed no electrolyte or acid-base abnormalities as well. Surgery was successfully completed and postoperative course was uncomplicated. Three days later patient was discharged home.

Discussion

Mannitol is commonly used in neuroanesthesia to reduce intracranial volume and pressure and can lead to serious electrolyte abnormalities [1]. Mannitol is an osmotic diuretic. It increases urinary losses of sodium and water. Mannitol is filtered by the glomerulus and does not undergo tubular reabsorption [2]. If very high doses of mannitol are administered, or if the drug is given to patients with preexisting renal insufficiency, it may be retained in the circulation [3]. The resulting elevation in plasma osmolality, similar to that created by elevated sodium, results in the passage of water and potassium out of cells. This may cause pulmonary edema, hyponatremia, dilutional metabolic acidosis and hyperkalemia [4]. Mannitol leads to increased serum osmolality, osmotic movement of water out of cells and hyponatremia secondary to dilution. The development of hyponatremia in a mannitol recipient is usually transient and sometime resolve in a short period of time with adequate hydration and diuresis [5]. In this case hyponatremia of 126 mmol/ml shortly resolved with 600 ml of intravenous fluid administration and subsequently ensued 200 ml diuresis. Sometime mannitol causes hypokalemic, hypochloremic alkalosis. This alkalosis usually is associated with volume contraction and diuresis. This alkalosis can be lessened if normovolemia is maintained [6]. In this case patient received adequate hydration throughout the case and alkalosis never ensued. Nevertheless severe hyperkalemia has developed. All commonly plausible causes of potassium increment resuscitation were carefully excluded. Making diagnosis by exclusion is not uncommon in anesthesiology practice. It is common to diagnose Cerebral Salt Waste Syndrome and Syndrome of Inappropriate Antidiuretic Hormone Secretion by exclusion [7,8].

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Data in this case suggests that potassium movement across cellular membrane into extracellular fluid was most probable cause of hyperkalemia. Interestingly enough, mannitol is routinely administered at lower doses (usually 12 to 25 grams totally) during kidney transplant in patients with ESRD to increase cadaveric kidney renal blood flow without any hyperkalemic sequel most of the time [9]. When mannitol is used to decrease intracranial pressure the dose is much higher (usually 0.5-1 gram per kilogram). In this case patient received 50 grams of mannitol making potassium movement across cellular membrane into extracellular fluid a most probable cause of hyperkalemia [6].

It was previously described that Mannitol in high doses can cause significant hyperkalemia and in low doses can cause hypokalemia [4]. The exact mechanism is yet to be understood. Mannitol induced transient increase in plasma osmolality, similar to that produced by hypernatremia, may result in the osmotic shift of water and potassium out of cells. This may cause extracellular fluid volume expansion, hyponatremia, metabolic acidosis and hyperkalemia. Mannitol may also cause phenomena leading to a shift of intracellular K^+ -rich fluid to the extracellular fluid compartment to maintain the tonicity. This can be caused by two possible mechanisms: (1) rise in cell potassium concentration induced by water loss favoring passive potassium exit through potassium channels in the cell membrane; and (2) the frictional forces between solvent (water) and solute can result in potassium being carried out through the water pores in the cell membrane (a process that is called solvent drag). A similar process occurs with acute hypernatremia and commonly blamed for the hyperkalemia observed with significant hyperglycemia in poorly managed diabetes mellitus [10]. Hassan et al. in the similar case report mentioned basically same two possible mechanisms to explain increase in the plasma potassium concentration after mannitol administration. First mechanism is the rise in cell potassium concentration induced by water loss favors passive potassium exit through potassium channels in the cell membrane. Second mechanism is the frictional forces between solvent (water) and solute can result in potassium being carried out through the water pores in the cell membrane [11]. There are other possible mechanisms by which mannitol increases serum potassium concentration. Those causes could include increased potassium intake, decreased urinary excretion or endocrine abnormalities. Hyperkalemia could be a result of dilutional acidosis caused by gain of extracellular fluid [4,6] 10 or by direct damage to red blood cells by mannitol [12]. None of those factors could be implicated in our case. We did not use any medication which could result in acutely developed hyperkalemia or hyponatremia. Preoperatively given Dexamethasone [13] and Phosphenytoin are not known for clinically significant hyperkalemia effects when single dose used [14]. Urine output was adequately maintained before, during and after mannitol infusion (Table 1). All this suggests that potassium movement across cellular membrane into extracellular fluid was probably a cause of hyperkalemia. Some factors can potentially facilitate the transcellular potassium movement: increase in plasma osmolality [4], hemolysis and rhabdomyolysis [15]. In our case there was no blood transfusion done, which makes potassium overload unlikely. There were no signs or symptoms suggestive of rhabdomyolysis or hemolysis as well. Giving these facts, the increase in plasma potassium concentration could be secondary to an increase in plasma osmolality. Rapid or repeated administration of mannitol is also known to increase likelihood of hyperkalemia development [16]. In our case Mannitol was administered over 30 minutes and no repeated doses were used.

Some previous case reports of complications during mannitol infusions described a range of adverse effects on cardiac rhythm [17-20]. Though our patient developed peaked T-waves, no arrhythmias or dysrhythmias followed as a result of hyperkalemia. Therapy of acute hyperkalemia, regardless of its cause, is first of all aimed at its untoward electrophysiologic effects on the myocardium. The major available modes of hyperkalemia treatment are to antagonize the effect of K on excitable cell membranes and to redistribute extracellular K into cells. In this case moderate hyperventilation was started and one gram of Calcium Chloride, 10 units of regular insulin and 25 ml of 50% Dextrose were administered. Calcium directly antagonizes the myocardial effects of hyperkalemia by reducing the threshold potential of cardiac myocytes, thereby restoring the normal gradient with the resting membrane potential that is distorted by hyperkalemia. The onset of action is usually with 2-3 minutes and the duration of action is usually 30 to 60 minutes. . Moderate hyperventilation and intravenous Insulin reliably shifts potassium ion into the cells. An intravenous dose of ten units of regular insulin given as a bolus along with an intravenous bolus of dextrose (25 g as a 50% solution) effectively lowers potassium. The onset of action is within 15 minutes and the effect is at maximum between 30 and 60 minutes after a single dose. Combined moderate hyperventilation, insulin and calcium therapy usually reliably lowers serum potassium concentration with 45 to 60 minutes [21]. It was postulated in literature that higher doses of mannitol are more likely to cause hyperkalemia. Apparently lower doses, such as 0.7 g/kg as in this case, could also result in hyperkalemia. Rapid or repeated administration of mannitol is also known to increase likelihood of hyperkalemia development [16]. In our case Mannitol was administered over 30 minutes and no repeated doses were used.

The risk factors for hyperkalemia after Mannitol administration have not yet been determined; literature and case reports on this topic remain to be scarce. Serum electrolyte levels, arterial blood gas analysis and vigilant electrocardiogram monitoring need to be carried out when using mannitol intraoperatively. This is especially important in an emergency situation such as intracranial hemorrhage, when mannitol infusion is done rapidly or when repeated doses of mannitol are given.

Conflict of Interest

Full consent was obtained from the patient. There was no conflict of interest whatsoever involved. All authors are contributors.

Events	Na mmol/ml	K mmol/ml	pH	PaO_2 mm Hg	$PaCO_2$ mm Hg	Glucose mg/dL	iCa mg/dL	Hct %	Urine mL	IVF (total) mL
Initial (9:08 am)	135	4.2	7.43	434	46	107	1.11	42	300	800
After mannitol (11:00 am)	126	7.2	7.42	307	42	126	1.0	41	550	1600
After Dextrose, Insulin and Calcium given (11:57 am)	133	4.2	7.44	319	37	138	1.46	39	750	2200
Follow up (12:30 pm)	135	4.1	7.47	304	36	57	1.48	40	770	2800

Table 1: Reports of complications.

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