Acute Hyperkalemia and Hyponatremia Following Intraoperative Mannitol Administration

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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. The risk factors for hyperkalemia after Mannitol administration have not been fully determined; literature and case reports on this topic are scant. 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.

Case

37 year old male with no previous medical, surgical or significant social history presented for scheduled craniotomy for intracranial hemorrhage. Intraoperative mannitol administration was not considered. Patient had no prior allergy. 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 100/70 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 Remiullentyn infusion were well maintained. Shortly after induction of anesthesia was induced a radial artery catheter was placed in the right wrist. Arterial Blood Gas (ABG) sample was sent for analysis at 09:48 am and all were within normal limits (Table 1). 1 mg of intravenous Dexamethasone were administered and slow intravenous infusion of 1 g of Phosphonate was used shortly prior to the start of surgery. The surgery then began at 09:32 am. At 09:45 am surgeon requested to give patient 50 ml of Mannitol (0.7 g/kg). Mannitol was intravenously infused in a period of 45 minutes. Infusion was complete by 10:30 am. All vital signs remained stable. At 10:45 peak T-waves were noted. ABG sample was immediately sent and revealed potassium level of 7.3 mmol/l and sodium level of 126 mmol/l. Another ABG sample revealed the same deranged electrolyte levels. One gram of Calcium Chloride, 10 units of regular insulin and 25 ml of D5W were administered and moderate hyperventilation was started. No dysrhythmias followed. Next ABG sample was at 11:57 am and showed potassium level of 4.2 mmol/l. EKG data shortly returned to the baseline. Adequate urine output and balanced intravascular fluid administration were maintained throughout the case. (Table) Follow up ABG sample 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, diabetic ketoacidosis and hyperkalemia [3]. Mannitol leads to increased serum osmolality, osmotic movement of water out of cells and hyponatremia secondary to dilution. The development of hyponatremia is usually transient and sometime resolves in a short period of time with adequate hydration and diuresis [3]. In this case hyponatremia of 126 mmol/l shortly resolved with 600 ml of intravenous fluid administration and subsequently ensued 200 ml diuresis. Sometimes Mannitol causes hypokalemia, hypochloremic alkalosis. This alkalosis usually is associated with volume contraction and diuresis. This alkalosis and can be lessened if normovolemic is maintained [4].

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 evaluated. Making diagnosis by exclusion is not uncommon in anesthesiology practice [2]. Data in this case suggests that potassium movement across cellular membrane into extracellular fluid was most probable cause of hyperkalemia. Interestingly enough, mannitol is ever 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 hyperkalemia sequel most of the time [4]. When mannitol is used to decrease intracranial pressure the dose is much higher (usually 0.5-1 gram per kilogram) [4]. It was previously described that in Mannitol in high doeses can cause significant hyperkalemia and low doeses 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 hypokalemia, 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 [4]. Possible mechanisms to explain increase in plasma potassium concentration after mannitol administration are: first the rise in cell potassium concentration induced by water loss passives potassium exit through potassium channels in the cell membrane and second is the frictional forces between solvent (water) and solute can result in potassium being carried out through the water pores in the cell membrane [4]. 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 transmembrane potassium movement.

Events

<table>
<thead>
<tr>
<th></th>
<th>Na (mmol/ml)</th>
<th>K (mmol/ml)</th>
<th>PaO2 (mmHg)</th>
<th>PaCO2 (mmHg)</th>
<th>Glu (mg/dl)</th>
<th>Ca (mg/dl)</th>
<th>Hct</th>
<th>Urine (mL)</th>
<th>IVF (total mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial (9:08am)</td>
<td>135</td>
<td>4.2</td>
<td>7.43</td>
<td>434</td>
<td>46</td>
<td>107</td>
<td>1.11</td>
<td>42</td>
<td>300</td>
</tr>
<tr>
<td>After mannitol (11:00am)</td>
<td>126</td>
<td>7.2</td>
<td>7.42</td>
<td>307</td>
<td>42</td>
<td>126</td>
<td>1.0</td>
<td>41</td>
<td>550</td>
</tr>
<tr>
<td>After Dextrase, insulin and Calcium given (11:57am)</td>
<td>135</td>
<td>4.2</td>
<td>7.44</td>
<td>319</td>
<td>37</td>
<td>138</td>
<td>1.46</td>
<td>39</td>
<td>750</td>
</tr>
<tr>
<td>Follow up (12:30pm)</td>
<td>135</td>
<td>4.1</td>
<td>7.47</td>
<td>304</td>
<td>36</td>
<td>57</td>
<td>1.48</td>
<td>40</td>
<td>770</td>
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References