Title: Hyperkalemia in the preoperative period: assessment, management, and creating a framework for decision making.

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Goals:
1) With stem cases, review assessment and contrast decision making.
2) Identify decision making branch points, including whole body K load, causes for the hyperkalemia, expected changes from the surgery and anesthesia, and others that the group may identify.
3) With input of the PBLD group, cooperatively build a ‘difficult electrolyte algorithm’ for assessment and pre-operative planning of patients with hyperkalemia.
4) Much is written about emergent management of the patient with hyperkalemia in the OR, for which the SPA and other organizations have published guidance. This is not our goal for discussion.

Two vignettes are provided. The goal is to use them collectively as cases in point, while we draw upon what is known about hyperkalemia, to build, if that is possible, an algorithmic approach to management of the preoperatively hyperkalemic patient.

Case 1:
A two month old has 3 days of projectile vomiting, weight loss, and lethargy. Pyloric stenosis is suspected and saline bolus of 20 ml/kg was administered in the ED last night, with a repeat of 10 ml/kg before transfer to the inpatient floor. An ultrasound of the abdomen was obtained confirming the diagnosis of pyloric hypertrophy. A colleague visited the patient and family last night, noting normal labs, good skin turgor, capillary refill <2 seconds, and flat but not sunken fontanelle. Family, birth, and neonatal history is unremarkable, and this child has had only mild diarrhea with emesis approximately two weeks prior. The child was kept NPO overnight, with no further emesis, and maintenance infusion of NS was provided. Labs are shown:

<table>
<thead>
<tr>
<th></th>
<th>Last night</th>
<th>This morning</th>
</tr>
</thead>
<tbody>
<tr>
<td>potassium</td>
<td>140</td>
<td>141</td>
</tr>
<tr>
<td>sodium</td>
<td>102</td>
<td>112</td>
</tr>
<tr>
<td>chloride</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>pH</td>
<td>6.2</td>
<td>5.9</td>
</tr>
<tr>
<td>bicarbonate</td>
<td>28</td>
<td>18</td>
</tr>
<tr>
<td>anion gap</td>
<td>0.4</td>
<td>0.3</td>
</tr>
</tbody>
</table>

Questions:
Is this typical for pyloric stenosis?
What are the expected effects of saline resuscitation?
What is the differential diagnosis and pathophysiology for hyperkalemia in this infant?
Can we stratify the risk of taking this patient to the OR with an elevated potassium?
Assuming a confirmed case of pyloric stenosis, do we proceed with surgery, or provide additional medical management?

Case 2:
A 5 year old with a history hemolytic uremic syndrome and subsequent chronic kidney disease requiring peritoneal dialysis is scheduled for replacement of a non-functioning peritoneal
catheter, and has not been dialyzed for 2 days. Her preoperative potassium level is 6.6. A preoperative ECG shows no abnormalities. She otherwise has no symptoms.

Questions:
Does the cause for hyperkalemia differ from Case 1, and what is the likely whole body potassium load?
Does a normal ECG provide the data we need?
Does the likely chronicity of the hyperkalemia matter?
Surgery facilitates potassium correction in this case; does this make proceeding more reasonable?

Discussion:
These cases highlight common situations encountered in the preoperative setting. The differential diagnosis for hyperkalemia includes possible undiagnosed genetic, metabolic, iatrogenic, or factitious causes of hyperkalemia as well as causes from underlying chronic conditions. Decision making regarding hyperkalemia sometimes may focus on the lab number to the exclusion of the broader consideration of cause and impact of associated conditions.

Acute Management of the Hyperkalemic Emergency:
The Critical Event Checklist (SPA web page) provides guidance on management once a critical event unfolds. Usually this pathway is activated after recognizing an ECG pattern typical for hyperkalemia, or through an arrest precipitated by hyperkalemia.

Preoperative considerations, before the situation is dire:
Potassium homeostasis is a complex balance of multiple electrolytes, acid – base status and buffer systems, whole body amounts of each ion, and balance of intracellular to extracellular concentrations. Typically lab panels allow only a small piece of the whole ion picture to be viewed. Extracellular potassium constitutes approximately 1-2% of total body potassium, with the intracellular/extracellular proportioning maintained by active energy dependent cell membrane based ion pumps. Passive flows of potassium and the other strong ions through gated channels and membrane leak then determine membrane resting potentials and action potentials.

ECG is often referenced as a tool to evaluate the physiologic effect of a particular potassium level, but it is uncertain whether a normal ECG may reliably be interpreted as indication of a normal electrophysiologic state. Conversely, it is clear that ECG is a poor predictor for potassium level, particularly in chronic renal failure, underscoring the physiologic complexity of the whole ion state. While ECG sensitivity may be low, reports exist in the literature demonstrate that elevated T wave does identify otherwise unsuspected hyperkalemia, with the double counting of heart rate being the first indicator to the clinician.

Treatment of hyperkalemia includes a mix of management, which have differing onsets and mechanisms depending on how critical the potassium level is, and the underlying cause. Typical interventions include:
Membrane stabilization, with calcium chloride or calcium gluconate, or hypertonic saline.

Potassium shift from extracellular to intracellular, with insulin/glucose, or beta agonist such as albuterol, or pH change with bicarbonate.

Enhanced excretion, if total body overload with potassium, using furosemide, sodium bicarbonate, ion exchange resin (e.g. Kayexalate), or dialysis.

References:


