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Getting the K^+ Down

We have seen several residents with hyperkalemia lately, so I thought the topic was worthy of a GeriJournal article. The two main conditions predisposing our residents to hyperkalemia are heart failure (HF) and chronic kidney disease (CKD). Some drugs used to treat these conditions; ACEI (e.g. ramipril, perindopril) and ARBs (candesartan, irbesartan, etc.) increase K^+ retention by the kidneys, further elevating serum K^+ levels.

ACEI and ARBs reduce or block angiotensin II (AG2), a blood component which constricts vessels, thereby increasing BP. A failing heart delivers reduced amounts of blood to the kidneys. The kidneys try to activate the circulation by releasing renin, which in turn increases AG2. Unfortunately, this is exactly what the weakened heart doesn't need (increased BP due to renin) and is why ACEI and ARBs are so helpful in HF.

AG2 further aggravates the situation by "telling" the adrenal glands to release aldosterone, a hormone which increases Na retention by the kidneys. Na pulls fluid into the bloodstream, and that is bad for the overwhelmed, failing heart. Since ACEI and ARBs block the formation or action of AG2, they reverse aldosterone linked Na retention. Aldosterone blockers, like spironolactone, also help eliminate Na and are used for HF treatment. While all these drugs reduce Na, the kidneys retain K^+ in exchange, with hyperkalemia a potential consequence.

Damaged kidneys may be a greater concern with regards to K^+ levels. We all know that individuals with CKD filter blood poorly, so waste products like creatinine accumulate. Another problem occurs when the filtering apparatus of the kidney, the glomerulus, is leaky. In such cases, large essential protein molecules can be lost in the urine. This loss is quantified by the ACR (urinary albumin/creatinine ratio). Ideally, the ratio would be low (below 3 is normal), indicating the kidneys are doing a good job of both keeping albumin, and getting rid of creatinine. Repeat levels between 3 and 30 signify moderate damage and greater than 30 show severe damage.

ACEI or ARB treatment reduces filtering pressure in the damaged glomeruli, so they slow progression when the kidneys are "spilling" protein (elevated ACR).

The challenge comes when the K^+ level becomes elevated. We are reluctant to stop or reduce the ACEI/ARB, because we don't want to leave the kidneys unprotected. Many guidelines deal with this issue, but in general they recommend that, ACEI/ARB not be started if K^+ is above 5 (some nephrologists will start these drugs with a K^+ up to 5.5). If K^+ is between 5.5 and 6 the drug should be stopped or dose reduced, and above 6 it should be stopped.

What other measures can we take to reduce K^+? Potassium supplements should be stopped and fruit/fruit juices limited or avoided. Lasix® can be added to increase urinary loss. Hyperglycemia moves K^+ into the bloodstream, so blood sugar must be controlled. Kayexalate is given, if necessary. It binds to K^+ in the intestinal tract.

Kayexalate should not be given within 3 hours of other meds, as it may bind to them. It is administered as 15 Gm (20ml in med cup) or 30 Gm, with a maximum daily dose of 60 Gm. It is mixed with roughly 50ml of water (never juice) per every 15 Gm of powder. Constipated residents must not take it, as it can cause impaction. It may increase Na levels and decrease Mg and Ca, so these should be watched. It can take hours or days to work, so critical K^+ levels (> 7.5) require rapid reduction with i.v. glucose and insulin in hospital.

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