

BRASH Syndrome

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Bradycardia, Renal Failure, AV blocker, Shock, Hyperkalemia

Underrecognized as a distinct clinical entity

High morbidity and mortality

Potential for rapid improvement

BRASH syndrome definition:

- Syndrome caused by a vicious cycle of *bradycardia* and *shock*, set up by a combination of *AV nodal blockade*, *hyperkalemia* and *renal failure*

Pathophysiology:

Renal failure, usually caused by a trigger that worsens kidney function. This leads to hyperkalemia and accumulation of AV nodal blockers that results in bradycardia, worsening renal hypoperfusion and shock. This further worsens renal failure and the cycle perpetuates. The key is that the hyperkalemia *synergizes* with the AV nodal blockade.

Evidence:

Most robust clinical review is by PulmCrit FOAM source (Dr. Josh Farkas) completed in 2016. This clinical review has been important in advancing BRASH syndrome as a distinct clinical entity. Overall, there is low quality evidence (mostly case reports) that make up small literature reviews.

| BRASH vs. HyperK | BRASH vs. AV nodal blocker OD |
|--|--|
| <ul style="list-style-type: none">- Concurrent presence of AV nodal blockers and renal failure- Degree of hyperkalemia is usually mild relative to bradycardia- ECG lacks <u>other</u> signs of hyperkalemia | <ul style="list-style-type: none">- Presence of hyperkalemia- Lacking history of large AV nodal blocker ingestion |

Epidemiology:

- Elderly (mean 72 yr)
- Female predominance (78%)
- Renal dysfunction (baseline and acute)
- Presence of AV nodal blockade

Clinical presentation:

Degree of bradycardia – HR 28-56

Type of bradycardia – Variable (junctional to 3rd deg), ECG AV nodal blockade in 78% of cases

Degree of hyperK – K \geq 6.3 in 55% of cases

Triggers:

Most common: Hypovolemia (eg: gastro, dehydration), up-titration of antihypertensives
Other: Fever, sepsis, tumor lysis syndrome, any other cause of AKI (meds, post-renal etc)

Management:

Spectrum of illness severity

- Most mild cases will respond to fluids +/- IV calcium
- More severe cases: three management priorities ----- >

HyperK

Stabilize → IV Calcium

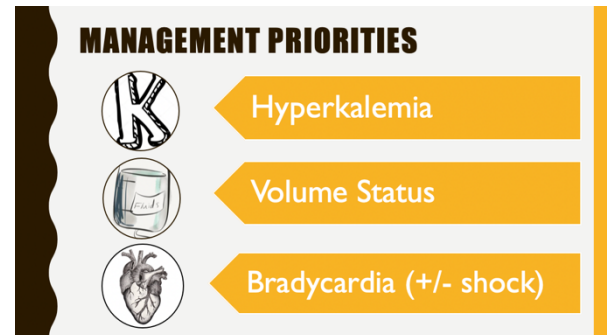
- Calcium gluconate 3g IV
- Calcium chloride 1g IV

Shift

- Insulin 5-10 units IV with 1-2 amps D50 IV
- Ventolin 10-20mg neb over 10 minutes
- NaHCO3 1-2 amp(s) IV

Eliminate

- Ensure adequate volume status (hydrate vs. lasix), if refractory then dialysis



Volume Status

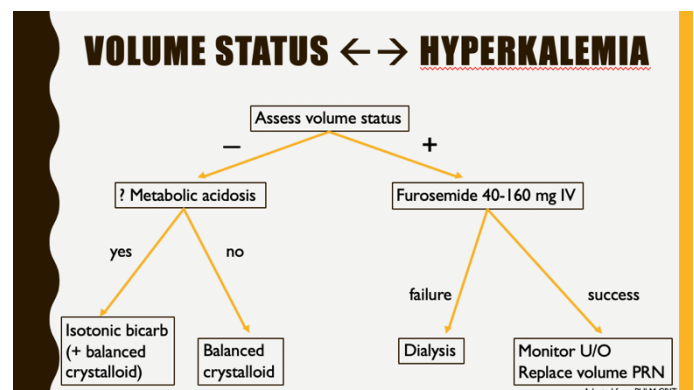
Volume assessment is key – spectrum of profound hypovolemia to anuric renal failure

- Concurrent hyperkalemia and (typically) lactic metabolic acidosis
- Goal is to address hypovolemia early to restore renal perfusion and halt BRASH cycle
- Fluid of choice = balanced crystalloid
 - Avoid NS due to creating further acidosis (hyperchloremic), hyperkalemia and worse renal outcomes

Approach:

Algorithm Adapted from PULM CRIT

- Consider isotonic bicarbonate (1L D5W with 3 amps of bicarb) @ <250cc's/hr and monitor Na to help normalize bicarb in severe metabolic acidosis, then balanced crystalloid
- Balance crystalloid in all others requiring fluids



Bradycardia/Shock:

EARLY initiation of pressors recommended as BRASH induced bradycardic shock generally responds well

| Meds | Rationale | Dosing |
|---|---|-----------------------------|
| Epinephrine (1 st line) | Use for sicker patients Shifts K into cells (more) | 2-10 mcg/min IV infusion |
| Isoproterenol (2 nd line) | Use for less sick patients Shifts K into cells (less) Likely safer for longer peripherally | 2-10 mcg/min IV infusion |

Consider more calcium above what is given to stabilize cardiac membrane if refractory to pressors

Treatments (typically) ineffective:

Atropine

Transcutaneous pacing

Transvenous pacing

High dose insulin and glucagon for BB or CCB overdose