**Introduction**

- BRASH syndrome: first coined by Dr. J. Farkas in 2016
- Bradycardia
- Renal failure
- AV blocking agent
- Shock
- Hyperkalemia

The aim is to review recommended therapeutic intervention.

**Case #1**

- 70-year-old male with history of CKD and CAD presented with AMS and AHRF.
- Medications: Carvedilol and Entresto
- V/S: HR:43, BP: 85/55, RR:25, SpO2: 80%
- PE: Drowsy, nonverbal, dry mucosal membranes, edematous, rales and crackles in lungs.
- K:6,5 mEq/L, Cr: 4,17 mg/dl (baseline of 1,7)

**Intervention:**
- Intubated and admitted to MICU
- Epinephrin gtt, hyperK treatment with Ca, bicarbonate, loop diuretics.
- Initiation of CRRT and HD (as hyperK was not controlled well)

**Case #2**

- 64-year-old male with ESRD (on HD), PAF presented with episodic confusion and generalized weakness.
- Medications: Diltiazem and metoprolol, (recently increased dose of both), losartan.
- V/S: HR:25, BP: 75/-, RR:15
- PE: alert, AAOX3, no neuro deficit
- K: 7,1 mEq/L, EKG: Afib, with tall T wave

**Intervention:**
- Atropine, pace pads, Epinephrin and dopamine gtt, bicarb, HyperK treatment, calcium, glucagon, urgent HD.

**Summary**

- Should not be managed as an isolated electrolyte abnormality, or pure bradycardia syndrome.
- The syndrome may be irreversible and fatal beyond a certain point, if not diagnosed, and managed well.
- In both of our cases, only after HD the vicious cycle was disrupted, and vasopressors could be stopped.
- Hypotension is mainly because of bradycardia
- Chronotropics like epinephrine or isoproterenol
- Organ hypoperfusion and worsening of renal failure may occur despite normal BP.

**Reference**