BRASH Syndrome

Section 1: Case Summary

Scenario Title:	BRASH Syndrome
Keywords:	BRASH Syndrome,
Brief Description of Case:	70yo male brought to the ED by EHS from his apartment with a history of several days of nausea/diarrhea/emesis. He called EHS today complaining of dizziness.

Goals and Objectives	
Educational Goal:	Approach to management of BRASH Syndrome
Objectives: (Medical and CRM)	CRM: Effectively lead team members through complex critical scenario. Medical: 1) Early recognition of BRASH Syndrome and its multifactorial nature 2) Check potassium early in bradycardia and have a low threshold for giving IV calcium even if the ECG doesn't show obvious hyperkalemia
EPAs Assessed:	F1 Initiating and assisting in resuscitation of critically ill patients

Learners, Setting and Personnel				
Target Learners:	☐ Junior Learners	☒ Senior Learners	☒ Staff	
	☐ Physicians	☐ Nurses	☐ RTs	☒ Inter-professional
	☐ Other Learners:			
Location:	☒ Sim Lab	☒ In Situ	☐ Other:	
Recommended Number of Facilitators:	Instructors: 1			
	Confederates:			
	Sim Techs: 1			

Scenario Development	
Date of Development:	March 26th 2022

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Scenario Developer(s):	Dr. Jeanne MacLeod (SPH Attending ED Physician) and Dr. Matei Stoian (CCFP-EM Resident)
Affiliations/Institutions(s):	UBC - SPH
Contact E-mail:	matei.stoian@medportal.ca jmacleod604@gmail.com
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Revised By:	Matei Stoian
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Section 2A: Initial Patient Information

A. Patient Chart					
Patient Name: John Doe			Age:70 years old	Gender: Male	Weight: 70kg
Presenting complaint: Arrived via EHS. Complaining of dizziness.					
Temp: 37.5	HR:38	BP:86/50	RR:20	O_2Sat:90% room air	FiO_2: R/A
Cap glucose: Not checked			GCS: (E V M) - 15		
Triage note: John is a 70yo male brought to the ED by EHS from his apartment with a history of several days of nausea/diarrhea/emesis. He called EHS today complaining of dizziness. EHS notes the above vitals. They have not provided any treatment. There is one 18gg IV in the right AC.					
Allergies: No known drug allergies					
Past Medical History (should be prompted by learners) -Previous STEMI with two stents placed 5 years prior -Hypertension -Diabetes (NIDDM)			Current Medications (should be prompted by learners) Atenolol 50mg PO OD Ramipril 10mg PO OD ASA 81mg PO OD Metformin 500mg PO BID		

Section 2B: Extra Patient Information

A. Further History (to be given in phase 1 or 2)	
His partner can arrive in the ED and provide this further history. -Has been slightly confused today -No known history of dementia -Mild abdominal cramps -No missed doses of medications, pills are all blister packed and no missing medications noted -Slowly getting worse over the last 3 days	
B. Physical Exam (to be given in phase 1 or 2)	
List any pertinent positive and negative findings	
Cardio: S1 + S2, no EHS, systolic ejection murmur. JVP is down.	Neuro: PEARL 4mm bilat. Decreased attention, not fully oriented to place or time. Grossly normal otherwise
Resp: Mild crackles to bases	Head & Neck: Dry mucous membranes.

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Abdo: Soft, mildly tender diffusely, non-distended	MSK/skin: Grossly normal. Appears generally unwell, disheveled.
Other: POCUS: FAST exam shows slow heart with poor contractility, no pericardial effusion, no right heart strain. IVC difficult to see but appears to be collapsing.	

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Section 3: Technical Requirements/Room Vision

A. Patient
☒ Mannequin *(adult)*
☐ Standardized Patient
☐ Task Trainer
☐ Hybrid

B. Special Equipment Required
None

C. Required Medications
Calcium (gluconate/chloride) Insulin, Dextrose, Ventolin Epinephrine infusion, Isoproteronol infusion Atropine

D. Moulage
None

E. Monitors at Case Onset
☐ Patient on monitor with vitals displayed ☒ Patient not yet on monitor

F. Patient Reactions and Exam
The patient is slightly confused. Unsure of the date, or what hospital he is at. Unsure of what medications he takes, not able to provide a good history.

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Section 4: Confederates and Standardized Patients

Confederate and Standardized Patient Roles and Scripts	
Role	*Description of role, expected behavior, and key moments to intervene/prompt learners. Include any script required (including conveying patient information if patient is unable)*
Patient's Spouse	Provides the history as seen above

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Section 5: Scenario Progression

Scenario States, Modifiers and Triggers				
Patient State/Vitals	Patient Status	Learner Actions, Modifiers & Triggers to Move to Next State		Facilitator Notes
1. Baseline State Rhythm: sinus HR: 38 BP: 86/50 RR: 20 O_2SAT: 90% r/a T: 37.5°C GCS: 15	70yo man brought by EHS from his apartment with several days of nausea/diarrhea/emesis. Called EMS complaining of dizziness	Expected Learner Actions -place monitors -two large bore IVs -Stat labs -Start fluid bolus -SAMPLE history directed at ruling out ACS -STAT ECG -Check bedside glucose -Should put pacer pads on the patient	Modifiers -If placed on oxygen 2-4L of oxygen, saturation up to 97% -If atropine given, no change in heart rate should occur Triggers -Once on monitors, with labs called for, and fluid bolus running, can give the learners the ECG -Once ECG given and interpreted can move to phase number 2.	-At this point atropine should be drawn up, but not necessary to give before ECG -If after two ineffective doses of atropine learners should look to set-up an epinephrine or isoproterenol infusion (or transcutaneous pacing)

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2. Acute deterioration Rhythm: sinus bradycardia HR: 32 BP: 72/50 RR: 24 O_2SAT: 97% on 2-4L T: 37.5°C GCS: 14	-Physical exam findings as in section 2B -History as in section 2B	Expected Learner Actions -Try atropine (but is unsuccessful) -Start chronotrope infusion -Can consider pacer pads -Can consider further fluid bolus -Can consider treatment for hyperkalemia on spec/AV nodal blocker toxicity on spec.	Modifiers -If pacer pads used without any sedation, patient reacts with pain -If only 1L of fluid given, IVC still collapsable, patient still appears dry - Triggers -If only atropine used, after 2 minutes the patient deteriorates to phase 3 -If pacer pads used, after 5 minutes they lose capture and if no other interventions have been taken the patient deteriorates to phase 3 -If started on an epinephrine or isoproteronol infusion, or given IV calcium can give lab work and progress to phase 4 -If phenylephrine given patient's HR should drop further, if not discontinued they should then deteriorate to phase 3	-The ECG can be interpreted as a rhythm around 60 bpm with severe QRS prolongation and prominent T waves. This might be sinus rhythm with very small P waves or atrial fibrillation with a relatively regular rate. These changes are typical of **severe hyperkalemia**. -Would be very reasonable for IV calcium to be given without the potassium result as the patient has a history of renally excreted beta-blocker (atenolol), bradycardia, and recent volume loss (diarrhea and emesis) -Pacer pads are an effective temporizing measure, but they are not the definitive management of BRASH. Transvenous pacing is more definitive but it is rarely necessary in BRASH and treating the root issues + temporizing with pressers is the preferred route of management
3. PEA Arrest Rhythm: junctional HR: 22 BP: 48/20 (pulseless) RR: n/a O_2SAT: 97% on NRB T: 36.5°C GCS: 3	PEA Arrest	Expected Learner Actions -No shocks given -2 rounds of 1mg epinephrine -High quality CPR with minimal interruptions -Recognizing this as a PEA arrest algorithm -At this point ideally calcium chloride is given, and more fluids	Modifiers -If learners prep for intubation they can be advised that the patient is maintaining good saturations and is easy to bag Triggers -Once two rounds of epinephrine given proceed to phase 4	-Bradycardic PEA arrest secondary to BRASH syndrome

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4. Stablization	The patient is less confused and feels somewhat better.	Expected Learner Actions	Modifiers	-Once the labs have returned on the patient the learners should identify that hyperkalemia is a contributing factor -The acidosis in this patient should be noted (wide anion gap secondary to uremia and lactate) -The mild troponin elevation in this case is meant to represent demand ischemia -The elevated white count is secondary to the gastroenteritis the patient initially presented with
Rhythm: junctional HR: 50 BP: 85/60 RR: 16 O_2SAT: 97% on 2-4L T: 37.5°C GCS: 15		-Calcium should Foley placement -Should shift potassium (salbutamol/insulin/dextrose) -Admission to monitored bed (cardiology/HAU/etc.) -Continued fluid resuscitation -Can mention options of glucagon, high dose insulin or fat emulsion therapy for beta-blocker toxicity should patient status worsen -Can consider furosemide -Should stop pacer pads after giving fluid resuscitation/pressors/potassium shift, myocardial stabilization with calcium)	-If admitting the patient should ask for details if not already addressed (e.g. admitting meds, follow-up diagnostic studies, admitting team, safety measures) -If at this point the learners still haven't asked for a medication list they should be prompted by the spouse with the medication list Triggers -If calcium not given promptly in response to the elevated potassium, should proceed to phase 3 -If calcium given, potassium shifted, ongoing fluid resuscitation, should prompt learner for their plan for admission for this patient	

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Appendix A: Laboratory Results

<u>CBC</u> WBC 15.4 Hgb 131 Plt 240 <u>Lytes</u> Na 134 **K 7.5** Cl 104 **HCO_3 10** **AG 20** Urea 18 Cr 210 Glucose 5.1 <u>Extended Lytes</u> Ca 2.2 Mg 0.80 PO_4 1.33 Albumin 40 TSH 2.1 <u>VBG</u> **pH 7.10** pCO_2 28 pO_2 88 **HCO_3 10** **Lactate 5**	<u>Cardiac/Coags</u> Trop 30 D-dimer 450 INR 1.2 aPTT 26 <u>Biliary</u> AST 21 ALT 42 GGT 46 ALP 89 Bili 9 Lipase 97 <u>Tox</u> EtOH <2 ASA 0 Tylenol 0 Dig level 0 Osmols N/A <u>Other</u> B-HCG N/A

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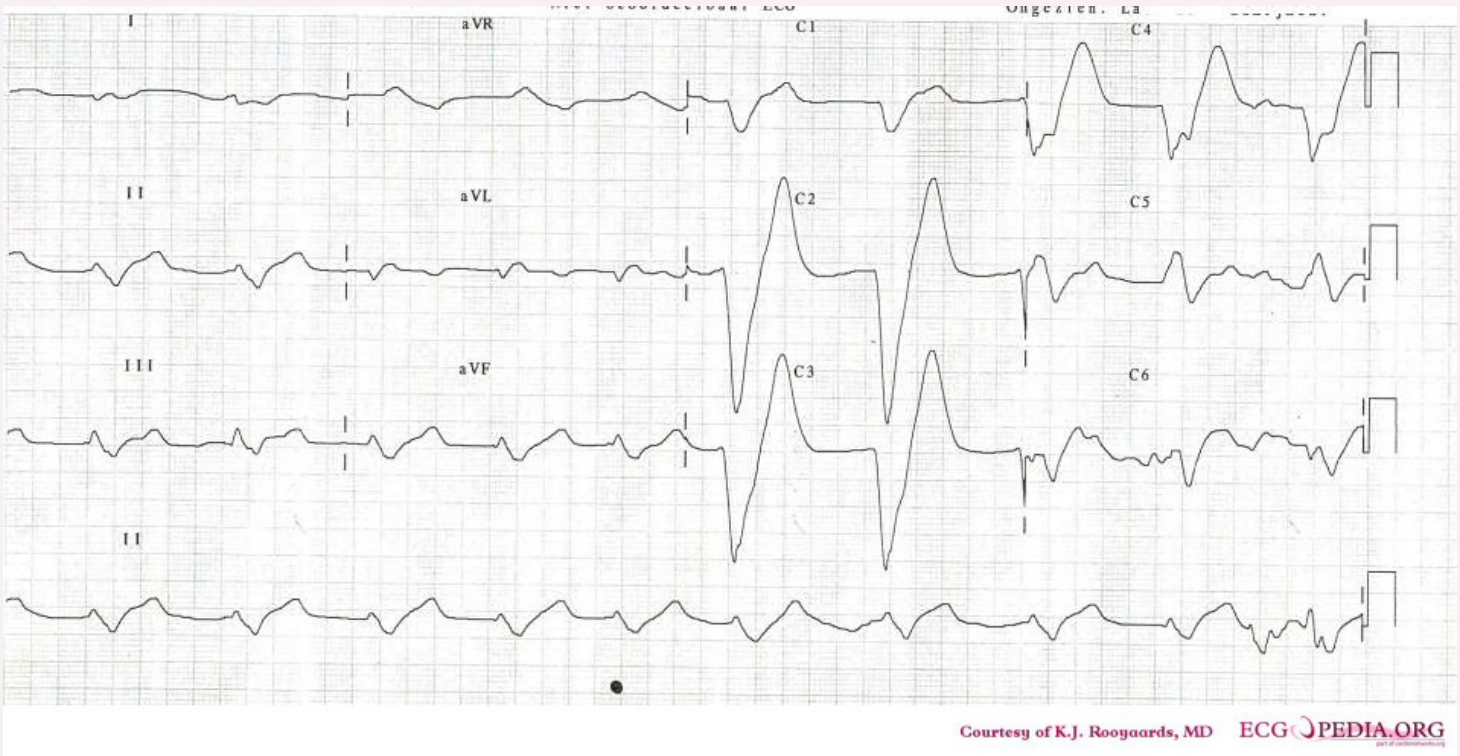
Appendix B: ECGs, X-rays, Ultrasounds and Pictures



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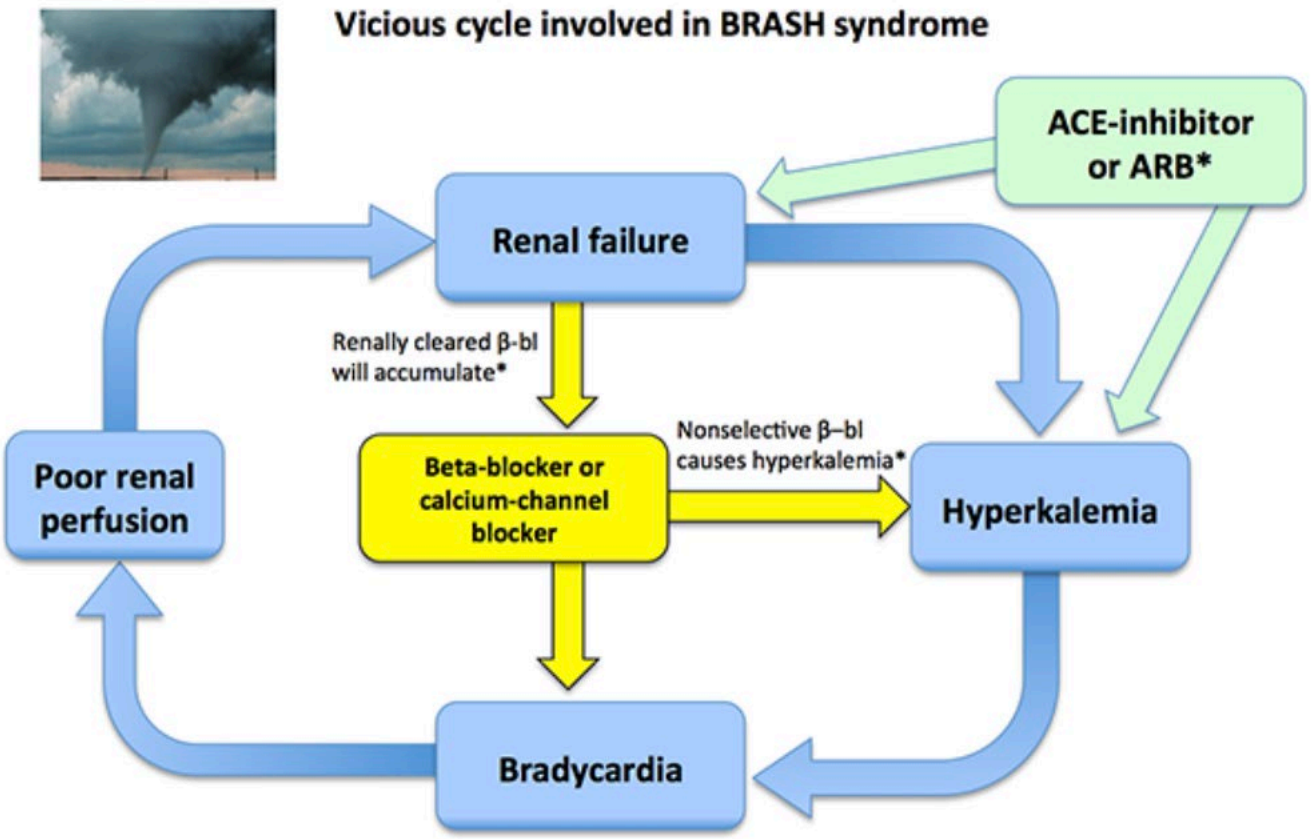
Appendix C: Facilitator Cheat Sheet & Debriefing Tips



* Indicates components which are not required, but may contribute in some cases

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Include key errors to watch for and common challenges with the case. List issues expected to be part of the debriefing discussion. Supplemental information regarding any relevant pathophysiology, guidelines, or management information that may be reviewed during debriefing should be provided for facilitators to have as a reference.

1. The patient in this scenario has BRASH syndrome. This syndrome is a constellation of abnormalities/issues that often co-occur resulting in significant hemodynamic instability. BRASH stands for Bradycardia, Renal Failure, AV Nodal Blockade, Shock, and Hyperkalemia
2. In this case, the patient had an initial infectious prodrome (gastroenteritis) which set off the course of events in which they had hypovolemia, resulting in a pre-renal AKI. This led to accumulation of their atenolol (renal excretion) as well as worsening hyperkalemia (further worsened by their ACEi). The hyperkalemia than further worsened the bradycardia. Finally, the hypovolemia and the bradycardia together led to shock, which further worsened renal perfusion leading to worsening kidney function, further beta-blocker toxicity, and further bradycardia (see previous page for graphic representation of this vicious circle)
3. Treatment of BRASH syndrome is aimed at holistic management of each of the specific concerns. The hyperkalemia is treated with IV calcium for myocardial stabilization (in this case perhaps even prior to classic ECG changes) and shifting (with insulin/salbutamol). The bradycardia and shock are temporized with pressors, fluids and TCP. The AV nodal blockade is addressed by holding the offending agent, and treating any potential toxicity (IV calcium first line, and if severe high dose insulin, fat emulsion, and glucagon for beta-blockers)
4. Because most patients with BRASH syndrome respond to resuscitation and have reversible causes, there is typically no need to treat with hemodialysis or transvenous pacing
5. For fluid resuscitation balanced solution is recommended as normal saline can worsen acidosis
6. Although it isn't the most emergent aspect of therapy, these patients will eventually need to be diuresed to excrete potassium. If dry they should be adequately fluid resuscitated prior.
7. Atropine isn't an incorrect choice and it IS part of the ACLS pathway, but it typically won't help treat the underlying problems in BRASH syndrome and there are better ways to improve the bradycardia. The standard ACLS bradycardia pathway fails to use calcium which is why this isn't an adequate algorithm in these patients
8. In terms of pressers, the ideal choices or either isoproterenol or epinephrine for their beta-agonism/chronotropy. Phenylephrine would be a bad option for this shock as it is pure alpha and could worsen bradycardia. Dobutamine is also an option.
9. The primary pitfall in the management of BRASH is focussing on just one issue (e.g. placing a transvenous pacemaker but not fluid resuscitating or treating the hyperkalemia). It is important to recognize the synergistic process for the treatment to be effective in these patients.
10. Although not in the mnemonic, acidosis is common in the patients (shock leading to lactic acidosis, AKI leading to uremic acidosis), and this in turn can worsen hyperkalemia through shifting potassium extracellularly.

References

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3. EM Cases Episode 154 - 4 Step Approach to Bradycardia: https://emergencymedicinecases.com/approach-bradycardia-bradydysrhythmias/

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