Brash Syndrome, Can You See the Cycle?

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Introduction: Bradycardia, Renal failure, Atrioventricular(AV)-nodal blockers, Shock and Hyperkalemia (BRASH) syndrome. Bradycardia is commonly caused by myocardial infarctions, arrhythmias, increased intracranial pressure, hyperkalemia or medication overdoses. BRASH syndrome describes a series of events, when combined with each other lead to a catastrophic cycle. Failure to recognize BRASH syndrome can lead to treatment failure and death. Case: An 80-year-old woman presented to the emergency room after being found down in her hallway confused. In the ambulance, she was found to be bradycardic to 33 with a low blood pressure. Known to our hospital system, she has a history of diabetes mellitus, atrial fibrillation on apixaban and prior non-hemorrhagic strokes. CT was obtained of her head, chest, and abdomen/pelvis which was negative for intracranial hemorrhage, pulmonary embolism, or intra abdominal pathologies. ACLS guided bradycardia with a pulse algorithm treatment was started. Transcutaneous pacing was started, shock developed and vasopressors were initiated. No infection or myocardial infarction were found. Labs demonstrated an anion gap metabolic acidosis with a potassium of 5.3 and creatinine of 1.9. Three pulseless electrical activity codes subsequently occurred with return of spontaneous circulation and she did not survive the fourth code. Discussion: BRASH syndrome’s pathophysiology is thought to act in a synergist way. It is believed to begin with an insult that causes hypo-perfusion, such as diarrhea. AV-nodal blocking medications prevent reflex tachycardia that can be needed to maintain adequate organ perfusion. Decreased renal perfusion leads to renal failure and hyperkalemia. AV-nodal blockers and hyperkalemia act synergisticly to cause bradycardia, even at normal potassium levels. Cardiogenic shock ultimately develops, which leads to worsening renal failure exacerbating the problem. Thus the cycle continues downward. This bradycardia is typically refractory to electrical conduction, and requires aggressive medical management. EKG degrangements expected (peaked T waves, or QRS widening) to cause bradycardia from hyperkalemia alone won’t be present. The degree of hyperkalemia is often how the diagnosis is missed. The syndrome lies between the synergistic effect of hyperkalemia and AV-nodal blockade. BRASH syndrome treatment is aggressive medical management targeted at hyperkalemia (calcium, insulin, hemodialysis) and shock reversal(volume, pressors). Conclusion: Elderly patients are more vulnerable to medication side effects. When ACLS treatment of bradycardia fails, having the knowledge of the pathophysiology and recognition of BRASH syndrome leads to alternative treatment options.

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