

BRASH: A Rare Syndrome Threatening Cardiovascular Collapse

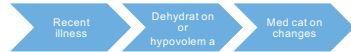
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BACKGROUND

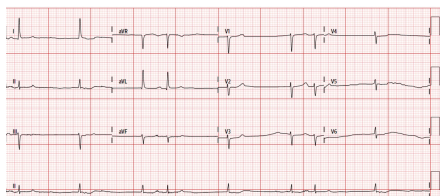
The synergistic interaction between the effects of AV nodal agents and hyperkalemia results in a rare life-threatening condition known as BRASH syndrome of which there are few cases reported. This condition is likely to become more common given the compounding comorbidities of aging populations and thus represents an under-recognized contributor to cardiovascular collapse and multi-system organ failure. This case is unique in that one of the few renally excreted beta-blockers (atenolol) was used in this patient which likely further worsened the patient's bradycardia.

Precipitants of BRASH syndrome:

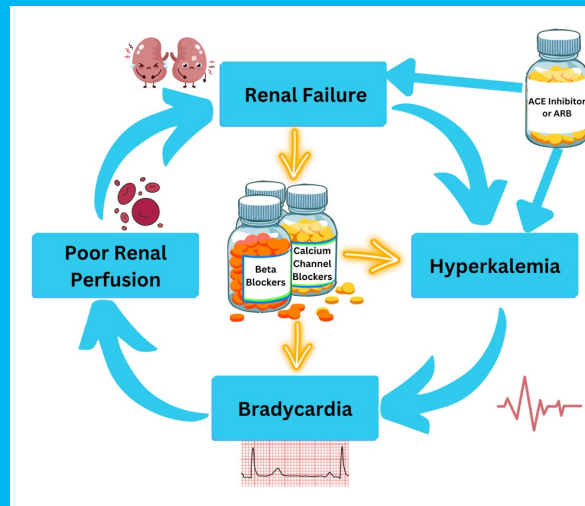


CASE PRESENTATION

A 72-year-old female with a history of lung cancer, hypertension, and stage III chronic kidney disease presented with acute, progressive weakness and dizziness in the setting of recent viral gastroenteritis. She had been taking her medications as prescribed, including atenolol for hypertension which she had been taking for years.

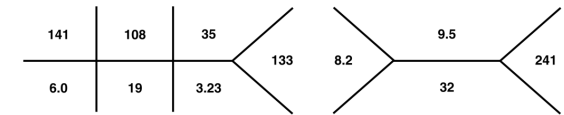


BRASH Syndrome is an under-recognized and potentially life-threatening condition which patients receiving AV-nodal blocking agents are at risk for developing without provider recognition and intervention.



Bradycardia
Renal failure
Av nodal blocking agents
Shock
Hyperkalemia

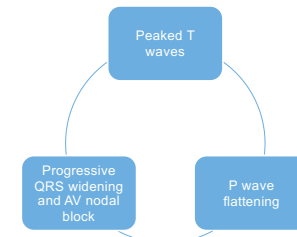
HOSPITAL COURSE



She was found to have significant acute kidney injury and hyperkalemia for which she received intravenous fluids, calcium gluconate, insulin/dextrose, and patiromer. Cardiology was consulted with plans for pacemaker placement, however the patient's bradycardia resolved with volume resuscitation and treatment of hyperkalemia in addition to holding atenolol. Her renal function improved, and atenolol therapy was discontinued at discharge as there was no cardiac indication for beta-blockade.

DISCUSSION

Typical findings of hyperkalemia:¹



BRASH syndrome does not typically demonstrate the above findings and the bradycardia is also out of proportion to the degree of hyperkalemia. Atenolol is hydrophilic and is predominantly eliminated via the kidneys; Elimination half-life of atenolol is prolonged as renal function decreases.² In this patient, acute kidney injury due to dehydration contributed to increased plasma atenolol levels. While this patient ultimately made a full recovery, BRASH syndrome goes under-recognized in many instances.

REFERENCES

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