An unusual case of BRASH syndrome precipitated by atrial fibrillation with a fast ventricular response

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Background

BRASH syndrome is a recently described clinical syndrome arising from a unique synergism between AV-blockade and hyperkalaemia, in turn producing profound bradycardia and cardiogenic shock (Fig 1)[1]. Often it is triggered by acute hypovolaemic events or medication changes[1,2]. Elderly with multiple comorbidities, particularly cardiorenal pathologies, and on AV-blockade are most at-risk[1]. BRASH syndrome is distinct as it is often refractory to traditional advance life support protocols[1,3].

Bradycardia
Renal insufficiency
AV blockade
Shock
Hyperkalaemia

Aims and objective

- Outline a unique case of BRASH syndrome precipitated by atrial fibrillation with fast ventricular response (fAF)
- Increase awareness of BRASH syndrome
- Highlight iatrogenic risk of BRASH when treating fAF

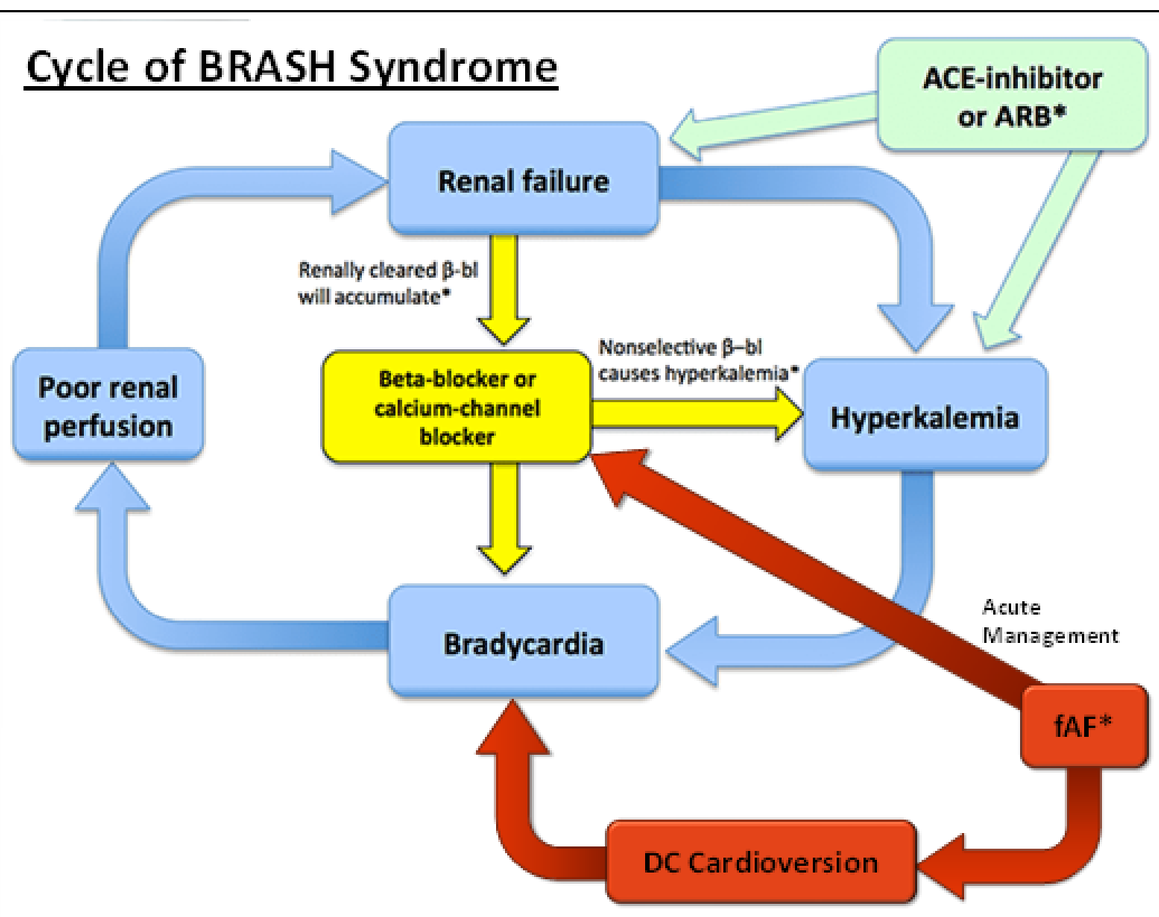

Figure 1: Adapted diagram of the cycle of BRASH syndrome (Farkas et al. 2020).

*Indicates components which are not required but may contribute to BRASH syndrome.

This figure proposes how atrial fibrillation with fast ventricular response (fAF) masks the bradycardia but may ultimately lead to worsened bradycardia via associated cardiomyopathy and management with beta-blocker.

Discussion

BRASH syndrome is an underdiagnosed and underreported syndrome. We suggest clinicians maintain a high degree of clinical suspicion in patients presenting with bradycardiac <60bpm and hyperkalaemia in at risk patients. Commonly reported presenting complaints of BRASH syndrome include falls and nausea/vomiting.

In this case hypothesise BRASH was precipitated by a combination of tachyarrhythmias-associated cardiomyopathy and iatrogenesis from high dose bisoprolol and DC cardioversion. Clinicians should consider the risk of BRASH when managing fAF, particularly with complex cardiac or renal histories.

Optimal management of BRASH syndrome deviates from traditional ALS protocol. With aggressive hyperkalaemia and reno-supportive measures, transcutaneous cardiac pacing can be avoided. In some cases medical therapy is effective but severe cases require CVVH[4,5]. Further characterisation with multi-centre case control study would help clarify presentation and management pathways

Case

Presentation

- 60 year old gentleman with complex comorbidities: hypertension, type 2 diabetes mellitus, ischaemic heart disease, previous myocardial infarction with triple vessel stenting, paroxysmal AF with ablation in 2018/20, epilepsy, obstructive sleep apnoea and hypothyroid
- No pre-existing renal insufficiency
- Key medication: bisoprolol 2.5mg and digoxin 125mcg
- Presents with shortness of breath for 1 week. ECG demonstrates fAF. Admission bloods unremarkable

Initial Management

- 10mg bisoprolol ineffective
- DC synchronised cardioversion

Rapid Deterioration Overnight

- 4h post cardioversion: vomiting, poor awareness and anuria >6h via catheter
- Bradycardic (30-40 bpm) and hypotensive (90/50 mmHg)
- Arterial blood gas shows hyperkalaemia: 7.3 mmol with no characteristic ECG changes

Acute Management

- 150 mcg/hr isoprenaline infusion restores normotension
- Slow response to insulin dextrose and calcium but K+ reduces to 5.5mmol/L

Reassessment

- AKI development: Creatinine 126umol/L (67umol/L) and urea 9 mmol/L (7 mmol/L)
- Positive fluid balance of +3440mls despite IV furosemide bolus
- Labile serum K+ and requiring 150mcg/hr of isoprenaline
- Temporary pacing considered but opted for continuous veno-venous haemofiltration (CVVH) due to severe renal failure

CVVH

- Rapid reduction in isoprenaline requirement and stopped <36h
- Patient restored to neutral cumulative fluid balance
- Electrolyte profile returned to normal

Ongoing Management

- Patient opts for permanent pacemaker on discussion with cardiology
- Episodes of fAF following procedure result in temporary increase in daily bisoprolol
- Usual bisoprolol and digoxin dose continue
- Patient remains well in the community

Conclusion

Tachyarrhythmias and its management can precipitate BRASH syndrome therefore practitioners should be aware of this potentially preventable iatrogenesis. Early recognition of BRASH is essential for initiating optimal management and preventing unnecessary interventions.

Acknowledgements

No declared conflicts of interest

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