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# BRASH Syndrome Masquerading as Septic Shock in a Patient with Pneumonia: A Case Report

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**KEYWORDS**

BRASH syndrome, Hyperkalaemia, Bradycardia, Acute kidney injury, AV nodal blockade, Haemodialysis, , PLAPS (posterolateral alveolar-pleural syndrome)

**ABSTRACT:**

Bradycardia with hypotension is a life-threatening emergency that mandates rapid stabilisation while simultaneously looking for reversible causes, particularly electrolyte disturbances and drug-related conduction suppression. Hyperkalaemia can precipitate malignant arrhythmias and cardiovascular collapse, and emergency management prioritises cardiac membrane stabilisation with intravenous calcium, intracellular potassium shifting with insulin–dextrose and inhaled β2-agonists, and definitive potassium removal when renal excretion is impaired. BRASH syndrome (Bradycardia, Renal failure, AV-nodal blockade, Shock, Hyperkalaemia) is an under-recognised but clinically important phenotype in which acute kidney injury reduces potassium clearance and promotes accumulation of AV-nodal–blocking agents, while hyperkalaemia potentiates their bradycardic effects, creating a self-amplifying cycle of hypoperfusion and worsening renal dysfunction. We reported a case managed in the Department of Emergency Medicine, Sri Ramachandra Institute of Higher Education and Research, Chennai. A 67-year-old man with type 2 diabetes mellitus and hypertension on metoprolol and enalapril presented with acute dyspnoea, productive cough, giddiness, and oliguria. He was hypotensive (80/60 mmHg) with marked sinus bradycardia (44/min) and wheeze, but no fever or chest pain. Arterial blood gas showed metabolic acidaemia (pH 7.30; $HCO_3^-$ 18.3 mmol/L) with hyperkalaemia ($K^+$ 6.97 mmol/L) and an elevated anion gap, and point-of-care testing showed acute kidney injury (creatinine 2.5 mg/dL). POCUS demonstrated preserved left ventricular function, no pericardial effusion, a collapsible inferior vena cava, and a right PLAPS-C pattern, leading to initial consideration of community-acquired pneumonia with shock and AKI. He received guideline-consistent hyperkalaemia therapy (IV calcium gluconate, insulin–dextrose, repeated high-dose nebulised salbutamol, and bicarbonate), fluid resuscitation, antibiotics, and vasopressors. Persistent hyperkalaemia and shock prompted ICU transfer and urgent haemodialysis, after which heart rate normalised (75/min) with sinus rhythm on ECG. This case underscores the need to recognise BRASH physiology early in unstable bradycardia, as syndrome-directed therapy—including timely renal replacement therapy—can be lifesaving.

## 1. Introduction

Bradycardia with hypotension represents a high-acuity presentation in emergency medicine because it can rapidly compromise cardiac output and precipitate end-organ hypoperfusion, metabolic derangements, and cardiac arrest if the underlying driver is not identified and treated promptly.(1) Advanced Cardiovascular Life Support (ACLS) guidance recommends an early 'treat-and-search' approach: clinicians first assess for instability (hypotension, altered mental status, signs of shock, ischemic chest discomfort, or acute heart failure), provide immediate supportive care (airway/oxygenation, monitoring, IV access), and initiate temporising therapy

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such as atropine and/or transcutaneous pacing and vasoactive infusions when clinically indicated.(2) Importantly, the same ACLS framework emphasises the need to concurrently evaluate reversible causes of bradycardia, including drug/toxicologic etiologies, hypoxia, and electrolyte abnormalities—especially hyperkalaemia—because definitive correction of the precipitant often determines outcome more than chronotropic therapy alone.(2)

Hyperkalaemia is among the most time-sensitive reversible causes of circulatory collapse encountered in the emergency department. It is well established that elevated serum potassium can depress myocardial excitability and impair conduction, producing bradyarrhythmias and malignant ventricular dysrhythmias.(3, 4) Lindner et al. (2020), Rafique et al. (2021) and Liu & Rafique (2019) recommend a parallel management strategy for clinically significant hyperkalaemia: (1) immediate cardiac membrane stabilisation with intravenous calcium when there is haemodynamic instability or electrocardiographic concern; (2) rapid intracellular potassium shifting with insulin–glucose and inhaled β2-agonists; and (3) definitive potassium removal through renal excretion (diuretics when appropriate), potassium binders in selected contexts, or renal replacement therapy when hyperkalaemia is refractory or renal failure is present.(3-5)

Within this landscape, BRASH syndrome—an acronym for Bradycardia, Renal failure, AV-nodal blockade, Shock, and Hyperkalaemia—has emerged as an important and under-recognised clinical entity that bridges toxicology, nephrology, and resuscitation medicine.(6) BRASH is conceptualised not as a single-cause diagnosis but as a synergistic vicious cycle: acute kidney injury reduces the clearance of AV-nodal–blocking drugs (most commonly β-blockers or non-dihydropyridine calcium-channel blockers) and diminishes potassium excretion; hyperkalaemia then potentiates the negative chronotropic and dromotropic effects of AV-nodal blockers, producing marked bradycardia; bradycardia and shock worsen renal hypoperfusion, further aggravating renal failure and hyperkalaemia, thereby amplifying the cycle.(7) A key clinical insight is that, in BRASH, the degree of bradycardia may appear disproportionate to the measured potassium level, and classic stepwise 'hyperkalaemia ECG' patterns may be absent or muted because the bradyarrhythmia reflects the combined effect of potassium and AV-nodal blockade rather than hyperkalaemia alone. It is particularly relevant in ageing populations and in patients with cardiometabolic comorbidities who are frequently prescribed β-blockers, calcium-channel blockers, and renin–angiotensin–aldosterone system inhibitors.

Against this clinical and pathophysiological background, we reported a case of a 67-year-old man who presented with acute dyspnoea, productive cough, giddiness, oliguria, and haemodynamic instability with marked bradycardia and hypotension, in whom investigations revealed metabolic acidaemia, hyperkalaemia, and acute kidney injury while on chronic AV-nodal blockade and ACE-inhibitor therapy. The case was identified and managed in the Department of Emergency Medicine, Sri Ramachandra Institute of Higher Education and Research, Chennai, and it underscored the diagnostic and therapeutic importance of recognising BRASH physiology early in the resuscitation pathway, particularly when concurrent infection or hypovolaemia plausibly precipitated renal dysfunction and triggered the syndrome's self-perpetuating cycle.

## 2. Case Report

A 67-year-old male presented to the emergency department with breathing difficulty for 1 day, cough with productive sputum for 3 days, and giddiness for 1 day. He denied fever and chest pain and also reported decreased urine output. His past medical history included type 2 diabetes mellitus for 3 years and hypertension for 2 years. He reported no known drug allergies and no history of alcohol intake, smoking, or illicit drug use. His regular medications included metformin 500 mg, enalapril 2.5 mg, and metoprolol 25 mg. On initial assessment, the airway was patent. He was breathing spontaneously with a respiratory rate of 24/min and maintained an oxygen saturation of 98% on 4 L/min oxygen(via Hudson mask). Chest examination revealed bilateral air entry with bilateral wheeze. Cardiovascular examination showed marked bradycardia with a heart rate of 44/min and hypotension with blood pressure of 80/60 mmHg; all peripheral pulses were palpable. Heart sounds (S1 and S2) were present with no murmurs, and the jugular venous pressure was not elevated. Neurological examination showed a Glasgow Coma

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Scale score of E4V5M6 with bilaterally 2 mm pupils reacting to light; capillary blood glucose was 160 mg/dL. He was afebrile, and general examination showed no pallor, icterus, cyanosis, clubbing, or pedal edema.

Initial investigations included a 12-lead electrocardiogram that demonstrated sinus bradycardia with no atrioventricular block and no ST-segment changes, with T-wave inversion in leads V1 and V2. Point-of-care venous blood gas analysis showed metabolic acidaemia with pH 7.301, $pCO_2$ 37.9 mmHg, $pO_2$ 28.3 mmHg, and bicarbonate 18.3 mmol/L, along with hyperkalaemia ($K^+$ 6.97 mmol/L), an elevated anion gap (24.5), and lactate of 2.83 mmol/L; blood glucose was 160 mg/dL. Point-of-care ultrasound revealed a right PLAPS-C profile, an inferior vena cava collapsing with respiration, adequate left ventricular function, no regional wall motion abnormality, no pericardial effusion, and no right atrial/right ventricular dilatation. A chest radiograph revealed consolidation in right lower lobe. Additional point-of-care tests showed creatinine 2.5 mg/dL, troponin-I 0.08, BNP 560, and Foley catheterisation yielded 25 mL of urine. Based on the presentation, initial differentials considered included community-acquired pneumonia with septic shock and acute kidney injury.

The patient received emergency management for hyperkalaemia with intravenous calcium gluconate (10 mL of 10%), intravenous human insulin (Actrapid) 10 units in 25% dextrose, and high-dose nebulised salbutamol 20 mg as needed (and repeated). He was also started on intravenous piperacillin–tazobactam 4.5 g, received intravenous fluids (normal saline 1 L bolus followed by 75 mL/hour), and vasopressor support with noradrenaline at 0.1 µg/kg/min; subsequently, adrenaline was started at 0.1 µg/kg/min, and sodium bicarbonate (75 mEq in 5% dextrose) was infused at 100 mL/hour. Repeat blood gas analysis showed persistent acidaemia and hyperkalaemia with pH 7.29, $pCO_2$ 40 mmHg, $pO_2$ 26.3 mmHg, bicarbonate 19.3 mmol/L, potassium 6.22 mmol/L (reported as 6.3 on repeat), glucose 116.3 mg/dL, anion gap 23.5, and lactate 1.54 mmol/L. He was shifted to the intensive care unit and initiated on haemodialysis, after which the heart rate improved to 75/min. A subsequent ECG (rate 77 bpm) showed sinus rhythm with premature supraventricular complexes and right bundle branch block (QRS 128 ms; QT/QTc 414/468 ms; PR 150 ms).

**Discussion**

The dominant physiology in this presentation is best explained by BRASH syndrome—a synergistic spiral of Bradycardia, Renal failure, AV-nodal blockade, Shock, and Hyperkalaemia—rather than isolated hyperkalaemia, isolated AV-nodal blocker toxicity, or ‘septic shock with AKI,’ as noted by Lehnhardt & Kemper (2011) and Shah et al. (2022).(8, 9) BRASH is increasingly recognised as a clinical phenotype in which relatively modest or moderate hyperkalaemia can produce disproportionate bradycardia when combined with AV-nodal–blocking drugs and impaired renal clearance, creating a self-reinforcing cycle of hypoperfusion and worsening potassium elevation.(10) In this case, several predisposing factors align tightly with BRASH. Older age, diabetes and hypertension increase baseline susceptibility to acute kidney injury during intercurrent illness (infection, dehydration).(8) The patient was taking metoprolol (AV-nodal blockade) and enalapril (RAAS blockade), a combination that can amplify bradycardia-shock physiology and promote hyperkalaemia, especially when renal function acutely declines. ACE inhibitors reduce aldosterone-mediated renal potassium excretion and can precipitate or worsen hyperkalaemia in the setting of renal impairment or acute illness; in corroboration with Khan et al. (2022).(11) Beta-blockers can contribute to hyperkalaemia by reducing cellular uptake of potassium (particularly via β2-blockade effects) and, more importantly for BRASH, by slowing AV-nodal conduction and sinus rate.(12, 13)

The clinical picture is also typical: marked bradycardia (44/min) with hypotension (80/60 mmHg) and oliguria with a point-of-care creatinine of 2.5 mg/dL indicates a low-flow state with renal hypoperfusion, which accelerates potassium accumulation and drug retention. Importantly, the ECG showed sinus bradycardia without AV block and without the classic progression of hyperkalaemic ECG changes; BRASH cases may not display ‘textbook’ hyperkalaemia ECG findings despite clinically significant potassium elevation, because the bradycardia is driven by the combined effect of potassium and AV-nodal blockade rather than potassium alone.(14) The post-dialysis improvement in heart rate to 75/min strongly supports a potassium- and renal-failure–driven mechanism, since reversal of hyperkalaemia and uraemic/acidotic physiology can rapidly restore nodal

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function and haemodynamics in BRASH, as noted by Subedi et al. (2025).(15)

A concurrent trigger still matters. The patient had cough with sputum, wheeze, and a lung ultrasound 'PLAPS-C' pattern (posterolateral alveolar-pleural syndrome with anterior consolidation profile), which in the BLUE (Bedside Lung Ultrasound in Emergency) framework supports pneumonia as a likely cause of acute respiratory symptoms.(16) Infection (or any volume-depleting illness) can initiate BRASH by causing dehydration, sepsis-associated renal hypoperfusion, and metabolic acidaemia, thereby worsening hyperkalaemia and drug accumulation; thus, pneumonia and BRASH are not mutually exclusive, and pneumonia may have been the precipitating stressor rather than the sole explanation for shock.(8) The collapsing IVC on POCUS is consistent with low preload/hypovolaemia, supporting prerenal contribution to AKI and the rationale for cautious fluid resuscitation while monitoring for fluid intolerance.

Management priorities in BRASH emphasise simultaneous treatment of hyperkalaemia, shock, and bradycardia, rather than focusing exclusively on ACLS pacing/atropine alone.(6) In life-threatening hyperkalaemia, immediate membrane stabilisation with intravenous calcium is recommended to reduce malignant arrhythmia risk. The administered 10 mL of 10% calcium gluconate is consistent with Khadka et al. (2024) that use calcium salts early when ECG/instability is present.(17) Potassium 'shifting' therapy with regular insulin plus dextrose and high-dose nebulised β2-agonist (albuterol/salbutamol) is also concordant with Eltom et al. (2025) for rapid intracellular redistribution of potassium.(18) The use of sodium bicarbonate is most physiologically justified when metabolic acidaemia coexists (as here, bicarbonate 18–19 mmol/L with elevated anion gap), because correction of acidaemia can facilitate potassium shift and improve catecholamine responsiveness, though bicarbonate is not a substitute for definitive potassium removal when renal failure is present. The persistent hyperkalaemia (6.2–6.3 mmol/L) and ongoing shock despite initial shifting measures appropriately prompted escalation to haemodialysis, which provides definitive potassium removal and correction of acid–base derangements when renal excretion is impaired.(19) In BRASH, dialysis can be particularly decisive because it simultaneously addresses multiple drivers of the vicious cycle—hyperkalaemia, uraemia, and acidaemia—thereby reversing bradycardia and improving perfusion, as observed with the heart rate rising to 75/min after dialysis.

The haemodynamic strategy in this case—fluids guided by ultrasound findings plus vasopressors—also matches contemporary BRASH thinking; restoring perfusion is essential to halt renal deterioration and ongoing potassium accumulation.(9) While atropine and pacing remain part of standard symptomatic bradycardia algorithms, BRASH bradycardia may respond incompletely to atropine because the primary mechanism is not increased vagal tone but impaired nodal conduction from hyperkalaemia and drug effect; therefore, parallel potassium-directed therapy is critical.(8) Current ACLS updates describe atropine as a first-line drug for symptomatic bradycardia at 1 mg IV every 3–5 minutes (maximum 3 mg), with infusion therapies (epinephrine or dopamine) and pacing as subsequent options when instability persists.(20) In practice, BRASH often improves most reliably when clinicians treat the underlying metabolic-renal cause and use vasoactive support to bridge perfusion while potassium is corrected.

Finally, several 'distractors' in the work-up can be reframed through the BRASH lens. Mild troponin elevation and BNP elevation can reflect demand ischemia, renal dysfunction, and myocardial strain in shock states rather than primary coronary occlusion, particularly when ECG lacks ischemic ST-segment changes and POCUS shows preserved LV function without regional wall-motion abnormality. The subsequent ECG demonstrating right bundle branch block and supraventricular ectopy after stabilisation may represent transient conduction vulnerability during metabolic derangement and catecholamine exposure, reinforcing the need for continuous monitoring during correction.(8) Overall, recognising BRASH early is clinically important because it prompts clinicians to stop AV-nodal/RAAS-blocking agents temporarily, aggressively treat hyperkalaemia, correct hypovolaemia/acidosis, and consider early dialysis when refractory, which can rapidly reverse the spiral and avert repeated ineffective bradycardia-only interventions.

**Conclusion**

This case highlights BRASH syndrome as a time-critical, synergistic cause of profound bradycardia and shock in

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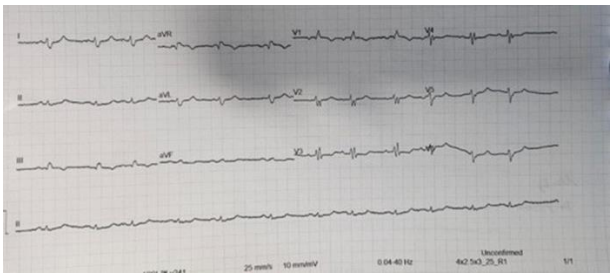
an older patient receiving AV nodal blockade and renin–angiotensin system inhibition, precipitated by acute kidney injury with hyperkalaemia and metabolic acidaemia. Although the initial presentation reasonably suggested infection with hypoperfusion, the combination of marked bradycardia disproportionate to the degree of hyperkalaemia, reduced urine output with rising creatinine, and chronic metoprolol/enalapril use supported BRASH physiology, in which renal dysfunction and hyperkalaemia amplify AV nodal blockade, leading to worsening hypoperfusion and further renal failure. Early recognition enabled targeted, simultaneous management—cardiac membrane stabilisation, intracellular potassium shifting, correction of acidaemia and perfusion, discontinuation of offending agents, and timely renal replacement therapy—resulting in rapid haemodynamic recovery and restoration of sinus rhythm. Clinicians should maintain a high index of suspicion for BRASH in patients with unexplained bradycardia and shock, particularly when renal dysfunction and AV nodal–blocking medications coexist, as prompt syndrome-directed treatment can be life-saving.



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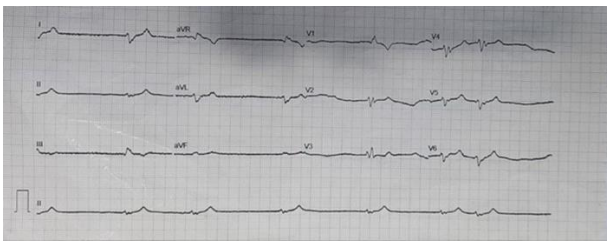

After Dialysis

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