



# Concurrent Digoxin Toxicity and BRASH syndrome

Natalia Paone MD, Jessica Duke MD, Erica Miller MD  
University of Rochester Medical Center Strong Memorial Hospital

## BACKGROUND

### DIGOXIN EFFECTS:

- ↑↑ Intracellular Ca<sup>2+</sup> and Arrhythmogenicity
- ↑↑ Vagal tone causing Bradycardia
- Narrow therapeutic window and Renally excreted

### BRASH SYNDROME:

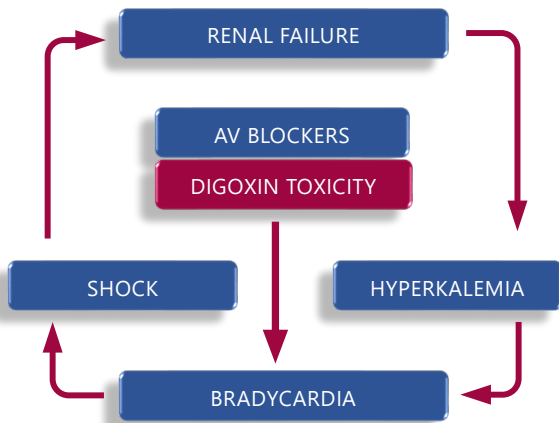


Figure 1: Pathophysiology of BRASH syndrome with concurrent digoxin toxicity<sup>1</sup>.

**Concurrent clinical suspicion for BRASH syndrome and digoxin toxicity can complicate diagnosis and clinical decision making.**

## CASE

A 74-year-old male with a history of non-ischemic cardiomyopathy and HFrEF s/p ICD, permanent atrial fibrillation, T2DM, CKD stage III, and recent COVID-19 infection presented with **progressive generalized weakness and lethargy**.

### Vitals

Heart Rate	30 bpm (via radial palpation)
Blood pressure	95/49 mmHg

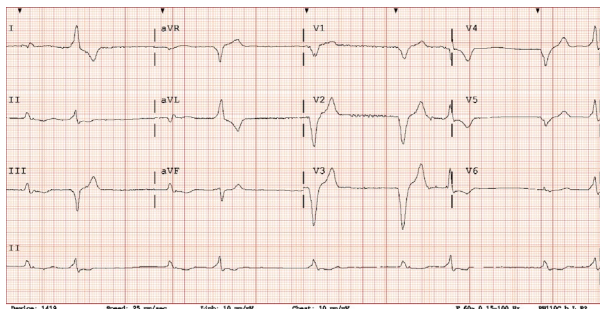
### Labs

K 6.1 mmol/L
Cr 4 mg/dL (baseline 1.6)
Lactate 1.0 mmol/L

### Home medications

Carvedilol 6.25 mg BID	Furosemide 40 mg daily	Spirolactone 25 mg daily
Digoxin 0.125 mg daily	Valsartan 20 mg daily	

**EKG:** heart rate of 55 bpm with ventricular paced complexes and PVCs, with PVCs contributing to underestimation of heart rate on radial palpation.



## DECISION MAKING

**THOUGHT PROCESS:** Acute renal failure, decreased oral intake precipitated by recent COVID-19 infection, and use of multiple AV nodal blocking agents were thought to cause BRASH syndrome. Digoxin toxicity was also suspected given his clinical history and elevated digoxin level.

The patient's **hyperkalemia** was of particular concern given the **direct correlation between hyperkalemia and mortality** in acute digoxin toxicity.

### WHAT WAS DONE:

- ✓ Home medications including carvedilol were held.
- ✓ Patient received **digoxin Immune Fab**, judicious fluid resuscitation, and calcium gluconate.
- ✓ **ICD back up rate was increased** to decrease ventricular ectopy and increase cardiac output.
- ✓ Once renal function and clinical status improved, the patient was **restarted on guideline-directed medical therapy** including carvedilol without complications.
- ✓ Patient was advised to **avoid digoxin** in the future.

## Conclusion

Digoxin Immune Fab is considered safe and effective treatment for severe toxicity, though it is costly and requires monitoring.<sup>2</sup>

**In this case, prompt recognition of concurrent BRASH syndrome guided initiation of supportive measures to interrupt the self-perpetuating cycle of bradycardia and renal hypoperfusion and decrease hyperkalemia-related mortality risk in the setting of acute digoxin toxicity.**

## References

1. Figure 1: BRASH syndrome with concurrent digoxin toxicity. Modified from: Farkas, J. D., Long, B., Koyfman, A., & Menson, K. (2020). BRASH Syndrome: Bradycardia, Renal Failure, AV Blockade, Shock, and Hyperkalemia. The Journal of emergency medicine, 59(2), 216-223. <https://doi.org/10.1016/j.ajem.2020.05.007>
2. Peters AE, Chiswell K, Hofmann J, Ambrosy A, Fudim M. Characteristics and Outcomes of Suspected Digoxin Toxicity and Immune Fab Treatment Over the Past Two Decades:2000-2020. Am J Cardiol. 2022 Nov 15;183:129-136. doi: 10.1016/j.amjcard.2022.08.004. Epub 2022 Sep 9. PMID: 36088419; PMCID: PMC9588603.