Chronic Hypotension Limiting GDMT in HFrEF with Underlying Adrenal Insufficiency

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Introduction

Guideline directed medical therapy (GDMT) with a betablocker, MRA, ARB/ARNI and SGLTi is known to improve morbidity and mortality in patients with HFrEF. As GDMT becomes more widely adopted it is increasingly common for patients with HFrEF to be treated with a variety of medications that all have the potential to lower systemic blood pressure. Unfortunately, target doses are often unachievable due to symptomatic hypotension, renal insufficiency, high cost, and many other factors contributing to medication nonadherence. We discuss possible underlying etiologies of chronic hypotension leading to drug class and dosage limitations in the treatment of chronic systolic heart failure.

Discussion

This patient's GDMT was gradually decreased prior to her admission, which may have contributed to worsening heart failure. The true effect of these dose adjustments is difficult to elucidate in cases such as this, which present with acute exacerbations of comorbid conditions that each may worsen cardiac function. An increase in her LVEF from 18% to 30% was noted over a two-day span during which she was treated with IV Hydrocortisone, without similar improvement in comorbid conditions.

If adrenal insufficiency is diagnosed, initiating treatment has been shown to quickly improve hemodynamic status.

Figure 1

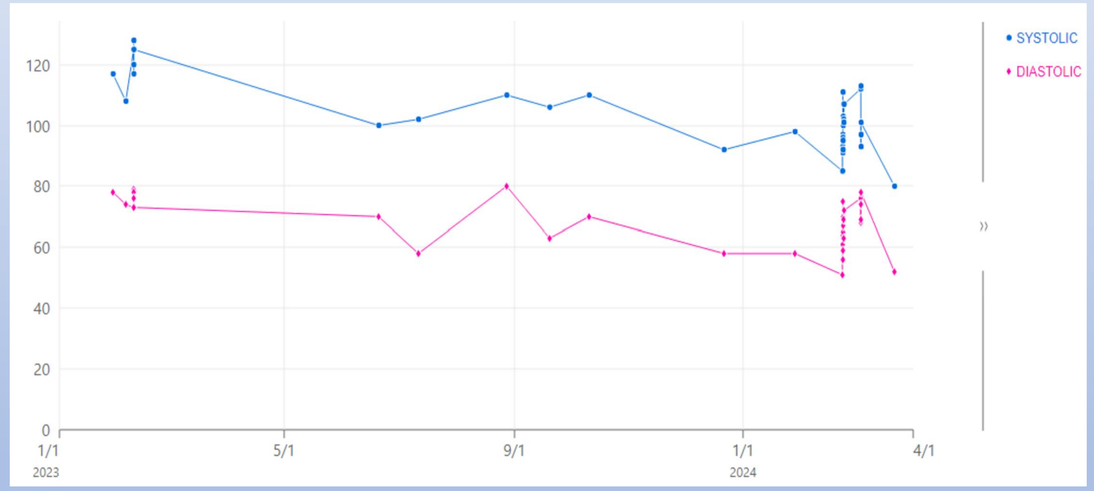

Figure 1: Ambulatory Blood Pressures

Figure 2

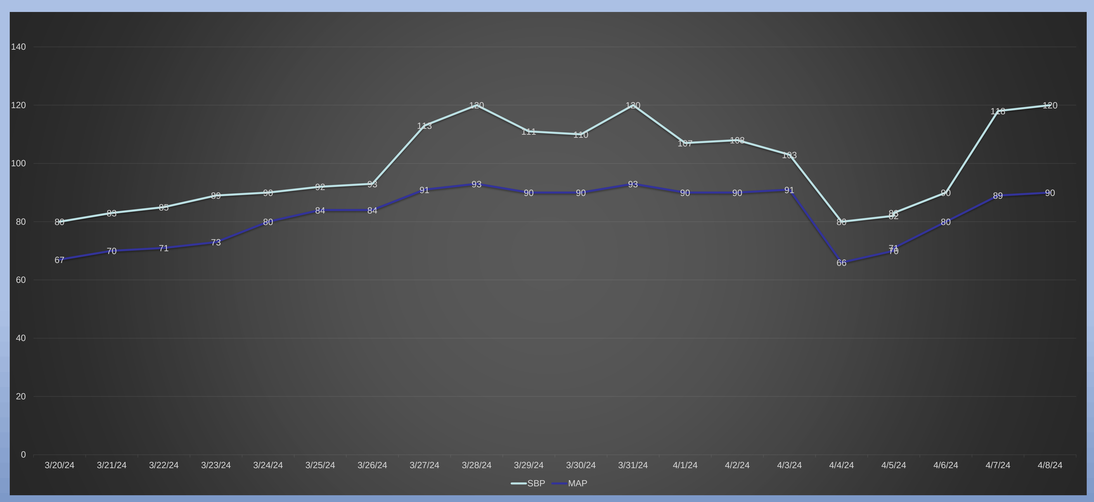

Figure 3: Inpatient Systolic Blood Pressure and MAP. IV Hydrocortisone was initiated on 03/27. Trial of PO Hydrocortisone was attempted on 04/04. IV was restarted on 04/06.

Case Presentation

A 67-year-old female with PMH of permanent Afib, ESRD s/p failure of remote deceased donor kidney transplant (2010) maintained on prednisone and tacrolimus for immunosupression, VT s/p dual chamber ICD (2007), vulvar cancer s/p radiation, breast cancer s/p combination chemotherapy, subsequent nonischemic cardiomyopathy from chemotherapeutic cardiotoxicity, and HFrEF (EF 30-40%). She also suffered from chronic hypotension with average systolic blood pressure in the 90s leading to discontinuation of carvedilol, reduction in her sacubitril-valsartan to 24/26mg BID, and initiation of proamatine 10 mg TID. Empagliflozin was discontinued due to worsening CrCl in the setting of failure of transplanted kidney.

She was admitted for hypotension, GI bleed, and ICD shock. She was treated with colestipol for chronic diarrhea and isavuconazonium for disseminated histoplasmosis. She developed worsening heart failure, with decrease in EF to 18%. This was felt to be due to worsening renal failure. She remained hypotensive with systolic blood pressure in the 80s. Adrenal insufficiency was considered given her long-term steroid use; however confirmative testing was not feasible given ongoing steroid treatment. Her blood pressure improved with empiric IV hydrocortisone. She elected to pursue peritoneal dialysis. Unfortunately, she developed respiratory distress secondary to pulmonary edema and required emergent intubation. Several days later she suffered a fatal cardiac arrest.

Conclusion

Testing for adrenal insufficiency is low-risk and should be considered in HFrEF patients with hypotension refractory to dose-reduction of medications with the potential to contribute to hypoperfusion.

Increased blood pressure following treatment of adrenal insufficiency may be sufficient to support dose escalation of GDMT while maintaining or even improving overall systemic prefusion.