Abstract Submission Form

The Women's Heart Center Program Committee is accepting abstract submission forms through **August 16, 2024**. Completed forms should be emailed to WHC@TheChristHospital.com.

Abstract submissions should be gender- and sex-specific research pertaining to one of the program topics outlined below.

The Program Committee wishes to encourage young scientific investigators and will reward up to 4 abstracts/posters submitted by presenters considered early career (definition provided below). First place will receive $1000, second place will receive $500, and two honorable mentions will each receive $250.

The presenting author will be sent an email with the status of the submission by **August 30, 2024**. If your abstract is accepted, your notification will contain complete presentation information. However, please note the following:

- All human subject research must conform to the principles of the Declaration of Helsinki of the World Medical Association.
- The presenting author should be able to provide documentation of IRB approval if requested.
- The Program Committee is unable to reimburse presenters for travel, hotel, or per diem expenses.
- Submission of an abstract constitutes a commitment by the presenting author (or designee) to present in-person at the symposium on October 11, 2024, during the following times:
    - Registration & Networking: 7:00 – 8:00 am
    - Networking Lunch: 12:00 – 1:00 pm
    - Poster Session Award Announcement: 3:40 – 4:00 pm
- All accepted abstract presenters must register for the symposium via Eventbrite and pay the applicable registration fees (trainees and invited speakers will have the registration fee waived).
- If an author wishes to withdraw an abstract, please email WHC@TheChristHospital.com.

Presenting Author Information

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Early Career (Defined as physicians, scientists, medical students, and other healthcare providers currently in residency or fellowship programs or within three years of training)? Yes ☒ No ☐

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Disclosures: Please list any relevant financial disclosures.
Nothing to disclose

Abstract Topic (must be gender- or sex-specific)

☐ Preventative cardiology	☒ General cardiology	☐ Interventional cardiology
☒ Heart failure	☐ Cardio-oncology	☐ Cardio-obstetrics
☐ Electrophysiology	☐ Cardiovascular Imaging	☐ Coronary Microvasculature
☐ Social Determinants of Health	☐ Mental Health	☐ Precision Medicine

Title: Include the full title as it will appear on the poster.
"Chronic Hypotension Limiting GDMT in HFrEF with Underlying Adrenal Insufficiency"

Background: In an initial paragraph, provide relevant information regarding the background and purpose of the study, preferably in no more than two to three sentences.

Guideline directed medical therapy (GDMT) with betablocker, MRA, ARB/ARNI and SGLTi are known improve morbidity and mortality in patients with systolic heart failure. 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. Consideration of dose-escalation is well studied, with recommendation for gradual escalation to a target dose of sacubitril-valsartan 97/103mg twice, metoprolol succinate 200 mg daily, carvedilol 50 mg twice, 10 mg daily for SGLTi and 50 mg daily for MRAs. Unfortunately, these target doses are frequently not meet for several reasons, including symptomatic hypotension, cost, medication adherence, and renal function. We discuss possible underlying etiologies of chronic hypotension leading to drug class and dosage limitations in the treatment of chronic systolic heart failure.

Case Presentation: Briefly state the methods used.

A 67-year-old female with a past medical history significant for permanent Afib, end stage renal disease status post failure of remote deceased donor kidney transplant (2010), dual chamber ICD placement in 2007 following an episode of VT, neurogenic bladder requiring frequent self straight-catheterization, vulvar cancer status post pelvic radiation, breast cancer status post combination chemotherapy, subsequent nonischemic cardiomyopathy from chemotherapeutic cardiotoxicity, and HFrEF (EF 30-40%). She also suffered from chronic hypotension with a systolic blood pressure averaging 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 creatinine clearance in the setting of failure of transplanted kidney. She was admitted to The Christ Hospital on for hypotension, gastrointestinal bleed, and ICD shock.

Colonoscopy was performed for chronic diarrhea (since 2020) and acute blood loss anemia in the setting of hematochezia and revealed innumerous mucosal lesions that when biopsied resulted with histoplasma capsulatum, diagnostic of disseminated histoplasmosis. She was started on colestipol for her chronic diarrhea and isavuconazonium sulfate for disseminated histoplasmosis. Over the next few days, she suffered from worsening peripheral edema and shortness of breath with her clinical status worsening from NYHA class II to NYHA class III-IV. This was felt to be secondary to her acute renal failure, as her creatinine continued to climb into the mid 6s up from her baseline of 1.70. Repeat TTE was obtained and showed acute worsening of her systolic function with an EF

estimated at 18% and severe diffuse hypokinesis. Her systolic blood pressure remained in the 80s. Her PTA prednisone was held due to her disseminated infection. Adrenal insufficiency was considered given her long-term steroid use; however confirmative testing was not feasible given ongoing steroid treatment. She was empirically placed on IV hydrocortisone and her systolic blood pressure subsequently improved to the 120s. Upon attempted transition to oral hydrocortisone, her systolic blood pressures once again dropped into the 80s. This lack of response was attributed to possible interaction with colestipol, as well as the possibility of malabsorption secondary to Histoplasma colitis. IV hydrocortisone was resumed with improvement in her blood pressures. She had no documented history of adrenal insufficiency, but did require stress-dosed steroids during a urologic procedure at UC a few years prior to presentation. She had been chronically immunosuppressed with prednisone 10 mg and tacrolimus since her transplant in 2007.

The patient elected to pursue peritoneal dialysis rather than hemodialysis. Unfortunately, following the placement of a PD catheter she developed respiratory distress secondary to pulmonary edema and required emergent intubation. Several days later she suffered a fatal cardiac arrest.

Discussion: Summarize the results in sufficient detail to support the conclusions.

Unfortunately, despite known benefit of GDMT, maximal dosing is precluded in many patients due to hypotension. This is case is a prime example of a possibly underdiagnosed cause of chronic hypotension in patients with heart failure, especially those on chronic steroids. Although data on the frequency of adrenal insufficiency in patients suffering from heart failure is lacking, there are isolated studies demonstrating acute onset heart failure with significant drops in LVEF secondary to adrenal insufficiency that improved with glucocorticoid administration. One such study, proposed that adrenal insufficiency should be suspected in cases of acute decompensated heart failure with concomitate severe hyponatremia defined as serum Na level less than 120 mEq/L. They acknowledge that severe hyponatremia occurs in 2-5% of all patients with acute decompensated heart failure, an extremely large number of people considering the increasing prevalence of heart failure (Fukuko Nagyra, Satoshi Kodera, Naoki Hayakawa and Juni Kanda, 2017).

This patient's GDMT medications were 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, and during which a similar improvement in her comorbid conditions was not seen.

The physiological effects of adrenal insufficiency are secondary to poor Na reabsorption in the distal tubule of the nephron caused by decreased aldosterone production with decreased or absent mineralocorticoid synthesis as well as decreased rennin production and decreased vascular response to catecholamines in the setting of glucocorticoid deficiency. This leads to a combination of decreased intravascular tone and decreased intravascular volume, which decrease circulating pressures and may be so severe that it affects organ prefusion. Testing for adrenal insufficiency is a low-risk diagnostic procedure which should be considered in HFrEF patients with hypotension refractory to dose-reduction of medications with the potential to contribute to hypoperfusion. Given the multitude of physiologic and pathologic variables affecting the free and bound cortisol levels and nuances related to HPA axis testing, consideration should be made to Endocrinology consultation for contextual interpretation of results, which are often complex. If adrenal insufficiency is diagnosed, initiating treatment has been shown to quickly improve hemodynamic status.

Conclusions: Concisely state the conclusions reached.

If adrenal insufficiency is suspected, proper confirmatory testing would allow appropriate treatment with glucocorticoid and mineralocorticoid replacement, which has been shown to reverse LVEF reduction in isolated case reports. The subsequent 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.

Tables/Figures/Graphics: Include images that are part of your submission here. Images should be high resolution and have a file type of “gif”, “jpg”, or “jpeg”.