



Catecholamine-induced cardiomyopathy in pheochromocytoma

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Introduction

Pheochromocytoma is a rare neuroendocrine tumor most commonly characterized by sustained or paroxysmal hypertension, headaches, diaphoresis and tachycardia due to excessive catecholamine secretion.

Case Presentation

Brief History:

A previously healthy 45-year-old man presented with urinary obstruction and crushing chest pain following an outpatient cystoscopy performed for urinary frequency and urgency. He reported 6 months of antecedent urinary frequency, and intermittent palpitations and headaches.

Physical Exam:

BP 193/119, HR 116, T 37.1C, RR 18, SpO2 92% on RA

Gen: diaphoretic man in mild distress

CV: tachycardic, no murmurs

Resp: normal breath sounds, CTAB

Abd: non distended, diffuse lower abdominal tenderness, no rebound/guarding

Neuro: No focal neurological deficits, motor strength and sensation grossly intact

Labs:

- CMP: Na 138, K 3.9, BUN 21, Cr 2.23, AST 36, ALT 64, ALP 102
- CBC: WBC 16.7, Hb 14.3, Plt 532
- Hs-Trop: 321 -> 913 -> 5756
- UDS negative
- TSH 1.25
- UA: 2+ prot, 3+ blood, trace ketones, 0-1 WBCs, >1000 RBC

EKG: Sinus tachycardia, RA enlargement, incomplete RBBB, age indeterminate septal infarct

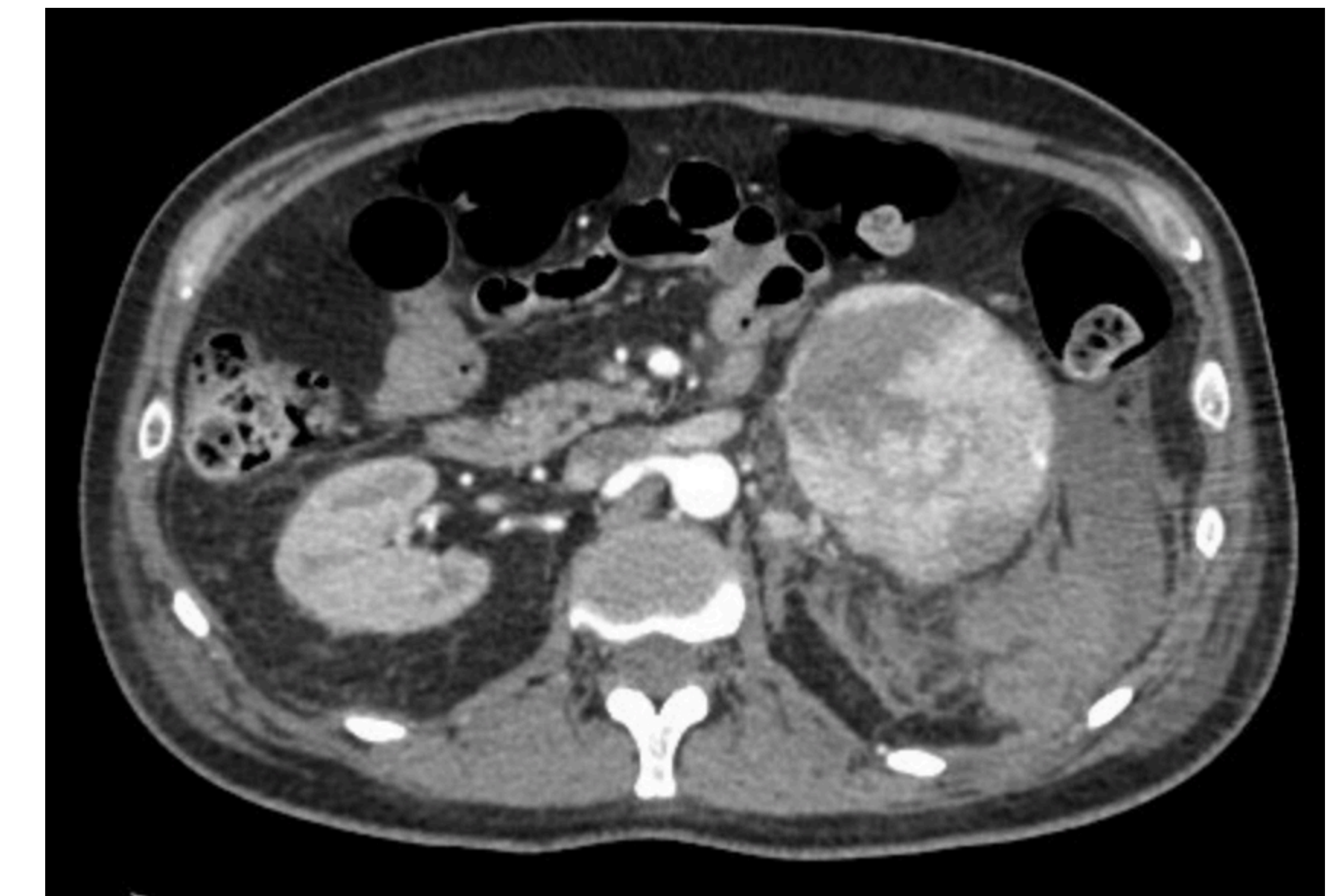
Imaging:

- CTA Chest: no acute abnormality, no evidence of central thromboembolic disease
- CT Abdo & Pelvis: large 8.6cm highly vascular left suprarenal mass likely adrenal in origin. Varices around mass and mass is displacing L kidney.
- TTE: global hypokinesis with apical ballooning, LVEF 20%
- Cardiac cath: normal coronary arteries

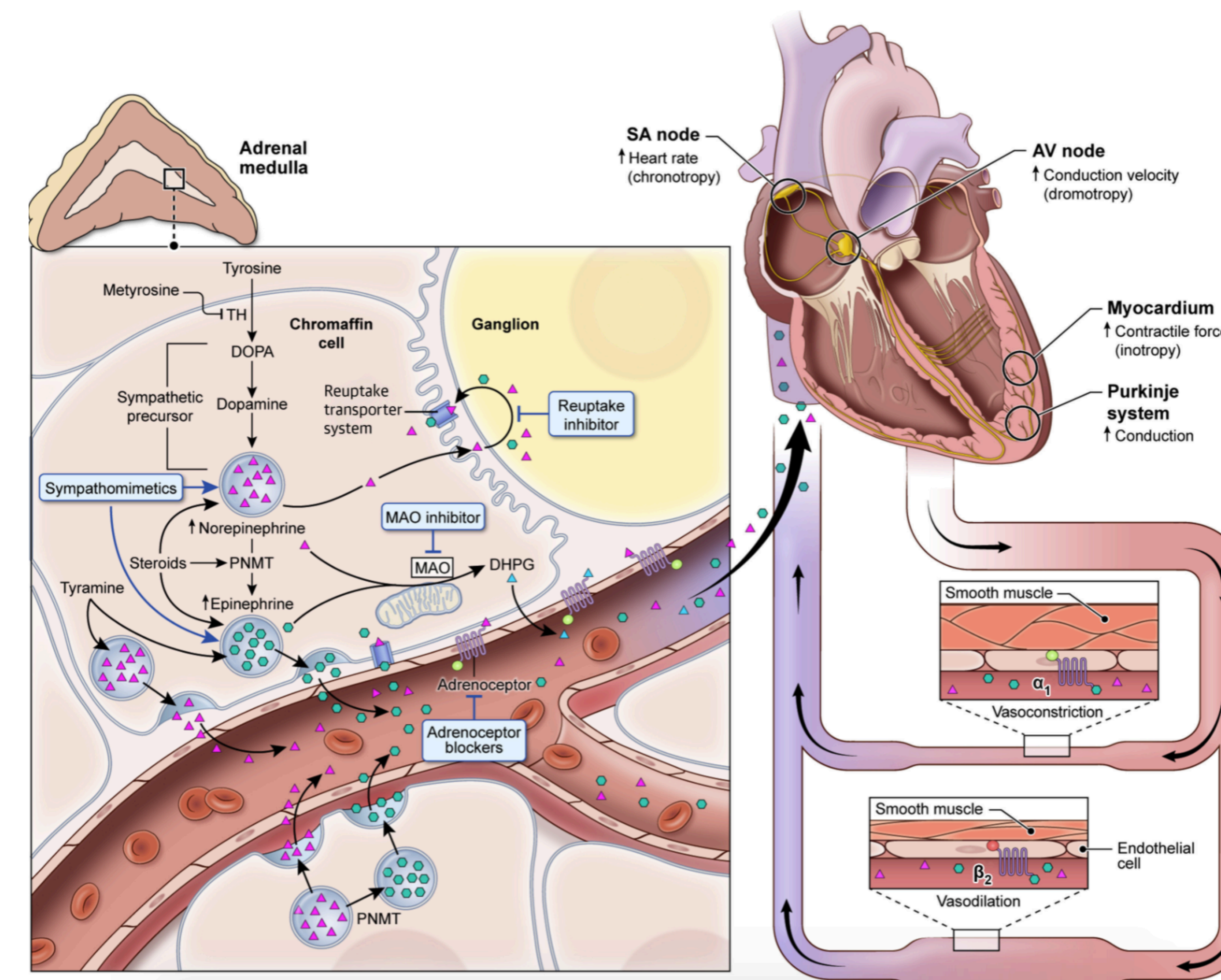
Hospital Course

- **Day 1:** Develops Afib w/ RVR and transferred to ICU for cardiogenic shock with pulmonary edema requiring pressors and HFNC
- **Day 2-3:** BPs stabilize and started on prazosin, renal artery duplex without signs of stenosis
- **Day 4:** Plasma metanephrine elevated to 4374 pg/mL. Develops hypertensive crisis with SBP 210, HR 160s (treated with prazosin, labetalol and esmolol) rapidly followed by hypotension requiring pressors. Excruciating L back and flank pain- imaging shows adrenal hemorrhage. Transferred to OHSU and started on phenoxybenzamine and metyrosine.
- **Day 20:** s/p open L adrenalectomy. Repeat TTE showing normal LV systolic function.

Imaging



Pathophysiology of pheochromocytoma induced cardiomyopathy



Discussion

- Pheochromocytoma can present with marked hemodynamic variation
- Cardiovascular complications may result from direct effects of excess catecholamines, such as myocardial stunning, or from end-organ complications, including adrenal hemorrhage.
- Catecholamine-mediated myocardial stunning leads to acute systolic dysfunction that may present with clinical features suggestive of acute myocardial infarction and cardiogenic shock.

Take Home Points

- Pheochromocytoma can cause acute systolic dysfunction presenting as an acute myocardial infarction or cardiogenic shock
- Myocardial stunning from excess catecholamines can present as stress-induced cardiomyopathy on TTE
- Early recognition requires imaging and urine/serum metanephrine levels
- Management involves alpha-adrenergic blockade followed by adrenalectomy
- Perioperative cardiac assessment and optimization is critical in preventing acute and long-term complications

References

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