Marathwada Medical & Research Institute
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The Complete Hospital
(NABH ACCREDITED HOSPITAL)

CARDIOLOGY CHRONICLES

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Conn's Syndrome Masquerading as Dilated Cardiomyopathy

A 41 y female was transferred from a nearby town for evaluation and management of left ventricular failure. On presentation to our hospital she had heart rate of 100 pm and BP of 190/105 mm Hg. ECG revealed presence of left atrial enlargement and non-specific ST-T changes. Echo showed a dilated LV (54 mm) with LVEF of 0.25 to 0.3 with generalised severe hypokinesis (Video 1). NT pro-BNP was markedly elevated (18000+). Cardiac Troponin was normal. She was treated with intravenous diuretics to control her left ventricular failure (LVF). Her serum potassium was 2.2 meq/L and was thought to be due to diuretics used for treatment of LVF. Blood pressure came down to 160/90 with long acting Nifedepine, ARNI and Spiranolactone. Her serum potassium coninued to remain low (less than 3 meq/L) despite oral and occasional IV supplementation. This raised the suspicion of secondary hypertension. Twenty four hours urinary metanephrines and VMA were normal. Renal ultrasound for renal artery stenosis was negative however a left suprarenal mass was seen. CT abdomen confirmed presence of a suprarenal mass on the left side (Fig 1,2,3). An adrenal adenoma (Conn's syndrome) was a strong possibility in view of an adrenal mass, persistent hypertension and refractory hypokalemia. Plasma Aldosterone concentration was elevated and direct renin levels were suppressed. This was highly suggestive of primary hyperaldosteronism or Conn's syndrome.

Gallium 68 Pentixafor PET-CT was performed to evaluate left adrenal nodule. This PET-CT assesses the CXCR4 (C-X-C motif chemokine receptor 4) expression in adrenal lesions & functioning adrenal adenomas have high expression of CXCR4.

Fig 1

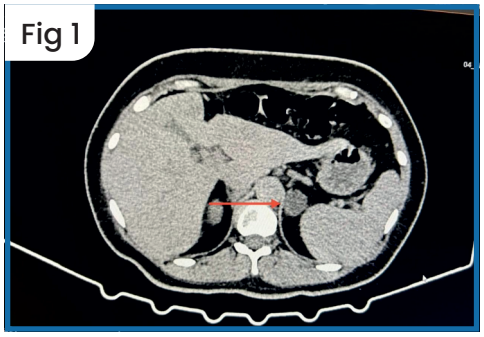


Fig 2

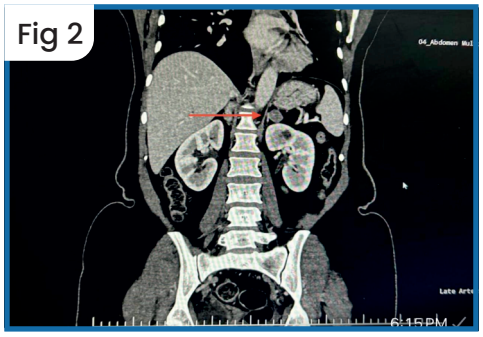


Fig 3

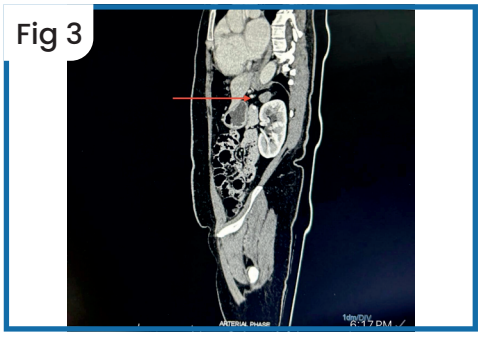


Fig 4

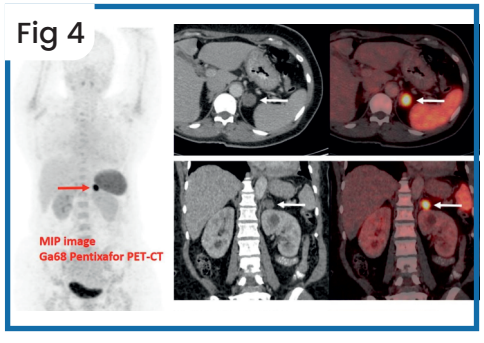
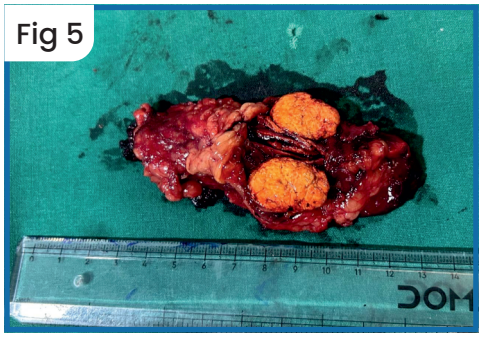
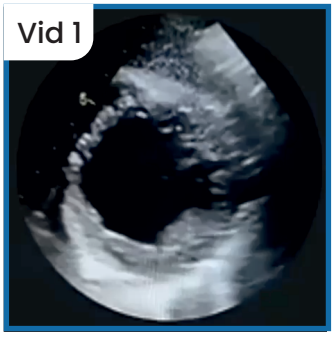


Fig 5



Vid 1



In this patient, the PET-CT showed intense, increased tracer uptake in 21 x 21 x 19 mm (ML X AP X CC) sized, left adrenal nodule (SUVmax= 26.6)- (Fig 4, arrow). This finding suggests presence of adrenal adenoma.

Patient underwent robotic left adrenelectomy and tumour excision 4 weeks ago (Fig 5). Histopathology confirmed the diagnosis. Blood pressure is now in the range of 140/90 with high dose of ARNI, Spiranolactone, Beta blockers and SGLT2-i. It's too early to see improvement in her LV function.

Key Learning Points

- Primary hyperaldosteronism is a common but frequently underdiagnosed cause of secondary hypertension.
- Persistent hypokalaemia despite potassium replacement should prompt investigation for mineralocorticoid excess.
- Conn's syndrome may rarely masquerade as dilated cardiomyopathy with severe LV systolic dysfunction. The mechanism of left ventricular dysfunction in Conn's syndrome extends well beyond pressure overload. Chronic aldosterone excess promotes myocardial fibrosis, oxidative stress, inflammation, endothelial dysfunction and adverse ventricular remodelling through activation of mineralocorticoid receptors. Aldosterone also enhances collagen synthesis and extracellular matrix deposition, leading to progressive myocardial stiffness and impaired contractility. Consequently, patients with primary hyperaldosteronism exhibit greater left ventricular hypertrophy, diastolic dysfunction, atrial enlargement, myocardial fibrosis and cardiovascular morbidity than patients with essential hypertension matched for blood pressure. Typically it takes several months for LV function to recover.
- Ga-68 Pentixafor PET-CT is an emerging functional imaging modality for localisation of aldosterone-producing adenomas.
- Surgical adrenalectomy is potentially curative in unilateral disease and may result in progressive improvement in cardiac function over subsequent months.

Overall, this case highlights the importance of maintaining a high index of suspicion for endocrine causes of cardiomyopathy. Recognition of primary hyperaldosteronism transformed the diagnosis from apparently irreversible dilated cardiomyopathy to a surgically treatable disease with the potential for meaningful recovery of myocardial function.

Note: We thank our chief radiologist Dr. Thomas George, chief nuclear medicine consultant Dr. Prathamesh Joshi, endocrinologist Dr. Lomte, Robotic surgeon Dr. Rajesh Saoji, our anaesthesia team and the pathologists team for helping us in managing this case.