Postural Orthostatic Tachycardia Syndrome unmasked by successful treatment of primary aldosteronism

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History:

50-year-old woman
History of chronic, symptomatic hypokalaemia and hypertension.
On examination weight 69.9kg, height 1.65 meters, BP 163/99, pulse 70,
Heart sounds normal and examination of the abdomen was unremarkable. ECG was normal.
Despite taking potassium supplements, repeat blood tests showed Sodium 148mmol/L, potassium 3.3mmol/L, Magnesium 0.87, Creatinine 56, eGFR > 90. Spot Urine potassium 25 mmol/L.
Primary aldosteronism was suspected
Further tests were arranged.

Renin was undetectable (< 1.1ng/L)
Serum aldosterone 772pmol/L.
Saline suppression test showed non-suppressible aldosterone levels.
Glucocorticoid-remediable aldosteronism was excluded. MRI failed to show a adrenal lesion.
Adrenal vein sampling confirmed a unilateral, right sided adrenal source for the aldosterone excess
Underwent successful right adrenalectomy.
Histology was supportive of the diagnosis

Progress:

Shortly after the operation she started to experience significant postural dizziness.
Relative suppression of aldosterone secretion by the contralateral gland suspected.
Fludrocortisone was started after a short synacthen test excluded adrenal deficiency.
She required increasing doses of fludrocortisone but despite this remained symptomatic.
Additionally she experienced headaches, fatigue, cold intolerance and breathlessness.
Therefore a neurocardiogenic process was suspected and she was referred to Cardiology.
Tilt table test showed her heart rate gradually rose to 138 bpm.
This was thought to be consistent with a POTs type response.

POTS:

Characterised by autonomic instability resulting in orthostatic intolerance.
Increase in Heart Rate > 30bpm or rate > 120bpm after standing (5 – 30mins).
Impaired venous innervation, alpha-1-adrehergic receptor denervation/insensitivity and/or beta-adrenergic receptor hypersensitivity contribute to reduce venous return on standing.
Associated symptoms include fatigue, sweating, palpitations, headaches, postprandial hypotension.
Typically affects women (5:1) aged 15 – 50 years.

Conclusions:

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Thus our patient had confirmed primary aldosteronism due to a adrenal adenoma, removal of which helped to unmask POTS.

We hypothesise that the excess mineralocorticoid activity from the adenoma hyper secreting aldosterone helped to mask the neurocardiogenic process.

References:

Agarwal et al. Postgrad Med J 2007; 83: 478-80
Abed et al. J Geriatr Cardiol 2012; 9: 61-7
Grubb et al. Circulation. 2008;117:2814-2817
Carew et al. Eurospace 2009; 11: 635-7

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