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Oedema in patients with Addison's disease on replacement therapy: glucocorticoid excess and mineralocorticoid deficiency?

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Abstract

Steroid hormones influence mechanisms related to oedema formation, including postural vasoconstriction and vascular tone. We studied fifteen patients (7 male, 8 female) with primary adrenal failure on clinically optimal replacement therapy. Five patients, all female, had clinically detectable oedema. Patients with oedema had evidence of mineralocorticoid deficiency, with increased supine and erect plasma renin activity and greater postural fall in blood pressure. Mean morning plasma cortisol levels were significantly higher in the group with oedema, suggesting they were receiving insufficient mineralocorticoid and a possible relative excess of glucocorticoid. There were no significant differences between patients with and without oedema in lower-limb cutaneous blood flow or in postural vasoconstrictor responses measured by laser Doppler flowmetry. The mechanism of oedema formation is unclear, but appears not to be modulated by haemodynamic mechanisms with expansion of intravascular volume or, in contrast to the known effects of sex hormones, by impairment of postural vasoconstriction. Theoretically, excess glucocorticoid replacement may result in oedema formation, by direct action on vascular tone, by altering capillary permeability, or by influencing other factors such as atrial natriuretic peptide. Measurement of plasma renin activity in conjunction with plasma cortisol profiles may be useful in adjusting replacement therapy in patients with Addison's disease and oedema.

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