

We suggest that there are two additional learning points from this case.

Firstly, phaeochromocytoma crisis with sustained hypotension is notoriously difficult to manage and there is normally a significant component of myocardial dysfunction due to catecholamine toxicity. An effective treatment (along with aggressive volume replacement) is some form of mechanical circulatory support such as cardiopulmonary bypass or veno-arterial extracorporeal membrane oxygenation.<sup>3,4</sup> The use of this type of circulatory support is strongly associated with improved survival in hypotensive phaeochromocytoma crisis.<sup>2</sup> If required, urgent surgery can be performed whilst on mechanical support.<sup>5</sup>

Secondly, the authors correctly point out that intravenous alpha blockade (phentolamine and phenoxybenzamine) is currently difficult to access in UK. In preference to using no alpha blocking agents, clinicians who find themselves in these circumstances should consider using intravenous magnesium for medical stabilisation.<sup>2,6</sup> There is an evidence base for intravenous magnesium<sup>7,8</sup> as an alternative to alpha blockade and importantly the drug is familiar to many intensivists due to its critical role in eclampsia treatment. ■

BENJAMIN C WHITELAW

Consultant endocrinologist, King's College Hospital NHS Foundation Trust, London, UK

JULIA K PRAGUE

Endocrinology registrar, Imperial College Healthcare NHS Trust, London, UK

OMAR G MUSTAFA

Consultant diabetes and endocrinology, King's College Hospital NHS Foundation Trust, London, UK

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## Response

We thank O'Toole, Brown and Drake for their informative response to our article.<sup>1</sup> We agree that it is important to highlight alternative parenteral regimens to alpha-blockers. In addition, volume expansion and careful management of fluid status is central to

successful management as in our case. We also thank Whitelaw, Prague and Mustafa for their insights into the use of mechanical circulatory support as rescue therapy. We acknowledge the association with myocardial dysfunction associated with catecholamine toxicity. In collaboration with colleagues in Oxford and London, we previously reported on the high prevalence of cardiac involvement in newly diagnosed phaeochromocytomas along with persistence of some parameters on cardiac magnetic resonance following successful surgery. We would advocate for the use of multi-centre registries for such rare conditions to improve treatment outcomes. ■

ZAKI HASSAN-SMITH

Consultant endocrinologist, Queen Elizabeth Hospital Birmingham, Birmingham, UK  
Visiting professor, Coventry University, Coventry, UK

SARAH FALOON

Foundation year doctor, Queen Elizabeth Hospital Birmingham, Birmingham, UK

NEIL GITTOES

Consultant and professor of endocrinology, Queen Elizabeth Hospital Birmingham, Birmingham, UK

JOHN AYUK

Consultant endocrinologist, Queen Elizabeth Hospital Birmingham, Birmingham, UK

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## CYP24A1 mutations and hypervitaminosis D

Editor – We read with interest the case report entitled 'Risks of the 'Sunshine pill' – a case of hypervitaminosis D'.<sup>1</sup> We wish to congratulate the authors on reporting this remarkable case, and hoped to make some additional contributions.

While noting that hypervitaminosis D is rare and can occur with excessively high doses of supplementation, they omit from their differential diagnoses the possibility of *CYP24A1* mutations, a well-described alternate cause of the phenotype described in their patient. Loss of function mutations in *CYP24A1* result in reduced action of 1,25-hydroxyvitamin-D<sub>3</sub>-24-hydroxylase, which usually inactivates active vitamin D. As well as a neonatal presentation, patients with *CYP24A1* mutations can present with adult-onset hypercalcaemia, together with low parathyroid hormone levels and high urinary calcium.<sup>2,3</sup> If this genetic condition is present, even modest vitamin D supplementation can lead to significant hypercalcaemia. Indeed, high levels of active vitamin D metabolites are found in some *CYP24A1*-deficient individuals even without supplementation.<sup>3</sup>

We acknowledge that in the case described by Ellis *et al* supplemental doses were truly high,<sup>1</sup> but the possibility of vitamin D unmasking *CYP24A1* mutations should have been considered. The identification of patients with *CYP24A1* mutations is