

Much more than gastro-enteritis: a severe case of hypo-pituitarism

Sebastian Vandermolen, Nelomi Anandagoda and Sarietha Krishnakumar

Renal Unit, Southend University Hospital, Southend on Sea SS0 0RY, UK

Corresponding author: Sarietha Kumar. Email: sarietha.kumar@southend.nhs.uk

Lesson

While well described in text books, acute presentations of hypo-pituitarism are rare and must be considered when patients present with vague symptoms with poor response to standard resuscitation procedures.

Keywords

postpartum fatigue, failure to lactate, postpartum weight loss, postpartum haemorrhage, Sheehan's syndrome

Case presentation

A 34-year-old woman with a history of polycystic ovarian syndrome self-presented to the emergency department, complaining of malaise, nausea and feeling cold. Her only previous hospital admission was for the birth of her first child one month earlier.

An obstetric history revealed that during the antenatal period, she developed severe headache and was investigated with a magnetic resonance imaging (MRI) brain in the 30th week of her pregnancy. This showed an enlarged pituitary gland with a cranio-caudal length of 12 mm. She was reviewed along with her imaging by the Neurologist, who felt the pituitary enlargement was physiological for her stage of pregnancy and the headache a manifestation of migraine.

She had a difficult vaginal delivery requiring the use of ventouse and forceps for shoulder dystocia with documented blood loss of 500 mL during delivery. Subsequent history from the patient revealed that during the in-hospital post-partum period, she experienced significant fatigue and generalised weakness. Further, the baby was commenced on formula feed due to lactation failure. The general malaise continued upon discharge, her mobility was poor, with muscle weakness and postural dizziness requiring assistance with various activities of daily living. She also noticed involution of her breasts and considerable weight loss of 6 kg from her pre-pregnancy weight. She spent a large proportion of the post-

natal period in bed, a considerable change from her previously active lifestyle with hobbies including hiking and rock climbing.

She consequently visited her local GP on numerous occasions and was diagnosed with post-natal depression and urinary tract infections. Around one month after delivery, she finally brought herself to hospital, as she was unable to cope with the symptoms of severe dizziness, nausea and vomiting. Initial observations suggested that she was in shock (heart rate 120 bpm, blood pressure 86/40, respiratory rate 18 and saturation 96% on air). Urine output was poor, but Glasgow Coma Scale was 15 throughout. Blood tests on arrival showed a normal haemoglobin and white cell count. There was evidence of acute kidney injury (AKI) with a creatinine of 128 $\mu\text{mol/L}$ (baseline around 70 $\mu\text{mol/L}$), with normal sodium and potassium levels. At this stage, she was aggressively fluid resuscitated with intravenous fluids. Upon review by the medical team, a diagnosis of gastro-enteritis and dehydration was made, and she was moved to a medical ward. That her AKI had not completely resolved, together with her ongoing general malaise, triggered the need for further detailed analysis. A thorough history including the details of labour revealed the possibility of a large post-partum bleed, which, together with the clinical picture, thus raised the possibility of Sheehan's syndrome. Her blood glucose was checked on the ward and revealed it to be low at 2.6 mmol/L. A hormonal screen was performed with results consistent with this diagnosis (Table 1). After initial treatment with intravenous corticosteroids, followed by oral levothyroxine, the patient made a rapid recovery. Within hours, she demonstrated both a subjective improvement in symptoms and objective improvement in physiological parameters (blood pressure and heart rate). Her cortisol rose to 1489 nmol/L (normal range 170–540 nmol/L – her high value was a consequence of high-dose glucocorticoid replacement) and her

Table 1. Biochemistry results on admission and one month post-discharge, with local normal reference ranges.

	On admission	After thyroid/cortisol replacement	Normal values
Clinical state	In shock, unwell	On ward, clinically better	
Free T4	11.6	14.1	12–22 pmol/L
TSH	1.10	6.49	0.27–4.2 U/L
Cortisol	7	1489	170–540 nmol/L
ACTH	<5	–	0–50
IGF	9.6		9–33.8 nmol/L
Estradiol	52		70–1480 pmol/L
Prolactin	<10	<10	0–492 mU/L

IGF: insulin-like growth factor; TSH: thyroid-stimulating hormone.

thyroid level returned to that within normal range. Her blood glucose was measured at 20.3 mmol/L. This high value was attributable to the intravenous glucose infusion. The glucose levels came down spontaneously after switching to Normal Saline.

Within two days, the patient was fit for discharge and was followed up on an outpatient basis within the endocrine department. Pituitary axis tests performed one month after discharge confirmed an ongoing cortisol deficiency. Low insulin-like growth factor further confirmed hypo-pituitarism. Prolactin remained low throughout the episode – this in itself a strong marker of a destructive process affecting the anterior pituitary. Estradiol was low, suggesting hypo-gonadotropic hypo-pituitarism secondary to pituitary infarction. Expectedly, luteinizing hormone and follicle stimulating hormone were low throughout this episode, and menses had not returned at endocrine outpatient follow-up one month later.

Discussion

Post-partum necrosis of the anterior pituitary was first described in 1937 by Sheehan.¹ While Sheehan's syndrome is rare in the developed world due to improvements in obstetric care, its prevalence is perhaps higher than expected, estimated at five per 100,000 patients in a recent study.²

The majority of patients has non-specific problems such as weakness and fatigue and thus go undiagnosed (therefore, untreated) for a many years post-partum. The case described here, with a presentation of acute hypo-pituitarism one month after delivery, is rare.³ The large variation in the duration between post-partum haemorrhage and clinical manifestation makes this a potentially difficult diagnosis to make.

However, severe pituitary insufficiency can result in coma or death, making it imperative for clinicians to recognise and instigate prompt management.

Clinical presentations are varied, with damage to more than 75% of the pituitary gland needed before clinical signs become apparent.⁴ Presenting features depend on the precise hormones involved. Growth hormone tends to be affected first and may cause fatigue and altered body composition.⁴ ACTH/cortisol deficiency will cause fatigue, weakness, weight loss and low blood pressure. Thyroid-stimulating hormone deficiency will result in a hypo-thyroid state, with fatigue, feeling cold and constipation. Prolactin deficiency will cause problems with lactation.⁴

In terms of imaging, as mentioned in the case presentation, this patient's MRI scan during her pregnancy confirmed enlargement of the pituitary gland. Pituitary enlargement during pregnancy is common due to increased vascularity⁵ and may well have contributed to increased susceptibility of the pituitary to infarction in the setting of acute blood loss during labour. Interestingly, when MRI pituitary was performed on this patient one month after presentation, pituitary size was described as normal. An 'empty sella' may be the expected MRI findings following pituitary necrosis, but the pituitary often undergoes enlargement followed by gradual shrinkage which can take months to years to appear radiologically.⁶

This case highlights the need for thorough assessment in particular with regard to the clinical history and the challenges of recognising the symptoms and signs of acute pan-hypo-pituitarism.

Declarations

Competing interests: None declared

Funding: None declared

Ethical approval: Written informed consent was obtained from the patient for publication of this case report.

Guarantor: SK

Contributorship: SV and NA contributed to the writing of this case report. SK was the patient's primary physician and reviewed and edited the manuscript.

Acknowledgements: Michael Almond, Renal Unit, for funding the publication.

Provenance: Not commissioned; peer-reviewed by Karl Metcalfe

References

1. Sheehan HL. Postpartum necrosis of anterior pituitary. *J Pathol Bacteriol* 1937; 45: 189–214.
2. Kristjansdottir HL, Bodvarsdottir SP and Sigurjonsdottir HA. Sheehan's syndrome in modern times: a nationwide retrospective study in Iceland. *Eur J Endocrinol* 2011; 164: 349–354.
3. Kelestimur F. Sheehan's syndrome. *Pituitary* 2003; 6: 181–188.
4. Stewart PM and Vance ML. *Hypopituitarism – Your Questions Answered*. See http://www.pituitarysociety.org/public/specific/hypopituitarism/PituitarySociety_HypopituitarismEnglish.pdf (last checked 26 February 2014).
5. Gonzalez JG, Elizondo G, Salvidar D, Nanez H, Todd LE and Villareal JZ. Pituitary gland growth during normal pregnancy: an in-vivo study using magnetic resonance imaging. *Am J Med* 1988; 85: 217–220.
6. Kaplun J, Fratila C, Ferenczj A, Yang WC, Lantos G, Fleckman AM, et al. Sequential pituitary MRI imaging in Sheehan syndrome: report of 2 cases. *AJNR* 2006; 29: 941–943.