A postpartum bald patch

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Case report

A 26-year-old primigravida with an uncomplicated pre-conception and antenatal history was induced at 41 weeks' gestation. Following a normal delivery, she suffered a life-threatening haemorrhage secondary to uterine atony. A B-lynch suture and bilateral uterine artery ligation proved ineffective and a hysterectomy was performed. The estimated blood loss was 7.1 litres.

She developed disseminated intravascular coagulation, with platelet levels dropping to 19 and LDH levels rising to 2,052. She received 23 units of blood, 10 units of cryoprecipitate, fresh frozen plasma, platelets and Factor VIIa.

There was ischaemic liver injury with ALT levels of 2,739. She also developed a femoral deep vein thrombosis. The biochemical markers improved on supportive therapy and warfarin. However, by 3 weeks postnatally, the patient continued to be lethargic and sleepy all day. The baby was bottle-fed, as the mother was too ill immediately postpartum.

At this time, she noticed an area of alopecia areata on the posterior aspect of her scalp measuring 5 cm in maximum diameter. An endocrine screen was arranged (Table I).

A diagnosis of Sheehan's syndrome was made based on these results and the alopecia was attributed to it. Treatment was instituted with 50 μg of thyroxin, 20 mg hydrocortisone in the morning and 10 mg hydrocortisone in the evening, and fludrocortisone. By the third day, there was dramatic improvement clinically. She was discharged home soon after with outpatient follow-up.

An MRI scan of the head showed a very small pituitary gland and the posterior pituitary could not be identified at all. There was no evidence of a pituitary adenoma.

At endocrine outpatient follow-up, she was found to have postural hypotension and polydipsia. Her quality of life index was poor. Hence she was started on desmopressin spray and growth hormone analogue after further biochemical assays. Her thrombophilia screen was negative and hormone replacement therapy with Climaval was started. Her symptoms improved on medication.

Table I. Results from endocrine screen

	Result	Normal range
Follicle stimulating hormone	0.2 IU/l	5–20 IU/l
Luteinising hormone	<0.1 IU/l	5–20 IU/l
Thyroid stimulating hormone	0.25 μ/l	0.4–5 μ/l
Cortisol	69 nmol/l	>180 nmol/l

Discussion

Sheehan's syndrome is characterised by hypopituitarism secondary to necrosis of the physiologically enlarged pituitary gland in pregnancy. This occurs due to massive peripartum blood loss (Sheehan 1954).

Often the diagnosis is delayed despite features such as failure of lactation, amenorrhoea, and loss of axillary and pubic hair, hyponatraemia and hypoglycaemia.

The endocrine features include secondary hypothyroidism, adrenal cortex failure, hypogonadotrophic hypogonadism and growth hormone deficiency (Sert et al. 2003).

Time to make a definitive diagnosis of the disease ranges between 5 and 25 years with a mean of 16.35 $\pm$ 4.74 years (Ozkan and Colak 2005). This may be because pituitary necrosis is frequently incomplete with selective loss of hormone secretion (Ozbey et al. 1994).

In our case, the diagnosis of an endocrine disorder was suspected at 26 days, when the isolated area of alopecia was seen, superimposed on constitutional symptoms of continued fatigue, well beyond her systemic thromboembolic and liver injury recovery. Although loss of pubic and axillary hair is common, as far as we are aware, a discrete area of scalp alopecia has not been previously described in association with Sheehan's syndrome. Whether this happened because of hormone deficiency of panhypopituitarism or as a result of the normal pregnancy changes affecting scalp hair growth patterns, is not clear. However, it was an unusual clinical sign that prompted further investigations that led to the diagnosis of Sheehan's syndrome.

The imaging studies demonstrated the classical partially empty sella described in the literature.

The biochemical manifestations of Sheehan's syndrome were so dramatic in this patient that stimulation tests were not felt to be of additional value in directing the management.

Our case illustrates the importance of unusual clinical signs in guiding the early diagnosis of this condition, now rare in the developed world due to availability of blood and oxytocics. Here, amenorrhoea would otherwise have been attributed to the hysterectomy and failure of lactation to maternal apathy. Although she received no lactation suppressant medication, there was no commencement of normal lactation.

References

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