occluded by thrombus from the bifurcation to the coeliac axis. Both renal arteries, the superior mesenteric, and the right vertebral arteries were thrombosed with the kidneys, small bowel, and part of the cerebellum infarcted. No venous thrombosis was present. The patient also had gall stones.

Comment

Oestrogen-containing contraceptives are well known to cause abnormalities of coagulation¹ and venous thromboembolic disorders.² Arterial thrombosis is less often encountered, though cerebral,² coronary,² mesenteric,³ and vertebral artery thromboses⁴ are described. But these events are relatively rare and usually occur singly. Our patient is unusual in that she experienced widespread large artery thrombosis without venous involvement, leading to ischaemia of the legs, infarction of kidneys and small bowel, and the lateral medullary syndrome. In the absence of other known factors influencing coagulation the oral contraceptive use is thought to be significant. Enhancing her susceptibility to oestrogen-associated thromboses would be her smoking habit, age of 33 years, and taking the pill for over five years.³ Two further adverse factors were obesity and hypertension.⁵ Massive arterial thrombosis is unusual and represents another major complication of oral contraception. It further emphasises the dangers of oestrogen-containing pills in women in the high-risk group defined by the Royal College of General Practitioners' report.³

We thank Mr D Charlesworth for allowing us to report this case.

¹ Poller L, Tabiwo A, Thomson JM. Effects of low-dose oral contraceptives on blood coagulation. *Br Med J* 1968;3:218-9.

² Inman WHW, Vessey MP. Investigation of deaths from pulmonary, coronary, and cerebral thrombosis and embolism in women of child-bearing age. *Br Med J* 1968;2:193-99.

³ Beral V, Kay CR. Mortality among oral-contraceptive users. Royal College of Practitioners' oral-contraception study. *Lancet* 1977;2:727-31.

⁴ Ask-Upmark E, Bickerstaff ER. Vertebral artery occlusion and oral contraceptives. *Br Med J* 1976;1:487-8.

⁵ Anonymous. The pill and raised blood pressure. *Br Med J* 1976;1:58-9.

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Department of Medicine, University Hospital of South Manchester, Manchester M20 8LR

P M HOLT, MB, MRCP, senior house officer (present appointment: registrar, University College Hospital, London)

D HOLLANDERS, MB, MRCP, lecturer in medicine

Postpartum cranial diabetes insipidus

Cranial diabetes insipidus is uncommon and is rarely associated with Sheehan's syndrome.¹ Isolated impairment of posterior pituitary function in the face of entirely normal anterior pituitary function is most unusual after postpartum haemorrhage. We here describe a patient with such abnormalities.

Case report

A 23-year-old Yemeni woman, gravida 5, para 3, ruptured her uterus during delivery of her fourth child at term, when she had a severe uterine haemorrhage and hypotension which required emergency hysterectomy. Postoperatively she developed sudden severe thirst, polydipsia, and polyuria. She was unable to establish lactation but her child thrived. During the next 12 months she lost 5 kg in weight, continued to pass 7-8 l urine/24 h, but took no medication. Physical examination showed no signs of hypopituitarism. She was clinically euthyroid; blood pressure was 120/75 mm Hg. Milk could not be expressed from her breasts. Urine output was 6.5 l/24 h with no glycosuria. Radiograph of sella turcica, perimetry of visual fields, and computerised tomography scan of head were all normal. Serum calcium, glucose, and electrolytes were within normal limits, as was thyroid function (thyroxine 112 nmol/l (8.7 µg/100 ml), triiodothyronine uptake 106%). Anterior pituitary stimulation by insulin-induced hypoglycaemia, thyrotrophin releasing hormone (TRH), and luteinising hormone-releasing hormone (LHRH) were entirely normal (table) and basal prolactin (230 mU/l) was not raised. But despite fluid restriction, which resulted in a 4.5% weight loss, and an increase in plasma osmolality of 10 mmol/kg there was little decrease in urine flow rate or increase in urine osmolality (table). Intranasal DDAVP (synthetic vasopressin analogue) 40 µg caused a prompt rise in urine osmolality. Furthermore, immunoreactive plasma vasopressin² was low (2.3 pg/ml) and failed to rise after osmotic stimulation with hypertonic saline which increased plasma osmolality by 23 mmol/kg. All the patient's symptoms were controlled with intranasal DDAVP 10 µg daily.

Results of anterior and posterior pituitary stimulation tests in case of postpartum cranial diabetes

	Time (min)	Glucose (mmol/l)	Cortisol (nmol/l)	GH (mU/l)	LH (U/l)	FSH (U/l)	TSH (mU/l)
Anterior pituitary	0*	5.2	290	5	12	7	2.3
	30	1.7	—	15	30	—	10.7
	60	2.3	730	14	14	—	8.4
	90	3.2	510	8	—	—	—
	120	4.5	510	7	—	—	—
	Time (h)	Weight (kg)	Plasma osmolality (mmol/kg)	Urine flow (ml/min)	Urine osmolality (mmol/kg)		
Posterior pituitary	0	39.8	293	—	70		
	1.5	—	—	8.9	73		
	2	38.9	299	9.5	107		
	3.5	38.2	—	3.9	190		
	5†	38.0	303	3.4	275		
	8	—	—	0.6	611		
	23	40.2	289	0.2	753		

*Soluble insulin 6 U, LHRH 100 µg, TRH 200 µg given intravenously at time 0.

†DDAVP 40 µg given intranasally at 5 h and patient allowed free fluids.

GH = growth hormone; LH = luteinising hormone; FSH = follicle-stimulating hormone; TSH = thyroid-stimulating hormone; LHRH = luteinising hormone releasing hormone; TRH = thyrotrophin releasing hormone.

Conversion: SI to traditional units—Glucose: 1 mmol/l ≈ 18 mg/100 ml. Cortisol: 1 nmol/l ≈ 0.32 µg/100 ml. Osmolality: 1 mmol/kg = 1 mosmol/kg.

Comment

This patient had clear evidence of cranial diabetes insipidus from the results of fluid restriction and subsequent response to DDAVP and also from the lack of vasopressin response to osmotic stimulation by hypertonic saline. Yet there was no suggestion of anterior pituitary dysfunction on either clinical or biochemical grounds. From the history the abnormalities apparently dated from the immediate postpartum period after uterine rupture. We were unable to find any other possible cause for the diabetes insipidus—particularly pituitary and suprasellar tumours that occasionally result in isolated diabetes insipidus. To our knowledge only one similar case has been reported³ and in that instance plasma vasopressin concentrations were not measured.

Pituitary dysfunction after postpartum haemorrhage usually affects only the anterior part of the gland. Nevertheless, despite the lack of clinical evidence of cranial diabetes insipidus, most patients with longstanding Sheehan's syndrome have neuronal degeneration of the supraoptic and paraventricular nuclei of the hypothalamus with associated changes in the posterior pituitary.⁴ Therefore other hypothalamic disorders might be expected with Sheehan's syndrome. Persistent hyperprolactinaemia and galactorrhoea in association with cranial diabetes insipidus but preserved anterior pituitary function have been described in only two patients.⁵ Why the posterior pituitary and hypothalamus, in contrast to the anterior pituitary, are so rarely affected by postpartum haemorrhage is not clear. The explanation may be due, in part, to a difference in blood supply, since the adenohypophysis is supported by the superior hypophyseal artery, whereas the neurohypophysis and hypothalamus are supplied by the inferior hypophyseal artery and branches from the circle of Willis. Whatever the explanation, our patient presents an unusual syndrome of isolated cranial diabetes insipidus with preserved anterior pituitary function after postpartum haemorrhage.

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² Baylis PH, Heath DA. The development of a radioimmunoassay for the measurement of human plasma arginine vasopressin. *Clin Endocrinol (Oxf)* 1977;7:91-102.

³ Collins ML, O'Brien P, Cline A. Diabetes insipidus following obstetric shock. *Obstet Gynecol (Suppl)* 1979;53:16s-17s.

⁴ Sheehan HL, Whitehead R. The neurohypophysis in post-partum hypopituitarism. *J Pathol* 1963;85:145-69.

⁵ Merker E, Futterweit W. Postpartum amenorrhoea, diabetes insipidus and galactorrhoea. *Am J Med* 1974;56:555-8.

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Department of Medicine, University of Birmingham, Queen Elizabeth Hospital, Birmingham B15 2TH, and Birmingham and Midland Hospital for Women, Sparkhill, Birmingham B11 4HL

P H BAYLIS, MD, MRCP, lecturer in medicine

J J MILLES, MB, MRCP, research fellow

D R LONDON, DM, FRCP, consultant physician

W R BUTT, D SC, FRCP, honorary professor of endocrinology