A 32 year-old lady had undergone an emergency caesarean section at 29 weeks’ gestation for an anterior uterine rupture with the placenta and fetus outside the uterus, membranes intact. The baby was transferred to the neonatal unit and the uterine dehiscence was repaired. Her estimated blood loss was 1500 ml and her lowest recorded blood pressure was 87/54, similar to her BP in the first trimester. She was transfused 3 units of blood and discharged home with a Hb level of 8.4 g/dl.

On the 7th post-operative day she was re-admitted with a 2-day history of extreme fatigue, severe headache, postural dizziness, vomiting, blurring of peripheral vision on the left with a painful left eye, and failure of lactation. She was clinically dehydrated and pale. Her BP was 98/60 mm Hg, HR was 80 bpm, RR was 14 breaths/min. She was alert and oriented. Neurological examination revealed a left VIth cranial nerve palsy. Visual fields were normal to confrontation with no papilloedema or haemorrhage seen on dilated fundoscopy.

Her serum sodium on admission was 116 mmol/l (last recorded serum sodium on the day of the Caesarean section was 138). Other tests showed potassium of 3.7 mmol/l, ACTH of <5ng/l, LH of <0.5 IU/L, FSH of 1.0 IU/L, TSH of 0.88 mU/l, free T4 of 9.1 pmol/l. The level of PRL was inappropriate for the postpartum period at 375 mU/l. A short Synacthen test was conducted: baseline cortisol of <30 nmol/l, 30 min 284 nmol/l, 60 min 348 nmol/l. Serum osmolality was 240 mOsm/kg (normal 275-295) and urine osmolality was 535 mOsm/kg (normal 150-1200). Her urine sodium was 91 mmol/l. A CT scan showed no abnormality. An MRI brain was consistent with central pituitary infarction. There were no features of intrapituitary haemorrhage or of a tumour.

The diagnosis of Sheehan’s syndrome with hypocortisolism was made. IV fluid resuscitation was initiated with 0.9% saline. She was given hydrocortisone and later levothyroxine. Her VIth cranial nerve palsy resolved within 24 hours, and her hyponatraemia improved to 122 within 48 hours. With this, her salt-wasting resolved, with the urine sodium falling to 38 mmol/l. She was discharged home on 20 mg hydrocortisone daily and levothyroxine 50 mcg.

When reviewed three months later, she was well and had of her own accord reduced her hydrocortisone to 10 mg daily and stopped the levothyroxine. She had had two spontaneous menses. Her BP was 110/70 mm Hg, HR 68 bpm. Laboratory results showed an LH of 2.7 IU/L, FSH of 10.0 IU/L, TSH of 2.6 mU/l, free T4 of 9.9 pmol/l, PRL of 237 mU/l, IGF-I of 12.8 nmol/l (normal 13-64), and sodium of 140 mmol/l. A cortisol day curve 24hrs off hydrocortisone showed resumption of circadian cortisol secretion: 9.35am – 853, 12.00pm – 406, 13.00pm – 223, 16.00pm – 73. A follow-up MRI revealed a markedly atrophic anterior pituitary with a thin rim enhancing normal pituitary. We plan to perform an insulin tolerance test to evaluate her response to hypoglycaemia.

This case therefore illustrates a case of Sheehan’s syndrome occurring in the context of peripartum haemorrhage. This manifested as hyponatraemia with salt wasting due to hypocortisolism. This patient has demonstrated spontaneous recovery of pituitary function within three months. Although the spontaneous recovery of pituitary function after lymphocytic hypophysitis is well described, only a few cases of partial recovery of pituitary function are described after Sheehan’s syndrome.