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Case Report

Pituitary Necrosis Revealed by Central Diabetes Insipidus Secondary to Postpartum Hemorrhagic Shock: A Case Report

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Abstract: Postpartum pituitary necrosis results in Sheehan's syndrome, giving a clinical picture of complete or dissociated panhypopitiutarism. It classically involves the anterior pituitary gland and is rarely associated with central diabetes insipidus. We report the case of a 26 year old primiparous woman admitted to intensive care for haemorrhagic shock secondary to post partum haemorrhage due to cervical tear. After resuscitation and haemodynamic stabilisation, she presented with retro-orbital headaches, an absence of milk production and primary hypotonic polyuria with a negative fluid balance. Fluid restriction without improvement and a positive desmopressin test led to the diagnosis of central diabetes insipidus. As part of the aetiological investigation, magnetic resonance imaging (MRI) showed pituitary necrosis. The presence of central diabetes insipidus in the context of post-partum haemorrhage should raise the possibility of pituitary necrosis, although this association is rare. MRI is a key element in the diagnosis, particularly in the acute phase, as the clinical picture of panhypopuitarism occurs much later.

Keywords: Diabetes insipidus - Sheehan syndrome - pituitary necrosis - pituitary MRI.

INTRODUCTION

Pituitary necrosis of the postpartum is most often part of Sheehan's syndrome. The latter performs a picture of complete or dissociated pituitary insufficiency in women following a hemorrhagic obstetric event. It is a rare condition, difficult to diagnose and often misunderstood [1]. It is estimated that a quarter of women who died within a month of giving birth showed signs of pituitary necrosis [2]. Necrosis classically affects the anterior pituitary gland, making central diabetes insipidus a rare manifestation of this syndrome [3].

We report a case of Sheehan syndrome revealed by central diabetes insipidus during postpartum hemorrhagic shock.

CASE PRESENTATION

A 26-year-old woman, primigest, primiparous with no past medical history was admitted to the obstetric emergency department for a three-hour premature rupture of membranes in a progressive intrauterine monofetal pregnancy of 37 weeks + 3 days not in labor. The patient also had gestational diabetes and moderate preeclampsia. After induction in the delivery room, she presented during an attempt at vaginal delivery, acute fetal distress with fetal bradycardia motivating the performance of a caesarean section.

The postoperative effects were marked by the onset of a state of hemorrhagic shock with tachycardia at 127 cpm, arterial hypotension at 73/35 mmHg, a shock index at 1.7 and oligoanuria associated with genital hemorrhage requiring a valve examination. The operation, performed under general anesthesia, found the presence of blood clots and active bleeding on a repaired cervical tear of about 2 cm. Intraoperatively, the patient benefited from volume expansion, a transfusion of 2 red blood cells and was put on norepinephrine, allowing hemodynamic stability after 40 minutes.

She was transferred to the post-operative intensive care unit, intubated and ventilated under residual sedation with a RASS score of -3, pupils in miosis, hemodynamically stable under norepinephrine 1.92 gamma/kg/min.

The postoperative assessment revealed normocytic normochromic anemia at 6.6 g/dl, thrombocytopenia at 124000/mm3, a prothrombin level

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at 53%, correct renal function with serum creatinine at 13 mg/L and urea at 0.22 g/L.

The intensive care unit had made it possible to stabilize the hemodynamic state, withdrawal from vasopressors and extubation on day 2 of hospitalization.

The course was marked by the occurrence of intermittent retroorbital headaches, an absence of milk build-up. On day 5 of hospitalization, a primary hypotonic polyuria was 7.69 ml/kg/h (urinary density 1.010; urinary osmolarity estimated at 314 mOsm/kgH20) with hypernatremia at 152 mmol/L and a negative fluid balance. The urinary ionogram showed: proteinuria at 1.08 g/24h, natriuria at 309.6 mmol/24h, kaliuria at 33.2 mmol/24h and chloride at 311.65 mmol/24h. Table I shows the evolution of diuresis and biological and urinary parameters.

Table I: Evolution of biological parameters during hospitalization

	DAY 2 Hospitalization	**DAY 5 Hospitalization**	**DAY 8 Hospitalization**
Diuresis ml/24h	2600	12000	4600
Urinary density	ND	1,003	1,010
urinary osmolarity mOsm/kgH2o	ND	282,6	314
Natraemia mmol/L	148	152	134
Kaliémie mmol/L	4,78	3,6	2,4
Chlorémie mmol/L	107	122,3	97,6
Natriurie mmol/24h	ND	309,6	ND
Kaliurie mmol/24h	ND	33,2	ND
Proteinuria g/24h	ND	1,08	ND
ASAT/UI/TOOL UI/L	ND	63/36	ND
PAL UI/L	ND	98,6	ND
Hémoglobine g/dL	6,6	7,1	9,1
TP %	54	76	87
Blood creatinine mg/L	13	7,5	10,5

The diagnostic hypotheses evoked given the context were training polyuria, central or nephrogenic diabetes insipidus in the context of acute hepatic steatosis gravidarum or pituitary necrosis.

Fluid restriction at 1000 ml/24 h was performed and came back negative with persistence of hypotonic polyuria, pointing to diabetes insipidus. An abdominal computed tomography (CT) scan did not show evidence of fatty liver disease (Figure 1). The liver test was also normal. A desmopressin test treatment introduced on day 7 of hospitalization proved positive and led to the retention of central diabetes insipidus.

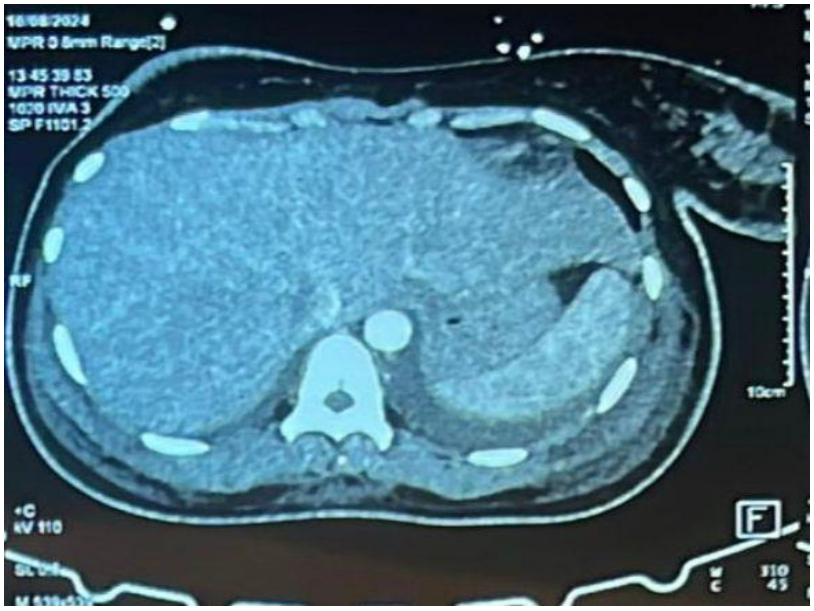

Figure 1: Absence of evidence of fatty liver disease. Normal-sized liver with regular contours, enhanced in mosaic

The etiological research led to the performance of a pituitary magnetic resonance imaging (MRI) at day 9 of hospitalization showing pituitary necrosis with thickening of the pituitary shaft (Figure 2).

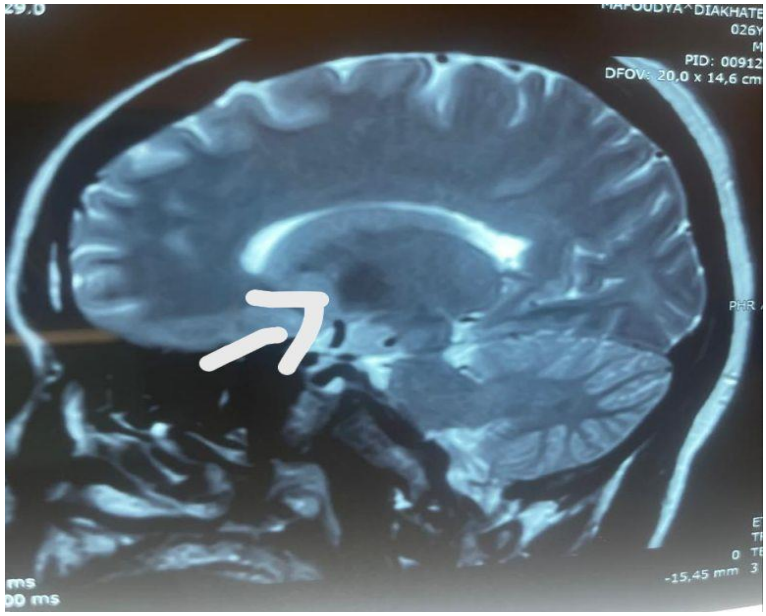

Figure 2: Pituitary MRI showing pituitary necrosis (Pituitary T2 hyposignal) with pituitary shaft thickening

The evolution was favorable on desmopressin with diuresis at 1.5 ml/kg/h, a normal blood ion test. The patient was transferred to the gynecology department and then referred to an endocrinology outpatient for hormonal assessment and subsequent follow-up.

DISCUSSION

The onset of diabetes insipidus in a postpartum context is primarily thought of gestational diabetes insipidus or in the context of acute steatosis gravidarum. This is all the more so since there is preeclampsia in the preparatum, evoking abnormalities related to the interactions between the hypothalamic-pituitary axis, the placenta and the maternal liver [4].

However, other diagnoses must be considered and sought, taking into account the context, clinical signs and diagnostic approach in the case of polyuropolydypsis syndrome. These include Sheehan's syndrome, which is very rarely diagnosed in the acute phase of the picture. The only early sign of this entity, the absence of milk onset indicating the deficiency of the lactotropic lineage, is often neglected [5]. Its diagnosis is often late, in half of the cases, it is not made until ten years after the obstetrical event, before the later onset of a picture of panhypopitiutarism [6].

Several authors note the existence, in reported cases, of isolated retroorbital or frontal headaches, or even of a meningeal syndrome inaugurating the picture of pituitary necrosis [1, 7]. The probable mechanism would be the tensioning and local inflammation of the dura mater bordering the pituitary compartment. In our case, the patient had retroorbital headaches upon waking.

The vulnerability of the pituitary gland to a decrease in its flow, in addition to its great dependence on its vascularization, is linked to hyperplasia of the latter occurring during pregnancy. Indeed, there is a proliferation of lactotropic cells due to the hormonal impregnation linked to pregnancy [2].

In addition, the factors that condition pituitary necrosis during the decrease in its blood flow are the existence of a spasm of the arteries anchored to the gland, the severity of this spasm, its duration and its distribution [7].

Few cases in the literature report the association with diabetes insipidus, a clinical expression of post-pituitary involvement. Authors estimate its prevalence at about 5% [10]. This association would depend on the proportion of necrotic pituitary gland. This is the case of our patient who presented with necrosis of the entire pituitary gland as well as a thickening of the pituitary shaft.

However, polyuria can be associated with pregnancy and persist until more than a month postpartum. It is thought to be related to diabetes insipidus due to a breakdown of vasopressin by placental vasopressinase [8].

For example, in a study comparing the power of urine concentration by fluid restriction test in patients with Sheehan's syndrome compared to a control group, Dejager *et al*., showed that there is a deficit in antidiuretic function in all patients with pituitary necrosis [9].

Antidiuretic hormone is at the heart of the pathophysiology of diabetes insipidus. It is synthesized by the supraoptic and paraventricular nuclei of the hypothalamus. It is released by the postpituitary gland and acts at the level of the collecting ducts by increasing water permeability [4].

In our case, diabetes insipid represented the circumstance of the discovery of pituitary necrosis with a polyuropolydipsic syndrome installed on the third postpartum day. Concordant deadlines are found in the literature. Indeed, in an analysis of 8 cases of association of diabetes insipidus with Sheehan's syndrome, Olmes *et al*., found time to onset between 1 and 19 days [10, 11].

Brain CT, when performed, is most often normal. Magnetic resonance imaging (MRI) may be the test of choice, but there is very little evidence of pituitary necrosis from the postpartum to the acute phase. In a recent observation, an MRI was performed on the sixth day postpartum and showed an intrasellar mass appearing as a homogeneous hypo-signal in T1 and a hyper-signal in T2 [9], suggestive of the diagnosis of hypophysitis. In our observation, the MRI performed on day 9 of hospitalization showed a thickening of the pituitary shaft with necrosis of the pituitary gland.

Endocrinological investigations in the acute phase of pituitary necrosis are poorly documented [4], sometimes not very helpful to the diagnosis.

CONCLUSION

Pituitary necrosis of the postpartum is a complication of postpartum hemorrhage. Its diagnosis is often late at the stage of panhypopituitarism. The presence of hypotonic polyuria in this context should lead to the suggestion and investigation of central diabetes insipidus, which may be the circumstance of discovery. Pituitary MRI is the radiological examination that confirms the diagnosis.

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