Case Study

Sheehan's Syndrome
A Case Report and Literature Review

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SUMMARY
Post-partum pituitary necrosis (Sheehan's syndrome) is a rare complication of post-partum hemorrhage. The diagnosis can be erratic and often delayed. In this case report of Sheehan's syndrome in the post-partum period, the signs were characterized by agalactia, severe hypoglycemia, and low serum levels of thyroid hormones, cortico-adrenal hormones, and gonadotrophin (FSH, LH). The hypophyseal magnetic resonance imaging confirmed the diagnosis of hypopituitarism secondary to pituitary necrosis.

INTRODUCTION
Sheehan's syndrome is a rare but potentially serious postpartum complication. It was first described in 1937 by Sheehan; it is the ischemic necrosis of the pituitary gland secondary to a brutal and extended shock due to obstetric hemorrhage [1].

OBSERVATION
A 40 year old patient, G3P3, with no significant medical or surgical history, was admitted to the intensive care unit with hemorrhagic shock due to postpartum hemorrhage following a normal delivery at home. The clinical examination on arrival confirmed the state of shock: an undetectable blood pressure, a tachycardia of 120 beats/min, cold peripheries, pale conjunctivae and polypnea. Immediate care involved vascular expansion with colloids via a central venous catheter, followed by a transfusion of 5 units of cross matched packed red cells since the haemoglobin level was 5g/dl. The short-term evolution was marked by lactational failure and hypoglycemic crisis (obnubilation, profuse sweats, agitation and convulsions) confirmed by glycemias of 0,4g/l and relieved by the administration of hypertonic glucose solutions. The medium-term evolution was marked by prolonged amenorrhea, fatigue and apathy. In the light of this clinical picture, Sheehan's syndrome was suspected and confirmed by a pituitary and cerebral MRI which showed an empty sella turcica (figure1). Hormone profiles including cortisol, plasma ACTH, thyroid hormones, TSH, FSH, LH and prolactin were all low as a result of panhypopituitarism (cortisol: 40ng/ml, plasma prolactin: 0ng/ml, TSH: 0,05UI/ml, FSH: 0,20mUI/ml, LH: 0,1mUI/ml, T3: 2pmol/l, T4: 4pmol/ml). The treatment is essential hormone replacement: thyroxin 50mg per day, hydrocortisone 40mg per day. The evolution was favorable.

DISCUSSION
Adenohypophyseal ischaemic necrosis following hypoperfusion is the most common cause of adenohypophysal insufficiency. It was first described in 1939 by Sheehan [2]. It occurs classically in postpartum hemorrhage with cardiovascular collapse. The diagnosis can be made reliably in the presence of lactational failure, prolonged amenorrhea and hypoglycaemic crises [3]. However, other signs of adenohypophysal insufficiency are often delayed and subtle leading to the diagnosis being missed. In some cases the pituitary necrosis is only partial and the syndrome can present in atypical and incomplete forms further complicating the diagnostic procedure [4]. Hypopituitarism has several possible etiologies. Apart from adenohypophysal necrosis, other causes are quoted: tumoral, immunological, iatrogenic, traumatic, infectious and genetic [2]. The MRI scan is the investigation of choice, but there is little data illustrating postpartum hypophyseal

necrosis in the acute phase. In case of hypophyseal necrosis, the early MRI highlights a pituitary gland of reduced size with segments of hypersignal in T1 and T2 and hyposignal without contrast. Later, the MRI shows an empty sella turcica following pituitary atrophy. Endocrine investigations during the acute phase of hypophyseal necroses are less well documented [5]. Corticotropin insufficiency could suggest the concept of low plasmatic cortisol, just as urinary free cortisol and ACTH would be low. Thyroid hormone levels are low. The basic blood level of growth hormone would be low [6]. Prolactin levels are important because they are physiologically very high at the end of pregnancy and return to normal approximately 6 weeks postpartum in women who do not breastfeed. An early drop in prolactin levels would therefore suggest adenohypophyseal insufficiency. Dynamic investigations are carried out, making it possible to confirm that target organs are normal and that the fault lies with an absence of activity at the pituitary gland. Once the diagnosis is established, treatment aims to correct life threatening endocrine imbalance: hypoglycaemia and adrenal insufficiency being the most urgent, meriting treatment even in anticipation of laboratory confirmation. Complete hormonal substitution aims to restore normal function in the thyroid, adrenal and ovarian axes. Subsequent pregnancies are achieved using ovarian stimulation techniques [7].

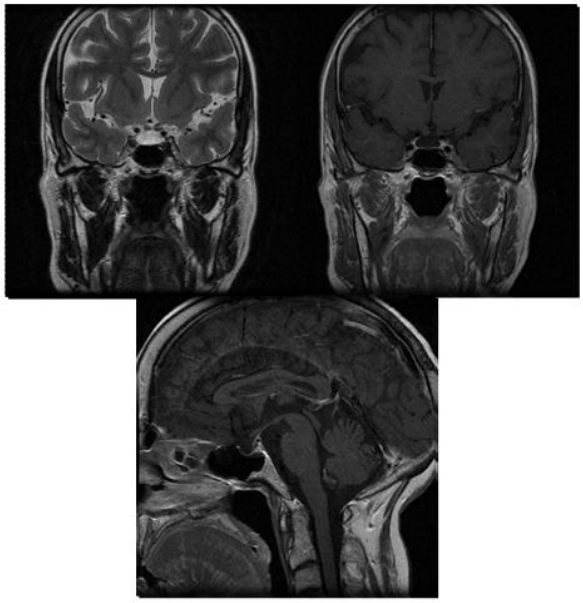

Figure 1: MRI scan showing an empty sella turcica

CONCLUSION

Postpartum pituitary necrosis is a known complication, but it is now rarely seen. Even if postpartum hemorrhage has been well managed, this complication cannot be excluded, and it can be life threatening. It is necessary to consider this diagnosis in all patients having presented with a cardiovascular collapse during childbirth, whatever the cause, and in the presence of the classic signs of pituitary insufficiency.

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