# Pituitary coma revealing Sheehan's syndrome: A case report

**Abstract:**

Introduction:

Postpartum hypopituitarism has been known as Sheehan syndrome since 1937. It is often an insidious revelation but can also be an acute one with coma and collapse, which was the case of our patient reported below.

Clinical case: Patient aged 37, admitted to the intensive care unit in a state of anasarca and coma. In view of her clinical features associated with hyponatremia, acute adrenal insufficiency was suspected. requiring degressive boluses of hydrocortisone hemisuccinate. The diagnosis was supported by biological evidence. Subsequent medical records revealed a haemorrhagic haemorrhagic childbirth dating back 6 years with absence of lactation and progressively incapacitating physical and psychological asthenia. Sheehan's syndrome was suspected in this clinical situation and confirmed on MRI. The patient was put on daily oral replacement of the deficient axes.

Discussion: Myxedema coma is exceptional in Sheehan's syndrome. Severe forms require an initial stay in intensive care, followed by multidisciplinary consultation in order to manage the multiple complications and to introduce hormone replacement therapy.

## Introduction :

Post-partum hypopituitarism has been known as Sheehan's syndrome since 1939, although it was first described in 1914 by Simmonds.

It is defined by necrosis and collapse of the gland during childbirth, generally after haemorrhage.

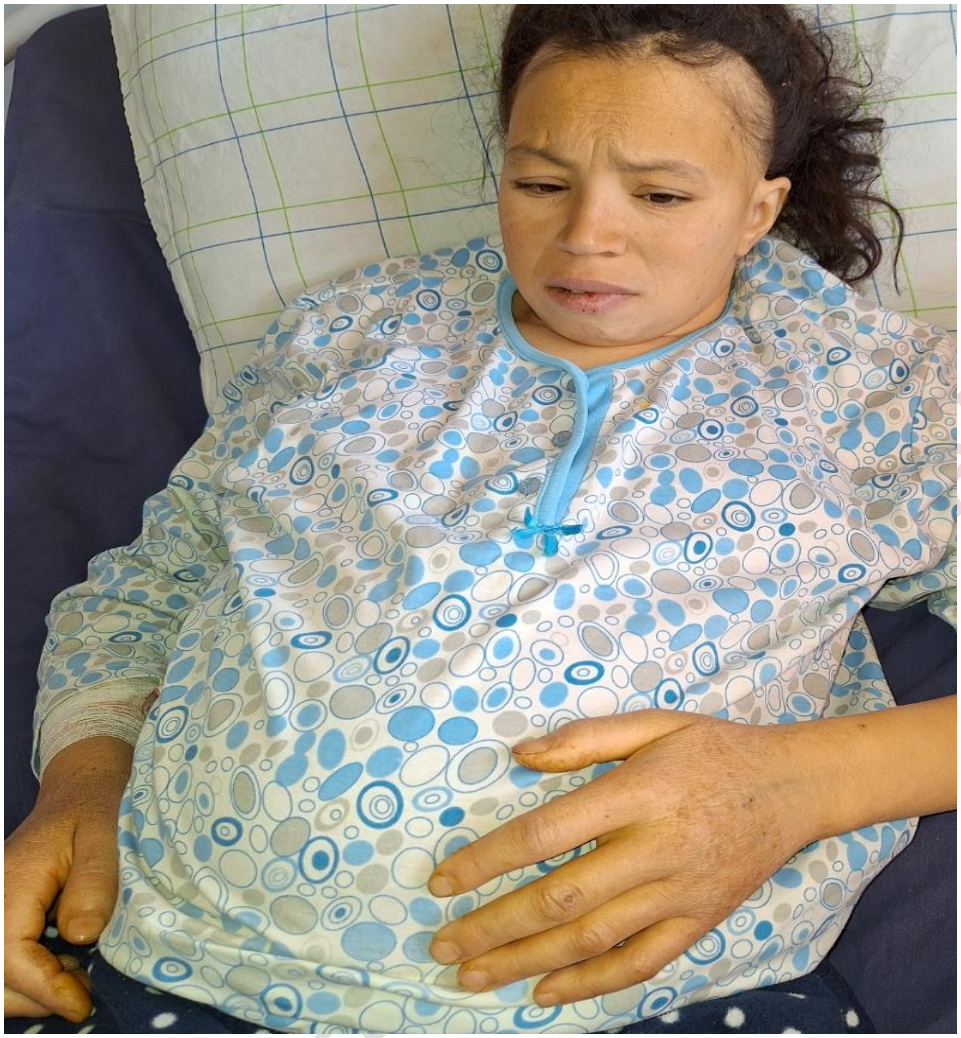
Its presentation varies from one clinical situation to another, and may involve acute pituitary insufficiency with ardiovascular collapse, or more commonly a patient who slowly develops fatigue and rather non-specific symptoms(1,2,3,4).

We report a clinical case in order to highlight this special endocrine condition,

**Case report:**

37-year-old patient was admitted to the emergency intensive care unit in a state of impaired consciousness with a Glasgow score of 8/15, hypoglycaemia at 0.4g/l and arterial hypotension of 60/30 mmhg, initially unresponsive to vasoactive substances.

The examination also revealed pallor areolar depigmentation, palmoplantar caroternoderma and myxedema (Figure N°1).



**Figue N°1: photography of the clinical features of the patient**

Confronted to this clinical picture associated with a hyponatremia of 130 mmol/l on the ionogram, acute adrenal insufficiency was suspected, necessitating degressive boluses of hemisuccinate of hydrocortisone. The diagnosis was supported by corticotropic, gonadotropic and thyroid deficiencies, with TSH: 1.11 uui/l and T4 under 5.14 pmol/l for which she received an initial bolus of 300 ug then 200ug/d by nasogastric tube.

Progress was marked by clinico-biological improvement. Post-treatment investigation revealed a history of haemorrhagic childbirth dating back 6 years with absence of and

progressively disabling physical and mental progressively invalidating physical and psychological asthenia.

After strong suspicion, Sheehan's syndrome syndrome was finally confirmed in view of the hypotrophic appearance of the pituitary gland on MRI (Figure N° 2). On discharge, the patient was put on daily oral substitution of deficient axes.

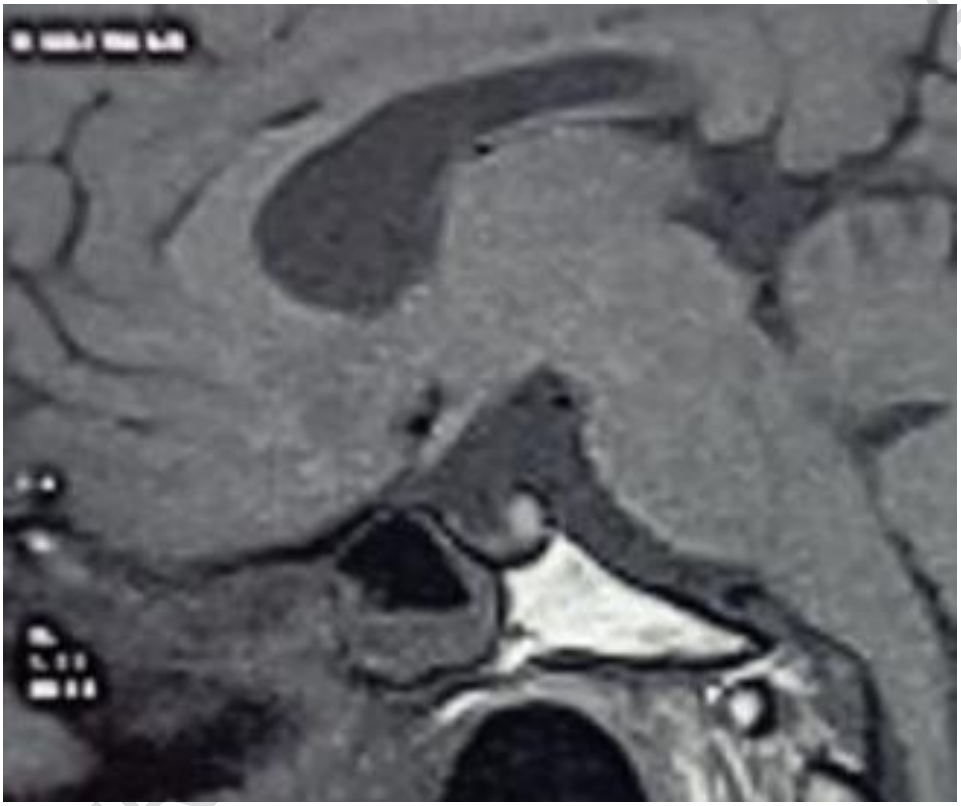


**Figure N°2: MRI of the pituitary in sagittal section**

**Discussion** :

From a pathophysiological point of view, major post-partum haemorrhage (PPH) causes spasm or thrombosis of the pituitary artery, contributing to hypoperfusion and then necrosis of the pituitary cells. During the last quarter of pregnancy, the pituitary gland enlarges with hyperplasia of lactotrophic cells, making it all even more vulnerable to these hypoperfusion

phenomena (4).

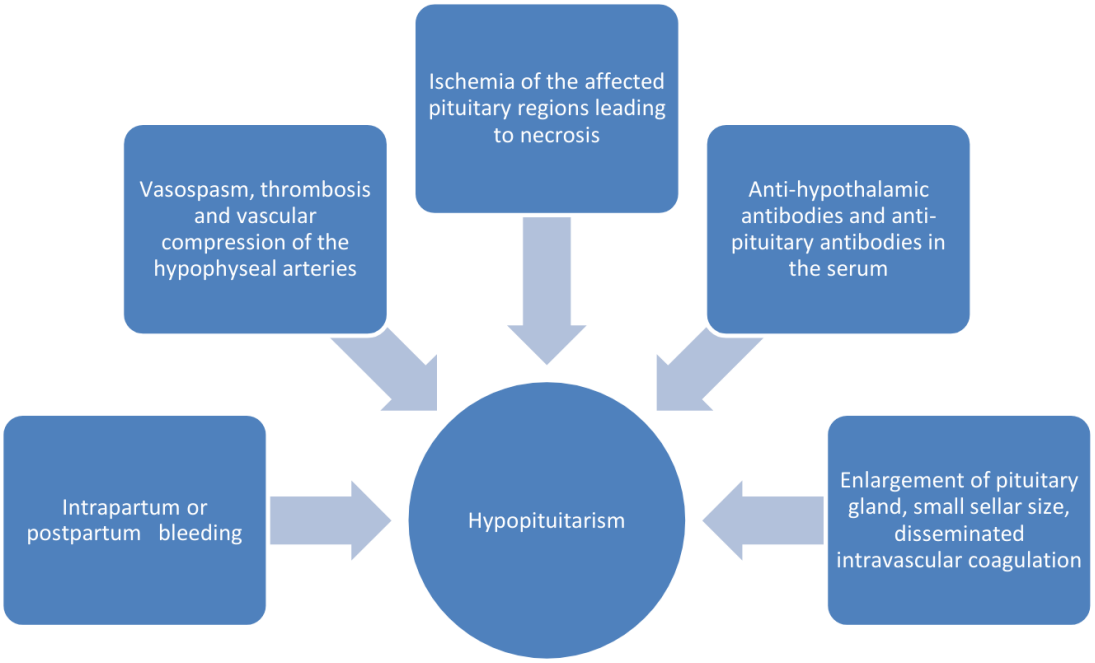



**Figure N°3: sheehan's syndrome pathophysiology (4)**

Sheehan's syndrome usually presents insidiously over a period of weeks or even months, and often remains clinically silent until a stressful event, such as a concomitant infection or trauma, reveals marked symptoms. This prolonged course is due to slow depletion of pituitary hormones. The endocrine organs also have other, albeit less effective, means of coping with the lack of pituitary input (for example, non-ACTH stimulation of cortisol by the adrenal gland or constitutive activation of the TSH receptor at a low level in the thyroid (1,2,3,5).

However, insidious and non-specific clinical pictures may sometimes be marked by psychiatric symptoms such as catatonia, depression, psychosis, etc.

It would also be wise not to overlook the possibility of post-pituitary insufficiency leading to diabetes insipidus (10).

In recent years, sheehan's syndrome has received increasing medical attention, thereby reducing the prevalence of pituitary coma, a secondary condition which is a feature of developing countries.

In order to establish a diagnosis, the pituitary gland should be examined, with cortisol levels being the most important and urgent, and thyroid hormone levels (TSH, FT3 and FT4) should also be measured, followed by FSH, LH, prolactin, oestrogen and then growth hormone. Non-specific biological elements pointing to pituitary insufficiency are also requested, in particular blood ionograms, blood formula counts and renal function tests (9).

As far as imaging is concerned, MRI at a distance from the acute phase following the immediate post-partum period is used to confirm the aetiology and generally shows pituitary hypoplasia or even an empty sellar zone (9,10).

In terms of management, hypopituitary coma presents a degree of delicacy and urgency. It often requires an initial intensive care unit placement to deal with hypovolaemia, hypoglycaemia and iterative hypotension, including rehydration regimens for adrenal acutisation, alternating saline/glucose serum with continuous monitoring (12,13).

This is followed by multidisciplinary consultation, particularly with endocrinologists, to initiate hormone replacement therapy, starting with an intensive supplementation regimen of 300 mg bolus initially, followed by degressive boluses of hydrocortisone hemisuccinates

Substitution of the thyrotropic axis, which increases the body's overall consumption and glucocorticoid requirements, should be delayed for 72 hours after corticotropic substitution (7).

Estrogen- progesterone and GH substitution should be postponed until after patients stabilisation (14).

The mortality rate of hypopituitary coma caused by Sheehan's syndromes has decreased significantly in recent decades with the development of medical care, giving way to a favourable outcome, as in the case of our patient.

**Conclusion:**

Severe comatogenic forms of Sheehan's syndrome, although less and less described, require a multicentric medical effort in order to avoid mortality and to hope for a better clinical improvement.

**Conflict of interest statement**

The authors declare that they have no conflict of interest.

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