Review Article

"THE SILENT DEATH OF THE PITUITARY": PATHOPHYSIOLOGY AND CLINICAL IMPACT OF SHEEHAN'S SYNDROME

Puhizë Osmanaj

Universiteti i Prishtinës

Correspondent E-mail:puhizaosm@gmail.com

ABSTRACT

Sheehan's syndrome, also called postpartum hypopituitarism, is a condition characterized by necrosis and impaired function of the anterior pituitary gland, typically resulting from severe blood loss during or shortly after childbirth. Its low incidence, prolonged course with nonspecific clinical manifestations, and higher prevalence among women in underdeveloped or developing countries often contribute to a delayed diagnosis. This condition is marked by reduced secretion of all anterior pituitary hormones, leading to clinical features such as failure of lactation in the postpartum phase to amenorrhea, and infertility. The diagnosis of Sheehan's syndrome is established through hormonal assays, dynamic stimulation tests, and imaging studies of the pituitary gland. Treatment consists of lifelong hormone replacement therapy.

Keywords. Sheehan's syndrome, postpartum hypopituitarism, pituitary necrosis, pituitary hormones, and empty sella.

"VDEKJA E HESHTUR E HIPOFIZËS": FIZIOPATOLOGJIA DHE NDIKIMI KLINIK I SINDROMËS SHEEHAN

ABSTRAKT

Sindroma e Sheehan-it, e quajtur edhe hipopituitarizëm pas lindjes, është një gjendje e karakterizuar nga nekroza dhe funksioni i dëmtuar i gjëndrës së hipofizës anteriore, që zakonisht rezulton nga humbja e rëndë e gjakut gjatë ose menjëherë pas lindjes së fëmijës.

Incidenca e ulët, ecuria e zgjatur me manifestime klinike jo specifike dhe prevalenca më e lartë tek gratë në vendet e pazhvilluara ose në zhvillim, shpesh kontribuojnë në një diagnozë të vonuar. Kjo gjendje karakterizohet nga sekretimi i zvogëluar i të gjitha hormoneve të hipofizës anteriore, duke çuar në karakteristika klinike të tilla si dështimi i laktacionit në fazën pas lindjes deri në amenorre dhe infertilitet. Diagnoza e sindromës së Sheehan-it vendoset përmes analizave hormonale, testeve dinamike të stimulimit hormonal dhe studimeve imazherike të gjëndrës së hipofizës. Trajtimi konsiston në terapi zëvendësuese hormonale gjatë gjithë jetës.

Fjalë kyçe. sindroma Sheehan, hipopituitarizmi pas lindjes, nekroza e hipofizës, hormonet e hipofizës dhe sella turcica e zbrazët.

INTRODUCTION

The pituitary gland is a classical endocrine gland, in the narrow sense that its special function is the production, release, and storage of hormones (1). Despite being very small and described as a "pea-sized gland", its importance in the organism is significant. It is in a part of the sphenoid bone, called the ''sella turcica", lying under the hypothalamus. Divided into two parts, the anterior and posterior pituitary, the anterior pituitary communicates with the hypothalamus via a specialized network of blood vessels called the hypothalamo-pituitary portal system. In contrast, the posterior pituitary communicates with the hypothalamus via a neural pathway (2). The hypothalamus releases several hormones specific to its connections with the anterior pituitary (Table 1).

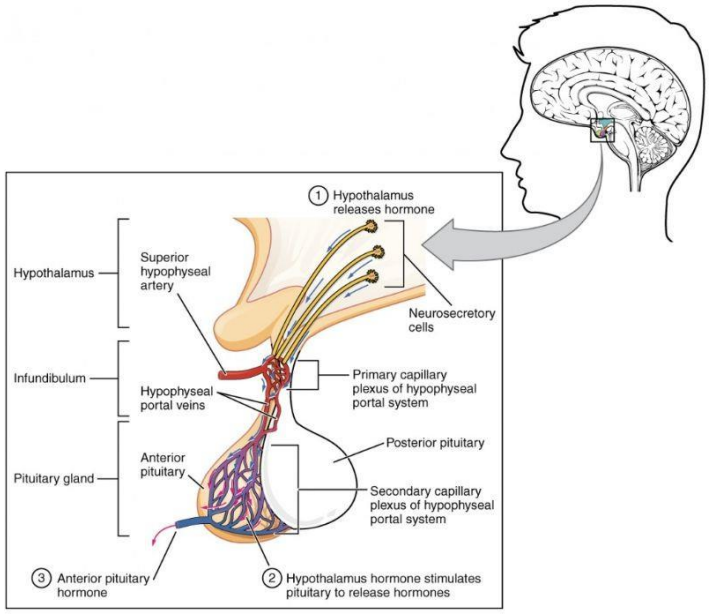

Figure 1. The hypothalamus produces separate hormones that stimulate or inhibit hormone production in the anterior pituitary. Hormones from the hypothalamus reach the anterior pituitary via the hypophyseal port.

Source: Adapted from StatPearls [Internet]. Endocrine Emergencies. Treasure Island (FL): StatPearls Publishing; 2024. Available from https://www.ncbi.nlm.nih.gov/books/NBK595005/ figure/ch9endocrine.F9.4/. Open-access content distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY 4.0) (https:// creativecommons.org/licenses/by/4.0/).

Pituitary gland size increases by approximately 0.08 mm per week during pregnancy. The pituitary gland enlarges throughout pregnancy but typically should not exceed 10 mm during most of this period. Postpartum pituitary size, up to 12 mm, is considered within normal limits (3). Sheehan's syndrome is caused by severe pituitary damage due to ischemic necrosis following significant postpartum hemorrhage (4). First described by Harold Leeming Sheehan in 1937, the condition often remains undiagnosed for decades, leaving patients without appropriate treatment (5).

Table 1. Hormonal signaling from the hypothalamus to target organs via the anterior pituitary.

Hypothalamus	Hormone	Pituitary (Adenohypophysis)	Pituitary Hormone	Target Organ
GnRH	Gonadotropin-Releasing Hormone	Anterior Lobe	FSH/LH	Gonads
CRH	Corticotropin-Releasing Hormone	Anterior Lobe	ACTH	Adrenal Cortex
TRH	Thyrotropin-Releasing Hormone	Anterior Lobe	TSH	Thyroid
PRH	Prolactin-Releasing Hormone	Anterior Lobe	Prolactin	Mammary Gland
GHRH	Growth Hormone-Releasing Hormone	Anterior Lobe	GH	Liver (+ body)

This article presents a review of the existing scientific literature on Sheehan's syndrome, drawing from relevant and reliable studies. For the literature search, major databases such as PubMed, Scopus, and Web of Science were consulted, including publications up to May 2025. Original articles, case reports, clinical studies, and systematic reviews were selected to summarize information on the etiology and pathogenesis of the disease, clinical presentation, diagnosis, differential diagnosis, treatment options, and prognosis. All sources referenced in the paper are cited in the References section.

Epidemiology.

Sheehan's syndrome is an endocrine disorder that is relatively rare in developed countries but remains a significant concern in underdeveloped and developing regions (6). A systematic study conducted at a tertiary care hospital identified Sheehan's syndrome as the leading cause of panhypopituitarism, accounting for 57.14% of cases (7).

Data from several studies indicate an incidence of 5.1 cases per 100,000 births in developed countries (8). In contrast, a study from India found that 3% of women over the age of 20 had

Sheehan's syndrome, with over half having delivered at home (9). Similarly, a study from Nigeria reported 11 cases, with an average age of 35.1 years at the time of diagnosis, around 6.9 years later between the inciting postpartum hemorrhage and diagnosis (10).

Etiology and Pathophysiology

During pregnancy, the pituitary gland undergoes hypertrophy in response to increased functional demands associated with fetal development (11). The primary factor driving its enlargement is the proliferation of lactotroph cells in response to elevated estrogen levels (12). The increase in estrogen during pregnancy comes mainly from the placenta, which converts androgens to estrogens from the adrenal cortex of the mother and fetus through the enzyme aromatase (13).

The enlargement of the pituitary gland during pregnancy is not accompanied by a proportional increase or expansion of its vascular network, which supplies the gland with blood. As a result, any condition that impairs blood flow or reduces oxygen delivery through these vessels places the pituitary at significant risk of ischemia and subsequent necrosis.

Conditions that may reduce perfusion to the anterior pituitary primarily involve severe postpartum hemorrhage but can also include prolonged maternal hypotension even in the absence of active bleeding (14,15).

Complicated births, such as multiple births, placental abnormalities, trauma during birth, uterine atony, and various coagulation disorders, are all factors that can be accompanied by large losses of blood volume, causing severe hypovolemia, and in some cases, ischemia of organs such as the anterior pituitary, leading to the so-called "Sheehan's Syndrome" (Table 2) (16,17).

Due to the nonspecific and gradual onset of symptoms, this disease is frequently diagnosed months or even years after onset; however, certain signs related to impaired hormone production can appear immediately.

Table 2. Risk factors of hemorrhage during labor.

Risk factors of hemorrhage during labor
Multiple pregnancy
Placental abnormalities
Trauma during birth
Uterine atony
Coagulation disorders

Clinical Presentation

Early signs of the disease

- Agalactia (Failure to produce breast milk) - due to lack of prolactin.
- Extreme fatigue, dizziness, and hypotension - due to lack of ACTH, the adrenal glands do not stimulate the production of cortisol, which under normal conditions is essential for maintaining blood pressure, coping with physical stress, and regulating metabolism through gluconeogenesis.
- Amenorrhea (absence of menstruation) results from insufficient FSH and LH levels, which inhibit ovarian follicle maturation, estrogen production, ovulation, and progesterone secretion by the corpus luteum, ultimately leading to anovulation and menstrual cycle cessation.
- Loss of secondary sexual characteristics (such as lack of pubic and axillary hair) - because of the lack of the hormones FSH, LH, through their influence on the production of estrogen and androgens (from the ovaries), and the lack of the hormone ACTH through its influence on the production of androgens (from the adrenal glands).
- Cold, pale, and reduced skin elasticity occurs due to deficiencies in TSH and cortisol, which affect metabolism, peripheral circulation, and connective tissue integrity (18,19,20).

These symptoms are often overlooked due to their nonspecific presentation, mistakenly attributed to the physiological adaptation of the mother's body after childbirth, a situation that leads to the appearance of later signs (Table 3).

Late signs of the disease

- Low libido and inability to get pregnant again - due to the decrease in FSH and LH, which affects the decrease in energy, function, and sexual desire.
- Hypoglycemia - due to the decrease in ACTH and cortisol, respectively, which leads to impaired gluconeogenesis by the liver.
- Pale skin - from lack of ACTH, leading to reduced stimulation of melanin production.
- Mood swings with depression and/or anxiety - from lack of TSH (decreased T3 and T4), lack of FSH and LH (decreased estrogen), and lack of ACTH (decreased cortisol), through their influence on metabolic processes in the brain and neurotransmitters that regulate emotions and stress coping.
- Anemia - because of a lack of TSH and estrogen, which normally stimulate the production of erythropoietin and bone marrow and improve iron absorption.

- Hyponatremia - lack of ACTH, which reduces cortisol, in severe cases can also affect the secondary reduction of aldosterone, thus causing hypotension, hyponatremia, and thirst (21, 22, 23).

Table 3. Early and Late Manifestations of Pituitary Hormone Deficiency.

Early Manifestations	Late Manifestations
Agalactia	Low libido
Extreme fatigue	Infertility
Dizziness	Hypoglycemia
Hypotension	Pale skin
Amenorrhea	Mood swings (depression and/or anxiety)
Loss of secondary sexual characteristics	Anemia
Cold, pale, and reduced skin elasticity	Hyponatremia

Diagnosis

The diagnosis of Sheehan's syndrome is established through a combination of disease history, physical examination, laboratory testing, and brain imaging such as MRI and CT (24).

According to the history of the disease, the patients reported heavy bleeding during or shortly after labor, lack of milk secretion, extreme fatigue, lack of menstrual cycle, decreased libido, and dizziness.

On examination, the skin appears cold, dry, and pale, and there's a lack of axillary and pubic hair, which is sometimes accompanied by a decrease in breast mass.

Laboratory tests reveal decreased levels of all anterior pituitary hormones: ACTH, FSH, LH, TSH, GH, and prolactin, accompanied by reduced cortisol, estrogen, T3, and T4 concentrations. (25). Other signs supporting the diagnosis are anemia, hypoglycemia, and hyponatremia (26).

Stimulation tests

To understand the levels of pituitary function, the so-called stimulation tests are used, which, in a state of normal functioning, stimulate the relevant organs to produce corresponding substances (27). These include the insulin stimulation test, which normally stimulates GH and cortisol; however, in Sheehan's disease, the hormones are absent due to the pituitary's failure to produce them.

TRH test: This test is used to stimulate the release of TSH and prolactin, but in Sheehan's disease, the response to these hormones is absent.

GnRH test: Under normal conditions, it would stimulate FSH and LH; however, in this case, the response is absent (28, 29).

Brain imaging

On postpartum MRI and CT imaging, due to enlargement of the pituitary gland, especially from the proliferation of prolactin-producing cells, the pituitary gland appears enlarged still. Following severe postpartum hemorrhage, the pituitary gland may exhibit inflammation and fluid accumulation. An enlarged appearance on imaging does not indicate increased functional pituitary tissue but rather reflects surrounding edema (30).

In the later phase (>6 months), the pituitary gland suffers a shrinkage due to necrosis, leaving an atrophic remnant on imaging (Fig. 2), called an empty sella or empty pituitary fossa, thus presenting permanent damage to the pituitary gland; the now empty space is filled by CSF, which can be seen on imaging (Fig. 3) (31,32, 33).

MRI is the gold standard, while CT is only used if MRI is contraindicated or unavailable.

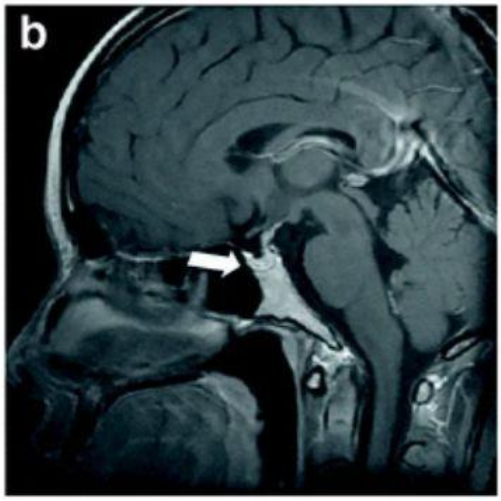

Figure 2. *Diminution in the pituitary gland size, observed 6 months postpartum. The white arrow indicates the pituitary gland*.

Source: Adapted from Matsuzaki S. et al., BMC Pregnancy and Childbirth (2017). Open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY 4.0). DOI: 10.1186/s12884-017-1380-y

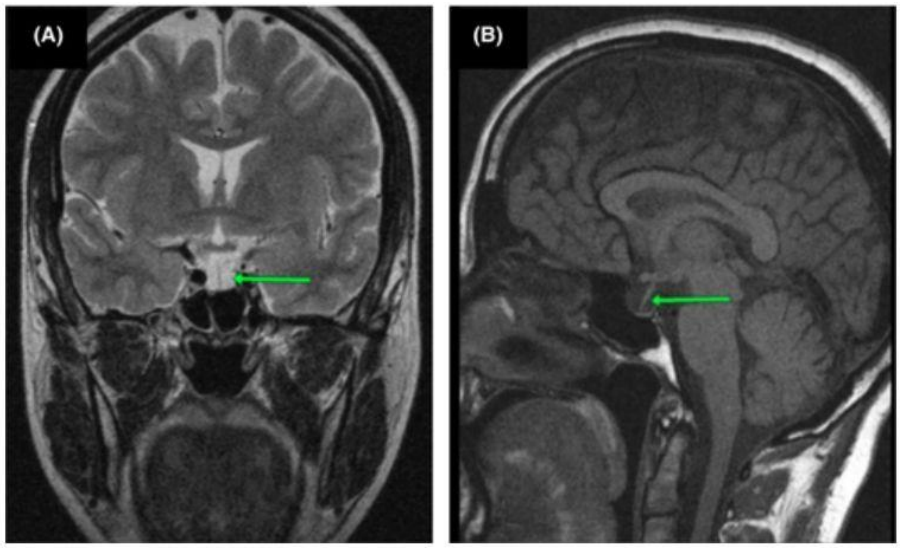

Figure 3*. Magnetic resonance imaging of sella. The arrow shows a CSF-filled sella with a thin rim of pituitary tissue and pituitary stalk, indicating an empty sella. (A) T2 coronal section, (B) T1 sagittal section. CSF, cerebrospinal fluid.*

Source: Adapted from Alamri M, et al. "A rare case of Sheehan's syndrome presenting with hyponatremia and hypoglycemia." Clinical Case Reports (2023). Open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY 4.0). DOI: 10.1002/ccr3.8521

Differential diagnosis

Early-stage differential diagnoses include macroadenomas, cysts, lymphocytic hypophysitis, infiltrative disorders, and pituitary apoplexy. (34)

In the late stages, Sheehan syndrome can be confused with diseases such as primary empty sella syndrome, non-Sheehan hypopituitarism due to radiation, Addison's disease, and postpartum depression (35).

Treatment

Treatment consists of lifelong replacement of deficient pituitary hormones to maintain physiological function (Table 4) (36, 37).

- For ACTH deficiency, corticosteroids such as hydrocortisone or prednisolone are prescribed in a regulated dose.
- For TSH deficiency, T4 (levothyroxine) is prescribed.
- For FSH/LH deficiency, estrogen and progesterone are administered if the patient has not yet reached menopause.

- For GH deficiency, when it is documented via stimulation tests, rhGH/somatropin is given, especially in cases of extreme fatigue, muscle wasting, or decreased bone density. To prevent adrenal crisis, we must make sure that adrenal insufficiency is treated before GH.

Table 4. Treatment of Sheehan's syndrome

Deficient Hormone	Prescribed Medication
ACTH	Corticosteroids
TSH	Levothyroxine
FSH/LH	Estrogen and Progesterone
GH	rhGH/somatropin

Prognosis and outcome

If diagnosed early, Sheehan's syndrome has a very good prognosis with hormone replacement therapy. In case of late detection, patients may face numerous problems resulting from the lack of appropriate hormones, leading to a poor prognosis with possible consequences, including death from adrenal crisis and multiorgan failure.

CONCLUSION

Sheehan's syndrome remains a significant issue in underdeveloped and developing countries due to multiple factors: limited professional awareness, particularly in primary care; poor socio - economic conditions; geographical and financial barriers to essential medical evaluations; restricted access to obstetric care; underreporting of postpartum hemorrhage and the lower socio - cultural status of women, who often perceive the symptoms as normal postpartum changes.

Timely recognition of Sheehan's syndrome, along with appropriate referral and early diagnosis, allows for restoration of the lost hormonal functions and can reduce or even completely prevent long-term complications.

Conflicts of interest: The authors declare that they have no conflicts of interest.

REFERENCES

1. Daniel PM. Anatomy of the hypothalamus and pituitary gland. J Clin Pathol Suppl (Assoc Clin Pathol). 1976;7:1-7. doi: 10.1136/jcp.s1-7.1.1. PMID: 1073162; PMCID: PMC1436118.
2. InformedHealth.org [Internet]. Cologne, Germany: Institute for Quality and Efficiency in Health Care (IQWiG); 2006-. In brief: How does the pituitary gland work? [Updated 2024 May 28]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK279389/
3. Elster AD, Sanders TG, Vines FS, Chen MY. Size and shape of the pituitary gland during pregnancy and post partum: measurement with MR imaging. Radiology. 1991 Nov;181(2):531-5. doi: 10.1148/radiology.181.2.1924800. PMID: 1924800.

4. JEFFCOATE TN. Post-partum necrosis of the pituitary. Ir J Med Sci. 1948 Jun;(270):256-63. doi: 10.1007/BF02956475. PMID: 18866111.
5. Wani AM, Hussain WM, Al Mejally MA, Banjar AA, Ali KS, Khoujah AM, Raja SH, Bafaraj MG, Al Miamini W, Akhtar M. Practice of symptomatic treatment in the era of evidence-based medicine: report of 2 cases of diagnosis of Sheehan's syndrome delayed till eighth decade. BMJ Case Rep. 2010 May 6;2010:bcr09.2009.2276. doi: 10.1136/bcr.09.2009.2276. PMID: 22736728; PMCID: PMC3047277.
6. Keleştimur F. Sheehan's syndrome. Pituitary. 2003;6(4):181-8. doi: 10.1023/b:pitu.0000023425.20854.8e. PMID: 15237929.
7. Mokta J, Ranjan A, Thakur S, Bhawani R, Mokta KK, Sharma JB, Kumar M. Sheehan's Syndrome-The Most Common Cause of Panhypopituitarism at Moderate Altitude: A Sub-Himalayan Study. J Assoc Physicians India. 2017 Dec;65(12):20-23. PMID: 29327517.
8. Kristjansdottir HL, Bodvarsdottir SP, Sigurjonsdottir HA. Sheehan's syndrome in modern times: a nationwide retrospective study in Iceland. Eur J Endocrinol. 2011 Mar;164(3):349-54. doi: 10.1530/EJE-10-1004. Epub 2010 Dec 23. PMID: 21183555.
9. Kumar S, Kumar KVSH. Pituitary Diseases in the Tropics. [Updated 2021 Mar 14]. In: Feingold KR, Ahmed SF, Anawalt B, et al., editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK568567/?utm_source=chatgpt.com
10. Famuyiwa OO, Bella AF, Akanji AO. Sheehan's syndrome in a developing country, Nigeria: a rare disease or problem of diagnosis? East Afr Med J. 1992 Jan;69(1):40-3. PMID: 1628549.
11. Benson JC, Malyuk DF, Madhavan A, Guerin JB, Krecke KN, Little JT, Passe TJ, DeLone DR, Lindell EP, Eckel LJ. Pituitary volume changes in pregnancy and the post-partum period. Neuroradiol J. 2024 Feb;37(1):39-42. doi: 10.1177/19714009231196470. Epub 2023 Aug 17. PMID: 37590100; PMCID: PMC10863577.
12. Stefaneanu L, Kovacs K, Lloyd RV, Scheithauer BW, Young WF Jr, Sano T, Jin L. Pituitary lactotrophs and somatotrophs in pregnancy: a correlative in situ hybridization and immunocytochemical study. Virchows Arch B Cell Pathol Incl Mol Pathol. 1992;62(5):291-6. doi: 10.1007/BF02899695. PMID: 1359702.
13. Alex A, Bhandary E, McGuire KP. Anatomy and Physiology of the Breast during Pregnancy and Lactation. Adv Exp Med Biol. 2020;1252:3-7. doi: 10.1007/978-3-030-41596-9_1. PMID: 32816256.
14. Shivaprasad, C.. Sheehan's syndrome: Newer advances. Indian Journal of Endocrinology and Metabolism 15(Suppl3):p S203-S207, September 2011. | DOI: 10.4103/2230-8210.84869
15. Kilicli, F., Dokmetas, H. S. and Acibucu, F. (2012) 'Sheehan's syndrome', *Gynecological Endocrinology*, 29(4), pp. 292–295. doi: 10.3109/09513590.2012.752454.
16. Feinberg EC, Molitch ME, Endres LK, Peaceman AM. The incidence of Sheehan's syndrome after obstetric hemorrhage. Fertil Steril. 2005 Oct;84(4):975-9. doi: 10.1016/j.fertnstert.2005.04.034. PMID: 16213852.
17. Pattanaungkul S, Chandraprasert S. Pregnancy in Sheehan's syndrome. J Med Assoc Thai. 1989 Jan;72(1):48-51. PMID: 2723567.
18. Mandal, Soumita; Mukhopadhyay, Pradip1; Banerjee, Mainak1; Ghosh, Sujoy1,. Clinical, Endocrine, Metabolic Profile, and Bone Health in Sheehan's Syndrome. Indian Journal of Endocrinology and Metabolism 24(4):p 338-342, Jul–Aug 2020. | DOI: 10.4103/ijem.IJEM_345_20

19. Dökmetaş HS, Kilicli F, Korkmaz S, Yonem O. Characteristic features of 20 patients with Sheehan's syndrome. Gynecol Endocrinol. 2006 May;22(5):279-83. doi: 10.1080/09513590600630504. PMID: 16785150.
20. Matsuzaki S, Endo M, Ueda Y, Mimura K, Kakigano A, Egawa-Takata T, Kumasawa K, Yoshino K, Kimura T. A case of acute Sheehan's syndrome and literature review: a rare but life-threatening complication of postpartum hemorrhage. BMC Pregnancy Childbirth. 2017 Jun 14;17(1):188. doi: 10.1186/s12884-017-1380-y. PMID: 28615049; PMCID: PMC5471854.
21. Melmed S, Polonsky KS, Larsen PR, Kronenberg HM. Williams textbook of endocrinology. Elsevier Health Sciences; 2015 Nov 30.
22. Molitch ME. Pituitary diseases in pregnancy. Semin Perinatol. 1998 Dec;22(6):457-70. doi: 10.1016/s0146-0005(98)80026-8. PMID: 9880116.
23. Agrawal P, Garg R, Agrawal M, Singh MK, Verma U, Chauhan R. Sheehan's Syndrome in India: Clinical Characteristics and Laboratory Evaluation. J Obstet Gynaecol India. 2023 Oct;73(Suppl 1):51-55. doi: 10.1007/s13224-023-01801-8. Epub 2023 Aug 23. PMID: 37916020; PMCID: PMC10616000.
24. Pandey A, Sindagi SM, Singh H, Singhal P, Bansal P, Negi M. A Diagnosis of Sheehan's Syndrome: Better Late Than Never. J Midlife Health. 2024 Apr-Jun;15(2):128-130. doi: 10.4103/jmh.jmh_66_24. Epub 2024 Jul 5. PMID: 39145272; PMCID: PMC11321522.
25. Furnica RM, Gadisseux P, Fernandez C, Dechambre S, Maiter D, Oriot P. Early diagnosis of Sheehan's syndrome. Anaesth Crit Care Pain Med. 2015 Feb;34(1):61-3. doi: 10.1016/j.accpm.2014.07.001. Epub 2015 Mar 5. PMID: 25829318.
26. Ramiandrasoa C, Castinetti F, Raingeard I, Fenichel P, Chabre O, Brue T, Courbière B. Delayed diagnosis of Sheehan's syndrome in a developed country: a retrospective cohort study. Eur J Endocrinol. 2013 Sep 12;169(4):431-8. doi: 10.1530/EJE-13-0279. PMID: 23864341.
27. Petersenn S, Quabbe HJ, Schöfl C, Stalla GK, von Werder K, Buchfelder M. The rational use of pituitary stimulation tests. Dtsch Arztebl Int. 2010 Jun;107(25):437-43. doi: 10.3238/arztebl.2010.0437. Epub 2010 Jun 25. PMID: 20644702; PMCID: PMC2905884.
28. Aguirre Sánchez-Covisa M, Bellido Guerrero D, Jaunsolo Barrenechea MA, et.al., La prueba combinada de triple estímulo (LHRH, TRH, insulina) en la evaluación clínica de la función adenohipofisaria [Combined triple-stimulation test (LHRH, TRH, insulin) in the clinical evaluation of pituitary function]. Rev Clin Esp. 1985 Sep;177(4):165-9. Spanish. PMID: 3934719.
29. Nakai T, Nomura F, Takekoshi K. [Measurements of serum pituitary hormones and dynamic tests to evaluate the pituitary functions]. Nihon Rinsho. 1993 Oct;51(10):2665-72. Japanese. PMID: 8254937.
30. Patel MC, Guneratne N, Haq N, West TE, Weetman AP, Clayton RN. Peripartum hypopituitarism and lymphocytic hypophysitis. QJM. 1995 Aug;88(8):571-80. PMID: 7648244.
31. Scheller TF, Nader S. Magnetic resonance imaging in Sheehan's syndrome: case report and literature review of imaging studies. Endocr Pract. 1997 Mar-Apr;3(2):82-4. doi: 10.4158/EP.3.2.82. PMID: 15251482.
32. Lee HC, Lee EJ, Lee KW, Ahn KJ, Jung TS, Kim DI, Huh KB. Computed tomographic correlation with pituitary function in Sheehan's syndrome. Korean J Intern Med. 1992 Jan;7(1):48-53. doi: 10.3904/kjim.1992.7.1.48. PMID: 1477030; PMCID: PMC4532096.

33. Bakiri F, Bendib SE, Maoui R, Bendib A, Benmiloud M. The sella turcica in Sheehan's syndrome: computerized tomographic study in 54 patients. J Endocrinol Invest. 1991 Mar;14(3):193-6. doi: 10.1007/BF03346787. PMID: 1906495.
34. Dejager S, Gerber S, Foubert L, Turpin G. Sheehan's syndrome: differential diagnosis in the acute phase. J Intern Med. 1998 Sep;244(3):261-6. doi: 10.1046/j.1365-2796.1998.00370.x. PMID: 9747750.
35. Jose M, Amir S, Desai R. Chronic Sheehan's Syndrome - A Differential to be Considered in Clinical Practice in Women with a History of Postpartum Hemorrhage. Cureus. 2019 Dec 4;11(12):e6290. doi: 10.7759/cureus.6290. PMID: 31938584; PMCID: PMC6942501.
36. Soares DV, Spina LD, de Lima Oliveira Brasil RR, Lobo PM, Salles E, Coeli CM, Conceição FL, Vaisman M. Two years of growth hormone replacement therapy in a group of patients with Sheehan's syndrome. Pituitary. 2006;9(2):127-35. doi: 10.1007/s11102-006-9990-9. PMID: 16944044.
37. Karaca Z, Kelestimur F. Sheehan syndrome: a current approach to a dormant disease. Pituitary. 2025 Jan 25;28(1):20. doi: 10.1007/s11102-024-01481-1. PMID: 39863703; PMCID: PMC11762620.