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CASE REPORT

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A Rare Endocrine Emergency in Pregnancy: A Case of Lymphocytic Hypophysitis Mimicking Adrenal Crisis Diagnosed Without Pituitary Imaging

Jyothsna Sree Kommaraju[1,*], Mahrukh Rasheed[2]

[1]Siddhartha Medical College, Gunadala, Vijayawada 520008, Andhra Pradesh, India
[2]Queen's Online Psychotherapy Lab, Kingston, Ontario, Canada

***Corresponding Author:**
Jyothsna Sree Kommaraju
Siddhartha Medical College,
Gunadala, Vijayawada 520008,
Andhra Pradesh, India
Email: kommarajujyothsnasree@gmail.com

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Abstract

Lymphocytic hypophysitis (LH) is a rare autoimmune inflammation of the pituitary gland, typically presenting during late pregnancy or postpartum. It can lead to life-threatening hypopituitarism, but diagnosis is often delayed, especially in settings where MRI and endocrine labs are limited. A 29-year-old primigravida at 34 weeks presented with fatigue, postural hypotension, nausea, and hyponatremia. Initial evaluation suggested adrenal insufficiency. Laboratory studies revealed low morning cortisol, undetectable ACTH, and suppressed TSH and free T4, indicating central adrenal and thyroid insufficiency. Due to limited imaging availability, diagnosis was established clinically and biochemically as lymphocytic hypophysitis. Treatment with hydrocortisone and levothyroxine led to full symptomatic resolution and a healthy term delivery. This case highlights the need for clinical vigilance and early hormone replacement therapy in pregnant patients presenting with unexplained hypotension and electrolyte abnormalities, even in the absence of imaging. Lymphocytic hypophysitis should be a key differential diagnosis in late pregnancy endocrine crises.

1. Introduction

Lymphocytic hypophysitis (LH) is an autoimmune inflammatory disorder of the pituitary gland, predominantly affecting women in late pregnancy or postpartum (Gubbi et al., 2019). It is characterized by lymphocytic infiltration of the pituitary, leading to varying degrees of anterior hypopituitarism (Kluczyński et al., 2019), and occasionally, diabetes insipidus if the posterior pituitary is involved (Iuliano and Laws, 2011). First described by Goudie and Pinkerton in 1962, LH remains a rare but important cause of hypopituitarism (Rao et al., 2016) lymphocytic and xanthomatous forms. Etiopathogenesis and the immunological differences among these is not well characterized. This study aims to explore the immunopathogenesis of granulomatous and lymphocytic forms of hypophysitis. Demographic, clinical, endocrine function and radiological features of 33 histologically confirmed cases of hypophysitis were reviewed. Immunophenotyping of inflammatory component was performed in 13/33 cases. Visual disturbances (46%, with an estimated incidence of 1 in 9 million individuals annually, although likely underreported due to diagnostic challenges.

LH is frequently mistaken for other causes of adrenal insufficiency or sellar masses such as pituitary adenomas (Gubbi et al., 2018). Its hallmark is the abrupt onset of fatigue, hypotension, nausea, and electrolyte abnormalities, usually in the context of pregnancy or autoimmune predisposition (Menotti et al., 2023). While pituitary MRI typically shows symmetrical enlargement or a homogenous mass, access to imaging is often limited in resource-constrained environments (Chaudhary and Bano, 2011). Therefore, hormonal assays and clinical context become critical in establishing a diagnosis and initiating life-saving therapy.

We present a case of LH presenting as adrenal crisis in the third trimester of pregnancy, successfully diagnosed without pituitary imaging and managed effectively with hormone replacement.

2. Case Report

A 29-year-old woman in her first pregnancy presented at 34 weeks of gestation to a district hospital with complaints of profound fatigue, dizziness, salt craving, postural light-headedness, and generalized weakness persisting for five days. She also reported nausea, loss of appetite, and a recent episode of syncope. There was no history of vomiting, fever, or vaginal bleeding. Her prenatal course had been uncomplicated, with no known autoimmune disorders or prior endocrine issues.

Based on the biochemical findings of markedly low morning cortisol and ACTH, central hypothyroidism with suppressed TSH and free T4, and the clinical context of late pregnancy, a provisional diagnosis of lymphocytic hypophysitis presenting as central adrenal and thyroid insufficiency was established. Given the potential for adrenal crisis, immediate therapy was initiated. The patient received intravenous hydrocortisone with a 100 mg bolus, followed by 50 mg every eight hours. Levothyroxine was introduced at a dose of 50 µg per day after ensuring adequate glucocorticoid coverage to avoid precipitating adrenal decompensation. Concurrently, intravenous fluids and electrolyte correction were administered to address hyponatremia and support hemodynamic stabilization.

Following the initiation of hormone replacement and supportive care, the patient exhibited rapid clinical improvement. Her energy levels increased significantly, postural symptoms resolved, and her blood pressure stabilized at 110/70 mmHg. Serum sodium normalized to 135 mmol/L within 48 hours of therapy. She continued to improve throughout the remainder of her pregnancy and successfully delivered a healthy infant via spontaneous vaginal delivery at 39 weeks of gestation. Upon discharge, she was prescribed oral hydrocortisone at a total daily dose of 20 mg, divided into 10 mg in the morning, 5 mg at noon, and 5 mg in the evening. Levothyroxine was increased to 75 µg daily based on clinical response. The patient was counseled regarding the importance of adherence to lifelong hormone replacement therapy and was referred to an endocrinologist for postpartum follow-up and further pituitary function assessment (Table 1, 2 & 3).

3. Discussion

This case emphasizes the need for clinical suspicion and biochemical evaluation in diagnosing lymphocytic hypophysitis, particularly when MRI is unavailable. LH is thought to be mediated by T-cell–driven autoimmune inflammation of the pituitary gland (Graham et al., 2021). In pregnant women, this may be triggered by placental antigens or fetal microchimerism (Jacobsen et al., 2025), resulting in an attack on pituitary tissue.

In previous studies, LH has been reported to cause central adrenal insufficiency and hypothyroidism (Gubbi

Table 1: Patient Demographics and Presentation

Parameter	Detail
Age/Sex	29 / Female
Gravida/Parity	G1P0
Gestational age	34 weeks
Past medical history	None
Presenting symptoms	Fatigue, nausea, dizziness, salt craving, postural hypotension
Vital signs	BP: 85/60 mmHg (supine), HR: 102 bpm, T: 36.5°C

Table 2: Initial Laboratory Evaluation

Test	Result	Reference Range	Interpretation
Serum Sodium	126 mmol/L	135–145 mmol/L	Hyponatremia
Blood Glucose	3.2 mmol/L	4.0–6.0 mmol/L	Hypoglycemia
Morning Cortisol	2.4 µg/dL	6.2–19.4 µg/dL	Low
ACTH	<5 pg/mL	10–60 pg/mL	Suppressed
TSH	0.2 µIU/mL	0.4–4.0 µIU/mL	Low
Free T4	0.5 ng/dL	0.8–1.8 ng/dL	Low
Prolactin	18 ng/mL	5–40 ng/mL	Normal (pregnancy-induced)

Table 3: Hormone Monitoring at Follow-up

Hormone	At 6 weeks postpartum	At 3 months postpartum	Reference Range	Interpretation
Morning Cortisol	14.1 µg/dL (on Rx)	13.5 µg/dL (on Rx)	6.2–19.4 µg/dL	Adequate replacement
ACTH	6 pg/mL	7 pg/mL	10–60 pg/mL	Still suppressed
TSH	2.1 µIU/mL	2.0 µIU/mL	0.4–4.0 µIU/mL	Normal (on Rx)
Free T4	1.3 ng/dL	1.4 ng/dL	0.8–1.8 ng/dL	Target achieved
Prolactin	4 ng/mL	3.5 ng/mL	5–40 ng/mL	Suppressed postpartum

et al., 2018; Torino et al., 2012). Prolactin levels may be preserved initially, especially during pregnancy, due to placental prolactin synthesis (Lopez-Vicchi et al., 2020), but generally fall postpartum.

MRI findings typically include symmetric enlargement of the pituitary with homogeneous enhancement (Caranci et al., 2020). However, in over 30% of cases, imaging may appear normal, particularly if performed after the acute phase. Therefore, hormonal assays remain the cornerstone of diagnosis, especially in settings lacking imaging capabilities.MRI findings typically include symmetric enlargement of the pituitary with homogeneous enhancement (Caranci et al., 2020). However, in over 30% of cases, imaging may appear normal, particularly if performed after the acute phase. Therefore, hormonal assays remain the cornerstone of diagnosis, especially in settings lacking imaging capabilities.

The clinical improvement following corticosteroid and levothyroxine replacement further supports the diagnosis. As with Sheehan's syndrome, lifelong hormone replacement may be necessary due to irreversible pituitary damage.

4. Conclusion

Lymphocytic hypophysitis is a critical yet underrecognized cause of central adrenal insufficiency in pregnancy. This case illustrates that a clinical and biochemical approach can be lifesaving in settings where imaging is unavailable. Prompt steroid replacement, even empirically, should be considered in pregnant women with unexplained hypotension and hyponatremia. Increased awareness and early hormonal testing can prevent morbidity and support safe maternal-fetal outcomes.

Declarations

Ethics approval statement

No ethical approval was required for the current study as it did not deal with any human or animal samples.

Consent to participate

Not applicable

Consent to publish

Not applicable

Data Availability Statement

The data are available from the corresponding author upon reasonable request

Competing Interests

The authors declare that they have no conflict of interest

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Author contribution

Conceptualization, Data curation, Investigation, Formal analysis: J.S.K, Writing—review and editing: M.R. All authors have read and agreed to the published version of the manuscript

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