Asian J Biol Life Sci., 2025; 14(3):806-809.
https://ajbls.com

Case Report

Clinical Diagnosis of Empty Sella Syndrome: A Case Report from Meghalaya

Kathrina Marbaniang[1], Ethan Van Lal Rinngheta[2], Rimiki Challam[3], Barry Cooper Hynniewta[4,*], Kyrshan Giri Lynrah[5], Lumlang Buhphang[6]

[1]Department of General Medicine, Shillong Civil Hospital, Shillong, Meghalaya, INDIA.
[2]School of Applied Sciences, REVA University, Bengaluru, Karnataka, INDIA.
[3]Department of Radiology, Shillong Civil Hospital, Shillong, Meghalaya, INDIA.
[4]Department of Clinical Embryology, MOMSOON Fertility and IVF Centre, Bengaluru, Karnataka, INDIA.
[5]Department of General Medicine, North-Eastern Indira Gandhi Regional Institute of Health and Medical Sciences (NEIGRIHMS), Shillong, Meghalaya, INDIA.
[6]Department of Anaesthesiology, Shillong Civil Hospital, Shillong, Meghalaya, INDIA.

ABSTRACT

Empty Sella Syndrome is a significant problem that can impair hormonal function in patients and is frequently misdiagnosed due to its common asymptomatic nature. A case study of a 60-year-old patient from Meghalaya with recurrent hypoglycaemia and hypothyroidism showed, through laboratory and imaging investigations, the presence of empty sella syndrome. Substantial data from clinical studies adds to the diagnosis of empty sella syndrome, however its particular aetiology remains a subject of considerable speculative interest. The assessment of fundamental serum investigations frequently results in the misdiagnosis of this illness, highlighting the need for targeted awareness programs for non-endocrinologists. Hormonal assessments of FSH, fT3, and fT4 must be accurately recorded for the appropriate diagnosis of empty sella syndrome prior to radiological studies. Corticosteroids, HRT and thyroxine supplementation are the first line of treatment.

Keywords: Empty sella syndrome, Endocrine hormones, Hypoglycaemia, Hypopituitarism, Hypothyroidism, Psychogenic.

Correspondence:
Dr. Barry Cooper Hynniewta
Department of Clinical Embryology, MOMSOON Fertility and IVF Centre, Bengaluru, Karnataka, INDIA.
Email: barrycooperhynniewta@gmail.com

Received: 12-09-2025;
Revised: 29-10-2025;
Accepted: 04-12-2025.

INTRODUCTION

Pituitary gland compression and flattening result from the subarachnoid space herniating into the sella turcica, a condition known as Empty Sella Syndrome (ESS) or arachnoidocele. The incidence of this uncommon illness ranges from 5.5% to 12% of autopsy cases and up to 12% in patients having neuroimaging.[1] With a 4-5:1 sex ratio, it is more prevalent in females.[2] About 70% of patients with Idiopathic Intracranial Hypertension (IIH) have an empty sella.[1] Hormonal deficits of various degrees can result from pituitary parenchymal compression and pituitary stalk involvement. These might result in a range of clinical presentations, frequently leading the family to believe that the symptoms are psychogenic, delaying necessary consultations or referrals. We document a middle-aged woman who had a history of numerous hospital stays and outpatient consultations who was properly evaluated and found to have ESS. Since treatment is frequently straightforward and highly successful, diagnosis is crucial.

PES has been reported to be associated with obesity and hypertension, particularly in females.[3] During pregnancy and lactation, the pituitary gland's size increases as its volume might double, causing the sella turcica to enlarge.[4] Primary Empty Sella (PES) Syndrome is typically asymptomatic and can be an accidental radiological result. Rather, the picture of "Primary Empty Sella Syndrome" is created when PES is linked to endocrine, neurological, ophthalmologic, and mental symptoms (produced by the aforesaid anatomic modification).[5]

It is not always easy to distinguish symptoms and biochemical findings that are the result of the empty sella from those that are discovered incidentally and are only the cause of a medical referral because the clinical picture in individuals with PES is frequently rather complex. As a result, people with PES may have the same symptom as a direct result or incidentally.[6] At the very least, Prolactin (PRL) dynamics vary between PES-affected patients before and after menopause because estrogen-mediated dopamine activation is likely of a lower magnitude in postmenopausal women. As a result, PRL response to Thyroid Releasing Hormone

OPEN ACCESS
ScienScript

DOI: 10.5530/ajbls.20250074

Publishing Partner : ScienScript Digital. [www.scienscript.com.sg]

(TRH) is frequently higher in postmenopausal PES patients than in normal subjects, and it is comparable to that of normal women in premenopausal PES patients.[7]

CASE REPORT

A 60-year-old Indian female of the Khasi tribes of Meghalaya presented to the Medicine Department with complaints of loose motion and hypoglycaemia. Previous histories of Hypoglycaemia were recorded and was admitted several times in the past one year. Her repeated hospitalisation had made relatives consider her as being psychogenic. Patient had a history of multiple pregnancy in which she was admitted in a tertiary care hospital during her deliveries due to postpartum haemorrhage. Blood transfusion was given accordingly. At the time of admission, the patient was administered IV fluids but was unconscious. Blood pressure was recorded 90/60 mmHg, pulse rate 87 bpm. Laboratory investigation was done as shown in Table 1.

Biochemical investigations reveal normal TSH but low T3T4 suggestive of secondary hypothyroidism. Low FSH and LH levels were suggestive of hypopituitarism and serum electrolytes (Sodium and Potassium ion levels were low). USG whole abdomen study was normal. CT scan showed inconclusive results and was further suggested for MRI scan.

Imaging with MRI showed that there was intrasellar herniation of suprasellar subarachnoid with enlargement of sella turcica was noted. Fluid with cerebrospinal fluid signals intensity was seen within the sella. Pituitary gland is poorly visualized and appears thin out at the posterior aspects of the sella. Diffused cerebral atrophy was seen suggestive of empty sella syndrome with diffused cerebral atrophy (Figure 1).

Wysolone (5 mg) was the initial steroid dose given to the patient; it has since been reduced to 2.5 mg. The patient experienced hypoglycaemia symptoms again a few days after the endocrinologist stopped the steroid, therefore the tapering dosage of steroids was resumed.

DISCUSSION

The clinical presentation and treatment of a PES patient were examined in this study. Due to the fluctuation of hormone levels ranging from low to high, the clinical picture of individuals with PES is highly complex. It is unclear whether the symptoms are caused by PES or are coincidental because the causation mechanism is unknown. Depending on the pituitary stalk compression by CSF or intracranial pressure, the incidence of endocrine disorders associated with PES varies greatly, ranging from normal pituitary function to specific categories of pituitary failure. The lack of established criteria for the diagnosis and treatment of PES, aside from a few thorough reviews detailing the clinical diagnosis and therapy, has led to non-endocrinologists treating PES cases and hormones being under evaluated.[8] The results can indicate subpar hormonal profile evaluation and inadequate case referrals to endocrinologists. According to a recent study, despite the fact that about 42% of the patients had symptoms and varied clinical features, one of the important findings of the other studies is the glaring under-evaluation of hormonal measurement.[9] According to research, women who have had at least one successful pregnancy in their physiological history are more likely to have PES. However, comparison with a control group would be required to show how pregnancy contributes to the pathophysiology of PES.[10] The patient did not receive any previous endocrine evaluation because it was believed to be psychogenic, however, on evaluation under our care, PES was discovered at a later stage during radiological imaging of brain studies. Steroid medications were administered along with

Table 1: Laboratory Investigation.

Investigations	Values	Reference Range
CBC		
Hb g/dL	12.1	12 - 15
WBC count (/μL)	7.9×10^3	$(4\text{-}10) \times 10^3$
Platelet count (/μL)	220×10^3	$(150\text{-}400) \times 10^3$
Serum laboratory tests		
Na^+ (mmol/L)	133	135 - 148
K^+ (mmol/L)	3.4	3.5 - 5.3
TSH (μIU/mL)	1.62	0.34 - 5.6
T3 (ng/mL)	0.31	0.87 - 1.78
T4 (μg/dL)	0.59	4.82 - 15.65
Cl^- (mg/dL)	95	90 - 107
FT4 (ng/dL)	<0.3	0.5 - 1.4
FSH (mIU/mL)	8.57	16.74.8-113.59 (post-menopausal)
LH (Miu/mL)	2.65	10.87 - 58.64 (post-menopausal)
Prolactin (ng/mL)	2.44	2 - 29
Urea (mg/dL)	17	15 - 39
Creatinine (mg/dL)	0.6	0.6 - 1.3
Others		
HbA_{1C} (%)	5.7	<5.7
Vitamin D (OH) (ng/mL)	33.5	30 - 100
Urine C/S	*Pseudomonas* spp	

CBC- Complete Blood Count, Hb - Hemoglobulin, WBC - White Blood Cell, Na^+ - Sodium ion, K^+ - Potassium ion, Cl^- - Chloride ion, TSH - Thyroid Stimulating Hormone, T3 - Triiodothyronine, T4 - Thyroxine, FT4 - Free Thyroxine, FSH - Follicle Stimulating Hormone, LH - Luteinizing Hormone, HbA_{1C} - Glycated Haemoglobin, Vitamin D (OH) - 25-hydroxyvitamin D, Urine C/S - Urine Culture and Sensitivity.

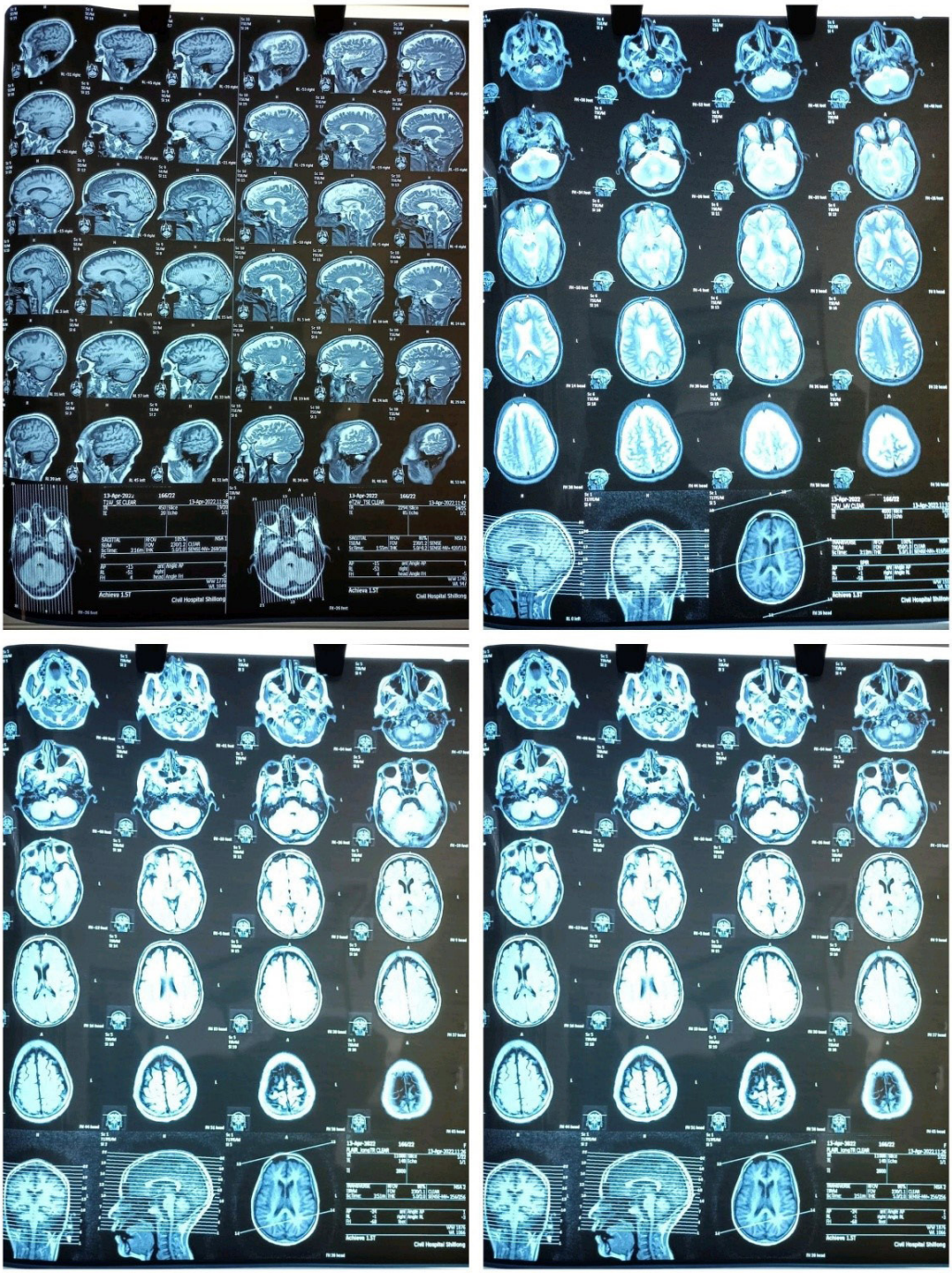

Figure 1: Axial T2W1 shows sella was expanded filled with CSF and infundibulum seen at the centre transversing the CSF, sagittal T1T2 weighted imaging, CSF filled sella with the gland seen pushed inferiority.

thyroid hormone supplementation. Once the patient's condition is stabilized, hormonal replacement therapy can be an adjunct therapy with follow-up with the endocrinologist which can improve symptoms and quality of life.

CONCLUSION

This case emphasizes the need for careful research and precise interpretation. Measurements of the entire Thyroid function profile (FT3FT4 and TSH) are necessary to avoid misdiagnosing dysfunctional thyroidism. Serum FSH levels below the menopausal range in patients with early menopause may aid in the diagnosis of hypogonadism in rare circumstances. Although hypopituitarism is difficult to diagnose, it is easily treated with corticosteroids, thyroxine, and hormonal replacement therapy, which can save the lives of patients that are diagnosed with this syndrome. Awareness programme for general physicians and other medical doctors on PES can promote early diagnosis in case of shortage of endocrinologists.

ABBREVIATIONS

ESS: Empty Sella Syndrome; **IIH:** Idiopathic Intracranial Hypertension; **PES:** Primary Empty Sella; **PRL:** Prolactin; **TRH:** Thyroid Releasing Hormone; **FSH:** Follicle Stimulating Hormone; **fT3:** free Triiodothyronine; **fT4:** free Thyroxine; **HRT:** Hormone Replacement Therapy; **IV:** Intravenous; **bpm:** beats per minute; **mmHg:** millimeters of mercury; **TSH:** Thyroid Stimulating Hormone; **T3:** Triiodothyronine; **T4:** Thyroxine; **LH:** Luteinizing Hormone; **USG:** Ultrasonography; **CT:** Computed Tomography; **MRI:** Magnetic Resonance Imaging; **CSF:** Cerebrospinal Fluid; **FT3FT4:** Free Triiodothyronine and Free Thyroxine; **AI:** Artificial Intelligence.

CONFLICT OF INTEREST

The authors declare that there was no conflict of interest.

ETHICAL APPROVAL

Institutional review port approval is not required.

DECLARATION OF PATIENT CONSENT

Institutional review board approval was not required. Institutional head approval was received.

USE OF ARTIFICIAL INTELLIGENCE (AI)

The authors confirm that there was no use of Artificial Intelligence (AI) - assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

REFERENCES

1. Chiloiro S, Giampietro A, Bianchi A, Tartaglione T, Capobianco A, Anile C, De Marinis L:Diagnosis of endocrine disease: primary empty sella: a comprehensive review. Eur J Endocrinol. 2017;177:R275-R285. 10.1530/EJE-17-0505.
2. Brodsky MC, Vaphiades M: Magnetic resonance imaging in pseudotumor cerebri. Ophthalmology. 1998;105:1686-93. 10.1016/S0161-6420(98)99039-X.
3. Brisman R, Huges JEO, Mount LA. Endocrine function in nineteen patients with empty sella syndrome Journal of Clinical Endocrinology & Metabolism, 1969;34:570-3.
4. Neelon FA, Goree JA, Lebovitz HE: The primary empty sella: Clinical and radiographic characteristics and endocrine function. Medicine 1973;52:73-92.
5. De Marinis L, Bonadonna S, Bianchi A, Maira G, Giustina A. Primary empty sella. Journal of Clinical Endocrinology & Metabolism 2005;90:5471-7.
6. Maira G, Anile C & Mangiola A. Primary empty sella syndrome in a series of 142 patients. Journal of Neurosurery 2005;103(5):831-6.
7. De Marinis L., Mancini A., D'amico C., Sambo P., Passeri M., Zuppi P., *et al.* Prolactin dynamics and cyrcadian rythm in postmenopausal primary empty sella: correlation with intracranial pressure. In Neuroendocrine system and aging, 1986; 365-70. Edn. Vezzadini P., Facchini A. & Labò G. Eurage
8. Chiloiro S, Giampietro A, Bianchi A, Tartaglione T, Capobianco A, Anile C, De Marinis L. Primary empty Sella: a comprehensive review. Eur J Endocrinol. 2017;177:275-85.
9. S Chiloiro, A Giampietro, A Bianchi, T Tartaglione, A Capobianco, C Anile, *et al*, Diagnosis of Endocrine Disease: Primary empty sella: a comprehensive review, European Journal of Endocrinology, 2017;(177)6:R275-R285, https://doi.org/10.1530/EJE-17-0505.
10. Komatsu M, Kondo T, Yamanouchi K, Yokokawa N, Ichikawa K, Ishihara M, *et al.* Antipituitary antibodies in patients with the primary empty sella syndrome. J Clin Endocrinol Metab, 1988;67:633-8.

Cite this article: Marbaniang K, Rinngheta EVL, Challam R, Hynniewta BC, Lynrah KG, Buhphang L. Clinical Diagnosis of Empty Sella Syndrome: A Case Report from Meghalaya. Asian J Biol Life Sci. 2025;14(3):806-9.