# Panhypopituitarism Caused by Empty Sella Syndrome in a Male Patient: A Case Report

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## Abstract

Background: Empty sella syndrome (ESS) is a rare condition characterized by herniation of cerebrospinal fluid into the sella turcica, resulting in compression of the pituitary gland. Although sometimes asymptomatic, ESS may lead to varying degrees of pituitary hormone deficiencies, including panhypopituitarism.
Methods: A 43-year-old male who was referred to the endocrinology clinic for evaluation of persistently low free thyroxine (T4) levels despite normal thyroid-stimulating hormone (TSH). The patient presented with chronic fatigue. Hormone assays and an electrolyte panel were requested alongside MRI pituitary.
Results: Laboratory investigations revealed secondary hypothyroidism, secondary adrenal insufficiency, hypogonadotropic hypogonadism, low insulin-like growth factor 1 (IGF-1), and hyperprolactinemia, which is consistent with panhypopituitarism. Pituitary MRI demonstrated empty sella syndrome. A DEXA scan was requested which revealed osteopenia. The patient was started on hydrocortisone replacement therapy, followed by thyroxine, testosterone, and cabergoline. At three-month follow-up, the patient reported symptomatic relief with normal laboratory results.
Conclusion: This case demonstrates that panhypopituitarism caused by primary empty sella syndrome can present with subtle symptoms. This stresses the importance of pituitary evaluation and MRI in diagnosing and guiding treatment in patients with hormonal abnormalities.

## Case Background

Empty sella syndrome (ESS) is a rare disorder in which the sella turcica appears “empty” on MRI or CT scan. The sella turcica is a depression in the sphenoid bone of the skull that normally houses the pituitary gland. The appearance of being empty is due to the herniation of cerebrospinal fluid (CSF) into the sellar space which results in the compression and in some cases displacement or damage of the pituitary gland(1). Moreover, involvement of the pituitary stalk, the connection between the pituitary gland and the hypothalamus in brain, could disrupt hormonal signalling and result in hormonal insufficiencies (2). When the pituitary gets compressed or damaged in ESS, patients may present with hormonal deficiencies and some cases develop panhypopituitarism where the pituitary gland fails to produce most or all of the hormones it usually secretes (3,4).

ESS can be classified into two types based on aetiology. Primary ESS is when there is a congenital or idiopathic defect in the diaphragma sella and/or an increase in CSF pressure causing the CSF to herniate into the sella turcica which compresses the pituitary gland (1,2). On the other hand, secondary ESS results from acquired pituitary damage such as radiation, tumours, or infarction (e.g. Sheehan’s syndrome) which leads to gland atrophy (1,2).
ESS manifests variably, in many cases especially cases of primary ESS, patients remain asymptomatic and are discovered incidentally on imaging (1,3).

While in other cases, patients may present with symptoms like headache, visual disturbances, and fatigability (1-3). Moreover ESS has a reported prevalence ranging from 8% to 35% and shows a marked female predominance with reported female-to-male ratio of approximately 5:1 (2,7).

## Case Details

A 43-year-old male was referred to the endocrinology department after repeated laboratory results of normal thyroid stimulating hormone (TSH) but low free T4. Despite the abnormal free T4 levels, initial reassurance was provided by his GP based on his normal TSH results. The patient only complained of chronic fatigue but otherwise felt well and did not perceive himself to have significant health concerns. On examination, the patient was found to have a low-normal blood pressure of 103/65 mmHg with no other remarkable findings.

A full hormonal assay was requested which showed TSH of 1.8 mIU/L (ref 0.4-4.0 mIU/L), free T4 of 0.5 ng/dL (ref 0.8-1.8 ng/dL), prolactin of 30 ng/mL (ref 2-18 ng/mL), total testosterone of 198 ng/dL (ref 300-1000 ng/dL), follicle stimulating hormone (FSH) of 0.8 IU/L (ref 1.5-12.4 IU/L), luteinising hormone (LH) of 0.7 IU/L (ref 1.7-8.6 IU/L), morning adrenocorticotropic hormone (ACTH) of 3 pg/mL (ref 7-63 pg/mL), morning cortisol of 2.8 g/dL (ref 5-25 g/dL), insulin-like growth factor (IGF-1) of 55 ng/mL (ref 94-284 ng/mL). In addition, an electrolyte panel was requested which revealed normal results. Furthermore, MRI pituitary was requested which revealed a total empty sella and the patient was diagnosed with panhypopituitarism caused by primary empty sella syndrome. A DEXA scan was also requested which revealed osteopenia. The patient was referred to the ophthalmologist to rule out visual field abnormalities, which were confirmed to be normal.

The patient was first started on hydrocortisone replacement therapy followed by thyroxine, testosterone and cabergoline (Dostinex). After a 3-month follow up visit, the patient showed marked symptomatic relief and laboratory tests showed normal results.

## Discussion

ESS is frequently diagnosed incidentally from radiological imaging, however it may be associated with clinically significant pituitary dysfunction (1,3). In this case, TSH remained repeatedly normal which masked the underlying pituitary pathology and delayed the patient's diagnosis. Furthermore, the presence of chronic fatigue and low-normal blood pressure prompted further endocrine evaluation which ultimately revealed multiple pituitary hormone deficiencies.

Hormonal assay in this patient showed inappropriately normal TSH despite low free T4 which suggests secondary hypothyroidism. Moreover, elevated prolactin is compatible with stalk involvement as it reduces the normal dopamine inhibition on prolactin (4,6). Low total testosterone, FSH and LH are also consistent with hypogonadotropic hypogonadism, which affects bone density causing osteopenia. Additionally, low morning ACTH and cortisol suggests secondary adrenal insufficiency, explaining the patient's low-normal blood pressure from cortisol deficiency. Low IGF-1 for the patients age also suggests growth hormone deficiency. Moreover, normal levels of electrolytes and no symptoms of diabetes insipidus localises the damage to the anterior pituitary (4). This multi-axis pituitary failure constitutes panhypopituitarism secondary to primary ESS which was confirmed by the pituitary MRI. The strong female predominance of ESS makes this male presentation noteworthy, highlighting ESS as an underrecognized cause of lethargy despite patient-reported wellness. This emphasises the importance of maintaining a high index of suspicion of central causes of hypothyroidism and performing comprehensive pituitary evaluation when thyroid function tests are discordant or clinical features are unexplained (4).

Similar cases have been reported in literature. Ngo EM reported a female referred for abnormal routine thyroid function tests and was found to have primary empty sella presenting with anterior hypopituitarism; similar to this case the patient showed subtle symptoms but was found to have multi-axis failure.

The sequence of hormone replacement is critical in managing hypopituitarism. This patient has established secondary adrenal insufficiency. Given that thyroxine increases metabolic rate and cortisol clearance, if the patient is started on thyroid hormone therapy without adequate glucocorticoid coverage, this could precipitate adrenal crisis and coma due to increased cortisol clearance (*5*). Accordingly, the patient was first started on hydrocortisone replacement therapy then was started on testosterone, thyroxine, and cabergoline. Three-month follow-up showed marked symptomatic relief with normalized laboratory findings overall treatment success.

## Conclusion

This case emphasises the critical need for diagnostic vigilance across genders despite the strong female predominance observed in empty sella syndrome. Panhypopituitarism secondary to ESS can present with subtle symptoms like fatigue and the resulting hormonal deficiencies may be masked by normal TSH. This underscores the need for comprehensive endocrine evaluation and pituitary MRI to diagnose and prevent life-threatening complications through appropriate hormone replacement.


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