

## CASE REPORT

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# Empty sella and reversible central adrenal insufficiency in treated primary hypothyroidism

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## ABSTRACT

**Introduction:** Primary hypothyroidism can be complicated by pituitary-related sequelae. Along with a few other reports, we documented pituitary dysfunction with empty sella in primary hypothyroidism; however, the spontaneous reversibility of the hypopituitarism is rarely reported. We aimed to describe a rare complication of primary hypothyroidism and to emphasize the importance of monitoring such complications.

**Case Report:** We report a previously healthy 59-year-old man who presented four years earlier with weight gain of several months duration. The investigations revealed primary hypothyroidism with thyroxine 5.2 pmol/L (7.8–16) and thyroid stimulating hormone >100 uIU/L (0.27–4.2). He responded well to the thyroxine replacement as he became asymptomatic with normalization of thyroid stimulating hormone (TSH). After a few years, the patient presented with lethargy and postural hypotension despite euthyroidism. The basal and stimulated cortisol levels were low 73 (185–624) and 185 nmol/L respectively, while the adrenocorticotrophic hormone was inappropriately normal at 16.1 pg/mL (10.00–46.00). All the other pituitary hormones were normal. Magnetic resonance imaging showed partial empty sella. The patient preferred conservative management. Subsequently, he showed progressive clinical and hormonal improvement. As scarcely

reported in the literature, primary hypothyroidism can be complicated by the development of empty sella, which can further develop pituitary endocrinopathies. Central adrenal insufficiency has been reported more often than other empty sella-related pituitary endocrinopathies, which might need long-term therapy.

**Conclusion:** We documented that empty sella and the associated hypopituitarism might complicate the primary hypothyroidism. This report encourages monitoring, and managing these complications in longer-term follow-up.

**Keywords:** Adrenal insufficiency, Empty sella syndrome, Hypothyroidism, Primary

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## INTRODUCTION

Primary hypothyroidism (PH) is a common problem in medical practice. The clinical course of compensated primary hypothyroidism can be rarely hindered by serious pituitary-related sequelae which complicate the management. Therefore, monitoring these complications including the pituitary function dynamics is advised to adjust the management plan accordingly.

Our case presented with primary hypothyroidism and responded well to thyroxine treatment as evidenced by clinical improvement and normalization of TSH. Later, the patient developed reversible central adrenal insufficiency (AI) coincidentally with partial empty sella (ES). The coincidence of the central AI and the partial

ES in treated primary hypothyroidism suggests the development of pituitary pathology which is related to the primary hypothyroidism concerning its underlying etiology, pathophysiology, or treatment.

Several pituitary-related conditions have been documented in the literature, to be associated with primary hypothyroidism. Pituitary hyperplasia secondary to primary hypothyroidism has been reported in the literature in both adults and children [1]. As pituitary hyperplasia regresses after thyroxine replacement, few case reports documented the development of empty sella [2]. Additionally, pituitary dysfunction and empty sella can be induced by an autoimmune process that causes autoimmune hypophysitis along with primary hypothyroidism [3]. Within the context of primary hypothyroidism, Idiopathic intracranial hypertension (IIH) can lead to empty sella and endocrine deficiencies [4]. Pituitary endocrinopathies can further complicate the course of treatment of primary hypothyroidism and empty sella. This case report replicated the scenario of some similar published reports, in the underlying pathophysiology and outcome, however, unlike the other cases, the pituitary dysfunction in our case was transient and did not necessitate hormonal replacement, which uncovers the dynamic pituitary function changes and emphasizes their careful monitoring in the course of treatment of primary hypothyroidism.

## CASE REPORT

Our case is a 59-year-old man who was unknown to have any medical illnesses and was referred from a primary care center for the management of primary hypothyroidism. He presented with weight gain, hypersomnia, lethargy, constipation, easy fatigability, and depression for several months duration; however, he described a good exercise tolerance. The patient has no relevant medical, surgical, family, or drug history. He is neither a smoker nor an ethanol consumer. The physical examination (P/E) revealed a body mass index (BMI) of 32.4, blood pressure (BP) of 106/73 mmHg, and regular pulse of 67 bpm. The patient was lethargic and had dry skin. The thyroid examination did not reveal any abnormalities.

His initial investigations revealed high total cholesterol of 6.38 mmol/L (N: < 5.2 mmol/L), high triglycerides of 2.5 mmol/L (TG: < 1.7), high initial erythrocyte sedimentation rate of 24 mm/h (0–15), low thyroxine level of 5.2 pmol/L (7.8–16 pmol/L), and a high thyroid stimulating hormone of >100 uIU/L (0.27–4.2 uIU/L), which confirmed primary hypothyroidism (Table 1). Anti-thyroid peroxidase antibodies (anti-TPO AB) titer was high at the level of: 1761.8 IU/mL (0–75) which is consistent with an autoimmune origin of primary hypothyroidism. Ultrasound of the thyroid has not been requested as thyroid examination did not reveal abnormalities. He was started on thyroxine with an initial dose of 50 mcg once a day for one week increased to 100

mcg thereafter and responded dramatically concerning his symptoms, improvement in his biochemistry, and normalization of TSH level (Table 1). Around three years later, the patient complained of lethargy, generalized weakness, occasional dizziness, poor erections, and irritability despite maintaining euthyroidism. Physical examination revealed a BP of 117/97 mmHg with postural drop. The investigations showed persistently high levels of platelets of  $472 \times 10^9$  (130–430) and a constant level of eosinophil count  $0.3 \times 10^9$  (0.02–0.5). Further workup included investigations of the adrenal axis and using the “chemiluminescent immunoassay,” the morning cortisol was found to be 73 nmol/L (185–624), which revealed adrenal insufficiency. The insulin tolerance test (ITT) at that time showed a subnormal response of cortisol (peak of 290 nmol/L) with an inappropriately normal adrenocorticotrophic hormone level of 16.1 pg/mL (10.00–46.00) (Table 1) which suggested central adrenal insufficiency. The anti-21-hydroxylase antibodies (anti-21-OHase AB) were negative which makes autoimmune primary AI a remote possibility. Growth hormone (GH) response was normal in the ITT. Short synacthen test (SST) revealed better cortisol response, which might be another clue to the central origin of adrenal insufficiency. The borderline cortisol response in SST might suggest the chronicity of adrenocorticotrophic hormone (ACTH) deficiency. No abdominal imaging for the adrenal gland has been requested. The testosterone and gonadotropins were at the lower limit of the normal range (Table 1). Magnetic resonance imaging (MRI) of sella which was requested after documentation of central adrenal insufficiency showed partial ES with a nodule more likely representing a small intraventricular sub-ependymoma (Figure 1A and B). Despite the symptoms, the patient thought that he could manage “naturally” and refused corticosteroid treatment. Repeated ITT, SST, and gonadal function tests after four years showed overall improvement except for the basal cortisol (Table 1) (Figure 2). During this period of clinical improvement, the patient had a temporary attack of blindness and was diagnosed with glaucoma which responded partially

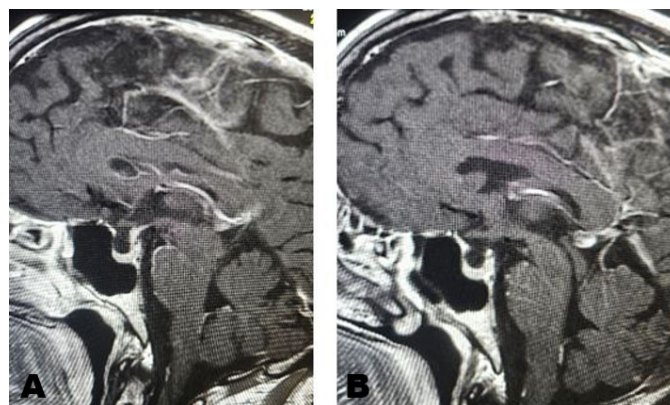


Figure 1: (A) Post-gadolinium-enhanced midline sagittal images reveal prominent CSF within the sella representing partial ES. (B) Right para sagittal image reveals non-enhancing intraventricular nodule.

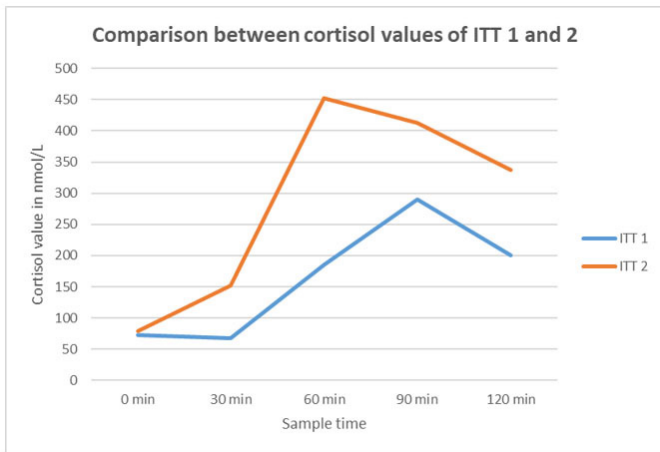


Figure 2: Comparison between the cortisol values in the corresponding sample of the initial (1) and the latest (2) ITT.

to treatment. In a subsequent visit, the BP was 132/90 mmHg with no postural hypotension. The patient felt improvement in his general condition but still complained of weight gain.

**DISCUSSION**

In this report, we presented a case of severe primary hypothyroidism (PH) which was found to have partial empty sella with central adrenal insufficiency after the attainment of euthyroidism by thyroxine replacement. Insulin tolerance test-stimulated cortisol and ACTH levels supported the central origin of adrenal insufficiency. Another indirect clue of AI is the eosinophil count which exceeded 3% of the total white blood cell (WBC). The combination of primary hypothyroidism,

Table 1: Illustration of biochemical investigations and hormonal profile over 9-year follow-up period

6	5	4	3	2	1	
13.8	13.9	15.3	13.9	15.4	15.6	Hb
8.17	8.06	8.6	7.04	7.4	9.05	WBC
0.3	0.3	0.2	0.3	0.1	0.3	Eosinophils
435	389	435	371	352	472	Platelets
					24	ESR
5.2	5.1	5.8	5.5	4.8	5	Glu
142	141	140	143	142	140	Na
	3.96	3.98	4.18	4.1	4.26	K
5.88	6.58	5.57		6.07	6.38	T chol
	3.08	2.89		1.72	2.5	TG
11.2	10.2	11.9	6.5	13.2	5.58	T4
4.81	0.834	0.681	34.6	0.51	>100	TSH
79			73, 67, 185, 290, 200			Cortisol (ITT 1)
79, 152, 453, 413.337						Cortisol (ITT 2)
			0.04, 0.05, 14.6, 20.33, 4.59			GH (ITT 1)
0.07, 2.02, 28.7, 12.5, 5.22						GH (ITT 2)
			183, 405, 330			SST 1 cortisol
62, 403, 491						SST 2 cortisol
11.4	15.11	11.02	8.1			T
2.4	2.2	2.1	1.7			LH
1.9	1.2	1.5	1.2			FSH
168	190	185	488			Prolactin

Hemoglobin (Hg): 13–17 g/L; White blood count (WBC): 4.5–5.5×10<sup>12</sup>/L; Eosinophils #: 0.02–0.5×10<sup>9</sup>/L. Platelets (Plt): 130–430×10<sup>9</sup>/L. Erythrocytes sedimentation rate (ESR): 0–15 mm/h. Glucose (Glu): 3.9–6.1 mmol/L; Na: 136–149 mmol/L; K: 3.5–5.1 mmol/L; Total cholesterol (T chol): <5.2 mmol/L; Triglycerides (TG): <1.7 mmol/L; Thyroxine (T4): 7.8–16 pmol/L; Thyroid stimulating hormone (TSH): 0.27–4.2 uIU/L; Cortisol: 185–624 nmol/L; Growth hormone (GH): 0.01–2.9 mIU/L; Adrenocorticotropic hormone (ACTH): 10.00–46.00 pg/mL; Testosterone: 6.1–27.1 nmol/L; Luteinizing hormone (LH): 1.24–8.62 IU/L; Follicular stimulating hormone (FSH): 1.27–19.26 IU/L; Prolactin: 57–281 mIU/L.

empty sella, and pituitary dysfunction can be explained in several ways. Pituitary hyperplasia accounts for 25–81% of hypothyroidism cases, especially in long-term primary hypothyroidism [2, 5]. We consider the partial ES in our case to be developed after involution of the presumptive hypothyroidism-induced pituitary hyperplasia, as the reported duration of hypothyroidism (a few months) could be sufficient to develop pituitary hyperplasia or might be even longer than what history of symptoms revealed before presentation. This condition has been reported in the literature in several studies [6]. Shimono et al. demonstrated that hypothyroid patients had significantly larger pituitary on MRI, in comparison to healthy euthyroid volunteers within 3–5 weeks of induction of hypothyroid state [7]. In 1851, Niépce et al. first reported cases of hypothyroidism characterized by pituitary hyperplasia; since then, there have been many related reports. A continuous lack of negative thyroid hormone feedback has been found to cause excessive thyrotropin-releasing hormone (TRH) secretion and stimulates thyroid gland development and enlargement [8]. Khawaja et al. reported that in 70% of thyroid hyperplasia, the TSH levels were  $\geq 50$   $\mu\text{IU/mL}$  [9]. In Khawaj’s study, regression of pituitary hyperplasia has been reported in 85% of patients who had a follow-up MRI. Thyroid hormone replacement has caused pituitary size regression in several studies. Most patients tend to respond anywhere between four weeks to three months, with documented regression in almost all cases. In one study, thyroxine replacement therapy brought TSH level within the normal range and prolactin came down simultaneously, as lactotrophs and/or thyrotrophs shrunk significantly in a short time as a result of the removed overstimulation on the pituitary gland [10]. After thyroxine treatment, the pituitary gland regresses in size and can result in empty sella. Kelestimur et al. reported a patient with primary hypothyroidism and an enlarged pituitary, who received thyroxine therapy,

the treatment resulted in regression of the enlarged pituitary with the development of simultaneous partial empty sella [11]. In the study of Stephens [12], the ES was revealed after four months. This phenomenon has been documented in another study [13]. In Stephen’s study [12], pituitary hypertrophy was not documented before the confirmation of the presence of ES, which has been found after several weeks of thyroxine therapy, however, the preceding period of nine years of uncontrolled primary hypothyroidism might involve the development of “feed-back” pituitary hypertrophy. In our case, the MRI was requested after around three years of euthyroidism, therefore, we cannot confirm the temporal relationship between these components, yet, can suggest a possible existence of pituitary hyperplasia and a potential role of thyroxine treatment.

Idiopathic intracranial hypertension (IIH) is associated with many endocrine diseases including hypothyroidism and thyroxine therapy [4]. At the presentation of our case, IIH was not suspected, however, the subsequent development of closed-angle glaucoma complicated by an attack of blindness could point to this possibility, as IIH has been associated with several ophthalmological abnormalities [14]. Another possible mechanism resulting in the empty sella within the context of primary hypothyroidism might be autoimmune hypophysitis which has been considered to include some of the classic cases of Sheehan’s syndrome [15]. The presence of anti-pituitary antibodies (AB) has been reported in cases of pituitary dysfunction [16]. Anti-pituitary antibody testing is not available in our laboratories. In several studies, autoimmune hypophysitis was found to end up with empty sella [17]. An illustrative table of the postulated risk factors of the development of empty sella within the context of primary hypothyroidism is provided (Table 2). Empty sella is commonly associated with normal pituitary functions; however, it can result in hypopituitarism. In a few reported cases, chronic

Table 2: Postulated risk factors of the development of empty sella within the context of primary hypothyroidism

Risk factor	Clue in the case	Correlation to PH	Correlation to ES	Literature evidence (Refs.)
High TSH level	>100 mIU/L	Associated with pituitary hypertrophy		[9]
Pituitary hypertrophy	High TSH level	Associations between PH and pituitary hypertrophy	ES is reported following regression of pituitary hypertrophy after treatment	[6–8]
Thyroxine replacement	50 mcg/D was initiated for 1 week and incremented to 100 mcg/D and increased accordingly till euthyroidism (in 6 weeks)		Rapid correction of PH was postulated to lead to ES	[4]
AH	Anti-TPO AB: 1761.8 IU/mL (0–75)	High titers are associated with the presence of anti-pituitary AB	AH has been reported to precede the development of empty sella (demonstrated in a mouse model)	[17]
IIH	Closed-angle glaucoma and the subsequent attack of bilateral blindness	Associated with a PH and full spectrum of visual complications	Postulated as a driving factor for the development of empty sella	[14]

Abbreviations: PH: primary hypothyroidism; ES: empty sella; AI: autoimmune hypophysitis; IIH: idiopathic intracranial hypertension.

untreated primary hypothyroidism with pituitary hyperplasia has caused long-term sequelae, including ESS and panhypopituitarism [18, 19]. In Stephens's case report [12], the patient developed secondary adrenal insufficiency as a consequence of the development of empty sella in a primary hypothyroidism case. In other cases of primary hypothyroidism, empty sella was reported in association with gonadotropin deficiency [19]. In Besci's study, up to 57% of all patients had growth hormone (GH) deficiency [20]. In other case reports empty sella has ended up in panhypopituitarism [19]. In our case, GH response was normal, which might reflect the extent of "emptiness," as, unlike our case, in Besci's study and another study most of the cases were complete empty sella [21]. Unlike the few reported cases, our case's sella-related endocrinopathies were transient and did not necessitate hormonal replacement therapy. Our patient had spontaneous improvement of the central adrenal insufficiency. The patient denied any previous corticosteroids or narcotic uses. Similarly, the gonadal functions have increased significantly from low normal levels concurrently with the improvement of adrenal function. The reversibility of the pituitary hormonal deficits is not unique to our case [22]. It can be explained by resolving hypophysitis or the disappearance of previously existing hypophysitis-related antibodies documented in three cases by Mori et al. [23]. Spontaneous resolution of hypophysitis has been documented in other studies [24]. The dynamics of testosterone and gonadotropins were parallel to that of adrenal function. Gonadotropin secretion is affected by cortisol insufficiency, in addition to thyroid function in some studies [25]. In Stephens's case [12], as it was iatrogenic, non-immune hypothyroidism, the ACTH deficiency might suggest a functional association between adrenal and thyroid function and more broadly, between other hypothalamus and pituitary hormones. Hashimoto et al. demonstrated that the cortisol insufficiency in isolated ACTH deficiency may affect the augmentation of gonadotropins, and the luteinizing hormone (LH) and follicular stimulating hormone (FSH) responses to gonadotropin-releasing hormone (GnRH) were enhanced in 20% and 15%, respectively [26]. Therefore in our case, it is postulated that the testosterone and gonadotropins represented a reciprocal relationship between ACTH cortisol and gonadotropins.

The endocrine processes in the current clinical setting are complex and dynamic, as primary hypothyroidism is commonly encountered, serious complications should be anticipated and the extended follow-up of the pituitary functions after treatment is recommended to monitor the changes in the potentially transient endocrine deficiencies. This report is influenced by several limitations, which interfere with our interpretations and weaken the drawn conclusions. These limitations include a lack of baseline pituitary MRI for comparison, unavailability of pituitary antibodies test, and loss of

patient follow-up for prolonged durations for various reasons including COVID-19 lockdown.

## CONCLUSION

Serious complications of primary hypothyroidism include the development of empty sella and hypopituitarism. This report raises awareness of the risk of this coincidence and calls for extended follow-up to screen for these complications, reevaluate the pituitary functions after replacement therapy, and adjust the management plan accordingly.

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### Author Contributions

Marwa MS Al-Qudheeb – Conception of the work, Design of the work, Analysis of data, Interpretation of

data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Hasan Ali Al-Tarrah – Acquisition of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Fayez Al-Azmi – Acquisition of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Shaikh Mehraj – Analysis of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

### Guarantor of Submission

The corresponding author is the guarantor of submission.

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### Consent Statement

Written informed consent was obtained from the patient for publication of this article.

### Conflict of Interest

Authors declare no conflict of interest.

### Data Availability

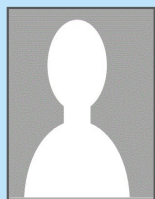
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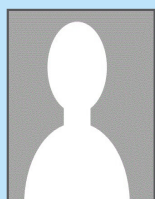
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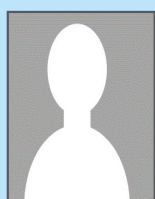
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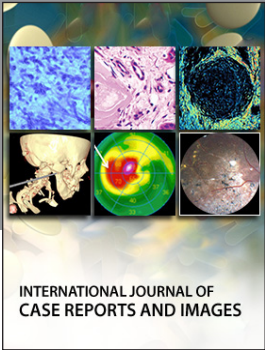
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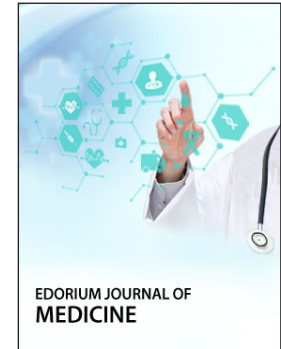
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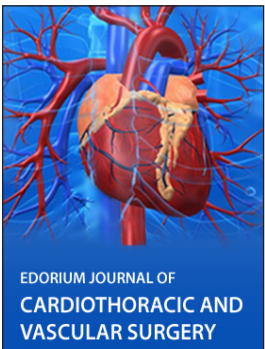
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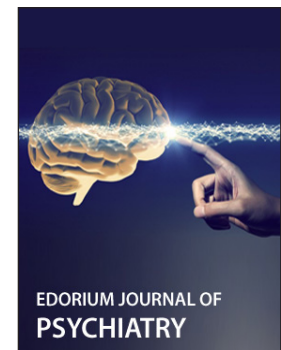
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