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CASE REPORT

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Hypopituitarism presenting with cardiovascular manifestations: a case report

Xiaowen Zhen[1], Yufang Li[1], Chenglong Ren[1] and Mengmei Li[1*]

Abstract

Hypopituitarism is a rare condition that can be particularly challenging to diagnose in individuals aged 65 and older. It is characterized by a reduced production of one or more hormones by the pituitary gland, resulting in a deficiency of the hormones that normally regulate various bodily functions. While hypopituitarism can affect multiple systems in the body, it is uncommon for it to present with cardiovascular symptoms. This rarity often leads to the condition being overlooked in clinical practice. Therefore, healthcare professionals must maintain a high level of suspicion for hypopituitarism to ensure timely diagnosis and appropriate management. In this study, we present a case of hypopituitarism caused by a tumor in the sellar region, which uniquely manifested with cardiovascular symptoms.

Keywords Hypopituitarism, Rathke cleft cyst, Glucocorticoid replacement therapy, Central hypothyroidism, Central hypoadrenalism

Introduction

Hypopituitarism refers to a condition characterized by a decrease or complete cessation of the production of one or more hormones by the pituitary gland. In older adults, the presentation of hypopituitarism can be atypical, with symptoms affecting various bodily systems. Depending on whether the condition impacts the anterior or posterior part of the pituitary, different hormones and their corresponding target glands may be affected, leading to a range of clinical symptoms. Anterior hypopituitarism results in deficiencies of growth hormone, adrenocorticotropic hormone, thyroid-stimulating hormone, and gonadotropins, while posterior hypopituitarism typically causes a deficiency in arginine vasopressin [1].

In rare instances, individuals with anterior hypopituitarism may exhibit cardiovascular symptoms, complicating the diagnostic process. Cardiovascular issues, including congestive heart failure, have been reported as potential manifestations of hypopituitarism, as highlighted in a specific case report [2, 3]. Other studies have described cardiac tamponade and pericardial effusion as rare but possible presentations [2]. Hormone replacement therapy, when appropriately administered, often leads to significant clinical improvement [2]. Known risk factors for cardiovascular disease in these patients include dyslipidemia, endothelial dysfunction, and vascular damage from radiation exposure [4].

The causes of anterior hypopituitarism include tumors, inflammation, infiltrative lesions, trauma, and radiation exposure affecting the hypothalamic-pituitary region [5]. The most common cause is non-functioning pituitary macroadenomas (≥10 mm) [5]. Macroadenomas may exert mass effects, manifesting as visual field abnormalities, headaches, and/or hypopituitarism [6]. Pituitary adenomas are classified into two categories: functioning and non-functioning. Functioning adenomas secrete excess hormones, such as prolactin, growth hormone,

*Correspondence:
Mengmei Li
sjogen@163.com
[1] Department of Emergency, Qingdao Central Hospital, University of Health and Rehabilitation Sciences, Qingdao, China

corticotropin, or thyrotropin, while non-functioning adenomas do not secrete hormones [6]. Rathke's cleft cysts (RCCs) are benign, sellar and/or suprasellar lesions that originate from remnants of Rathke's pouch [7]. When symptomatic, RCCs may cause headaches, hormonal imbalances, and occasionally visual disturbances. Surgical removal, typically via an endonasal transsphenoidal approach with microscopic or endoscopic techniques, is the preferred treatment for symptomatic RCCs [8]. This intervention can effectively alleviate symptoms such as headaches and visual disturbances and, in some cases, improve hormonal imbalances [8].

In this study, we report the case of an elderly female patient who presented with vomiting and heart failure with preserved ejection fraction(HFpEF). The diagnosis of hypopituitarism was confirmed, with the underlying cause identified as Rathke's cleft cysts in the sellar and suprasellar regions.

Case presentation

A 75-year-old female was admitted to the emergency department (ED) due to dizziness, headache, nausea, and vomiting that had persisted for one year. The patient had been previously healthy, with no significant family history relevant to her current condition. She had no history of medication use. On examination, the patient appeared apathetic and was afebrile. Her blood pressure was 128/72 mmHg, and her heart rate was 77 bpm, both of which were within normal limits. The electrocardiogram (Fig. 1) revealed sinus arrhythmia, low QRS waves in the limb leads, and flat T waves across all leads. Her volume status was considered normal, given her normal blood pressure, heart rate, and consciousness. Liver,

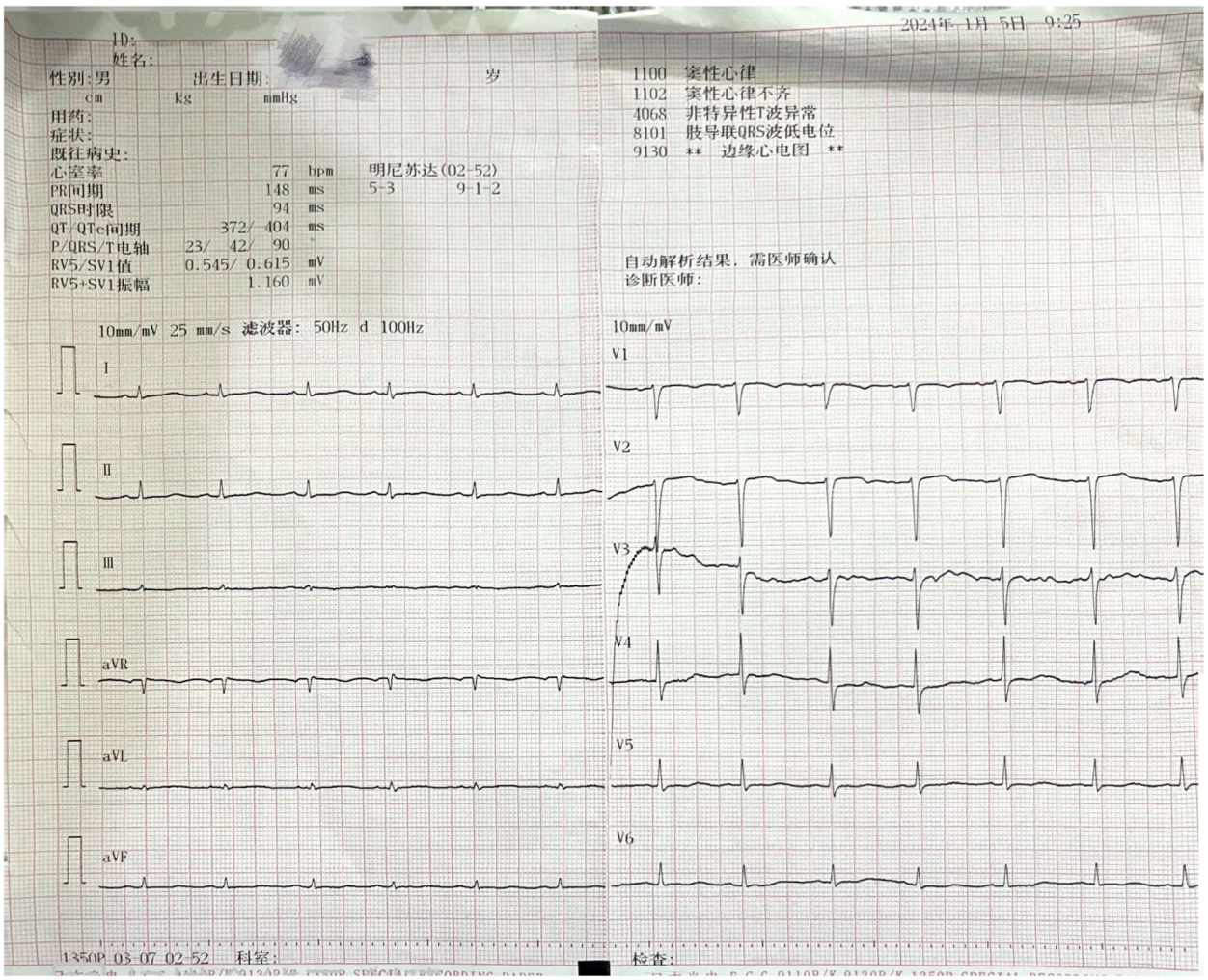

Fig. 1 ECG of this patient

renal, and electrolyte tests were ordered before starting intravenous fluids, which revealed severe hyponatremia but no abnormalities in liver or renal function. Despite administering fluids, the adjusted serum sodium level did not change (Table 1). The calculated osmotic pressure was 259.8 mOsm/L, lower than normal (Table 1). Initially, hypotonic hyponatremia due to severe vomiting was suspected. The patient had been vomiting extensively and was unable to eat. As a result, parenteral nutritional support and intravenous sodium supplements were initiated. Hiatal hernia, gastrointestinal and neurologic diseases were considered as potential underlying causes. A head, chest, and abdominal CT scan was performed, revealing a cerebral lacunar lesion, bilateral pneumonia, and thickening of the gastric antrum wall. A gastroscopy was planned for further evaluation.

While waiting for the gastroscopy, the patient developed a fever, reaching a maximum temperature of 38.3 °C and coughing up white sputum. Pneumonia was diagnosed, and cefoperazone/sulbactam 6 g/d was administered intravenously, along with sputum cultures for bacteria and fungi. The patient became afebrile three days later, and the fungal culture results returned, showing Candida glabrata (2+). Consequently, fluconazole 0.4 g was given on the first day, followed by 0.2 g/d intravenously. The gastroscopy revealed a submucosal protrusive lesion in the gastric antrum, chronic non-atrophic gastritis, and reflux esophagitis.

However, the patient continued to suffer from dizziness and vomiting and was unable to eat. A brain MRI was subsequently ordered, which showed a partial empty sella. Despite extensive testing, the cause of the patient's symptoms remained unclear. Considering the hyponatremia and the empty sella, pituitary disorders were suspected. After consulting an endocrinologist, measurements were taken for serum levels of six sex hormones, renin, aldosterone, angiotensin II, cortisol, adrenocorticotropic hormone (ACTH), FT3, FT4, and TSH. The results indicated central hypothyroidism, central hypogonadism, and central adrenal insufficiency (Table 1). As a result, anterior hypopituitarism was diagnosed as the underlying cause. Initial treatment

Table 1 Laboratory Results of the Patient

Items	Result	Reference	Time	On intravenous fluids
Serum sodium(mmol/L)	120	137–147	First day	No
Serum glucose(mmol/L)	6.0	3.9–6.1	First day	No
Serum Pottasium(mmol/L)	4.1	3.5–5.3	First day	No
Adjusted sodium(mmol/L)	120.1	137–147	First day	No
Osmotic pressure(Mosm/L)	259.8	275–300	First day	No
Leukocyte(10^9)	4.19	3.5–9.5	Third day	No
Creatinine(mmol/L)	42.7	41–81	First day	No
NT-proBNP(pg/ml)	8230	300–450	Seventh day	Yes
Troponin I(ng/ml)	0.259	0–0.034	Seventh day	Yes
Testosterone(ng/ml)	< 0.025	0.029–0.408	Fifth day	No
FSH(mIU/ml)	3.03	25.8–134.8	Fifth day	No
LH(mIU/ml)	0.58	7.7–58.5	Fifth day	No
Prolactin(mIU/ml)	1466	102–496	Fifth day	No
Estrogen(ng/L)	< 5	< 5–138	Fifth day	No
Progesterone(ng/ml)	< 0.05	< 0.05–0.126	Fifth day	No
Renin(uIU/ml)	8.18	3.0–40.9	Fifth day	No
Aldosterone(pg/ml)	55.75	29–240	Fifth day	No
ACTH(pg/ml)	11.64	7–65	Fifth day	No
Cortisol(ug/dl)	1.1	6.4–22.8	Fifth day	No
FT3(pmol/L)	2.73	3.1–6.8	Fifth day	No
FT4(pmol/L)	8.82	12–22	Fifth day	No
TSH(uIU/ml)	2.97	0.27–4.2	Fifth day	No
24 h urine sodium(mmol/L)	168	130–260	Sixth day	Yes
24 h urine pottasium(mmol/L)	64	25–100	Sixth day	Yes

NT-proBNP pro-B type natriuretic peptide, *FSH* Follicle-stimulating hormone, *LH* Luteinizing hormone, *ACTH* Adrenocorticotropic hormone, *TSH* Thyroid-stimulating hormone

with prednisone 7.5 mg/d was started, and levothyroxine sodium 25 μg/d was added two days later, considering potential interactions between the hormones. In light of the brain MRI showing partial empty sella, a more focused MRI of the sellar region was ordered to determine the etiology. However, before the results were available, the patient suddenly developed dyspnea. On auscultation, bilateral rales and wheezing were noted. Dihydroxypropyl theophylline 0.5 g was administered intravenously. Emergency tests for NT-proBNP and troponin I revealed elevated levels (Table 1), leading to a diagnosis of acute left heart failure. Sodium nitroprusside, cedilanid, and morphine were administered to manage the symptoms.

The patient's dyspnea improved, and cardiac echocardiography showed enlargement of the left atrium, with minor pericardial effusion (Table 2). The ejection fraction (EF) was 50%, and the diagnosis of heart failure with preserved ejection fraction (HFpEF) was made, likely secondary to hypopituitarism. According to recent studies, hormone replacement therapy can improve HFpEF. Within three days, the patient's vomiting and dyspnea were alleviated. The sellar MRI results showed an empty sella with occupying lesions in the sellar and suprasellar regions, compressing the optic chiasm. A Rathke cleft cyst was suspected. A neurosurgeon was consulted and recommended surgical resection once the patient's condition stabilized. The patient's serum electrolytes were rechecked, and the sodium levels returned to normal. The patient was discharged from the hospital with instructions to continue levothyroxine and prednisone. She was advised to have her renin, aldosterone, angiotensin II, cortisol, ACTH, FT3, FT4, and TSH levels checked one month later to adjust medication doses. Additionally, she was scheduled to undergo surgical resection of the tumor once her hormone levels had normalized.

Table 2 Echocardiography results of the patient

Items	Result	Reference Range	Time
LA(mm)	49	Female < 38;Male < 40	Eighth day
LV(mm)	44	Female < 50,Male < 53	Eighth day
IVS (mm)	10.8	Female < 11,Male < 12	Eighth day
EDV(ml)	96	84–132	Eighth day
RA(mm)	33*46	Female < 42*52,Male < 45*55	Eighth day
RV(mm)	21	Female < 28,Male < 30	Eighth day
PA(mm)	26	Female < 27,Male < 27	e
CO(L/min)	3.9	3.5–5.5	Eighth day
EF(%)	50	50–70	Eighth day
PAP(mmHg)	23	< 25	Eighth day

LA Left atrium, *LV* Left ventricle, *IVS* Interventricular septum, *EDV* End diastolic volume, *RA* Right atrium, *RV* Right ventricle, *PA* Pulmonary artery, *CO* Cardiac output, *EF* Ejection fraction, *PAP* Pulmonary artery pressure

Discussion

Hypopituitarism is a condition in which the pituitary gland fails to produce adequate amounts of certain hormones. These hormones include adrenocorticotropic hormone, thyroid-stimulating hormone, gonadotropins, growth hormone, prolactin, melanocyte-stimulating hormone, and oxytocin and vasopressin. Each of these hormones has distinct pathways and targets specific organs throughout the body [9, 10].

Pituitary hormone deficiency can have a significant impact on the cardiovascular system. As the central regulator of hormone levels, the pituitary gland controls the synthesis and release of critical hormones essential for various bodily functions. Disruptions in producing these hormones can lead to widespread effects, particularly on the cardiovascular systems.

Growth hormone (GH) is critical in regulating metabolism, body composition, and cardiovascular health [11]. In adults, GH deficiency is associated with an increased risk of cardiovascular disease (CVD) [12], primarily through indirect mechanisms such as dyslipidemia, increased fat mass, reduced lean body mass [13], insulin resistance, and elevated inflammatory markers. These factors contribute to metabolic syndrome and increase the risk of atherosclerosis [14]. These metabolic disturbances are well-established contributors to the development of ischemic heart disease and other cardiovascular conditions in individuals with GH deficiency. Additionally, GH is involved in the metabolic processes of cardiac cells and serves protective functions for the heart [15]. A deficiency in GH may elevate the risk of arrhythmias and ischemic heart disease due to its impact on cardiac muscle metabolism and blood vessel function [16]. It is important to differentiate these effects from the cardiovascular manifestations associated with acromegaly, a condition caused by prolonged exposure to elevated GH and IGF-1 levels [17]. In acromegaly, direct cardiovascular effects such as left ventricular hypertrophy (LVH), arrhythmias, and diastolic dysfunction are commonly seen due to chronic overactivation of the GH-IGF-1 axis [18]. These effects are distinct from those seen in GH deficiency, where cardiovascular alterations are primarily indirect, arising from metabolic dysfunction rather than structural or electrical changes in the myocardium [19]. In this case, the patient presented with symptoms of HFpEF, which were attributed to multiple hormonal deficiencies resulting from hypopituitarism. The combination of hypocortisolism, hypothyroidism, and GH deficiency likely contributed to impaired myocardial relaxation, metabolic disturbances, and increased cardiovascular

risk. However, the absence of structural cardiac abnormalities, such as LVH or arrhythmias, is consistent with the profile of GH deficiency rather than acromegaly, which underscores the need to distinguish between the distinct cardiovascular risks associated with these two conditions. IGF-1, a key mediator of GH effects, plays an important role in cardiovascular health. Due to GH insufficiency, its deficiency could contribute to increased cardiovascular risk through impaired endothelial function, reduced lean muscle mass, and metabolic dysregulation [20]. In this case, GH deficiency was not explicitly assessed through IGF-1 levels, which may be because IGF-1, while a useful marker of GH activity, is not always measured during the diagnostic process for hypopituitarism when other clinical and biochemical findings—such as central adrenal insufficiency and central hypothyroidism—already strongly point to the condition. The patient presented with acute cardiovascular symptoms (HFpEF), which likely shifted the immediate clinical focus toward stabilizing critical endocrine deficiencies like cortisol and thyroid hormones, with more direct and urgent cardiovascular implications. Hormone replacement therapy (HRT) with cortisol and thyroid hormones was initiated first, as these were more immediately critical. GH replacement therapy could be considered later, particularly if IGF-1 deficiency is confirmed. HRT for GH, aimed at restoring IGF-1 levels, might improve muscle mass, reduce fat accumulation, and lower cardiovascular risks associated with hypopituitarism. If clinically indicated, periodic monitoring of IGF-1 and GH stimulation testing should be part of the ongoing endocrine evaluation in hypopituitarism cases to ensure comprehensive care. However, GH HRT is contraindicated in patients with active malignancy [20], and a malignant pituitary tumor could not be completely ruled out in this patient. Therefore, GH replacement therapy may be considered in the future, but only after confirming that the patient's condition is stable and that there are no contraindications. In this case, the absence of IGF-1 measurement does not invalidate the treatment plan but highlights an area for further investigation and optimization of endocrine replacement therapy.

ACTH stimulates the production of cortisol [21], and a cortisol deficiency can lead to hypotension, as cortisol is essential for maintaining vascular tone and the heart's responsiveness to catecholamines [22]. There are two main types of adrenal insufficiency: primary and secondary, each with distinct causes and clinical features. Primary adrenal insufficiency (PAI), also known as Addison's disease, is caused by direct damage to or dysfunction of the adrenal glands [23]. Common etiologies include autoimmune destruction, infections like tuberculosis, adrenal hemorrhage, and infiltrative disorders [24]. PAI is characterized by deficiencies in both glucocorticoids (cortisol) and mineralocorticoids (aldosterone) [25]. Patients often present with hyperpigmentation due to elevated ACTH levels, along with symptoms such as hypotension, hyponatremia, hyperkalemia, and fatigue [26]. Secondary adrenal insufficiency (SAI), on the other hand, occurs due to insufficient ACTH production by the pituitary gland, often resulting from pituitary or hypothalamic disorders such as tumors, trauma, or radiation therapy [27]. Unlike PAI, mineralocorticoid production is typically preserved in SAI because aldosterone secretion is mainly regulated by the renin–angiotensin–aldosterone system rather than ACTH [28]. As a result, hyperkalemia is usually absent, but patients may still experience symptoms such as fatigue, hypotension, hypoglycemia, and weight loss [29]. SAI may also lack hyperpigmentation, as ACTH levels are not significantly elevated [30]. In this case, the patient exhibited secondary adrenal insufficiency as part of anterior hypopituitarism [29]. Laboratory findings demonstrated low cortisol levels with low-normal ACTH, consistent with SAI. The absence of hyperkalemia and hyperpigmentation further supports this diagnosis. The patient's symptoms and acute heart failure were effectively managed with glucocorticoid replacement therapy, underscoring the importance of identifying and treating hormonal deficiencies in hypopituitarism. Individuals with secondary adrenal insufficiency, resulting from a deficiency in pituitary ACTH, can experience cortisol shortages, particularly during periods of stress when the body requires increased cortisol production [31]. These individuals may exhibit reduced cardiac output and deficient response to stress, increasing the risk of cardiovascular collapse under severe conditions [32]. The echocardiography of this patient showed HFpEF and a low-normal ejection fraction, indicating diastolic dysfunction and reduced cardiac output, consistent with cortisol deficiency. The patient also presented with dyspnea, nausea, and vomiting, all of which are characteristic of cortisol deficiency. Prednisone was administered to address these issues. The choice of prednisone may have been based on specific patient factors such as compliance and comorbidities. Prednisone has a longer half-life than hydrocortisone and can be administered as a single daily dose, which simplifies treatment, particularly for older patients who may struggle with complex medication regimens. In our region, prednisone is more cost-effective and readily available than hydrocortisone. The price of hydrocortisone is 23 times higher than that of prednisone, and it can only be obtained from an online pharmacy, which would delay treatment by two days. Though not identical to hydrocortisone in pharmacokinetics, prednisone can achieve a similar physiological effect when given in an

equivalent dose. Thus, prednisone did not attenuate the treatment effect in this patient. Hypopituitarism, especially in the context of secondary adrenal insufficiency, can have profound effects on multiple systems, including the cardiovascular system. In this case, we believe that an adrenal crisis, precipitated by the patient's acute clinical deterioration, may have contributed to the onset of acute heart failure. Cortisol is essential for maintaining vascular tone and myocardial function. In patients with adrenal insufficiency, the lack of sufficient cortisol impairs the body's ability to respond to stress, leading to hypotension, electrolyte disturbances, and reduced myocardial contractility [33]. This deficiency is particularly pronounced in secondary adrenal insufficiency, where there is a deficiency in ACTH due to pituitary dysfunction. The patient's acute illness, characterized by vomiting, hyponatremia, and gastrointestinal distress, likely represented an acute stress event that exacerbated her adrenal insufficiency, potentially triggering an adrenal crisis. Under normal circumstances, cortisol plays a critical role in the body's stress response, particularly in maintaining cardiovascular stability. In the absence of cortisol, patients are unable to mount an appropriate response to stress, leading to hypotension, reduced cardiac output, and acute heart failure [34]. This was likely the case for our patient, whose heart failure was precipitated by hypocortisolism, impairing her cardiovascular response to the acute illness. The development of HFpEF in this patient is consistent with previous studies showing that hypocortisolism can impair myocardial relaxation and increase filling pressures, both hallmarks of diastolic dysfunction [33]. Additionally, pericardial effusion, a known complication of hypocortisolism, was also observed in this patient, likely contributing to the development of heart failure. The normal ejection fraction (EF) in this patient, as opposed to a reduced EF, further supports the diagnosis of HFpEF, where the issue lies in the heart's ability to relax and fill adequately rather than in systolic function. The patient's symptoms improved significantly after initiating glucocorticoid replacement therapy (prednisone). This clinical improvement supports the hypothesis that adrenal crisis due to secondary adrenal insufficiency played a significant role in the development of her acute heart failure. The hormonal replacement therapy effectively restored cortisol levels, alleviating the symptoms of heart failure, reducing dyspnea, and improving overall clinical status.

TSH triggers the synthesis and secretion of thyroid hormones, which exert multiple effects on the cardiovascular system [35]. Thyroid hormones increase heart rate and contractility, enhance cardiac output, and promote vasodilation, thereby improving blood circulation [35]. Consequently, a deficiency in TSH, leading to hypothyroidism, can result in bradycardia (slower heart rate), reduced myocardial contractility, increased systemic vascular resistance, and potentially heart failure [36]. Hypothyroidism also impairs myocardial relaxation, leading to diastolic dysfunction and the development of pericardial effusion. These processes likely contributed to the development of HFpEF in this patient, consistent with the symptoms of hypothyroidism. Additionally, the patient's pericardial effusion further supports the diagnosis of hypothyroidism as a contributing factor.

The patient's laboratory examination indicates partial anterior hypopituitarism, with deficiencies in cortisol, thyroid hormones, and sex hormones, consistent with dysfunction of the anterior pituitary gland. This partial involvement suggests that not all pituitary functions were impaired, and only specific hormonal axes were affected. The patient did not exhibit complete pituitary failure, typically involving deficiencies in all anterior pituitary hormones. The patient was diagnosed with HFpEF, a condition in which the heart muscle can contract normally but has difficulty relaxing. This impaired relaxation restricts the heart's ability to fill with blood between beats, leading to symptoms like shortness of breath, fatigue, and fluid accumulation, particularly after physical exertion. Hypocortisolism is known to impair myocardial function, especially myocardial relaxation, which plays a key role in the development of HFpEF [33]. In this case, the lack of cortisol likely impaired myocardial relaxation and fluid retention, resulting in pericardial effusion. These factors collectively worsened the patient's cardiovascular status and contributed to the development of HFpEF. Hypothyroidism also impairs myocardial relaxation, leading to diastolic dysfunction and pericardial effusion. The patient's diagnosis of HFpEF was therefore consistent with her hypothyroidism, as well as the presence of pericardial effusion, which further supports this connection. Given these factors, the patient's cardiac manifestations align with findings in the literature regarding HFpEF due to hypopituitarism. Hormonal replacement therapy should be initiated as part of the treatment plan to address the underlying hormonal deficiencies and improve the patient's overall cardiovascular function.

The patient in this case also presented with severe nausea and vomiting, with no organic abnormalities identified. A similar case reported nausea and vomiting in patients with Addison's disease. Nausea and vomiting in the context of hypopituitarism are particularly intriguing, as these symptoms can be both direct and indirect consequences of hormonal deficiencies. For example, adrenal insufficiency due to a lack of ACTH can precipitate nausea, which is often exacerbated during stress or illness. This patient had secondary hypocortisolism, which may

have been the underlying cause of her nausea and vomiting. Additionally, thyroid hormone deficiencies can slow gastric motility, potentially leading to nausea and vomiting. The patient was diagnosed with central hypothyroidism, which likely contributed to her gastrointestinal symptoms. The patient's symptoms were alleviated after initiating hormone replacement therapy. The secondary effects of hormone deficiencies further compound the complexity of this case. For instance, while deficiencies in growth hormone (GH) or gonadotropins may not directly cause nausea, their overall impact on well-being could contribute to conditions such as gastroparesis or changes in appetite, which indirectly influence nausea and vomiting. Hormone replacement therapy can improve the prognosis in such cases. This patient's glucocorticoid and thyroid hormone replacement were particularly relevant in improving her prognosis. Cortisol plays a critical role in maintaining vascular tone, myocardial function, and the body's ability to respond to stress [32, 33]. Glucocorticoid therapy helps restore normal cardiovascular function, especially in cases of hypocortisolism, as seen in this patient. Similarly, thyroid hormones are essential for maintaining normal heart rate, cardiac output, and vascular tone. Inadequate thyroid hormone levels can lead to diastolic dysfunction and heart failure [36], which were observed in this patient.

The elevated prolactin levels in this patient could be attributed to hypopituitarism resulting from the Rathke cleft cyst, which may have disrupted the normal pituitary function. In cases of pituitary dysfunction, particularly with lesions affecting the anterior pituitary, there can be a disruption in the inhibitory regulation of prolactin by dopamine. This disruption can lead to hyperprolactinemia. Additionally, secondary hypothyroidism can also contribute to elevated prolactin levels, as low thyroid hormone levels can enhance the release of prolactin by increasing the sensitivity of the pituitary to thyrotropin-releasing hormone (TRH), which stimulates prolactin secretion. Thus, the elevated prolactin in this patient is likely a consequence of the combined effects of pituitary dysfunction and thyroid hormone deficiency.

Identifying hypopituitarism can be challenging due to the often vague and nonspecific nature of its symptoms, as the pituitary gland plays a central role in regulating hormones across various bodily systems. The presentation of symptoms in patients can vary widely, ranging from mild to potentially fatal, depending on the speed and extent of hormonal deficiencies. Signs such as fatigue, frailty, loss of appetite, illness, impaired stress response, and low serum glucose levels may indicate a deficiency in cortisol [37] or growth hormone [38]. Conversely, polyuria and polydipsia may suggest a deficiency in antidiuretic hormone (ADH) [39]. The primary treatment for hypopituitarism is hormone replacement therapy (HRT), which is tailored to address each patient's specific hormonal deficiencies [40]. The hormones that may require replacement include cortisol, thyroid hormone, sex hormones (estrogen, progesterone, and testosterone), and, in some cases, growth hormone and antidiuretic hormone [41]. Hormone replacement therapy typically leads to significant improvement in symptoms, including the alleviation of cardiovascular manifestations.

In conclusion, hypopituitarism results in impaired production of several key hormones, including adrenocorticotropic hormone, thyroid-stimulating hormone, gonadotropins, growth hormone, prolactin, melanocyte-stimulating hormone, and oxytocin/vasopressin. This hormonal deficiency leads to a range of clinical manifestations, with cardiac symptoms being relatively rare but notable. Cardiac manifestations of hypopituitarism may include heart failure, increased risk of coronary artery disease, hypotension, pericardial effusion, and bradycardia. The diagnosis of hypopituitarism can be challenging and relies on the measurement of target hormones produced by the end organs affected by pituitary dysfunction. The primary treatment for hypopituitarism is hormone replacement therapy (HRT), which can effectively alleviate many symptoms, including those related to the cardiovascular system. In cases where a pituitary tumor is present, surgical resection may also help to reduce symptoms resulting from hormonal deficiencies. Physicians should focus on accurate diagnosis and targeted hormone replacement rather than merely addressing symptoms, as this approach can lead to more comprehensive and effective management of the condition.

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Ethics and dissemination
Informed written consent was obtained from the patient. The case was approved to be published by the ethics committee of Qingdao Central Hospital.

Authors' contributions
Mengmei Li and Xiaowen Zhen wrote the main manuscript; Yufang Li and Chenglong Ren prepared the discussion part and table1-2. All authors reviewed the manuscript.

Data availability
No datasets were generated or analysed during the current study.

Declarations

Competing interests
The authors declare no competing interests.

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