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CASE REPORT

Amenorrhea Induced by Hydrocephalus Due to Tuberculous Meningitis: A Rare Case Report and Literature

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Abstract: Secondary amenorrhea is commonly associated with endocrine or gynecological disorders; however, central nervous system diseases may also disrupt the hypothalamic–pituitary–ovarian (HPO) axis and lead to hypogonadotropic hypogonadism. Hydrocephalus caused by tuberculous meningitis represents a rare but important neurological cause of menstrual disturbance. We report the case of a 24-year-old Indonesian woman of Southeast Asian ethnicity presenting with secondary amenorrhea for 2.5 years following a history of tuberculous meningitis complicated by communicating hydrocephalus and ventriculoperitoneal shunt placement. Hormonal evaluation revealed low gonadotropin and estradiol levels consistent with hypogonadotropic hypogonadism. Neuroimaging demonstrated ischemic lesions involving the thalamic and basal ganglia regions with radiological features of hydrocephalus and tuberculoma-related meningeal enhancement. Transrectal ultrasonography showed preserved ovarian morphology with antral follicles, supporting a central rather than ovarian etiology. Bone mineral density assessment revealed reduced bone mass associated with prolonged hypoestrogenism. A review of the literature highlights several proposed mechanisms linking hydrocephalus and hypothalamic dysfunction, including increased intracranial pressure and structural compression of hypothalamic pathways. This case emphasizes that neurological conditions such as tuberculous meningitis with hydrocephalus may cause secondary amenorrhea through disruption of hypothalamic–pituitary regulation. Early recognition of neuroendocrine complications is essential to prevent delayed diagnosis and long-term reproductive or metabolic consequences.

Keywords: secondary amenorrhea, hypogonadotropic hypogonadism, hydrocephalus, tuberculous meningitis, ventriculoperitoneal shunt

Introduction

Amenorrhea is defined as the absence or abnormal cessation of menstruation, which may be complex and concerning for many women. Secondary amenorrhea, in which amenorrhea happens after the initiation of regular periods, may be accounted by secondary hypogonadotropic hypogonadism, which is characterized by disrupted gonadotropin-releasing hormone (GnRH) secretion. This mechanism represents nearly 15–20% of all secondary amenorrhea cases and is often caused by structural central nervous system pathologies.[1] Chronic hydrocephalus, particularly those that are caused by obstruction as a result of aqueductal stenosis, may impair pulsatile GnRH release, which eventually leads to missed periods. Interventions such as ventriculostomy and ventriculoperitoneal shunting, which helps relief increased intracranial pressure, has been found to restore menstrual cycle in more than 75% of cases, which brings about the idea on how these pathologies are plausibly reversible.[2]

In general, tuberculosis infection may contribute to amenorrhea through malnutrition and chronic inflammation. Moreover, tuberculous meningitis, which represents 1–5% of all extrapulmonary tuberculosis cases, may initiate cerebrospinal fluid

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(CSF) buildup as a result of arachnoid fibrosis and basal cistern exudates, which manifests into hydrocephalus in 30–80% patients.[3] Additionally, tuberculosis infection produces inflammatory reactions and cytokines such as tumor necrosis factoralpha (TNF-a) and interleukin-6 (IL-6), which leads to ventriculomegaly through impairment of CSF absorption, and concurrently decreases GnRH neuronal activity. Previous studies have found how 31% of women with central nervous system (CNS) tuberculosis present with menstrual irregularities, preceding neurological symptoms.[4]

Diagnostic challenges are often seen in patients with tuberculous meningitis and hydrocephalus. Basal meningeal enhancement on magnetic resonance imaging (MRI), often seen in the majority of tuberculous meningitis-related hydrocephalus cases, is associated with CSF obstruction and hypothalamic distortion. A case report demonstrated the rapid progression of obstructive hydrocephalus after the diagnosis of tuberculous meningitis, which increases the urgency of early diagnosing before symptoms worsen and permanent hypothalamic damage occurs. Treatment of tuberculous meningitis requires strict regimens of antituberculosis therapy and early diversion of CSF, if needed.[2,3,5]

Despite these associations, amenorrhea as a result of tuberculous meningitis is seldom discussed in papers, which reflects the lack of recognition of this condition particularly in under resourced settings. If any, most cases often highlight the neurological symptoms of tuberculous meningitis and not in a gynecologic standpoint.[6] This study presents a rare case of secondary amenorrhea as a result of hydrocephalus due to meningitis tuberculosis from Hasan Sadikin Hospital, Bandung, Indonesia, which aims to increase awareness on how missed menstrual periods may indicate a more complex pathophysiology that clinicians should not miss. To contextualize this case, we conducted a narrative review of the literature focusing on reported cases of secondary amenorrhea associated with hydrocephalus or central nervous system infections. Articles were identified through PubMed using keywords including “hydrocephalus”, “amenorrhea”, and “tuberculous meningitis”. Relevant reports were reviewed to summarize proposed mechanisms and clinical outcomes.

Case Illustration

A 24-year-old Indonesian woman of Southeast Asian ethnicity from Sayang Hospital, Cianjur, was referred due to secondary amenorrhea persisting for 2.5 years, potentially attributed to a pituitary disorder suspected to be associated with tuberculous meningitis. The patient underwent antituberculosis treatment for 12 months. Regrettably, the data pertaining to tuberculosis were insufficient (the records from the prior hospital were limited). From the physical examination, her Tanner stage is mammae 4, pubic 4; this gives us an insight that she had a prior history of estrogen and androgen exposure (Figure 1). The ultrasound images provided show a transrectal sonographic evaluation of the uterus and ovaries. The uterus appears retroflexed, with size 4.83 × 2.18 × 2.98 cm, EL 0.22 cm, right ovary with size 2.32 × 1.52 × 1.63 cm, vol 3.010 cm3, with antral follicle; left ovary with size 1.45 × 1.18 × 1.77 cm, with antral follicle, with fluid collection in the pouch of Douglas (Figure 2). Data from a prior computed tomography (CT-scan) conducted in 2022 revealed ischemic lesions in the bilateral thalamus, bilateral basal ganglia, and bilateral cortical-subcortical frontoparietal lobes, indicating communicant hydrocephalus (Figure 3). A ventriculoperitoneal shunt was subsequently placed in the patient (Santosa Hospital, March 3rd, 2022). A follow-up CT scan was conducted on April 29th, 2025, and gave a result of subacute infarct on the left cortical subcortical lobes of parietooccipital, inhomogeneous multiple lesions, multiloculated, irregular edge on the left hypophysis and medulla oblongata with meningeal enhancement, especially on the cisterna basalis due to meningitis with caseating phase tuberculoma (Figure 4). We also performed an immunological assessment for tuberculosis utilizing the IGRA (IFN-Gamma Release Assay) test and found that the results were positively related to tuberculosis infection. She additionally states that her prior condition has resulted in paralysis of her lower extremities and hearing impairment. Prior to the illness, she had a regular menstrual cycle that lasted eight days and happened every 28 days. Her sexual development was normal, and she started having periods at the age of 13.

Laboratory examination revealed pregnancy test was negative; FSH level of 3.37 mIU/mL (reference range: 3–10 mIU/mL); LH level of 0.59 mIU/mL (reference range: 2–15 mIU/mL); PRL level of 17.25 ng/mL (reference range: 5–25 ng/mL); estradiol level of 18.49 pg/mL (reference range: 30–400 pg/mL in reproductive women); FT4 level of 1.09 ng/mL; TSH level of 0.972 ng/mL; AMH level of 3.17 ng/mL, suggestive of hypogonadotropic hypogonadism. The bone mass density (BMD) assessment, conducted according to WHO criteria and the International Society of Clinical Densitometry (ISCD) 2007, yielded a z-score of −2.9 SD. Based on the transrectal sonography biophysical profile, hormonal findings, and supplementary examination, we classified her condition as secondary amenorrhea due to

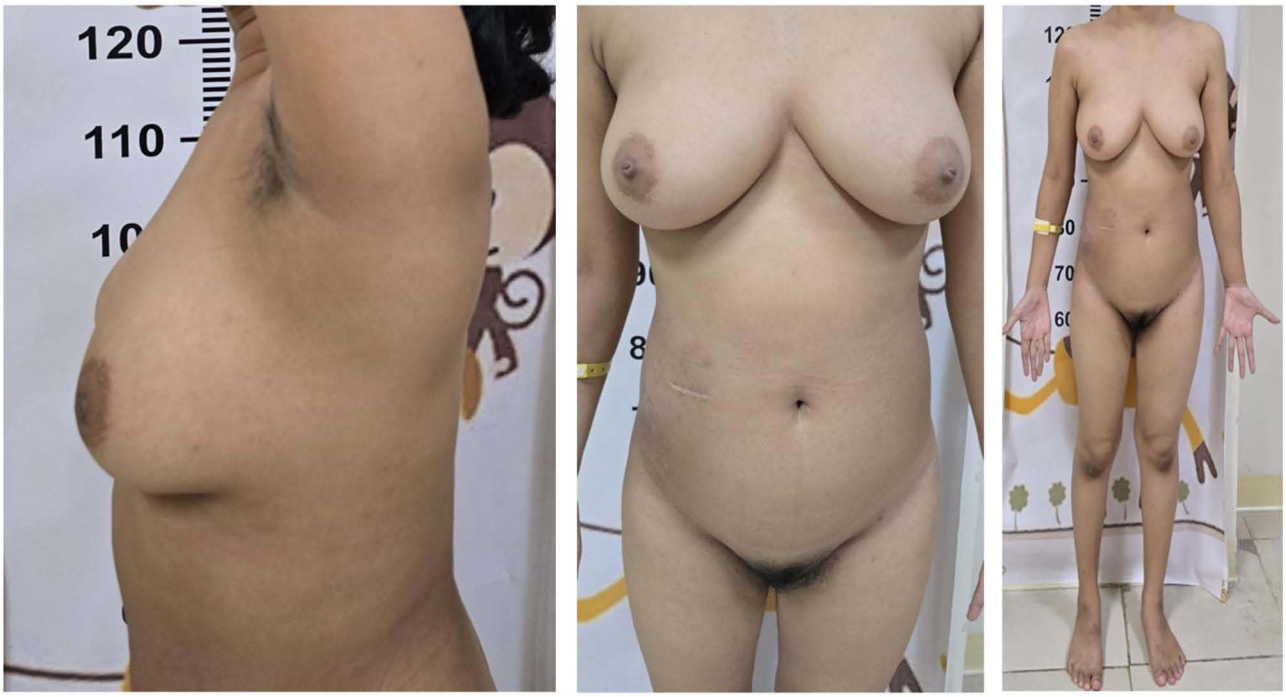

Figure 1 Tanner stage and physical examination of the patient.

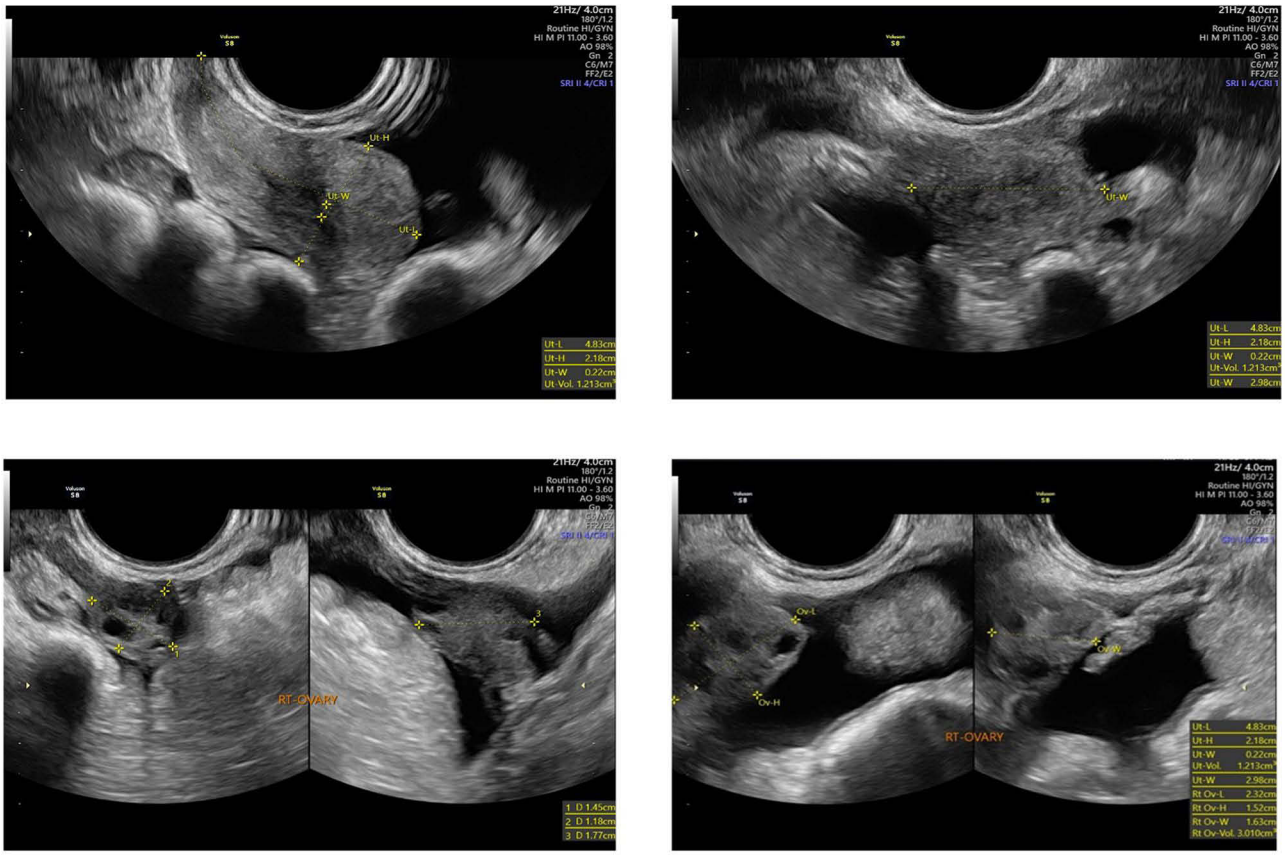

Figure 2 The transrectal ultrasonography.

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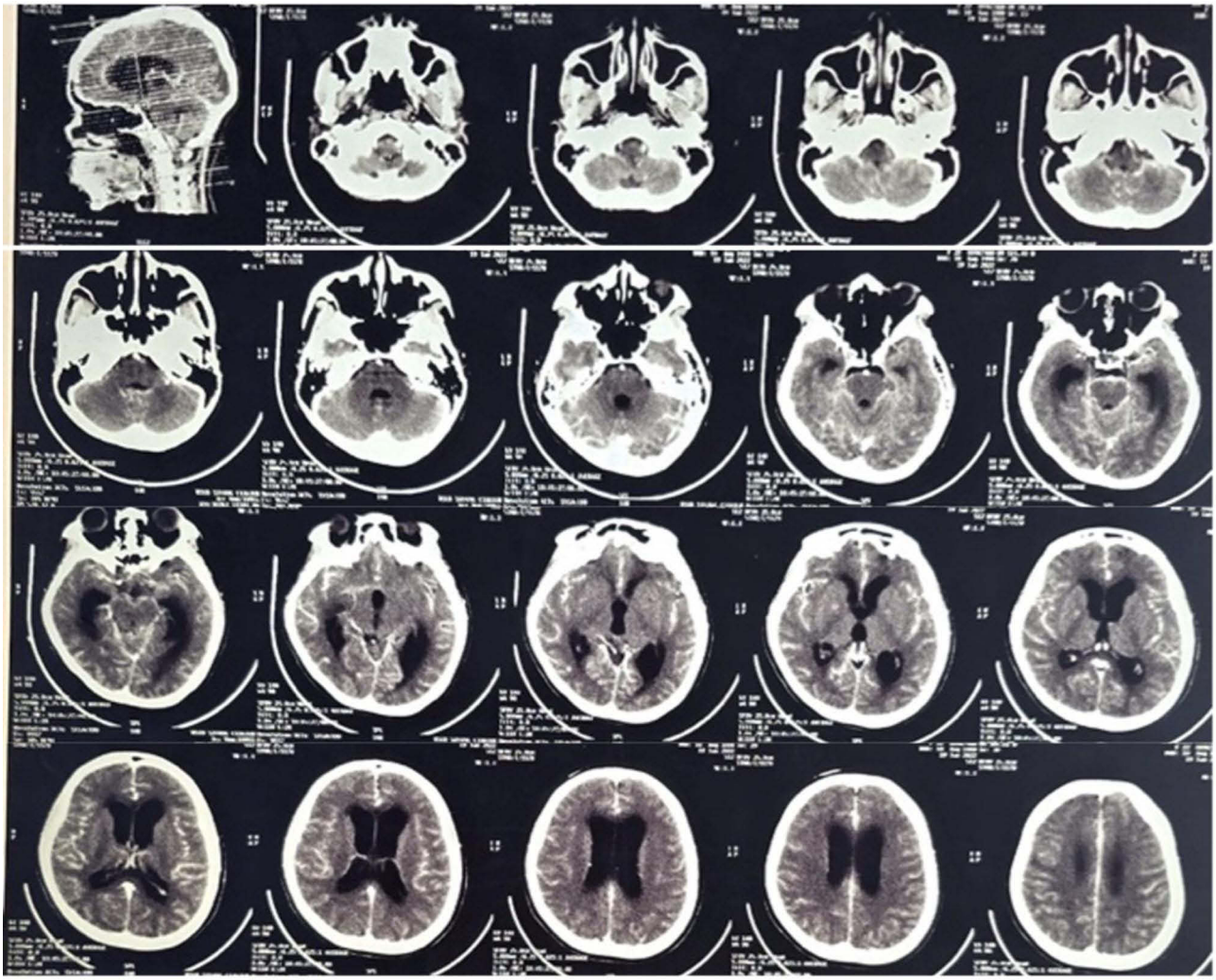

Figure 3 Brain CT scan performed in 2022 before ventriculoperitoneal shunt showing ischemic lesions in bilateral thalamus, basal ganglia and cortical–subcortical frontoparietal lobes suggesting communicating hydrocephalus.

hypothalamic dysfunction (hypogonadotropic hypogonadism) resulting from hydrocephalus induced by tuberculosis meningitis infection. And we conduct the management for her condition by giving her estradiol valerate 2 mg/daily to induce the proliferative state of the endometrium and observe the effect on this patient.

A chronological summary of the patient's clinical course is presented in Table 1 to clarify the sequence of neurological and endocrine events.

Discussion

Hypogonadotropic hypogonadism is characterized by impaired secretion of GnRH and/or gonadotropins, usually leading to diminished levels of both FSH and LH.[7] A structural abnormality of the pituitary gland linked to infection is an uncommon condition that can cause hypogonadotropic hypogonadism leading to secondary amenorrhea, especially when caused by tuberculous meningitis, comprising 1–5% of all tuberculosis cases.[8,9] Hydrocephalus is the most common complication of tuberculosis meningitis. Reports indicate that around 80% of patients with tuberculosis meningitis display signs of ventricular enlargement in the early stages, with hydrocephalus serving as a critical prognostic marker. The types associated with this condition include communicative, obstructive, and mixed.[8,10] The prior CT scan from 2022 revealed the existence of communicating hydrocephalus, likely linked to this condition.

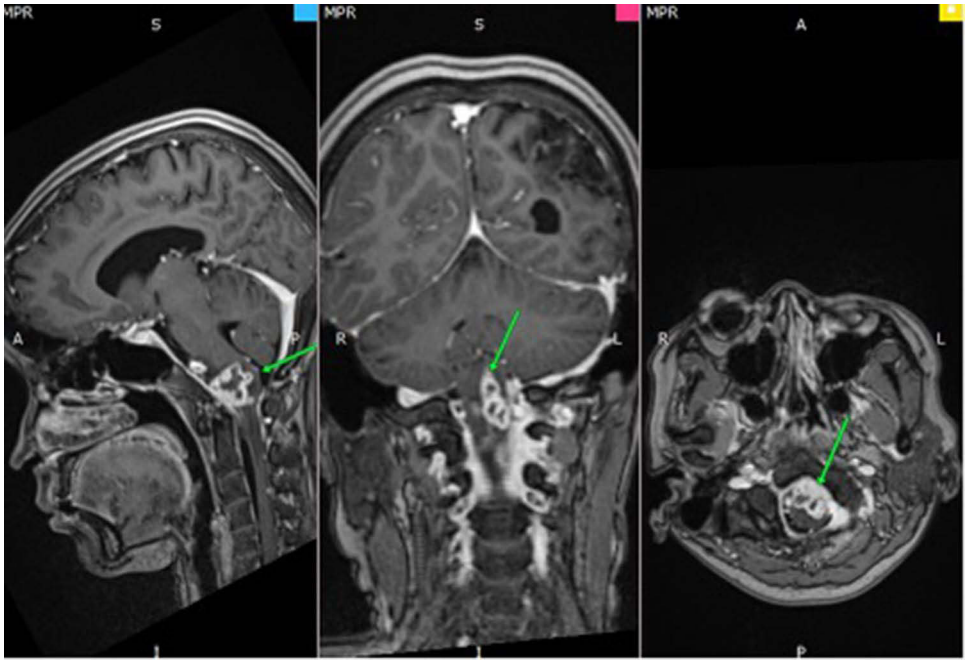

Figure 4 Brain CT scan performed on April 29, 2025 showing subacute infarct in the left parietooccipital cortical–subcortical region. Green arrows indicate multiloculated lesions with meningeal enhancement in the cisterna basalis suggestive of caseating tuberculoma.

Although the pathophysiology of hydrocephalus-induced amenorrhea is still not fully comprehended, the case report by Lee et al[2] indicates that the pathogenesis of hydrocephalus does not involve the hypothalamic process causing the inhibitory effect on the hypothalamic-pituitary-ovary axis at the hypothalamic level, but rather the elevated intracranial pressure caused by hydrocephalus. Coenegracht et al[11] postulated that a functional disruption limited to the gonadotropin-releasing hormone (GnRH)-secreting neurons in the ventral hypothalamus, rather than the median eminence, might be the cause of a relative GnRH deficiency. Lowry et al[12] said that, it is possible that noncommunicating hydrocephalus leads to amenorrhea by dilation of the third ventricle and distention of the periventricular and medial basal regions of the hypothalamus, where one finds the ventromedial and arcuate nuclei in which GnRH-producing

Table 1 Clinical Timeline of the Patient

Time	Clinical Events
2022	Patient was diagnosed with tuberculous meningitis after presenting with neurological symptoms.
	Brain CT scan revealed communicating hydrocephalus with ischemic lesions involving the bilateral thalamus, basal ganglia, and frontoparietal regions.
	Ventriculoperitoneal (VP) shunt was placed to manage hydrocephalus.
	Following the neurological illness, the patient began experiencing secondary amenorrhea.
2023-2024	Amenorrhea persisted without menstrual recovery.
29th April, 2025	Follow-up CT scan showed subacute infarction in the left parieto-occipital cortical–subcortical region with multiloculated lesions and meningeal enhancement consistent with tuberculoma in the caseating phase.
2025	Hormonal evaluation demonstrated low gonadotropin and estradiol levels consistent with hypogonadotropic hypogonadism.
	Transrectal ultrasonography showed a retroflexed uterus with preserved ovarian morphology and antral follicles, suggesting a central cause of amenorrhea.
	Bone mineral density assessment revealed decreased bone mass associated with prolonged hypoestrogenism.

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parvocellular neurons are located. If progressive dilation of the third ventricle is the cause, it is possible that GnRH release is impaired solely in the ventral hypothalamus, while GnRH production in other regions remains unaffected, resulting in a relative deficiency of GnRH. Conversely, secondary mechanisms, including global or focal ischemia, may result in axonal damage, reduced neuronal growth, and compromised axonal transport. The impaired axonal transport may have a deleterious effect on the transport of GnRH through the fibers that are coursing through the floor or the third ventricle and the pituitary stalk.[13]

The primary objective is to alleviate intracranial pressure to restore the condition and resume normal gonadotropin pulsatility. A case report by Touraine et al[14] supports the assertion that performing a ventriculocisternostomy to alleviate elevated intracranial pressure results in a gradual restoration of gonadotropin pulsatility, with normal menstruation accompanying the enhancement of LH pulsatility. Unfortunately, despite the patient's ventricular shunt insertion in 2022, she did not exhibit any restoration of pulsatility. The likelihood of shunt malfunction is a considerable concern; therefore, we referred the patient to a neurosurgeon for further evaluation.

A complex relationship entangles tuberculous-meningitis induced hydrocephalus and secondary hypogonadotropic hypogonadism. Hydrocephalus, as seen in this patient, and observed in the majority of tuberculous meningitis patients, exerts direct pressure on the median eminence and infundibular recess of the brain, which disrupts GnRH pulsatility, affecting the hypothalamic-pituitaryovarian (HPO) axis. Arachnoid fibrosis, as a result of inflammatory reactions in the meninges, perpetuates this obstruction through impairing CSF absorption.[15–17]

The impaired feedback of the HPO axis inhibits the ovarian granulosa cells from receiving signal associated with estradiol production, which eventually leads to the lack of endometrial thickening during the follicular phase in a healthy woman.[18,19] However, the disruption of this axis leads to clinical symptoms beyond amenorrhea. This includes reduced bone mineral density, increased cardiovascular risk, as well as psychological distress.[15–17] Chronic hypoestrogenism predisposes patients to osteopenia with a 2.5-fold increased fracture risk in patients who are left untreated. Patients with estrogen deficiency experience increased osteoclastic activity, which leads to a 7.4% annual decline in lumbar spine bone mineral density. Tuberculosis infection, in itself, also exacerbates skeletal fragility through chronic inflammation, inhibiting osteoblast differentiation and initiating osteoclastogenesis.[20–22]

Additionally, an inflammatory condition drives patients to a disrupted metabolic state, leading to insulin resistance, dyslipidemia, and a pro-atherogenic profile. Hypoestrogenism impairs insulin sensitivity by impairing GLUT4 translocation in adipocytes, which leads to a 34% higher prevalence of individuals with insulin resistance in comparison to healthy subjects. Tuberculosis has been associated with elevated LDL-C and a decline in HDL-C levels, attributable to cytokine-mediated suppression of lipoprotein lipase. Leptin levels are also further suppressed by TB-induced cachexia, which leads to a vicious cycle of metabolic dysregulation.[23–25]

Patients may also experience neuropsychiatric symptoms including depression and cognitive decline, which are often underrecognized yet debilitating. Estrogen is a crucial neuroprotector, and the lack of estrogen leads to increase of hippocampal glutamate toxicity and a reduction in serotonin synthesis, associated with a 3.1-fold increase in major depressive disorder cases among individuals with hypogonadotropic hypogonadism. Individuals also experience cognitive deficits, particularly associated with verbal memory and executive function, which is a result of decreased hippocampal neurogenesis and prefrontal cortex dendritic atrophy. In patients with tuberculosis, the blood-brain barrier is disrupted, which leads to increased quinolic acid levels, promoting neurotoxicity.[26–28]

Treatment strategies for hypogonadotropic hypogonadism should aim to restore hormonal status while treating underlying etiology. In male patients, testosterone replacement should be prioritized as first-line symptom relief. Transdermal testosterone may help restore libido, muscle mass, and bone density within 6–12 months. Among female patients, estrogen-progesterone regimens, such as transdermal 17B-estradiol + cyclic progesterone are preferable given thromboembolic risks in estrogen-only treatments. Hormone replacement therapy is crucial as initiation within 5 years of amenorrhea onset halts progression of bone loss in 92% cases.[29–33]

In patients aiming for fertility restoration, gonadrotropin therapy is essential. Patients may be advised to combine human chorionic gonadotropin (hCG; 1000–2500 IU biweekly) and follicle stimulating hormone (FSH: 75–150 IU 3x/week), with studies reporting spermatogenesis in 86% of males within 12–24 months. For patients with hypothalamic hypogonadotropic hypogonadism, pulsatile GnRH pumps (100–400 ng/kg/2 hours) normalize gonadotropin pulsatility with 60.7% patients

who failed the first regimen achieving spermatogenesis. Additionally, clomiphene citrate, a selective estrogen receptor modulator, may augment endogenous GnRH release by blocking hypothalamic estrogen feedback.[31,34,35]

Therapeutic efficacy is highly dependent on factors including baseline testicular volume, body mass index, and treatment duration. Obesity, or a BMI of 30 kg/m2 and above, may lead to hypogonadism as a result of leptin resistance and aromatase-driven estrogen excess, which suppresses gonadotropin and GnRH pulsatility.[36]

Concurrent antituberculosis therapy and CSF diversion remain a critical portion of care. First line antituberculosis regimens (isoniazid, rifampicin, pyrazinamide, and ethambutol) must be initiated promptly to avoid progression in arachnoid fibrosis and hypothalamic damage. Additionally, corticosteroids should be initiated to reduce inflammatory processes responsible for CSF obstruction.

Surgical procedures, including ventriculoperitoneal shunting (VP), help alleviate intracranial hypertension and subsequent mechanical compression. A previous study has found that VP shunting within 2 weeks of antituberculosis initiation normalizes intracranial pressure in 92.9% of tuberculous meningitis-related hydrocephalus cases, with more than half of all patients achieving long-term functional recovery. A different study highlighted the importance of early shunting as delayed shunting of 4 weeks and above increases risk of irreversibility due to hypothalamic ischemia. These two mechanisms, when combined synergistically, restore pulsatile gonadotropic secretion, with menstrual resumption observed in 78% of patients within 6 months post-shunt.[8,10,37,38] However, given the chronic state of the patient in this case, pulsatility was not observed after CSF diversion, which further emphasizes the importance of early treatment.

Conclusion

This case highlights a rare presentation of secondary amenorrhea resulting from hypogonadotropic hypogonadism associated with hydrocephalus secondary to tuberculous meningitis. Disruption of hypothalamic–pituitary regulation due to increased intracranial pressure and central nervous system inflammation may impair gonadotropin secretion and lead to menstrual disturbances. Importantly, endocrine manifestations such as secondary amenorrhea may occur before or alongside neurological complications. Therefore, early recognition and evaluation of hormonal abnormalities in patients with central nervous system infections are essential to prevent delayed diagnosis and potential long-term reproductive or metabolic consequences.

Informed Consent Patient Statement

No formal ethical clearance was required for the publication of this case. The authors confirm that written informed consent for publication of this case report and any accompanying images was obtained from the patient and her parents. The patient was informed in detail about the case content and agreed to its publication. All personal identifiers have been removed to ensure patient anonymity.

Disclosure

The authors report no conflicts of interest in this work.

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