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The Quiet Gland: Central Hypopituitarism Masquerading as Depression and Refractory Shock in a Critically Ill Woman

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Abstract

Background: *Central hypopituitarism is an under-recognized mimic of far more common inpatient diagnoses. Because secondary adrenal insufficiency and central hypothyroidism blunt the classic biochemical alarms of primary endocrine failure, the presentation can be misattributed to psychiatric decompensation, sepsis, or simple deconditioning — delaying a diagnosis that is, once suspected, straightforward to confirm and treat.*

Case Presentation: *A 68-year-old woman with COPD, bipolar disorder, chronic liver disease, and a recent tracheostomy for laryngeal cancer was admitted for acute hypoxic respiratory failure secondary to COPD exacerbation. Her respiratory status improved with bronchodilators, antibiotics, and steroids, but she developed progressive weakness, hypotension refractory to fluid resuscitation, and an acute change in affect — becoming mute and withdrawn, initially raising concern for depression. Laboratory evaluation revealed an inappropriately normal TSH in the setting of low free T4 and T3, a low random cortisol, and a blunted (rather than absent) response on ACTH stimulation testing — a biochemical signature pointing away from primary organ failure and toward a central process. MRI confirmed a 4 mm nonenhancing pituitary lesion. Glucocorticoid replacement was initiated before thyroid hormone repletion to avoid precipitating adrenal crisis, with prompt clinical recovery.*

Conclusion: *This case illustrates how central hypopituitarism can masquerade as psychiatric illness or refractory shock, and underscores two teaching points with direct bedside impact: thyroid function tests must be interpreted as a pair rather than in isolation, and hormone replacement sequencing — steroids before thyroid hormone — is a patient-safety issue, not an academic nicety.*

Keywords

Adrenal insufficiency, Hypotension, Pituitary tumor, Microadenoma, Macroadenoma, Central hypopituitarism.

Clinical Case

Patient is a 62-year-old woman with past medical history of laryngeal cancer status post tracheostomy not currently on any treatment, COPD, bipolar disorder, and chronic liver disease who presents to the ED with complaints of hypoxia. Your patient initially reported a fall 2 days ago with associated weakness. That time, patient continued to experience shortness of breath. The EMS was called who found the patient to be hypoxic at 70% on the seen-on room air via tracheostomy. Review of outside records indicate that patient had a tracheostomy placed last month at Piedmont Midtown due to laryngeal cancer. It appears patient was discharged home on a tracheostomy collar but patient states that she was not aware she was supposed to be using oxygen. Patient reports improvement in her shortness of breath following breathing treatments and initiation of oxygen. Patient denies any other concerns at this time.

Patient's saturations improved following initiation of oxygen via T-piece. Patient was found to be hypokalemic in the ED for which she was given repletion with potassium. CT head showed no acute

intracranial abnormalities. Chest x-ray showed no abnormalities. Patient was placed for admission to the Internal Medicine Service for further management. CT Angio chest was negative for pulmonary embolism. Circumferential wall thickening involving the upper thoracic esophagus without obstructing component. This could be inflammatory in nature. An infiltrating lesion cannot be completely excluded.

For her COPD exacerbation, she was treated with Ipratropium-Albuterol 3ml every 4h PRN, Prednisolone 40 mg oral, Antibiotics - Ceftriaxone 1g IV daily 24h, 3L oxygen pulmonary hygiene, Deep breathing exercises. We monitored her Respiratory status, oxygen saturation, work of breathing and response to treatment. She developed persistent hypotension and we checked morning cortisone random cortisol ACTH which confirmed the diagnosis of adrenal insufficiency. We started the patient on hydrocortisone 100 mg IV We also checked TSH T3-T4 which showed thyroid insufficiency. Next, we checked the ACTH stimulation test which confirmed the diagnosis of central hypopituitarism. Once the patient is stabilized, we converted to oral hydrocortisone 40 mg at 20 mg in the morning 10 mg showed afternoon and 10 mg in the evening. MRI of the brain showed 4 mm right pituitary non-enhancing focus (Figure 1). Questionable microadenoma. Correlate with hypothalamic pituitary axis dysfunction. She's advised to follow up with outpatient endocrinology for management of for pituitary microadenoma and associated adrenal insufficiency and hypothyroidism. Have potassium was decreased to 2.5 the next day and we repleted to potassium and performed regular monitoring with BMP to make sure the potassium is greater than 3. An outpatient appointment with endocrinologist as well as Oncology appointment for her laryngeal cancer was arranged. All the patient's instructions were clearly explained to the patient and the patient verbalized understanding. If symptoms recur, she's advised to return to ED for further evaluation and management.

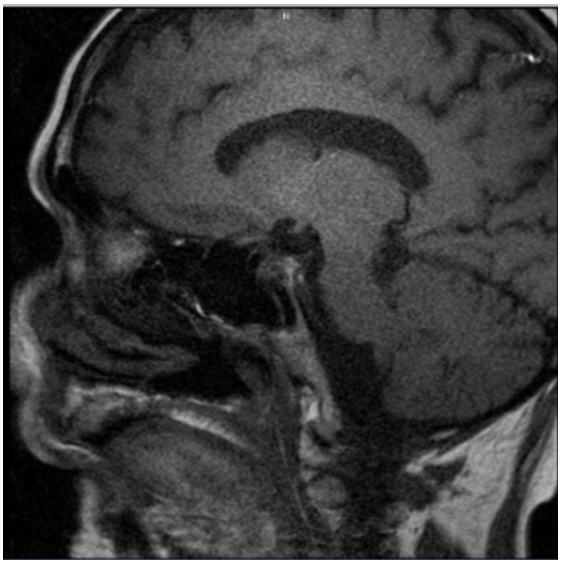


Figure 1: MRI Brain Scan showing 4mm pituitary adenoma.

Introduction

Pituitary adenomas are predominantly benign neoplasms arising from adenohypophyseal cells and represent a common cause of sellar lesions. They may present as functioning tumors due to excess hormone secretion, as nonfunctioning lesions, or through manifestations related to mass effect and pituitary hormone deficiency. Pituitary adenomas measuring less than 10 mm are classified as microadenomas and are frequently discovered incidentally on neuroimaging performed for unrelated indications [1,2].

Hypopituitarism is more commonly associated with pituitary macroadenomas because larger lesions can compress normal pituitary tissue and interfere with hypothalamic-pituitary function. In contrast, hormonal deficiencies are considerably less common in patients with pituitary microadenomas, particularly very small lesions. Even so, the degree of pituitary dysfunction does not track reliably with tumor dimensions, and biochemical deterioration can occur independently of measurable growth [3]. Nevertheless, pituitary dysfunction can occur even in patients with microadenomas, highlighting the importance of biochemical evaluation when clinical findings suggest hormonal deficiency regardless of lesion size [2].

Central adrenal insufficiency is an important manifestation of hypothalamic-pituitary dysfunction because cortisol deficiency can present with nonspecific findings, including fatigue, weakness, and hypotension, and may become clinically significant during acute illness. Concurrent central hypothyroidism may provide additional evidence of anterior pituitary dysfunction. Here, we describe a patient presenting with persistent hypotension during hospitalization who was subsequently found to have adrenal insufficiency and thyroid-axis dysfunction, prompting pituitary imaging that demonstrated a small pituitary lesion suspicious for a microadenoma.

Discussion

We present a clinical case where patient presented with hypotension. Workup including morning cortisol, ACTH stimulation test and thyroid function tests revealed the presence of adrenal insufficiency and hypothyroidism, thus raising the suspicion for central mediated causes for patient's symptoms. Accordingly, an MRI brain revealed the presence of pituitary microadenoma 4mm. Patient is subsequently referred to endocrinology outpatient follow up for evaluation and management of her pituitary microadenoma.

Pituitary adenomas account for 17.2% of the all-central nervous system brain tumors. There are the third most common brain tumors next to gliomas and meningiomas, with them being regarded as most common neuroendocrine tumors [4,5]. Pituitary adenomas are classified into microadenomas (< 10 mm), macroadenomas (> 10 mm) and gaint adenomas (> 40 mm) [6,7]. Their incidence and prevalence are 16.7% and 14.4% respectively [8-11]. Depending on their secretory capacity, these are further classified into functioning and non-functioning adenomas. Functioning pituitary adenomas are further classified based on the hormone secreted

– Somatotroph adenomas (GH, 11%), Corticotroph adenomas (ACTH, 5%), Thyrotroph adenomas (TSH), gonadotroph adenomas (FSH & LH), Prolactinomas (Prolactin, 50%) and Nonfunctioning adenomas (30%) [12,13]. Pituitary adenomas manifest itself due to either hormonal hypersecretion, hormonal hyposecretion or mass effect on the neighboring critical brain structures. It is not uncommon for them to go undetected to escape detection, until they are being detected in the brain scans ordered for other clinical presentations in older patients (30-60 years) [12]. In such clinical cases, these are asymptomatic and are designated as incidentalomas [12]. If they do become symptomatic, they most commonly present with symptoms of hormonal excess or deficiency (prolactin, TSH, ACTH, GH, FSH and LSH), headache, visual disturbances, nausea, and vomiting. Rarely, some of the pituitary adenomas develops internal hemorrhage, thereby presenting with mass effect or hormonal deficit [12].

The pathophysiology has been understood due to combination of familial preponderance, epigenetic changes and mutations [12-15]. In few cases, disruption of physiological cell changes due to epigenetic can be partially responsible for its causation [14]. In other instances, mutations including germline and somatic mutations can be implicated [12]. MEN1 [Multiple endocrine neoplasia type 1], MEN4 [Multiple endocrine neoplasia type 4], Carney complex and Familial isolated pituitary adenomas are some of germline mutations that are implicated in the causation of pituitary adenomas [12,16-18]. Our patient also has a past history of laryngeal cancer. A speculative hypothesis for inception of pituitary tumor would be paraneoplastic syndrome where anti-tumor autoimmune assault against the pituitary gland can be blamed for its occurrence [19]. In this regard, antigen mimicry between the tumor cells and anterior pituitary cells can serve as a springboard for initiation and propagation of tumorigenesis [20].

A pituitary adenoma secreting GH becomes symptomatic with sequelae of GH excess including increased limb size, macroglossia and increased jaw size whereas TSH secreting adenoma is characterized by as palpitation, excessive sweating, arrythmias, and diarrhea [21]. ACTH secreting pituitary might be characterized by headache, dizziness, double vision, mydriasis and nasal stuffiness [22-24]. Prolactin secreting adenomas might be associated galactorrhea, loss of libido, infertility, menstrual irregularities, obesity and metabolic syndrome [25]. In the contrary, pituitary adenoma might compress the pituitary tissue, thus interfering with hypothalamic signals reaching the anterior and posterior pituitary [26]. The later mechanism is the underlying reason for patients presenting with symptoms of anterior or posterior hormone deficiency including adrenal insufficiency, or thyroid deficiency as described in our current clinical case.

Diagnostic imaging, including MRI and CT scans, plays a crucial role in assessing pituitary macroadenomas' size, location, and extent [27]. Patient clinical presentation with signs of pituitary hormones over secretion or under secretion, should prompt clinical investigation to diagnose the central causes of hyper or hypo pituitarism. In this regard, MRI and CT scan brain would be helpful and provide the necessary information regarding the size, location and extent of pituitary adenoma [27]. Classifying pituitary adenoma based on the size is important as helpful in making further clinical decisions on future course of clinical management.

Non-functioning pituitary microadenomas are usually monitored with serial MRI imaging performed every 2-3 years [27,28]. In the contrary, pituitary macroadenomas are usually monitored with MRI imaging at 1-year intervals with those specifically close to the optic chiasm monitored at 6 months intervals [27,28].

However, pituitary adenomas associated with visual disturbances, progressive enlarging tumor masses, active hemorrhage with adenoma, or loss of endocrine function should be recommended for surgical intervention [28]. The preferred modality of surgery in these cases is the trans-nasal trans-sphenoidal endoscopic approach which is associated with lesser morbidity and mortality. Transspenoidal surgery is the first line of treatment GH oversecreting, ACTH oversecreting and TSH oversecreting pituitary adenomas [28]. In contrast, medical management is the primary preferred management in prolactin adenomas with surgery reserved for medically refractory cases [28]. Active collaboration between endocrinologists, neurologists and radiologists in imperative for improving clinical outcomes and prognosis in surgically treated cases [27,28].

Prolactin oversecreting adenomas can be treated with dopamine agonist such as bromocriptine or cabergoline [28,25]. GH hormone over secreting adenomas usually respond to somatostain analogs such as octreotide [29,30]. Radiation therapy is usually resorted when tumor recurs despite surgery and failure of medical management [28].

Future Research

Pertinently it has to be emphasized that focused research particularly on the surgical techniques, peri-operative care and post-operative care of pituitary adenomas is indispensable for bringing to light non-invasive, less expensive and novel therapeutic approaches for enhancing the standard of care and prolonging life in these patients [27]. Lastly, bench research to delve into the downstream signaling pathways responsible for triggering the origination and progression of these pituitary adenomas needs to be prioritized. This will uncover molecular targets upon which novel therapeutic interventions can be designed. Clinical trials based on these newly developed therapeutics can bring new treatment modalities to the clinic

Take home messages

- Pituitary adenomas are the third most common brain tumors after meningiomas, gliomas.
- Pituitary adenomas are classified into microadenomas and macroadenomas based on their size.
- Pituitary adenomas are further classified based on the hormone secreted into prolactinomas, GH or ACTH or TSH secreting adenomas.
- In few instances they squeeze the pituitary tissues thus

impairing the blood supply and barricading the hypothalamic pituitary pathways, thus giving rise to hypopituitarism.

- In patients presenting with hypotension, a thorough work up should be initiated to exclude central causes including hypopituitarism.
- MRI/CT scan should be informational in providing the size, location and extent of tumors
- Trans-sphenoidal surgery is the preferred line of treatment for macroadenomas as well as GH oversecreting, ACTH oversecreting and TSH oversecreting pituitary adenomas.
- Medical management with dopamine agonists are the preferred treatment modalities for prolactin secreting adenomas.
- Active collaboration between endocrinologists, neurologists and radiologists is indispensable for improving clinical outcomes in these patients.
- Lastly, clinical research for refining surgical techniques will be beneficial in enhancing clinical recovery and reducing post operative complications.
- Bench research in pathophysiological basis of pituitary adenomas would uncover new molecular targets for crafting new therapeutic modalities in pituitary adenomas.

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