

Acne, Anxiety, and Adenoma

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Introduction

Cushing's syndrome is defined as a collection of signs and symptoms due to prolonged exposure to glucocorticoids, namely cortisol. Whereas Cushing's disease describes when Cushing's syndrome is caused by an excess of ACTH, likely from a pituitary adenoma. This case highlights notable signs, symptoms, and recognition of Cushing's disease, inpatient workup, and inpatient management. Additionally, this emphasizes the importance of maintaining a broad differential diagnosis and the role family physicians play in facilitating patient care.

Case Study

AM is a 22-year-old female with no significant PMH, complaining of 3-4 months of increasing anxiety, weakness, headaches with blurred vision, facial acne, weight gain, and more abundant stretch marks, who presented to our ED on the recommendation of her endocrinologist. A few months prior, her PCP referred her to a dermatologist and was given a trial of SSRIs, which did not improve symptoms. Additional outpatient labs for PCOS, revealed elevated testosterone. She was subsequently referred to endocrine. Further lab findings prompted the specialist to refer the patient to the hospital for immediate treatment. Her blood pressure had been increasing over the last month as well. Of note, the patient has a history of Phentermine use >1 year ago.

Physical Exam:

Vitals: T 98.3 HR 82, RR 18, BP 174/134 100% on RA
General: Well developed, female with rounded facies, facial hirsutism, facial acne, with central obesity
Cardiovascular: RRR. Normal S1/S2. No murmurs/rubs/gallops or S3/S4 noted
Respiratory: CTAB. No wheezes/rhonchi/rales
Gastrointestinal: Soft, NTND
Musculoskeletal: Moving all 4 limbs. Normal strength and tone.
Extremities: Thin appearing UE and LE compared to abdomen. No edema of peripheral extremities
Neuro: Alert and oriented. No focal deficits. CN II-XII grossly intact. Strength 5/5 bilateral UE & LE
Skin: multiple dark lower abdominal and lower back striae; erythematous patches noted on face, throughout back, and lower extremities

Diagnosis and Management

DDx:

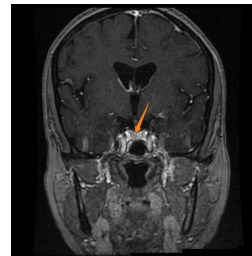
Cushing's Syndrome vs. Cushing's Disease, consider adrenal mass as possible cause vs. exogenous use

Labs:

CBC unremarkable. CMP for mild hypokalemia. Free cortisol urine: 41H, AM cortisol (post dexamethasone) 16.7H, Prolactin 18.4 wnl, ACTH 56.6H, DHEA-S 322.0 wnl

Imaging:

MRI Pituitary: No large pituitary masses were seen. There is a small area of decreased enhancement within the left side of the pituitary on image 8 of series 12 measuring 2 mm. This may represent an artifact, or a tiny pituitary microadenoma. The remainder of the brain was normal.



Final Diagnosis:

Cushing's Disease 2/2 pituitary microadenoma

Treatment/Management:

Inpatient endocrine and neurosurgery were consulted immediately. After further imaging, the patient underwent urgent transsphenoidal resection of the pituitary. She was started on hydrocortisone replacement therapy, and was discharged from the hospital shortly after, with slow improvement in symptoms overall. She currently continues low dose steroids and has close follow up with PCP and endocrinology.

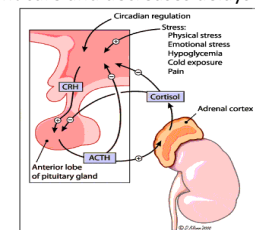


Discussion

Corticotropin releasing hormone (CRH) is released from the hypothalamus, which stimulates ACTH secretion from the anterior pituitary gland. ACTH signals positive feedback to the adrenal cortex, which releases cortisol and regulates this pathway via a negative feedback loop to the hypothalamus. In Cushing's disease, a pituitary adenoma can signal excessive ACTH release, leading to high levels of cortisol.

The family physician played an important role in first recognizing the patient's earliest symptoms. Patients with Cushing's disease, can present with signs such as depression, increased striae, decreased libido, menstrual irregularities, acne, hirsutism, fatigue, and most commonly—weight gain. Given the wide range of symptoms, it is easy to overlook the primary diagnosis. This patient had initially been referred to multiple specialists, but after subsequent workup, revealed hypercortisolism. If hypercortisolism is not attributed to exogenous use, further workup is warranted. The family physician inpatient team enabled multispecialty care during admission, which lead to urgent surgical intervention.

Ultimately, this case underscores the importance of maintaining a broad differential. When working up a patient with such physical exam and biochemical findings, avoidance of anchoring and recognizing personal bias helps facilitate adequate patient care and decreases delays in diagnoses.



References

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