Case Report: A Brief Study of Prolactinoma

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Abstract: <u>Background</u>: Pituitary tumors are common in sellar area. The prevalence of clinically apparent pituitary lesions is estimated to comprise approximately 10% of all intracranial lesions, while incidental pituitary tumors are detected in approximately 11% of individuals at autopsy. Pituitary tumors are mostly found to be benign adenomas, however pituitary carcinoma has been reported to comprise about 0.5% of pituitary tumors. <u>Aims and Objectives</u>: To study the MRI features, Lab investigation to rule out pituitary macroadenomas. <u>Materials and Methods</u>: We studied clinical history, laboratory and MRI features of 35 year old female with features of pituitary macroadenomas who presented to King Gerge Medical University, Lucknow. MRI was carried out .Other significant lab investigation was done in repeated series that help in making diagnosis of prolactionoma.

Keywords: Pituitary adenoma, diagnosis, Serum prolactin

1. Introduction

PROLACTINOMAS represent the most common type of pituitary tumor. However, they are relatively rare in males; more than 70% of the cases occur in women. As a consequence, only a few large series of men with prolactinomas have been reported, most of them more than 10 yr ago. From previous reports, it is clear that the clinical setting of PRL-secreting pituitary tumors varies greatly with sex. Women usually present with microadenomas revealed by the classic amenorrhea-galactorrhea syndrome. The clinical presentation of hyperprolactinemic men is much more polymorphic and can be misleading . Impotence and decreased libido are the most frequent symptoms, but the diagnosis is often made when signs of compression due to the tumor developbrain herniation, requiring emergent ventriculostomy and intubation, led to the clinical suspicion of a more sinister diagnosis. Transnasal biopsy of the mass was consistent with sinonasal neuroendocrine carcinoma, and chemotherapy was planned. Laboratory testing, however, revealed an elevated prolactin (27,400 ng/mL, after 1:100 dilution). Re-review of pathology with additional immunohistochemical staining was requested and confirmed the diagnosis of prolactinoma.

2. Case Study

A 35-year-old female who was previously healthy presented to the medicine department with acute history of headache, visual blurring, and vomiting following a one month history of intermittent frontal headache and blurred vision, no galactorrhea or gynectomastia. She was not taking any medication. Her family history was unremarkable for endocrinopathies. Vital signs at the time of admission showed blood pressure 118/65 mmHg, heart rate 74/min, temperature 36.8C.

Physical examination showed a Glasscow Coma scale of E4V5M6.

CT/MRI showed a well defined enhaching space occupying lesion in sellar and suprasellar causing widening of the

sellaturcica with feature as described suggesting a neoplastic etiology (possibility of Pitutarymicroadenoma)

Laboratory investigations (Table 1) revealed high prolactin (PRL) level of 250.12 ng/ml, thyroid stimulation hormone (TSH) 3.76 mu/L and free thyroxin (F T4) 0.8ng/dl (indicating secondary hypothyroidism).Serum cortisol 5.9 nmoL/L ($0.3 \mu g/dl$) (indicating central adrenal insufficiency), luteinizing hormone (LH) 0.09 mIU/L, follicular stimulation hormone (FSH) 8.79 mIU/L, and testosterone 0.91nmoL/L (181.1ng/dl).

Liver and renal function were normal with serum glucose 4.4mmol/l (79mg/dl). Serum sodium 137mmol/l with normal serum osmolality.

During follow up patient has no significant symptoms and sign of orthostatic hypotension, vertigo, nausea and vomiting. Importantly, the left eye vision was partially restored with remaining temporal hemianopia, and complete improvement of the right eye vision. Growth velocity and puberty continued to progress normally. Unfortunately the patient lost to follow up after the 6 months visit. The family were contacted several but they did not respond.

3. Result

A series of serum prolactin test done using 5 different dilution have values we got was 210.75 ng/ml (on 1:5 dilution), 214.30 ng/ml (on 1:10 dilution), 214.25 (on 1:25 dilution), 229.5 ng/ml (on 1:50 dilution), 238.0 ng/ml (9on 1:100 dilution) and 187.84 ng/ml was obtained on undilution.

Every result show high level of serum prolactin along with low serum LH along with CT findings help in making the diagnosis of prolactionoma (Pituitary macroadenoma)

4. Discussion

Pituitary tumors comprise 10-15% of intracranial neoplasm. ^[3] They can be broadly classified on the basis of

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tumor size. Micro-adenomas are less than 10 mm in diameter and those of more than 10 mm are called macroadenomas. The tumors can also be classified as chromophobic, acidophilic and basophilic adenoma on the basis of their histologic appearance. On the basis of immunohistochemical staining or by serum hormone measurement, tumors can be divided into secreting and nonsecreting types. The secreting (functional tumor) comprises of 75% of pituitary adenomas. They include:

- Growth hormone (GH) cell adenoma
- PRL cell adenoma/prolactinoma
- Mixed GH and PRL adenoma
- Thyrotropin releasing hormone cell adenoma
- Adrenocorticotropic hormone (ACTH) cell adenoma
- Gonadotroph (LSH) and (follicle-stimulating hormone) cell adenoma.

Symptoms of these pituitary neoplasms depend on the presence of pituitary hypersecretion or hyposecretion caused by destruction of pituitary gland or direction of tumoral expansion and invasion of adjacent structures.

Very occasionally, some pituitary tumors demonstrate their functional differentiation toward the production of hormones belonging to different cell lineages, i.e., ACTHomas with GH production, GHomas with ACTH production. It has been postulated that aberrant expressions of transcription factors could be the cause of this abnormal differentiation in the tumors.

Prolactinomas constitutes 40% to 50% of pituitary adenoma. PRL secreting micro-adenomas generally occur in reproductive-aged females and they manifest with amenorrhea, galactorrhea or both. In males and postmenopausal females, prolactinomas often appear to be clinically non-functional, growing to macro-adenoma and exhibit invasion. Due to various syndromes produced by secreting tumors, they are detected early. Non-secreting tumors are larger when diagnosed and present with various symptoms and signs such as headache, visual field defects, typically bi-temporal field loss and cranial nerve palsies, due to invasion into cavernous sinus or with epistaxis due to downward extension through the floor of sella. The mass can extend to orbit leading to proptosis. They can present with sudden onset of headache/loss of vision due to hemorrhage or necrosis of tumor as pituitary apoplexy. [7]

The diagnosis of prolactinoma is based on measurement of serum PRL level and neuroradiological imaging. Hyperprolactinemia at level less than 150 ng/ml does not indicate tumoral PRL production. Instead it may be the result of stalk section effect.

5. Questions

1) What are diagnostic features of prolactinoma ?

Laboratory evaluation should include a repeat serum prolactin test, measurements of TSH and free T4, and a pregnancy test. (A serum testosterone level should be checked in men.) If the results come back normal and if other diagnoses are excluded, the most likely diagnosis is a prolactinoma. In this case, a pituitary MRI should be obtained. Visual field testing can be performed in individuals with specific visual complaints, especially loss or impairment of peripheral vision.

2) What are the causes of prolactinoma?

There are many causes of hyperprolactinemia. Factors that can increase prolactin secretion include pregnancy, nursing, physiologic stress, estrogen use, polycystic ovary syndrome, hypothyroidism, and chronic renal or hepatic failure. Head trauma, use of certain medications (verapamil, neuroleptics, antipsychotics, and antidepressants), and presence of nonsecretorysellar or suprasellar masses can also increase prolactin levels.

3) What is the incidence of prolactinoma in the general population?

Prolactin-secreting adenomas, or prolactinomas, are the most common type of pituitary adenoma, accounting for approximately 60% overall.¹ They occur at a frequency of six to 10 cases per million each year.² Prolactinomas are almost always benign; malignant tumors are extremely rare.

4) Classify prolactinoms?

Tumors are classified as microadenomas or macroadenomas, depending on the size. A microadenoma is defined as an intrasellar mass less than 10 mm in diameter. A macroadenoma, defined as larger than 10 mm in diameter, can cause enlargement of the sellaturcica. The larger the size of the prolactinoma, the greater the prolactin level and higher the likelihood of mass-effect symptoms.

5) What are the differential diagnosis ?

Differential diagnoses of pituitary adenoma include other brain tumours, such as craniopharyngioma, meningioma, glioma, bilateral tilted disc syndrome, optic nerve coloboma, nasal retinoschisis, nasal retinitis pigmentosa and masquerade syndromes (chronic retrobulbar optic neuritis, nutritional amblyopia, uncorrected refractiveerror, normal tension glaucoma and agerelatedmaculopathy.

6. Conclusion

Pituitary tumors are most frequently encountered sellar neoplasms. They exhibit a wide range of biological behavior in terms of hormone production and tumor growth. The young patients with pituitary adenoma should be thoroughly evaluated for the association with genetic syndromes such as MEN-1, FIPA, CNC and McCune-Albright syndrome. The family of a young patient diagnosed with pituitary adenoma as a part of genetic syndrome should be offered genetic counseling. Recent advances in immunohistochemistry and molecular techniques have improved our concepts regarding pathogenesis of tumors. Classification of these tumors is likely to develop in future with growing knowledge of pathways of adenohypophysealcy to differentiation.

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