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Case Study

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A CASE REPORT ON PITUTARY MICRO ADENOMA

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ABSTRACT

The tumors that occur in the pituitary gland are referred to as adenomas out of which they are classified on the basis of the size of growth. Mostly, Pituitary tumors are seen in the sellar region. The prevalence of pituitary lesions comprises to about 10 percent of all intracranial lesions and most of them are benign in nature but 0.5 percent of pituitary tumors are reported as carcinogenic. The development in the medical sciences in relation with improvement in diagnostic examination and advances in treatment resulted in the successful therapeutic outcome in dealing with these tumors. A case report regarding pituitary microadenoma of a 29-year-old female is discussed here with details of her clinical presentation.

KEYWORDS: Pituitary Microadenoma, therapeutic outcome.

INTRODUCTION

The tumors that occur in the pituitary gland are referred to as adenomas out of which they are classified on the basis of the size of growth. If the size of the tumor is below 10mm then it is characterized as microadenoma whereas those above 10mm of size are named as macroadenomas.[1] Mostly, Pituitary tumors are seen in the sellar region. The prevalence of pituitary lesions comprises to about 10 percent of all intracranial lesions and most of them are benign in nature but 0.5 percent of pituitary tumors are reported as carcinogenic.[2] Diffuse adenomas are the major reason for sellar expansion as it compresses the residual gland into a thin membrane. On the contrary, massive adenomas often replace the sellar floor and displace surrounding structures and undergo suprasellar extension. Most frequently microadenomas are frequently diagnosed as a result of investigating hormonal imbalance.

The development in the medical sciences in relation with improvement in diagnostic examination and advances in treatment resulted in the successful therapeutic outcome in dealing with these tumors. A case report regarding pituitary microadenoma of a 29-year-old female is discussed here with details of her clinical presentation.

CASE REPORT

A 29 year old female patient presented to General Medicine Department with complaints of episodic palpitations, sweating, flushing, wheezing, headache and decreased vison for about 6 months. Moreover, she was having cyclical Cushing's syndrome, Diabetes mellitus, Bronchial Asthma, Hypertension and Seizures for last 2 years. General examination was found to be Normal. No thyroid enlargement was found. Systemic examination was found to be normal. Ocular examination revealed that visual acuity in the right eye was 6/6, in the left eye was 9/36 respectively. Extraocular movements were of full range.

On investigation MRI showed focal convex bulge in the left side of the pituitary gland in its posterior aspect with an ill-defined lesion which stand out as an area of decreased enhancement on dynamic contrast sequencers representing microadenoma.

Hormonal assay was carried out and cortisol level were found to be 37ng/ml (normal range 5-25ng/ml), prolactin level was found to be 42ng/ml (normal value 2-29 ng/ml). Thyroid profile was found to be normal and with the light shed by the investigations the clinical diagnosis of Pituitary microadenoma was confirmed and initiated conservative management.

DISCUSSION

About 10-15 % of intracranial neoplasm are constituted by pituitary tumors.[2] They can be broadly classified on the basis of tumor size. The tumors can also be classified as chromophobic, acidophilic and basophilic adenoma on the basis of their histologic appearance. Besides this, tumors can be classified as secreting and non-secreting types based on immunohistochemical staining or by serum hormone measurement. The secreting type of functional tumor constitutes about 75 percent of the total.[2] These include Growth hormone (GH) cell

adenoma, PRL cell adenoma or prolactinoma, Thyrotropin releasing hormone cell adenoma and so on.

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.[3] Prolactinomas constitutes 40% to 50% of pituitary adenoma. Prolactin secreting micro-adenomas generally occur in reproductive-aged females and they manifest with symptoms such as amenorrhea, galactorrhea or both. In males and post-menopausal females, prolactinomas often appear to be clinically non-functional which eventually grows to macro-adenoma and exhibit invasion. The secreting tumors are detected early due to various syndromes produced whereas Non-secreting tumors are larger when diagnosed and present with varying symptoms and signs such as headache, visual field defects, typically bi-temporal field loss and cranial nerve palsies which is due to invasion into cavernous sinus or with epistaxis due to downward extension through the floor of sella.[4] The mass can extend to orbit leading to proptosis.[5] They can present with sudden onset of headache/loss of vision due to hemorrhage or necrosis of tumor as pituitary apoplexy.[6]

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 prolactin production. Instead, it may be the result of stalk section effect.Pituitary adenomas are recently classified by their hormonal content. The hormonal activity is the basis for the diagnosis and treatment from the clinical perspective. Majority of pituitary adenoma formation is dependent on a no of oncogenes and tumor suppressor gene such as cyclinD1, multiple endocrine neoplasia type 1 (MEN-1), RAS, P53, retinoblastoma gene.[7] Pituitary adenomas that occur in a familial setting account for 4-5% of all pituitary adenomas.[8] They can be a part of endocrine related tumor syndromes such as MEN-1.

CONCLUSION

The tumors found in the pituitary gland 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 and so on. The family of a young patient diagnosed with pituitary adenoma as a part of genetic syndrome should be offered genetic counseling which may help in improving the conditions of others in the family. Conservational treatment can help in alleviating the existing difficulties and surgical prophylaxis could be advised if there occurs further progression in the size of the existing adenoma.

REFERENCES

1. Vasenwala SM, Ansari H, Basari R, Srivastava V K. Pituitary adenoma: A case report with special emphasis on approach to diagnosis. Clin Cancer Investig J., 2014; 3: 264-7.
2. Raju KV and Ruby MV. Pituitary macroadenoma: A case presentation. Kerala Journal of Ophthalmology, 2006; 18: 58-60.
3. Ezzat S, Asa SL, Couldwell WT, Barr CE, Dodge WE, Vance ML, *et al*. The prevalence of pituitary adenomas: A systematic review. Cancer, 2004; 101: 613-9.
4. Kovacs K, Horvath E. Pathology of pituitary tumors. Endocrinol Metab Clin North Am, 1987; 16: 529-51.
5. CPMC neurosurgery pituitary tumour, Department of Neurosurgery, Columbia Presbyterion (Medline).
6. Riskind PN, Richardson EP. Clinicopathologic conference-SN. N Engl J Med., 1986; 314: 229.
7. Landis CA, Masters SB, Spada A, Pace AM, Bourne HR, Vallar L. GTPase inhibiting mutations activate the alpha chain of Gs and stimulate adenylyl cyclase in human pituitary tumours. Nature, 1989; 340: 692-6.
8. Kovals K, Horvath E. Tumours of the pituitary gland. In: Hartman WH, Sobin LH, editors. Atlas of Tumour Pathology. Second series; Fascicle 21. Washington, D.C: Armed Force Institute of Pathology, 1986.
9. Tahara S, Kurotani R, Ishii Y, Sanno N, Teramoto A, Osamura RY. A case of Cushing′s disease caused by pituitary adenoma producing adrenocorticotropic hormone and growth hormone concomitantly: Aberrant expression of transcription factors NeuroD1 and Pit-1 as a proposed mechanism. Mod Pathol, 2002; 15: 1102-5.
10. Vandeva S, Jaffrain-Rea ML, Daly AF, Tichomirowa M, Zacharieva S, Beckers A. The genetics of pituitary adenomas. Best Pract Res Clin Endocrinol Metab, Jun, 2010; 24(3): 461-76.
11. Molitch ME. Diagnosis and Treatment of Pituitary Adenomas: A Review. JAMA, Feb 07, 2017; 317(5): 516-524.