Brief Communication

Tuberous sclerosis: An uncommon cause of hyperprolactinemia

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ABSTRACT

Tuberous Sclerosis is a multi system genetic disorder affecting skin, central nervous system,kidney,heart and lungs. We present a case report of a 26 year old female with tuberous sclerosis who presented with galactorrhea and menstrual irregularities due to hyperprolactinemia.

Key words: Tuberous sclerosis, hyperprolactinemia, galactorrhoea

Introduction

Tuberous sclerosis (TSC) is a multi system genetic disorder affecting skin, brain/nervous system, kidneys, heart and lung and leading to growth of non malignant tumors. A combination of symptoms may include seizures, developmental delay, behavioral problems, skin abnormalities, lung and kidney disease. Tuberous sclerosis lesions infrequently cause symptoms in the endocrine, gastrointestinal (GI), and lymphatic systems. We hereby report a case of tuberous sclerosis presenting with hyperprolactinemia.

Case Report

A 26-year-old female presented with galactorrhea for the past 10 days and menstrual irregularities over the past six months. Galactorrhea was spontaneous. Her last childbirth four years ago was uneventful. She had no head ache, vomiting, and visual impairment. She denied any history of substance abuse, drug intake (antipsychotic, anti emetic, hormonal preparations), hypothyroidism, chronic liver or kidney diseases, epilepsy. There was no significant family history. She was a well nourished female with mild pallor, tiny nodule on face, subungual fibroma in hands. There was spontaneous galactorrhea and mildly tender breasts without any signs of inflammation. Systemic examination was entirely normal with normal IQ. Ophthalmologic evaluation revealed white disk shaped retinal hamartoma. Routine laboratory investigations including renal and liver function tests, thyroid profile were normal. Serum prolactin was 85 ng/mL with FSH-4.66 and LH- 4.21 mIU/ml. In view of the theoretical possibility of involvement of cell groups of other anterior pituitary lineage, tests for evaluation of other anterior pituitary hormones were carried out and were found to be normal. Abdominal and pelvic ultrasound revealed no abnormality. Chest X-ray showed bilateral interstitial infiltrates. Echocardiogram of heart was normal. Computed tomography (CT) scan revealed multiple intracerebral calcifications. These calcified lesions/subependymal hamartomas are seen along the lateral surface of the lateral ventricles giving rise to characteristic candle dripping appearance. Magnetic resonance imaging (MRI) of the brain ruled out the presence of any pituitary mass. The combined clinical scenario along with the radiologic findings leads to the diagnosis of TSC with hyperprolactinemia. Patient was prescribed cabergoline 0.5 mg twice daily, which resulted in amelioration of galactorrhea and regularization of menses.

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Discussion

There is a growing body of evidence that TSC patients develop hormone secreting tumors involving the neuroendocrine system at higher frequency than the general population. Cushing's disease,[1] hypoglycemia secondary to insulinomas,[2] precocious puberty, thyrotoxicosis, hypercalcemia secondary to parathyroid adenomas,[3] hyperprolactinemia[4] and acromegaly have all been reported in TSC patients. The circulating prolactin of our patient may be of pituitary origin or may possibly be secreted ectopically by a hamartoma. There has been evidence linking neuroendocrine tumors to the AKT/mTOR/S6 kinase pathway that is regulated by the hamartin/tuberin (TSC1/TSC2) complex. Thus, it is beginning to appear that the occurrence of these neuroendocrine tumors (NET) in TSC is more than random coincidence.

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