Coexistence of pituitary macroadenoma with systemic lupus erythematosus and antiphospholipid syndrome

Sir,

Prolactin is a cytokine like hormone that is associated with autoimmune diseases such as systemic lupus erythematosus (SLE) and celiac disease.\(^1\) It causes proliferation of immune cells and cytokine release.\(^1\) Hyperprolactinemia is seen in 20–30% of SLE patients.\(^2\) Some case reports of SLE associated with prolactinoma are found in the literature, but there is no cause and effect relationship between them.\(^2,3\) The degree of hyperprolactinemia may be associated with SLE activity and this finding may have a therapeutic role in the management of patients.\(^2,4\) Elevated prolactin levels in SLE may result from a nonfunctioning pituitary tumor.

A 35-year-old woman presented with hemiplegia of left upper limb and dysarthria. Dysarthria was resolved spontaneously after half an hour. Past medical history was positive for three spontaneous abortions, Bell's palsy, appendicectomy, and one episode of seizure. Her menstrual cycle was regular. She had reduced force of proximal and distal muscles of left upper limb, increased deep tendon reflexes (DTR) of the same limb and right side Bell's palsy on examination. Vital signs were normal. Consciousness, sensation, Romberg, vibration, and cerebellar tests were all intact, but cognition was minimally impaired. Brain magnetic resonance imaging (MRI) revealed mild hydrocephaly with abnormal hyperintense lesions in periventricular white matter, centrum semioval, precentral and postcentral gyrus in T2-weighted images [Figure 1]. Cerebellar white matter was also involved and an abnormal mass in pituitary fossa was discovered. MRI of sella turcica in sagittal and coronal views demonstrated pituitary macroadenoma with chiasmatic compression [Figure 2a and b]. The findings on brain magnetic resonance angiography and magnetic resonance venography (MRA and MRV) were normal. Electromyography and nerve conduction velocity (EMG-NCV) showed upper motor lesion in left side upper extremity.

Laboratory results showed the following: WBC 3300/μl [72% polymorphonuclear leukocytes (PMN) and 18.7% lymphocytes], lymphocytes 617/μl, hemoglobin (Hb) 8.9 g/dl, mean cell volume (MCV) 75, hematocrit (HCT) 26.4%, platelet (Plt) 46,000/μl, reticulocyte count (Ret C) 0.3%, erythrocyte sedimentation rate (ESR) 55 mm/hour, C-reactive protein (CRP) +1, ferritin 64 ng/ml (normal level 7.4–73), fibrinogen degradation products (FDP) 0.18 mg/dl (up to 0.2) and lactate dehydrogenase (LDH) 802 U/l (normal level <480). Blood sugar, calcium, magnesium, urine SG, renal and
liver function tests and 24-hour urine protein level were all normal.

Endocrine and rheumatologic tests showed mild hyperprolactinemia [prolactin 31.6 ng/ml (normal level 4.8–23.3)], normal thyroid function, normal C3 and C4 levels, positive antinuclear antibody (ANA; 1.35 IU/ml, normal level <1), positive anti-ds DNA (2.2 IU/ml, normal level <0.9), high anticardiolipin (IgG) level (78 GPL/ml, normal level <15), high titer of antiphospholipid Ab (IgG) (106 U/ml, normal level <10), positive lupus anticoagulant and negative antineutrophil cytoplasmic and perinuclear antibody (P-ANCA and C-ANCA).

The diagnosis of antiphospholipid syndrome (APS) secondary to SLE and nonfunctioning pituitary macroadenoma (NFPA) was made. The diagnosis of SLE was based on the presence of high titers of ANA, positive anti-ds DNA, leukopenia (lymphopenia) and CNS involvement. Because of mildly elevated prolactin level and on the basis of MRI finding, the diagnosis of NFPA was also made. Hyperprolactinemia in this patient originated from a nonfunctioning pituitary tumor. Hyperprolactinemia in SLE cannot be explained by the autoimmune nature of the disease or by its other known complications. We cannot confidently comment on the cause and effect relationship between hyperprolactinemia and the risk of SLE occurrence. We conclude that regardless of the autoimmune nature of SLE, pituitary imaging should be performed in all SLE patients with any degree of hyperprolactinemia to search for pituitary tumors.

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Sheehan’s syndrome: Presented with hyponatremia and hypoglycemia after 14 years from delivery

Sir,
Sheehan’s syndrome (SS) is postpartum pituitary necrosis due to bleeding and hypovolemia. According to the degree of damage to the pituitary, the symptoms may occur immediately or later. The main symptoms are loss of libido, cessation of lactation, and secondary amenorrhea. The first sign of symptoms may associate with hypoglycemia and hyponatremia. There are reports that it is a relatively common cause of hypoglycemia and hyponatremia. SS should be considered in the differential diagnosis of hyponatremia and hypoglycemia.

A 49-year-old woman was admitted to our clinic with weight loss, nausea, and vomiting. We learned that currettage had been performed because of stillbirth and total abdominal hysterectomy had been performed because of excessive bleeding 14 years ago. Hypoglycemia and hyponatremia were detected. Hormone profiles including cortisol, plasma ACTH, thyroid hormones, TSH, FSH, LH, and prolactin were all low. SS was suspected and confirmed by a pituitary and cerebral MRI which showed an empty sella turcica. We diagnosed pituitary insufficiency and started essential hormone replacement treatment. The patient’s clinical picture improved dramatically after hormone replacement therapy. It is difficult to determine the real incidence of SS because of geographic and racial differences.

We want to highlight the fact that Sheehan’s is seen not only in developing countries, but also in Europe. SS may manifest long after the delivery with hypoglycemia and hyponatremia like our case.