Obesity as the Presenting Feature of Sellar-Suprasellar Tuberculoma

Sir,

A 9-year-old boy presented with intense hyperphagia, rapid weight gain (23 kg over 3 years), and diminution of vision. His daily food intake had trebled over 3 years, and he showed aggressive behavior if the food was denied. The loss of vision was gradual, painless, and bilateral. Parents also noted roughening and darkening of the skin in the flexural areas, and snoring during sleep for 6 months. There was no history of a headache, vomiting, seizures, fever, or contact with tuberculosis. Physical examination showed generalized body fat distribution and acanthosis nigricans in neck and axillae [Figure 1a and b]. His weight, height, and body mass index were 46 kg (+2.53 Z-score), 119.2 cm (−2.18 Z-score), and 32.4 kg/m² (+4.16 Z-score), respectively. Systemic examination was unremarkable except for hepatomegaly. Ophthalmological evaluation revealed markedly reduced visual acuity and bilateral optic atrophy. A provisional diagnosis of hypothalamic obesity due to a mass lesion was considered.

Magnetic resonance imaging of the brain showed multiple coalescing ring-enhancing lesions in the sellar and suprasellar area, and in right frontal and bilateral temporal lobes with moderate hydrocephalus [Figure 1c and d]. Cerebrospinal fluid examination showed no cells, glucose 86 mg/dL, protein 34 mg/dL, and adenosine deaminase 7.0 U/L. Chest radiograph was normal. Three sputum examinations for acid-fast bacilli and family screen for tuberculosis were negative. Mantoux test was strongly positive (induration 18 mm × 20 mm). Total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein, and triglyceride levels were 202, 109, 220, and 49 mg/dL, respectively. Serum triiodothyronine, thyroxine, and thyroid-stimulating hormone levels were 1.11 ng/mL, 91.9 ng/mL, and 4.17 μIU/mL, respectively. Fasting and...
2-h postglucose load blood sugars were 92 and 154 mg/dL, respectively. Glycosylated hemoglobin was 7.1%. Abdominal ultrasound revealed hepatic steatosis with span of 18 cm (normal range 10–14.1 cm). Serum transaminases were raised. Four-drug antitubercular therapy along with steroids was initiated. At first follow-up after 6 weeks, a normal appetite and weight loss of 2 kg were reported. He was subsequently lost to follow-up.

Sellar-suprasellar tuberculosis (SST) is a rare form of tuberculosis with <10 cases reported in children.[1] The usual manifestations include a headache, vomiting, seizures, decrease or loss of vision, and varying degrees of pituitary dysfunction.[1,2] Hyperphagia resulting in severe obesity has never been reported in SST although weight gain was observed in one patient previously.[3] This is intriguing because patients with mass lesions such as craniopharyngiomas with suprasellar extension commonly present with obesity due to impairment in the hypothalamic regulatory centers of body weight and energy expenditure resulting from structural damage to the hypothalamus.[4] The loss of sensitivity of appetite-regulating network in the hypothalamus to afferent peripheral humoral signals causes hyperphagia.[4] We presume that the tuberculosis damaged the hypothalamic nuclei or their connections involved in appetite regulation that resulted in intense hyperphagia and severe obesity in our child. In addition, several components of obesity-related metabolic syndrome were present.[3] To the best of our knowledge, this is the first patient to develop severe obesity due to SST.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

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