Reply to “How prevalent are depression and anxiety symptoms in hypothyroidism?”

Sir,

In response to letter to the editor by Praharaj et al. in Indian Journal of Endocrinology and Metabolism titled, “How prevalent are depression and anxiety symptoms in hypothyroidism?” The comments raised by the author are welcome.

We have noted the various points raised by the author in their letter and offer the appropriate explanations.

The patients with a history of anxiety and depression were excluded from our study as the aim of the study was to assess prevalence among the patients diagnosed with hypothyroid and developed the anxiety and depressive symptoms later during illness (i.e., hypothyroid). The prevalence of depressive symptoms in hypothyroid population (more than 60%) is far more than that in general population (range from 1.5 to 19%).

Patients with below primary education were excluded so as to be able to differentiate between the onset of symptoms and the illness. However, we agree that these exclusions might have affected the actual prevalence of symptoms.

Although Hamilton depression rating scale and Hamilton anxiety rating scale are 17 points scale, the items which were not scored at all (i.e. zero) were not included in the statistics.

The recommendations by the authors are beyond the aims of the study; however, we had suggested this in view of the patient’s response to the treatment and only a clinical tip for both the psychiatrist and the endocrinologists. This was done so as to emphasize the establishment of a liaison between the two.

The comparison with euthyroid individuals as controls would have led to the inclusion of other factors contributing to the depressive and anxiety symptoms. The details of the diagnosis of hypothyroid patients and the details of psychiatric diagnosis were beyond the scope of this study. Moreover, this has already been mentioned as the limitations of the study.

However, the points raised in the letter to the editor by Praharaj et al. with respect to the use of Bonferroni correction and also the Hospital Anxiety and Depression Scale are well taken up.

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Conflicts of interest
There are no conflicts of interest.

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Letters to the Editor

Sheehan’s syndrome in two generations

Sir,

In developing countries, Sheehan’s syndrome (SS) continues to be a common cause of hypopituitarism in women of child-bearing age.\[1\] SS in two generations has not been reported; we present the clinical course of a mother and daughter, both presenting with SS.

A 55-year-old female, para 4, delivered her last child at home apparently not associated with postpartum hemorrhage (PPH). She had lactation failure and did not menstruate after the last childbirth. Examination at the time of presentation revealed anemia, loss of axillary and pubic hair, breast atrophy, blood pressure of 130/80 mmHg, and delayed relaxation of deep tendon jerks. Hormonal evaluation revealed evidence of lactotroph, corticotroph, somatotroph, and gonadotroph failure (T4 of 2 μg/dl, thyroid-stimulating hormone [TSH] of 1.8 U/L, luteinizing hormone [LH] of 0.55 U/L, follicle-stimulating hormone [FSH] of 1.23 U/L, peak cortisol of <1 μg/dl, peak prolactin of 1.15 ng/ml, and peak GH of 0.1 ng/ml after insulin tolerance test). Magnetic resonance imaging (MRI) of pituitary revealed the evidence of empty sella. With the diagnosis of SS, the patient was given glucocorticoid and thyroxine replacement therapy in appropriate doses and is on follow-up. Six years later in April 2014, her 35-year-old daughter presented to our outpatient services with features of hypopituitarism. History revealed lactation failure and amenorrhea following 4th childbirth though there was no history of PPH. Hormonal evaluation revealed the evidence of thyrotroph, lactotroph, corticotroph, gonadotroph, and somatotroph failure (T4 of 2.30 μg/dl, TSH of 5.7 U/L, LH of 1.83 U/L, FSH of 4.6 U/L, peak cortisol of 2 μg/dl, peak prolactin of 1.7 ng/ml, and peak GH of 0.07 ng/ml after insulin tolerance test). MRI pituitary revealed partial empty sella.

Hypopituitarism occurs in nearly one-third of the patients with severe postpartum hemorrhage, and the extent of pituitary gland involvement is variable. Furthermore, history of PPH may not be available in home-conducted deliveries.\[2,3\] The presence of SS in mother and daughter is quite interesting as it has not been reported previously. Whether such an association is incidental or has some familial or genetic basis is not clear at present. In adult population with sporadic idiopathic pituitary insufficiency, genetic cause for hypopituitarism is supposed to be rare. Nyström et al. studied 25 patients of idiopathic pituitary insufficiency for genotyping of all coding exons of HESX1, LH X4, PROP1, POU1 F1, and GH 1 genes. The rate of mutation was higher (50%) in familial cases compared to low frequency of 8% in nonfamilial cases. Based on these observations, it can be concluded that familial cases of adult idiopathic hypopituitarism can be considered for genetic screening, it is not required in adults with nonfamilial idiopathic adult hypopituitarism.\[4\]

Kajita et al. studied antipituitary antibodies in twenty patients with hypopituitarism and their family members (including two patients of SS). Two types of antibodies such as pituitary cell and pituitary cell surface antibodies were measured with indirect immunofluorescence. Among two patients of SS, one had antibody positive for pituitary cytoplasmic antigen 13 years later and against pituitary cell cytoplasmic antigen 15 years after obstetric insult. Antibody against pituitary cytoplasmic antigen was also positive in her brother and one of two sons who demonstrated subnormal GH response to arginine.\[5\]

The significance of antipituitary antibodies in women with SS is being debated. Although not previously reported, SS in two generations may point to a familial or genetic basis.

References

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