STRESS-INDUCED CYCLICAL HYPREPROLACTINEMIA

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ABSTRACT

Both hyperprolactinemia and depressive disorder are known manifestations of hypothyroidism. However, hyperprolactinemia with overt symptomatology per se has not been reported to precede all episodes of organic depressive disorder. We report a similar presentation.

Key words : hyperprolactinemia, hypothyroidism, organic depressive disorder, stress

Hyperprolactinemia in women manifests as galactorrhoea, menstrual irregularities, reduced libido, and other bodily changes (Molitch, 1995). Hypothyroidism, which causes various psychiatric manifestations including depressive disorder (Lishman, 1998), is a cause for hyperprolactinemia (Molitch, 1995). Nevertheless, a presentation in which typical and recurring features of hyperprolactinemia preceded all episodes of depression, following stressful life events in a hypothyroid patient, is not mentioned in the literature.

CASE REPORT

A 38-year-old woman, well adjusted premorbidly, having history of manic episode in a paternal cousin presented with following history. Her problems appeared for the first time, six years ago, following a severe quarrel with her husband. Within few days, she observed whitish discharge from her breasts and decreased sexual desire. This was followed five to six days later by insomnia, irritability, marked sadness, loss of interest, delusion of infidelity, increased religiosity and second person auditory hallucinations. However, a month later, all these symptoms disappeared spontaneously. After an interval of four years, following a severe physical illness in her son, she had second episode for four weeks with similar presentation that resolved again spontaneously.

Third episode with similar picture occurred following another stress of similar nature, a year later. Presence of marked depressive symptoms and a suicidal attempt that followed mammary discharge warranted first psychiatric consultation. She was prescribed dothiepin 75 mg/day, haloperidol 0.25 mg/day and alprazolam 0.5 mg/day. A hormonal assay was ordered following history of menorrhagia and weight gain for two years. She was found to be hypothyroid (T3 3.2 (µg/dl, normal value 5-11.7 (µg/dl; TSH 5.1 (µu/ml, normal value 0.4-3.8(µu/ml) and hyperprolactinemic (serum prolactin 50.6 ng/ml, normal maximum value 27.7 ng/ml). Thyroid supplementation (thryoxin 0.1 mg/day) resulted in complete clinical recovery within a month and her hormonal values (T3 9.82 (µg/dl, TSH 1.12 (µg/dl, prolactin 16 5ng/ml) returned to normalcy. Subsequently, alprazolam, dothiepin and haloperidol were stopped but thyroid supplement continued.

However, after an interval of three months, she presented to Central Institute of Psychiatry (CIP) OPD with her fourth episode with similar symptomatology of seven days duration, which occurred following her son’s brief-lasting but serious physical illness. Detailed
history and psychiatric examination revealed mammary discharge preceding lack of interest, low mood, easy fatigability, insomnia, poor appetite, persecutory delusion and auditory and visual hallucinations. Other than hoarseness, neurological examination was unremarkable. She was provisionally diagnosed as a case of organic depressive disorder (ICD-10, World Health Organization, 1992). Her MRI brain was unremarkable, started on risperidone 2-mg/day and fluoxetine 20-mg/day along with ongoing thyroxin supplementation. A month later, she recovered completely.

DISCUSSION

In this patient, the diagnosis of hypothyroidism was based on both clinical and laboratory evidences. Though her hyperprolactinemia was evidence-based but it was mainly diagnosed on clinical grounds. She was on psychotropic medication with hyperprolactinemic potential (Molitch, 1995), nonetheless it was unlikely to be drug-induced since galactorrhoea preceded all episodes spontaneously and psychotropic medicines was given only during the third episode. Even during this episode, mammary discharge began before the depressive syndrome set in. Secondly, apart from hypothyroidism, she had no other condition that might result in hyperprolactinemia. Hence, hypothyroidism was presumed as the most probable cause for hyperprolactinemia.

An important clinical problem, in this patient, has been determining the cause for depressive episodes, whether it is due to hypothyroidism, hyperprolactinemia or stress, either alone or in combination. Although anxiety, depression and hostility can occur in hyperprolactinemic states (Kellner et al., 1984), nothing is clear about the role of prolactin in causing discrete psychiatric syndromes (Funente & Rosenbaum, 1981). Hence, hyperprolactinemia may not offer a comprehensive explanation for severe depressive episodes. Similarly, hypothyroidism was treated and reached normalcy and hence, it may not explain subsequent clinical decompensation, which occurred inspite of continued thyroid supplement. In this case, it would be more logical to consider the role of stress that is known to inhibit thyroid releasing and stimulating hormones, and increase prolactin level (Ganong, 1995; Molitch, 1995). Stress, might have been etiologically significant in causing hypothyroid state, recurrent depression and cyclical hyperprolactinemia. Similarly, improvement in both clinical hyperprolactinemia and depression could be the consequence of disappearance of stress, which is evidenced by the spontaneous recovery noted in her first two episodes.

In summary, this report illustrates a clinical problem, which may arise while managing patients with endocrinological abnormalities. Additionally, it also highlights the significance of stress in precipitating clinical illness in susceptible individuals. This report suggests that clinicians handling patients with endocrinological abnormalities should look for evidence of stress if a well-settled patient relapses acutely without an appropriate reason. In addition to medications, stress reduction strategies would be more helpful in reducing subsequent relapses and recurrences.

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