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A Case of Thyroid Storm Due to Thyrotoxicosis Factitia

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We describe a case of thyroid storm due to thyrotoxicosis factitia, which was caused by the ingestion of excessive quantities of exogenous thyroid hormone for the purpose of reducing weight. An 18-year-old female was admitted to the hospital 24 hours after taking up to 50 tablets of synthyroid (1 tablet of synthyroid : levothyroxine 100 μg). Because of her stuporous mental state and acute respiratory failure, she was intubated and treated in the intensive care unit. After reviewing her history carefully and examining plasma thyroid hormone levels, we diagnosed this case as a thyroid storm due to thyrotoxicosis factitia. Her thyroid function test revealed that T3 was 305 ng/dL, T4 was 24.9 μg/dl, FT4 was 7.7 ng/dL, TSH was 0.05 μIU/mL and TBG was 12.84 μg/mL (normal range: 11.3 - 28.9). TSH receptor antibody, antimicrosomal antibody, and antithyroglobulin antibody were negative. She was recovered by treatment, namely, steroid and propranolol, and was discharged 8 days after admission. Thyroid storm due to thyrotoxicosis factitia caused by the ingestion of excessive thyroid hormone is rarely reported worldwide. Therefore, we now report a case of thyroid storm that resulted from thyrotoxicosis factitia caused by the ingestion of a massive amount of thyroid hormone over a period of 6 months.

Key Words: Thyroid storm, thyrotoxicosis factitia, ingestion of excessive thyroid hormone

INTRODUCTION

Thyrotoxicosis factitia is a disease resulting from taking a excessive thyroid hormone, which is also often referred to as alimentary thyrotoxicosis, exogenous thyrotoxicosis, occult factitial thyrotoxicosis or thyreoidismus medicamentosus.[1,2] This disease normally develops, due to excessive thyroid hormone preparations taking, in patients with an unstable mental state or a neuropsychotic disorder.[2-4] Thyrotoxicosis often occurs due to the taking of medication to reduce weight and the condition has been reported to be induced by the administering of excessive thyroid hormone to subjects for medical research purposes.[3,5] An outbreak of thyrotoxicosis was once reported to have been caused by the consumption of bovine thyroid gland in ground beef.[6] However, thyrotoxic storm due to thyrotoxicosis factitia has rarely been reported, worldwide. The authors experienced one case of thyrotoxic storm, which occurred after ingesting thyroid hormone over a 6-month period, we report this case with reviewing references.

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CASE REPORT

An 18-year-old female presented to our hospital with drowsy consciousness and dyspnea. She had taken thyroid hormone for 6 months by herself. She had been previously healthy, but was under mental stress due to college entrance examinations. To reduce weight, she had taken synthyroid (levothyroxine). Three months before admission, suffering from symptoms such as anxiety, irritability, tachycardia, and insomnia, she went to a private clinic. After being diagnosed as having hyperthyroidism, she received a prescription of PTU or methimazole, and in spite of a recommendation to keep to the medication, she took it on only one occasion. Afterwards, she usually

took 0.6 - 0.9 mg per day of synthyroid defying the doctor's recommendation, and she had even taken 50 tablets of synthyroid (levothyroxine 5 mg) on one occasion. One day before admission, she had taken 50 tablets of synthyroid at one time and 15 hours before admission she had been found unconsciousness. After gastric lavage at near hospital she was transferred to our hospital.

At the time of admission, she appeared acutely ill and revealed systolic hypertension (180/74 mm Hg), tachycardia (125 beats per minute), tachypnea (36 times per minute), and a stuporous mental status. Her thyroid was not visible or palpable, and no nodules were detected. She did not exhibit exophalmos, and there was no specific finding on physical examination of the thorax, abdomen, or other parts.

Laboratory findings at the time of admission, on a peripheral blood smear, WBC count was 14,580/mm^3 (neutrophil 95.8%, lymphocyte 3.6%, monocyte 0.1%), hemoglobin 11.1 g/dL, hematocrit 31.9%, and platelet 265,000/mm^3. Biochemical analysis of serum revealed, BUN 6.8 mg/dL, creatinine 0.4 mg/dL, total protein 6.1 g/dL, albumin 3.3 g/dL, uric acid 2.9 mg/dL, sodium 137 mmol/L, pottasium 3.6 mmol/dL, chloride 103 mmol/L, calcium 9.0 mg/dL, phosphate 3.8 mg/dL, total cholesterol 119 mg/dL, AST 128 U/L, ALT 70 U/L, total bilirubin 0.5 mg/dL, and alkaline phosphatase 209 U/L. Her AST and ALT returned to a normal level after 2 months. Urinalysis showed no specific finding. On blood coagulation testing, the prothrombin time was 91%, and the aPTT 35.1 sec. Serum thyroid hormone level showed that T3 305 ng/dL, T4 24.9 μg/dl, FT4 7.7 ng/dL, TSH 0.05 μIU/mL and TBG 12.84 μg/mL (normal range: 11.3 - 28.9). TSH receptor antibody, antimicrosomal antibody, and antithyroglobulin antibody were negative. CNF analysis showed, that it was clear and transparent, protein 17.9 mg/dL, glucose 81 mg/dL, WBC count 5/uL, RBC 0/uL, and CSF pressure 240 mmHg. In the culture study, which was done at the time of admission, no bacteria grew on the sputum, blood, or urine. Chest X-ray showed normal findings and computed tomography of her brain revealed diffuse ischemic change on both cerebral hemispheres. Electrocardiography showed sinus tachycardia.

At the time of admission, the patient showed mental confusion and acute respiratory failure, therefore, endotracheal intubation was performed and mechanical ventilator care was undertaken in the intensive care unit. To reduce cerebral pressure, intravenous mannitol at 600 cc per 6 hour was infused from hospital day 1 to hospital day 3. Oral propranolol 40 mg per 6 hour was given via a ventral tube for 5 days and intravenous dexamethasone 2 mg per 6 hour for 2 days was tried, with sufficient fluid and diuretics. Two days after admission, her acute respiratory failure was improved and her mental state had returned to normal, and she was extubated. Eight days after admission, her symptoms recovered, and she was discharged. Whilst being observed on an outpatient basis, she was readmitted to our hospital due to generalized tonic clonic seizure 3 months after discharge, because she started retaking synthyroid. Serum thyroid hormone levels at the time of readmission revealed that T3 was 330.7 ng/dL, FT4 4.5 ng/dL, and TSH 0.01 μIU/mL. She was discharged 7 days after readmission, and 2 months after the second discharge her serum thyroid hormone levels were, T3 96.4 ng/dL, FT4 1.4 ng/dL, and TSH 1.41 μIU/mL.

DISCUSSION

Thyrotoxicosis is a syndrome, which includes a series of symptoms stemming from an overreaction of thyroid hormone due to excessive thyroid hormone in the blood, and thyrotoxicosis factitia is a thyrotoxicosis a result of excessive thyroid hormone intake. It is very rare to be presented with thyrotoxic storm due to thyrotoxicosis factitia, as in this case. In terms of the published literature, Braustein et al. reported 5 cases of unintentional thyrotoxicosis factitia, where all of the patients ingested thyroid hormone pills because they believed that they were nontoxic natural substance that would allow them to lose an appreciable amount of weight.[7] However we found no report about thyrotoxicosis factitia leading to thyroid storm. Generally thyrotoxicosis factitia is thought to be caused by the secret ingestion of excessive amounts of thyroid hormone by neuropsychotic patients,[3] but it is possible that it

could be caused by an excessive intake of thyroid hormone in nontoxic goiter patients being administered thyroid hormone. As for the cases in the States, about 5600 children with thyrotoxicosis are reported each year. Also in the States, between April 1984 to August 1985, 121 patients came down with thyrotoxicosis factitia due to consumption of bovine thyroid gland in ground beef.[6] Another reported found that infants had ingested thyroid hormone by mistake,[4] and there is even a report that thyrotoxicosis was caused by mistakenly taking thyroxine that was intended for a dog.[8] It was also reported that while taking thyroid hormone, after a diagnosis of hypothyrodism, thyrotoxicosis factitia occurred during the course of an operation.[9,10]

We believe that our case concerns a possible cause that deserves some attention. The patient was admitted to hospital due to a stuporous mental state. Therefore, having considered documented cases and the present case, we believe that thyrotoxicosis factitia should be considered as a possibility when a patient presents with an unexplained mental disturbance, even though the patient history provides no indication.

In terms of the symptoms associated with taking a lot of thyroxine, it is believed likely that children could show tachycardia, fever, nausea, seizure, diarrhea, or irritable activity, and that even fatalities could occur.[4] Adults, on the other hand, could have tachyarrhythmia, heart failure, myocardiac infarction,[11] cerebral infarction, or psychosis, or even death.[4,12] One reported case involved acute myocardial infarction in a patient taking thyroid hormone after a diagnosis of hypothyroidism due to a rapid increase in oxygen requirement.[11] To diagnose thyrotoxicosis factitia, it is necessary to differentiate several diseases, such as Graves disease, subacute thyroditis, subacute lymphocytic thyroiditis, thyrotoxic multinodular goiter, simple thyrotoxic nodule, and hyperthyroidism due to taking excessive iodide, Hydatidiform mole, and choriocarcinoma.[13] Differentiating points are that in the case of patients taking much thyroid hormone, symptoms of thyrotoxicosis can be observed, and there is no evidence of other thyroid disease, though there might be history of thyroid medication, the serum thyroid hormone levelwould be increased, TSH suppressed or undetectable, thyroid autoantibody undectable, serum thyroglobulin decreased or undetectable,[2,5,14-17] iodine uptake low or suppressed,[16] and urine iodine excretion normal, with no hypervascularity and a normal peak systolic velocity, by color flow Doppler sonography (CFDS).[18,19]

The self-administration of thyroid hormones should be considered in patients whose diagnosis looks like clinical hyperthyroidism and whose mental state is abnormal.

Considering the patient's history and thyroid function test, the case described here is compatible with a finding of thyrotoxicosis factitia. To our knowledge, this is the first report of thyrotoxicosis factitia caused by chronic thyroxine overdosage leading to thyrotoxic storm.

REFERENCES

1. Rose E, Sanders TP, Webb WL, Hines RC. Occult factital thyrotoxicosis. Ann Intern Med 1969;71:309-15.
2. Cohen JH, Ingbar SH, Braverman LE. Thyrotoxicosis due to ingestion of excess thyroid hormone. Endocr Rev 1989;10:113-24.
3. Harvey RF. Thyroxine "addicts". Br Med J 1973;2:35-6.
4. Bartalena L, Bogazzi F, Martino E. Adverse effects of thyroid hormone preparations and antithyroid drugs. Drug Saf 1996;15:53-63.
5. Mariotti S, Martino E, Cupini C, Lari R, Giani C, Baschieri L, et al. Low serum thyroglobulin as a clue to the diagnosis of thyrotoxicosis factitia. N Engl J Med 1982;307:410-2.
6. Hedberg CW, Fishbein DB, Janssen RS, Meyers B, McMillen JM, MacDonald KL, et al. An outbreak of thyrotoxicosis caused by the consumption of bovine thyroid gland in ground beef. N Engl J Med 1987;316: 993-8.
7. Braunstein GD, Koblin R, Sugawara M, Pekary AE, Hershman JM. Unintentional thyrotoxicosis factitia due to a diet pill. West J Med 1986;145:388-91.
8. Feit S. Thyrotoxicosis factitia veterinarius. Ann Intern Med 1997;127:168.
9. Ambus T, Evans S, Smith NT. Thyrotoxicosis factitia in the anesthetized patient. Anesthesiology 1994;81:254-6.
10. Redahan C, Karski JM. Thyrotoxicosis factitia in a post-aortocoronary bypass patient. Can J Anaeth 1994; 41:969-72.
11. Locker GJ, Kotzmann H, Frey B, Messina FC, Sterz FR, Weissel M, et al. Factitious hyperthyroidism causing acute myocardial infarction. Thyroid 1995;5:465-7.
12. Vlase H, Lungu G, Vlase L. Cardiac disturbances in thyrotoxicosis: Diagnosis, Incidence, Clinical features

and management. Rom J Endocrinol 1991;29:155-60.
13. Stein PP, Tanoue LT. Factitious thyortoxicosis: Searching for the source of this disorder. Consultant 1988; 28:64-75.
14. Pearce CJ, Himsworth RL. Thyrotoxicosis factitia. N Engl J Med 1982;307:1708-9.
15. Pacini F, Pinchera A. Serum and tissue thyroglobulin measurement: Clinical applications in thyroid disease. Biochimie 1999;81:463-7.
16. Refetoff S, Lever EG. The value of serum thyroglobulin measurement in clinical practice. JAMA 1983;250:2352-7.
17. Matsubara S, Inoh M, Tarumi Y, Sato M, Takahara J. An outbreak (159 cases) of transient thyrotoxicosis without hyperthyroidism in Japan. Intern Med 1995;34: 514-9.
18. Bogazzi F, Bartalena L, Vitti P, Rago T, Brogioni S, Martino E. Color flow doppler sonography in thyrotoxicosis factitia. J Endocrinol Invest 1996;19:603-6.
19. Bogazzi F, Bartalena L, Brogioni S, Burelli A, Manetti L, Tanda ML, et al. Thyroid vascularity and blood flow are not dependent on serum thyroid hormone levels: studies in vivo by color flow doppler sonography. Eur J Endocrinol 1999;140:452-6.