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Case Report

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Old Man Presented with Thyrotoxic Vomiting

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Thyrotoxicsis has proved to be a challenging and intresting disorder. It can present in many ways though vomiting is uncommon as a main presenting symptom, which may lead to a delay in diagnosis. We are reporting a 74 years old male who presented with persistent vomiting, nausea and 10 kg weight loss. Investigations for causes of vomiting were negative except for hyperthyroidism. His symptoms resolved after treatment with carbimazole and B Blockers. Recurrence of thyrotoxicosis and vomiting occurred just after Radioiodine ablation therapy. To the best of our knowledge, this is the first case of thyrotoxic vomiting in a male patient of this age group, in addition to recurrence of thyrotoxic vomiting post radioiodine ablation therapy.

Key words: Thyrotoxicsis, vomiting, radiation thyroiditis

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INTRODUCTION

The clinical manifestations of hyperthyroidism are largely independent of its cause. However not every patient present with typical presentation. This might cause a delay in diagnosis which could be determental especially to eldery patients (Arthurs *et al*., 1997). We report an elderly male patient with hyperthyroidism whose main complaint was persistent vomiting. Increasing awareness of such presentation among health professionals will help in recognizing thyrotoxicosis earlier and, Hence preventing its complications.

CASE REPORT

A 74 year old man was referred to the gastroenterology center because of persistent vomiting and weight loss for two weeks. Physical examination showed a moderately ill patient who weighed 60 kg, he lost around 10 kg in 2 weeks. His blood pressure was 110/60 mm Hg with a pulse rate of 108 beats/min, regular. There was no orthostatic hypotension, moderate thyroid swelling with no eye signs nor tremors. Examination of the abdomen showed no abnormalities. Laboratory evaluation showed prerenal azotemia with no electrolyte disorders (Table 1). He was thoroughly investigated including scopic and radiological imaging however no cause was found. he did not respond to treatment with antacids or prokinetic agents. Thyroid functions was requested which revealed thyrotoxic state, at that time he was referred to endocrine OPD and diagnosis was confirmed (April, 2005). Thyroid auto-antibodies (antiperoxidase and antithyroglobulin) were positive. Thyroid U/S showed moderately enlarged diffuse thyroid gland. thyroid scan showed diffuse increased uptake with no cold nodules. Thyroid uptake was 35% at 2 and 67% at 24 h (N 10-35%). Diffuse toxic goiter was diagnosed. He was kept on carbimazole 5 mg tab tds together with propranolone 20 mg tds. the vomiting stopped within one week. He continued the antithyroid medications for 10 months. The antithyroid medications were titrated according to clinical and thyroid functions and completely stopped after 10 months, at that time the patient was clinically and biochemically euthyroid (TSH 0. 44, FT4 6.43), he gained 8 kg during this period (Feb 2006). After 2 months the patient became biochemically thyrotoxic (TSH 0. 01, FT4 18. 6) during evaluation of his thyroid functions without vomiting, at that time the relapse was considered and failure to medical treatment was settled (April 2006). We have restarted the carbimazole therapy as preparatory step to radioactive iodine ablation of the thyroid gland. Two months later the patient became biochemically euthyroid and he was referred to nuclear medicine department and he was advised to stop the carbimazol for one week before the ablation after which radioactive iodine was given (June 2006). One day Post radioactiveiodine ablation the patient had repeated vomiting which did not stop for one week for which he was admitted to hospital with 110 pulse rate and prerenal azotemia. TFT showed thyrotoxicosis (Jun 2006). He lost 5 kg during this week. He was afebrile with mild thyroidal pain. Radiation thyroiditis was considered, the patient was treated with short coarse of steroids, B blocker and IV fluids, with marked improvement. the vomiting was stopped within 2 days. Six weeks later, thyroid functions was done which revealed hypothyroid state and thyroxine therapy was initiated.

Table 1: Laboratory data of the patient

Parameters	April 2005	Jun 2006	Jun 2006 postiodine
Glucose (3.9-6.1 mmol L^{-1})	4.80	5.70	6.10
BUN (2.5-7.1 mmol L^{-1})	8.10	6.80	10.60
Creatinine (62-115 umol L^{-1})	75.00	70.00	88.00
Na (136-144 mmol L^{-1})	137.00	135.00	140.00
K (3.6-5.1 mmol L^{-1})	4.10	4.70	4.30
T. Bilirubin (3-25 μmol L^{-1})	22.00	21.00	24.00
AST (10-42 IU L^{-1})	39.00	25.00	40.00
ALT (10-60 IU L^{-1})	53.00	28.00	55.00
Alkaline phos (56-119 IU L^{-1})	72.00	90.00	102.00
Amylase (25-125 IU L^{-1})	90.00	110.00	105.00
Corrected Ca (2.1-2.6 mmol L^{-1})	2.43	2.34	2.49
Mg (0.74 1.2 mmol L^{-1})	0.83	0.91	0.80
Hb % (140-170 g L^{-1})	10.10	11.30	10.50
WBC (4-11)	5.20	6.10	7.20
RBC (4.5-6.2)	3.40	3.90	3.60
Plat. (150-450)	169.00	191.00	234.00
TSH (0.43-4.1 uIU mL^{-1})	0.01	0.45	0.01
FT4 (5.69-13.44 pmol L^{-1})	52.80	8.21	77.20

DISCUSSION

Thyrotoxicosis refers to the hypermetabolic clinical syndrome resulting from serum elevations in thyroid hormone levels, specifically free thyroxine (T4), triiodothyronine (T3), or both. Hyperthyroidism is a type of thyrotoxicosis in which accelerated thyroid hormone biosynthesis and secretion by the thyroid gland produce thyrotoxicosis. However, hyperthyroidism and thyrotoxicosis are not synonymous. After reviewing the literature, this is the only case report of thyrotoxic vomiting after age of 70. He was presented with vomiting two times not explained by any causes except for hyperthyroisism and he was proved to be thyrotoxic at both times. Thyrotoxicosis has a variety of typical presentations. Vomiting is not considered one of them. It's rarely mentioned in standard textbooks of medicine and endocrinology. A total of 33 such cases have been reported in literature. The mean age of the patients was

46±14 year with a range of 19 to 68 years. Only 4 patients were male. Weight loss was found in about half of them and might be an important clue. Thyrotoxicosis should be considered in differential diagnosis of unexplained vomiting (Chen *et al*., 2003). Rosenthal *et al*. (1976), reported that vomiting can be an important presenting symptom of thyrotoxicosis. The mechanism that causes vomiting in thyrotoxicosis is not clear. It has been suggested that direct action of excess thyroid hormone on gastrointestinal motility or thyroid hormone stimulation of a chemoreceptor trigger zone in the central nervous system may play a role (Sellin and Vassilopoulou-Sellin, 2000). Another possible mechanism behind thyrotoxic vomiting, could be the increase in B adrenergic activity in hyperthyroidism due to an increased number of B adrenergic receptors (Bilezikian and Loeb, 1983). The increase in B adrenergic activity is responsible for many of the other symptoms associated with hyperthyroidism. It also explains the ability of B blockers to cause a rapid improvement in many of the symptoms, including palpitations, tachycardia, tremulousness, anxiety and heat intolerance. In support of the theory that vomiting is caused by B adrenergic stimulation is the observation Dreyfuss made in a 53 year old woman who suffered from epigastric pain and vomiting due to hyperthyroidism (Dreyfuss, 1984). He noted improvement after 36 h of treatment with propanolol and propylthiouracil. Hypercalcemia, which is reported to be associated with thyrotoxicosis (Gordon *et al*., 1974; Skrabanek, 1976) may be associated with vomiting, also hypomagnesia was attributed for thyrotoxic vomiting in one case (Skrabanek, 1976) In our case, both serum magnesium and serum calcium level were normal. Hepatitis is usually associated with vomiting, Bellassoued et al reported a case of thyrotoxic hepatitis with elevated liver enzymes and mild cholestasis (Bellassoued *et al*., 2001), how ever liver enzymes were normal in our patient.

Aging men may exhibit higher estrogen and gonadotropines level, both were proposed as responsible for thyrotoxic vomiting on the grounds that it is similar to hyperemesis gravidarum (Hershman, 1999), however serum estradiol level, FSH and LH were not measured in our patient.

Approximately 1 percent of patients who have radioactive iodine therapy for hyperthyroidism develop radiation thyroiditis between five and 10 days after the procedure. The rapid destruction of the thyroid parenchyma results in pain, tenderness, and an exacerbation of hyperthyroidism from the release of stored T4 and T3. A brief course of NSAIDs or, rarely, prednisone in dosages of 40 to 60 mg per day may be used to alleviate pain; a beta blocker often is required to block the peripheral effects of the thyroid hormone. The gland eventually undergoes extensive fibrosis in approximately six to 18 weeks (Ginsberg, 2003), in our patient radiation thyroiditis was suspected and the patient was managed accordingly with marked improvement of the patient symptoms, he turned hypothyroid 6 weeks later.

CONCLUSION

Persistent vomiting could be a symptom of thyrotoxicosis. The symptom resolves quickly and completely with treatment with antithyroid drugs and B blocking agents.

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