Vomiting as harbinger for Graves’ disease

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Introduction: Isolated vomiting is a rare often forgotten presentation of Graves’s disease1 and can lead to delayed diagnosis. Multiple case reports quote vomiting in thyrotoxicosis co-presenting with Addisonian crisis2, diabetic ketoadiposis3,4 or with abnormal liver function and jaundice5,6. Another common association is hyperemesis gravidarum. Vomiting in paraneoplastic hyperthyroidism occurs through a similar mechanism of beta-HCG secretion, mainly related to germ-line tumours7. We present a case of severe prolonged vomiting caused by Graves’ disease.

Case presentation: A 46 years old postmenopausal woman of Afro-Caribbean origin presented with unremittent vomiting and dramatic 38 Kilograms weight loss over 2-3 months. She reported tiredness, headaches and anxiety. Her heart rate was 91/min sinus rhythm, BP 115/80, temperature 37.2°C. She had no palpable thyroid nor thyroid eye signs. Thyroid function tests showed suppressed TSH<0.01 mIU/L and elevated FT4 86.9 pmol/L. The Burch and Wartofsky’s score was 20 (temperature 37.2, heart rate 91 in sinus rhythm, absent agitation or CNS symptoms), indicating this was not a thyroid storm presentation.

Further investigations: Thyroid uptake scan and anti-thyroid antibodies confirmed Graves’s disease. Liver function tests were normal. Thyrotoxicosis related hypercalcaemia was noted (corrected calcium 2.75 mmol/l with low parathyroid hormone 1.3 pmol/l). Coeliac, autoimmune screen and beta-HCG were normal. Gastroscopy with duodenal biopsy and CT body imaging were normal.

Conclusion: Vomiting is commonly associated with endocrine disorders. Literature review reports female predominance of thyrotoxic vomiting. Previous case reports of isolated vomiting have no Graves’s thyroid eye signs making early diagnosis challenging. Typically, they were associated with significant weight loss in the context of a delayed diagnosis, and persistent tachycardia as diagnostic clues. Characteristically such vomiting fails to respond to usual antietemics, but responds to antithyroid medications. Glucocorticoids are also useful adjuncts in bringing symptoms under control11,12.

Mechanisms of thyrotoxic vomiting

| Increased beta-adrenergic activity | Increases the number of beta-adrenergic receptors8,9 |
| Increased thyroid hormones | Stimulates the chemical trigger zone10 |
| Decreases gastric emptying and causes pyloric sphincter malfunction11,12 |
| Increases magnesium urinary excretion. Hypermagnesaemia inhibits gastric smooth muscle13,14 |
| Increases oestrogen level in patients of both sexes10,12 |
| Raised oestrogen level may induce nausea and vomiting only in susceptible patients17 |
| Functional duodenal obstruction | Hypermetally induced volvulus of duodenal third portion has been described16 |

Recommendations: Increased awareness of vomiting as presentation of thyrotoxicosis is necessary in order to avoid a delayed diagnosis and over-investigations. There is a potential risk of precipitating a thyroid storm while exposing patients unnecessarily to invasive investigations such as endoscopy or exploratory laparoscopy.

Ruling out alternative aetiologies is also important. Rare paraneoplastic thyrotoxic conditions may present as such.

References: