INTRODUCTION

Persistent nausea and vomiting are debilitating to any patient. Vomiting can be caused by a number of causes from metabolic abnormality to structural or neurological causes resulting in poor oral intake and malnutrition. We highlight a case of secondary adrenal insufficiency as a cause of persistent vomiting.

CASE REPORT

A 65-year-old female, homemaker, presented with easy fatiguability, generalised weakness and multiple episodes of vomiting of 20 days duration. Vomiting was predominantly post-prandial, was not associated with pain abdomen and reflux symptoms. Patient had history of hypertension, chronic obstructive pulmonary disease (COPD) and osteoarthritis for the past 10 years for which she was treated with oral amlodipine 10 mg once daily, oral salbutamol and theophylline. She attained menopause at age of 42 years, had 2 children and had no problems with lactation. On examination her vital parameters were stable; blood pressure was 130/80mmHg. General physical examination revealed oral candidiasis and, angular cheilosis. Skin was dry and thin. Abdominal examination was normal.

Laboratory investigations revealed normal haemogram; serum sodium was 134mmol/L, serum potassium was 3.6mmol/L. Fasting blood glucose was 92mg/dL. Renal function and liver function parameters were within normal limits. human immunodeficiency virus (HIV) serology was non-reactive. Ultrasonography of abdomen was normal.

Vomiting persisted despite treatment with injectable antiemetics. Upper gastrointestinal endoscopy showed oesophageal candidiasis, rest of the examination was normal. Her thyroid function tests were normal.

Candidiasis was treated with oral fluconazole for 14 days. Because of persistent nausea and vomiting serum cortisol was measured to rule out adrenal insufficiency. Early morning cortisol was 3 µg/dL. On subsequent review of medications, patient admitted to have taken oral corticosteroids, (prednisolone 10 mg once daily over the counter), for 10 years for COPD which was stopped a month prior to admission during...
the episode of fever. She was started on intravenous hydrocortisone 100 mg thrice-daily and subsequently shifted to oral prednisolone 10 mg once-a-day. Symptoms disappeared on the first day of replacement therapy. Currently patient is doing well, is on oral corticosteroids. A tapering course of corticosteroid treatment has been planned.

DISCUSSION

Corticosteroids are used in a wide range of inflammatory and neoplastic diseases. Despite various side effects which include secondary diabetes mellitus, osteoporosis, cardiovascular abnormalities and infections, they are often used for inappropriate duration. Suppression of hypothalamic–pituitary axis is known with chronic steroid usage since ages, is the most hazardous complication associated. This effect reduces patient’s ability to respond to stress.

Suppression of hypothalamic-pituitary-adrenal (HPA) axis is dose and duration dependent. Several studies\(^1\)-\(^4\) have shown that suppression of HPA axis can occur with high dose corticosteroids for shorter duration and small dose for longer duration. HPA axis suppression is inevitable in patients on 15 mg or more of prednisolone or equivalent for more than 3 weeks and with lower doses of 5-15 mg/day, suppression is variable.\(^5\) Even inhalational and topical steroid use have been associated with suppression of HPA axis and symptoms of adrenal insufficiency.\(^6\)-\(^7\) Our patient was receiving oral prednisolone 10 mg/day for more than 10 years.

Patients on chronic glucocorticoid therapy on dose reduction may present with steroid withdrawal syndrome, adrenal insufficiency or flare up of disease. Steroid withdrawal syndrome is characterized by physical or psychological dependence. Non-specific symptoms like nausea, feeling of not being well, arthralgia. Exact physiological basis is not known. Symptoms may be seen even at supra-physiologic doses of steroids during rapid dose reduction despite intact HPA axis.\(^8\) It may be due to increased levels of circulating interleukin-6 levels which are normally suppressed by glucocorticoids.\(^9\),\(^10\)

Symptoms of steroid insufficiency (secondary adrenal insufficiency) occur due to suppression of HPA axis and vary widely. Patients can present acutely with circulatory collapse\(^11\) or with chronic symptoms like anorexia, nausea, vomiting, constipation, abdominal pain, diarrhea, lethargy, weight loss, desquamation and arthralgia. Secondary adrenal insufficiency is differentiated from primary by absence of hyperpigmentation and findings of mineralocorticoid deficiency like hyponatremia, hyperkalemia. Our patient presented with nausea, vomiting and easy fatiguability.

Chronic adrenal insufficiency is treated best with oral hydrocortisone 15-20 mg per day, with two-third of the total dose administered in the morning and 2 divided doses are administered 4 and 8 hours apart from the morning dose.\(^12\) Corticosteroids should be withdrawn cautiously to prevent adrenal insufficiency over months. Recovery of HPA axis suppression can take more than 9 months.\(^12\) Dose should be reduced from pharmacological dose to physiological dose if disease condition permits over few weeks. Once patient is on physiological dose (prednisolone 7.5 mg/day or equivalent), dose should be reduced by 1 mg/day of prednisolone every 2-4 weeks with monitoring for any symptoms of steroid insufficiency and stopped. Alternatively patient can be started on equivalent dose of hydrocortisone and dose can be tapered by 2.5 mg/week till dose of 10 mg single morning dose and maintained till recovery of HPA axis. HPA axis recovery is documented by short synacthen test. Serum cortisol levels greater than 18 µg/dL at 60 minutes after 250 µg of intramuscular injection of synacthen suggests HPA axis recovery.
REFERENCES


