Case Report:

Unusual cause of nausea and vomiting

B. Nandakrishna, Varma Muralidhar, Sudha Vidyasagar

Department of Medicine, Kasturba Medical College, Manipal

ABSTRACT

Adrenal insufficiency can manifest with various symptoms ranging from haemodynamic compromise to non-specific symptoms like myalgia, anorexia, and loss of weight. Aetiology can be primary adrenal disorder or secondary to suppression of HPA axis. We report a case of secondary adrenal insufficiency presenting with persistent nausea and vomiting.

Key words: Adrenal Insufficiency, substance withdrawl syndrome, Nausea, Vomiting

Nandakrishna B, Muralidhar V, Vidyasagar S. Unusual cause of nausea and vomiting. J Clin Sci Res 2016;5:192-4. DOI: http://dx.doi.org/10.15380/2277-5706.JCSR.15.071.

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 postprandial, 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 $3 \mu g/dL$. 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

Received: November 20, 2015; Revised manuscript received: March 19, 2016; Accepted: April 02, 2016.

Corresponding author: Dr Sudha Vidyasagar, Professor, Department of Medicine, Kasturba Medical College, Manipal, India. **e-mail:** vsagar33@yahoo.com



Online access http://svimstpt.ap.nic.in/jcsr/jul-sep16_files/4cr.15.071.pdf DOI: http://dx.doi.org/10.15380/2277-5706.JCSR.15.071 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 studies¹⁻⁴ 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 supraphysiologic 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 collapse¹¹ 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 μ g/dL at 60 minutes after 250 µg of intramuscular injection of synacthen suggests HPA axis recovery.

REFERENCES

- 1. Christy NP, Wallace EZ, Jailer JW. Comparative effects of prednisone and cortisone in suppressing the response of the adrenal cortex to exogenous adrenocorticotropin. J Clin Endocrinol Metab1956;16:1059-74.
- 2. Plager JE, Cushman P Jr. Suppression of the pituitary-ACTH response in man by administration of ACTH or cortisol. J Clin Endocrinol Metab 1962;22:147-54.
- 3. Grant SD, Forsham PH, DiRaimondo VC. Suppression of 17-hydroxycorticosteroids in plasma and urine by single and divided doses of triamcinolone. N Engl J Med 1965; 273:1115-8.
- 4. Streck WF, Lockwood DH. Pitutary adrenal recovery following short term suppression with corticosteroids. Am J Med 1979; 66:910-4.
- 5. Christy NP. Corticosteroid withdrawal. In: BardinCW, editor. Current therapy in endocrinology and metabolism, 3rd edition. New York: BC Decker;1988.p.113-20.
- 6. Mortimer KJ, Tata LJ, Smith CJ, West J, HarrisonTW, Tattersfield AE, et al. Oral and inhaled corticosteroids and adrenal insufficiency: a case control study. Thorax 2006;61:405-8.
- 7. Böckle BC, Jara D, Nindl W, Aberer W, Sepp NT.Adrenal insufficiency as a result of long-term

misuse of topical corticosteroids. Dermatology 2014;228:289-93.

- Dixon RB, Christy NP. On the various forms of corticosteroid withdrawal syndrome. Am J Med1980;68:224-30.
- 9. Schlagheckhe R, Kornely E, Santen RT, Ridderskamp P. The effect of long term glucocorticoid therapy on pituitary adrenal response to exogenous corticotropin releasing hormone. N Engl J Med 1992;326:226-30.
- 10. Papanicolaou DA, Wilder RL, Manolagas SC, Chrousos GP. The pathophysiologic roles of interleukin-6 in human disease. Ann Intern Med1998;128:127-37.
- 11. Fraser CG, Preuss FS, Bigford WD. Adrenal atrophy and irreversible shock associated with cortisone therapy. J Am Med Assoc 1952;149:1542-3.
- 12. Arlt W. The approach to the adult with newly diagnosed adrenal insufficiency. J Clin Endocrinol Metab 2009 ;94:1059-67.
- 13. Graber AL, Ney RL, Nicholson WE, Island DP, Liddle GW. Natural history of pituitary-adrenal recovery following long-term suppression with corticosteroids. J Clin Endocrinol Metab 1965;25:11-16.