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Case Study

Pantoprazole - Induced Hyperhydrosis: A Case Report

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ABSTRACT

Pantoprazole is a proton-pump inhibitor (PPI) widely used both in inpatient and outpatient settings which acts by blocking the gastric parietal cells' H+/K+ ATPase pump. A 56-year-old female with a medical history of chronic liver disease was admitted. When intravenous pantoprazole 40mg was initiated on her admission, she experienced hyperhidrosis, which was ruled out after pertinent investigations. Therefore, despite appearing to be a rare side effect, PPI-induced hyperhidrosis is more likely to occur in clinical practice due to the medication's extensive use. If there are no obvious reasons why a patient on PPI therapy has hyperhidrosis, a trial of quitting PPI can be conducted to determine if symptoms can resolve. Hence, healthcare professionals should be mindful of the possibility of an uncommon adverse reaction of pantoprazole-induced hyperhidrosis, which could be confused with other causes of the condition. Prospective studies are necessary to assess the incidence of PPI-induced hyperhidrosis and its mechanism.

INTRODUCTION

The Food and Drug Administration (FDA) has approved proton-pump inhibitor (PPI) for the treatment of pathological hyper secretory conditions like Zollinger-Ellison syndrome, gastroesophageal reflux disease and erosive esophagitis. [1] As of 2015 The FDA has approved the following PPIs: Omeprazole, Esomeprazole, Lansoprazole, Dexlansoprazole, Pantoprazole, Rabeprazole. [2]

Excessive perspiration beyond what is required to keep the body temperature steady is known as hyperhidrosis. [3] Several widely used medicines can lead to hyperhidrosis. These consist of antidepressants, antihypertensive medications, antineoplastic agents, antimigraine drugs, antipyretics, cholinergic agonists, hypoglycemic agents, hormonal therapies, immunosuppressant, opioids, and protease inhibitors. Also, endocrine disorders associated with hyperhidrosis include

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Thyrotoxicosis, Hypoglycemia, Diabetic peripheral and autonomic neuropathy, Acromegaly, Pheochromocytoma/Paraganglioma, Menopause, Male hypogonadism, Carcinoid syndrome, Diabetes insipidus. [4]

The food and drug administration adverse event reporting system (FAERS) database contains adverse events (AEs) reported to the FDA by different countries regarding post marketing drugs [5]. Through the FAERS database from Q4/2003-Q3/2024, we retrieved a total of 141920 adverse event reports related to hyperhidrosis. [6] Herein, we present a case report of a 56-year-old female who developed hyperhidrosis during her stay in hospital.

CASE PRESENTATION

A 56-year-old female with Medical history significant for chronic liver disease was admitted with the complaints of bilateral lower limb swelling, facial puffiness, yellow discoloration of sclera and associated with vomiting 2 episodes, diarrhea of 3 to 4 times per day. The final diagnosis was decompensated chronic liver disease (DCLD), which is not linked to hepatatorenal syndrome (HRS) and was accompanied by portal hypertension and atrophic pancreatitis with no other comorbidities. she was not a smoker and alcoholic. Examination revealed a well-appearing, moderately built, nourished and well oriented to time female. Blood pressure was 116/80 mm Hg, pulse 88 beats per minute (bpm) and body mass index (BMI) 22.5 kg/m2. There were S1, S2 cardiac murmurs with presence of rhonchi and lungs were clear.

Laboratory tests, including electrocardiogram (ECG), 2D echocardiography (2D echo), Ultrasound Sonography (USG) abdomen and pelvis were performed. The test results are as following: ECG showed sinus tachycardia and 2D Echo showed normal left ventricular systolic function (LVSF), no regional wall motion abnormality (RWMA), ejection fraction (EF) 55%, Pulmonary artery systolic pressure (PASP) 30 mmHg. USG abdomen and pelvis showed chronic parenchymal liver disease with portal hypertension, splenomegaly and mild asities. CEA (carcinoembryonic antigen) level was found to be 14.6 ng/ml. Amylase, lipase was found to be in normal limits.

When pantoprazole 40mg iv was initiated on her admission, she reported heat intolerance and sweats starting on the face and spreading towards her upper part of the body (right and left arm pits). Following the adverse event, the patient's vital signs were assessed and revealed as blood pressure 100/80 mm Hg, pulse rate 122 beats per minute (BPM), respiratory rate 22 cycles per minute (CPM). The further daily orders for injection pantoprazole was stopped and managed by administering 1-pint bolus normal saline after transferring patient to ICU.

DISSCUSSION

Excessive sweating (hyperhidrosis) seems to be an occasional problem with omeprazole and lansoprazole. We are aware of 3 cases in the literature of PPI induced hyperhidrosis involving omeprazole and lansoprazole. Case 1 describes a 62-year-old man with medical history of gastroesophageal reflux disease (GERD) was on lansoprazole 15 mg for 5 years. Post which, he reported heat intolerance and sweats starting on face and spreading towards his legs that occurred mostly at night. Case 2 describes an 83-year-old man with significant medical history for esophagitis and had been on omeprazole 20 mg orally twice daily for 9 months, later developed hyperhidrosis comprising the upper chest and antecubital fossae. [4] Case 3 describes a 50-year-old man with liable blood pressure and non-ulcer dyspepsia developed hyperhidrosis with

omeprazole 20 mg orally daily taken for about 3 years. [7] In the three cases described above, cessation of PPIs alleviated hyperhidrosis symptoms; however, upon resuming the PPI, hyperhidrosis recurred.

PPI-induced hyperhidrosis seems to be rare, and exact mechanism has not been established.

Hitherto, to our knowledge this is the first case of hyperhidrosis involving pantoprazole. PPIs work as antacids by blocking the gastric parietal cells' H+/K+ ATPase pump. An increase in gastric pH causes G cells to release more gastrin, which in turn triggers the release of histamine by activating the cholecystokinin 2 receptor on enterochromaffin-like (ECL) cells. Histamine then lowers the pH of the stomach by acting on parietal cells. PPIs impede the negative feedback on gastrin synthesis, which causes hyperplasia and increases the functional capacity of parietal and ECL cells. The possibility that histamine contributes to PPI-associated hyperhidrosis is raised by the increase in histamine levels. The other possible mechanisms include, the influence of gastrin on ECL cells, which exhibits enhanced transcription of the CgA gene, resulting in increased CgA synthesis. The markedly increased CgA levels with PPI therapy when it is used for as little as one week. As CgA expression has also been identified in sweat glands and ducts, it can be postulated that elevations in CgA levels may play a role in linking hyperhidrosis and PPI therapy. [4]

There are no concomitant medications or contributory variables from comorbid diseases, which may be linked to hyperhidrosis. Also, patient had elevated CEA levels, for which research indicates that elevated levels of CEA in the blood can be associated with a condition called Acquired Idiopathic Generalized Anhidrosis (AIGA), which is characterized by significantly reduced sweating. Therefore, high CEA levels can be indirectly linked to decreased sweating, not increased sweating. Hyperhidrosis was experienced by the patient despite having an increased CEA antigen. [8] After ruling out other potential causes, we discerned that pantoprazole might be the cause, as there aren't any other ones.

Our study involves few limitations. Firstly, reintroduction of pantoprazole which strengthens the casual relationship was not performed, considering the economical constrains due to transfer-in from ward to ICU. Secondly, CgA levels was not obtained as the patient had been on PPI for a shorter period of time.

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