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Case Report

An unusual case of hematemesis in a patient with type 1 diabetes mellitus

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Abstract

Diabetes is a metabolic disorder characterised by hyperglycemia, with disturbances of carbohydrate, fat and protein metabolism. Here is the report of an unusual case of hematemesis in a patient with type 1 diabetes mellitus. Hematemesis refers to vomiting of blood and can be explained as the backward flow of blood through the upper gastrointestinal tract. Hematemesis is an extremely serious condition that should be treated at the earliest. Diabetics are prone to opportunistic infections by Candida, which can range from oral candidiasis to oesophageal candidiasis. Oesophageal candidiasis causes erosion of the stomach and oesophageal wall which can lead to gastritis and hematemesis.

Keywords: Type 1 Diabetes Mellitus, Hematemesis, Oesophageal Candidiasis

1. Introduction

Hematemesis refers to the vomiting of blood from the upper gastrointestinal (GI) tract, which includes the mouth, pharynx, oesophagus, stomach, and the small intestine. The most common causes of acute upper GI bleeding are peptic ulcer, gastritis, and oesophagitis[1]. Manifestation of bleeding depends on the source, rate of bleeding, and underlying or coexistent disease. Often coexistent conditions like diabetes can manifest with gastrointestinal tract complications (in 75% of cases) and even lead to hematemesis[2]. Below we report an interesting case of a Type 1 diabetes mellitus with hematemesis.

2. Case Report

A 28-year-old woman presented to the emergency ward with complaints of 15 -20 episodes of coffee coloured vomitus since a day. She was a known case of Type 1 diabetes that was on insulin and had poorly controlled blood sugar levels. At the time of admission her blood sugar levels were high and she had acidotic breathing, a further evaluation revealed deranged metabolic parameters (ketone bodies +) and a diagnosis of Diabetic Ketoacidosis (DKA) was made. She was started on insulin infusion and her metabolic parameter stabilized. Detailed history revealed that she was not on any NSAIDS or other drugs causing gastric irritation and bleeding diathesis.

She is a non-alcoholic, non- smoke. Her family history revealed nothing significant. There was no history suggestive of Acid peptic disease (APD) or abdominal surgeries.

Her height: 159 cm and body Weight: 47 kg.
BMI – 18.59.Hb -11.2 gm/dl.
Total counts -14,300/µL,
Platelets -4,59,000/µL,
Red blood cells -4,28,000/µ L,
Peripheral smear –normocytic normochromic blood picture with neutrophilicleucocytosis and thrombocytosis.
HIV, HBSAG –negative.
Urine routine:
Albumin –traces,
Sugar -0.5%,
Acetone –present,
WBC-3-4,
RBC–Nil,
Epithelial cells -2-3,
Casts, crystals –Nil.
Random blood sugar -244mg/dl,
Blood urea -39 mg/dl,
Serum creatinine -1.0 mg/dl,
Sodium -144 meq/L,

Potassium-4.3meq/L.
USG abdomen – normal.
ECG- Normal sinus rhythm,
Chest X ray – normal.
Lipid parameters – within normal limit.

On further evaluation an endoscopy was undertaken which revealed whitish patches seen in the oesophagus, with pale and edematous stomach wall confirming oesophageal candidiasis, gastritis and duodenitis. *Helicobacter Pylori* colonization was absent as shown by a negative urease breath test. Patient was successfully managed with oral fluconazole, proton pump inhibitors, euglycemic treatment and correction of metabolic parameters.

3. Discussion

Diabetes is a metabolic disorder characterised by hyperglycaemia, with disturbances of carbohydrate, fat and protein metabolism, resulting from defects in insulin secretion, insulin action or both. It is known that diabetes can present with gastrointestinal symptoms. In the upper gastrointestinal tract these complications could manifest as dyspepsia, abdominal pain, nausea, vomiting and anorexia. Poor glycemic control also predisposes to infection by candida. Candidiasis is one of the most common infections associated with diabetes. Often oesophageal and oral candidiasis can be manifestations present in diabetes in mellitus even the absence of any other secondary causes like immunosuppressant therapy, malignant disease or use of intravenous hyperalimention. Carcinomas, diabetes mellitus, corticosteroid and antibiotic therapy are the major risk factors for candida esophagitis[3].

Risk factors for Candida esophagitis have been documented in several series. These include pharmacological suppression of gastric acid production, use of antibiotics, previous vagotomy, functional or mechanical oesophageal abnormalities, and endocrine diseases such as diabetes mellitus, hypothyroidism and hypoparathyroidism.[4] Malnutrition, alcoholism, advanced age, and therapy with corticosteroids - either systemic or inhaled - has also been implicated[4].

Biological analyses have revealed by which hyperglycemia triggers candidiasis specifically to diabetic patients

1) Neutrophil dysfunction
2) Impaired opsonisation because of glucose binding to the third component
3) Virulence of the pathogens that enables them to grow rapidly in the hyperglycaemic environment.

Candida albicans, the most frequently candida species in oral mucosa specimens from diabetic patients contains a glucose – inducible protein that inhibits phagocytosis. Thus apart from neutrophil dysfunction, hyperglycaemia itself may be the risk factor of candidiasis[5].

Infective esophagitis is a rare disease, affecting mostly immunocompromised patients. *Candida esophagitis* is one of the most common opportunistic infections in patients with impaired immunity and the most common cause of oesophageal disease in patients. It also occurs in debilitated patients who have received broad-spectrum antibiotics, steroids and immunosuppressants.[3]

Candida esophagitis was graded as the following:
Grade 1: as scattered mucosal plaques involving less than 50 % of the esophageal mucosa,
Grade 2: as mucosal plaques involving more than 50%esophagealmucosa,
Grade 3: as confluent plaque material circumferentially coating at least 50%of the esophageal mucosa but without luminal impingement,
Grade 4: as circumferential plaque mat coating at least 50 % of the esophageal mucosa with luminal impingement despite air insufflations.[3]

In most cases, an ulcer could be readily distinguished endoscopically by the marked hyperaemia and granularity of the ulcer base from the surrounding *Candidaesophagitis*[3].

All patients diagnosed with Candida esophagitis did not have oral thrush[3].Diabetic patients complicated with *Candida esophagitis* had uncontrolled diabetes at the time of presentation, irrespective of its type[3]. Epigastric pain has been known to be associated with *candida* infection. *Candida esophagitis* should be considered early in patients who have been on steroids and antibiotic treatment and presented with upper gastro-intestinal symptoms.

Oral candidiasis does not accompany *candida esophagitis*. Our study showed that *candida esophagitis* by itself was an easily managed complication.

4. Conclusion

Candida esophagitis is more common due to chronic diseases, poor glycemic control, corticosteroid and antibiotic therapy which impair the immune system[3]. It occurs in the absence of local obstructive lesions and responds to treatment with nystatin and fluconazole.

Neuropathic complications in diabetes frequently result in a mild loss of oesophageal contraction amplitude, a slowing of the velocity of peristalsis, and a resultant delay in oesophageal emptying. But more severe abnormalities are uncommon, and that degree of oesophageal motor change does not cause symptoms. When dysphagia or chest pain occur in diabetics, they should be thoroughly investigated with X-ray, endoscopy, and, if warranted, motility. The only condition which probably occurs with greater frequency in diabetes is *oesophageal candidiasis*.[6]

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