

Trying to stomach multiple myeloma: A case report

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ABSTRACT

We report a case of a 51-year-old male with minimal past medical history who presented to his primary care provider with nausea, vomiting and constipation in the outpatient setting. Concomitantly, he was found to have a renal injury, anemia and lytic lesions which were confirmed to be due to multiple myeloma. After further investigation of the gastrointestinal symptoms, he was diagnosed with gastroparesis. This case represents an unusual presentation of gastroparesis, diagnosed at the same time as multiple myeloma, for which there has yet to be a published association. Here we detail the case, review gastric emptying physiology, the diagnostic criteria for gastroparesis and hypothesize the connection if it might have with multiple myeloma.

Keywords: Gastric emptying study, gastric motility, gastroparesis, hypercalcemia, multiple myeloma

Introduction

Gastroparesis is a disorder of gut motility in the absence of a physical obstruction.^[1] Symptoms range from nausea, vomiting, postprandial bloating, inability to finish a modest-sized meal and in prolonged/severe cases: significant weight loss.^[1] It is most commonly associated as a complication of diabetes; however, there are other well-established offenders such as scleroderma, medications, Parkinsons and even critical illness alone.^[2,3] The prevalence of gastroparesis is not known but limited studies show 37.8/100,000 women suffer from gastroparesis and 9.6/100,000 of men.^[4] Despite this, there is concern that gastroparesis might be significantly underdiagnosed in the primary care setting.^[5]

This report details a unique case of the concomitant diagnosis of gastroparesis and multiple myeloma (MM). While hypercalcemia from MM can have gastrointestinal manifestations such as nausea and constipation, MM is not an established cause of gastroparesis.^[6] This presentation is of

particular significance to the primary care physician (PCP) as a majority of patients with MM are diagnosed in the outpatient setting.^[7] Recognizing that gastroparesis could be related to an uncovered disease such as MM might lead to earlier diagnosis and treatment.

Case History

A 51-year-old male presented to his PCP with intractable postprandial vomiting as well as left lower quadrant abdominal pain, bloating, early satiety, and constipation for about 1 month. A review of systems yielded only severe fatigue. Upon presentation, his vitals were normal aside from mild hypertension, 142/79. Physical exam was notable for poor dentition, dry mucous membranes and decreased bowel sounds. Laboratory studies were significant for: normocytic anemia (hemoglobin of 8.9 g/dL, RDW of 17.5%, and normal iron studies), calcium of 13.5 mg/dL, BUN of 30 mg/dL and creatinine of 3.0 mg/dL. His PCP sent him to the hospital for treatment of hypercalcemia and an acute kidney injury (AKI).

Upon admission to the hospital, he received a CT abdomen which was negative aside from atherosclerosis. For the hypercalcemia and AKI, he was started on intravenous fluids

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and calcitonin with incremental improvement [Figure 1]. Given his (C) hypercalcemia, (R) renal dysfunction, and (A) anemia, a peripheral blood smear, SPEP, UPEP and bone marrow biopsy were conducted. These showed elevated serum and urine-free light lambda chains, elevated beta-2 microglobulin and peripheral smear without increased rouleaux formation. The bone marrow biopsy revealed hypocellular with plasma cell dyscrasia and flow cytometry for 1.6% abnormal CD56/CD28 positive plasma cells and monoclonal lambda, confirming MM. A skeletal survey done afterwards found multiple lytic (B) bone lesions [Figure 2], completing the C.R.A.B. presentation.

Despite successful treatment of his hypercalcemia, his gastrointestinal symptoms persisted prompting further investigation. An esophagogastroduodenoscopy (EGD) and a gastric emptying study (GES) revealed findings consistent with gastritis, no obstruction, and markedly impaired gastric emptying (8% emptying at 2 hours and – 3% at 4 hours) [Figure 3]. He was diagnosed with gastroparesis for which he was started on a modified diet and metoclopramide. Over the following 3 days, there was progressive improvement in gastrointestinal symptoms. He was discharged with metoclopramide, a chemotherapy regimen and appointment for stem cell transplant evaluation. His metoclopramide was prescribed for a limited time after discharge while receiving chemotherapy and after 2 months his gastrointestinal symptoms only consisted of mild nausea while receiving chemotherapy.

Discussion

In a normal functioning stomach, chyme enters in the proximal stomach, which serves primarily as a reservoir, and then travels to the distal part which contracts to grind the meal and empty it into the duodenum. Gastric contractions are regulated by interstitial cells of Cajal (ICC). These cells are mesodermal in origin and located near the myenteric plexus primarily in the greater curvature of the stomach. They receive input from the

vagus nerve and enteric nerves to create depolarizations along the cellular membranes known as slow waves at the rate of about 3 beats per minute and directly correlate with gastric contractions. The contractions create a peristaltic wave from the mid-corpus to the pylorus which grind food in the antrum and afterwards evacuate into the duodenum. The rate of emptying is variable and dependent on multiple factors including the consistency of the meal and calorie content.^[1,8]

The evaluation of gastroparesis starts with ruling out organic causes such as physical obstruction with an EGD and correcting electrolytes as done with our patient above.^[8] His symptoms were initially attributed to hypercalcemia which is known to cause nausea and constipation, especially when above 12 mg/dL; however, he remained symptomatic after swift correction.^[6] The diagnosis of gastroparesis is confirmed with a GES based on the percentage of gastric content retention after 4 hours: mild: less than 15%, moderate: 15%-35%, and severe: greater than 35%.^[9] His GES demonstrated no emptying at 4 hours consistent with severe gastroparesis. Typical treatment for gastroparesis is diet modification (smaller meals), symptom management and metoclopramide (a prokinetic agent and only FDA-approved medication for gastroparesis).

Our case of gastroparesis is unique in that it was diagnosed at the same time as MM. The pathophysiology of gastroparesis is not entirely clear; however, there is no established connection with MM. Our current explanation is multifactorial with proposed mechanisms ranging from dysfunction of smooth muscle, ICC and nearby macrophages, and/or myenteric neurons.^[2,10,11] The relationship between these two diseases may be related to the elevated inflammatory state of MM that has previously been studied.^[12] In one study where mice with diabetes were examined, delayed gastric was found to be associated with increased pro-inflammatory factors, decreased inhibiting inflammatory factors by gastric macrophages and decreased ICC.^[11] Another cause may be related to nerve damage. There have been some studies of myeloid nerve deposition in MM, which may have directly affected the stomach's autonomic system, myenteric plexus, enteric nerves or the vagus nerve.^[13,14]

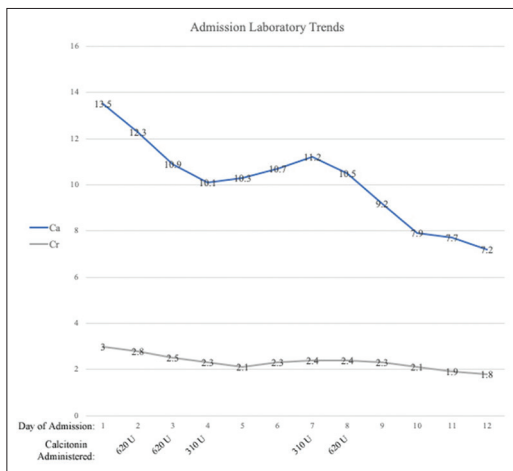


Figure 1: The patient's serum calcium and creatinine trend each day of admission with treatment

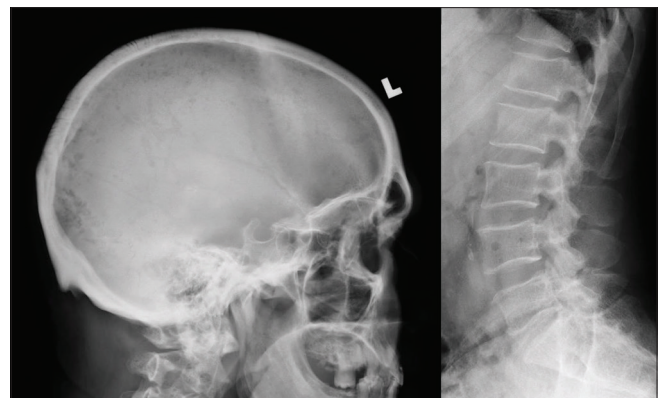


Figure 2: Cranial and lumbar spinal lytic bone lesions

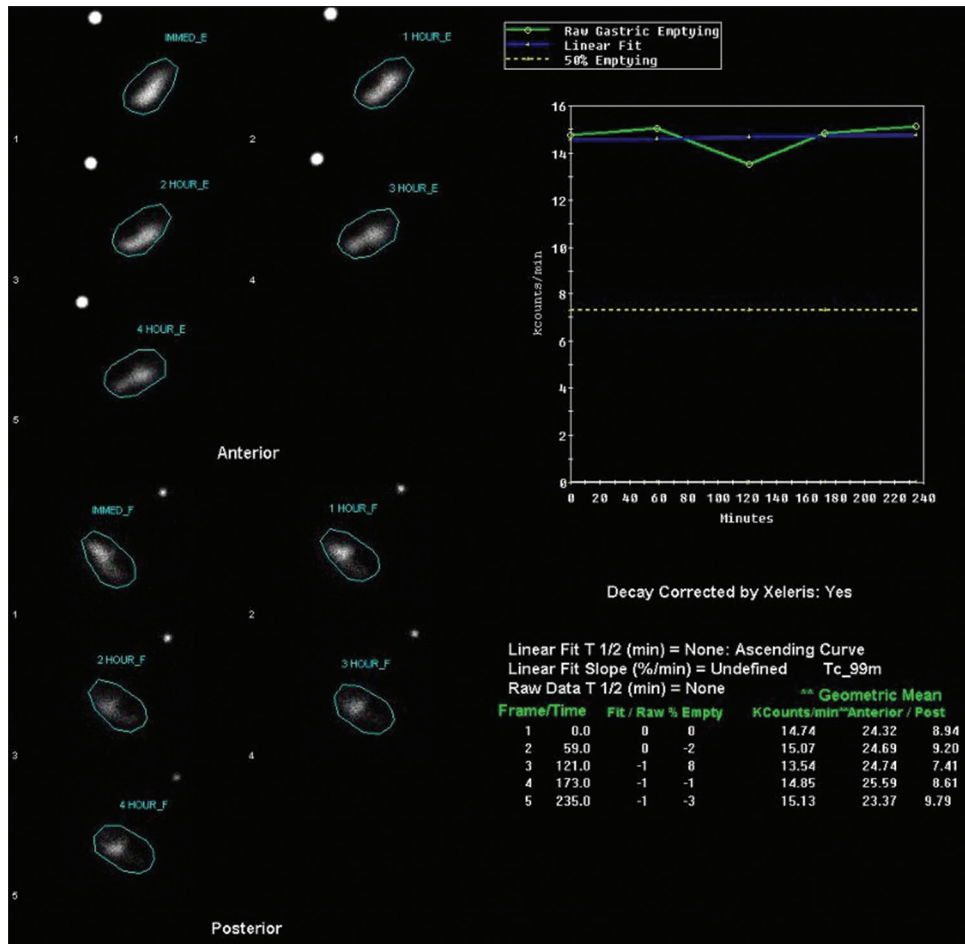


Figure 3: Gastric emptying study consistent with severe gastroparesis: -1% emptying after 4 hours

The physiology of a normal emptying stomach and its intricacies helps one understand why a systemic illness might cause dysfunction. We are unlikely to know the exact cause of this patient’s gastroparesis but the case highlights how MM truly a disease of protean manifestations is. PCPs should be vigilant for both gastroparesis and MM and now possibly at the same time.

Key Messages

A unique presentation of gastroparesis diagnosed at the same time as multiple myeloma. This case lends the opportunity to review gastric motility physiology as well as how to diagnose gastroparesis. A potential relationship between the two diseases is also discussed in this study.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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