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An unusual manifestation of diabetic ketoacidosis and acute colonic pseudo-obstruction

Jake Natalini, Hala Borno, Lan Jin and Trevor Jensen

Department of Medicine, University of California, San Francisco, CA 94143, USA Corresponding author: Trevor Jensen. Email: trevor.jensen@ucsf.edu

Lesson

Patients presenting with diabetic ketoacidosis and acute colonic pseudo-obstruction should undergo a focused evaluation to identify underlying precipitants.

Keywords acute colonic pseudo-obstruction, pheochromocytoma, diabetic ketoacidosis

Case

A 50-year-old Filipino woman with a history of type II diabetes mellitus and hypertension presented to the emergency department with three days of generalised weakness and abdominal discomfort. She also described decreased oral intake for several days prior to presentation due to persistent nausea and intermittent facial flushing since the onset of her symptoms. She denied fevers, chest pain, dyspnoea, cough, headache, and dysuria. Her last bowel movement was four days prior to presentation. She took metformin daily and glipizide only when her blood glucose was higher than 140 mg/dL. Her only other medication was lisinopril. She denied alcohol, tobacco, or illicit drug use.

The patient's vital signs on presentation were a temperature of 37.1°C, heart rate of 152 beats/min, respiratory rate of 16 breaths/min, blood pressure of 175/79 mmHg, and oxygen saturation of 100% on ambient air. She had facial and neck flushing. Other pertinent findings on physical examination included dry mucous membranes, tachycardia, a regular heart rhythm, and a normal S1 and S2 without murmurs. She had hypoactive bowel sounds with a moderately distended and diffusely tender abdomen.

Laboratory tests demonstrated a serum sodium of 120 mmol/L, potassium of 5.3 mmol/L, chloride of 76 mmol/L, carbon dioxide of 25 mmol/L, urea nitrogen of 30 mg/dL, creatinine of 0.94 mg/dL, glucose of 612 mg/dL, and lactate of 2.1 mmol/L. Her anion gap was 19 (24 if corrected for an albumin of 2.1 g/dL). Her white blood cell count was 19.3×10^9 cells/L.

Her haemoglobin and platelet counts were normal. Her initial troponin I was $0.07 \,\mu\text{g/L}$ (reference range: $<0.04 \,\mu g/L$). Urinalysis demonstrated a glu- $\cos e > 500 \text{ mg/dL}$ and ketones of 20 mg/dL. The serum beta-hydroxybutyrate was 3.67 mmol/L. A haemoglobin A1c was 9.2%. Her serum lipase was within normal limits. Her chest radiograph was normal. An acute abdominal series demonstrated dilation of the large bowel measuring up to 7 cm within the region of the transverse colon (Figure 1).

The patient was aggressively resuscitated with isotonic crystalloid solution and was started on an insulin infusion. Her hyponatremia and anion gap resolved over the ensuing 24-48 h. Despite resuscitative efforts, the patient remained persistently tachycardic with a heart rate ranging 140-160 beats/min. She was treated empirically for sepsis with broadspectrum antibiotics. Her serum troponin I peaked at 0.10 µg/L. Serial electrocardiograms demonstrated sinus tachycardia and Q waves in the inferior and anteroseptal leads suggestive of prior infarct, but there were no acute ST segment or dynamic T wave changes. A trans-thoracic echocardiogram demonstrated preserved left ventricular systolic function with an estimated ejection fraction of 60%-65% without wall motion abnormalities. Two sets of blood cultures drawn at the time of presentation remained negative.

A CT scan of the abdomen and pelvis, which was ordered to further assess the X-ray findings, confirmed diffuse massive dilation of the colon with an abrupt transition point at the splenic flexure and decompression of the distal colon. No anatomic obstruction was identified. The combination of the hypoactive bowel sounds and CT imaging pointed to acute colonic pseudo-obstruction. Her CT scan also demonstrated a 6.9×7.0 -cm heterogeneous, partially necrotic mass within the right adrenal gland (Figure 2). In the context of hypertension, tachycardia, nausea, and acute-onset facial flushing, pheochromocytoma was suspected. Plasma total free metanephrines and 24-h urine total metanephrines

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Figure 1. Abdominal radiograph demonstrating dilatation of predominantly large bowel, measuring up to 7 cm within the region of the transverse colon (indicated by the white arrow).



Figure 2. Abdominal CT scan revealing a 6.9×7.0 -cm heterogeneous, partially necrotic mass, identified within the right adrenal gland (indicated by the white arrow), as well as diffuse dilatation of the colon, measuring up to 7 cm within the region of the transverse colon. There is an abrupt transition point at the splenic flexure with decompression of the distal colon without identification of an obstructing mass (not visualised here).



were markedly elevated (21,282 pg/mL) and 42,638 mcg/24 h, respectively).

The patient's blood pressure and heart rate remained persistently elevated. She was started on phenoxybenzamine and a nicardipine infusion and had marked improvement of her vital signs. Her acute colonic pseudo-obstruction was reversed with the administration of phenoxybenzamine and an aggressive bowel regimen. She underwent a right adrenalectomy, which confirmed the diagnosis of pheochromocytoma. Since surgery, she has had no recurrence of abdominal symptoms. In addition, her diabetes and hypertension have improved dramatically.

Discussion

This case highlights unique catecholamine-driven manifestations of pheochromocytoma, as well as an unusual manifestation of an otherwise common inpatient diagnosis. Pheochromocytoma is a rare functional neuroendocrine tumour arising from the chromaffin cells of the adrenal medulla. Amongst patients with hypertension, estimated prevalence ranges from 0.1% to 0.6%.^{1,2} It is thought to be much rarer in the general population. Clinical manifestations of pheochromocytoma are varied. The classic triad of symptoms is episodic headaches, palpitations, and diaphoresis, all of which occur in response to the intermittent release of epinephrine and norepinephrine into the bloodstream.^{3,4}

Overt diabetes mellitus can occur in patients with pheochromocytoma because catecholamines antagonise insulin release.⁵ Furthermore, previous case reports have described diabetic ketoacidosis as a rare manifestation of pheochromocytoma.^{6,7} Acute colonic pseudo-obstruction has been observed in approximately 25% of cases of pheochromocytoma.⁸ Though the mechanism is somewhat controversial, high levels of circulating catecholamines are thought to cause intestinal smooth muscle relaxation, thereby causing non-mechanical obstruction.

Acute colonic pseudo-obstruction, also referred to as Ogilvie's syndrome, is a condition in which the large bowel acutely dilates in the absence of an obstructing anatomic lesion. Although it can involve the entire colon, it typically presents with dilation of the caecum and right hemicolon. The condition mimics mechanical large bowel obstruction, causing symptoms of nausea, vomiting, and abdominal pain and distention.9 Common precipitants are major surgery, severe illness, neurologic conditions, and medications such as opioids, calcium channel blockers, alpha-2-adrenergic agonists, and epidural analgesics.^{9,10} The condition is more common in men and predominantly affects patients over the age of 60 years. Treatment is primarily supportive, along with decompression of the bowel and removal of the causative agent. In addition, current literature underscores the importance of early surgical consultation due to the high risk for bowel perforation.⁸

In conclusion, this case highlights how the investigation of a clinical precipitant is a crucial component in the management of diabetic ketoacidosis. A focused evaluation for potential aetiologies of diabetic ketoacidosis is warranted in all patients in order to not miss rare causes. This case also highlights the salient features of acute colonic pseudo-obstruction, a condition uncommonly managed by internists.

Declarations

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Guarantor: TJ.

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