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Letters to the editor

A case of sumatriptan-induced intestinal ischemia

To the Editor: Sumatriptan succinate, a serotonin-l (5-hydroxytryptamine-1) receptor agonist, is an antimigraine drug that is reported to act by selectively constricting intracranial arteries. Recently, vasopressor responses that are distinct from the cranial circulation have been demonstrated to occur in the systemic, pulmonary, and coronary circulations. A series of eight cases of ischemic colitis in the setting of sumatriptan have been reported previously.[1] We present a case report of focal segmental ischemia of the small bowel in the setting of migraine headaches in a patient treated with sumatriptan.

A 20-year-old female patient was admitted to the hospital in June 2005 with severe abdominal pain, nausea, and vomiting. She had been having intermittent but worsening symptoms for 4 months before admission. She described her abdominal pain as dull, periumbilical, without radiation, with no relation to food or bowel movements. Patient denied diarrhea, constipation, melena, or hematochezia. She had a 20-pound weight loss over the past 4 months. Past medical history was significant for migraine headaches. She did not have any prior abdominal surgeries. She was taking Topiramate and Imitrex (sumatriptan). She denied any use of oral contraceptives. She denied smoking tobacco or drinking alcohol. Family history was significant for Crohn's disease (maternal uncle). The patient disclosed that she had been using 50 mg sumatriptan prn for migraine headaches. She reported that she consistently took sumatriptan for migraine headaches and that her abdominal complaints usually started approximately 24 h after her intake of sumatriptan. She had visited the ER for severe migraine the day before her symptoms became severe and was given sumatriptan.

Physical examination was significant for mild periumbilical tenderness with no rebound. No masses were felt, and there were good bowel sounds. She was afebrile and her blood pressure and heart rate were within normal limits. Her serum chemistry values, including liver function tests, were within normal limits. Amylase was slightly elevated at 127 (30–110). Lipase was within normal limits. Hemogram showed leukocytosis with a white count of 12.7 thousand with 80% neutrophils. Hematocrit and platelet count were within normal limits. She had normal serum protein electrophoresis. ESR and CRP were within normal limits. Her stool was positive for occult blood.

She had a KUB (plain abdominal film) film and a right upper quadrant ultrasound done, which were normal. She had an abdominal computed tomography (CT) scan, which showed thickening of terminal ileum suspicious for Crohn's (Fig. 1). She had a small bowel follow-through that showed a focal edematous loop of small bowel in the left lower quadrant approximately 5 cm in length. She underwent a colonoscopy that showed mild erythema and nodularity of her terminal ileum. She had a capsule endoscopy and a small bowel magnetic resonance imaging, (MRI), both of which were normal. Her terminal ileal biopsies showed a normal villous pattern without ileitis. Serological markers for irritable bowel syndrome (IBD) including ASCA, OmpC IgA, and pANCA were negative.

Due to her intractable symptoms, she was started on TPN and on IV antibiotics including ciprofloxacin and metronidazole, along with other supportive measures. Given her abdominal CT findings suspicious for Crohn's, she was started on prednisone.

She was seen at our clinic, where we diagnosed her symptoms to be secondary to sumatriptan use. We tapered her prednisone and advised her not to take any more sumatriptan. She remains symptom free since that time.

Vascular insults to short segments of small bowel produce a broad spectrum of clinical features without the life-threatening complications that are associated with more extensive ischemia. The causes of focal segmental ischemia (FSI) of the small bowel include atheromatous emboli, strangulated hernias, immune complex disorders and vasculitis, blunt abdominal trauma, segmental

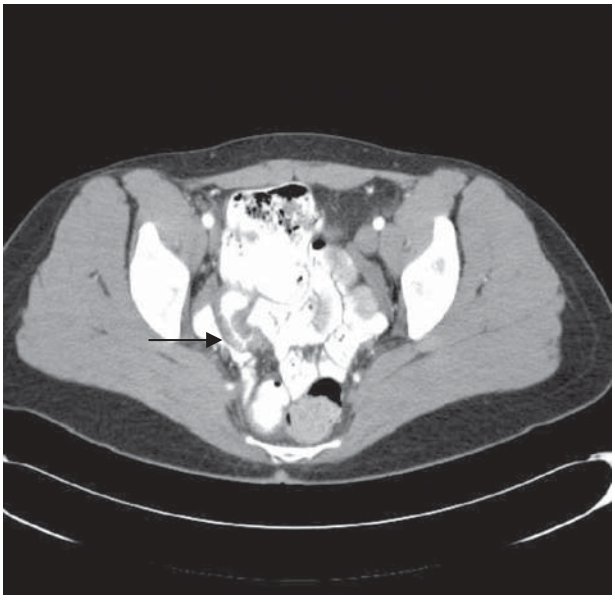

Fig. 1. Computed tomographic imaging showing thickening of terminal ileum

venous thrombosis, radiation therapy, and drugs including oral contraceptives, among others. With FSI there is usually adequate collateral circulation to prevent transmural infarction; the most common lesion is partial bowel wall necrosis. Limited necrosis may present as acute enteritis, chronic enteritis, or a stricture. In the acute pattern, abdominal pain often simulates acute appendicitis. Physical findings are those of acute abdomen, and an inflammatory mass may be palpated. The chronic enteritis pattern may be indistinguishable from that of Crohn's disease and includes crampy abdominal pain, diarrhea, fever, and weight loss[2].

Given the fact that the patient's biopsies did not show any evidence of Crohn's and that her serologies were negative along with normal ESR and CRP at the time of admission, we believe that she does not have Crohn's. On the other hand, given the temporal association between her abdominal pain and use of sumatriptan, we think that this is focal segmental ischemia of her small intestine secondary to vasospasm induced by sumatriptan. There have been multiple cases of colonic ischemia reported with sumatriptan use, but so far there has been only one report of small bowel involvement with sumatriptan use.[3] Thus, focal segmental ischemia of the small intestine should be considered in the differential in suspected cases of Crohn's disease in patients on triptans.

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Solid serous adenoma of the pancreas: a solid variant of serous cystadenoma or a separate disease entity?

To the editor: Solid serous adenoma of the pancreas has been reported to be a solid variant of serous cystadenoma (SC) of the pancreas because this peculiar pancreatic neoplasm has a solid appearance and is formed by clear cells indistinguishable from those lining the cysts of SC.[1] In this report, we demonstrate a case of this rare pancreatic benign tumor and discuss whether it is a variant form of SC or a separate disease entity.

A 58-year-old woman was referred to our institution as having a well-defined hypoechoic mass in the body of her pancreas. There were no symptoms and no abnormalities such as tumor markers or islet hormone levels in blood tests. Computed tomography of the pancreas revealed a well-enhanced solid mass in the body with a dilated distal pancreatic duct, so we suspected a malignant islet cell neoplasm (Fig. 1). A distal pancreatectomy was performed. A cut section of the resected specimen showed a well-demarcated, solid, nodular lesion measuring 2.0 cm × 1.8 cm in the body of the pancreas. Microscopic study revealed an encapsulated tumor composed of several vague lobules with dense collagenous septa. Each lobule exhibited a collection of tubular glands (Fig. 2a). The tumor cells had small round nuclei and clear or pale eosinophilic cytoplasm (Fig. 2b). No pleomorphism, necrosis, or mitosis was identified. Some tumor cells contained glycogen, as shown by the presence of granules, revealed by

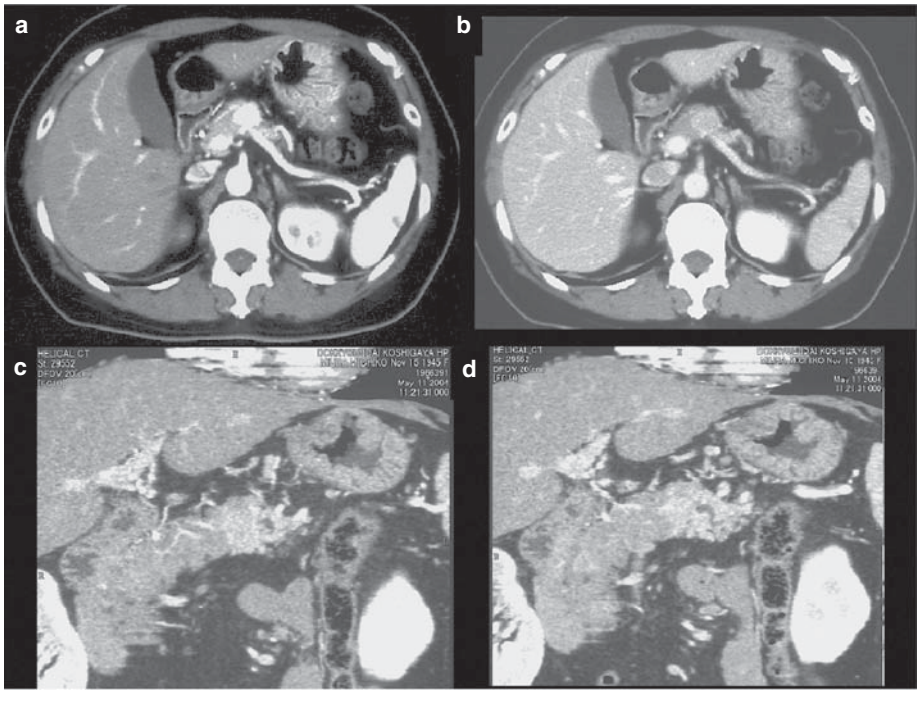

Fig. 1a–d. Computed tomographic images of the pancreas showing a well-enhanced solid tumor in the body (**a** early phase, **b** late phase) and the dilated distal pancreatic duct (**c, d**)