Abdominal Pain and Weight Loss in New-Onset Type 1 Diabetes

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Patients who are newly diagnosed with type 1 diabetes are routinely counseled by their health care professionals to make lifestyle changes and take other measures to improve their glycemic control and prevent long-term complications. However, the rapid achievement of metabolic control can lead to unforeseen consequences. We recently identified one such case, in which rapid improvement in metabolic control precipitated insulin neuritis with associated weight loss, resulting in extensive and unnecessary investigations.

PRESENTATION
A 31-year-old man presented to our hospital emergency department with new-onset type 1 diabetes complicated by ketoacidosis. He responded to intravenous fluids and insulin and was discharge on day 2 on a basal-bolus insulin regimen using premeal insulin aspart three times daily and insulin detemir at bedtime. Under the supervision of a diabetes specialist nurse, his metabolic control improved, with self-monitoring of blood glucose results between 90 and 126 mg/dl. The patient did, however, develop severe right-side lower abdominal pain that was associated with weight loss, and was accordingly referred to a gastroenterologist. Testing was performed to exclude celiac disease and was accordingly referred to a gastroenterologist. Testing was performed to exclude celiac disease, and the patient also underwent numerous tests including urine culture, abdominal ultrasound, CT scan of the abdomen, colonoscopy, and barium meal follow-through, all of which yielded normal or negative results.

The patient was seen in the diabetes clinic 2 months after the episode of ketoacidosis and still complained of sharp pain over his right side. There was no aggravating factor, but he reported nocturnal exacerbation of pain that disturbed his sleep. It transpired that his appetite had reduced, and he had lost ~15 lb in weight since his diagnosis, despite having good glycemic control. His A1C had fallen from 14.4 to 7.1% within that 2-month period. During the consultation, he denied body image problems or excessive exercise or self-induced vomiting. On further examination, he had mild tenderness over the right iliac fossa region and had altered sensation in the dermatome supplied by the right T12 and L1 nerve root. He was diagnosed with “truncal neuropathy” due to “insulin neuritis” causing pain and cachexia. The patient was prescribed amitriptyline, 25 mg at night, with the dose gradually increasing to 75 mg. His pain and appetite improved, and he gained 13 lb within a month.

QUESTIONS
1. Should blood glucose be lowered gradually in all cases to avoid “insulin neuritis”?
2. Is there any association between weight loss and “truncal neuropathy”?
3. Could the patient in this case have had an underlying behavior disorder?
4. Were all of the invasive investigations performed in this case necessary for a 31-year-old man?

COMMENTARY
Acute neuropathy resulting from rapid glycemic control has been reported in literature as “insulin neuritis” that usually manifests with severe excruciating neuropathic pain in the first month of insulin therapy. Symptoms can last up to 6 months and respond to treatment, which is usually required for a similar period. In one observational study, six patients with diabetes experienced severe neuropathic pain, mostly in their feet. The pain started within 2–4 weeks of initiation of intensive diabetes therapy, during which blood glucose levels dropped up to one-fifth of initial levels.

The patient in this case developed localized pain in his abdominal wall within 4 weeks of rapid correction of blood glucose. Similar abdominal wall pain has been reported after rapid reduction of A1C from 12 to 7.5% in a patient with type 2 diabetes. Development of acute painful neuropathy after rapid glycemic control suggests that blood glucose flux is responsible for the pain. Tesfaye et al. elegantly demonstrated several structural abnormalities in the sural nerve, including arteriolar attenuation, tortuosity, and arterio-venous shunting with new vessel formation in patients with insulin neuritis. The combination of structural and
functional changes in the nerves is possibly the cause of neuropathic pain in insulin neuritis.4

Our patient experienced weight loss associated with neuropathic pain, which resulted in a number of clinical investigations. Weight loss associated with painful diabetic neuropathy has been reported in the literature as “diabetic neuropathic cachexia,” which can last up to 1 year. Most patients respond well to neuropathic pain treatment, which provides pain relief and assists in increasing weight. The exact mechanism and cause are unknown.1

In one observational study,9 nine patients with diabetes reported to have painful neuropathy with constant discomfort, and profound weight loss was noted, along with depression and impotence. The severe manifestation subsided in all cases within 10 months and in most cases within 6 months. One case has been reported4 in which the patient presented with profound weight loss associated with painful neuropathy in the abdomen, as was the case with our patient.

The abdominal pain in our case resulted from truncal neuropathy, a condition that manifests with neuropathic pain such as a hypoesthesia, regional hyperalgesia, allodynia, and sometime focal weakness in a specific dermatome region.1,8 The onset is sub-acute, and symptoms are usually unilateral but can be bilateral.

There are many possible causes of pain in the abdominal or thoracic wall; thus, patients with such symptoms often undergo numerous investigations.8–10 There are several cases in which investigations led to a misdiagnosis of hernia, angina, or choledocholithiasis, with patients subsequently failing to respond to treatment for those conditions.8,9,11

The diagnosis of truncal neuropathy is made on clinical grounds with a good history and physical examination. The pain is neuropathic in character (i.e., burning and stabbing), localized to a dermatome, and often associated with altered sensation.8 Most people respond well to neuropathic treatment within 3–12 months.

CLINICAL PEARLS

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