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ENDOCRINE IMAGING

Severe gastrointestinal autonomic dysfunction in a diabetic boy

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Autonomic neuropathies are normally subclinical in diabetic children and adolescents, but in poorly controlled patients may be extremely symptomatic [1]. Gastrointestinal (GI) autonomic dysfunction may include esophageal dysmotility, gastroparesis, dysfunction of intestinal neurons, and decreased/absence of gastrocolic reflex [1, 2]. We report a case of a diabetic boy who had a severe GI dysautonomia related with longstanding poorly controlled diabetes, which reverted following the restoration of glycemic control.

A 15-year-old boy recurred to our hospital emergency unit due to persistent nausea and vomiting, anorexia, weight loss and constipation, with one-week onset and progressive worsening. His medical past was remarkable for type 1 diabetes under intensive insulin therapy, with irregular therapeutical compliance. He confessed rare use of prandial insulin which motivated several past admissions due to diabetic ketoacidosis. At admission, physical examination was noteworthy for abdominal distention with pain, lack of GI sounds, and increased tympanism. Laboratorial data were remarkable for hyperglycemia (290 mg/dL) and ketonemia (0.9 mmol/L) with no acidosis (pH 7.46); glycated hemoglobin was 17.5 %. Abdominal X-ray and computed tomography (CT) scan revealed exuberant gastric and bowel distention with stasis (Fig. 1). An endoscopic study showed alimentary impaction at stomach. The patient was managed with continuous intravenous insulin, parenteral nutrition, metoclopramide, and erythromycin. Glycemia was fastly controlled and patient́s complaints significantly improved. Ten days after, he started oral feeding with tolerance and resolution of dyspepsia and constipation was perceived. Abdominal X-ray and barium esophagram confirmed resolution of GI tract distention and stasis. The patient was discharged two weeks after the admission absolutely asymptomatic.

Gastric emptying and GI motility involve complex coordination of enteric nervous, hormonal, and smooth muscle systems [2]. The pathogenesis of GI dysautonomia is multifactorial involving cellular changes, increased oxidative stress, autoimmune and inflammatory factors, impaired hormonal regulation, and genetic predisposition [1]. Acute hyperglycemia has been demonstrated to induce relaxation of the stomach, increased pyloric tone, and suppression of peristalsis [3]. Moreover, acute glycemic fluctuations may adversely affect GI function [3]. GI dysmotility may lead to unpleasant GI manifestations, deterioration of nutritional status, or unpredictable oscillations of glycemia due to irregular absorption of food/medication [1, 2]. However, rarely can be life-threatening and may lead to long periods of debilitating GI symptoms requiring prolonged hospital admissions [2, 3]. Gastroparesis is the commonest GI dysautonomia, but constipation, diarrhea, and more rarely megacolon or colonic pseudo-obstruction, stercoral ulcer or viscera perforation may occur [3]. The chronic nonadherence to

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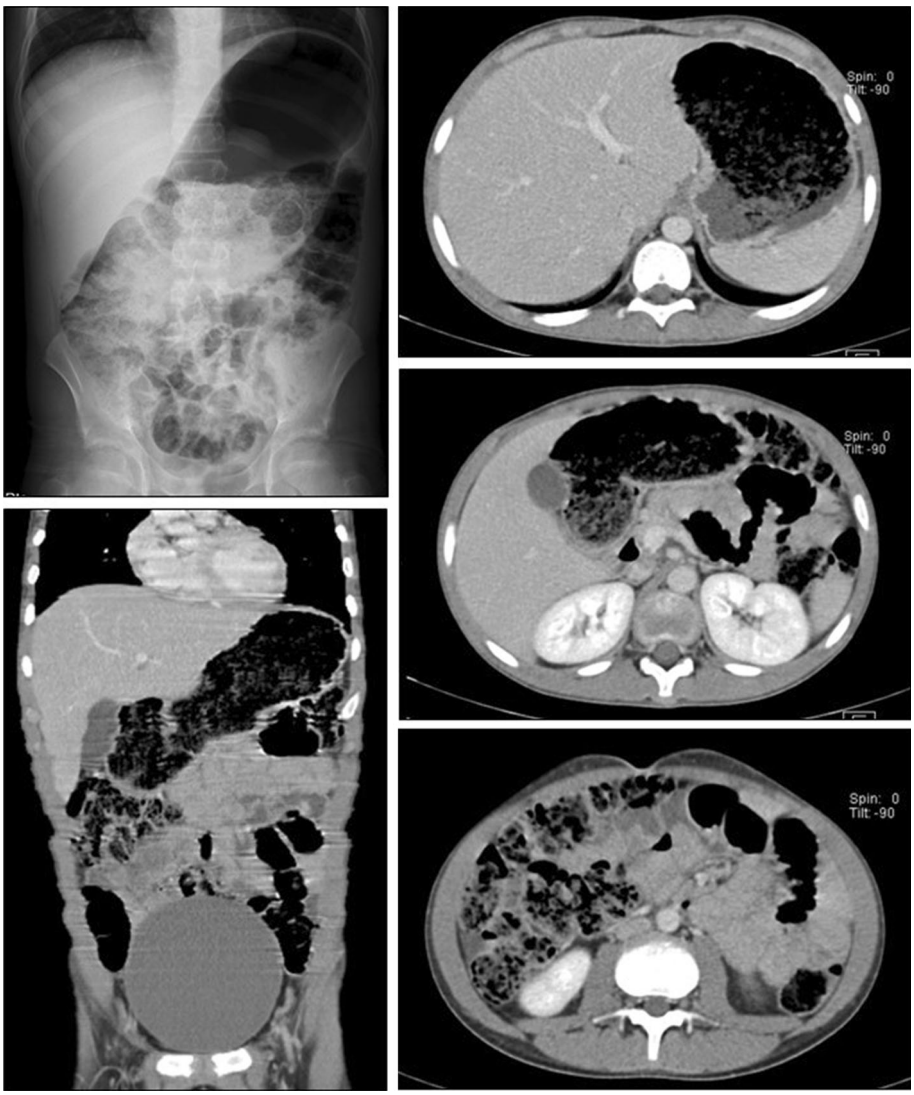

Fig. 1 Abdominal X-ray and computed tomography scan images of our patient showing exuberant gastric and bowel distention with stasis

insulin therapy, with consequent poor glycemic control, predisposed our patient to this severe GI dysautonomia.

The management of severe GI dysautonomia may involve support therapy (fluids, parenteral nutrition), symptomatic therapy (analgesics, antiemetics, prokinetics, erythromycin, proton pump inhibitors, laxatives), but the mainstay approach consists in the restoration of euglycemia. It has been reported that continuous subcutaneous insulin infusion in diabetic gastroparesis improves the glycemic control, reduces glycemic variability, and the frequency of hypoglycemia, as well as decreases the number of hospitalization days [3]. In our patient, the use of continuous intravenous insulin infusion allows us to obtain glycemic control, and together with prokinetics agents, reverted fastly the severe GI condition.

Conflict of interest The authors declare that they have no conflict of interest.

Informed consent Informed consent was obtained from the patient and his legal representant.

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