

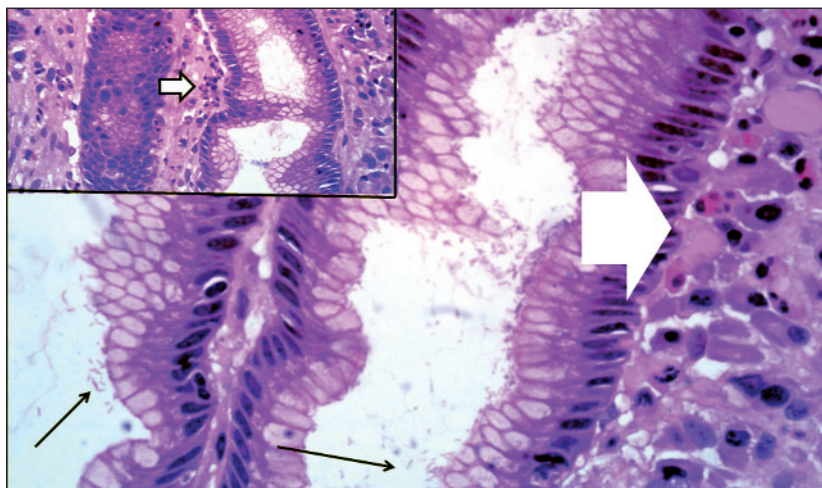


# Diabetes Vignette

## Recurrent diabetic ketoacidosis related to acute gastritis

A 16 year-old-male patient with type 1 diabetes mellitus (DM) presented with epigastric pain and recurrent vomiting for five days. He was regular on insulin and his blood glucose was reasonably controlled with a recent HbA<sub>1c</sub> level of 7.3% (56mmol/mol). Clinically, he was fully conscious, dehydrated and afebrile. He was neither pale nor jaundiced, had a normal chest and heart examination, and got mild epigastric tenderness. Random blood glucose was 22mmol/L. Investigations showed room air arterial blood gases: PaO<sub>2</sub> 8.5KPa, PaCO<sub>2</sub> 3.8KPa, bicarbonate 14mmol/L, and pH 7.26; urine acetone 3+; electrolytes: sodium 136mmol/L, potassium 4.9mmol/L, chloride 106mmol/L, and bicarbonate 14mmol/L; and anion gap 16mmol/L. Renal and hepatic profiles as well as serum amylase were normal. Abdominal ultrasonography was also normal.

A diagnosis of diabetic ketoacidosis (DKA) was made, but the precipitating factor was initially unclear. A thorough search for any evidence of infection, poor compliance or any form of stress was negative. The patient responded well to IV fluids and insulin infusion with reversal of ketoacidosis, dehydration, and hyperglycaemia. Three days after discharge, he was re-admitted, again with DKA. Plasma cortisol, 24-hour urinary catecholamines, plasma insulin like growth factor1, and plasma thyroid hormones and thyroid stimulating hormone were all normal. Treatment with IV ranitidine (50mg every 6 hours) and metoclopramide (10mg every 8 hours) was given along with measures to treat DKA. In spite of ranitidine therapy and the full reversal of DKA, he continued to have epigastric pain with vomiting. While arranging for an upper gastrointestinal (GI) endoscopy, and despite being on regular insulin scale in the ward, he developed an astonishing third episode of DKA. Endoscopy showed severe pangastritis with positive antral biopsy for *Helicobacter pylori* (Figure 1). *H. pylori* eradication treatment was given in the form of omeprazole (20mg twice daily), amoxicillin (1g twice daily), and clarithromycin (500mg twice daily) for 14 days. One week after starting eradication therapy, the patient's epigastric pain and vomiting totally resolved. He



**Figure 1.** Gastric mucosal biopsy with high and medium (inset) power fields showing inflammatory cell infiltration (white arrows) and *H. pylori* organisms (black arrows)

was discharged on his previous doses of insulin and was followed for a period of six months without any attacks of DKA.

Recurrent DKA may be defined as three or more episodes occurring within a period of four years.<sup>1</sup> Recurrence of DKA is found in patients with brittle DM, and much less commonly in patients with untreated bacterial infections, especially chronic cryptic infections (such as sinusitis, osteomyelitis, perinephric abscess, or lung abscess).

The evaluation of GI symptoms in patients with DKA is not infrequently a challenging issue. Although the presentation with epigastric pain and vomiting can be attributed to the DKA, abdominal symptoms may be a manifestation of a serious intra-abdominal process.<sup>2</sup> Clinically, if there is no localised tenderness, rebound tenderness or rigidity, then the likelihood of a localised intra-abdominal process is diminished.<sup>2</sup> Abdominal pain and vomiting which occur secondary to DKA alone resolve with improvement in the metabolic picture.<sup>2</sup> Barrett *et al.* suggested that, when GI symptoms persist beyond the first 12–24 hours after beginning treatment of DKA, additional aetiologies should be actively sought.<sup>2</sup> In a study by Umpierrez *et al.*, a strong association was observed between abdominal pain and metabolic acidosis.<sup>3</sup> Patients without abdominal pain had serum bicarbonate  $15 \pm 1$  and pH  $7.24 \pm 0.09$ , while patients with abdominal

pain had serum bicarbonate  $9 \pm 1$  and pH  $7.12 \pm 0.02$ .<sup>3</sup>

In our patient, the presence of abdominal pain and the severity of vomiting were unlikely to be solely explained by the associated degree of metabolic acidosis. Also, the poor response of the GI complaints to correction of ketoacidosis raised the suspicion of an associated intra-abdominal process. Acute *H. pylori*-related gastritis was the aetiological factor underlying three successive episodes of DKA, in spite of proper fluid and insulin therapy. Such aetiology could be missed, as abdominal symptoms may be attributed to DKA. An upper GI endoscopy may be included in the list of investigations to evaluate abdominal pain with DKA. In contrast to the high association with chronic gastritis, *H. pylori* infection is less frequently reported with acute gastritis.<sup>4</sup> Treatment of that form of acute gastritis with antisecretory therapy alone may not be effective as eradication therapy is required to control symptoms and prevent complications (e.g. recurrent DKA).<sup>5</sup>

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### References

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