Gastric mucosal calcinosis

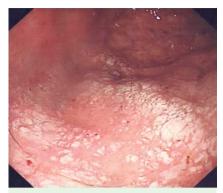


Fig. 1 Endoscopic appearance in a 68-year-old woman with end-stage kidney disease showing varying numbers of 1 – 3-mm, white, flat lesions in the gastric body.

A 68-year-old woman with end-stage kidney disease due to hypertension presented with nausea, vomiting, dyspepsia, and epigastric pain. Endoscopy revealed varying numbers of 1 – 3-mm diameter, white, flat plaques in the gastric body (**• Fig. 1**). Laboratory results included the following (normal ranges are given in brackets): blood urea 86.2 mg/dL (6.0 – 21.0 mg/dL); creatinine 4.2 mg/dL (0.6 – 1.1 mg/dL); calcium 9.2 mg/dL (8.9 – 10.1 mg/dL), corrected calcium 10.08 mg/dL; phosphorus 8.7 mg/dL (2.5 – 4.5 mg/dL); and albumin 2.9 g/dL (3.5 – 5.0 mg/dL). Urinalysis showed proteinuria (+++). The calcium–phosphate product was 87.6 mmol/L (normal < 60) and plasma intact parathyroid hormone level was 121 pg/mL (9.5 – 75 pg/mL).

Histologic examination of gastric biopsies showed widespread deposits of subepithelial microcalcification in the superficial gastric mucosa. The deposits of microcalcification were irregular, amorphous and extended into the lamina propria. Calcium was colored as black pigment in the von Kossa stain (• Fig. 2). Stains for cytomegalovirus, herpes, and iron were negative. *Helicobacter pylori* was also identified in the biopsy material.

Calcific deposits in the gastric mucosa are only rarely found in routine biopsies: metastatic calcification is the most common type of gastric mucosal calcinosis, with other underlying diseases being uremia and chronic renal disease. Hypercalcemia,

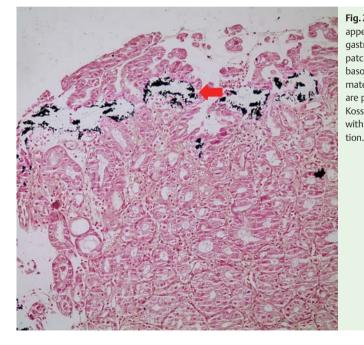


Fig. 2 Histologic appearance of the gastric biopsy showing patches of irregular, basophilic, amorphous material (arrow) that are positive with a von Kossa stain, consistent with calcium deposition. hyperphosphatemia, and an elevated calcium – phosphate product have been suggested as being the most important factors in the development of metastatic calcification [1-3]. *H. pylori* was positive in the present case; however, it is not significantly associated with gastric calcinosis. Although several drugs have been suspected of having a role in the etiology [4], there was no history of administration of alendronate, oral iron, aluminum-containing antacids, or sucralfate in our case.

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Competing interests: None

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