



Case Report

Gastric Mucosal Calcinosis

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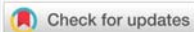
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Abstract

Gastric mucosal calcinosis is a very rare pathology of the gastric mucosa. It may develop secondary to several diseases but may also be idiopathic in some cases. In this case, gastric mucosal calcinosis was diagnosed with endoscopic biopsy performed for a patient who presented to our clinic with heartburn and abdominal discomfort. This case involves a very rare gastric pathology, and is being studied here with reference to literature data.

Case Report

The endoscopy performed for the 43-years-old male patient who presented to our clinic with dyspeptic and epigastric pain demonstrated Mallory-Weiss syndrome and chronic gastritis. No white, flat plaques were seen with endoscopy. The result of the biopsy with haematoxylin and eosin (H&E) staining performed on the material taken during endoscopy was reported as chronic gastritis and gastric mucosal calcinosis characterized by chronic infiltrate. Helicobacter pylori was negative and no atrophy, intestinal metaplasia or dysplasia was detected. Staining with Von Cossa clearly demonstrated gastric mucosal calcinosis with large, dense, irregular C accumulation [Figure 1A, 1B, 2A, 2B]. Pathology department's opinion was that the mucosal calcinosis observed in the corpus mucosa could be associated with calcium dysregulation and

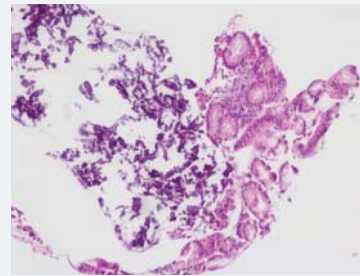


Figure 1A: Amorphous basophilic calcium deposits in gastric mucosa (A, HE, x100) verified with Voncossa stain (B, Von Cossa, x200).



Figure 1B: Von Cossa x 20 Amorphous basophilic calcium deposits in gastric mucosa (A, HE, x100) verified with Voncossa stain (B, Von Cossa, x200).

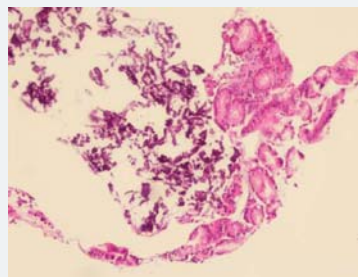


Figure 2A: Amorphous basophilic calcium deposits in gastric mucosa (A, HE, x100) verified with Voncossa stain (B, Von Cossa, x200). H&Ex10.

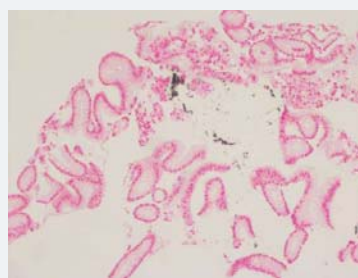


Figure 2B: Amorphous basophilic calcium deposits in gastric mucosa (A, HE, x100) verified with Voncossa stain (B, Von Cossa, x200).

could also be secondary to medication, and recommended analyzing correlation with clinical findings. Patient's physical examination and routine hematology yielded results that were within normal ranges. USG revealed hepatosteatosi. Abdominal computed tomography (CT) also yielded normal results. Serum PTH was 31.96 pg/ml [N: 15-65 pg/ml], serum calcium was 10.69 mg/dl [N: 8.6-10 mg/dl] and serum phosphorus was 4.88 mg/dl [N: 2.6-4.5 mg/dl]. The opinion of endocrinology was therefore sought. Endocrinology department doctor's said that there is no hyperparathyroidism. Therefore, hyperparathyroidism was not reported. It was considered to be due to the antacids the patient was taking. The patient was given empiric 40 mg pantoprazole to be taken in the morning before breakfast on empty stomach and 20 ng famotidine to be taken at bedtime. When the patient returned for follow-up, he was free of gastric complaints. Gastric mucosal calcinosis is a very rare pathologic diagnosis and we aimed to present this case with reference to the literature data.

Discussion

Generally, gastric mucosal calcinosis (GMC) is only rarely encountered in routine biopsies. They have been associated with a wide spectrum of clinical conditions. The underlying mechanism by which a given cell or group of cells accumulate calcium is unclear. It has been suggested that calcium enters the cell by passive diffusion attributable to the concentration gradient across the gastric cell membrane, and that accumulation occurs when the ability to extrude these salts is exceeded. Gastric mucosal calcinosis (GMC) may be secondary to hypercalcemia, hyperphosphatemia, atrophic gastritis, hypervitaminosis A, chronic kidney disease, tumor lysis syndrome, organ transplantation, gastric neoplasia, or may be associated with use of citrate-containing blood products, aluminum-containing antacids, sucralfate or bismuth [1-6]. GMC may rarely cause symptoms such as epigastric pain or dyspepsia [7,8]. The clinical relevance of GMC remains to be seen. In theory, however, accelerated bone demineralization via loss of phosphates and absorption of aluminum in the gastrointestinal tract may be a consequence of long-term aluminum-containing antacid or sucralfate therapy. Furthermore, some examples of systemic calcification are reversible with normalization of biochemical parameters, which highlights the need for pathologists to highlight this

finding when encountered in a premortem gastric biopsy. Our patient had no history of renal failure, organ transplantation, gastric malignancy or drug use.

Gastric mucosal calcinosis is categorized by some investigators into three subtypes as metastatic, dystrophic and idiopathic. Metastatic calcification is defined as the deposition of calcium salt in normal tissue due to an abnormal serum biochemical environment such as hypercalcaemia or hyperphosphataemia [1,2,7]. Metastatic calcification is the most common subtype and is seen in the presence of hypercalcemia, hyperphosphatemia or high levels of CaPO_4 products [9,15]. In cases with systemic calcification, gastric calcinosis improves when metabolic parameters improve. Dystrophic calcification develops in inflammatory, fibrotic and other pathological conditions. Metastatic calcification develops also in the kidneys, heart and liver. Our patient had no cancer, euthyroid lysis syndrome, antacid use or any of the other known risk factors. Our case was a very rare one and no etiology could be found for gastric mucosal calcinosis. The patient was recommended to return for endoscopic and clinic follow-up in 6 to 12-month intervals.

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