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What is behind Calcium Deposition over Gastric Mucosa?

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Abstract

Calcinosis cutis can be categorized into dystrophic, metastatic, idiopathic, and iatrogenic. Dystrophic calcification is the most common subtype and results from local tissue injury. When calcinosis cutis is noted over gastric mucosa, it is called gastric mucosal calcinosis (GMC). GMC has only three subtypes, including dystrophic, metastatic, and idiopathic. Metastatic subtype is the most common one. It is associated with abnormal calcium and phosphate metabolism. The incidence of GMC varies with the population being studied. GMC is usually demonstrated as white flat plaques or nodules by esophagogastroduodenoscopy. Hyperphosphatemia can lead to high mortality and cardiovascular event. Thus, when calcium deposition was found over gastric mucosa, we should look for possible etiologies and check laboratory data including calcium and phosphate.

Key Words: Gastric mucosal calcinosis, calcinosis cutis, metastatic calcinosis, chronic kidney disease, hemodialysis, hyperphosphatemia

Abbreviation: EGD: esophagogastroduodenoscopy, GMC: gastric mucosal calcinosis, CV: cardiovascular

Introduction

Calcium deposition over gastric mucosa, also known as gastric mucosal calcinosis (GMC), is a rare condition and mostly found incidentally by esophagogastroduodenoscopy (EGD). Although only a few cases have been reported, GMC has been recognized for over 70 years[1]. It is usually associated with abnormal calcium and phosphate metabolism. Thus, we presented a typical case of GMC.

Case report

A 52-year-old male had a medical history of hypertension, hyperlipidemia, coronary artery disease, gastroesophageal reflux disease, and end-

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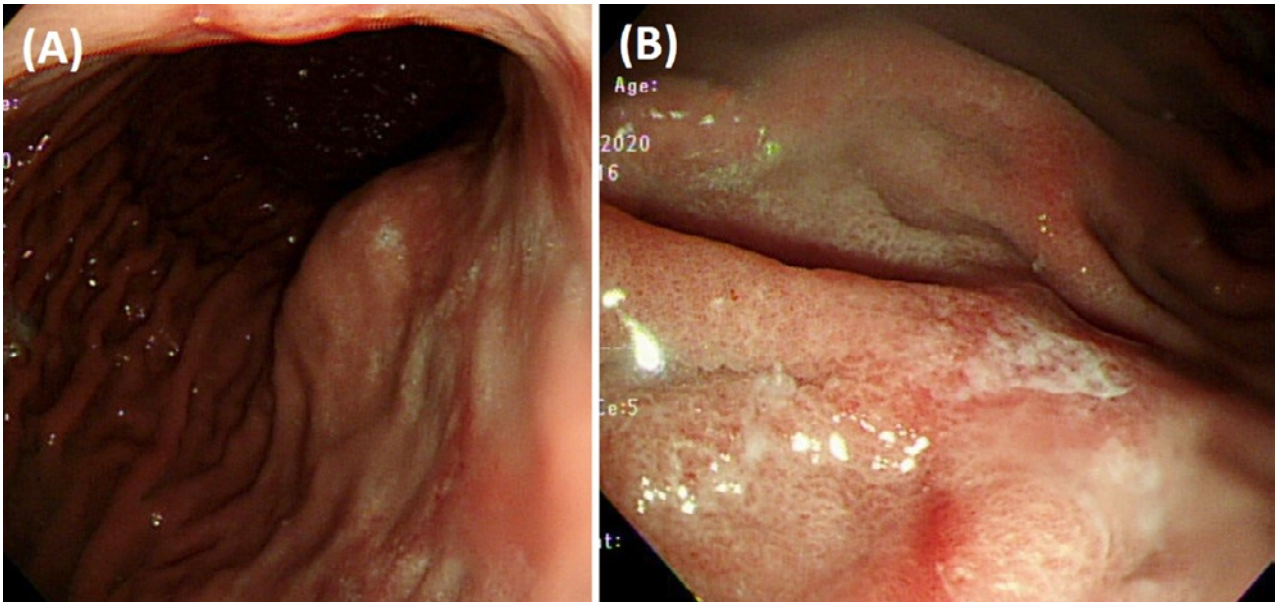

Figure 1. (A) (B) A few whitish plaques over greater curvature (GC) side and posterior wall (PW) of gastric body on esophagogastroduodenoscopy.

stage kidney disease under regular hemodialysis. He visited the outpatient department due to dyspepsia. Physical examination was unremarkable. EGD showed a few whitish plaques over greater curvature side and posterior wall of gastric body (Figure 1A, 1B). The histology showed calcium deposits in lamina propria with mild acute and chronic inflammation (Figure 2). There was no helicobacter pylori or malignancy.

He has suffered from hypocalcemia and hyperphosphatemia (serum calcium 5.7 mg/dL, inorganic phosphorus >11 mg/dL) for 2 years before hemodialysis. After hemodialysis, hypocalcemia subsided but hyperphosphatemia persisted. The laboratory data before the endoscopic biopsy was serum calcium 9.1 mg/dL, inorganic phosphorus 5.5 mg/dL, intact-parathyroid hormone 757 pg/mL, and alkaline phosphatase 58 U/L.

Discussion

The deposition of insoluble calcium salt in the cutaneous or subcutaneous tissue is called calcinosis cutis. Based on the etiology, calcinosis cutis can be divided into four subtypes: dystrophic, metastatic, idiopathic, and iatrogenic[2]. Dystrophic calcification, the most common subtype, results from local tissue injury. The calcium deposited in fibrotic or inflamed tissue[3,4]. Calcium and phosphate metabolism is normal. It often develops in patients with autoimmune connective tissue disease, such as systemic sclerosis and dermatomyositis. It can also occur in patients with panniculitis, porphyria cutanea tarda, cutaneous neoplasms, infections, or trauma[2]. Metastatic calcification is caused by abnormal calcium and/or phosphate metabolism. There is usually an underlying disease that cause metabolic disorder. The most common cause is chronic kidney disease. Hypervitaminosis D, milk-alkali syndrome, neoplasms, and sarcoidosis may also be the cause. The calcification can be seen over blood vessels, kidneys, lungs, and gastric mucosa[2], where is relative intracellular alkalinity[3]. Idiopathic calcification occurs without local tissue injury or abnormal calcium and/or phosphate metabolism. Idiopathic calcification of the scrotum, subepidermal calcified nodule, and tumoral calcinosis could be categorized in this subtype[2]. Finally, iatrogenic calcification happens after extravasation of intra-

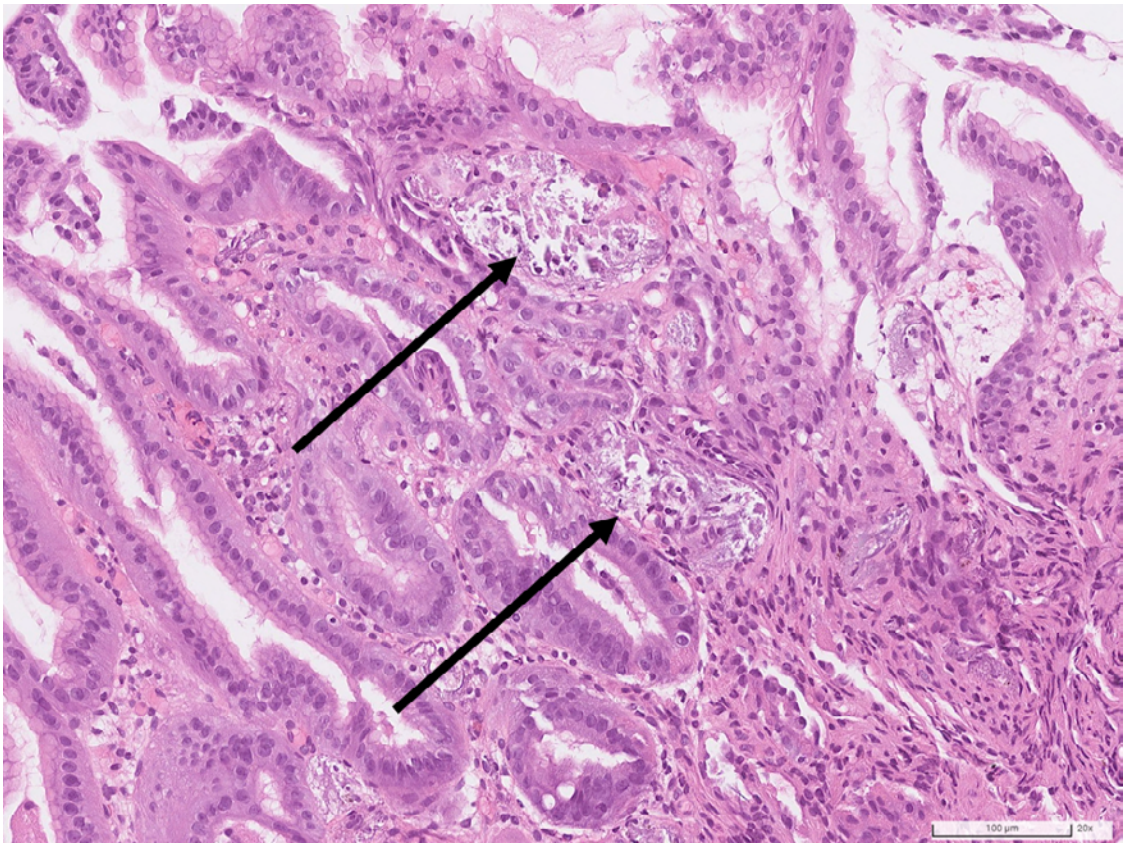

Figure 2. Calcium deposits in lamina propria of gastric mucosa. (Hematoxylin and eosin stain, 20X)

venous calcium chloride or calcium gluconate. The mechanism is thought to be both tissue damage and metabolic disorder.

On the other hand, GMC can be categorized into metastatic, dystrophic, and idiopathic. Metastatic calcinosis is the most common subtype, account for 70% of GMC[3]. Calcium deposition is found in normal tissue with abnormal serum biochemistry (hypercalcemia, hyperphosphatemia, and an elevated Ca X PO4 product). Due to the silent clinical course, the incidence of GMC is based on the population being studied, ranging from 13% in patients with chronic kidney disease[1], 14.6% in renal transplant patients[5], to 60% in chronic dialysis patients[6]. GMC is usually found incidentally.

EGD usually shows 1-5 mm of white flat plaques or nodules[3], which are finally diagnosed as GMC. It can also be erythema[4], patch-like mucosal changes in watermelon pattern[7], or linear whitish plaques[8].

Currently, there is no study available regarding the prognosis of GMC. Since GMC is often found incidentally by gastric biopsy, most of the articles are case reports. The prognosis of GMC remains uncertain. Hyperphosphatemia has been associated with higher mortality rate and cardiovascular (CV) events in many studies[9,10]. One study has presented the connection between soft tissue calcification and cause of death in dialysis patients. 3 of 23 (13%) patients without cardiac calcification and 9 of 33 (27%) patients with cardiac calcification were died from cardiac disease[6]. Pulmonary calcification can also be life-threatening[4]. Hyperphosphatemia, cardiac calcification, and pulmonary calcification could lead to poor prognosis.

Considering the medical history of hemodi-

alysis and long-term abnormal mineral metabolism, our case is most likely metastatic type GMC. Chronic kidney disease-mineral and bone disorder plays an important role on calcium deposition. Several cardiovascular examinations were reviewed. Cardiac catheterization showed coronary artery disease with triple vessel disease. Percutaneous transluminal coronary angioplasty and stents placement were performed. Chest computed tomography for lung nodule study revealed calcification of coronary arteries, aorta, and bilateral renal arteries. There was no evidence of calcification of pulmonary arteries. Echocardiogram showed no valvular calcification.

Calcimimetics with cinacalcet and vitamin D analogs with calcitriol were prescribed for lowering parathyroid hormone. Phosphate binders was used for hyperphosphatemia. During the last follow-up, the laboratory data was serum calcium 9.5 mg/dL, inorganic phosphorus 5 mg/dL, intact-parathyroid hormone 584 pg/mL, and alkaline phosphatase 54 U/L. There was no CV event occurred during the 2.5-year follow-up.

Conclusion

When calcium deposition is found over gastric mucosa, we should search for the possible etiology. History including underlying disease may be reviewed, and lab data including calcium and phosphorus should be checked. Since hyperphosphatemia is associated with higher mortality rate and CV events, electrolytes imbalance should be corrected as possible.

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Conflict of Interest

The authors declare no conflict of interest.

References

1. MULLIGAN RM. Metastatic calcification. Arch Pathol (Chic) 1947;43(2):177-230.
2. Walsh JS, Fairley JA. Calcifying disorders of the skin. J Am Acad Dermatol 1995;33(5 Pt 1):693-706; quiz 707-10.
3. Gorospe M, Fadare O. Gastric mucosal calcinosis: clinicopathologic considerations. Adv Anat Pathol 2007;14(3):224-8.
4. Kosuru V, Mohammed A, Kapoor R, et al. Metastatic Calcinosis of Gastric Mucosa. J Investig Med High Impact Case Rep 2020;8:2324709620940482.
5. Stroehlein KB, Stroehlein JR, Kahan BD, Gruber SA. Gastric mucosal calcinosis in renal transplant patients. Transplant Proc 1999;31(5):2124-6.
6. Kuzela DC, Huffer WE, Conger JD, Winter SD, Hammond WS. Soft tissue calcification in chronic dialysis patients. Am J Pathol 1977;86(2):403-24.
7. Roh J, Noh CK. Unusual stomach and duodenum mucosal changes in a kidney transplant patient after long-term peritoneal dialysis. Gut 2022;71(7):1276-425.
8. Wiggins, T. and A. Thomas, Gastric Mucosal Calcinosis in a Patient with Abdominal Pain. The American College of Gastroenterology, 2010;105:S176-7.
9. Block GA, Klassen PS, Lazarus JM, Ofsthun N, Lowrie EG, Chertow GM. Mineral metabolism, mortality, and morbidity in maintenance hemodialysis. J Am Soc Nephrol 2004;15(8):2208-18.
10. McGovern AP, de Lusignan S, van Vlymen J, et al. Serum phosphate as a risk factor for cardiovascular events in people with and without chronic kidney disease: a large community based cohort study. PLoS One 2013;8(9):e74996.

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摘要

皮膚鈣化 (Calcinosis cutis) 可以分成失養性 (dystrophic)、代謝性 (metastatic)、不明性及醫源性。失養性最常見，由局部組織損傷所致。當鈣化發生在胃黏膜時，則稱為胃黏膜鈣質沉積症 (Gastric mucosal calcinosis, GMC)，其包含 3 種亞型：失養性、代謝性和不明性，其中代謝性最常見，和鈣磷代謝異常有關。GMC 發生率因研究族群而異，在胃鏡下通常呈現白色平坦斑塊或結節。高血磷可能導致高死亡率及心血管事件，因此當發現胃黏膜有鈣沉積時，應盡可能找出病因，並抽血確認鈣磷之數值以加以矯正。