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Gastric wall calcification in gastric cancer relapse: Case report

Nüks mide kanserinde mide duvar kalsifikasyonu: Vaka sunumu

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We present the case of a 53-year-old male with subtotal gastrectomy and gastrojejunostomy due to gastric cancer who later developed cancer relapse and diffuse plaque-like calcification in the residual gastric tissue. As far as we know, this is the first case in the English literature in whom gastric tumor calcification developed one year after gastric cancer operation. We also discuss possible mechanisms of gastric wall calcification in such cases.

Key words: Gastric cancer, calcification

Mide adenokanseri nedeniyle subtotal gastrektomi ve gastrojejunostomi yapıldıktan bir sene sonra nüks eden ve midede diffuz, plak tarzında kalsifikasyon gelişen 53 yaşındaki erkek hastayı sunduk. Bu vaka literatürde mide kanseri operasyonundan bir yıl sonra midede tümör kalsifikasyonu tanımlanan ilk vakadır. Bu vaka sebebiyle mide duvar kalsifikasyonu nedenlerini tartıştık.

Anahtar kelimeler: Mide kanseri, kalsifikasyon

INTRODUCTION

Unlike in thyroid, breast and ovarian neoplasms, the digestive tract cancers are rare examples of tumors that show calcification (1-3). Gastrointestinal leiomyomas, angiomas, carcinoid tumors and more rarely mucinoid adenocarcinomas of the stomach and colon are distinctive examples of tumors that may undergo calcification (1, 4, 5).

Gastric carcinomas show some characteristic features in respect to tumoral calcification. Calcifications within primary gastric cancer are a rare finding. Most of the cases described are mucinous adenocarcinomas. These tumors rarely calcify in their mucinous pools in advanced stages of tumoral progression (6-8). The patients with calcified gastric cancer are relatively young in age and probably have a better survival than those gastric cancer cases without calcification (7).

In this report, we present a gastric cancer case who interestingly developed plaque-like gastric wall calcification extending into relapsed gastric tumor after a noncurative resection operation, and we discuss the case with the literature regarding the genesis of calcification.

CASE REPORT

A 53-year-old male admitted to our hospital in June 2001 because of epigastric pain, anorexia, nausea and vomiting lasting for three months. He had lost nearly 10 kg during this period. His previous medical history was uneventful. His basic physical examination and routine blood tests were completely normal. Nevertheless, an upper gastrointestinal endoscopy revealed a large polypoid tumor located at the distal stomach. Endoscopic biopsies revealed an adenocarcinoma. Computerized tomography (CT) of the whole abdomen showed diffuse and irregular thickening of gastric antrum wall without any sign of metastasis. He underwent distal gastrectomy, gastrojejunostomy and regional lymph node dissection. Surgical pathologic examination revealed a moderately well differentiated adenocarcinoma tissue infiltrating gastric serosa and three regional lymph nodes. Tumor tissue contained signet ring cells and small extracellular mucinous islets. There was no sign of calcification on histopathologic examination. The resection borders were free of tumor infiltration. Thereafter, the patient refused any type of adju-

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vant medical treatment and was therefore discharged from the hospital. One year later, he readmitted with jaundice. An upper endoscopy revealed mass lesion at the gastroenterostomy anostomosis line, and endoscopic biopsies documented reappearance of adenocarcinoma. The abdominal tomography clearly documented relapsed gastric tumor mass located at the gastroenterostomy site. There were conglomerated lymph nodes at the portal hilus and possibly due to this, the intrahepatic bile ducts were found to be dilated. CT also documented plaque-like calcification in the gastric wall (Figure 1). His serum calcium and phosphorus levels were within the normal range. There was no sign of calcification on abdominal X-ray. He was inoperable at this stage and a percutaneous approach was used to insert an expandable biliary wall stent. After the procedure, his jaundice resolved completely, but the patient died six months later.

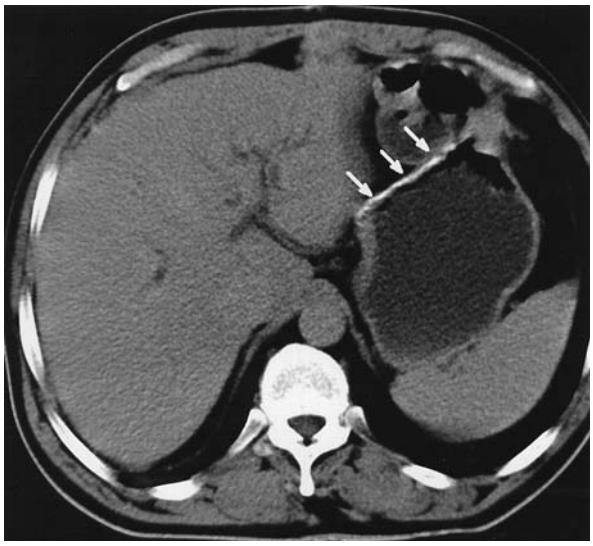

Figure 1. Plaque-like calcification in the gastric wall is demonstrated by white arrows

DISCUSSION

Gastric tumor calcification is a rare event. In the literature, the present knowledge on this topic comes primarily from reports on mucinous adenocarcinoma cases. These cases were relatively young patients who interestingly showed a good surveillance (7). Our case was the first in the literature who developed diffuse plaque-like calcification in the remnant gastric wall one year after subtotal gastrectomy and gastroenterostomy operation.

There are few special pathophysiologic mechanisms suggested in the development of gastric cancer calcification (1-3, 6, 7, 9). As it is generally known, dystrophic calcification occurs in ischemic and necrotic tissue samples. Denatured proteins bind specially to phosphate ions and thereafter react with calcium ions to form calcium phosphate precipitates. A relatively alkaline environment helps this precipitation to occur easily. In this situation, the plasma calcium level is usually within the normal range. On the contrary, metastatic calcification occurs due to precipitation of calcium ions in the presence of high serum calcium levels. Finally, mucin-forming tissues are prone to develop calcification. Mucinous adenocarcinomas are likely to develop calcification due to the presence of mucinous material and a relatively alkaline environment (10). The acid pH is a relatively soluble environment for calcium phosphate and calcium carbonate salts. However, calcium salts undergo precipitation at alkaline pH.

Gastric wall calcification in the present case apparently developed during the postoperative period. Moreover, as seen from the CT films, gastric wall calcification extended into the tumoral tissue. Though elucidation of the tumoral calcification in the present case is difficult, we can suggest some mechanisms as possible explanations. First, tumor histopathology had shown the presence of mucinous material, which can trap calcium ions. Second, postoperative circulatory changes in the gastric wall containing neoplastic tissue may potentiate dystrophic calcification. As seen from Figure 1, presence of calcification only at the tumoral site of the gastric wall supports this idea. Additionally, shift of the pH of the stomach lumen towards the alkaline site might play a role in the pathogenesis. In other words, the mutual effects of these proposed mechanisms may act together to result in gastric calcification.

We believe that each gastric tumor presents with interesting minor differences in each patient. To collect and understand these minor details may help us to cover the entire nature of tumor behavior.

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