Xanthogranulomatous gastritis: Association with xanthogranulomatous cholecystitis

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Abstract

A case of xanthogranulomatous inflammation of the gastric wall is reported. The lesion was associated with adherent xanthogranulomatous cholecystitis and simulated, clinically, a malignant neoplasm. Histologically, foamy histiocytes, multinucleated giant cells, other inflammatory cells, fibrous reaction with spindle cells and cholesterol clefts were found.

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Xanthogranulomatous inflammation is characterised by varying amounts of foamy histiocytes, inflammatory cells, fibrous reaction and sometimes multinucleated giant cells. Typically, the process causes destruction and effacement of the normal structures of the affected organ and may simulate a neoplasm.¹

We report an unusual xanthogranulomatous process of the gastric wall in a patient with xanthogranulomatous cholecystitis. To the best of our knowledge a similar case has not been reported before.

Case report

A 79 year old man presented with a two month history of gastric discomfort, anorexia, and

weight loss. Physical examination showed an ill defined mass in the region of the right hypochondrium. Laboratory values were as follows: red blood cells $4.46 \times 10^{12}/l$; haemoglobin concentration 129 g/l; haematocrit 39.9%; platelet count of $243 \times 10^9/l$. White cell count ($5.5 \times 10^9/l$) was within normal limits. The erythrocyte sedimentation rate was 26/first hour. Urine had a pH of 5; specific gravity was 1018; there was nothing in the sediment. Prothrombin was 100%, with prothrombin time of 28. Glucose concentration was 6.1 mmol/l. Urea excretion was 68; creatinine concentration was $93 \mu\text{mol/l}$; albumin 57%; α_1 3%, α_2 9%, β 11%, γ 20%. An x ray picture showed a luminal stenosis in the antropylic region of the stomach, suspicious of a malignant neoplasm. At surgery the gall bladder was found to be adherent to the gastric antrum by means of firm tissue. Several gallstones were also palpable, so a partial gastrectomy using Billroth's I procedure, and a cholecystectomy were performed. The postoperative course was free of complications and the patient recovered well.

Results

The gastrectomy specimen consisted of a 12×10 cm segment of stomach with irregular mucosal and serosal surfaces. On sectioning of the antropylic region, a firm, yellowish-white

Figure 1 Gross sections of the antropylic region showing wall thickening with ill defined nodularity.

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Figure 2 The gastric wall has been largely replaced by a xanthogranulomatous infiltrate with cholesterol clefts (A), foam cells, lymphocytes, multinucleated giant cells (B) and spindle cells (C).

vaguely nodular area of wall thickening, measuring up to 1.7 cm was seen (fig 1). The gall bladder, received separately, measured 6 cm in length and contained several polyhedral calculi. The serosal surface was irregular and the wall was thickened and brownish. Histological sections from the gall bladder and antropylic region of stomach showed the same picture characterised by transmural granulomatous inflammation. Nodular collections of foamy histiocytes together with many lymphocytes, plasma cells, granulocytes, giant cells containing cholesterol clefts and spindle cells were seen (fig 2). The overlying gastric mucosa showed superficial erosions and small ulcers. Immunohistochemical reactivity for α -1-antichymotrypsin and lysozyme was present in xanthomatous cells, granulocytes, and giant cells. Mac 387 strongly stained granulocytes

and some giant cells. Plasma cells and lymphocytes were polyclonal for κ and λ light chains. The spindle cells were reactive with vimentin (V9), muscle specific actin (HHF-35), and, focally, with desmin (DER-11).

Discussion

The clinical and gross features of the gastric lesion in the case described here implicated malignancy. Nevertheless, microscopic examination disclosed a fully developed xanthogranulomatous inflammation with foamy histiocytes, multinucleated giant cells, and cholesterol clefts both in the gastric and gall bladder walls.

Because xanthogranulomatous cholecystitis is rare, it is unlikely that these findings are unrelated. Presence of cholesterol crystals in

the inflammatory infiltrate of the stomach could implicate a bile induced pathogenesis² for the gastric lesion too. Rupture of Rokitan-sky-Aschoff sinuses with subsequent extravasa-tion of bile and a histiocytic granulomatous reaction is the mechanism implicated in the development of xanthogranulomatous cholecystitis.¹ Adhesions to surrounding tissues have been found in other cases of xanthogranulomatous cholecystitis at surgery.² It is conceivable that deepening of the xantho-granulomatous process in the gall bladder and adhesions to the stomach may result in a transmural involvement of gastric wall, accounting for the findings observed in our case.

Bile reflux into the stomach damages gastric mucosa and there is evidence that the compo-sition of bile salts in the refluxed fluid is an important factor in the development of gastric damage.³ It is tempting, therefore, to speculate that a peculiar composition of bile may damage directly the mucosa of both the gall bladder and the stomach, resulting in similar lesions. Ulceration would allow refluxed bile to enter the subepithelial tissues, giving rise to the xanthogranulomatous process.¹ Indeed, bile reflux has been presumed to have a role in the pathogenesis of gastric xanthoma,⁴ a lesion that shares some similarities with xanthogranu-lomatous inflammation. However, evidence of bile reflux in the stomach was absent in our patient.

Apart from the xanthoma, which comprises collections of foamy cells,⁴ the differential diagnosis of this gastric lesion should include several granulomatous lesions.

A diagnosis of idiopathic granulomatous gastritis⁵ is made when non-caseating granu-lomas are seen and Crohn's disease and sarcoidosis have been excluded. Barium granu-loma, a rare complication of x ray examination, shows collections of macrophages and multi-nucleated giant cells filled with granular material.⁶ Hyalinoid giant cell gastritis is char-acterised by eosinophilic hyalinoid degenera-tion of smooth muscle which elicits a giant cell granulomatous reaction.⁷ However, in all the aforementioned conditions, a prominent foamy cell infiltrate, as seen in the present case, was not observed. Malacoplakia is charac-terised by accumulations of macrophages with

granular, acidophilic cytoplasm containing laminated structures called Michaelis-Gut-mann bodies. Although malacoplakia and xanthogranulomatous processes are morpho-logically and perhaps pathogenetically similar, accounting for the existence of transitional forms,⁸ Michaelis-Gutmann bodies are found only in malacoplakia. Inflammatory pseudotu-mour or plasma cell granuloma may be present as a mass in many sites, including the stomach, and shows a variable admixture of plasma cells, spindle cells of myofibroblastic type, and foamy histiocytes.⁹ Thus the histological pic-ture may be very suggestive of a xanthogranu-lomatous process. Spindle cells expressing vimentin, muscle-specific actin and desmin, consistent with myofibroblasts,⁹ were also found in our case. This was not surprising as myofibroblasts have been found in juvenile xanthogranuloma,¹⁰ a lesion closely related to a xanthogranulomatous process. In spite of these resemblances in our case prominent foreign body giant cells and cholesterol clefts were present and this is not a feature of plasma cell granuloma.

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