Russell Body Gastritis: Case Report and Review of the Literature

Pinar Karabagli[1], Huseyin Savas Gokturk[2]

1) Department of Pathology, Selcuk University, Selcuklu Medical Faculty, Konya; 2) Department of Gastroenterology, Baskent University, Faculty of Medicine, Konya, Turkey

Abstract

An unusual and rare gastric mucosal lesion histologically consisting of Russell bodies and plasma cell infiltration and termed as Russell body gastritis is presented. In the literature there are only ten such case reports and of these, seven including this present case were associated with Helicobacter pylori infection. The high rate of Helicobacter pylori infections in cases of Russell body gastritis suggests that the correlation is not merely coincidental. All data in the literature related to Russell body gastritis were scanned, and histopathologic findings and the clinical characteristics associated with such lesions were discussed.

Key words

Russell body – gastritis – Helicobacter pylori – plasma cells - histopathology.

Introduction

The Russell Body gastritis (RBG) is a recently recognized pseudotumoral lesion of the gastric mucosa with unknown etiology. It is characterized by the accumulation of numerous plasma cells containing Russell bodies (RBs) with the expression of kappa and lambda light chains [1-3]. It was labelled as RBG by Tazawa and Tsutsumi in 1998 [1]. There are only ten case reports in the English literature, and seven of them were associated with Helicobacter pylori (H. pylori) infection [1, 4-8].

In the present case, RBG was confirmed by clinical, morphological and immunohistochemical studies. Characteristic features and differential diagnosis of this disease are discussed in light of the pertinent literature.

Received: 27.11.2011 Accepted: 22.12.2011
J Gastrointestin Liver Dis
March 2012 Vol. 21 No 1, 97-100
Address for correspondence: Pinar Karabagli
Department of Pathology, Selcuk University, Selcuklu Medical Faculty, Selcuklu, Konya 42075, Turkey
Email: pinarkarabagli@yahoo.com

Case report

A 60-year-old man was admitted to the clinic complaining of dyspepsia. Physical examination and laboratory data exhibited no abnormalities. Upper gastrointestinal endoscopy revealed erythema in the gastric body and a large, irregular, fungating, ulcerated, nearly circumferential mass at the incisura angularis of the stomach. Consequently, multiple biopsies were obtained (Fig. 1).

Fig 1. Endoscopy: large, irregular and deep ulcerated mass lesion with central ulceration at the incisura angularis.

Histopathological examination of the gastric mucosa biopsies revealed mononuclear cellular infiltration with numerous spherical eosinophilic globules in the lamina propria and polymorphonuclear leukocyte infiltration of the glandular epithelium in the antrum and corpus mucosa. In the surface epithelium of antral mucosa, mild foveolar hyperplasia, focal intestinal metaplasia and mild dysplasia were observed (Fig. 2).

Under high power, the plasma cells were found as containing numerous large intracytoplasmic globules, homogeneous, mainly round to oval, and pushing the nucleus toward the periphery (Fig. 3). They were evidenced as RBs -containing plasma cells, all of which were intensely PAS-positive (Fig. 4). Thin wavy blue rods indicating H. pylori

presence were readily identified in the mucus overlying or attached to the foveolar epithelium on H&E and modified Giemsa stained slides.

İmmunohistochemically, RBs-containing cells were stained with CD45, but showed immunonegativity with AE1/AE3. Furthermore, immunohistochemical staining against immunoglobulin K and λ light chains showed a polyclonal pattern confirming the plasma cells to be non-neoplastic (Fig. 5A, B).

Helicobacter pylori eradication treatment was administered to the patient, and the patient was followed up because of dysplasia and a suspicion of malignancy. Upper gastrointestinal endoscopy was repeated after 3 months and 6 months. On post-eradication endoscopic controls, the macroscopic findings had resolved completely (Fig. 6). As a result of follow-up biopsies, both the density of RBs, and the active inflammation and focally mild dysplasia were detected as decreasing gradually and then disappearing completely. Helicobacter pylori was negative. However, mild mononuclear inflammatory cell infiltration was observed to be present at the final biopsy.

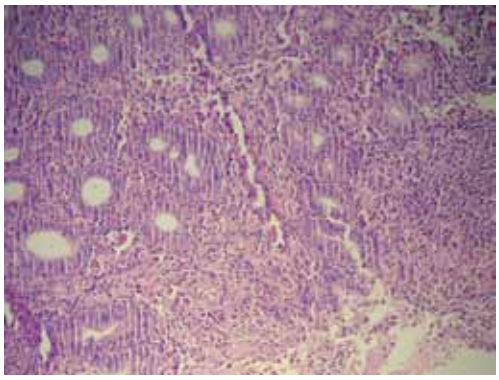

Fig 2. Gastric biopsy showing diffuse infiltration of lamina propria by numerous spherical eosinophilic globules (H&E, 200x).

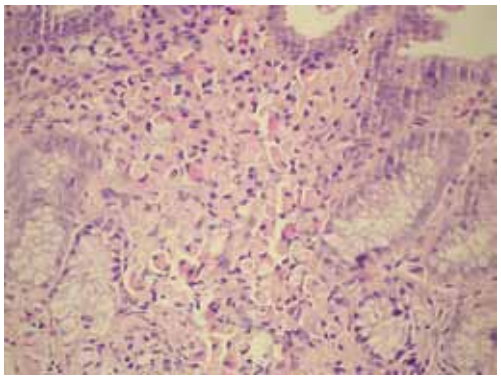

Fig 3. High-power view of large intracytoplasmic globules (H&E, 400x).

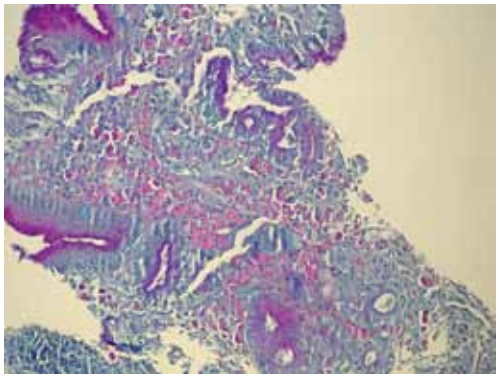

Fig 4. Strong PAS positivity of plasma cell inclusions (PAS, 200x).

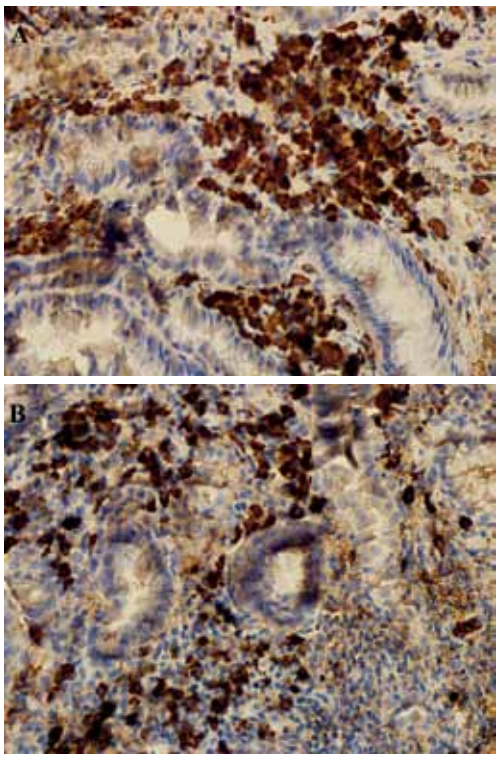

Fig 5. İnfiltrating cells showing cytoplasmic positivity with A: immunoglobulin kappa light chains (400x); B: immunoglobulin lambda light chains (400x).

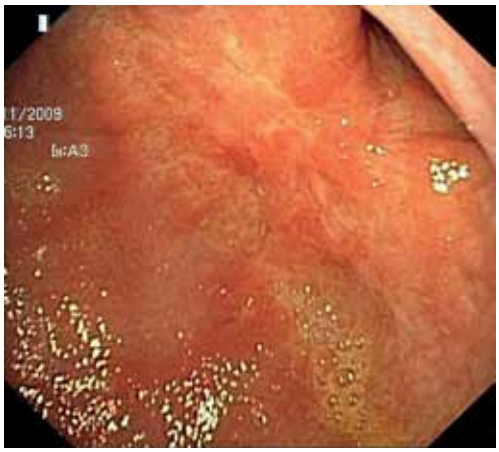

Fig 6. Endoscopic appearance after 3 months.

Discussion

Russel bodies are considered to be an accumulation of condensed immunoglobulin byproducts within the perinuclear

cistern of the smooth endoplasmic reticulum [7, 9, 10]. The neoplastic cells in plasmacytoma and in states of chronic hyperimmunization, including lymphoid malignancies, such as myeloma and B cell lymphomas occasionally contain RBs. However, plasma cells with RBs may also be seen in such disorders as Hashimoto's thyroiditis, rheumatoid arthritis and ulcerative colitis [2, 5, 6]. Empirically, a small number of RBs are occasionally identified in either chronic follicular gastritis or gastric MALT lymphoma. In these conditions, RBs are usually found dispersed in small numbers among a larger component of polymorphous lymphocytic infiltrate [5]. However, in 1998, Tazawa and Tsutsumi described a very peculiar, localized accumulation of plasma cells with RBs in the gastric mucosa named RBG [1]. The reason for the accumulation of RBs stil remains unclear.

Russell body gastritis is very rare and there are only ten case reports in the English literature. The 10 patients were 4 men and 6 women aged from 34 to 80 years (Table I).

Table I. Comparison of data of RBG in literature and present case

Case	Author (Reference)	Age/sex	H. pylori	History
1	Tazawa [1]	53/M	+	
2	Erbersdobler [2]	80/F	-	Candida esophagitis-alcohol and analgesic abuse
3	Ensari A [4]	70/M	+	
4	Drut [3]	34/M	-	HIV +
5	Paik S [5]	47/F	+	
6	Paik S [5]	53/F	+	
7	Wolkersdörfer [6]	54/F	+	monoclonal gammopathy of undetermined significance
8	Pizzolitto [7]	60/F	+	
9	Licci [8]	59/M	+	HIV +
10	Del Gobbo [10]	78/F	-	
11	Karabagli (present)	60/M	+	

Seven of the ten cases including the present case were associated with H.pylori infection [1, 4-8]. Helicobacter pylori is known to play a causative role in chronic gastritis, particularly in chronic follicular gastritis characterised by hyperplastic lymphoid follicles and to have an etiological role in MALT lymphoma. Eradication of H. pylori infections not only results in the decrease of these reactive lymphoid follicles and aggregates, but in the regression of early-stage MALT lymphoma, as well [6].

Russell body gastritis may develop in the setting of chronic gastritis associated with H. pylori infection. It is possible that the chronic infection with H. pylori may stimulate plasma-cell hyperactivation and consequently hyperproduction of immunoglobulins with numerous Russell body formations. The disappearance of Russell bodies in the follow-up biopsies after the eradication of H. pylori in our case and the report by Tazawa and Tsutsumi lends further support to the causative role of H. pylori in the development of RBG [1, 5]. However, the presence of RBs in H. pylori gastritis seems to be a very rare finding, thus a direct role played by this pathogen in the formation of RBs is highly unlikely [4].

In a study performed by Wolkersdörfer et al, a case of H. pylori-positive RBG was found to be associated with monoclonal gammopathy. They concluded that the genetic alterations in the Ig locus caused an excess of unsecreted immunoglobulin and lead to the formation of RBs [6].

In the literature, two cases of HIV-positive RBG were encountered, and one of them was also H. pylori-negative [3, 8]. One of the other two H. pylori-negative RBG reported in the literature was candida esophagitis with a history of alcoholic and analgesic abuse [2]. In light of the data, both patients with H. pylori-negative may be considered to be immunosuppressed. There was no particular history in the other H. pylori-negative case [10].

Unfortunately, the clinical significance of RBs in gastritis still remains unknown. The lesion can be confused with a neoplastic process. On low-power view, plasma cells with RBs may resemble signet ring cell carcinoma. Signet ring cell carcinoma usually has a dramatic endoscopic appearance and is often associated with linitis plastica of the stomach. Although the morphologic features can be subtle, carcinomatous cells may display features of malignancy, including destructive infiltrative growth, nuclear atypia and mitotic activity. Furthermore, the cytoplasm of signet ring-like carcinomatous cells is distended by optically clear and round vacuoles, whereas the RBs in plasma cells are seen in the form of homogeneous and round to ovoid eosinophilic inclusions. Definite exclusion of carcinoma can be obtained by immunohistochemistry, since signet ring cell carcinoma is positive for keratins, a reaction not expected for plasma cells [7]. Malignant lymphoma, especially mucosa-associated lymphatic tissue (MALT) lymphoma, is also a serious consideration, but the lack of lymphoepithelial lesion, nuclear atypia, or mitoses favors a reactive nature of the infiltrating cells. Plasmacytoma can occur in the stomach, yet the immunohistochemistry against immunoglobulin light chains demonstrates a polyclonal pattern [2, 4, 5].

Russell bodies represent a general response of the cell to the accumulation of abundant, non-degradable immunoglobulin. Considering the other cases reported in the literature, it seems as if H. pylori plays an active role in the etiology of the condition, either by promoting it or by modulating the extension of the disease locally. Chronic H. pylori infection could be the antigenic stimulus for the formation and accumulation of RBs in the gastric mucosa. Although RBG is by itself a benign condition, its long-term effect, such as its possible increased risk for the development of neoplasia, is unknown [5]. Clinically, it may be worthwhile to implement H. pylori eradication treatment in this peculiar disease entity.

Conclusion

The differential diagnosis of RBG is significant, since the associated microscopic views may be confused with a neoplastic disease, especially when lesions are extensive or the endoscopic features suggest a suspected neoplasm. The compilation of more cases, including endoscopic and histopathological controls, may help to elucidate the backround of this condition.

Acknowledgement

We thank Numan Duran for language editing.

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