

“Russell Body” Gastritis: A Case Report

“Russell Body” Gastriti: Olgu Sunumu

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ABSTRACT

Our case was a 50-year-old female who presented at the outpatients department with dyspeptic symptoms. Microscopical examination of mucosal samples from the corpus and antrum showed widespread *Helicobacter pylori* within the superficial mucus network, marked neutrophilic cryptitis, widespread reactive/regenerative crypt hyperplasia, intestinal metaplasia, and increased lymphoplasmocytoid cells and plasma cells full of immunoglobulin in the lamina propria. Immunohistochemical staining showed the plasma cells to be CD3 (-), CD20 (-), CD79a (+), CD45 (+), Kappa (+), and Lambda (+).

It is possible for a dense accumulation of Russell bodies to be observed on a background of *H. pylori* taking into account that Russell bodies contain immunoglobulin aggregates and side products of immunoglobulin synthesis. However, there are only a limited number of articles evaluating this aspect.

We present a case to contribute to the few articles on *Helicobacter gastritis* characterized by an inflammatory reaction rich in “Russell” bodies.

Key Words: Russell body, Gastritis, *Helicobacter pylori*, Mott cells

ÖZ

Olğumuz dispeptik yakınmaları nedeni ile kliniğe müracaat eden 50 yaşında kadın hastadır. Antrum ve korpusta ait mukozal örneklerinin mikroskopik incelemesinde yüzey mukus ağı içinde yaygın *Helicobacter pylori*, belirgin nötrofilik kriptit, yaygın reaktif/rejeneratif kript hiperplazisi, intestinal metaplazi, lamina propriada içleri immünglobülin ile dolu plazma hücreleri ve lenfoplazmositer hücre artışı izlenmiştir. Olguya uygulanan immünhistokimyasal boyalarda immünglobülin ile dolu plazma hücrelerinin CD3 (-), CD20 (-), CD79 a(+), CD45 (+), Kappa (+), Lambda (+) olduğu saptanmıştır.

Russell cisimciklerinin immünglobülin agregatları ve immünglobülin sentezinin yan ürünleri içerdiği düşünülürse *H.pylori* zemininde Russell cisimciklerinin yoğun olarak saptanması olasıdır. Ancak günümüze değin bu açıdan değerlendirilmiş sınırlı sayıda yayın bulunmaktadır.

Literatürde “Russell” cisimciklerinden zengin inflamatuvar reaksiyonla karakterize *Helikobakter gastritinin* nadir bildirilmiş olması nedeni ile olğumuz sunulmuştur.

Anahtar Sözcükler: Russell body, Gastrit, *Helikobakter pylori*, Mott hücresi

INTRODUCTION

Johansen A et al have studied the diagnostic significance of Russell body-containing cells in endoscopic biopsies in 1977 (1). However, the term “Russell body gastritis” has first been used by Tazawa K et al (2).

Russell bodies occur as a result of a block in the normal pathways of immunoglobulin secretion in plasma cells. Mott cells are plasma cells filled with Russell bodies. Russell bodies have been reported in many types of chronic inflammation and in various B cell lymphocytic neoplasms (3-7). Plasma cells are found together with lymphocytes, neutrophils and eosinophils in chronic gastritis. However, only 9 cases of chronic gastritis with plasma cell infiltration where Mott cells are dominant have been reported (8-12).

We describe a case in light of the literature as *H. pylori* gastritis characterized by an inflammatory reaction with widespread distribution of plasma cells full of Russell bodies (Mott cells) in the lamina propria.

CASE REPORT

Our case was a 50-year-old female with dyspeptic symptoms.

Endoscopic Findings: These consisted of hyperemia of fundus and antrum mucosa, prominent submucosal vessel network in the fundus and mucosal irregularity at the posterior wall.

Microscopic Findings: Mucosal samples of the antrum and corpus showed: *H. pylori* in the superficial mucus network,

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neutrophilic cryptitis in the gland and surface epithelium, widespread reactive/regenerative crypt hyperplasia, multifocal atrophy, intestinal metaplasia (Figure 1), plasma cells containing homogenous eosinophilic cytoplasmic globules in the lamina propria (Figure 2).

Immunohistochemistry Panel: CD20 and CD3 (-), and CD45 and CD79a (+) immune reaction in the plasma cells (Figures 3), positive immune reaction in some plasma cells with Kappa and Lambda (Figure 4).

Pathology Diagnosis: "Russell body" gastritis, multifocal atrophy, intestinal metaplasia, and *H. pylori* (+)

DISCUSSION

"Russell body gastritis" has been considered to be a different presentation of *Helicobacter pylori* gastritis in our case and

all other published cases (2,8-12). The mechanism may be plasma cell hyperactivation caused by chronic *H. pylori* infection and the immunoglobulin overproduction leading to Russell body formation. Johansen A et al. reported that Mott cells are found in significantly higher number at the periphery of cancerous samples but they have not mentioned the *H. pylori* state in their study (1). An esophageal and a cervical case characterized by infiltration with plasma cells filled with Russell bodies have also been reported (3,4). Such similar lesions outside the stomach indicate that Russell body formation may be associated with *Helicobacter pylori* or may develop due an antigenic stimulus as a result of chronic infection or some other reason. The disappearance of Mott cells during *H. pylori* eradication in the stomach also shows that the factor causing Russell body formation is *Helicobacter pylori* (2).

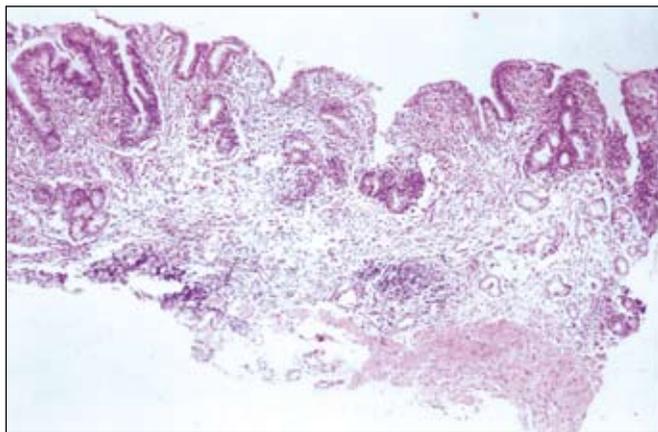


Figure 1: Widespread reactive/regenerative crypt hyperplasia, multifocal atrophy, complete colonic type intestinal metaplasia, and neutrophilic cryptitis in the gland and surface epithelium (H&E, x40).

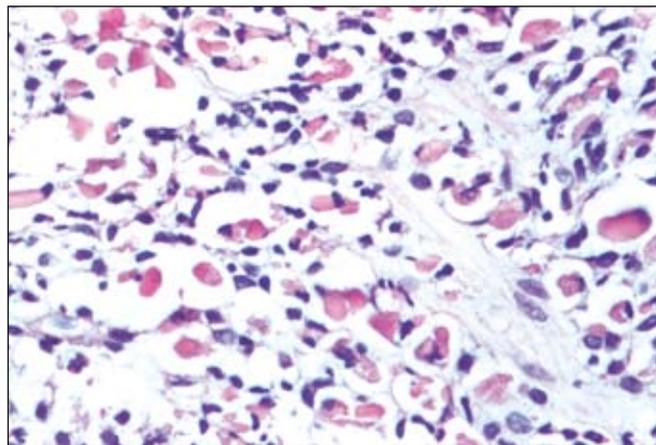


Figure 2: Plasma cells containing homogenous eosinophilic globules in the lamina propria (H&E, x200).

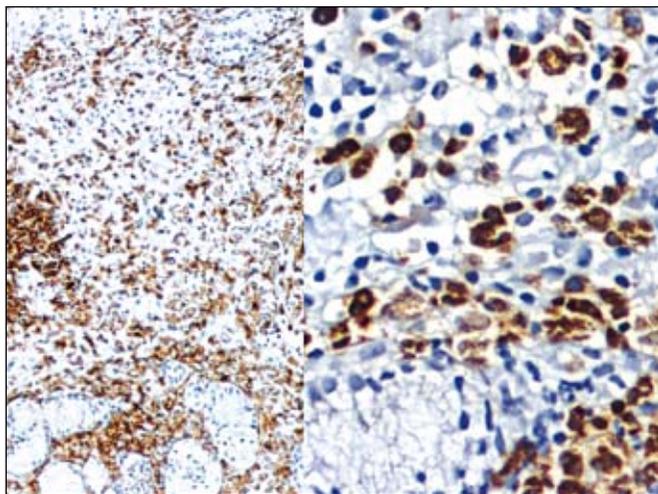


Figure 3: CD45 (left, DAB, x100) and CD79a (right, DAB, x200) positive lymphocytes.

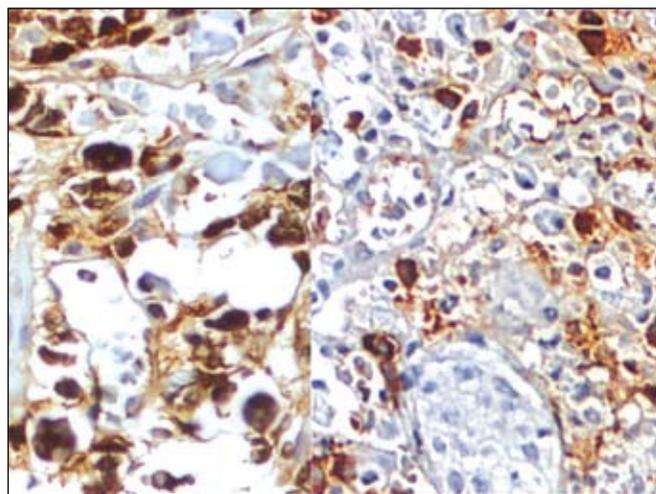


Figure 4: Positive immune reaction in some plasma cells with kappa (left, DAB, x400) and lambda (right, DAB, x200).

It is reported that plasma cell infiltration increases 2.5 times while the lymphocyte ratio stays constant in atrophic antral gastritis due to *H. pylori* (13). The immunoglobulin response developing against *H. pylori* is accepted to start first in the gastric mucosa in this disorder. This response is reported to end with an IgA dominant response in the mucosa and an IgG dominant systemic response outside the mucosa (14). Taking into account that Russell bodies contain immunoglobulin aggregates or side products of immunoglobulin synthesis, it is normal for Russell bodies to be densely located on a background of *H. pylori*. However, there are only a limited number of studies on the incidence of plasma cells containing Russell bodies in the gastric mucosa.

Johansen A et al have compared the peritumoral mucosa in patients with carcinoma (n=315) and gastric biopsies in patients without carcinoma (n=786) for the density of plasma cells containing Russell bodies. The significantly higher density of plasma cells containing Russell bodies in this study led to the statement that these cells could be important for adenocarcinoma development (1). We did not see any neoplastic progression in any of the 3 control endoscopies within the last 2 years. Fujiyoshi Y. has defined a Mott cell tumor developing due to the abnormal proliferation of IgG kappa monotypic plasma cells containing Russell bodies on a background of *H. pylori* (6). We thought that this tumor that had metastasized to the perigastric lymph nodes could be the MALT-lymphoma type developing from mucosa-dependent lymphoid tissue. We found polyclonality with the kappa and lambda immunohistochemistry stain used in our case and did not observe lymphoma development during follow-up.

Diffuse infiltration of plasma cells with Russell bodies in the gastric mucosa requires differentiating between several diagnoses (5,6). Cytokeratin negativity excludes signet-ring cell carcinoma while the kappa and lambda polyclonal immunoreactive pattern excludes lymphoplasmacytic lymphoma and plasmacytoma. The lesion can be differentiated from mucosa-associated lymphoid tissue (MALT) lymphoma with extreme plasmacytic differentiation with the absence of cytologic atypia, lymphoepithelial lesions, centrocyte-like cells and monocytoid cells.

Russell body gastritis needs to be kept in mind as it may be difficult to diagnose in biopsies consisting of small fragments.

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