

Russell body gastritis resolved after *Helicobacter pylori* eradication: a case report and review of the literature

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ABSTRACT

Russell body gastritis (RBG) is a rare gastric inflammatory lesion characterized by dense infiltration of plasma cells containing Russell bodies (RBs) known as Mott cells. Here, we report a case of RBG that showed resolution 2 months after *Helicobacter pylori* eradication. A gastric biopsy revealed numerous eosinophilic globular RBs and signet ring cell-like Mott cells. *H. pylori* infection was identified on the surface of foveolar epithelial cells. Alcian blue staining was negative for RBs. Periodic acid–Schiff staining showed that most RBs were negative, with only a few RBs and Mott cells showing weak positivity. Immunohistochemical staining for immunoglobulin G (IgG), kappa, and lambda light chains was performed on paraffin-embedded tissue. Plasma and Mott cells were positive for CD79a, CD138, IgG, kappa, and lambda. However, despite being traditionally considered as aggregates of immunoglobulin molecules, the majority of RBs are negative for IgG and light chain determinants. The marked reduction in RBs and reappearance of normal plasma cells following *H. pylori* eradication suggest that RB formation is a reversible and reactive process. However, the lack of staining in certain immunohistochemical analyses indicated the presence of unexpected alterations in immunoglobulin composition. The precise biochemical nature and fate of RBs warrant further investigation.

Keywords: Russell Body; Gastritis; *Helicobacter pylori*; Case Reports

Introduction

Russell bodies (RBs) are eosinophilic globular inclusions composed of immunoglobulins that accumulate within vacuoles of the rough endoplasmic reticulum (ER) of plasma cells. Plasma cells containing Russell's bodies are known as Mott cells [1,2].

Russell body gastritis (RBG), a rare finding on upper gastrointestinal endoscopy, is characterized by Mott cell accumulation in the gastric mucosa. It was first reported by Tazawa and Tsutsumi [3] in 1998. RBG is a form of chronic gastritis that is particularly

associated with *Helicobacter pylori* infection [1].

Endoscopically, RBG commonly present with mucosal erosion, swelling, erythema, and discoloration. Other reported findings include multiple ulcers and raised lesions, most frequently located in the gastric antrum [4-6].

Some studies have noted the resolution of these characteristic lesions following *H. pylori* eradication therapy [3]. Histologically, RBs are rarely observed in both the upper and lower gastrointestinal tracts.

This paper presents a case of RBG detected during upper en-

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doscopy that showed marked regression after *H. pylori* eradication, with discussion on the nature and implications of RBs.

Case Report

A 47-year-old woman visited Guro Sungsim Medical Center for routine national health screening. She had no significant medical history, including diabetes or hypertension, and was not taking any medication. Her surgical history was unremarkable, except for a prior ankle ligament surgery. She denied excessive alcohol consumption, smoking, or any known drug or food allergies. Apart from a family history of hypertension, no family history of malignancies was observed. Physical examinations revealed no abnormalities. Mammography performed at another institution

revealed microcalcifications, prompting a scheduled follow-up breast ultrasound.

Laboratory investigations revealed hemoglobin, 11.5 g/dL; aspartate aminotransferase, 18 IU/L; alanine aminotransferase, 11 IU/L; gamma-glutamyl transpeptidase level of 11 IU/L; and creatinine 0.62 mg/dL—all within normal limits. Chest radiographic findings were unremarkable.

During the clinical interview, the patient reported occasional stress-related epigastric cramps, however, had never taken medication for the symptoms.

Esophagogastroduodenoscopy revealed a 2 cm, map-like, white, flat granular lesion on the anterior wall of the gastric antrum, and a biopsy was performed (Figure 1A). The patient had undergone gastroscopy at our institution approximately 4 years earlier, however, had never undergone colonoscopy.

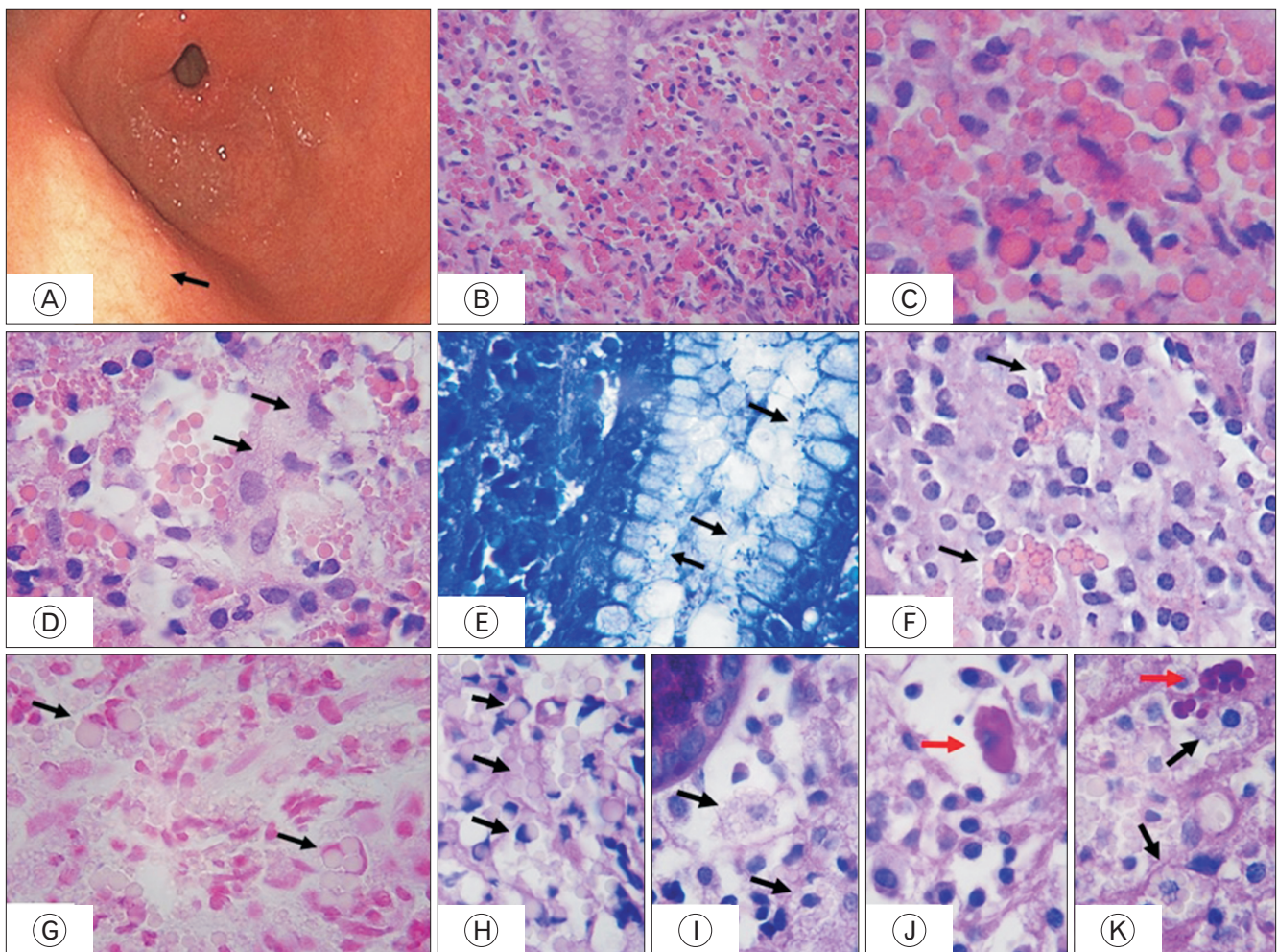


Figure 1. Clinical and pathological findings of Russell body gastritis. (A) Gastrointestinal endoscopic view (arrow). (B) Numerous variable-sized Russell bodies are present in the gastric mucosa ($\times 400$). (C) High-power view ($\times 1,000$). (D) Small vacuolar structures (arrows) are present in the cytoplasm of macrophages, suggesting phagolysosomal structures of the Russell bodies ($\times 1,000$). (E) Moderate degree of *Helicobacter pylori* infection on the surface mucous epithelium in the crypt of gastric mucosa (arrows). Giemsa stain ($\times 1,000$). (F) The gastric mucosa after *Helicobacter pylori* treatment shows a markedly decreased number of Russell bodies and Mott cells (arrows) ($\times 1,000$). (G) Alcian blue stain for signet ring-like Russell bodies is negative for mucin (arrows). Alcian blue stain ($\times 1,000$). (H–K) Periodic acid–Schiff staining ($\times 1,000$); (H) most Russell bodies, and signet ring-like Mott cells (arrows); (I) most Mott cells are negative (arrows); (J) only a few cells are positive (red arrow) in Mott cells; and (K) only a few positive Russell bodies (red arrow), negative in Mott cells (black arrows).

Histopathological examination of a gastric biopsy specimen revealed chronic gastritis with atrophic mucosa, superficial edema, erosion, and focal fibrosis. The most notable finding was the massive accumulation of eosinophilic globular bodies of variable sizes, the RBs, and a few plasma cells (Figure 1B, C). Only a few lymphocytes and macrophages with cytoplasmic vesicular structures suggestive of phagolysosomes were identified (Figure 1D). Moderate *H. pylori* infection was confirmed by Giemsa staining (Figure 1E), and post-treatment specimens were negative for *H. pylori*. Following the eradication therapy, histological findings showed a marked decrease in RBs and numerous plasma cells without RBs, with only a few Mott cells remaining (Figure 1F). Alcian blue staining for mucin within RBs was negative (Figure 1G). Periodic acid–Schiff (PAS) staining for immunoglobulins and glycoproteins demonstrated that nearly all RBs and Mott cells were negative, with only a few RBs and Mott cells showing weak positivity (Figure 1H–K).

Immunohistochemical staining was performed for plasma cell markers, including CD79a (Figure 2A), CD138, immunoglobulin G (IgG) (Figure 2B), and light chains (kappa, Figure 2D–F; lambda, Figure 2G–I). Pretreatment samples showed diffuse and evenly distributed staining without light chain restriction (Figure 2D–I). After treatment, the CD79a, CD138, kappa, and lambda staining intensities markedly decreased (Figure 2D–I).

Additionally, immunohistochemistry for IgG (Figure 2B), kappa (Figure 2D–F, before *H. pylori* treatment), and lambda (Figure 2G–I, before *H. pylori* treatment) showed that nearly all RBs were negative and most Mott cells also showed negative staining with rare-positive cells.

The patient was prescribed amoxicillin 1,000 mg, clarithromycin 500 mg, and esomeprazole 20 mg, each administered twice daily for 10 days. Approximately 1 month after completion, the urea breath test confirmed the successful eradication of *H. pylori*.

A follow-up biopsy of the previously identified white flat gran-

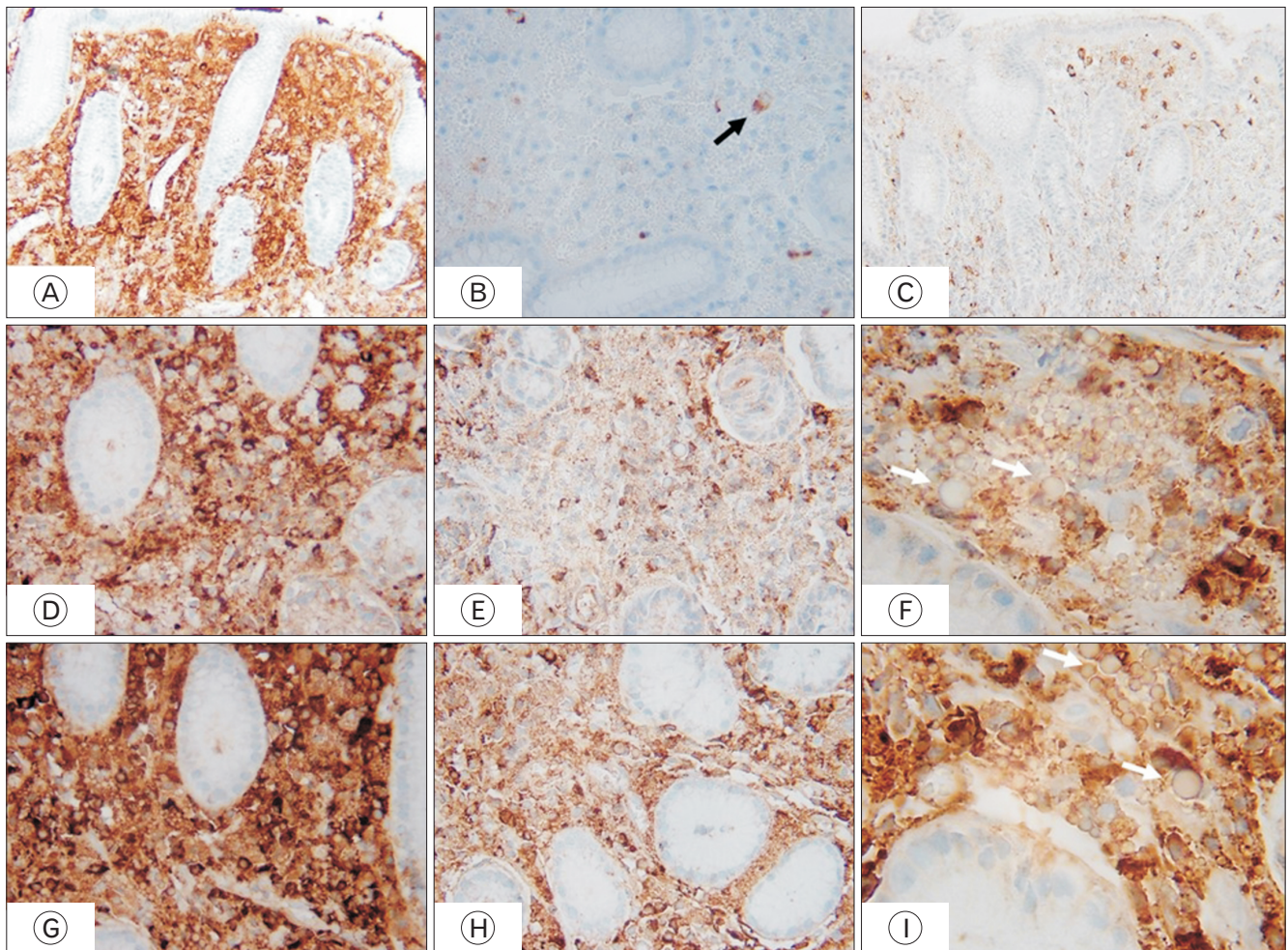


Figure 2. Immunohistochemical stains of Russell bodies. Immunohistochemical stain for CD79a ($\times 200$) (A), immunoglobulin G (IgG) ($\times 200$) (B), and CD68 ($\times 200$) (C). (B) Immunohistochemical stain for IgG shows a few positive in plasma cells not in Russell bodies (black arrow). (D, E) Immunohistochemical stain for kappa chain, before ($\times 400$) (D) and after ($\times 400$) *Helicobacter pylori* treatment (E). (F) Negative for kappa chain in Russell bodies (white arrows) ($\times 1,000$, before *H. pylori* treatment). (G, H) Immunohistochemical stain for lambda chain, before ($\times 400$) (G) and after ($\times 400$) *H. pylori* treatment (H). (I) Negative for lambda chain in Russell bodies (white arrows), ($\times 1,000$, before *H. pylori* treatment).

ular lesion revealed a marked reduction in both RBs and Mott cells with diffuse infiltration of plasma cells (Figure 1F).

Despite these histological improvements, some of the previously observed white nodular lesions persisted on follow-up endoscopy approximately 2 months later. However, tissue analysis confirmed a significant reduction in RBs and *H. pylori* was no longer detected.

The patient provided written informed consent for the publication of the research details and clinical images.

Discussion

RBG is a rare inflammatory condition with unclear prevalence both domestically and internationally. It is histologically diagnosed based on the presence of numerous red blood cells and Mott cell infiltration in the gastric mucosa.

RBs can occasionally be observed in lymphoid malignancies, such as plasmacytomas, multiple myeloma, and B-cell lymphomas, as well as in chronic hyperimmune states. Additionally, plasma cells containing RBs have been reported in autoimmune diseases, such as Hashimoto's thyroiditis, rheumatoid arthritis, and ulcerative colitis [2,7,8]. In this case, immunohistochemical staining for immunoglobulin kappa and lambda light chains was performed to differentiate between mucosa-associated lymphoid tissue lymphoma with plasmacytic differentiation and plasmacytoma. The results revealed a polyclonal pattern, confirming a non-neoplastic process (Figure 2A, D–I).

The exact pathogenesis of RBG remains unclear. However, numerous reports have been associated RBG with *H. pylori* infection, with approximately two-thirds of the reported cases being *H. pylori*-positive [6].

Nevertheless, cases of RBG without *H. pylori* infection have also been documented. Given its occurrence in patients with human immunodeficiency virus infection, post-transplant immunosuppression, alcohol or drug abuse, and monoclonal gammopathy, RBG may have broader etiological associations beyond *H. pylori* [7–10]. In *H. pylori*-negative infection, proton pump inhibitor therapy has been reported to reduce plasma cells containing RBs [11].

In the present case, a map-like, white, flat, granular lesion was identified in the gastric antrum. A review of the literature shows that such lesions are more commonly observed in the antrum than in the body [6]. This distribution is linked to *H. pylori* infection. In 2007, Soltermann et al. [12] reported that *H. pylori* strains with the VacA m1 genotype produce more RBs in the gastric antrum than in the body.

H. pylori causes chronic inflammation of the gastric mucosa. In our case, following eradication therapy, *H. pylori* was no longer detected, and follow-up histology revealed a marked decrease in RBs. This suggests a reduction in inflammatory response. Similarly, immunohistochemical staining for kappa and lambda light

chains showed decreased staining intensity, further indicating attenuation of immune and inflammatory responses after treatment.

RBs accumulate intracellularly and are not secreted during normal physiological processes. The exact mechanism underlying clearance after eradication therapy remains unknown. Tooze et al. [13] reported that RBs, as intracisternal granules, can be enclosed within autophagic vacuoles and subsequently degraded through fusion with lysosomes. In our case, some macrophages exhibited prominent vacuolar structures within their cytoplasm, suggesting phagocytosis and lysosomal degradation of RBs via phagolysosome formation.

Although the disappearance of RBs after *H. pylori* eradication remains largely unclear, it may be related to reduced immune stimulation, normalization of plasma cell function, and autolysis and heterolysis by macrophages.

The RBs were negatively stained for PAS (Figure 1H–K), IgG (Figure 2B), and light chains (kappa, Figure 2D–F; lambda, Figure 2G–I). Although their precise biochemical composition remains controversial, RBs are widely regarded as immunoglobulin aggregates. Matthews [14] reported that RBs are immunohistochemically positive for immunoglobulin heavy chains and kappa and lambda light chains, both intracellularly and extracellularly. These findings suggest that RBs, regardless of their size or location, are associated with intact immunoglobulin molecules.

However, Hsu et al. [15] reported cases in which RBs were not stained for immunoglobulins under light microscopy. Their study of gut lymphoma containing RBs using immunoelectron microscopy demonstrated the presence of immunoglobulin-positive flocculent material. They hypothesized that the lack of staining might have resulted from limited reagent penetration owing to the condensation of intercisternal immunoglobulins. These findings suggest that the principal biochemical composition of RBs may not be intact immunoglobulin aggregates, though rather modified immunoglobulin or byproducts of immunoglobulin synthesis. A similar mechanism may account for the lack of staining observed in this case.

Mattioli et al. [16] described RBs as dilated ER cisternae filled with mutant immunoglobulins. These mutant immunoglobulin- μ chains, lacking the first constant domain (μ CH1), are unable to reach the Golgi apparatus for secretion or the cytosol for degradation, leading to the accumulation of detergent-insoluble aggregates in the ER and formation of RBs.

Valetti et al. [17] experimentally induced RBs in both lymphoid and non-lymphoid cells by overexpressing mutant immunoglobulin heavy chains lacking the CH1 domain. These mutant proteins were partially degraded in the pre-Golgi compartments, forming insoluble aggregates that accumulated in the ER. Thus, RB formation represents a general cellular response to the overproduction of non-secretable and non-degradable immunoglobulins, resulting in the formation of Mott cells.

Such immunoglobulin variants lacking the CH1 domain con-

dense within the dilated cisternae of the early secretory pathway (ER), forming RBs that are commonly observed in plasma cell dyscrasias, autoimmune diseases, and chronic infections [18].

In conclusion, our findings suggest that protein aggregation in the ER arises from multiple cooperating factors, including excessive synthesis and insufficient degradation of proteins with an intrinsic tendency to aggregate, reduced chaperone binding, and altered interactions with ancillary proteins that facilitate condensation [19]. In addition, RBs are generally considered reactive and non-harmful phenomena. Despite the presence of large protein aggregates within the ER, Mott cells remain viable [20]. In our case, the disappearance of RBs and re-emergence of normal plasma cells after *H. pylori* eradication supports the hypothesis that RB formation represents a reversible, non-damaging, reactive, non-neoplastic process.

This case is of particular interest because of the divergent immunoglobulin staining findings compared to those described in previous reports. Given the limited understanding of underlying mechanisms, further case-based analyses and comprehensive studies are required. Additionally, further investigations using histochemical and immunohistochemical techniques at both the light and electron microscopic levels are necessary to clarify the biochemical and structural nature of RBs.

Article Information

Conflict of interest

No potential conflict of interest relevant to this article was reported.

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Data availability

Data of this research are available from the corresponding author upon reasonable request.

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KYS. Methodology: JEP, YBK. Validation: SAH. Writing—original draft: JHJ. Writing—review & editing: JHJ, JEP, KYS, YBK, SAH. Final approval of the manuscript: all authors.

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