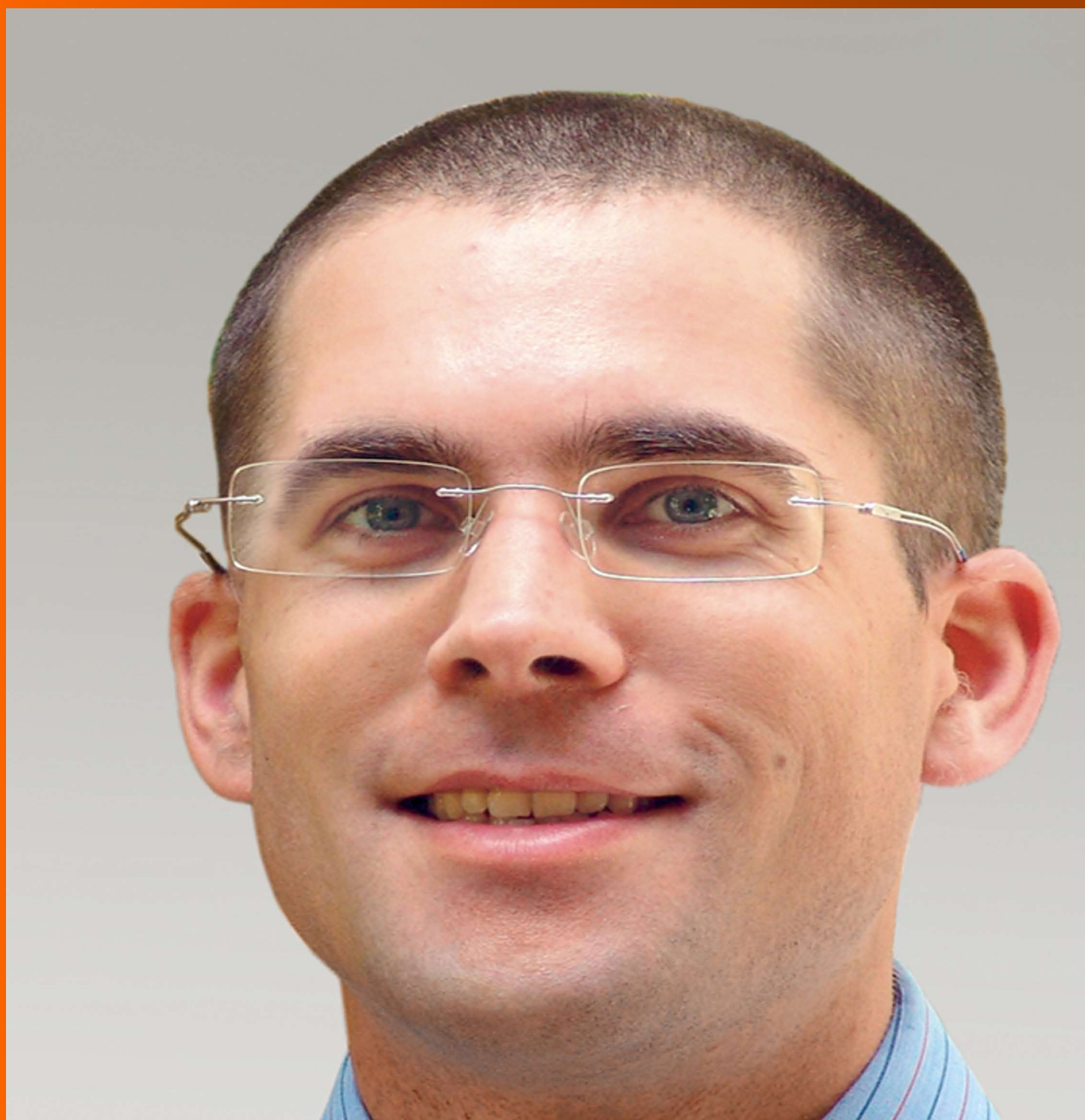


World Journal of *Gastrointestinal Endoscopy*

World J Gastrointest Endosc 2017 August 16; 9(8): 346-427



REVIEW

- 346 Terahertz endoscopic imaging for colorectal cancer detection: Current status and future perspectives
Doradla P, Joseph C, Giles RH

MINIREVIEWS

- 359 Use and barriers to chromoendoscopy for dysplasia surveillance in inflammatory bowel disease
Shukla R, Salem M, Hou JK
- 368 Evolution of stereoscopic imaging in surgery and recent advances
Schwab K, Smith R, Brown V, Whyte M, Jourdan I
- 378 Endoscopic ultrasonography - emerging applications in hepatology
Magalhães J, Monteiro S, Xavier S, Leite S, de Castro FD, Cotter J

ORIGINAL ARTICLE

Retrospective Cohort Study

- 389 Clinical impact of confocal laser endomicroscopy in the management of gastrointestinal lesions with an uncertain diagnosis
Robles-Medrand C, Vargas M, Ospina J, Puga-Tejada M, Valero M, Soria M, Bravo G, Robles-Jara C, Lukashok HP
- 396 Impact of laparoscopic liver resection on bleeding complications in patients receiving antithrombotics
Fujikawa T, Kawamoto H, Kawamura Y, Emoto N, Sakamoto Y, Tanaka A

Retrospective Study

- 405 Correlation of abnormal histology with endoscopic findings among mycophenolate mofetil treated patients
Izower MA, Rahman M, Molmenti EP, Bhaskaran MC, Amin VG, Khan S, Sultan K
- 411 Usefulness of the Hook knife in flexible endoscopic myotomy for Zenker's diverticulum
Rouquette O, Abergel A, Mulliez A, Poincloux L

CASE REPORT

- 417 Russell body gastritis with Dutcher bodies evaluated using magnification endoscopy
Yorita K, Iwasaki T, Uchita K, Kuroda N, Kojima K, Iwamura S, Tsutsumi Y, Ohno A, Kataoka H
- 425 Simultaneous Courvoisier's and double duct signs
Agrawal S, Vohra S

Contents

World Journal of Gastrointestinal Endoscopy
Volume 9 Number 8 August 16, 2017

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World Journal of Gastrointestinal Endoscopy
Baishideng Publishing Group Inc
7901 Stoneridge Drive, Suite 501, Pleasanton, CA 94588, USA
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Pleasanton, CA 94588, USA
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Russell body gastritis with Dutcher bodies evaluated using magnification endoscopy

Kenji Yorita, Takehiro Iwasaki, Kuniyoshi Uchida, Naoto Kuroda, Koji Kojima, Shinichi Iwamura, Yutaka Tsutsumi, Akinobu Ohno, Hiroaki Kataoka

Kenji Yorita, Naoto Kuroda, Department of Diagnostic Pathology, Japanese Red Cross Kochi Hospital, Kochi-city, Kochi 780-8562, Japan

Takehiro Iwasaki, Kuniyoshi Uchida, Koji Kojima, Shinichi Iwamura, Department of Internal Medicine, Japanese Red Cross Kochi Hospital, Kochi-city, Kochi 780-8562, Japan

Yutaka Tsutsumi, Department of Pathology, Fujita Health University School of Medicine, Toyoake, Aichi 470-1192, Japan

Akinobu Ohno, Pathology Section, University of Miyazaki Hospital, Miyazaki 889-1601, Japan

Hiroaki Kataoka, Section of Oncopathology and Regenerative Biology, Department of Pathology, Faculty of Medicine, University of Miyazaki, Miyazaki 889-1601, Japan

Author contributions: Yorita K performed pathological diagnosis, collected and analyzed the data, and wrote the paper; Iwasaki T, Uchida K, Kojima K and Iwamura S performed the clinical diagnosis, endoscopic submucosal resection of the lesion, and clinical follow-up; Kuroda N, Tsutsumi Y, Ohno A and Kataoka H performed the pathological diagnosis.

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Correspondence to: Kenji Yorita, MD, PhD, Department of Diagnostic Pathology, Japanese Red Cross Kochi Hospital, 2-13-51, Shinonmachi, Kochi-city, Kochi 780-8562, Japan. kenjiyorita@gmail.com
Telephone: +81-88-8221201
Fax: +81-88-8221056

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Abstract

Russell body gastritis (RBG) is an unusual type of chronic gastritis characterized by marked infiltration of Mott cells, which are plasma cells filled with spherical eosinophilic bodies referred to as Russell bodies. It was initially thought that *Helicobacter pylori* (*H. pylori*) infection was a major cause of RBG and that the infiltrating Mott cells were polyphenotypic; however, a number of cases of RBG without *H. pylori* infection or with monoclonal Mott cells have been reported. Thus, diagnostic difficulty exists in distinguishing RBG with monoclonal Mott cells from malignant lymphoma. Here, we report an unusual case of an 86-year-old-Japanese man with *H. pylori*-positive RBG. During the examination of melena, endoscopic evaluation confirmed a 13-mm whitish, flat lesion in the gastric antrum. Magnification endoscopy with narrow-band imaging suggested that the lesion was most likely a poorly differentiated adenocarcinoma. Biopsy findings were consistent with chronic gastritis with many Mott cells with intranuclear inclusions referred to as Dutcher bodies. Endoscopic submucosal dissection confirmed the diagnosis of RBG with kappa-restricted monoclonal

Mott cells. Malignant lymphoma was unlikely given the paucity of cytological atypia and Ki-67 immunoreactivity of monoclonal Mott cells. This is the first reported case of RBG with endoscopic diagnosis of malignant tumor and the presence of Dutcher bodies.

Key words: Russell body gastritis; Mott cell; Dutcher body; Mucosa-associated lymphoid tissue lymphoma; Plasmacytoma; Magnification endoscopy with narrow-band imaging

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Core tip: We report Russell body gastritis (RBG) evaluated by magnification endoscopy with narrow band imaging and pathological evaluation by endoscopic submucosal dissection. The endoscopic features of RBG are exclusively inflammatory; however, our detailed endoscopic evaluation led to misdiagnosis of the lesion as poorly differentiated adenocarcinoma. The histological features of RBG were also unique because the presence of Mott cells with light chain restriction and Dutcher bodies suggested malignant lymphoma. Pathologists should be aware of the existence of this pathological entity, and clinicians should consider RBG as a differential diagnosis in cases where detailed endoscopic examination reveals poorly differentiated early gastric cancer.

Yorita K, Iwasaki T, Uchita K, Kuroda N, Kojima K, Iwamura S, Tsutsumi Y, Ohno A, Kataoka H. Russell body gastritis with Dutcher bodies evaluated using magnification endoscopy. *World J Gastrointest Endosc* 2017; 9(8): 417-424 Available from: URL: <http://www.wjgnet.com/1948-5190/full/v9/i8/417.htm> DOI: <http://dx.doi.org/10.4253/wjge.v9.i8.417>

INTRODUCTION

Russell body gastritis (RBG) was first described by Tazawa *et al.*^[1] in 1998 and is considered as a unique form of chronic gastritis characterized by infiltration of plasma cells filled with spherical eosinophilic cytoplasmic globules, referred to as Russell bodies. The endoscopic features of RBG are most in keeping with an inflammatory condition, and biopsy is required to confirm the pathological diagnosis. RBG is considered a reactive condition; however, monoclonal Mott cells have been shown in a number of cases of RBG, and therefore whether or not RBG with monoclonal Mott cells is benign remains debatable. This uncertainty is due to the fact that monoclonal Mott cells can show regional lymph node metastasis^[2] and have been identified in mucosa-associated lymphoid tissue (MALT) lymphoma with extreme plasmacytoid differentiation^[3]. Here, we report a unique case of RBG with endoscopic features of a malignant tumor, which consisted of monoclonal Mott cells with Dutcher bodies identified histologically following endoscopic submucosal resection.

CASE REPORT

An 86-year-old Japanese man with a history of rheumatoid arthritis, type 2 diabetes mellitus, and hypertension was referred to our medical center with melena. He took non-steroidal anti-inflammatory drugs for arthralgia but did not take immunosuppressive drugs. Physiological examination was unremarkable. Laboratory findings revealed anemia (Hb 6.5 g/dL) and no other abnormal results. Endoscopic evaluation of the upper and lower digestive tracts did not reveal any active bleeding. *Helicobacter pylori* (*H. pylori*) infection was confirmed on a positive serum anti-*H. pylori* antibody test. The patient was initially diagnosed with atrophic and erosive gastritis secondary to *H. pylori* infection, and eradication therapy was initiated. The first esophagogastroduodenoscopy revealed a 13-mm flat lesion (Figure 1A and B) of white and slightly brown discoloration in the lesser curvature of the antrum. A magnification endoscope (Gastrointestinal fiber-H260Z, Olympus, Tokyo, Japan) with a narrow-band imaging (NBI) system (EVIS LUCERA SPECTRUM ELITE system, Olympus) was used, and magnification endoscopy with NBI (M-NBI) of the lesion showed loss or irregularity of microsurface pattern, irregular microvascular proliferation, and a demarcation line (Figure 1C and D). Poorly differentiated adenocarcinoma was suspected, and a biopsy was performed. The duodenum appeared to be intact. The biopsy specimen (Figure 2A-D) showed chronic gastritis with infiltration of lymphocytes, plasma cells, eosinophils, and small-to large-sized granulated cells (Figure 2B-D). Spiral-shaped bacilli were focally located in the mucin on the foveolar epithelium, confirming the diagnosis of *H. pylori*. Substantial amounts of granulated cells with eosinophilic cytoplasmic granules and eccentric nuclei were seen (Figure 2B), which were of a similar size to the eosinophilic granules (Figure 2C). The small-sized granulated cells showed plasmacytoid morphology with eccentric and cartwheel-like nuclei, while ballooning large granulated cells showed histiocytoid morphology (Figure 2C). Intranuclear eosinophilic granules were also observed in some granulated cells (Figure 2D) but were not apparent in infiltrating plasma cells without cytoplasmic granules. Lymphoid follicles were not observed. Neither lymphoepithelial lesions nor monotonous proliferation of centrocyte-like cells or monocytoid-B cells were observed. The cytoplasmic granules were stained by phosphotungstic acid-hematoxylin (PTAH) and periodic acid Schiff with or without diastase treatment. Immunohistochemically, the granulated cells including histiocytoid cells were positive for CD79a (Clone HM57, DAKO, Glostrup, Denmark) and multiple myeloma oncogene 1 (Clone MUM1p, DAKO, Glostrup, Denmark) and negative for pancytokeratin (Clone CAM5.2, Becton Dickinson, CA), CD20 (Clone L26, DAKO, Glostrup, Denmark), CD138 (Clone MI15, DAKO, Glostrup, Denmark), CD68 (Clone KP-1, DAKO, Glostrup, Denmark), CD163

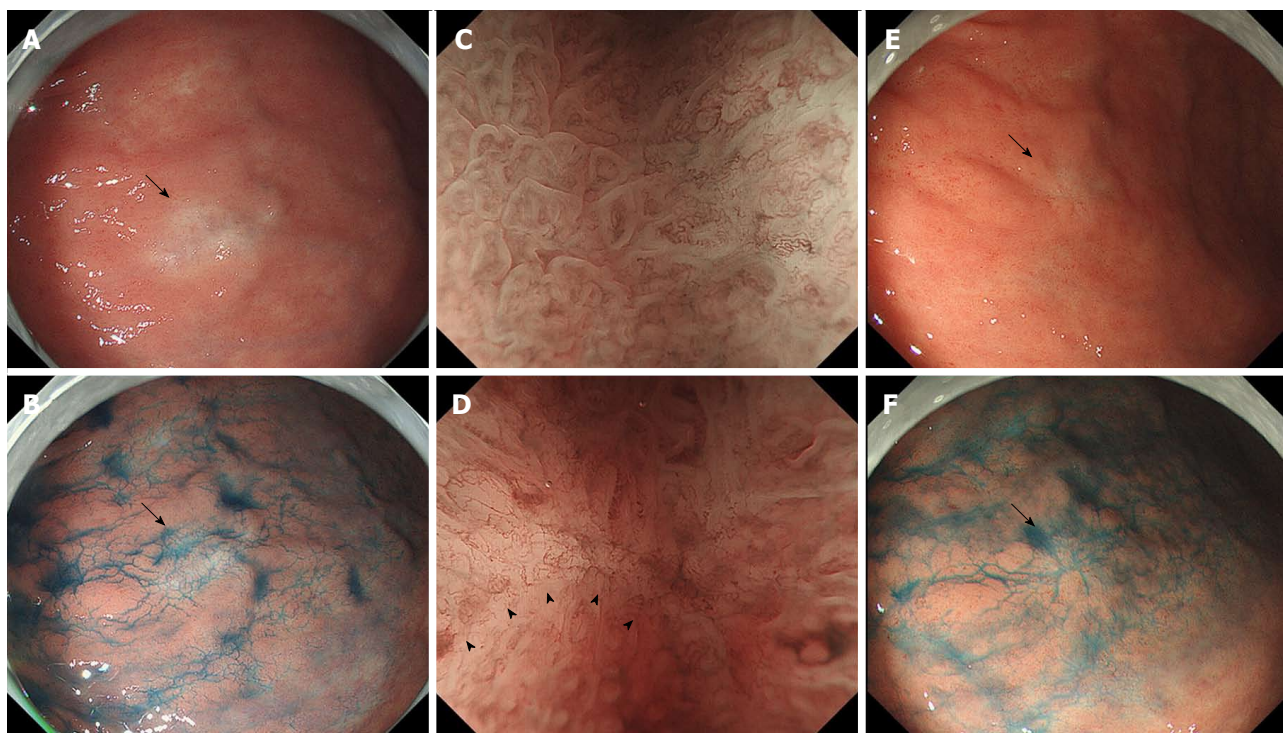


Figure 1 Endoscopic imaging of the antral lesion before and after the eradication. A: An endoscopic image before the eradication shows a 13-mm whitish/slightly brownish lesion (arrow) on the lesser curvature of the antrum; B: Indigo carmine dye spray reveals that the antral lesion (arrow) is almost flat; C: Magnification endoscopy with narrow-band imaging shows the lesion (C, right side) exhibits loss or irregularity of the microsurface pattern and irregular microvascular proliferation compared to the background mucosa (C, left side). A demarcation line (D, arrowheads) is seen at the periphery of the lesion. E-F: Endoscopic images after the eradication therapy exhibit that the mucosal lesion (arrow) decreases in size to 7 mm in diameter (E) and preserves the gross appearance with indigo carmine dye spray (F).

(10D6, Thermo scientific), CD1a (Clone 10, DAKO, CA), S100 (polyclonal, DAKO, Glostrup, Denmark), c-KIT (polyclonal, DAKO, Glostrup, Denmark), mast cell tryptase (Clone AA1, DAKO, Glostrup, Denmark), and Ki-67 (Clone MIB-1, DAKO, Glostrup, Denmark). Thus, the granulated cells were considered to be plasma cells and identified as Mott cells with cytoplasmic eosinophilic globules (Russell bodies). Intranuclear eosinophilic granules in some Mott cells were considered to be Dutcher bodies. Immunohistochemically, the Mott cells appeared to be negative for light chains and immunoglobulin G (IgG, polyclonal, Novocastra Laboratories Ltd., Newcastle, United Kingdom), IgA (polyclonal, Novocastra Laboratories Ltd., Newcastle, United Kingdom), and IgM (polyclonal, Novocastra Laboratories Ltd., Newcastle, United Kingdom), while plasma cells without Russell bodies showed polytypic light chain staining pattern and were reactive for IgG, IgA, or IgM in varying proportions. Taken together, these findings were most in keeping with a diagnosis of RBG, but malignant lymphoma, particularly MALT lymphoma, was a differential diagnosis. However, chest-abdomen-pelvis computed tomography showed no abnormal mass, no swollen lymph nodes, and no lytic bone lesion. Two months after completion of eradication therapy, the patient consented to undergo endoscopic submucosal dissection (ESD), which was performed with the aim of histologically evaluating the entire lesion.

After eradication therapy, the mucosal lesion decreased in size to 7 mm in diameter (Figure 1E-F) and loss or irregularity of the microsurface pattern, irregular microvascular proliferation, and a demarcation line with M-NBI were seen. ESD was successfully performed. The patient was discharged without any complications and there was no endoscopic evidence of recurrence 14 mo after the ESD treatment.

Pathological diagnosis on ESD specimens

The endoscopically resected tissue was extended on a board with pins, fixed in 10% formalin for 24 h, cut into 2- to 3-mm thick sections, and embedded in paraffin. Four μ m-thick sections were obtained from the paraffin blocks and stained with hematoxylin and eosin. Pathology showed that the mucosal lesion had regional accumulation of substantial amounts of granulated cells associated with mild lymphocytic and plasma cell infiltration (Figure 2E and F). The immunohistochemical results of ESD were similar to those of the biopsy specimens (Figure 2G and H). In situ hybridization revealed that the Mott cells showed kappa light chain restriction (Figure 2I and J), whereas the plasma cells without Russell bodies were polyphenotypic. Cellular atypia and mitosis were not seen in the plasma cells or Mott cells, and less than 1% of the infiltrating cells were Ki67-positive, whereas no Mott cells were Ki67-positive. At the periphery of the lesion and in the background

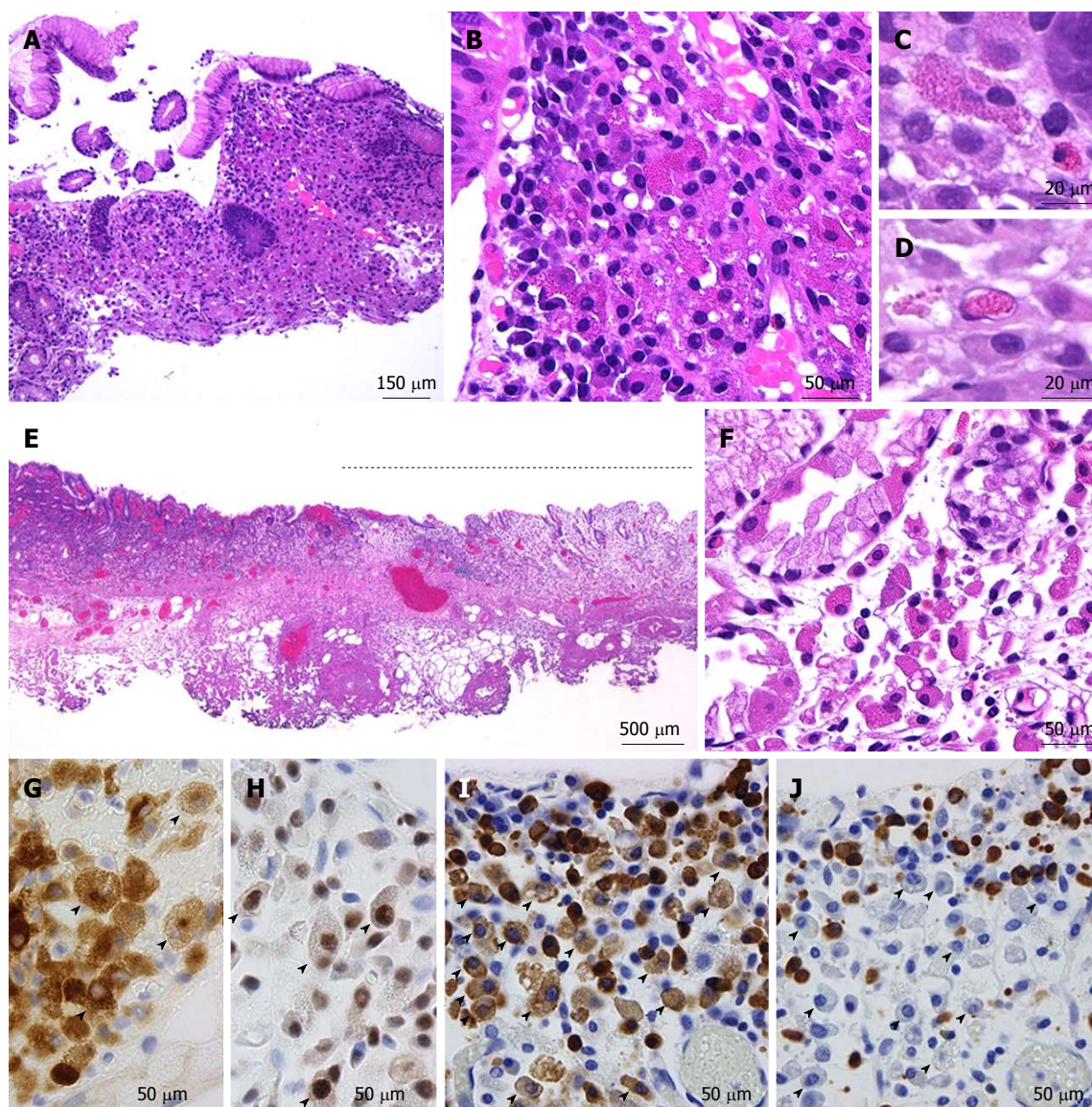


Figure 2 Pathological features of the antral lesion. The biopsy specimen stained by hematoxylin and eosin shows chronic gastritis with crowded granulated cells (A, B). The histiocytoid cells have fine eosinophilic granules similar to those of eosinophils (C). Intranuclear cytoplasmic granules suggesting Dutcher bodies are observed (D). E-F: The ESD specimen stained by hematoxylin and eosin shows that the lesion (E, indicated by a dotted line) is edematous and demarcated and includes many granulated cells (F). G-J: Immunohistochemical (G-H) and in situ hybridization (I-J) findings with ESD specimens show that the granulated cells (arrowheads) are positive for CD79a (G), multiple myeloma oncogene 1 (H), and kappa (I) and negative for lambda (J), while plasma cells without cytoplasmic granules are polyphenotypic.

mucosa and submucosa, Mott cells and Dutcher bodies were absent. Lymphoid follicles were observed in the periphery of the lesion and the background mucosa, but not within the collection of Mott cells. There were no pathological features suggestive of carcinoma or MALT lymphoma. Amyloid deposition was unlikely because no amorphous materials were identified, and there was no characteristic apple green birefringence noted when the Congo Red stained section was viewed under polarized light. Thus, our final diagnosis was Russell body gastritis. However, the diagnostic assessment utilized had several limitations, as we did not assess for the presence of M protein in the serum, Bence-Jones protein in the urine, or genetic alterations in the immunoglobulin locus, and we did not perform

cytopathological examination of the bone marrow.

DISCUSSION

We presented a unique case of RBG with *H. pylori* infection. Russell body gastritis is characterized by a dense accumulation of plasma cells with Russell bodies, referred to as Mott cells^[1]. Russell bodies are eosinophilic spherical or globular cytoplasmic inclusions in plasma cells that were first described by Russell *et al*^[4] in 1890. The globules differ from normal secretory granules in that they are larger and much more electron dense, and they lie in the distended cisternae of the rough endoplasmic reticulum^[5]. These globules mainly consist of condensed immunoglobulin^[6] and are

related to abnormalities in the synthesis, trafficking, or excretion of immunoglobulin^[7]. The Russell bodies seen in the present case were much smaller compared to those in previous reports; however, the granules were stained positive by periodic acid Schiff and PTAH, as described in previous case reports of RBG^[8,9].

Table 1 summarizes the results of 31 previously published cases of RBG, including our own^[1,8-25]. We excluded two cases reported as RBG^[26] and Russell body carditis^[27] because in these cases the inflammation site was at the esophagogastric junction. One case of RBG associated with advanced signet ring cell cancer^[28] was also excluded because the authors did not describe whether or not RBG was present in the background gastric mucosa. The mean age of the patients described in previous case reports of RBG was 60 (range 24 to 86) years old, and our patient was the oldest. The male ($n = 19$) to female ($n = 12$) ratio was approximately 2:1. Twenty-one cases (68%) of RBG accompanied *H. pylori* infection, and the majority of patients in all cases presented with non-specific symptoms. The gastric antrum appears to be the predominant location for RBG, although there is one reported case of an immunocompromised patient with RBG associated with Russell body enterocolitis^[25].

The endoscopic features of RBG are non-specific, and in most cases, the inflammatory phenotype of the condition has been attributed largely to the presence of swollen, erythematous mucosa. The pathological features of RBG can mimic those of gastric xanthoma^[19]. To the best of our knowledge, this is the first case of RBG with a clinical diagnosis highly suggestive of a malignant tumor. In the present case, M-NBI confirmed a flat mucosal lesion in the antrum with a demarcated line, loss of microsurface pattern, and irregular microvascular proliferation. Nishimura *et al.*^[29] also reported that partial loss of microsurface structure and abnormal microvessels were observed in a case of RBG with M-NBI, but the demarcation line of RBG was not reported. Magnification endoscopy permits visualization of mucosal details that cannot be seen with standard endoscopy, and NBI is a novel endoscopic approach to visualize the microvasculature on the tissue surface. M-NBI has been proven to be highly sensitive, specific, and accurate at detecting early gastric neoplasms. Pathological evaluation revealed that the lesion was characterized by regional and intramucosal accumulation of Mott cells. The proliferation of Mott cells might induce expansion of the lamina propria, decrease the density of gastric glands and pits, and influence the microvasculature of the lesion. At present, RBG is considered a rare entity, but its incidence may increase in the future secondary to the increased use of M-NBI. It may be better that clinicians raise RBG as a differential diagnosis of poorly differentiated early gastric cancer upon examination by M-NBI.

Of pathological interest, Mott cells were monoclonal and had Dutcher bodies, which caused difficulty in making a histological diagnosis. Only two cases^[8,12]

of RBG and Dutcher bodies have been described in the literature to date, and both failed to identify a Dutcher body in the lesion. Considering the presence of monoclonal Mott cells and Dutcher bodies, the differential diagnoses that we considered were plasma cell neoplasm, MALT lymphoma, and lymphoplasmacytic lymphoma. In the present case, MALT lymphoma could not be diagnosed on account of the lack of histological features suggestive of MALT lymphoma, such as a lymphoepithelial lesion and proliferation of monocytoid B-cells or centrocyte-like cells. Moreover, Mott cells showed neither cellular atypia nor mitotic activity. Plasma cell neoplasm was excluded due to hypoproteinemia, lack of osteolysis in computed tomography images, and lack of nuclear atypia or mitotic activity of monoclonal Mott cells, although paraproteinemia was not evaluated *via* serum protein electrophoresis. Lymphoplasmacytic lymphoma was ruled out because of an absence of splenomegaly and lymphadenopathy. Monoclonal gammopathy of undetermined significance^[13], which can be associated with *H. pylori*-positive RBG, could not be evaluated in this case.

The pathogenesis of RBG remains unknown. Tazawa *et al.*^[1] firstly postulated that RBG might be induced by *H. pylori* infection, and this is in keeping with previously published literature, in which two thirds of RBG cases were *H. pylori*-positive (Table 1). A recent study^[30] showed that *H. pylori* with vacA m1 genotype produces more prominent Russell bodies in the antrum but not in the body. Indeed, the literature review revealed that the incidence of antral RBG was higher in *H. pylori*-positive cases (70%) than in *H. pylori*-negative cases (40%). On the other hand, our case showed endoscopic regression of the lesion from 13 mm to 7 mm at 2 mo after eradication of *H. pylori*, and similar findings were observed in previous studies^[16,18,19,21]. These observations suggest that the successful eradication of *H. pylori* may eliminate the proliferative stimulation of Mott cells. However, case reports of RBG without *H. pylori* infection have also been described, although those cases were complicated by HIV infection^[11], post-transplant status^[25], or monoclonal gammopathy of uncertain significance^[13]. Therefore, it is likely that *H. pylori*-unrelated pathogenesis of RBG is present, and an immunocompromised status may be partly related to the occurrence of RBG. In our case, the patient was elderly and had a history of rheumatoid arthritis; therefore, it is possible that the occurrence of RBG in this patient may have been due, in part, to his immunocompromised status.

RBG is considered to be a benign condition, and clinical follow-up data of 19 cases of RBG to date has failed to reveal any malignant change (Table 1). Among the 19 cases described in the literature, ten cases received *H. pylori* eradication therapy after diagnosis of RBG, and most of these showed endoscopic or histological resolution at three months, whether or not monoclonal Mott cells were present. Thus, follow-

Table 1 Literature review of clinicopathological findings of Russell body gastritis

Ref.	Age/sex	Endoscopic finding or diagnosis (size)	Site of mott cells	HP	Mott cells	ET and follow-up
Tazawa <i>et al</i> ^[1] (1998)	53/M	Multiple ulcer scars with redness and swelling	Antrum	Yes	Poly	Follow-up biopsy after ET showed no RBG Follow-up period was not available
Erbersdobler <i>et al</i> ^[8] (2004)	80/F	Circumscribed irregular swelling (30 mm)	Fundus	No	Poly	NA
Ensari <i>et al</i> ^[10] (2005)	70/M	Pangastritis/flattened, edematous gastric folds	Body and antrum	Yes	Poly	ET was performed, but patient refused to be re-examined endoscopically
Drut <i>et al</i> ^[11] (2006)	34/M	A raised, swollen area (20 mm)	Body	No	Poly	NA
Paik <i>et al</i> ^[12] (2006)	47/F	Focal erythematous swelling	Antrum	Yes	Poly	ET was performed. Follow-up data: NA
	53/F	A geographical yellowish raised lesion (25 mm)	Body	Yes	Poly	ET was performed Follow-up data: NA
Wolkersdorfer <i>et al</i> ^[13] (2006)	54/M	Mild erythema and small erosions with slight edema	Antrum	Yes	Mono (λ chain)	One year after ET, the lesion had not resolved macroscopically, but biopsy found resolution of Mott cells
Pizzolitto <i>et al</i> ^[14] (2007)	60/F	Minute-raised granular areas	Antrum	Yes	Poly	ET was performed, and clinical follow-up was uneventful
Licci <i>et al</i> ^[15] (2008)	59/M	Mild hyperemia	Antrum	Yes	Poly	Mott cells were absent in biopsy specimen taken 3 mo after ET
Tabata <i>et al</i> ^[16] (2010)	72/M	Multiple ulcers	Body and antrum	Yes	Mono (κ chain, IgG)	Mott cells were absent in biopsy specimen taken 3 mo after ET
Habib <i>et al</i> ^[17] (2010)	75/M	Nodular chronic active gastritis	Antrum	No	Poly	NA
Miura <i>et al</i> ^[18] (2012)	63/F	Low elevated lesions in the antrum	Antrum	Yes	Mono (λ chain)	Mott cells were absent in biopsy specimen taken 4 mo after ET
Yoon <i>et al</i> ^[19] (2012)	57/M	A slightly raised whitish lesion with a mild central depression (20 mm)	Body	Yes	Poly	The lesions were cleared 3 mo after ET. A follow-up biopsy was not performed
	43/M	A whitish oval shaped flat lesion with a slight central depression (20 mm)	Antrum	Yes	Poly	The lesions were cleared 2 mo after ET. A follow-up biopsy was not performed
Choi <i>et al</i> ^[20] (2012)	55/M	A mucosal elevation with a central depression (10 mm)	Antrum	Yes	Mono (λ chain)	NA
Karabagli <i>et al</i> ^[21] (2012)	60/M	Erythema (body) and ulcer (incisura angularis)	Body and antrum	Yes	Poly	Three months and 6 mo after ET, Mott cells were decreased and absent in biopsy specimens, respectively
Coyne <i>et al</i> ^[22] (2012)	49/M	Severe, raised, erosive gastritis	NA (Biopsy site; NA)	No	Mono (κ chain, IgM)	NA
Araki <i>et al</i> ^[9] (2013)	74/F	Open ulcer	Gastric angle	Yes	Mono (κ chain, IgM)	NA
Zhang <i>et al</i> ^[23] (2014)	78/F	Uneven mucosa	Body, incisura angularis, antrum	No	Mono (κ chain)	Clinical follow-up evaluations were uneventful
	77/F	Uneven mucosa	Incisura angularis	Yes	Mono (κ chain)	
	77/F	punctiform erosion	Body	Yes	Mono (κ chain)	
	56/M	Raised erosions	Antrum	Yes	Mono (κ chain)	
	76/M	Erythema	Body	Yes	Mono (κ chain)	
	50/M	Flat and raised erosions	Antrum	Yes	Mono (κ chain)	
	28/M	Erythema	Antrum	No	Mono (κ chain)	
	24/F	Erythema	Antrum	No	Mono (κ chain)	
	66/M	Ulcer, stage A2	Incisura angularis	No	NA	
Klair <i>et al</i> ^[24] (2014)	76/F	Cobblestoned, whitish, raised, and irregular mucosa	Fundus	No	Poly	NA
Muthukumarana <i>et al</i> ^[25] (2015)	44/M	Diffuse mild erythematous gastric mucosa	Stomach, duodenum, terminal ileum, colon	No	Poly	NA
Nishimura <i>et al</i> ^[29] (2016)	64/F	A white, granular lesion (2 cm)	Body	Yes	Poly	The lesion had grown larger 15 mo after the diagnosis, and the lesion had disappeared 15 mo after eradication
Present case	86/M	A demarcated whitish flat lesion (13 mm)	Antrum	Yes	Mono (κ chain)	Two months after ET, the lesion decreased in size. There was no evidence of recurrence 14 mo after ESD

HP: *Helicobacter pylori*; ET: Eradication therapy; Poly: Polyclonal; Mono: Monoclonal; RBG: Russell body gastritis; NA: Not available; Ig: Immunoglobulin.

up might be a better approach for the present case. However, whether or not RBG with monoclonal Mott cells is a reactive or neoplastic phenomenon remains debatable. To date, a total of 15 cases (50%, 15/30) of RBG with monoclonal Mott cells, including our case, have been described, and all of these RBG cases had uneventful clinical follow-up. Brink *et al.*^[31] reported a case with a rectal tubulovillous adenoma accompanied by a proliferation of monoclonal Mott cells and concluded that the phenomenon indicated an inflammatory response. As Araki *et al.*^[9] discussed, monoclonal B cell proliferation can be seen in cases of chronic inflammation such as lymphoid follicle-forming gastritis^[32], Sjögren's syndrome^[33], Hashimoto's thyroiditis^[34], and chronic liver disease secondary to hepatitis C virus^[35]. Thus, the above data support the theory that RBG with monoclonal Mott cells is a benign condition. However, Fujiyoshi *et al.*^[2] described a gastric tumor consisting of monoclonal Mott cells which metastasized to the perigastric lymph nodes, and they concluded that the gastric tumor was most likely an extramedullary plasmacytoma or a MALT lymphoma. Joo^[3] and Kai *et al.*^[36] also showed that monoclonal Mott cells can be a neoplastic component of MALT lymphoma with extreme plasmacytoid differentiation. Fujiyoshi *et al.*^[2], Joo^[3], and Kai *et al.*^[36] did not describe cellular atypia or mitotic activity in monoclonal Mott cells in their cases, and therefore the histopathological distinction of monoclonal Mott cells in RBG and in MALT lymphoma and plasmacytoma remains unknown. In the present case, we concluded that the monoclonal Mott cells were non-neoplastic cells mainly because of the absence of cellular atypia and mitotic activity. Nevertheless, future studies are warranted to clarify the distinction between RBG with monoclonal Mott cells and MALT lymphoma or plasmacytoma.

In conclusion, we presented an unusual case of an 86-year-old man with *H. pylori*-positive RBG. This is a valuable case of RBG evaluated by M-NBI and pathological evaluation of the entire lesion. The detailed endoscopic evaluation led to the clinical diagnosis of a poorly differentiated adenocarcinoma, despite the fact that previous endoscopically diagnosed cases of RBG were exclusively inflammatory. Pathological evaluation of ESD-obtained specimens confirmed the presence of regional proliferation of kappa-restricted Mott cells within the mucosal lesion and failed to identify an epithelial malignancy. RBG is a rare condition whose incidence is expected to increase in proportion to the increased use of M-NBI. Therefore, it is of great clinical importance to increase our understanding of the pathological features of RBG in order to effectively diagnose and manage future cases.

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COMMENTS

Case characteristics

An 86-year-old Japanese man with melena and a history of rheumatoid arthritis and type 2 diabetes mellitus.

Clinical diagnosis

The patient was initially diagnosed with atrophic and erosive gastritis secondary to *Helicobacter pylori* (*H. pylori*) infection, and the first esophagogastroduodenoscopy revealed a 13-mm flat lesion of white and slightly brown discoloration in the lesser curvature of the antrum.

Differential diagnosis

Findings of magnification endoscopy with a narrow-band imaging (M-NBI) of the flat gastric lesion suggested poorly differentiated adenocarcinoma.

Laboratory diagnosis

Laboratory findings revealed anemia and a positive serum anti-*H. pylori* antibody test.

Imaging diagnosis

M-NBI of the lesion showed loss or irregularity of microsurface pattern, irregular microvascular proliferation, and a demarcation line, which suggested poorly differentiated early gastric cancer.

Pathological diagnosis

The final diagnosis was Russell body gastritis (RBG) with substantial infiltration of granulated plasma cells. Although the granulated plasma cells showed kappa light chain restriction and the presence of Dutcher bodies, malignant lymphoma was unlikely partly because of the paucity of the cellular atypia and mitotic activity.

Treatment

Endoscopic submucosal dissection (ESD) was selected for therapeutic diagnosis.

Related reports

Thirty-one previously published cases of RBG, including the authors own, has been reported, and this is the first reported case of RBG with the endoscopic diagnosis of malignant tumor with M-NBI, pathological evaluation of the entire lesion with ESD-obtained specimens, and the presence of Dutcher bodies.

Term explanation

RBG is considered as a unique form of chronic gastritis characterized by infiltration of plasma cells filled with spherical eosinophilic cytoplasmic globules, referred to as Russell bodies.

Experiences and lessons

The endoscopic features of RBG are exclusively inflammatory; however, clinicians should consider RBG as a differential diagnosis in cases where detailed endoscopic examination reveals poorly differentiated early gastric cancer. Pathologists should be aware of the existence of this pathological entity, because histological features of RBG can overlap with those of malignant lymphoma.

Peer-review

Excellent case report article.

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