Case Report

Russell body gastritis

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Summary

Russell body gastritis is caused by an accumulation of plasma cells within the gastric mucosa. These plasma cells are characterized by eosinophilic cytoplasmic inclusions of immunoglobulin which are called “Russell bodies.” We report a case of Russell body gastritis in a 28-year-old male who presented with abdominal pain and rectal bleeding. Endoscopy showed erosions with edema and vascular congestion in the gastric body and antrum. The biopsy showed chronic gastritis with plasma cell infiltration of the lamina propria. Many plasma cells contained cytoplasmic Russell bodies which stained positive for CD138, CD79a, Kappa and lambda light chains. The Russell bodies were negative for pancytokeratin, excluding signet ring cell carcinoma. Russell body gastritis is an uncommon, benign reactive condition.

Key words

Gastric biopsy • Gastritis • Plasma cells • Russell body gastritis

Russell body gastritis is characterized by the presence of plasma cells with eosinophilic cytoplasmic inclusions of immunoglobulin in the gastric lamina propria. The process is thought to be a benign reactive process. Initial case reports suggest the association of this entity with infections, particularly Helicobacter pylori infections.¹² We report a case of a 28-year-old male with a history of human immunodeficiency virus (HIV) who presented with abdominal pain, fatigue and a one week history of rectal bleeding. Complete blood count showed pancytopenia. Computerized tomography (CT) scan showed moderate splenomegaly and mild hepatomegaly. Endoscopy revealed erosions, erythematous mucosa, and vascular congestion in the gastric body and antrum. Microscopic examination showed an accumulation of plasma cells with cytoplasmic Russell bodies in the lamina propria. The plasma cells had eosinophilic cytoplasm and were present in a background of chronic inactive gastritis (Fig. 1). Immunohistochemical stains were positive for CD138, CD79a and they show polyclonal expression of kappa and lambda light chains (Fig. 2). The Russell bodies were negative for pancytokeratin, excluding a signet ring cell carcinoma. Giemsa stain is negative for H. pylori organisms. There are occasional case reports of Russell body gastritis in the literature and some describe an association with H. pylori infection.²⁵ Theories regarding the cause include chronic gastritis leading to either under-secretion of immunoglobulin by plasma cells or over-production of plasma cells with increased formation of Russell bodies.⁵⁶ Some cases have been described in H. pylori negative patients⁹¹³ and occasional cases have been reported in HIV positive patients⁴⁷¹⁰. Shinozaki et al. reported two cases of Epstein-Barr virus positive stomach carcinoma with associated accumulation of Mott cells.⁸ Altindag SD et al. studied 11 cases of Russell body gastritis and observed one case of concomitant carcinoma and plasma cell neoplasm.¹⁴ The presenting symptoms were variable and mostly non-specific.¹⁴ Endoscopic examination can show features of gastritis and erythematous mucosal changes or present as nodular or raised lesion.¹²¹⁵ Follow-up is suggested due to occasional reported cases with associated concurrent neoplastic processes.¹⁴¹⁶ The differential diagnosis includes plasma cell neoplasms, other hematologic neoplasms, histiocytic pro-

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**Fig. 1.** Gastric mucosa with chronic inflammation and presence of plasma cells with intracytoplasmic eosinophilic globules. (A): 100X, (B & C): 200X and (D): Pancytokeratin immunohistochemical stain which is negative in the globules (400x).

**Fig. 2.** These eosinophilic globules are positive for CD79a and CD138 (A & B, 400X, respectively). Lambda and kappa immunohistochemical stains are also positive (C & D, 200X, respectively).
cesses, and signet ring cell adenocarcinoma. Immunohistochemical stains are helpful in excluding the above entities as diagnostic possibilities. Wolkersdörfer et al. described concomitant presence of monoclonal gammopathy of undetermined significance, *H. pylori* infection and Russell body gastritis. Plasma cell neoplasms are monoclonal and they express either kappa or lambda light chains. Our case shows expression of both kappa and lambda light chains which is an expected finding in Russell body gastritis unassociated with a plasma cell neoplasm. There have been case reports of Russell body gastritis with monoclonal expression of kappa or lambda light chain. Histiocytic infiltrates may have a similar histologic appearance with eosinophilic cytoplasm and immunohistochemical stains for CD68 and CD138, a plasma cell marker can aid in the differential diagnosis. Signet ring cell adenocarcinoma may have a similar appearance, with the malignant cells often having nuclear atypia and cytoplasmic mucin secretion. Immunohistochemical stains for keratin are positive in signet ring cell adenocarcinoma and negative in Russell body gastritis. We report a case of Russell body gastritis in an *H. pylori* negative patient who is HIV positive and on HAART (highly active antiretroviral therapy) therapy. Russell body gastritis is considered to be a reactive process.

Conflict of interest statement

None declared.

References