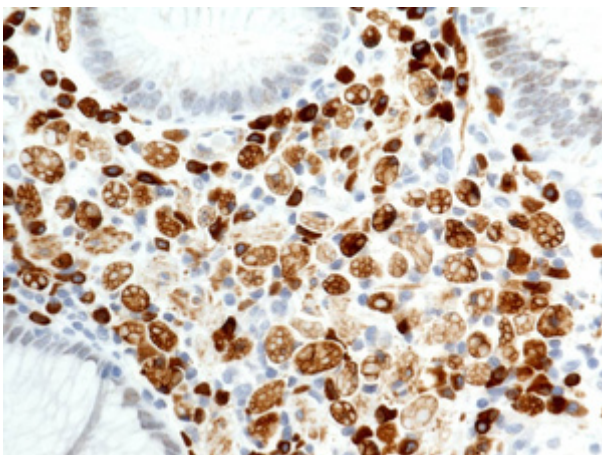
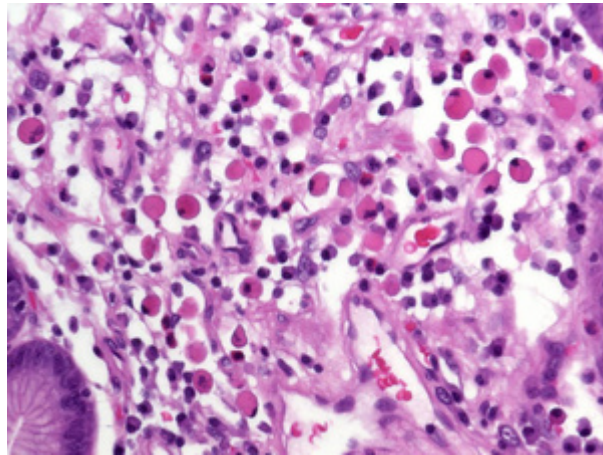
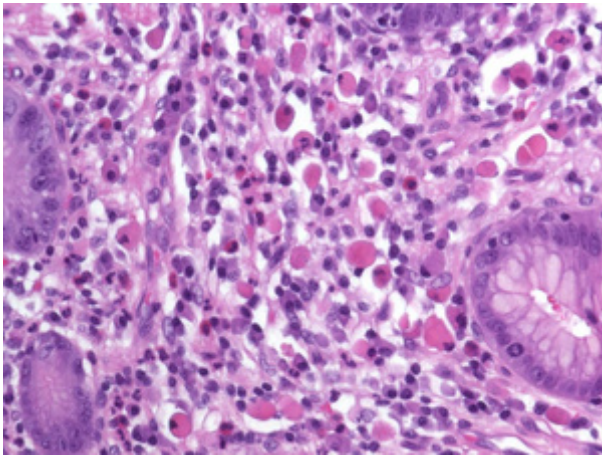
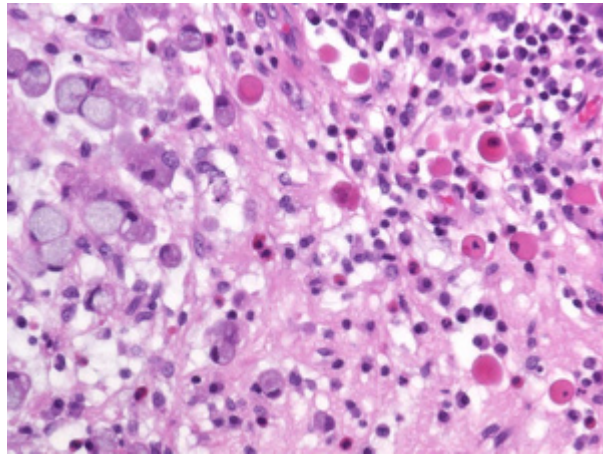
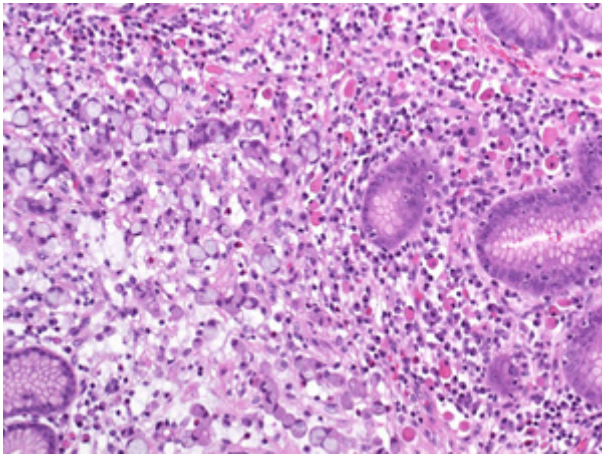


October 2014

Biopsy specimens from a gastric ulcer in a 66-year-old male.

What is your diagnosis?



Diagnosis:

Russell Body gastritis and signet ring cell carcinoma

Comment:

Panels A and B (different magnifications) show diffusely infiltrating atypical cells with hyperchromatic nuclei and prominent intracytoplasmic mucin - with characteristic displacement and moulding of the nuclei. These cells qualify for a diagnosis of signet ring cell carcinoma. But these panels also show chronic-active gastritis

(positive for *Helicobacter pylori*) and, on the right side of both images, a diffuse infiltration of the tunica propria by cells with abundant eosinophilic cytoplasm and eccentric pyknotic nuclei. Higher magnification of these cells (Panels C and D) reveals closely packed hypereosinophilic intracytoplasmic globules. Immunohistochemically, these cells are strongly positive for CD79a (Panel E), kappa and lambda light chains (not shown), thereby identifying them as plasma cells with rounded globular cytoplasmic immunoglobulin ("Russell bodies").

Russell bodies were first described more than a century ago, the term Russell body gastritis, however, was coined by Tazawa and Tsutsumi in 1998. Please note, plasma cells filled with Russell bodies have also been called "Mott cells", and Mott cell gastritis is therefore used as an alternative term. Neil Shepherd coined the term "thesaurocytosis" for these "cells with treasures" (personal communication).

Russell body gastritis is regarded as a response to chronic inflammation caused by *Helicobacter pylori* infection (in particular highly pathogenic genotypes *vacA* and *cagA*) or possibly other infectious agents. It is believed that chronic antigenic stimulation activates the immune response and causes plasma cells to overproduce immunoglobulins. This overstimulation of plasma cells leads to the accumulation of abundant, non-degradable, condensing immunoglobulins inside rough endoplasmic cisternae.

Although Russell body gastritis represents an entirely benign lesion, its occurrence in patients with gastric cancer has been reported also by others: Shinozaki et al. observed prominent Mott cell proliferation in Epstein-Barr virus (EBV)-associated gastric carcinoma and explained this finding as reactive phenomenon caused by virus infection. It is of note that in situ hybridization for EBV-encoded small RNA (EBER) labelled the carcinoma cells, but not the inflammatory cells.

For further reading:

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- › Shinozaki A, Ushiku T, Fukayama M. Prominent Mott cell proliferation in Epstein-Barr virus-associated gastric carcinoma. *Hum Pathol.* 2010; 41: 134-8.
- › Wolf EM, Mrak K, Tschmelitsch J, Langner C. Signet ring cell cancer in a patient with Russell body gastritis-- a possible diagnostic pitfall. *Histopathology.* 2011; 58: 1178-80.
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- › Yoon JB, Lee TY, Lee JS, Yoon JM, Jang SW, Kim MJ, Lee SJ, Kim TO. Two Cases of Russell Body Gastritis Treated by *Helicobacter pylori* Eradication. *Clin Endosc.* 2012; 45: 412-6.

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