Olmesartan-induced collagenous gastritis

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See Online for appendix

A 63-year-old woman presented to our department with a 6-month history of recurrent vomiting and loss of 5 kg in weight. The patient had a medical history of dyslipidaemia and hypertension; she was prescribed rosuvastatin and olmesartan. She had no history of gastrointestinal disorders, recent foreign travel, or infection with *Helicobacter pylori*.

On examination, she was generally unwell and underweight; her blood pressure was 150/88 mm Hg, pulse was 70 beats per min, oxygen saturation was 99%, and BMI was 16 kg/m². We found no lymphadenopathy, and abdominal examination found no masses or ascites.

Laboratory investigations showed normal complete blood count—including a typical eosinophil count; carcinoembryonic antigen, soluble interleukin-2 receptor, and antineutrophil cytoplasmic antibody tests were within normal range. Antinuclear antibody titre was 1:160 (normal value <1:40) and serum immunoglobulin E was 700 IU/mL (typical range 0–100).

An oesophagogastroduodenoscopy (OGD) showed a distended stomach with linear scars and a rough atrophic mucosa that bled easily upon contact (figure). Histopathological examination of a sample obtained by gastric biopsy showed subepithelial collagen deposition with inflammatory cell infiltration—including eosinophils (figure); the small and large intestines showed no specific findings.

At this stage, our working diagnosis included collagenous gastritis, eosinophilic gastritis, and autoimmune gastritis. And based on these working diagnoses, the patient was prescribed a proton pump inhibitor—esomeprazole 20 mg orally—which did not result in any improvement.

Further review of the medical history found that the patient had been taking olmesartan 20 mg daily for the past decade and we considered that it may have been responsible for the patient's symptoms; we therefore decided to stop it.

2 weeks later, the patient stopped vomiting and within 2 months she had gained 7 kg. A repeat OGD, 6 months post-discontinuation, showed a substantial improvement in mucosal inflammation; histopathological examination of samples obtained by biopsies, showed no evidence of residual collagen bands or significant inflammatory cell infiltration (appendix). And at 2 years after discontinuation of the olmesartan no recurrence was seen. Regarding the patient's hypertension, azelnidipine 8 mg daily was prescribed as an alternative treatment.

Collagenous gastritis is characterised by patchy subepithelial collagen bands and inflammatory cell infiltration, including eosinophils; such features can appear in other types of gastritis with similar inflammatory cell infiltration—including eosinophilic gastritis, coeliac disease, and autoimmune gastritis—necessitating a high index of suspicion to make an accurate pathological diagnosis. The precise pathogenesis of collagenous gastritis remains poorly understood and no available treatments are established owing to its rarity; associations with certain medications—including olmesartan as in our patient—have been suggested.

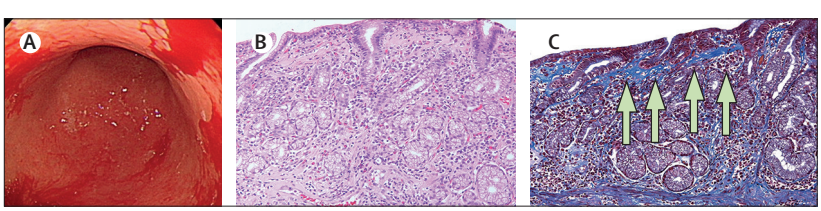

Figure: **Olmesartan-induced collagenous gastritis with inflammatory cell infiltration**
(A) Oesophagogastroduodenoscopy shows a distended stomach with linear scars on a rough, atrophic mucosa that bled easily on contact. Histopathological examination of a sample obtained from gastric biopsy shows (B) inflammatory cell infiltration, including eosinophils, and stromal fibrotic changes (haematoxylin and eosin staining). 100× magnification; and (C) markedly thickened subepithelial collagen bands (arrows; Masson's trichome staining). 100× magnification.

Contributors
AH cared for the patient. AH and NM collected the patient data and drafted and edited the manuscript. MK collected the patient data. KK and YF provided critical comments regarding the manuscript. All authors approved the final version of the manuscript. Written consent for publication was obtained from all patients.

Declaration of interests
We declare no competing interests.