Case Series

Vonoprazan Associated Gastric Mucosal Changes in Bangladeshi Patients: A Case Series

S Dey[1], SMA Hasan[2], SK Bhowmick[3]

Abstract

Background: Vonoprazan, a potassium-competitive acid blocker (P-CAB), has emerged as an effective therapy for gastroesophageal reflux disease (GERD) and Helicobacter pylori eradication. Since its global launch in 2015, several endoscopic mucosal changes associated with its long-term use have been reported, including white globe appearance (WGA), web-like mucosa (WLM), and gastric cracked mucosa (GCM). In Bangladesh, vonoprazan has only recently been introduced, and limited data exist regarding its mucosal effects.

Objective: To report three cases from Bangladesh showing characteristic gastric mucosal changes following vonoprazan therapy.

Result: All three patients developed distinct mucosal changes after vonoprazan use, including longitudinal erythema, web-like mucosa, and hemorrhagic gastric polyps. Mucosal abnormalities regressed or improved following cessation of vonoprazan and initiation of alternative acid-suppressive therapy (proton pump inhibitors or H2 blockers). All these mucosal changes occur at upper part of stomach.

Conclusion: This case series highlights the emergence of vonoprazan-associated gastric mucosal changes in Bangladeshi patients. Clinicians should remain vigilant and consider endoscopic follow-up during long-term P-CAB therapy.

Keywords: Vonoprazan, P-CAB, Web-like mucosa, White globe appearance, Gastric cracked mucosa, GERD, Helicobacter pylori, Bangladesh.

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Introduction:

Potassium-competitive acid blockers (P-CABs) represent a novel class of potent acid-suppressive medications. Vonoprazan, introduced in Japan in 2015, is the most clinically established P-CAB to date and is widely used for treating GERD, Helicobacter pylori infection, and PPI-resistant esophagitis.[1] Unlike PPIs, vonoprazan directly inhibits gastric H+/K+ ATPase without requiring activation under acidic conditions, resulting in rapid and sustained acid suppression.

Long-term use of vonoprazan has been associated with unique gastric mucosal changes. Recent literature describes newly recognized endoscopic findings such as web-like mucosa (WLM), white globe appearance (WGA), stardust gastric mucosa, and gastric cracked mucosa (GCM), which are distinct from changes due to autoimmune gastritis or Helicobacter pylori infection. The mechanisms remain unclear but may involve hypergastrinemia, epithelial remodeling, and microbiota alterations due to profound hypochlorhydria.

In Bangladesh, vonoprazan is relatively new, and data on its adverse mucosal effects are scarce. This case series presents three patients from Bangladesh who developed characteristic gastric mucosal changes during vonoprazan therapy.

Case 1:

A 36-year-old female presented with upper abdominal discomfort and heartburn persisting for the last six months, predominantly occurring after meals. She had a medical history of hypothyroidism and nonalcoholic fatty liver disease. Her medications included thyroxine (50 mcg daily), ursodeoxycholic acid (300 mg twice daily), vonoprazan (20 mg daily), domperidone (10 mg as needed), and losartan (50 mg daily). On clinical examination, her BMI was 31 kg/m². Blood pressure was 140/90 mmHg, heart rate was 110 bpm, and mild hepatomegaly was noted without tenderness. Laboratory tests showed hemoglobin of 13 g/dL, ALT 51 U/L, random blood sugar 6.8 mmol/L, HbA1c 5.4%, and TSH 8 mIU/mL. Ultrasonography confirmed grade II fatty liver disease. Fibroscan revealed a CAP score of 369 dB/m and liver stiffness of 7.4 kPa. An upper GI endoscopy performed six months ago showed Grade B reflux esophagitis and rapid urease test was negative. On follow-up endoscopy, conducted while she was on vonoprazan, longitudinal erythema was noted in the gastric body and fundus. Vonoprazan was discontinued, and esomeprazole was prescribed. Repeat endoscopy performed two months later showed complete resolution of the mucosal changes (Figure 1).

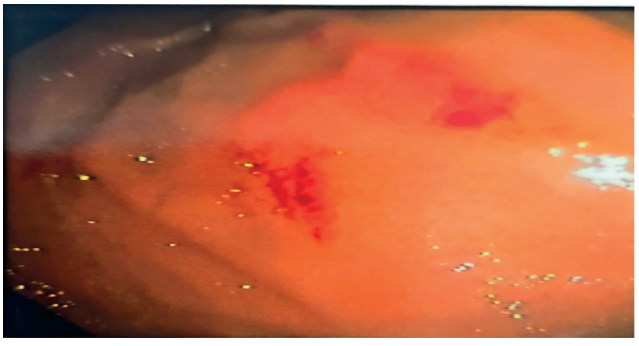

Figure 1: The Stomach shows longitudinal erythema in the fundus after 6 months of vonoprazan intake.

Case 2:

An 84-year-old male, a retired farmer and chronic smoker, presented with a six-month history of productive cough and epigastric pain that worsened with food intake. He denied vomiting, weight loss, hematemesis, or altered bowel habits. On examination, his BMI was 19 kg/m². Vital signs were stable. Respiratory examination revealed prolonged expiration. Laboratory investigations revealed hemoglobin of 12 g/dL and ALT of 34 U/L. Chest X-ray demonstrated hyperinflated lung fields. ECG showed right bundle branch block. Abdominal ultrasonography was normal. An endoscopy performed four months prior had shown mild antral gastritis, and the CLO test was negative. A follow-up endoscopy, done after four months of vonoprazan therapy, revealed a thick, adherent web-like mucosa in the body and fundus of the stomach. The patient had been receiving vonoprazan 20 mg twice daily for acid-related dyspepsia. He was switched to H2-receptor antagonists, and a follow-up endoscopy was scheduled to monitor the mucosal changes (Figure 2).

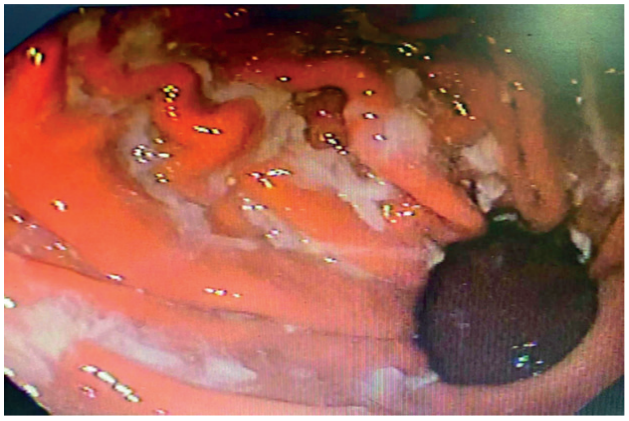

Figure 2: Web-like mucosa in a patient following vonoprazan intake.

Case 3:

A 32-year-old male known case of spondyloarthritis presented with burning upper abdominal pain for six months. He had been on sulfasalazine and intermittent indomethacin for joint symptoms. Physical examination revealed a BMI of 20 kg/m² and grade I joint tenderness. Laboratory investigations showed hemoglobin of 10.5 g/dL, CRP 20 mg/L, and positive HLA-B27. Abdominal ultrasonography was normal. Initial endoscopy revealed antral erosions with multiple duodenal ulcers. He was started on vonoprazan-based triple therapy by a local physician. After four months, follow-up endoscopy showed a bleeding sessile polyp at the fundus. Histopathology confirmed it to be a hyperplastic polyp, and the rapid urease test was negative. Vonoprazan was discontinued, and esomeprazole therapy was initiated. The patient was advised to undergo surveillance endoscopy (Figure 3).

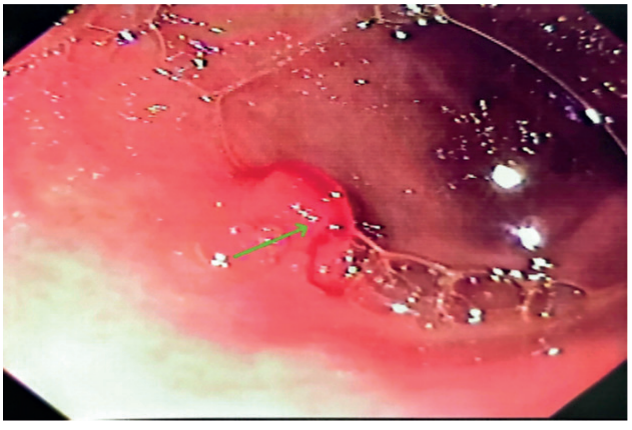

Figure 3: A hemorrhagic gastric polyp in a patient following 4 months of vonoprazan.

Discussion:

Vonoprazan's strong acid suppression efficacy makes it suitable for GERD and *Helicobacter pylori* eradication, particularly in PPI-resistant cases.[2] P-CAB drugs have similar efficacy to PPI in triple therapy.[3,4] Vonoprazan can be used even at a low dose for PPI-refractory reflux esophagitis.[5,6] However, with its increased use, novel gastric mucosal findings have emerged. These include web-like mucosa, described as a spider web or clot-like mucus pattern in the upper stomach and reported in 19 to 21 percent of users; white globe appearance, consisting of white, globe-like protrusions; gastric cracked mucosa, characterized by linear mucosal breaks possibly related to chronic hypochlorhydria; stardust gastric mucosa, a white granular appearance and hemorrhagic gastric polyp more commonly seen in long-term users and females patients.[7-11]

The pathogenesis of these mucosal changes is not fully understood but may involve hypergastrinemia-induced epithelial proliferation, hypochlorhydria-related alterations in gastric flora, and mucosal remodeling.[12-14] Notably. most of these mucosal changes resolve upon discontinuation of vonoprazan and substitution with PPIs or H_2 blockers. Our case series is consistent with these international findings, with patients developing reversible mucosal changes following vonoprazan therapy.

Conclusion:

Vonoprazan-associated mucosal changes are now being recognized globally. Here we have documented these changes for the first time in Bangladeshi patients. So endoscopic surveillance may be warranted in long-term users of vonoprazan. Further multicentric, prospective studies are necessary to define pathogenesis, risk factors, and optimal management strategies in this regard.

Conflicts of Interest: There is no conflict of interest.

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