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From Refractory Esophagitis to Scurvy: A Diagnostic Challenge

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Abstract

Background: Vitamin C deficiency is an underrecognized but potentially reversible cause of impaired gastrointestinal mucosal healing. Although proton pump inhibitors (PPIs) are highly effective in erosive reflux disease, persistent ulcerative esophagitis despite optimized therapy should prompt investigation for mechanisms of defective mucosal repair beyond uncontrolled acid exposure. The implication of scurvy in refractory peptic esophagitis remains rarely reported, but the biological plausibility is strong given the central role of ascorbic acid in collagen synthesis, extracellular matrix integrity, angiogenesis, and antioxidant protection. **Aim:** The aim of this report is to highlight vitamin C deficiency as a reversible and potentially underdiagnosed cause of persistent ulcerative esophagitis, and to emphasize the diagnostic value of a structured etiological work-up in apparently refractory disease. **Case Presentation:** A 54-year-old man was followed for recurrent and persistent ulcerative esophagitis despite optimized PPI therapy. Several elements prompted investigation for vitamin C deficiency: the unusual severity of the lesions, their persistence and recurrence despite adequate acid suppression, the discrepancy between appropriate treatment and the absence of expected mucosal healing, and a context of fatigue, asthenia, alcohol consumption, and undernutrition in a socially precarious setting. An extended etiological work-up was therefore undertaken, including plasma vitamin C measurement, which showed a pretreatment level below the normal range (<23 μmol/L). Differential diagnoses of refractory esophagitis, particularly eosinophilic esophagitis, were explored, including esophageal biopsies. Oral ascorbic acid supplementation at 1 g/day was prescribed for three months together with pantoprazole at double dose. Follow-up upper gastrointestinal endoscopy showed marked regression of the lesions, with only a small residual distal ulceration corresponding to Los Angeles grade A esophagitis. The chronological relationship between correction of a documented deficiency and clear clinico-endoscopic improvement supports a possible contributory role of vitamin C deficiency in the persistence of the esoph-

ageal lesions. **Conclusion:** In patients with recurrent or PPI-refractory peptic esophagitis, vitamin C deficiency should be considered once common differential diagnoses have been excluded. Identifying and correcting this deficiency may reveal a reversible mechanism of persistent mucosal injury.

Subject Areas

Gastroenterology & Hepatology

Keywords

Esophagitis, Proton Pump Inhibitors, Vitamin C Deficiency, Scurvy, Case Report

1. Introduction

Peptic esophagitis related to gastroesophageal reflux disease is a common disorder, and its endoscopic severity is conventionally graded using the Los Angeles classification [1]. Proton pump inhibitors remain the cornerstone of therapy and achieve mucosal healing in most patients with erosive reflux disease within a few weeks [2]. However, a subset of patients develops persistent or recurrent esophagitis despite optimized therapy, raising the possibility of refractory disease and requiring reassessment of adherence, drug exposure, reflux burden, competing diagnoses, and mechanisms of impaired mucosal repair [1]-[4].

Among nutritional factors that may alter tissue restitution, vitamin C deficiency deserves particular attention. Ascorbic acid is essential for collagen hydroxylation, extracellular matrix assembly, fibroblast function, angiogenesis, and oxidative stress control, all of which are fundamental to wound healing [3] [4]. Contemporary literature confirms that scurvy persists in modern practice, often with incomplete or atypical presentations, and remains underrecognized because clinicians may not suspect it in the absence of classic gingival or cutaneous signs [5]-[7].

The gastrointestinal relevance of vitamin C deficiency is biologically plausible yet probably underestimated. In damaged digestive mucosa, where continuous repair is required to counterbalance chemical and mechanical injury, deficiency of a key cofactor in connective tissue synthesis may contribute to chronic non-healing [3] [4]. This hypothesis becomes even more relevant in patients exposed to long-term PPI therapy, since pharmacologic acid suppression may reduce dietary vitamin C bioavailability by altering gastric chemistry and the stability of ascorbic acid [8]-[10].

This case report describes persistent severe ulcerative esophagitis in a patient who failed to heal as expected despite optimized PPI therapy. The decision to investigate scurvy was driven not by a stereotypical presentation of classic systemic deficiency, but by the endoscopic behavior of the disease itself: unusual severity, chronicity, and failure of mucosal restitution despite apparently adequate acid suppression. The combination of documented vitamin C deficiency, exclusion of

key differential diagnoses, and marked improvement after ascorbic acid supplementation supports a causal implication of scurvy in refractory ulcerative esophagitis.

2. Case Presentation

A 54-year-old man was followed in our gastroenterology department for recurrent and persistent ulcerative peptic esophagitis despite optimized proton pump inhibitor therapy. He reported fatigue and asthenia, and the clinical context included undernutrition in a socially precarious setting as well as alcohol consumption. At reassessment, upper gastrointestinal endoscopy showed severe erosive esophagitis classified as Los Angeles grade D, with confluent circumferential ulcerations involving the distal esophagus (**Figure 1**). The most remarkable feature of the case was the persistence and recurrence of severe ulcerative lesions despite what was considered adequate acid suppression.

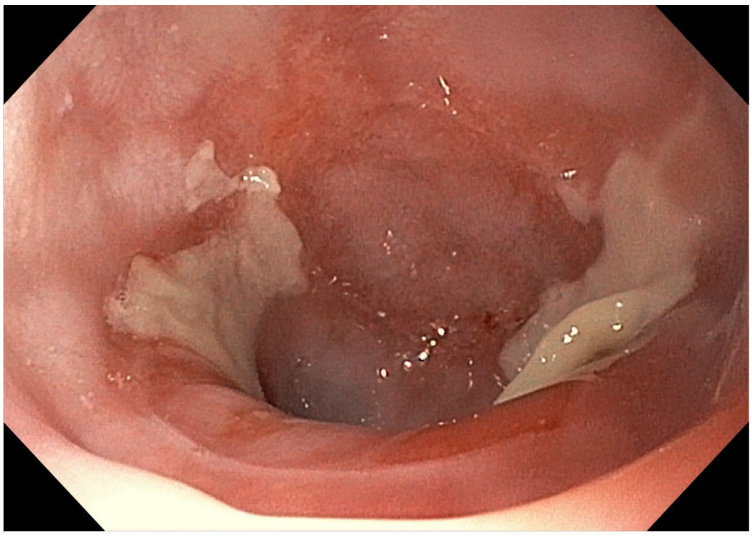


Figure 1. Upper gastrointestinal endoscopy showing severe erosive esophagitis, classified as Los Angeles grade D.

The available medical records did not identify another immediately obvious explanation for this refractory course. No specific mucocutaneous abnormalities suggestive of scurvy were documented on physical examination. Nevertheless, several arguments prompted investigation for vitamin C deficiency. First, the severity of the lesions was disproportionate to the expected course under prolonged optimized PPI therapy. Second, mucosal healing failed to occur despite continuation of acid suppression, suggesting that the problem was not simply uncontrolled acidity but a broader defect in tissue restitution. Third, the ulcerative phenotype raised the possibility of impaired connective tissue repair. Fourth, among reversible causes of non-healing mucosal lesions, nutritional deficiency represented a plausible and clinically actionable hypothesis.

On this basis, an extended etiological work-up was undertaken, including

plasma vitamin C measurement, which showed a pretreatment level below the normal range (<23 µmol/L). At the same time, differential diagnoses of refractory esophagitis were reassessed. Esophageal biopsies were performed during the diagnostic evaluation, and eosinophilic esophagitis was excluded. Other relevant differential diagnoses were explored during the etiological work-up, and no competing etiology better accounted for the severity and persistence of the ulcerative endoscopic pattern. The retained diagnosis was persistent ulcerative peptic esophagitis associated with vitamin C deficiency.

Oral ascorbic acid supplementation at 1 g/day was initiated for three months together with pantoprazole at double dose. Follow-up upper gastrointestinal endoscopy showed marked mucosal improvement, with regression to a small residual distal esophageal ulceration corresponding to Los Angeles grade A esophagitis (Figure 2), without stricture or other complication. This substantial clinico-endoscopic improvement after correction of a documented deficiency suggests that vitamin C deficiency may have contributed to the persistence of the esophageal lesions. No adverse event related to supplementation was reported.

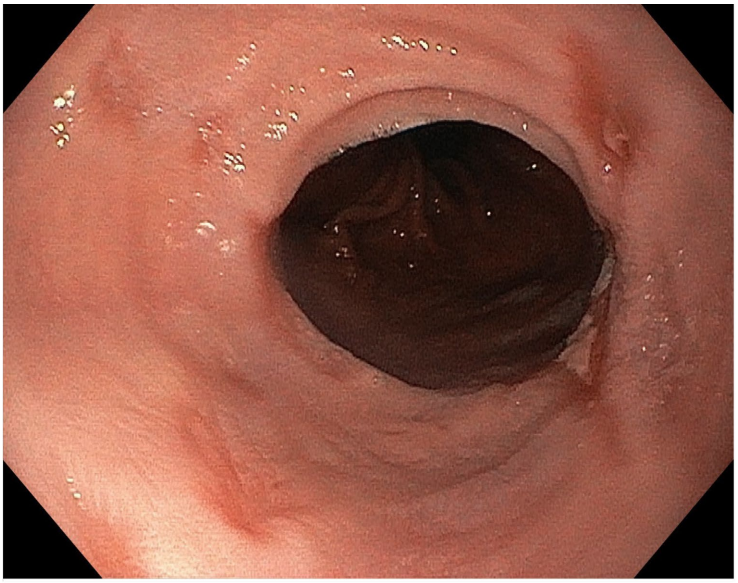


Figure 2. Follow-up upper gastrointestinal endoscopy after three months of oral vitamin C supplementation showing grade A peptic esophagitis.

3. Discussion

This case highlights a diagnostic and pathophysiological point that deserves greater attention in gastroenterology: vitamin C deficiency may impair esophageal mucosal healing and contribute to persistent ulcerative esophagitis despite optimized acid suppression. The importance of this observation lies not simply in the coexistence of hypovitaminosis C and esophagitis, but in the coherence of the clinical sequence: unusually severe lesions, an unexpectedly refractory course, exploration of relevant alternative diagnoses, biological confirmation of deficiency, and improvement after targeted correction.

In most modern adult reports, scurvy is still diagnosed late because constitutional, dermatologic, musculoskeletal, or mucosal manifestations are either nonspecific or incompletely expressed. In the present case, the key clue was the mismatch between therapeutic adequacy and endoscopic outcome. A patient receiving optimized PPI therapy would usually be expected to demonstrate substantial healing of erosive reflux disease over time. Persistent grade D ulcerative esophagitis under such conditions therefore raised the possibility of an underlying failure of tissue repair rather than insufficient acid suppression alone. This diagnostic reasoning is supported by current knowledge of vitamin C physiology. Ascorbic acid is required for hydroxylation of proline and lysine residues during collagen synthesis, thereby ensuring the structural stability and tensile strength of connective tissue. It also participates in angiogenesis, fibroblast proliferation, epithelial repair, and antioxidant defense, all of which are essential for wound healing. In a chronically inflamed esophageal mucosa exposed to reflux-related injury, deficiency of such a cofactor may plausibly perpetuate ulceration by preventing effective restitution even when acid injury is being pharmacologically reduced.

The literature on vitamin C and tissue repair provides additional support for this interpretation. A 2022 systematic review concluded that vitamin C plays a clinically relevant role in tissue healing and that supplementation improved healing outcomes in several settings, particularly when deficiency or high tissue demand was likely. Although esophageal-specific data remain limited, the underlying biological mechanisms are directly applicable to upper digestive mucosa, where collagen remodeling, vascular integrity, and oxidative stress control are central to lesion repair. Thus, in this patient, vitamin C deficiency should not be regarded merely as a biochemical epiphenomenon but as a possible contributor to persistent mucosal injury.

Another major strength of the case is the diagnostic work-up directed at differential diagnoses. In PPI-refractory esophagitis, eosinophilic esophagitis is among the most important competing etiologies and must be considered systematically. In the present case, this possibility was specifically explored by esophageal biopsies during the etiological assessment. Once eosinophilic esophagitis and other retained differentials have been reasonably excluded, the significance of a documented and correctable nutritional deficiency becomes stronger.

The therapeutic response further supports the hypothesis of a contributory role. In contemporary case reports of adult scurvy, improvement after ascorbic acid replacement is often rapid and diagnostically informative, even when the baseline presentation is atypical. In the present observation, correction of vitamin C deficiency was followed by marked endoscopic regression from Los Angeles grade D ulcerative esophagitis to grade A disease.

Long-term PPI exposure may also provide an important mechanistic background. Human data have shown that omeprazole can reduce plasma vitamin C concentrations and impair the bioavailability of dietary ascorbic acid [8]. This phenomenon is thought to be related to elevation of intragastric pH and reduced

stability of vitamin C in less acidic conditions. More recent reviews on PPI safety continue to recognize nutrient-related consequences of prolonged acid suppression, including effects on vitamin C handling in predisposed patients. Accordingly, in a patient with persistent esophageal injury, chronic PPI exposure may paradoxically treat acid-mediated damage while simultaneously creating a biochemical environment less favorable to optimal ascorbic acid availability and mucosal regeneration [8].

The present case also broadens the clinical spectrum of gastrointestinal scurvy. Published reports more often emphasize bleeding stigmata, perifollicular purpura, gingival abnormalities, anemia, constitutional symptoms, or musculoskeletal complaints. Esophageal involvement is rarely foregrounded, although one case report described severe scurvy in a patient with eosinophilic esophagitis and restricted intake, illustrating that significant upper gastrointestinal disease can coexist with profound vitamin C deficiency. The originality of the current observation is that persistent ulcerative esophagitis itself served as the major clue leading to diagnosis. This suggests that in some patients, non-healing digestive mucosal disease may be one of the clinically relevant manifestations of vitamin C deficiency even in the absence of classic systemic semiology [10].

The clinico-biological course supports a possible relationship between vitamin C deficiency and persistent ulcerative esophagitis in this patient. The lesions remained severe and non-healing despite optimized PPI therapy, eosinophilic esophagitis and other relevant competing diagnoses were explored, plasma vitamin C deficiency was documented, and marked clinico-endoscopic improvement occurred after targeted ascorbic acid supplementation given together with pantoprazole. This response to supplementation, together with the established role of vitamin C in collagen synthesis, epithelial repair, and antioxidant protection, supports the hypothesis that deficiency contributed to defective esophageal mucosal healing rather than representing an incidental biological finding.

4. Conclusion

Vitamin C deficiency should be considered in patients with severe, recurrent, or PPI-refractory peptic esophagitis when lesion persistence suggests impaired mucosal healing rather than uncontrolled acid exposure. The most compelling clues are disproportionate lesion severity, absence of expected healing under optimized therapy, exclusion of usual differential diagnoses such as eosinophilic esophagitis, and the presence of a biologically plausible reversible defect in tissue repair. In such situations, targeted vitamin C supplementation may do more than accompany improvement; it may correct one of the principal drivers of disease persistence.

Patient Perspective

The patient reported concern because the esophagitis persisted despite ongoing PPI therapy. Oral vitamin C supplementation was well tolerated, and the marked endoscopic improvement at follow-up was experienced as reassuring.

Statement of Informed Consent

Written informed consent was obtained from the patient for publication of this case report and the accompanying clinical material.

Author Contributions

Nihad Lachkar: Conceptualization, clinical data collection, literature review, manuscript drafting, and preparation of the final manuscript.

Maha Lahkim Bennani: Clinical data interpretation, critical revision of the manuscript, and intellectual contributions.

Yosra Aouni: Scientific supervision, critical revision of the manuscript, and final approval of the manuscript.

Conflicts of Interest

The authors have no conflicts of interest to declare.

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