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Case Study

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EoE: Evidence of Healing and Possible Sustained Untreated Remission

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Clinical Observation

The course of eosinophilic oesophagitis (EoE) in this case raises an important question: how should suppression of disease activity, healing of the oesophageal mucosa, and possible sustained untreated remission be distinguished? The EoE had previously required active swallowed topical corticosteroid therapy, initially with budesonide orodispersible treatment and subsequently with swallowed fluticasone (Flixotide) each evening. This therapy was directed at controlling eosinophilic inflammation within the oesophageal mucosa, rather than merely relieving symptoms. There has now been an approximately eight-week period during which no EoE-specific swallowed corticosteroid therapy has been required and no recognisable flare-up has occurred. The previous symptomatic pattern has not apparently recurred. This raises the possibility that the oesophagus is maintaining remission after treatment withdrawal and, more cautiously, that a degree of sustained treatment-independent recovery may be occurring. This observation is clinically encouraging, but it must be interpreted in the context of the known biology and natural history of EoE.

1. EoE as a Chronic but Potentially Reversible Inflammatory Disorder

EoE is classified as a chronic, immune-mediated inflammatory disease of the oesophagus. Contemporary guidelines therefore generally recommend maintenance of an effective therapy after remission has been achieved, rather than assuming that successful induction treatment has eradicated the disease [1]. Chronicity, however, should not be equated with irreversibility. The inflammatory component of EoE can heal substantially. Effective treatment can markedly reduce mucosal eosinophilia, restore epithelial integrity, reduce oedema and inflammatory activity, improve endoscopic abnormalities, and modify some of the tissue-remodelling processes associated with prolonged disease [2,3]. This distinction is central to the present case. Swallowed corticosteroid therapy does not merely mask symptoms while leaving the underlying inflammation unchanged. In treatment responders, the inflammatory process itself can be substantially suppressed and the oesophageal epithelium can undergo genuine biological repair.

Budesonide orodispersible therapy provides particularly strong evidence for this effect. In a randomised, placebo-controlled trial of adults with active EoE, 93% of patients receiving budesonide orodispersible tablets achieved histological remission after six weeks, compared with none receiving placebo; complete clinical and histological remission occurred in 58% at six weeks and increased further with continued treatment [2]. Swallowed fluticasone likewise reduces oesophageal eosinophilia and can induce histological remission. The sequence observed in this case is therefore biologically plausible:
active eosinophilic inflammation → topical corticosteroid suppression → marked reduction in eosinophils → epithelial healing and reduced inflammatory signalling → clinical remission → persistence of remission after treatment withdrawal.
The unresolved issue is the final step: whether persistence after withdrawal represents temporary post-treatment remission or a more durable, treatment-independent change in disease activity.

2. Evidence that Oesophageal Healing can Extend Beyond Symptom Control

Studies of oesophageal remodelling show that successful anti-inflammatory treatment can produce changes extending beneath the surface epithelium. Aceves and colleagues demonstrated that patients responding to topical budesonide had reductions in subepithelial fibrosis, transforming growth factor-beta signalling, and vascular activation [3]. Other work has similarly shown that epithelial-mesenchymal transition, a process implicated in fibrotic remodelling, can diminish with successful EoE treatment [4]. These findings are important because they demonstrate that the oesophagus retains meaningful capacity for structural recovery when eosinophilic inflammation is successfully controlled. The degree of reversibility depends partly on disease stage. Inflammatory abnormalities such as oedema, exudates, epithelial injury, and furrowing appear considerably more reversible than

longstanding fixed rings, stenoses, or established fibrotic strictures. Prolonged untreated EoE is associated with progressively greater fibrostenotic disease; in one major cohort, oesophageal strictures were present in approximately 17% of patients with a diagnostic delay of 0–2 years, compared with approximately 71% when symptoms had preceded diagnosis by more than 20 years [5]. Accordingly, where the predominant disease process is inflammatory rather than advanced fibrostenotic disease, substantial recovery of oesophageal tissue is biologically credible.

3. Interpreting the Present Eight-Week Treatment-Free Period

The current eight-week period without EoE-specific medication and without a recognised flare-up provides evidence of clinical treatment-free remission. Importantly, treatment withdrawal has not produced an immediate return to the previous symptomatic state. It does not, however, establish permanent disease resolution. The most informative comparison is a randomised maintenance study involving adults who had already achieved clinical and histological remission with budesonide. Participants were then assigned either to continued budesonide or to placebo. Among those switched to placebo, the median time to relapse was 87 days—approximately 12.5 weeks. At 48 weeks, only 4.4% of the placebo group remained in persistent remission, compared with approximately three-quarters of those continuing budesonide [6].

The present eight-week observation therefore remains within a period during which many patients who ultimately relapse would still be expected to remain clinically well. A second prospective study followed patients who had achieved a histological response with either oral viscous budesonide or swallowed fluticasone and then discontinued treatment. Fifty-seven percent experienced recurrent symptoms within one year. The median time to symptom recurrence was 244 days, or approximately eight months; when symptoms returned, 78% had histological relapse [7]. Long-term observational evidence points in the same direction. Greuter and colleagues identified deep remission—defined as sustained clinical, endoscopic, and histological remission—in only 33 of 351 patients treated with swallowed topical corticosteroids. Following treatment withdrawal, most subsequently relapsed, indicating that even deep treatment-induced remission does not usually constitute permanent eradication of EoE [8].

Eight symptom-free weeks are therefore significant and encouraging, but remain too early to distinguish confidently among:

- continuing post-treatment remission;
- asymptomatic microscopic recurrence; and
- genuinely prolonged treatment-independent remission.

4. The Symptom–Histology Disconnect

A particular difficulty in evaluating EoE is that symptoms do not correlate reliably with biological disease activity. In a prospective study of 269 adults with EoE, symptom scores had only modest ability to distinguish histological or endoscopic remission. The investigators concluded that absence of symptoms could not reliably be used to infer absence of biological EoE activity [9]. This has direct implications for the present case.

Clinical remission may reflect continuing histological healing of the oesophageal epithelium. Alternatively, low-grade eosinophilic inflammation may already be returning while remaining below the threshold required to produce recognisable symptoms.

Accordingly: absence of symptoms is evidence of clinical remission, but biopsy is required to establish whether histological remission is also persisting.

This distinction is especially important when the objective is to investigate possible self-correction or sustained untreated remission, rather than simply satisfactory symptom control.

5. Molecular Memory Despite Histological Remission

Recent molecular evidence adds a further layer to this question. Ruffner and colleagues examined oesophageal biopsies from adults and children with EoE who were already in histological remission. Remarkably, EoE-associated abnormalities in gene expression remained detectable even in patients whose biopsy specimens contained zero eosinophils per high-power field [10]. This suggests that an oesophagus can reach very deep histological remission while retaining a molecular signature of its previous inflammatory disease.

This concept may help explain why EoE frequently relapses after apparently successful treatment. The epithelium may heal sufficiently to appear microscopically normal by conventional eosinophil counting while retaining an altered molecular or epigenetic state that predisposes it to renewed inflammation when relevant immune or environmental stimuli recur. For the present case, this is an important caution against equating apparent healing with complete biological erasure of EoE.

At the same time, it reinforces the reality that substantial healing can occur. An oesophagus with no active eosinophilic inflammation is in genuine biological remission, even if a residual molecular predisposition remains.

6. Is Spontaneous or Sustained Untreated Improvement Possible?

Although established symptomatic EoE usually behaves as a relapsing chronic disorder, its natural history is not identical in every patient. Cohorts involving asymptomatic oesophageal eosinophilia and minimally symptomatic EoE demonstrate that spontaneous improvement is biologically possible. Suzuki and colleagues followed untreated individuals with asymptomatic oesophageal eosinophilia for more than five years. Among 21 patients receiving no medication, seven (33.3%) demonstrated spontaneous endoscopic and pathological improvement, 12 remained unchanged, and two worsened [11]. A separate long-term study of untreated asymptomatic or minimally symptomatic oesophageal eosinophilia found that localised disease could remain stable or gradually improve over approximately seven years of observation [12]. These populations are not equivalent to a patient with previously symptomatic, treatment-requiring EoE.

Nevertheless, they demonstrate that eosinophilic oesophageal inflammation is biologically capable of spontaneous attenuation in at least some circumstances.

It would therefore be too categorical to state that self-correction of EoE is impossible.

A more defensible evidence-based conclusion is that permanent spontaneous remission appears uncommon in established symptomatic EoE, but prolonged or spontaneous reduction in disease activity can occur and warrants recognition when objectively documented.

7. A Proposed Interpretation of the Present Case

The current clinical course is most consistently interpreted as continuing treatment-free clinical remission following successful anti-inflammatory therapy.
One plausible biological sequence is:
EoE inflammatory activation
↓
budesonide treatment
↓
major reduction in mucosal eosinophilia
↓
swallowed fluticasone maintenance/suppression

↓
progressive epithelial repair and reduced inflammatory signalling
↓
treatment withdrawal
↓
continued symptom-free oesophageal function for approximately eight weeks.

Nothing in this sequence requires active corticosteroid to remain within the oesophagus. Once inflammation has been suppressed and epithelial healing has occurred, the repaired state may persist for some time after the medication itself has cleared.

The clinically important uncertainty is whether the immune processes responsible for EoE will eventually re-establish themselves. Three hypotheses therefore remain open.

Hypothesis 1: Continuing post-treatment remission.
Previous topical corticosteroid therapy has induced substantial mucosal healing, and that healed state is persisting temporarily without medication. On current evidence, this is the most conservative interpretation.

Hypothesis 2: Sustained untreated remission.
Treatment has been followed by a more durable resetting of oesophageal inflammatory activity, such that histological as well as clinical remission persists for many months without further pharmacological intervention. This would be less common, but remains biologically credible.

Hypothesis 3: Silent histological recurrence.
Symptoms remain absent while eosinophilic inflammation has begun to return microscopically. Because symptom–histology correlation is imperfect, this possibility cannot be excluded clinically.

8. How Sustained Untreated Remission could be Demonstrated

The present observation would become substantially stronger if documented prospectively.
The most informative assessment would be an upper gastrointestinal endoscopy with systematic oesophageal biopsies during the treatment-free period, if clinically appropriate and agreed by the treating gastroenterologist.
Evidence of:

- minimal or absent eosinophilic infiltration;
- normalisation of the oesophageal epithelium;
- resolution or marked reduction of oedema, exudates, and furrowing;
- absence of progressive rings or stenosis; and
- persistent clinical absence of dysphagia or food impaction

would demonstrate that treatment-free remission was occurring not only symptomatically, but also histologically and endoscopically.

Duration would then become an important part of the evidence. Eight weeks establishes an initial observation. Persistence beyond approximately three months would extend the observation beyond the median placebo relapse interval reported in the budesonide maintenance trial.[6] Persistence at six months would be increasingly notable. Demonstration of clinical, endoscopic, and histological remission at 12 months without EoE-specific therapy would provide substantially stronger evidence of sustained untreated remission.

Even then, sustained untreated remission would be scientifically preferable to cure, because recurrence after prolonged remission remains biologically possible.

9. Case-Study Conclusion

This case provides a clinically important prospective observation concerning recovery from EoE. Following a period in which eosinophilic oesophageal disease required swallowed topical corticosteroid therapy, there has now been an approximately eight-week interval without EoE-specific medication and without recognised clinical recurrence. The medical literature provides strong evidence that topical budesonide and fluticasone can produce genuine histological and epithelial healing rather than merely symptomatic suppression. Reduction in eosinophilia can also be accompanied by reversal of elements of oesophageal remodelling. Accordingly, the current symptom-free interval can reasonably be interpreted as evidence that substantial oesophageal healing may have occurred. It cannot yet be interpreted as evidence that EoE has permanently disappeared. Most patients with established EoE eventually develop recurrent disease activity after withdrawal of successful treatment, and histological recurrence may precede, or occur without, recognisable symptoms. Nevertheless, spontaneous attenuation of oesophageal eosinophilic inflammation has been

documented in longitudinal cohorts, and the possibility of sustained untreated remission should not be excluded.

The appropriate working conclusion is:
The oesophagus appears clinically to be maintaining remission following withdrawal of swallowed corticosteroid therapy. This is compatible with genuine post-treatment mucosal healing. Whether the observation represents temporary remission or a less common process of sustained treatment-independent recovery cannot yet be determined. Continued longitudinal observation, particularly with off-treatment endoscopic and histological assessment where clinically appropriate, would allow this distinction to be tested objectively.

The significance of the present case therefore lies not in asserting that EoE has self-corrected, but in prospectively documenting whether a previously treatment-dependent inflammatory disorder is now showing increasingly prolonged evidence of clinical—and potentially histological—treatment-free remission.

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