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Case Report

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Gastroautonomic Dysfunction, Pyloric Dysmotility, and Eosinophilic Esophagitis: A Narrative-Clinical Integration of Motility Restoration and Targeted Anti-Eosinophilic Therapy

Bruce H Knox*

Independent Scholar, Auckland, New Zealand

***Corresponding Author**
Bruce H Knox, Independent Scholar, Auckland, New Zealand.

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Abstract

Gastrointestinal symptom complexes involving dysphagia, nocturnal oesophageal pain, reflux-like symptoms, and impaired gastric emptying are frequently approached as isolated pathological entities. However, emerging evidence supports an integrated gastroautonomic axis, in which autonomic dysfunction, allergic inflammation, and motility impairment interact dynamically and bidirectionally. This paper presents a narrative-informed clinical reconstruction, uniquely conveyed through musical lyrics authored by the patient and serving as structured longitudinal clinical data [1]. The case integrates Eosinophilic Esophagitis (EoE), gastroautonomic dysfunction, and pyloric sphincter dysmotility, demonstrating that symptom evolution reflects a complex neuro-immune-mechanical interaction. The findings indicate that effective therapeutic intervention requires a combined strategy involving topical corticosteroids (Fluticasone, Budesonide/Jorveza) and pyloric-directed intervention via Gastric Peroral Endoscopic Myotomy (G-POEM), thereby supporting a unified model of disease rather than compartmentalised treatment pathways.

Keywords: Eosinophilic Esophagitis (EoE), Gastroautonomic Dysfunction, Gastric Peroral Endoscopic Myotomy (G-POEM), Pyloric Dysfunction, Gastroparesis, Autonomic Nervous System, Aeroallergen-Triggered Inflammation, Narrative Medicine

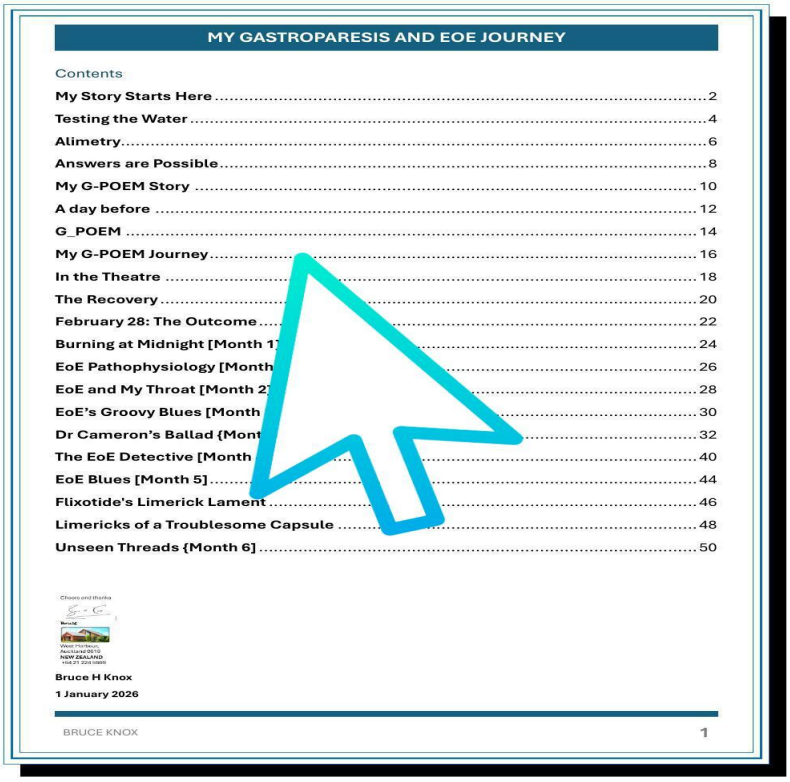
1. Introduction

1.1. Musical Narrative as Clinical Data

This work is intentionally constructed and presented through **musical lyrical compositions**, which serve not merely as artistic expression but as a structured and temporally organised form of narrative medicine and phenomenological clinical documentation [1]. Unlike conventional symptom reporting, the lyrical framework enables the capture of experiential nuance, including temporal progression, sensory perception, autonomic awareness, and therapeutic response, in a manner that is both sequential and internally coherent.

Within this framework, the opening lyric —
"My throat's a blaze, it screams tonight / A quiet war no end in sight" [1]
— functions as a clinically meaningful descriptor of inflammatory dysesthesia, encapsulating both the intensity and persistence of symptoms associated with eosinophilic esophagitis, while simultaneously signalling that the pathology extends beyond a purely structural disorder.

As the lyrical narrative unfolds across successive temporal phases, it becomes evident that the condition cannot be adequately explained by isolated models of reflux disease, mucosal inflammation, or food-triggered allergy alone. Rather, the narrative reveals a multi-system disorder that necessitates interpretation through an integrated lens combining gastroenterology, immunology, neurophysiology, and autonomic science. In this context, the lyrical compositions are treated as primary qualitative clinical data, enabling the alignment of subjective experience with established and emerging pathophysiological frameworks.



MY GASTROPARESIS AND EOE JOURNEY

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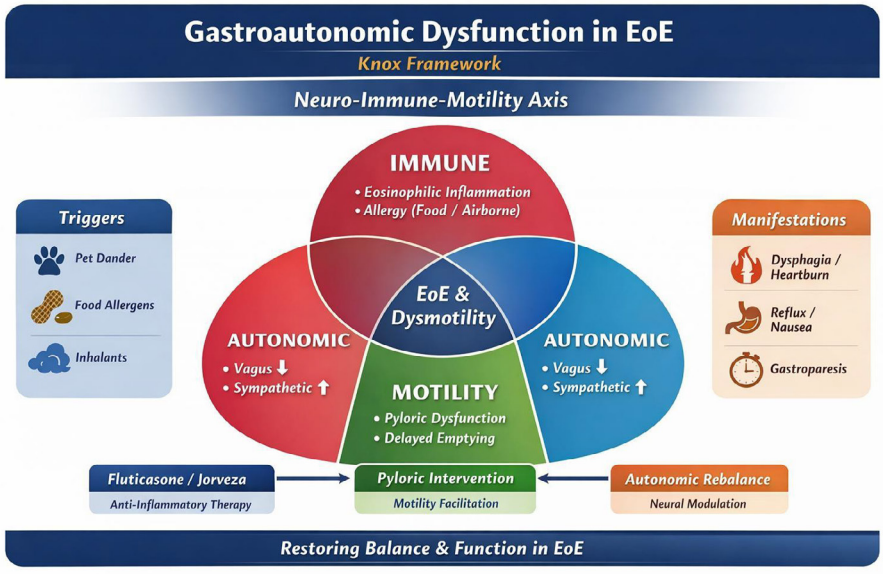
1.2. Eosinophilic Esophagitis: Immunological Foundation

The immunopathological basis of the condition is explicitly articulated within the narrative:
"Eosinophils flood my oesophagus… a response to allergens that my body misidentifies as threats" [1].

This description aligns with current understanding of eosinophilic esophagitis as a chronic, antigen-driven, T-helper type 2 (Th2)–mediated inflammatory disease, characterised by cytokine signalling involving interleukin-5 and interleukin-13, and eosinophil recruitment via eotaxin-3 [2]. These immunological processes lead to epithelial barrier dysfunction, tissue remodelling, and eventual fibrotic change [3,4].

The experiential correlate of this pathology is captured in the lyric:
"Every swallow a wound that won't heal" [1]
which reflects ongoing mucosal injury, impaired epithelial repair, and the persistence of inflammatory insult.



1.3. Gastroautonomic Dysfunction

A central contribution of this work is the identification of autonomic nervous system involvement as a key driver of symptom persistence and disease progression. This is articulated within the narrative:

"*Autonomic dysfunction… creates now motility function" [1]*

and more explicitly:

"The vagus nerve weakens, the sympathetic takes hold" [1]

These descriptions correspond to reduced parasympathetic (vagal) tone and relative sympathetic dominance, resulting in impaired gastrointestinal motility, reduced peristaltic coordination, and altered visceral sensitivity [5]. This autonomic imbalance provides a unifying explanation for several otherwise incongruent clinical observations, including symptom persistence despite reflux control, variability independent of dietary triggers, and nocturnal exacerbation.

1.4. Symptom Reinterpretation: Beyond the Esophagus

A critical diagnostic shift emerges within the narrative:

"But it's not my throat, it's deeper down below / Where food should gently flow" [1]

This reflects the phenomenon of symptom mislocalisation, in which oesophageal discomfort originates from distal gastrointestinal dysfunction. This is well recognised in conditions such as gastroparesis and autonomic neuropathy, where afferent signalling pathways obscure anatomical origin [6,7]. This insight represents a key turning point in the clinical reasoning process, redirecting attention toward gastric motility and pyloric function.

1.5. Pyloric Dysfunction and Motility Failure

The narrative provides explicit confirmation of pyloric involvement:

"My pyloric sphincter… would not open" [1]

This finding is consistent with pyloric dysfunction, including pylorospasm or impaired pyloric relaxation, which are increasingly recognised contributors to delayed gastric emptying and gastroparesis spectrum disorders [8]. Such dysfunction is frequently associated with autonomic neuropathy, particularly involving vagal impairment.

The clinical interpretation –

"Understanding neuropathy and gastro implications was no joke" [1]

further supports the presence of a neurogenic motility disorder linking autonomic dysfunction to impaired gastric transit.

1.6. Therapeutic Intervention: G-POEM

The intervention described within the narrative:

"This muscle to be helped… contents through" [1]

corresponds clinically to Gastric Peroral Endoscopic Myotomy (G-POEM), a minimally invasive endoscopic procedure designed to improve gastric emptying by performing a targeted pyloric myotomy. G-POEM has demonstrated efficacy in improving gastric emptying, reducing reflux-like symptoms, decreasing upstream esophageal pressure burden, and enhancing patient quality of life [8]. Within the context of this case, G-POEM represents the **mechanical correction** within the broader gastroautonomic model.

This therapeutic effect is reflected in the narrative:

"Less reflux… quality of life enhanced" [1]

1.7. Environmental Trigger Identification

A major diagnostic breakthrough is captured in the lyric:

"Dog dander floats… could it be stirring a battle inferred?" [1]

This hypothesis is subsequently confirmed through experiential observation:

"Two weeks away… zero EoE attacks… returned… every night" [1]

This temporal association strongly supports aeroallergen-driven eosinophilic esophagitis, which is increasingly recognised in sensitised individuals [9]. The identification of this trigger represents a critical step toward personalised disease management.

1.8. Pharmacological Management

Pharmacological intervention in this case aligns with established best practice for EoE.

1.8.1. Fluticasone (Flixotide)

"Two puffs… swallowed once daily" [1]

Swallowed topical corticosteroids reduce eosinophilic inflammation, promote mucosal healing, and improve symptom control [10].

1.8.2. Budesonide Orodispersible (Jorveza)

"When Jorveza arrives, a path shining through" [1]

Budesonide orodispersible tablets provide enhanced mucosal contact and have demonstrated efficacy in achieving both symptomatic and histological remission in EoE [2].

1.9. Integrated Gastroautonomic Model

The central conceptual insight of this work is captured in the narrative:

"Not just the eosinophils… but autonomic threads" [1]

This supports a three-layer disease model, in which immune, neural, and mechanical dysfunction interact to produce the observed clinical phenotype. Specifically, eosinophilic inflammation represents the immune layer, autonomic imbalance constitutes the neural layer, and pyloric dysfunction defines the mechanical layer.

The progression described—

"From intermittent flares to the endless dark night" [1]

—reflects a process of phenoconversion, whereby episodic dysfunction evolves into sustained pathological dysregulation.

2. Discussion

This case demonstrates that gastrointestinal disease should not be approached in isolation, as immune-mediated inflammation, autonomic regulation, and mechanical function are deeply interconnected. Failure to recognise this integration may result in incomplete diagnosis and suboptimal treatment. Furthermore, the use of musical narrative as a data source highlights the value of patient-authored qualitative evidence in identifying patterns and

mechanisms that may not be readily apparent through conventional clinical assessment [10,11]

3. Conclusion

In conclusion, this study demonstrates that eosinophilic esophagitis, when associated with gastroautonomic dysfunction and pyloric dysmotility, represents a complex multi-system disorder requiring an integrated therapeutic approach. Effective management must therefore address environmental triggers, immune activation, autonomic regulation, and mechanical obstruction simultaneously. The narrative insight—

"To treat what's below… what hides from the eye" [1]

—serves as both a clinical and conceptual summary of this integrated model [12,13].

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