

Rising Incidence and Prevalence of Eosinophilic Esophagitis in the United Arab Emirates, Comorbidities, and Clinical Outcomes: A Single-Center Cohort Study

Eyad Alakrad

eakrad@yahoo.co.uk

Sheikh Shakhbout Medical City

Evan S. Dellon

University of North Carolina School of Medicine

Shaima Wasim Khan

Sheikh Shakhbout Medical City

Khalifa Saleh Al-Tenaiji

Sheikh Shakhbout Medical City

Mohamed Al Zaabi

Sheikh Shakhbout Medical City

Hosameldin Abdelrahman

Sheikh Shakhbout Medical City

Ameirah Bader Al Ali

Sheikh Shakhbout Medical City

Thaer Khaleel Swaid

Sheikh Shakhbout Medical City

Mostafa Ahmad Shehata

Sheikh Shakhbout Medical City

Research Article

Keywords: Eosinophilic Esophagitis, Gastroesophageal reflux disease, UAE, Dupilumab

Posted Date: October 19th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10999082/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: Competing interest reported. Eyad Alakrad has received speaker honoraria from Sanofi, Abbott, and Acino. Evan S. Dellon reports research funding from Adare/Ellodi, Allakos, Arena/Pfizer, AstraZeneca, Celldex, Dr. Falk Pharma, Eupraxia, Ferring, GSK, Meritage, Miraca, Nutricia, Celgene/Receptos/BMS, Phathom, Regeneron, Revolo, Sanofi, Shire/Takeda, Uniquity; consulting relationships with Abbvie, Adare/Ellodi, Alfasigma, ALK, Amgen, Anaptysbio/First Tracks bio, Apogee, Apollo, Aqilion, Arena/Pfizer, AstraZeneca, Bethanamist, Biocryst, Bryn, Calypso, Celgene/Receptos/BMS, Celldex, Cytel, Domain, Eli Lilly, EsoCap, Eupraxia, Dr. Falk Pharma, Ferring, GI Reviewers, GSK, Holoclara, Johnson & Johnson, Knightpoint, Kymera, LucidDx, Noa, Nexstone Immunology/Uniquity, Nutricia, Parexel/Calyx, Phathom, Regeneron, Revolo, Robarts/Alimentiv, Roivant, Sanofi, Shire/Takeda, Target RWE, Third Harmonic Bio; and educational grants from Allakos, Aqilion, Holoclara, and Invea. The remaining authors declare no conflicts of interest.

Abstract

Background

Eosinophilic esophagitis (EoE) is a chronic, immune-mediated esophageal disease with a rapidly increasing global incidence. However, data from the Middle East remain limited.

Objective

To characterize the clinical, diagnostic, and treatment characteristics of patients with EoE at a large tertiary referral center in the United Arab Emirates, and secondarily to evaluate temporal trends in center-level incidence and prevalence.

Methods

We conducted a retrospective cohort study of patients diagnosed with EoE between 2019 and 2025 in the United Arab Emirates (UAE). Patients were identified using ICD-10 codes and diagnoses were confirmed through guideline-based criteria. Extracted data included demographics, clinical presentation, endoscopic and histologic findings, and treatment outcomes.

Results

The annual EoE incidence increased > 23 fold, from 0.2 per 100,000 population in 2019 to 4.7 per 100,000 in 2024. Prevalence increased in parallel, from 0.2 per 100,000 in 2019 to 17.0 per 100,000. In the 102 newly diagnosed EoE patients included (mean age 26.6 years; 68% male), dysphagia (67%) and food impaction (35%) were the most common presenting symptoms. Patients without concomitant GERD were more likely to require esophageal dilation compared to those with GERD (13% vs. 2%, $p = 0.03$). In an exploratory analysis, histologic response was higher among patients treated with biologic therapy compared with those not receiving biologic therapy (88% vs. 41%; $p = 0.02$). In a subanalysis limited to dupilumab, a trend toward higher histologic response was also observed (83% vs. 45%; $p = 0.09$).

Conclusion

EoE is an emerging disease in the Middle East with a rapidly increasing incidence and prevalence. Patient characteristics in the UAE are similar to those reported elsewhere around the world, and those treated with biologics had a higher treatment response.

INTRODUCTION

Eosinophilic esophagitis (EoE) is a chronic, allergen-driven, type 2 immune-mediated disease characterized by symptoms of esophageal dysfunction and eosinophil-predominant inflammation of the esophagus [1]. EoE is now a major cause of upper gastrointestinal morbidity, with a marked increase in incidence and prevalence globally [2, 3]. This rise has been attributed to an interplay of environmental exposures and genetic susceptibility, with disease progression characterized by a transition from an inflammatory phenotype to a fibrostenotic state with diagnostic delay or when left untreated [4].

Despite this growing global recognition, data from the Middle East remain limited. A recent systematic review encompassing 67 studies across the region highlighted heterogeneity in diagnostic practices, study design, and reporting standards, with concerns regarding underdiagnosis and misclassification of disease [5]. Similarly, expert consensus from the Gulf region has identified gaps in disease awareness, variability in biopsy practices, and limited epidemiological data as key barriers to accurate disease characterization [6]. Contributing factors include inconsistent use of esophageal biopsies, particularly in patients with presumed gastroesophageal reflux disease (GERD), as well as limited awareness among healthcare providers. Accordingly, reported prevalence rates remain comparatively low, potentially reflecting under-recognition. However, available regional cohorts suggest that the clinical phenotype of EoE in Middle Eastern populations mirrors that observed in Western populations [7, 8].

In this context, our primary aim was to characterize the clinical, diagnostic, endoscopic, histologic, and treatment characteristics of patients with confirmed EoE managed at a large tertiary referral center in the UAE. A secondary aim was to describe temporal trends in the incidence and prevalence of EoE within the center's estimated catchment population. We also performed exploratory subgroup analyses according to concurrent GERD diagnosis, biologic therapy, and sex, with an additional subanalysis according to dupilumab use. By addressing these gaps, this work could contribute to a more precise understanding of EoE in the region and support improved recognition and management of this increasingly prevalent disease.

METHODS

Study Design, Setting, and Patient Identification

This was a retrospective cohort study conducted at Sheikh Shakhbout Medical City (SSMC), a large tertiary referral center in Abu Dhabi, United Arab Emirates. The study period extended from January 2019 to October 5, 2025.

Patients were identified through the institutional electronic medical record (EMR) system (Salamtak) using the International Classification of Diseases, Tenth Revision (ICD-10) code for eosinophilic esophagitis (K20.0). After initial identification, the diagnosis of EoE was confirmed in accordance with current guidelines [1], and patients were included if they had: symptoms of esophageal dysfunction; ≥ 15 eosinophils per high-power field (eos/hpf) on esophageal biopsy; and exclusion of alternative causes of esophageal eosinophilia. Patients were excluded if: electronic medical records were incomplete or

insufficient for analysis; they had a concomitant non-EoE eosinophilic gastrointestinal disease; or diagnostic criteria for eosinophilic esophagitis were not met. After EoE patient identification, follow-up data were collected when available.

At our institution, the standard biopsy protocol for suspected EoE includes obtaining at least six esophageal biopsies from the proximal and distal esophagus, and all patients included in this study had biopsies obtained from both levels. Histologic assessment was performed by institutional general pathologists using a standardized high-power-field area, with eosinophil counts reported as eos/hpf. Alternative causes of esophageal eosinophilia were excluded through review of the clinical history, endoscopic and histologic findings, and relevant clinical investigations.

Data Collection

Data were collected through systematic retrospective review of EMR records and compiled into a structured database. Extracted variables included demographic characteristics such as age, sex, nationality, and body mass index, as well as clinical features including presenting symptoms and duration of symptoms prior to EoE diagnosis. Comorbid conditions were documented, with particular focus on atopic diseases including asthma, allergic rhinitis, food allergy, and environmental allergy.

Typical endoscopic findings of EoE were recorded, including edema, rings, exudates, furrows, and stricture, and whether dilation was performed. Because EREFS was not routinely documented at our center, we were not able to present these data. Histologic data included eosinophil counts from proximal and distal esophageal biopsies, as well as peak eosinophil count from all locations. Data related to additional investigations, such as esophageal high-resolution manometry, were obtained when available.

Treatment data included use of dietary therapies, proton pump inhibitors (PPIs), swallowed topical corticosteroids, and biologic agents. Procedural interventions, including esophageal dilation, were also reported. Follow-up data included repeat endoscopic findings and histologic outcomes when available. Given the retrospective nature of the study, detailed information regarding treatment dose, duration, sequencing, and combination regimens was not systematically available and was therefore not analyzed. Treatment adherence was assessed based on documentation in the medical record when available.

Clinical Practice Patterns

At our center, management practices reflected real-world clinical practice at our tertiary referral center and were guided by contemporary guideline-based recommendations. Treatment decisions were individualized through a shared-decision-making approach, following detailed discussions with patients regarding the natural history of the disease, as well as the risks, benefits, and alternatives of available therapeutic options. Initial therapy most commonly consisted of PPI, STC, dietary therapy or biologics

such as dupilumab. The latter is frequently reserved for patients with advanced fibrostenotic disease, failure of initial therapy, or concomitant asthma or atopy.

Follow-up endoscopy with esophageal biopsies was generally recommended within 3 to 6 months after initiation or modification of therapy, or for a change in symptoms to assess endoscopic and histologic response. However, given that these were real-world practice data, endoscopy was not uniformly performed in all patients due to physician discretion or patient adherence. Esophageal dilation was performed in patients with symptomatic strictures or at the discretion of the treating endoscopist, based on clinical presentation and endoscopic findings, with EndoFLIP utilized when available to aid in detecting subtle esophageal strictures.

Definitions and Outcome Measures

Endoscopic findings associated with EoE, including edema, rings, exudates, furrows, and strictures, were recorded when documented by the endoscopist. Although these findings represent the individual components of EREFS [9], EREFS was not routinely reported during the study period and a composite score was therefore not retrospectively calculated. Histologic response was defined as a peak eosinophil count of < 15 eos/hpf on follow-up biopsy [10].

The diagnosis of gastroesophageal reflux disease (GERD) was based on a diagnosis documented by the treating gastroenterologist in the medical record. When objective testing was available within our institutional records, this included erosive esophagitis of Los Angeles grade B, C, or D and/or abnormal ambulatory reflux monitoring. As this was a retrospective study, some patients had previously undergone diagnostic evaluation outside our institution, and the results of these studies were not always available for independent review; in these cases, the documented diagnosis of GERD by the treating gastroenterologist was used.

Statistical Analysis

Incidence and prevalence rates were estimated using an institutional catchment population of approximately 600,000 individuals, based on estimates provided by the SSMC Statistical Department and Patient Experience Department. The catchment population was considered stable over the study period and was used as the population at risk because the study was based on patients diagnosed at SSMC rather than the overall UAE population. Annual incidence was calculated as the number of newly diagnosed cases each year divided by the estimated catchment population and expressed per 100,000 individuals. Cumulative prevalence was calculated using the cumulative number of diagnosed cases divided by the same estimated catchment population and expressed per 100,000 individuals.

Patient characteristics were summarized with descriptive statistics. Continuous variables are presented as mean $\pm$ standard deviation and were compared using appropriate parametric or non-parametric tests based on distribution. Categorical variables are presented as frequencies and percentages. EoE patients

with and without GERD were compared, as were those who did and did not receive biologics and male versus female patients. In a secondary analysis, we compared only those patients who did and did not receive dupilumab. For these comparisons, continuous variables were analyzed with two-sample t-tests, and proportions were compared using chi-square. Stata version 12.0 (College Station, TX) was used for analyses.

Ethical Considerations

The study was approved by the Institutional Review Board of Sheikh Shakhboub Medical City (SSMCREC-495). Given the retrospective design and use of anonymized data, the requirement for informed consent was waived in accordance with institutional and international ethical standards.

RESULTS

Incidence and Prevalence

Between 2019 and 2025, we identified 102 patients who were newly diagnosed with confirmed eosinophilic esophagitis (EoE) at our center. Based on an estimated catchment population of approximately 600,000, the annual incidence of EoE was calculated at 0.2 per 100,000 population in 2019, which increased more than 23 fold to a peak of 4.7 per 100,000 in 2024 (Supplemental Table 1, Fig. 1). Cumulative prevalence increased in parallel, from 0.2 per 100,000 in 2019 to 17.0 per 100,000 by 2025.

Patient Demographics and Characteristics

The mean age at diagnosis was 26.6 ± 17.1 years, with 69 patients (68%) being male. The vast majority of patients were of Emirati nationality (81, 79%; Table 1). Allergic rhinitis was the most frequently identified atopic condition (37%), followed by asthma (29%) and food allergy (26%). Dysphagia was the dominant presenting symptom (67%), followed by food impaction (35%). A concurrent diagnosis of GERD was found in 54%. The mean duration of symptoms prior to diagnosis was 14.2 ± 26.8 months (median 5 months, IQR 1–24 months).

Table 1

Baseline demographic and clinical characteristics of patients with eosinophilic esophagitis (n = 102)

Variable	Value
Demographics	
Age at diagnosis (years, mean $\pm$ SD)	26.6 $\pm$ 17.1
Male sex, n (%)	69 (68)
Female sex, n (%)	33 (32)
Emirati nationality, n (%)	81 (79)
BMI (kg/m ² , mean $\pm$ SD)	24.0 $\pm$ 7.0
Atopy	
Food allergy, n (%)	27 (26)
Environmental allergy, n (%)	10 (10)
Asthma, n (%)	30 (29)
Allergic rhinitis, n (%)	37 (37)
Comorbidities	
Diabetes, n (%)	7 (7)
GERD diagnosis, n (%)	55 (54)
Symptoms	
Dysphagia	68 (67)
Odynophagia	10 (10)
Food impaction	36 (35)
Weight loss	24 (24)
Failure to thrive	18 (18)
Vomiting	20 (20)
Symptom duration prior to establishing diagnosis	
Mean $\pm$ SD (months)	14.2 $\pm$ 26.8
Median (IQR)	5 (1–24)
Data are presented as mean $\pm$ standard deviation or n (%). BMI, body mass index; GERD, gastroesophageal reflux disease; EoE, eosinophilic esophagitis.	

At the index endoscopy, typical EoE findings were common and included edema (36%), rings (49%), exudates (29%), furrows (50%), and strictures (14%); 7% required esophageal dilation at the time of their first procedure (Table 2). The mean peak eosinophil count was 38.5 ± 35.6 eos/hpf, with similar values distally and proximally.

Table 2

Diagnostic testing, endoscopic, histologic, reflux-related, treatment, and follow-up characteristics of patients with eosinophilic esophagitis (n = 102)

Variable	Value
Testing	
EGD performed, n (%)	102 (100)
Upper GI series, n (%)	26 (25)
Esophageal manometry, n (%)	11 (11)
Skin prick allergy tests, n (%)	2 (2)
Blood allergy tests, n (%)	0 (0)
GERD-related findings	
Positive pH study, n/N (%)	4/8 (50)
Erosive esophagitis LA Grade, n/N (%)	of 55 patients with GERD Diagnosis
None	36 (65)
A	12 (22)
B	5 (9)
C	0 (0)
D	2 (4)
Hiatal hernia on endoscopy, n/N (%)	24/55 (44)
Index EGD findings	
On PPI at first EGD, n (%)	49 (48)
Normal examination, n (%)	14 (14)
Edema, n (%)	37 (36)
Rings, n (%)	50 (49)
Exudates, n (%)	30 (29)
Furrows, n (%)	51 (50)
Stricture, n (%)	14 (14)

Data are presented as mean $\pm$ standard deviation or n (%) unless otherwise indicated. Ambulatory pH testing data were available for 8 of 55 patients with GERD. Therapy compliance data were available for 31 patients. Follow-up EGD was done or offered within 6 months in 33 patients. Follow-up biopsy data were available for 35 patients. Histologic response was defined as < 15 eos/hpf. BMI, body mass index; EGD, esophagogastroduodenoscopy; eos/hpf, eosinophils per high-power field; GERD, gastroesophageal reflux disease; PPI, proton pump inhibitor; EoE, eosinophilic esophagitis.

Variable	Value
Dilation performed, n (%)	7 (7)
Histologic findings at index EGD (eos/hpf, mean $\pm$ SD)	
Distal	33.0 $\pm$ 29.3
Proximal	32.4 $\pm$ 35.6
Peak	38.5 $\pm$ 35.6
Treatment after first EGD	
PPI, n (%)	95 (93)
Elimination diet, n (%)	18 (18)
Elemental formula, n (%)	0 (0)
Topical steroids (off-label), n (%)	22 (22)
Budesonide orodispersible tablet, n (%)	15 (15)
Systemic steroid, n (%)	1 (1)
Biologic, n (%)	18 (18)
Dupilumab, n/N (%)	15/18 (83)
Infliximab, n/N (%)	1/18 (6)
Omalizumab, n/N (%)	1/18 (6)
Vedolizumab, n/N (%)	1/18 (6)
Therapy compliance, n/N (%)	22/31 (71)
Follow-up	
Follow-up EGD done/offered within 6 months, n (%)	33 (32)
Months to follow-up EGD (mean $\pm$ SD)	13.9 $\pm$ 23.2
Follow-up eosinophil count (eos/hpf, mean $\pm$ SD)	
Distal	20.8 $\pm$ 28.6
Proximal	14.1 $\pm$ 21.4
Data are presented as mean $\pm$ standard deviation or n (%) unless otherwise indicated. Ambulatory pH testing data were available for 8 of 55 patients with GERD. Therapy compliance data were available for 31 patients. Follow-up EGD was done or offered within 6 months in 33 patients. Follow-up biopsy data were available for 35 patients. Histologic response was defined as < 15 eos/hpf. BMI, body mass index; EGD, esophagogastroduodenoscopy; eos/hpf, eosinophils per high-power field; GERD, gastroesophageal reflux disease; PPI, proton pump inhibitor; EoE, eosinophilic esophagitis.	

Variable	Value
Peak	23.7 ± 31.1
Histologic response, n/N (%)	18/35 (51)
Data are presented as mean ± standard deviation or n (%) unless otherwise indicated. Ambulatory pH testing data were available for 8 of 55 patients with GERD. Therapy compliance data were available for 31 patients. Follow-up EGD was done or offered within 6 months in 33 patients. Follow-up biopsy data were available for 35 patients. Histologic response was defined as < 15 eos/hpf. BMI, body mass index; EGD, esophagogastroduodenoscopy; eos/hpf, eosinophils per high-power field; GERD, gastroesophageal reflux disease; PPI, proton pump inhibitor; EoE, eosinophilic esophagitis.	

Treatments and outcomes

PPIs were the most widely prescribed first-line treatment, initiated in 95 patients (93%). Topical corticosteroids were used off-label in 22%, while budesonide orodispersible tablets were prescribed in 15%, and 18% selected dietary elimination (Table 2). Biologic therapy was prescribed in 18 patients (18%), 15 of whom received dupilumab. The remaining three patients received infliximab, omalizumab, and vedolizumab, prescribed for other concomitant diseases. Follow-up EGD was done or offered within 6 months in 33 patients (32%). The mean interval to follow-up EGD was 13.9 ± 23.2 months. Mean follow-up peak eosinophil count was 23.7 ± 31.1 eos/hpf, and histologic response was achieved in 18 of 35 patients (51%) with available follow-up biopsy data.

Comparative Analysis by Concurrent GERD Diagnosis

Patients with concomitant EoE and GERD were significantly older at diagnosis (mean 31.8 ± 17.1 vs. 20.4 ± 14.9 years; $p < 0.001$) and had significantly higher BMI (25.9 ± 6.6 vs. 21.6 ± 6.8 kg/m²; $p = 0.002$) than patients with EoE alone, and diabetes was exclusively present in the GERD group (13% vs. 0%; $p = 0.01$) (Table 3). In contrast, food allergy was more prevalent in the non-GERD group (38% vs. 16%; $p = 0.01$). Duration of symptoms prior to diagnosis was comparable between the GERD and non-GERD subgroups (15.7 ± 20.6 vs. 12.5 ± 32.8 months; $p = 0.55$), PPI use at the time of index endoscopy was substantially higher in the GERD group (69% vs. 23%; $p < 0.001$), while esophageal dilation was performed significantly more frequently in non-GERD patients (13% vs. 2%; $p = 0.03$). Histologic response rates at follow-up were similarly comparable between groups (55% vs. 46%; $p = 0.63$).

Table 3
Comparison of clinical, endoscopic, and treatment characteristics by GERD status

Age at diagnosis (mean years $\pm$ SD)	No GERD (n = 47)	GERD (n = 55)	p
	20.4 $\pm$ 14.9	31.8 $\pm$ 17.1	< 0.001
Male (n, %)	34 (72)	35 (64)	0.35
Emirati nationality (n, %)	36 (77)	45 (82)	0.52
BMI (mean $\pm$ SD)	21.6 $\pm$ 6.8	25.9 $\pm$ 6.6	0.002
Atopy (n, %)			
Food allergy	18 (38)	9 (16)	0.01
Environmental allergy	5 (11)	5 (9)	0.79
Asthma	12 (26)	18 (33)	0.43
Allergic rhinitis	15 (32)	22 (40)	0.40
Diabetes (n, %)	0 (0)	7 (13)	0.01
Symptoms (n, %)			
Dysphagia	27 (57)	41 (75)	0.07
Odynophagia	4 (9)	6 (11)	0.69
Food impaction	19 (40)	17 (31)	0.32
Weight loss	12 (26)	12 (22)	0.66
Failure to thrive	10 (21)	8 (15)	0.37
Vomiting	10 (21)	10 (18)	0.70
Symptom length prior to diagnosis (mean months $\pm$ SD)	12.5 $\pm$ 32.8	15.7 $\pm$ 20.6	0.55
Testing (n, %)			
EGD	47 (100)	55 (100)	--
UGI series	20 (43)	6 (11)	< 0.001
Esophageal manometry	7 (15)	4 (7)	0.22

Data are presented as mean $\pm$ standard deviation or n (%) unless otherwise indicated. Follow-up biopsy data were available for 13 patients without GERD and 22 patients with GERD. Histologic response was defined as < 15 eos/hpf. BMI, body mass index; EGD, esophagogastroduodenoscopy; eos/hpf, eosinophils per high-power field; GERD, gastroesophageal reflux disease; PPI, proton pump inhibitor; EoE, eosinophilic esophagitis.

Age at diagnosis (mean years $\pm$ SD)	No GERD (n = 47)	GERD (n = 55)	p
	20.4 $\pm$ 14.9	31.8 $\pm$ 17.1	< 0.001
Skin prick allergy tests	0 (0)	2 (4)	0.19
Blood allergy tests	0 (0)	0 (0)	--
First EGD details (n, %)			
On PPI	11 (23)	38 (69)	< 0.001
Normal exam	4 (9)	10 (18)	0.16
Edema	15 (32)	22 (40)	0.40
Rings	24 (51)	26 (47)	0.70
Exudates	16 (34)	14 (25)	0.34
Furrows	26 (55)	25 (45)	0.32
Stricture	9 (19)	5 (9)	0.14
Dilation	6 (13)	1 (2)	0.03
Eosinophil count (mean eos/hpf $\pm$ SD)			
Distal	29.0 $\pm$ 25.1	36.5 $\pm$ 32.4	0.20
Proximal	34.2 $\pm$ 34.3	31.0 $\pm$ 37.0	0.66
Peak	36.7 $\pm$ 34.0	40.1 $\pm$ 37.2	0.63
Treatment after first EGD (n, %)			
PPI	40 (85)	55 (100)	0.003
Elimination diet	10 (21)	8 (15)	0.37
Elemental formula	0 (0)	0 (0)	--
Topical steroids (off-label)	13 (28)	9 (16)	0.17
Budesonide orodispersible tablet	7 (15)	8 (15)	0.96
Systemic steroid	1 (2)	0 (0)	0.28
Biologic	7 (15)	11 (20)	0.50

Data are presented as mean $\pm$ standard deviation or n (%) unless otherwise indicated. Follow-up biopsy data were available for 13 patients without GERD and 22 patients with GERD. Histologic response was defined as < 15 eos/hpf. BMI, body mass index; EGD, esophagogastroduodenoscopy; eos/hpf, eosinophils per high-power field; GERD, gastroesophageal reflux disease; PPI, proton pump inhibitor; EoE, eosinophilic esophagitis.

Age at diagnosis (mean years $\pm$ SD)	No GERD (n = 47)	GERD (n = 55)	p
	20.4 $\pm$ 14.9	31.8 $\pm$ 17.1	< 0.001
Follow-up eosinophil count (mean eos/hpf $\pm$ SD)			
Distal	25.5 $\pm$ 36.7	18.0 $\pm$ 23.2	0.46
Proximal	18.4 $\pm$ 22.0	11.3 $\pm$ 21.0	0.36
Peak	27.5 $\pm$ 37.0	21.4 $\pm$ 27.8	0.58
Histologic response (n, %; <15 eos/hpf)	6 (46)	12 (55)	0.63
Data are presented as mean $\pm$ standard deviation or n (%) unless otherwise indicated. Follow-up biopsy data were available for 13 patients without GERD and 22 patients with GERD. Histologic response was defined as < 15 eos/hpf. BMI, body mass index; EGD, esophagogastroduodenoscopy; eos/hpf, eosinophils per high-power field; GERD, gastroesophageal reflux disease; PPI, proton pump inhibitor; EoE, eosinophilic esophagitis.			

Comparative Analysis by Biologic Therapy

Patients who received biologic therapy (n = 18) had a significantly higher prevalence of allergic rhinitis (67% vs. 30%; p = 0.003) and a trend toward greater food allergy burden (44% vs. 23%; p = 0.06). Dysphagia was significantly more common in the biologic group (89% vs. 62%; p = 0.03). Histologic response was higher in the biologic-treated group (88% vs. 41%; p = 0.02) (Table 4). In a secondary analysis limited to dupilumab, patients who received dupilumab had a higher prevalence of allergic rhinitis (73% vs. 30%; p = 0.001), food allergy (53% vs. 22%; p = 0.01), and dysphagia (93% vs. 62%; p = 0.02). There was also a trend toward higher histologic response among patients receiving dupilumab (83% vs. 45%; p = 0.09) (Supplemental Table 2).

Table 4
Comparison of clinical characteristics and outcomes by biologic therapy use

Age at diagnosis (mean years $\pm$ SD)	No biologic (n = 84)	Biologic (n = 18)	p
	27.0 $\pm$ 17.5	24.3 $\pm$ 14.9	0.55
Male (n, %)	58 (69)	11 (61)	0.51
Emirati nationality (n, %)	69 (82)	12 (67)	0.14
BMI (mean $\pm$ SD)	24.0 $\pm$ 7.0	23.6 $\pm$ 7.3	0.80
Atopy (n, %)			
Food allergy	19 (23)	8 (44)	0.06
Environmental allergy	7 (8)	3 (17)	0.28
Asthma	23 (27)	7 (39)	0.33
Allergic rhinitis	25 (30)	12 (67)	0.003
Diabetes (n, %)	6 (7)	1 (6)	0.81
GERD diagnosis	44 (52)	11 (61)	0.50
Symptoms (n, %)			
Dysphagia	52 (62)	16 (89)	0.03
Odynophagia	6 (7)	4 (22)	0.05
Food impaction	28 (33)	8 (44)	0.37
Weight loss	18 (21)	6 (33)	0.28
Failure to thrive	14 (17)	4 (22)	0.58
Vomiting	15 (18)	5 (28)	0.34
Symptom length prior to diagnosis (mean months $\pm$ SD)	12.3 $\pm$ 27.4	23.2 $\pm$ 22.9	0.12
Testing (n, %)			
EGD	84 (100)	18 (100)	--
UGI series	22 (26)	4 (22)	0.73

Data are presented as mean $\pm$ standard deviation or n (%) unless otherwise indicated. Follow-up biopsy data were available for 27 patients in the no-biologic group and 8 patients in the biologic group. Histologic response was defined as < 15 eos/hpf. BMI, body mass index; EGD, esophagogastroduodenoscopy; eos/hpf, eosinophils per high-power field; GERD, gastroesophageal reflux disease; PPI, proton pump inhibitor; EoE, eosinophilic esophagitis.

Age at diagnosis (mean years ± SD)	No biologic (n = 84)	Biologic (n = 18)	p
	27.0 ± 17.5	24.3 ± 14.9	0.55
Esophageal manometry	9 (11)	2 (11)	0.96
Skin prick allergy tests	2 (2)	0 (0)	0.51
Blood allergy tests	0 (0)	0 (0)	--
First EGD details (n, %)			
On PPI	38 (45)	11 (61)	0.22
Normal exam	10 (12)	4 (22)	0.25
Edema	32 (38)	5 (28)	0.41
Rings	42 (50)	8 (44)	0.67
Exudates	27 (32)	3 (17)	0.19
Furrows	43 (51)	8 (44)	0.60
Stricture	12 (14)	2 (11)	0.72
Dilation	5 (6)	2 (11)	0.43
Eosinophil count (mean eos/hpf ± SD)			
Distal	32.9 ± 29.4	33.7 ± 29.8	0.92
Proximal	35.3 ± 38.4	19.2 ± 11.2	0.09
Peak	38.8 ± 37.3	37.3 ± 27.3	0.87
Treatment after first EGD (n, %)			
PPI	79 (94)	16 (89)	0.43
Elimination diet	13 (15)	5 (28)	0.21
Elemental formula	0 (0)	0 (0)	--
Topical steroids (off-label)	19 (23)	3 (17)	0.58
Budesonide orodispersible tablet	12 (14)	3 (17)	0.80
Systemic steroid	1 (1)	0 (0)	0.64

Data are presented as mean ± standard deviation or n (%) unless otherwise indicated. Follow-up biopsy data were available for 27 patients in the no-biologic group and 8 patients in the biologic group. Histologic response was defined as < 15 eos/hpf. BMI, body mass index; EGD, esophagogastroduodenoscopy; eos/hpf, eosinophils per high-power field; GERD, gastroesophageal reflux disease; PPI, proton pump inhibitor; EoE, eosinophilic esophagitis.

Age at diagnosis (mean years $\pm$ SD)	No biologic (n = 84)	Biologic (n = 18)	p
	27.0 $\pm$ 17.5	24.3 $\pm$ 14.9	0.55
Follow-up eosinophil count (mean eos/hpf $\pm$ SD)			
Distal	25.0 $\pm$ 31.4	6.6 $\pm$ 6.3	0.11
Proximal	17.2 $\pm$ 23.6	4.4 $\pm$ 5.8	0.14
Peak	28.7 $\pm$ 33.8	6.6 $\pm$ 6.3	0.08
Histologic response (n, %; <15 eos/hpf)	11 (41)	7 (88)	0.02
Data are presented as mean $\pm$ standard deviation or n (%) unless otherwise indicated. Follow-up biopsy data were available for 27 patients in the no-biologic group and 8 patients in the biologic group. Histologic response was defined as < 15 eos/hpf. BMI, body mass index; EGD, esophagogastroduodenoscopy; eos/hpf, eosinophils per high-power field; GERD, gastroesophageal reflux disease; PPI, proton pump inhibitor; EoE, eosinophilic esophagitis.			

Comparative Analysis by Sex

Male and female patients with EoE were generally similar with respect to age at diagnosis, BMI, atopic comorbidities, symptom burden, or eosinophil counts (Table 5). Treatment strategies and histological response rates at follow-up were also comparable between sexes (56% in females vs. 50% in males; p = 0.77).

Table 5
Sex-based comparison of clinical, endoscopic, and treatment characteristics in eosinophilic esophagitis
(n = 102)

Age at diagnosis (mean years $\pm$ SD)	Female (n = 33)	Male (n = 69)	p
	27.4 $\pm$ 15.9	26.1 $\pm$ 17.7	0.72
Emirati nationality (n, %)	27 (82)	54 (78)	0.68
BMI (mean $\pm$ SD)	24.8 $\pm$ 7.9	23.6 $\pm$ 6.5	0.43
Atopy (n, %)			
Food allergy	11 (33)	16 (23)	0.28
Environmental allergy	6 (18)	4 (6)	0.05
Asthma	10 (30)	20 (29)	0.89
Allergic rhinitis	13 (39)	24 (35)	0.65
Diabetes (n, %)	2 (6)	5 (7)	0.83
GERD diagnosis	20 (61)	35 (51)	0.35
Symptoms (n, %)			
Dysphagia	22 (67)	46 (67)	1.0
Odynophagia	3 (9)	7 (10)	0.87
Food impaction	10 (30)	26 (38)	0.47
Weight loss	10 (30)	14 (20)	0.27
Failure to thrive	4 (12)	14 (20)	0.31
Vomiting	7 (21)	13 (19)	0.78
Symptom length prior to diagnosis (mean months $\pm$ SD)	14.6 $\pm$ 19.6	14.1 $\pm$ 29.8	0.93
Testing (n, %)			
EGD	33 (100)	69 (100)	--
UGI series	8 (24)	18 (26)	0.84
Data are presented as mean $\pm$ standard deviation or n (%) unless otherwise indicated. Follow-up biopsy data were available for 9 female patients and 26 male patients. Histologic response was defined as < 15 eos/hpf. BMI, body mass index; EGD, esophagogastroduodenoscopy; eos/hpf, eosinophils per high-power field; GERD, gastroesophageal reflux disease; PPI, proton pump inhibitor; EoE, eosinophilic esophagitis.			

Age at diagnosis (mean years $\pm$ SD)	Female (n = 33)	Male (n = 69)	p
	27.4 $\pm$ 15.9	26.1 $\pm$ 17.7	0.72
Esophageal manometry	5 (15)	6 (9)	0.33
Skin prick allergy tests	2 (6)	0 (0)	0.04
Blood allergy tests	0 (0)	0 (0)	--
First EGD details (n, %)			
On PPI	20 (61)	29 (42)	0.07
Normal exam	5 (15)	9 (13)	0.77
Edema	12 (36)	25 (36)	0.99
Rings	15 (45)	35 (51)	0.62
Exudates	4 (12)	26 (38)	0.008
Furrows	14 (42)	37 (54)	0.29
Stricture	4 (12)	10 (14)	0.75
Dilation	3 (9)	4 (6)	0.54
Eosinophil count (mean eos/hpf $\pm$ SD)			
Distal	35.8 $\pm$ 30.2	31.7 $\pm$ 29.0	0.51
Proximal	30.8 $\pm$ 28.7	33.2 $\pm$ 38.6	0.76
Peak	40.5 $\pm$ 31.9	37.6 $\pm$ 37.5	0.71
Treatment after first EGD (n, %)			
PPI	29 (88)	66 (96)	0.15
Elimination diet	7 (21)	11 (16)	0.51
Elemental formula	0 (0)	0 (0)	--
Topical steroids (off-label)	8 (24)	14 (20)	0.65
Budesonide orodispersible tablet	6 (18)	9 (13)	0.49
Systemic steroid	0 (0)	1 (1)	0.49
Data are presented as mean $\pm$ standard deviation or n (%) unless otherwise indicated. Follow-up biopsy data were available for 9 female patients and 26 male patients. Histologic response was defined as < 15 eos/hpf. BMI, body mass index; EGD, esophagogastroduodenoscopy; eos/hpf, eosinophils per high-power field; GERD, gastroesophageal reflux disease; PPI, proton pump inhibitor; EoE, eosinophilic esophagitis.			

Age at diagnosis (mean years $\pm$ SD)	Female (n = 33)	Male (n = 69)	p
	27.4 $\pm$ 15.9	26.1 $\pm$ 17.7	0.72
Biologic	7 (21)	11 (16)	0.51
Follow-up eosinophil count (mean eos/hpf $\pm$ SD)			
Distal	15.4 $\pm$ 16.1	22.6 $\pm$ 31.9	0.53
Proximal	8.9 $\pm$ 9.4	16.0 $\pm$ 24.3	0.40
Peak	15.4 $\pm$ 16.1	16.5 $\pm$ 34.7	0.36
Histologic response (n, %; <15 eos/hpf)	5 (56)	13 (50)	0.77
Data are presented as mean $\pm$ standard deviation or n (%) unless otherwise indicated. Follow-up biopsy data were available for 9 female patients and 26 male patients. Histologic response was defined as < 15 eos/hpf. BMI, body mass index; EGD, esophagogastroduodenoscopy; eos/hpf, eosinophils per high-power field; GERD, gastroesophageal reflux disease; PPI, proton pump inhibitor; EoE, eosinophilic esophagitis.			

DISCUSSION

In this retrospective cohort study from a large tertiary referral center in the United Arab Emirates, we noted a substantial increase in the incidence of eosinophilic esophagitis (EoE) over the study period, with a corresponding increase in prevalence. These findings are consistent with patterns reported worldwide [2, 3, 11]. The clinical and demographic features of our cohort were similar to those reported in other regions, including a predominance of male patients and dysphagia as the most common presenting symptom [5–8], suggesting commonalities between EoE in the Middle East and elsewhere in the world.

Though the number of EoE cases has markedly increased at our center, the cumulative prevalence observed in our cohort remains relatively lower than that reported in Western populations [3], but this may reflect under-recognition of the disease, variability in biopsy practices, and low awareness among clinicians. For example, other studies have noted that diagnostic delay remains a well-recognized challenge in EoE, with delays of several years to a decade between symptom onset and diagnosis [12–14]. Similarly, in our cohort the mean duration of symptoms prior to diagnosis was 14.2 $\pm$ 26.8 months. However, the rapid increase in incidence over a relatively short time in our center likely relates, at least in part, to improved recognition and lower threshold to obtain esophageal biopsies upon clinical suspicion by endoscopist. Changes in case ascertainment likely contributed to the observed increase, although a true increase in disease occurrence cannot be determined from our data.

A notable observation in our study was the overlap between EoE and GERD, with more than half of the study cohort carrying a diagnosis of GERD. Patients without GERD were more likely to undergo

esophageal dilation, suggesting a higher burden of fibrostenotic disease in this subgroup. As would be expected, patients with GERD were more commonly treated with PPIs prior to diagnosis, and PPIs were the most commonly used first-line therapy for our patients. This reflects their accessibility, cost-effectiveness, and established role in initial management [1].

In terms of other EoE treatments, use of dietary therapy, swallowed topical corticosteroids and biologics varied, suggesting differences in physician preference and patient factors. This variability highlights the need for more standardized approaches to management in the region. Notably, patients who received biologic therapy had a higher prevalence of atopic disease, particularly allergic rhinitis, and were more likely to present with dysphagia. This suggests that biologic therapy was more often used in patients with a stronger atopic background and more symptomatic disease. We also observed a higher histologic response in the biologic-treated group. In the secondary analysis limited to dupilumab, there was a strong trend toward higher histologic response (83% vs. 45%; $p = 0.09$), which may have been limited by the small sample size. This finding is consistent with regional and worldwide published data on dupilumab [15–17]. Given the small number of patients treated with dupilumab and the non-randomized treatment selection, this analysis was exploratory and was not designed to compare treatment effectiveness.

This study has several strengths. It includes both pediatric and adult patients and applies strict clinicopathologic criteria for diagnosis rather than relying solely on administrative coding. This approach provides a more accurate assessment of disease burden and clinical characteristics. Which enabled us to more accurately report the incidence and prevalence of EoE in our center's catchment population. We also performed analyses comparing EoE patients with and without GERD, those who were and were not treated with biologics, and males and females with EoE. There are also limitations. This is a single-center retrospective study, and findings may not be generalizable to other settings. Follow-up endoscopy with biopsies was not performed uniformly, often due to physician decision-making or patient non-adherence, rather than lack of data availability. Similarly, investigations such as ambulatory pH monitoring were performed selectively. These factors may affect assessment of treatment response.

Conclusion

We found that EoE is increasingly recognized in the Middle East, with rising incidence and prevalence at our center, but with clinical characteristics similar to those reported internationally. We found that patients with EoE and GERD overlap required less frequent dilation but had similar treatment outcomes compared to those without GERD overlap, and that biologic-treated patients had the most comorbid atopic conditions but also the highest rates of response. Given heterogeneity in care patterns at our center, earlier recognition of EoE, appropriate diagnostic evaluation, individualized treatment approaches, and standardized outcome assessment will be important to improve patient outcomes.

Abbreviations

EoE
Eosinophilic Esophagitis
SSMC
Sheikh Shakhbout Medical City
EPI
Enterprise Patient Identifier
EMR
Electronic Medical Record
UAE
United Arab Emirates

Declarations

Funding: This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflict-of-Interest Statement: Eyad Alakrad has received speaker honoraria from Sanofi, Abbott, and Acino. Evan S. Dellon reports research funding from Adare/Ellodi, Allakos, Arena/Pfizer, AstraZeneca, Celldex, Dr. Falk Pharma, Eupraxia, Ferring, GSK, Meritage, Miraca, Nutricia, Celgene/Receptos/BMS, Phathom, Regeneron, Revolo, Sanofi, Shire/Takeda, Uniquity; consulting relationships with Abbvie, Adare/Ellodi, Alfasigma, ALK, Amgen, Anaptysbio/First Tracks bio, Apogee, Apollo, Aqilion, Arena/Pfizer, AstraZeneca, Bethanamist, Biocryst, Bryn, Calypso, Celgene/Receptos/BMS, Celldex, Cytel, Domain, Eli Lilly, EsoCap, Eupraxia, Dr. Falk Pharma, Ferring, GI Reviewers, GSK, Holoclara, Johnson & Johnson, Knightpoint, Kymera, LucidDx, Noa, Nexstone Immunology/Uniquity, Nutricia, Parexel/Calyx, Phathom, Regeneron, Revolo, Robarts/Alimentiv, Roivant, Sanofi, Shire/Takeda, Target RWE, Third Harmonic Bio; and educational grants from Allakos, Aqilion, Holoclara, and Invea. The remaining authors declare no conflicts of interest.

Author Contributions: Alakrad E initiated and designed the study, obtained institutional review board approval, developed the data collection framework, supervised the project, and contributed to manuscript drafting, review, and editing. Dellon ES contributed to study design, performed the statistical analyses, contributed to manuscript drafting, and provided senior critical review and editing. Shehata MA contributed to data review, literature review and reference collection, and drafting portions of the Results and Discussion. Khan SW performed data collection and data curation, compiled the study data, and contributed to project administration and preparation of the manuscript for submission. Al-Tenaiji KS, Al Zaabi M, Abdelrahman H, Al Ali AB, and Swaid TK contributed to data collection and data curation. All authors reviewed and approved the final manuscript.

Author Responsibility Statement: All authors reviewed and approved the final manuscript and take responsibility for the accuracy and integrity of its content.

Data Sharing Statement: Deidentified data may be available from the corresponding author upon reasonable request, subject to institutional approval and applicable privacy requirements.

Institutional Review Board Statement: The study was approved by the Sheikh Shakhbout Medical City Institutional Review Board (SSMCREC-495).

Informed Consent Statement: The requirement for informed consent was waived because the study used retrospective, anonymized data.

Patient Privacy Statement: No directly or potentially identifiable patient information is presented in this manuscript.

References

1. Dellon ES, Muir AB, Katzka DA, Shah SC, Sauer BG, Aceves SS, et al. ACG Clinical Guideline: Diagnosis and Management of Eosinophilic Esophagitis. *Am J Gastroenterol*. 2025;120(1):31–59.
2. Dellon ES, Hirano I. Epidemiology and Natural History of Eosinophilic Esophagitis. *Gastroenterology*. 2018;154(2):319–e3323.
3. Visaggi P, Dellon ES. Epidemiology, Natural History, and Treatment of Eosinophilic Gastrointestinal Diseases. *Gastroenterology*. 2026;170:476–94.
4. Schoepfer AM, Safroneeva E, Bussmann C, Kuchen T, Portmann S, Simon HU, et al. Delay in diagnosis of eosinophilic esophagitis increases risk for stricture formation in a time-dependent manner. *Gastroenterology*. 2013;145(6):1230–e12362.
5. Alwisi N, Abdul Moqeeth S, Al-Mohannadi M. Eosinophilic Esophagitis in the Middle East: Insight From a Comprehensive Systematic Review. *JGH Open*. 2026;10:e70305.
6. Jazzar A, Al-Darmaki A, Dellon ES, et al. Expert Opinion on the Management, Challenges, and Knowledge Gaps Pertaining to Eosinophilic Esophagitis Among Adults in the Greater Gulf Region. *Clin Exp Gastroenterol*. 2025;18:91–102.
7. Alzahrani MA, Alshehri MA, Rassam LAH, et al. Epidemiologic Characteristics and Clinical Pattern of Eosinophilic Esophagitis: Single Centre Experience. *Bahrain Med Bull*. 2022;44(1):832–5.
8. Abuzakouk M, Yousef O, Fahmy M, Chouikrat Z. Descriptive Analysis of Pre-existing Data on Eosinophilic Esophagitis and Associated Morbidities in Cleveland Clinic Abu Dhabi, United Arab Emirates. *Cureus*. 2024;16(1):e51493.
9. Hirano I, Moy N, Heckman MG, Thomas CS, Gonsalves N, Achem SR. Endoscopic assessment of the oesophageal features of eosinophilic esophagitis: validation of a novel classification and grading system. *Gut*. 2013;62(4):489–95.
10. Reed CC, Wolf WA, Cotton CC, Rusin S, Perjar I, Hollyfield J, et al. Optimal histologic cutpoints for treatment response in patients with eosinophilic esophagitis: analysis of data from a prospective cohort study. *Clin Gastroenterol Hepatol*. 2018;16(2):226–e2332.

11. Hahn JW, Lee K, Shin JI, Cho SH, Turner S, Shin JU, et al. Global incidence and prevalence of eosinophilic esophagitis, 1976–2022: a systematic review and meta-analysis. *Clin Gastroenterol Hepatol*. 2023;21(13):3270–e328477.
12. Melgaard D, Westmark S, Laurberg PT, Krarup AL. A diagnostic delay of 10 years in the DanEoE cohort calls for focus on education: a population-based cross-sectional study of incidence, diagnostic process, and complications of eosinophilic oesophagitis in the North Denmark Region. *United Eur Gastroenterol J*. 2021;9(6):688–98.
13. Reed CC, Koutlas NT, Robey BS, Hansen J, Dellon ES. Prolonged time to diagnosis of eosinophilic esophagitis despite increasing knowledge of the disease. *Clin Gastroenterol Hepatol*. 2018;16(10):1667–9.
14. Murray FR, Kreienbuehl AS, Greuter T, Nennstiel S, Safroneeva E, Saner C, et al. Diagnostic delay in patients with eosinophilic esophagitis has not changed since the first description 30 years ago. *Am J Gastroenterol*. 2022;117(11):1772–9.
15. Dellon ES, Rothenberg ME, Collins MH, et al. Dupilumab in adults and adolescents with eosinophilic esophagitis. *N Engl J Med*. 2022;387(25):2317–30.
16. Hasosah M, Sukkar G, AlSahafi A, et al. Dupilumab in children with eosinophilic esophagitis: a retrospective multicenter study. *BMC Pediatr*. 2025;25:100.
17. Collins MH, Rothenberg ME, Dellon ES, Bredenoord AJ, Hirano I, Leung J, et al. Dupilumab improves histopathologic features in patients with eosinophilic esophagitis: LIBERTY EoE TREET study results. *Gastro Hep Adv*. 2025;4(6):100646.

Figures

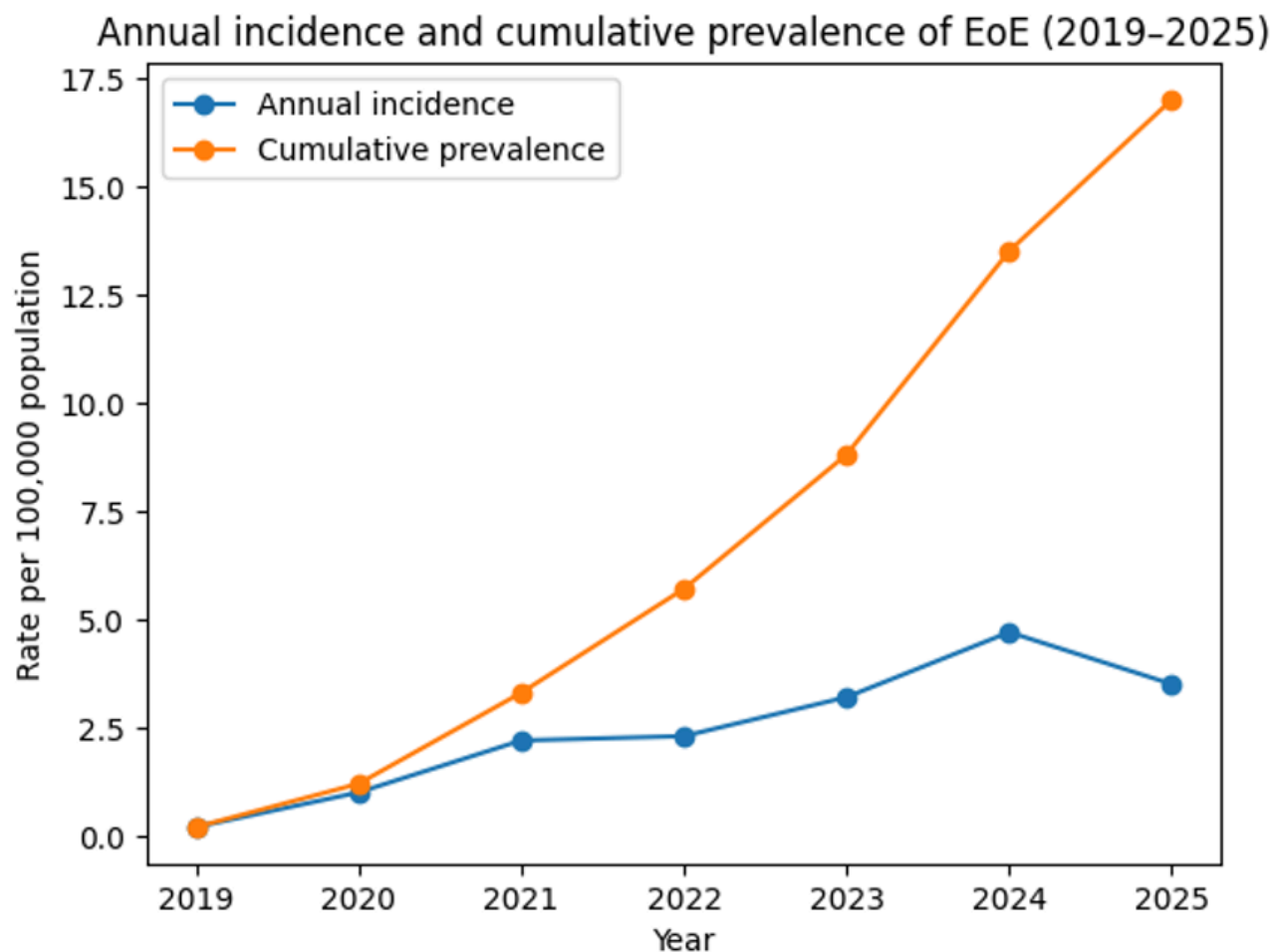


Figure 1

Annual incidence and cumulative prevalence of eosinophilic esophagitis at a tertiary referral center in Abu Dhabi, United Arab Emirates (2019–2025). Rates are expressed per 100,000 population based on the estimated institutional catchment population.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [SupplementalTables.docx](#)