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UPPER GASTRO-INTESTINAL ENDOSCOPY

Clinical presentation and spectrum of comorbidity in a South African achalasia cohort

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Background: Achalasia is a rare oesophageal motility disorder characterised by aperistalsis and failure of lower oesophageal sphincter (LOS) relaxation. While the aetiology remains unclear, autoimmune myenteric neuronal degeneration is suspected.
Methods: A retrospective review of the symptomatology and comorbidities present in a South African achalasia cohort undergoing high-resolution oesophageal manometry (HRM) between January 2012 and December 2024.
Results: During the 12-year study period, 258 patients with achalasia were included, with 181 (70.1%) newly diagnosed cases and 77 (29.8%) cases with an intervention prior to HRM. The median age was 47 (IQR 35–69) years with a slight female majority (n = 136; 52.7%). Median symptom duration prior to HRM was 36 months (IQR 12–84 months) with a median Eckhardt score of 7 (IQR 6–9). At time of achalasia diagnosis 12 patients (4.6%) had a known autoimmune condition.
Of the 217 patients having a oesophagogastroduodenoscopy (EGD) prior to their HRM, 54 (24.9%) were reported normal. Only seven (3.2%) of 219 contrast swallows performed were reported normal. Type I achalasia was most prevalent (n = 131, 50.8%), followed by type II (n = 121, 46.9%) and type III (n = 6, 2.3%). Compared to type II achalasia, type I had a significantly longer symptom duration prior to HRM (48 vs 24 months, p = 0.026) and lower rate of normal gastroscopy findings prior to HRM (12.2% vs 29.8%, p = 0.004).
Conclusion: The clinical and manometric presentation of achalasia in South Africa is similar to previous international reports, however the associated presence of autoimmune conditions within this cohort is much less than detailed elsewhere.
Keywords: achalasia, clinical presentation, comorbidities, manometry

Introduction

Achalasia is a rare oesophageal motility disorder with a global incidence of 0.78 cases per 100 000 person-years.[1] While the incidence in Africa is reportedly the lowest (0.09 cases per 100 000 person-years), this may be an underrepresentation due to a significant scarcity of data from the continent.[1] Achalasia is characterised by abnormal peristalsis and failure of lower oesophageal sphincter (LOS) relaxation due to the loss of inhibitory innervation of the myenteric plexus of Auerbach.[2,3] Although chronic inflammation of this nerve plexus has been described, the aetiology of primary achalasia is likely multifactorial. There appears be an association with autoimmune conditions as histological examination of the LOS shows evidence of an inflammatory response with T-cell infiltrates, with achalasia 3.6 times more likely to occur in patients with a co-existing autoimmune condition.[4-7] There exists a possible genetic predisposition as the condition has been reported in first degree relatives, monozygotic twins and patients with chromosomal abnormalities such as Down's syndrome.[8,9] Viral pathogens have also been postulated to trigger the development of achalasia.[10-12]

While achalasia may present at any age, the peak incidence of presentation is between 30 and 60 years of age.[13] The dominant symptom of dysphagia, typically for both fluids and solids, is gradually progressive and usually presents after some years of symptom compensation often culminating in unintentional loss of weight. Regurgitation of undigested food and commonly frothy saliva are regular complaints, especially when supine. Chest pain with achalasia may be due to a combination of progressive oesophageal distention, stasis oesophagitis, candidiasis or pill oesophagitis.[14,15] Endoscopy, contrast swallow and high-resolution oesophageal manometry (HRM) are integral in the diagnostic work-up. Evaluation by HRM

remains the gold standard with classification presently still into three manometric sub-types according to Chicago 4.0; type I (absent contractility), type II (pan-oesophageal pressurisation) and type III (spastic).[16,17] This differentiation into sub-types helps explain differences in presentation, treatment options and outcomes.

Our study aimed to evaluate presentation, endoscopic, radiological and manometric findings within a sub-Saharan achalasia cohort. We compared clinical and diagnostic variables between the three manometric achalasia subtypes and reviewed the prevalence of autoimmune conditions.

Methods

Study design

This was a retrospective descriptive cohort study of all patients diagnosed on high-resolution oesophageal manometry (HRM) with achalasia by the Oesophageal Motility Unit at Groote Schuur Hospital between 1 January 2012 and 31 December 2024. The objectives of this study were to document the symptomatology and spectrum of comorbidities present within a South African cohort of confirmed achalasia patients, including the clinical presentation, the duration of symptoms at time of diagnosis on initial HRM, patient comorbidities, and associated investigations (oesophagogastroduodenoscopy [EGD], HRM and contrast swallow). However, due to the shortage of motility services available in South Africa, this initial HRM followed prior intervention for achalasia (where achalasia diagnosis was initially based only on contrast swallow and EGD) in selected cases. LOS metrics, including the median resting LOS pressure and the median integrated relaxation pressure (IRP) were only included for analysis in patients without prior intervention for achalasia.

Study population

All adult patients (> 18 years of age) diagnosed with achalasia on oesophageal HRM during the study period were evaluated for possible inclusion. Patients with oesophageal motility disorders not classified as achalasia were excluded. Duration of presenting symptoms prior to diagnosis of achalasia was determined as the period prior to the initial confirmatory HRM only.

Patient demographics and results of relevant investigations (HRM, EGD and contrast swallow) of confirmed achalasia patients were documented with patient folder review confirming the presence of any comorbidities. In addition, chronic prescriptions were assessed with relevance to the treatment of any additional comorbidities.

Definitions

Achalasia diagnosis by high resolution manometry

HRM was performed using the Solar™ GI HRM system from Laborie Medical Technologies Corporation with a 24-channel water-perfused catheter, according to the modified protocol initially taken from the Chicago Classification version 3.0 and more recently version 4.0.[16] LOS metrics were captured and depending on the manometric pattern, categorised into achalasia sub-types (Figure 1).

Symptom duration and severity

Symptom duration was defined in months from symptom onset (as reported by the patient) to achalasia confirmation on HRM. The Eckardt score was used to define symptom severity (Addendum 1).[17]

Endoscopy

A retrospective review of available EGD reports were assessed as to whether the EGD was deemed normal by the attending endoscopist or if findings were noted in keeping-

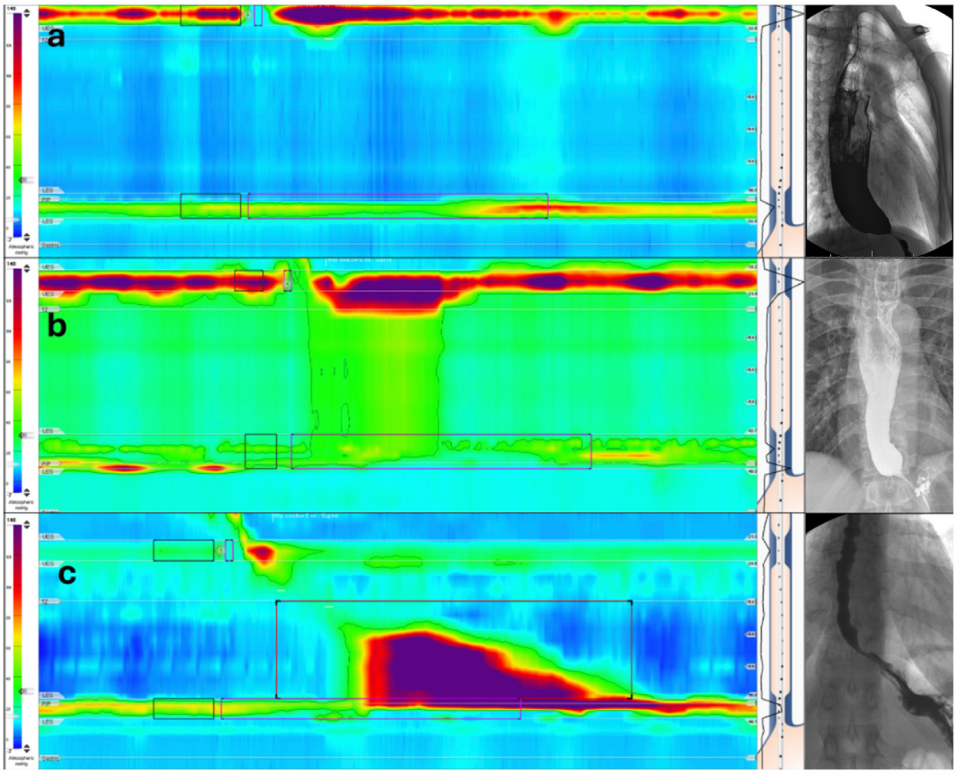

Figure 1: Characteristic high-resolution oesophageal manometry and contrast swallow findings in the three types of achalasia

In all three the median integrated relaxation pressure is above normal (> 18mmHg with 24 channel water-perfused catheter in a supine position using MMS Laborie software). a. Type I achalasia: 100% failed peristalsis; very dilated, "bird's beak". b. Type II achalasia: ≥ 20% pan-oesophageal pressurisation (note also the oesophageal shortening demonstrated by the upward movement of the lower oesophageal sphincter pressure line); moderate to very dilated, significant stasis. c. Type III achalasia (≥ 20% premature (spastic) contractions (distal latency in this image was 4.2 seconds (normal > 4.5 seconds); less dilatation, "cork-screw", tertiary contractions

with a possible oesophageal dysmotility disorder (including dilatation or tortuosity of the oesophagus, retained saliva/fluid/food, stasis oesophagitis and/or oesophageal candidiasis and a "tight" but intubatable OGJ).

Contrast swallow

Contrast swallow oesophagograms and reports were retrospectively reviewed on Picture Archiving and Communicating System (PACS) where available. Most were simple contrast swallows and not formal timed contrast swallow studies. While a bird's beak or rat's tail sign is almost pathognomonic of achalasia, if oesophageal dilatation, tortuosity, delayed oesophageal emptying with a contrast-fluid level or tertiary contractions were reported, a possible oesophageal dysmotility disorder was considered.[17] Normal contrast swallow reports by the reporting radiologist were noted as such.

Presence of comorbidities

Patient folders and prescription charts were retrospectively reviewed for the presence of any comorbidities. Within the comorbidities, special note was made of any autoimmune conditions.

Data analysis

Data analysis was done using Microsoft Excel and IBM SPSS Statistics (version 28.0.1.1). The following data categories were described using simple descriptive statistics: patient demographics, medical comorbidities, clinical presentation, including Eckhardt score and dysphagia duration and grade at presentation. Manometry, contrast swallow and EGD findings were also included. Parametric data were described using mean with standard deviation and non-parametric data were described using median with inter-quartile range. Univariate analysis to assess for associations between numerous parameters and type of achalasia was performed using the chi-squared test for categorical data and the Kruskal-Wallis test to allow for comparison of multiple groups with non-parametric data. Further post-hoc multiple comparison analyses using the Mann-Whitney U test were performed on any variables shown to be statistically significant. Spearman's Rank Correlation was used to assess for correlation between HRM metrics (LOSP and IRP) and total Eckardt symptom score. A p - value of < 0.05 was considered statistically significant.

Results

Patient demographics, presentation and comorbidities

During the study period, a total of 258 patients were diagnosed with achalasia on HRM (Table I). Within this achalasia cohort, 181 patients (70.2%) were newly diagnosed, and 77 (29.8%) underwent HRM only post-intervention (including 26 dilatations, 32 laparoscopic Heller's myotomies and 19 patients with both previous dilatations and Heller's myotomies). The age at initial diagnosis of achalasia was 47.0 years (IQR 35.0–69.3), with a narrow majority of female patients (136; 52.7%). Type III achalasia patients tended to be older at diagnosis, but this was not determined to be significant (Table II).

In the newly diagnosed group, the duration of symptoms overall was a median of 36.0 months (IQR 12.0–84.0 months). When symptom duration was reviewed as per achalasia subtype, type I and III achalasia patients had a longer symptom duration compared to type II achalasia. On post-hoc analysis the symptom duration difference was found to be statistically significant only for type I compared to type II ($p = 0.026$). Overall symptom severity at time of achalasia diagnosis on HRM was a median Eckardt score of 7 (IQR 6–9). While type II had a higher median Eckardt score, this was not determined significant when evaluated across the three achalasia sub-types (Table II).

At the time of achalasia diagnosis, 147 patients (57.0%) were being treated for one or more chronic comorbidities, most commonly hypertension, asthma and diabetes mellitus. Twelve patients (4.7%) had a known autoimmune condition, with a higher prevalence in type III achalasia but this did not reach statistical significance ($p = 0.337$) (Table II).

Table I: Patient demographics, comorbidities and symptom history at time of achalasia diagnosis on high resolution manometry (HRM)

Demographics	**Median**	**IQR**
Age (years)	47.0	35.0–69.3
	n	**%**
Female	136	52.7
Male	122	47.3
Comorbidities		
Hypertension	67	26.0
Diabetes mellitus	21	8.1
Asthma	22	8.5
Ischaemic heart disease	17	6.6
Tuberculosis	15	5.8
Mental healthcare user	12	4.7
HIV[a] positive	12	4.7
Chronic kidney disease	9	3.5
Other	76	29.5
Autoimmune conditions	12	4.7
Myasthenia gravis	2	
Polyarthritis rheumatica	2	
Hashimoto's thyroiditis	1	
Sarcoidosis	1	
Systemic lupus erythematosus	1	
Stephen-Johnsons syndrome	1	
Systemic sclerosis	1	
Type I diabetes mellitus	1	
Ulcerative colitis	1	
Von Willebrand disease	1	
Symptom History	**Median**	**IQR**
Symptom Duration Prior to HRM (months)	36.0	12.0–84.0
Total Eckardt Score	7.0	6.0–9.0

HIV – human immunodeficiency virus, HRM – high-resolution manometry

Table II: Univariate analysis of numerous factors for association with achalasia type

	Type I		Type II		Type III		*p*-value[#]	post-hoc *p*-value[$]		
	n	%	*n*	%	*n*	%		I vs II	I vs III	II vs III
Female Sex	73	55.7%	60	49.6%	3	50.0%	0.617			
Hypertension	36	27.5%	28	23.1%	3	50.0%	0.293			
Diabetes	9	6.9%	10	8.3%	2	33.3%	0.069			
Chronic kidney disease	4	3.1%	4	3.3%	1	16.7%	0.205			
Ischaemic heart disease	9	6.9%	7	5.8%	1	16.7%	0.569			
Mental healthcare user	8	6.1%	4	3.3%	0	0.0%	0.495			
HIV[a] Positive	6	4.6%	6	5.0%	0	0.0%	0.853			
Tuberculosis	11	8.4%	4	3.3%	0	0.0%	0.188			
Asthma	7	5.3%	14	11.6%	1	16.7%	0.162			
Autoimmune condition	5	3.8%	6	5.0%	1	16.7%	0.337			
Hiatus hernia on HRM[b] (*n* = 173)	15	11.5%	16	13.2%	2	33.3%	0.445			
Normal endoscopy	16	12.2%	36	29.8%	2	33.3%	**0.012**	**0.004**	0.158	0.736
Normal contrast swallow	3	2.3%	4	3.3%	0	0.0%	0.85			
Age	46.0 (35.0–61.0)		46.0 (36.0–62.0)		66.0 (60.0–75.0)		0.108			
Symptom duration (months)	48.0 (12.0–96.0)		24.0 (12.0–72.0)		96.0 (24.0–120.0)		**0.041**	**0.026**	0.365	0.133
Eckardt total score	7.0 (5.0–9.0)		8.0 (6.0–9.0)		6.5 (6.0–11.0)		0.478			
IRP[c] (mmHg) (*n* = 142)	25.0 (19.0–31.0)		28.0 (20.0–37.0)		23.0 (22.0–39.0)		0.156			
Resting LOSP[d] (mmHg) (*n* = 142)	30.5 (25.0–47.0)		41.0 (29.0–52.0)		36.0 (33.0–39.0)		0.093			

All numerical date represented as median with interquartile range. [a]HIV – human immunodeficiency virus, [b]HRM – high-resolution manometry, [c]IRP – integrated relaxation pressure, [d]LOSP – lower oesophageal sphincter pressure, [#]Kruskal-Wallis test, [$]Mann-Whitney U test for post-hoc analysis

Oesophagogastroduodenoscopy and contrast swallow

Of the 217 patients having EGD prior to their HRM, 54 (24.9%) were reported as normal. Normal endoscopy differed significantly across the three subtypes ($p = 0.012$). Compared to type II achalasia, type I achalasia had a statistically significantly smaller percentage of reported normal EGDs (12.2% vs 29.8%, $p = 0.004$) (Table II). While EGD is an integral investigation in dysphagia, in 41 patients the actual endoscopy reports were not available for review.

In 39 patients a contrast swallow was either not performed, or the imaging/report was unavailable for review. Of the contrast swallows accessible for review, only seven (3.2%) were reported normal. EGD was falsely negative (normal) in 24.9% of patients compared to only 3.2% of contrast swallows.

High-resolution manometry

HRM identified type I achalasia as most prevalent (50.8%; $n = 131$), followed by type II achalasia (46.9%; $n = 121$). Type III achalasia was confirmed on only 6 HRMs (2.3%).

LOS metrics were analysed in 142 patients (Table II). Although both the integrated relaxation pressure (IRP) and lower oesophageal sphincter pressure (LOSP) were higher in type II achalasia, this did not reach statistical significance. The presence of a hiatus hernia on HRM was noted in 33 patients (12.8%). The remaining 116 HRMs were excluded due to inaccurate LOS metrics (77 patients presented for HRM after a prior intervention for achalasia; in 39 patients the manometry catheter could not cross the OGJ).

Increasing IRP and Eckhardt's score demonstrated a weak positive correlation (Spearman's $p = 0.175$; $p = 0.039$) based on 140 paired observations. Contrastingly, LOSP and symptom severity had only a very weak positive, but not statistically significant, correlation (Spearman's $p = 0.038$; $p = 0.658$).

Discussion

This study on the clinical presentation of a manometrically confirmed achalasia cohort is the largest to our knowledge from sub-Saharan Africa. Only two additional regional studies exist, one from Algeria and a single sub-Saharan study from Zimbabwe (from 1985), highlighting the near complete absence of data from the African continent as regards achalasia.[18,19]

Within our cohort, the demographics and spread across the three achalasia subtypes was similar to international reports. With achalasia usually diagnosed between 25–60 years, our type I and II achalasia patients fell well within this age range, but the type III achalasia group were noticeably older, as has been documented elsewhere before.[20] Our overall incidence of associated autoimmune disease was only 4.7%, much lower than the 12.3–16.7% in other series.[4-7,21,22] While our type III achalasia patients had a higher prevalence of autoimmune disorders, our small numbers limit meaningful comparison. This unexpectedly low autoimmune disease incidence within our achalasia population may point to possibly underdiagnosing autoimmune disease locally, a different genetic population or may be highlighting poor documentation as a limitation of this retrospective study, with no clear conclusions possible.

Due to its rarity and initially insidious onset, achalasia has a mean diagnostic delay commonly reported internationally between two to five years.[6,23] Within this cohort the duration of reported symptoms was 3 years prior to presentation for diagnostic HRM which is somewhat surprising considering the very limited availability of oesophageal motility services in South Africa. This likely reflects referral bias due to the retrospective nature of this study in reviewing preselected

patients. The shorter preceding symptom duration and higher IRP and LOSP noted in the type II achalasia group (although the IRP and LOSP comparisons did not reach statistical significance) are in keeping with the accepted theory that type II achalasia is the precursor to type I achalasia.[24] As such higher pressures should be associated positively with severity of symptoms, keeping in mind that morphological shape with associated distal stasis is responsible for many of the chronic symptoms in achalasia.[25,26]

While HRM is accepted as the gold standard for diagnosing achalasia, locally EGD and contrast swallows still hold much diagnostic weight when suspecting an oesophageal dysmotility.[27] Our results suggest that early achalasia may be frequently missed (29.4%) on EGD. Type I achalasia, with the associated stasis and dilatation is more endoscopically obvious. Within this cohort, normal EGDs were reported not infrequently in type II and III achalasia. While this study was not designed to assess diagnostic accuracy, contrast swallow had a much-improved diagnostic rate due to its ability to simultaneously functionally assess oesophageal transit and review oesophageal anatomy, with only 3.2% of contrast swallows missing an oesophageal dysmotility within this cohort.

Despite limitations due to its retrospective nature and referral bias, this study is the first clinical description of a South African manometrically confirmed achalasia cohort. There are similarities in demographics, clinical presentation, investigative outcomes and achalasia subtype rates compared to international studies. Similar delays in diagnosis and availability of specialised investigations are noted. Our findings support the local relevance of contrast swallows complementing EGD when investigating for oesophageal dysmotility. This study documents the real-world scenario where lack of access to high-resolution oesophageal manometry in South Africa may result in patients diagnosed and treated for achalasia without prior HRM. This study should prompt further research into achalasia on the African continent and highlight the need to expand specialised oesophageal motility services across sub-Saharan Africa.

Conflict of interest

The authors declare no conflict of interest.

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Ethical approval

This study was approved by the Human Research Ethics Committee of the University of Cape Town (HREC 130/2022).

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Addendum 1: Eckardt Score[17]

Score	Dysphagia	Retrosternal pain	Regurgitation	Weight loss
0	None	None	None	None
1	Occasional	Occasional	Occasional	< 5kg
2	Daily	Daily	Daily	5–10kg
3	Each meal	Each meal	Each meal	> 10kg