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Concise Report

Clinical science

Anxiety and depression in patients with giant cell arteritis

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Abstract

Objectives: To compare the prevalence of anxiety and depression in patients with GCA with that in the general population, using the Hospital Anxiety and Depression Scale (HADS), and to identify independent predictors of these psychiatric manifestations in patients with GCA.

Methods: We conducted a cross-sectional study including all patients diagnosed with GCA followed during 1 year in a vasculitis outpatient clinic. The HADS and 36-item Short Form (SF-36) questionnaires were prospectively collected. Patients' HADS results were compared with an age- and gender-matched control group. HADS anxiety (HADS-A) and HADS depression (HADS-D) scores between 8 and 10 defined possible anxiety and depression and ≥11 defined probable anxiety and depression, respectively.

Results: We included 72 patients and 288 controls. Compared with controls, patients with GCA had a statistically significant higher prevalence of HADS-A ≥8 (48.6% *vs* 26.4%), HADS-A ≥11 (30.6% *vs* 12.2%) and HADS-D ≥11 (33.3% *vs* 18.1%). GCA was an independent predictor of HADS-A ≥8 [odds ratio (OR) 3.3 (95% CI 1.9, 5.9)], HADS-A ≥11 [OR 3.8 (95% CI 2.0, 7.4)] and HADS-D ≥11 [OR 2.6 (95% CI 1.4, 4.7)]. Among patients with GCA, a negative correlation was observed between HADS-A/D and SF-36 mental health scores ($r = -0.780$ and $r = -0.742$, respectively). Glucocorticoid therapy was a predictor of HADS-A ≥8 [OR 10.4 (95% CI 1.2, 94.2)] and older age of HADS-D ≥8 [OR 1.2 (95% CI 1.1, 1.3)] and HADS-D ≥11 [OR 1.1 (95% CI 1.0, 1.2)].

Conclusions: Compared with the general population, patients with GCA have a higher prevalence of anxiety and depression and GCA is an independent predictor of these symptoms. Glucocorticoid treatment and older age are predictors of anxiety and depression, respectively, in patients with GCA.

Lay Summary

What does this mean for patients?

Giant cell arteritis (GCA) is a condition that causes inflammation of the arteries, most frequently in the head and neck. It affects predominantly the elderly and can potentially lead to stroke and blindness. Both GCA-related symptoms and treatment with glucocorticoids can impact mental health. We compared the prevalence of anxiety and depression in individuals with GCA with that of the general population using the Hospital Anxiety and Depression Scale (HADS). We found that GCA was an important contributor to a higher prevalence of anxiety and depression in this group compared with the general population. We also explored which aspects contributed the most to anxiety and depression in people with GCA. We found that treatment with glucocorticoids was a predictor for anxiety and older age was a predictor for both anxiety and depression. Moreover, we described a negative correlation between anxiety/depression scores and quality of life among people with GCA, which suggests an impact of mental health on overall well-being. These findings highlight the burden of anxiety and depression in GCA, emphasizing the need for awareness and physician attention to mental health in this population in order to improve overall care and elevate the quality of life for individuals with GCA.

Keywords: giant cell arteritis, anxiety, depression, Hospital Anxiety and Depression Scale.

Key messages

- Patients with GCA have a higher prevalence of anxiety and depression than the general population.
- HADS anxiety and depression scores have a strong negative correlation with SF-36 scores in patients with GCA.
- Glucocorticoid treatment and older age are predictors of anxiety and depression, respectively, in GCA.

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Introduction

GCA is the primary systemic vasculitis affecting the elderly [1]. It typically presents with an abrupt onset and potentially severe manifestations, including permanent visual loss and stroke, which can negatively impact quality of life (QOL) and mental well-being [1, 2]. Well-established research has underscored the intricate relationship between chronic systemic inflammation, found in conditions like GCA, and the increased risk of depression and anxiety [3]. Moreover, its treatment, which includes glucocorticoids (GCs), can induce these same psychiatric manifestations [1, 4]. Therefore, both GCA and its treatment may impact mental health.

Depression is known to negatively impact QOL, treatment adherence and mortality [5, 6]. Higher rates of depression have been previously reported in patients with primary systemic vasculitis compared with RA, PsA and AS [7]. A few studies have specifically explored the occurrence of depression in GCA but relied on self-reporting or inadequately validated measuring tools [8–10]. In addition, the majority of studies currently assessing QOL and mental status in patients with GCA use the 36-item Short Form Health Survey (SF-36), which is a generic and non-disease-specific patient-reported outcome (PRO) with a Mental Health subscale (SF-36 MH) [11, 12]. In order to mitigate these methodological shortcomings in the evaluation of anxiety and depression in GCA, other forms of assessment are warranted. The Hospital Anxiety and Depression Scale (HADS) is a validated PRO [13] that has been successfully used to evaluate the mental health of patients with Takayasu arteritis, Behçet's disease and ANCA-associated vasculitis [14–16]. However, to the best of our knowledge, its value in patients with GCA remains unexplored.

This study aims to compare the prevalence of anxiety and depression in patients with GCA with that of the general population using the HADS questionnaire and to identify disease features associated with these psychiatric manifestations in GCA.

Materials and methods

Study population

All patients with an established diagnosis of GCA followed between January 2019 and January 2020 in the vasculitis outpatient clinic of a university hospital in Lisbon, Portugal, were included in this cross-sectional study. Patients had to fulfil either the ACR 1990 classification criteria for GCA, have a temporal artery biopsy consistent with GCA or have the presence of a halo sign in an ultrasound of the temporal or axillary arteries. An age- and gender-matched control group, with a ratio of 1 case per 4 controls, was retrieved from the general population included in the EpiReumaPT study, the largest Portuguese epidemiologic study on rheumatic diseases [17].

Data collection and assessment of PROs

Data regarding demographic, clinical and laboratory characteristics of patients with GCA at baseline were obtained from the Rheumatic Diseases Portuguese Register of Vasculitis (Reuma.pt/Vasculitis) [18]. Demographic information, past medical history data and HADS results from participants in the EpiReumaPt without inflammatory rheumatic diseases were also retrieved [17].

All patients with a diagnosis of GCA underwent prospective assessment using two questionnaires—HADS and SF-36—at a single timepoint during the study period, regardless of the disease duration or activity. Both questionnaires were administered during a unique medical consultation. In instances where patients encountered difficulties completing the questionnaires autonomously, the physician provided assistance to ensure accurate and thorough responses. The HADS questionnaire is divided into two domains: HADS-A, for anxiety, and HADS-D, for depression. HADS-A and HADS-D scores of 8–10 defined possible cases of anxiety and depression and scores $\geq$11 defined probable cases of anxiety and depression [13]. The SF-36 comprises eight domains: four physical [physical functioning (PF), role physical (RP), bodily pain (BP) and general health (GH)] and four mental [vitality (VT), social functioning (SF), role emotional (RE) and mental health (MH)]. Physical and mental component summaries and each individual scale can be calculated, ranging from 0 to 100, with higher scores indicating a better QOL.

Statistical analysis

Univariate analysis was performed using the chi-squared, Fisher's exact, Student's *t* or Mann–Whitney U test. The association between continuous variables was assessed using Spearman's correlation coefficient. Multivariate analysis, using binary logistic regression modelling, was performed to investigate if GCA was a predictor of anxiety and depression and also to identify independent predictors of HADS $\geq$8 and HADS $\geq$11 in patients with GCA. The continuous variable's linearity regarding the dependent variable's logit was assessed via the Box–Tidwell procedure. SPSS version 25 (IBM, Armonk, NY, USA) was used for statistical analysis and the significance level was defined as a two-sided *P*-value <0.05.

Ethical approval

This study was approved by the Lisbon Academic Medical Centre Ethics Committee (reference 254/19). All patients provided written informed consent before inclusion in the study.

Results

Patient characteristics

We included 72 patients with a diagnosis of GCA, 52 (72.2%) females, with a mean age of 78.3 years (S.D. 7.7) and a median time of disease of 2.7 years [interquartile range (IQR) 5.2] (Table 1). The matched control group consisted of 288 individuals. Compared with controls, patients with GCA had a higher prevalence of neoplastic disease and a lower prevalence of hyperuricaemia/gout and history of mental disease. No other significant differences between the two groups were found regarding comorbidities.

Anxiety and depression in patients with GCA compared with controls

Regarding anxiety in GCA patients, 35 (48.6%) had a HADS-A score $\geq$8 and 22 (30.6%) had a HADS-A score $\geq$11. When compared with the control population, patients with GCA had higher median HADS-A scores [7 (IQR 7) *vs* 5

Table 1. Clinical features of patients with GCA and matched controls

Characteristics	Patients ($n=72$)	Controls ($n=288$)	*P*-value
Demographics			
Age, years, mean (S.D.)	78.3 (7.7)	77.4 (8.4)	0.186
Female, *n* (%)	52 (72.2)	208 (72.2)	1.000
Comorbidities, *n* (%)			
Arterial hypertension	49 (68.0)	171 (60.0)	0.209
Diabetes mellitus	21 (29.2)	63 (22.2)	0.213
Hyperuricemia/gout	2 (2.8)	30 (10.8)	**0.036**
Neoplastic disease	13 (18.1)	19 (6.6)	**0.002**
Thyroid disease	5 (6.9)	28 (9.9)	0.442
Previous history of mental disease	4 (5.6)	47 (16.4)	**0.018**
HADS score			
HADS-A, median (IQR)	7 (7)	5 (5)	**<0.001**
HADS-A ≥8, *n* (%)	35 (48.6)	76 (26.4)	**<0.001**
HADS-A ≥11, *n* (%)	22 (30.6)	35 (12.2)	**<0.001**
HADS-D, median (IQR)	7 (7)	5 (5)	**0.013**
HADS-D ≥8, *n* (%)	35 (48.6)	107 (37.2)	0.075
HADS-D ≥11, *n* (%)	24 (33.3)	52 (18.1)	**0.004**

Statistically significant differences ($P<0.05$) in bold.

(IQR 5), $P<0.001$] and a higher prevalence of HADS-A ≥8 (48.6% *vs* 26.4%, $P<0.001$) and HADS-A ≥11 (30.6% *vs* 12.2%, $P<0.001$). Similarly, in terms of depression in GCA patients, HADS-D ≥8 was observed in 35 (48.6%) patients and HADS-D ≥11 in 24 (33.3%) patients. Moreover, patients with GCA had higher median HADS-D scores than controls [7 (IQR 7) *vs* 5 (IQR 5), $P=0.013$], as well as a higher frequency of HADS-D ≥11 (33.3% *vs* 18.1%, $P=0.004$). The proportion of patients with HADS-D ≥8 did not differ significantly between groups (48.6% *vs* 37.2%, $P=0.075$), despite the numerically higher prevalence in the GCA group (Table 1).

Multivariate analyses adjusted for GCA, neoplastic disease, hyperuricaemia/gout and history of mental disease showed that GCA and a history of mental disease were independent predictors of HADS-A ≥8 [OR 3.3 (95% CI 1.9, 5.9) and OR 4.9 (95% CI 2.5, 9.5), respectively] and HADS-A ≥11 [OR 3.8 (95% CI 2.0, 7.4) and OR 2.5 (95% CI 1.1, 5.5), respectively] (Supplementary Table S1, available at *Rheumatology Advances in Practice* online). Only GCA was a predictor of HADS-D ≥11 [OR 2.6 (95% CI 1.4, 4.7)] (Supplementary Table S1, available at *Rheumatology Advances in Practice* online).

Association between HADS and SF-36 scores in patients with GCA

Patients with GCA with HADS-A ≥8 and HADS-A ≥11 had lower SF-36 scores in all categories when compared with patients with HADS-A <8 and HADS-A <11 (only the RP limitation category did not reach statistical significance). Similarly, patients with HADS-D ≥8 and HADS-D ≥11 had lower SF-36 scores in all categories compared with patients with HADS-D <8 and HADS-D <11 (Table 2). A correlation between HADS-A/D and SF-36 mental health scores was observed (HADS-A: $r=-0.780$, HADS-D: $r=-0.742$; both $P<0.001$).

Contributing factors for anxiety and depression in GCA

Patients with HADS-A or HADS-D ≥8 were more frequently under GC treatment than patients with HADS-A or HADS-D <8 (97.1% *vs* 78.4% for both; $P=0.028$) (Table 2). All patients with HADS-A or HADS-D ≥11 were on GCs, unlike those with HADS-A or HADS-D <11 (100% *vs* 82.0%, $P=0.049$ and 100% *vs* 81.3%, $P=0.023$, respectively).

Patients with HADS-A ≥8 had higher median ESRs (36 *vs* 25 mm/h, $P=0.027$) and a lower prevalence of diabetes mellitus (40.5% *vs* 17.1%, $P=0.029$) compared with patients with HADS-A <8. Patients with HADS-D scores ≥8 and ≥11 were more likely to be older than patients with HADS-D scores <8 and <11 (81.9 *vs* 74.9 years, $P<0.001$ and 82.3 *vs* 76.3 years, $P<0.001$, respectively). Permanent visual loss, stroke, disease duration for at least 1 year, current treatment with high doses of GCs (>30 mg/day) or treatment with GCs for at least 1 year did not significantly impact the HADS scores. Of note, when analysing the influence of these same variables in the SF-36 scores, we observe that patients receiving high doses of GCs (>30 mg/day) had higher SF-36 GH scores compared with those not undergoing this treatment (50.9 *vs* 35.7, $P=0.002$). Conversely, patients under GC treatment for at least one year had lower SF-36 GH scores compared with those with <1 year of GCs (36.0 *vs* 47.9, $P=0.012$). The remaining variables showed no significant impact on the SF-36 scores (Supplementary Table S2, available at *Rheumatology Advances in Practice* online).

Multivariate analyses adjusted for sex, age, disease duration >1 year and treatment with GCs showed that only GC therapy was a predictor of HADS-A ≥8 [OR 10.4 (95% CI 1.2, 94.2)] and only age was a predictor of HADS-D ≥8 [OR 1.2 (95% CI 1.1, 1.3)] and HADS-D ≥11 [OR 1.1 (95% CI 1.0, 1.2)]; no predictors were identified for HADS-A ≥11 (Supplementary Table S3, available at *Rheumatology Advances in Practice* online). Moreover, age and HADS-A and HADS-D scores showed a positive correlation ($r=0.26$, $P=0.03$ and $r=0.53$, $P<0.001$, respectively).

Discussion

This study shows that anxiety and depression are more prevalent in patients with GCA compared with the general population, with almost double the prevalence, as indicated by the HADS questionnaire. These results are consistent with previous studies showing an increased risk of depression in patients with GCA compared with controls [7, 9, 10]. However, many of these studies relied on self-reporting rather than employing reliable measurement techniques to determine the presence of depression [10]. In addition, in the study conducted by Brezinova *et al.* [9], depression was evaluated in only 13 patients with GCA and using the Beck Depression Inventory (BDI), a self-report questionnaire designed to assess the severity of depressive symptoms, including cognitive, affective and somatic symptoms. This contrasts with the HADS questionnaire, which was developed to exclude physical indicators of psychological distress, making it potentially more suitable for patients with GCA, where somatic features may overlap with psychological symptoms [13].

Table 2. Results of univariate analysis comparing patients with HADS ≥8 and HADS <8 and HADS ≥11 and HADS <11.

Characteristics	HADS-A						HADS-D					
	HADS-A <8 ($n=37$)	HADS-A ≥8 ($n=35$)	*P*-value	HADS-A <11 ($n=50$)	HADS-A ≥11 ($n=22$)	*P*-value	HADS-D <8 ($n=37$)	HADS-D ≥8 ($n=35$)	*P*-value	HADS-D <11 ($n=48$)	HADS-D ≥11 ($n=24$)	*P*-value
Demographic data												
Age, years, mean (S.D.)	76.8 (7.3)	79.9 (8.0)	0.093	77.7 (7.5)	79.6 (8.3)	0.304	74.9 (7.4)	81.9 (6.4)	**0.001**	76.3 (7.5)	82.3 (6.8)	**0.001**
Female, *n* (%)	27 (73.0)	25 (71.4)	0.884	34 (68)	18 (81.8)	0.228	26 (70.3)	26 (74.3)	0.704	35 (72.9)	17 (70.8)	0.852
Clinical characteristics of the disease												
Permanent vision loss, *n* (%)	6 (16.2)	6 (17.1)	0.916	9 (18.0)	3 (13.6)	0.647	6 (16.2)	6 (17.1)	0.916	8 (16.7)	4 (16.7)	1.000
Stroke, *n* (%)	0 (0)	4 (11.4)	0.051	3 (6.0)	1 (4.5)	1.000	0 (0)	4 (11.4)	0.051	2 (4.2)	2 (8.3)	0.597
Large vessel involvement by imaging, *n* (%)	11 (29.7)	10 (28.6)	0.914	15 (30.0)	6 (27.3)	0.815	12 (32.4)	9 (25.7)	0.531	14 (29.2)	7 (29.2)	1.000
Disease duration, years, median (IQR)	1.0 (4.2)	2.9 (4.9)	0.383	2.1 (5.3)	3.1 (4.5)	0.334	1.0 (4.0)	3.1 (6.5)	0.481	2.5 (5.2)	2.8 (6.6)	0.282
Disease duration >3 months, *n* (%)	28 (75.7)	29 (82.9)	0.453	38 (76.0)	19 (86.4)	0.529	29 (78.4)	28 (80.0)	0.866	36 (75.0)	21 (87.5)	0.218
Disease duration >1 year, *n* (%)	22 (59.5)	25 (71.4)	0.286	24 (57.1)	16 (72.7)	0.221	22 (59.5)	25 (71.4)	0.286	23 (57.5)	17 (70.8)	0.286
Comorbidities, *n* (%)												
Atherosclerosis	0 (0)	3 (8.6)	0.110	2 (4)	1 (4.5)	1.000	1 (2.7)	2 (5.7)	0.609	2 (4.2)	1 (4.2)	1.000
Atrial fibrillation	1 (2.7)	1 (2.9)	1.000	1 (2)	1 (4.5)	0.521	0 (0.0)	2 (5.7)	0.233	0 (0)	2 (8.3)	0.108
Cerebrovascular disease	2 (5.4)	4 (11.4)	0.423	3 (6)	3 (13.6)	0.361	1 (2.7)	5 (14.3)	0.102	2 (4.2)	4 (16.7)	0.091
Chronic renal disease	3 (8.1)	1 (2.9)	0.615	3 (6.0)	1 (4.5)	1.000	3 (8.1)	1 (2.9)	0.615	3 (6.3)	1 (4.2)	1.000
Diabetes mellitus	15 (40.5)	6 (17.1)	**0.029**	18 (36.0)	3 (13.6)	0.054	12 (32.4)	9 (25.7)	0.531	15 (31.3)	6 (25)	0.582
Hypercholesterolaemia	5 (13.5)	4 (11.4)	1.000	7 (14)	2 (9.1)	0.712	5 (13.5)	4 (11.4)	1.000	7 (14.6)	2 (8.3)	0.708
Hypertension	26 (70.3)	23 (65.7)	0.679	35 (70)	14 (63.6)	0.594	25 (67.6)	24 (68.6)	0.927	33 (68.8)	16 (66.7)	0.858
Hyperuricaemia/gout	2 (5.4)	0 (0)	0.493	2 (4.0)	0 (0)	1.000	1 (2.7)	1 (2.9)	1.000	1 (2.1)	1 (4.2)	1.000
Ischaemic cardiac disease	1 (2.7)	3 (8.6)	0.350	3 (6)	1 (84.5)	1.000	1 (2.7)	3 (8.6)	0.350	2 (4.2)	2 (8.3)	0.597
Mourning	1 (2.7)	5 (14.7)	0.098	2 (4.1)	4 (18.2)	0.070	2 (5.6)	4 (11.4)	0.429	3 (6.4)	3 (12.5)	0.399
Neoplastic disease	5 (13.5)	8 (22.9)	0.303	23 (7.6)	9 (15.8)	0.053	5 (13.5)	8 (22.9)	0.303	25 (8.8)	7 (9.2)	0.761
Obesity	0 (0)	1 (2.9)	0.486	0 (0)	1 (4.5)	0.306	0 (0.0)	1 (2.9)	0.486	0 (0)	1 (4.2)	0.333
Peripheral arterial disease	1 (2.7)	2 (5.7)	0.609	2 (4)	1 (4.5)	1.000	0 (0.0)	3 (8.6)	0.110	1 (2.1)	2 (8.3)	0.256
Previous history of mental disease	2 (5.4)	2 (5.7)	1.000	3 (6.0)	1 (4.5)	1.000	1 (2.7)	3 (8.6)	0.350	3 (6.3)	1 (4.2)	1.000
Thyroid disease	3 (8.1)	2 (5.7)	1.000	3 (6.0)	2 (9.1)	0.638	3 (8.1)	2 (5.7)	1.000	4 (8.3)	1 (4.2)	0.659
Laboratory results, median (IQR)												
CRP, mg/dl	0.4 (0.5)	0.5 (1.2)	0.556	0.4 (0.6)	0.4 (1.2)	0.959	0.4 (0.5)	0.4 (0.9)	0.581	0.4 (0.5)	0.3 (1.2)	0.311
ESR, mm/h	25 (20)	36 (33)	**0.027**	28 (35)	35 (25)	0.530	25.5 (26.5)	35 (43.5)	0.318	28 (28)	35 (45)	0.985
GC treatment at the time of questionnaires												
Patients on GCs, *n* (%)	29 (78.4)	34 (97.1)	**0.028**	41 (82.0)	22 (100)	**0.049**	29 (78.4)	34 (97.1)	**0.028**	39 (81.3)	24 (100)	**0.023**
Current GC dose, mg, median (IQR)[a]	10 (42.5)	7.5 (42.5)	0.994	10 (45)	7.5 (27.5)	0.459	18.8 (42.5)	7.5 (42.5)	0.827	12.5 (45)	7.5 (40)	0.631
Patients on GCs >30 mg, *n* (%)[a]	11 (37.9)	10 (31.3)	0.583	15 (38.5)	15 (38.5)	0.377	11 (39.3)	10 (30.3)	0.462	14 (36.8)	7 (30.4)	0.610
Treatment with GCs ≥1 year, *n* (%)	16 (53.3)	24 (70.6)	0.155	30 (60)	17 (77.3)	0.156	16 (55.2)	24 (68.6)	0.270	29 (60.4)	18 (75)	0.220
SF-36 questionnaire, median (IQR)												
Physical function	55.0 (52.5)	25.0 (55.0)	**0.003**	55.0 (46.3)	20.0 (51.3)	**0.003**	70.0 (50.0)	20.0 (50.0)	**0.001**	55.0 (48.8)	10.0 (53.8)	**0.001**
Role limitation (physical)	50.0 (75.0)	25.0 (50.0)	0.103	40.6 (64.1)	9.4 (32.8)	**0.018**	50.0 (59.4)	18.8 (31.2)	**0.001**	50.0 (62.5)	9.4 (25.0)	**0.001**
Role limitation (emotional)	58.3 (75.0)	25.0 (66.7)	**0.006**	50.0 (75.0)	20.8 (66.7)	**0.012**	66.7 (66.7)	25.0 (41.7)	**0.001**	58.3 (72.9)	16.7 (25.0)	**0.001**
Social function	87.5 (50.0)	50.0 (37.5)	**0.001**	75.0 (50.0)	37.5 (22.0)	**0.001**	87.5 (37.5)	50.0 (25.0)	**0.001**	75.0 (50.0)	43.8 (34.4)	**0.001**
Pain	62.0 (43.0)	31.0 (29.0)	**0.001**	51.0 (40.0)	31.0 (42.5)	**0.017**	62.0 (43.0)	31.0 (40.0)	**0.001**	51.0 (39.0)	26.5 (47.3)	**0.006**
Vitality	56.3 (42.5)	25.0 (33.8)	**0.001**	50.0 (38.8)	18.8 (25.0)	**0.001**	56.2 (32.5)	25.0 (27.5)	**0.001**	50.0 (38.4)	16.9 (25.0)	**0.001**
Mental health	80.0 (26.5)	40.0 (26.4)	**0.001**	69.0 (32.6)	30.0 (27.8)	**0.001**	76.0 (32.4)	40.0 (32.0)	**0.001**	69.0 (35.2)	30.0 (30.2)	**0.001**
General health	50.0 (27.5)	30.0 (20.0)	**0.001**	47.5 (25.0)	30.0 (26.3)	**0.001**	50.0 (30.0)	30.0 (20.0)	**0.001**	45.0 (25.0)	30.0 (23.8)	**0.001**

Statistically significant differences ($P < 0.05$) in bold.

[a] Prednisolone equivalent.

To our knowledge, our study is the first to evaluate mental health in GCA using the HADS. So far, measurements of QOL and mental status in GCA have mainly been based on generic PROs, namely the SF-36 [2, 11, 12, 19]. Due to its lack of specificity, the SF-36 has demonstrated limitations in detecting disability related to crucial features of GCA, such as visual loss [19]. However, it is a well-recognized and validated tool for measuring QOL. In our cohort, the HADS and SF-36 scores showed a strong negative correlation, supporting the idea that the HADS is a suitable PRO for patients with GCA and highlighting the fact that anxiety and depression are associated with worse QOL. Moreover, our findings using the HADS in patients with GCA align with other studies on systemic vasculitis, such as Takayasu arteritis [14] and Behçet's disease [15]. These studies have also reported higher rates of anxiety and depression measured by the HADS compared with control groups, further supporting the usefulness of this PRO in assessing psychological well-being in vasculitis.

Regarding the contributing factors for anxiety and depression in patients with GCA, our study revealed an association between higher ESR and anxiety, suggesting that systemic inflammation may contribute to the development of psychiatric symptoms [3]. Moreover, we demonstrated that elderly patients are at a higher risk of experiencing depression, which could be attributed to a greater prevalence of risk factors such as bereavement, social isolation, impairment and somatic diseases [16]. Lastly, the significant association found between GC treatment and anxiety and depression is to be expected, as these are known possible adverse events of this therapy [4].

Some potential limitations of this study should be noted. The HADS assessment, regardless of disease duration or activity, and the relatively small sample size might have limited the ability to identify additional contributing factors of anxiety and depression linked to GCA. Moreover, our analyses focused solely on results from the HADS questionnaire, lacking final diagnostic confirmation of depression or anxiety by a clinician. To delve deeper into the correlation between GCA and mental health conditions, larger-scale investigations with formal clinical assessments at specific time points during the disease course are imperative. Future studies should also investigate whether providing timely psychological support to individuals with elevated HADS scores can improve the overall outcomes in GCA. Nevertheless, our study was able to make a valuable contribution to the management of GCA by revealing a strong association between this disease and anxiety and depression. As a practical result, physicians should now be encouraged to incorporate the assessment of these psychiatric manifestations in the routine care of patients with GCA, particularly in patients with prior mental conditions and older patients undergoing GC treatment. The HADS questionnaire could be easily applied in clinical practice, with patients scoring higher values potentially benefiting from psychiatric assessment and subsequent treatment.

In conclusion, this study sheds light on the significant burden of anxiety and depression in patients with GCA and identifies potential factors associated with these psychiatric symptoms. These findings underscore the importance of addressing mental well-being in managing this disease and providing appropriate support to enhance patients' overall health and QOL.

Supplementary material

Supplementary material is available at *Rheumatology Advances in Practice* online.

Data availability

The data underlying this article will be shared upon reasonable request to the corresponding author.

Authors' contributions

Joana Martins-Martinho: conceptualization; methodology; writing original draft; André Ponte: conceptualization; methodology; review and editing; Eduardo Dourado: investigation; formal analysis; review and editing; Nikita Khmelinskii: investigation; review and editing; Sofia C Barreira: investigation; review and editing; Ana R Cruz-Machado: investigation; review and editing Carla Macieira: investigation; review and editing; Vítor Teixeira: investigation; review and editing; Ana M Rodrigues: investigation; review and editing; Diogo Telles-Correia: conceptualization; review and editing; João E Fonseca: supervision; review and editing; Cristina Ponte: conceptualization; methodology; supervision; review and editing.

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