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DEPRESSION AND DEPRESSIVE SYMPTOMS IN RHEUMATOID ARTHRITIS PATIENTS: AN ANALYSIS OF THEIR OCCURRENCE AND DETERMINANTS

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SUMMARY

The objectives were to determine the differences in depressive symptoms and depression between rheumatoid arthritis (RA) and osteoarthritis (OA) patients, and to analyse the contribution of sociodemographic and clinical variables to depression in RA patients. The responses of 60 Egyptian RA patients and 40 patients with OA of the knees to the Symptom Checklist-90-R Depression subscale were compared. The proportions of patients from both groups confirmed by a psychiatric interview to be clinically depressed according to the DSM-III-R criteria were also compared. The contributions of sociodemographic and disease variables to depressive symptoms and clinical depression in RA patients were explored by multiple linear and logistic regression, respectively. RA patients showed significantly higher depression scores than OA patients ($P = 0.001$). The difference was unaffected by controlling for the effects of age, sex, disease duration and the sociodemographic covariates. A depressive disorder was clinically confirmed in 23% of RA patients and 10% of OA patients. The erythrocyte sedimentation rate (ESR), being unmarried and an urban residence were significant predictors of depressive symptoms ($P < 0.05$), while being unmarried ($P < 0.05$, OR = 2.1) and HAQ disability ($P < 0.01$, OR = 3.8) were significant predictors of clinical depression in RA patients. RA patients have significantly more depressive symptoms and tend to be more clinically depressed than OA patients. The contribution of some sociodemographic and clinical variables to depression in RA patients was modest, albeit significant.

KEY WORDS: Rheumatoid arthritis, Osteoarthritis, Depression, Psychosocial.

DEPRESSION has long been recognized in rheumatoid arthritis (RA). In 1969, a leading article in the *British Medical Journal* [1] claimed that it is a feature of the disease, although at that time there was no clinical evidence for this. Subsequently, a multitude of studies were published estimating the prevalence of depression in RA patients from 14 to 46% [2–8].

Two methodological factors may explain this disparity of prevalence figures. The first is the use of clinical interviews *vs* questionnaires and psychological inventories. Standardized interviews applying criteria for the diagnosis of depression in RA have generally produced lower prevalence rates than questionnaires [9], which indicates that depressive symptoms are more common in RA patients than clinical depression. The second factor is 'criterion contamination', which is the overlap between depressive symptoms and the manifestations and consequences of RA (fatigue; sleeplessness; work disability), as found in many commonly used psychological inventories [10–12]. Therefore, these inventories would overestimate the occurrence of depression in RA patients [13]. Clinical interviews are also not immune to criterion contamination, since the type of criteria and the instructions for their application may overestimate depression in RA patients [5, 6, 14].

In studies which have adequately assessed depression in RA independently of physical health, prevalence figures converged at around 20%, which is 3–5 times higher than the rates for the normal population [6, 15]. However, while some chronic non-rheumatic diseases would not differ in the prevalence of depression from RA [6, 16], essentially no studies have attempted to determine the situation in more prevalent rheumatic diseases such as osteoarthritis (OA) [7]. Also, most of the studies examining depression in RA have failed to determine the relative contribution of disease and sociodemographic variables to the depressed mood of RA patients [17].

This study was therefore undertaken to compare depressive symptoms and depression in RA and OA patients, and to determine the disease and sociodemographic variables that contribute significantly to depressive symptoms and depression in RA patients. A contemporary view of depression is that it is not a disease, but the extreme end of a continuum of psychological distress [6]; therefore, high scores on a valid depression measure would imply, if not detect, a depressive disorder. However, to avoid the pitfalls of other studies, a differentiation between depressive symptoms (scores on the depression measure) and clinical depression (confirmed by a psychiatrist) was made throughout this work.

PATIENTS AND METHODS

Patients

Subjects included in the study were a primary group of 60 RA patients, diagnosed according to the 1987

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ARA criteria [18], and a comparative group of 40 patients with OA of the knees, diagnosed according to the clinical and radiographic criteria [19]. Both groups were recruited consecutively from the rheumatology and rehabilitation out-patient clinics of Cairo and Minia university hospitals in Egypt. Exclusion criteria included organic brain disease, major communicative disorders and chronic disabling conditions other than the diseases involved. Informed consent was taken from the patients before being studied. All patients approached agreed to participate.

Methods

Interviews. An interview by one of the authors (AMA) was chosen as the primary method for obtaining data. The interviewer administered the checklists and examined the patients to establish the diagnosis according to the criteria and record the clinical data. The interview and examination took $\sim$1.5 h for each patient, and were carried out on the same day. A second interview by a psychiatrist, who was blinded to the patients' diagnoses and scores on the depression questionnaire, was subsequently arranged for patients who were found to have high scores on the depression scale (according to the cut-off point described later). Diagnosis was determined according to the DSM-III-R criteria [20].

Clinimetry. The Ritchie articular index (RAI) [21] and the grip strength were calculated. Full general and locomotor system examinations were also carried out to determine the presence of extra-articular manifestations.

Investigations. Complete blood counts, 1 h erythrocyte sedimentation rate (ESR) (Westergren) and rheumatoid factor (latex) were performed on the day of the interview in both groups. X-rays of the hands and feet were taken for the RA group, and of the knees for the OA group.

Disease variables. Pain was assessed by a Numerical Rating Scale (NRS), whereby patients rated their pain grade on the interview day verbally, from 0 to 10. In populations with a high rate of illiteracy, the NRS would be more applicable and reliable than the more popular Visual Analogue Scale [22]. RA activity was assessed by a modification of the composite Index of Disease Activity [23], comprising the ESR, grip strength, pain grade, articular index and morning stiffness. The scores of the five individual measures were also used separately in the analysis. Disease severity was assessed by the ARA X-ray staging [24], as well as by rheumatoid factor seropositivity and the presence of extra-articular manifestations in the RA group. Functional assessment was carried out according to the ARA classification [24]. In addition, an Arabic version of the Health Assessment Questionnaire-Disability Index (HAQ) [25] was applied. This measure rates patients' self-reports of limitations in activities of daily living, yielding a total score from 0 (no disability) to 3 (maximum disability).

Assessment of depression. The Arabic version [26] of the depression subscale of the Symptom Checklist-90-Revised (SCL-90-R-D) [27] was used to assess depressive symptoms in both groups of patients. This subscale includes 13 items which rate depressive symptoms during the past week from 0 (never) to 4 (always). The SCL-90-R has undergone extensive comparison and validation with other measures. Reliability and validity are established for medical as well as psychiatric populations, with an internal consistency coefficient for the depression subscale of 0.90 [27, 28]. The SCL-90-R was found to be reliable, valid and applicable in RA patients, as it primarily measures psychological distress independent of RA disease factors [29].

Statistical analysis

Data analysis was by the Statistical Package of the Social Sciences (SPSS for Windows 6.1) [30]. Two-tailed tests were used and statistical significance was set at the conventional 0.05 level.

Group differences. The raw scores of both groups on the SCL-90-R-D were compared by Student's *t*-test, and subsequently used in an analysis of covariance to test the difference in depressive symptoms between the two groups, controlling for the effects of covariates. A cut-off point of 60 on the *T*-scores of the SCL-90-R-D was used to identify patients with a probable depressive disorder [29]. The proportions of patients confirmed to be clinically depressed by psychiatric interview were compared by the χ^2 test and the odds ratio (OR). Since this study has a case–control design, the OR can be looked on as the relative risk of depression in one group *vs* the other [31].

Determinants of depression in RA. Pearson product moment correlations were carried out prior to regression analysis. Two regression models, linear and logistic, were used to analyse the predictors of depressive symptoms and clinical depression, respectively. A hierarchical stepwise technique (demographic variables entered in the first block) with subsequent backward elimination was used. Forced entry of highly correlated variables would yield an artificially high R^2 with little significance of independent variables due to multicollinearity [32]. The choice of independent variables was governed by three issues: clinical significance (thus variables like age, sex, disease duration and the demographic variables were all chosen and entered in the first block regardless of the correlational analysis); statistical significance ($P < 0.1$) in the correlational analysis; multicollinearity (thus single variables, having the highest associations, were chosen to represent each of disease activity and disability). The linear regression coefficient (R^2) for a logistic model and the probabilities of depression at different levels of the independent variables were calculated as described by Agresti [33].

RESULTS

RA patients had significantly higher mean scores on the SCL-90-R-D scale than OA patients ($P = 0.001$), even after controlling for age, sex and disease duration (Table I). On the other hand, although more RA patients (23.3%) were diagnosed as having clinical

TABLE I
Comparison of depressive symptoms and depression in RA and OA patients

		RA ($n = 60$)	OA ($n = 40$)	Difference statistic	P	CI
Depressive symptoms						
SCL-90-R-D scores	Range	5–43	2–38			
	Mean ± S.D.	22.6 ± 9.3	16.3 ± 7.8	$t = 3.6$	0.001	2.8, 9.9
T-scores	Mean ± S.D.	52.7 ± 10.0	45.9 ± 8.5	$t = 3.6$	0.001	3.0, 10.7
Adjusted* scores	Mean	22.9	16.0	$F = 12.3$	0.001	1.5, 5.4
Depression						
Prevalence†	Frequency (%)	14 (23.3%)	4 (10%)	$\chi^2 = 2.9$	0.09	
Risk				OR‡ = 2.7		0.8, 9.0

*Adjusted to the covariates age, sex and disease duration by analysis of covariance.
†Determined by a cut-off point of 60 in *T*-scores, confirmed by clinical interview and diagnosis by DSM-III-R criteria.
‡Odds ratio and confidence interval for depression in RA *vs* OA.

depression than OA patients (10%), with the risk of having depression in RA patients 2.7 times that in OA patients, this difference was not statistically significant. All the 18 patients from both groups identified by the cut-off point were found to have some form of clinical depression in the psychiatric interview.

The clinical variables of RA patients and their correlations with depressive symptoms and depression are shown in Table II. The RA sample revealed, on average, a moderately active and severe disease. However, none of the variables tested were significantly correlated with depressive scores. On the other hand, while the associations with clinical depression remained essentially similar to the associations with depressive scores for most variables, the associations of the ESR, pain and HAQ-Disability with clinical depression became statistically significant. This change was most prominent for the HAQ scores ($P < 0.01$).

When age, sex, disease duration, literacy, marital status, residence and income were entered in the first block of the regression analysis with the SCL-90-R-D scores as the dependent variable, only urban residence and being unmarried remained after elimination of non-significant variables, each contributing 7% to the variance of depression scores. In the second block, disease variables were entered, of which only the ESR remained, contributing a further 6% in the variance of depression scores, after controlling for the demographic variables. Results of the final model are shown in Table III, with the three included variables contributing 20% to the variance of depressive symptoms.

In the logistic model with clinical depression (depressed *vs* non-depressed) as the dependent variable (Table IV), only being unmarried and HAQ-Disability were significant predictors of clinical depression among the variables tested. Being unmarried doubled the odds

TABLE II
Clinical variables in RA patients and their correlation with depressive symptoms and depression

		Value	Depressive symptoms r (P)	Depression r (P)
Age (yr)	Mean ± S.D.	39.7 ± 10.9	− 0.03 (0.82)	− 0.03 (0.82)
Age at onset (yr)	Mean ± S.D.	33.4 ± 11.6	0.05 (0.73)	− 0.09 (0.48)
Duration (yr)	Mean ± S.D.	6.3 ± 5.4	−0.03 (0.77)	0.14 (0.29)
Sex	Females	48 (80%)	0.14 (0.29)	− 0.07 (0.55)
Disease activity				
IDA (1–4)	Mean ± S.D.	2.7 ± 0.5	0.18 (0.17)	0.23 (0.07)
Ritchie index (0–78)	Mean ± S.D.	28.3 ± 13.1	0.19 (0.15)	0.19 (0.15)
GS (mmHg)	Mean ± S.D.	60.0 ± 35.6	0.04 (0.75)	− 0.03 (0.85)
MS (min)	Mean ± S.D.	65.3 ± 89.5	− 0.03 (0.82)	− 0.08 (0.54)
ESR (mm/h)	Mean ± S.D.	50.2 ± 29.9	0.25 (0.06)	0.26 (0.05)
NRS-Pain (0–10)	Mean ± S.D.	6.3 ± 2.3	0.09 (0.51)	0.26 (0.05)
Disease severity				
Radiological*	Stage 3 and 4	34 (56.6%)	− 0.02 (0.87)	0.01 (0.97)
Rheumatoid factor	Positive	53 (88.3%)	− 0.02 (0.87)	0.05 (0.73)
EAM	One or more	15 (25%)	0.03 (0.82)	− 0.05 (0.73)
Disability				
Functional grades*	Grade 3 and 4	22 (36.6%)	0.17 (0.20)	0.23 (0.07)
HAQ-DI (0–3)	Mean ± S.D.	1.3 ± 0.8	0.24 (0.07)	0.35 (0.006)

IDA, Index of Disease Activity; GS, grip strength; MS, morning stiffness; ESR, erythrocyte sedimentation rate; NRS, Numerical Rating Scale; EAM, extra-articular manifestations; HAQ-DI, Health Assessment Questionnaire Disability Index.
*Radiological and functional grades are treated as dichotomous variables. Results did not differ when analysing radiological stages and functional grades as ordinal variables with four categories.

TABLE III
Determinants of depressive symptoms in RA patients as analysed by a stepwise multiple regression model, with the SCL-90-R-D scores as the dependent variable

Dependent	Model	Independent	β	t	P
		Included			
Depressive symptoms	$R^2 = 0.20$	Urban/rural	0.29	2.4	0.02
	Adjusted $R^2 = 0.16$	Unmarried/married	0.27	2.2	0.03
	$F = 4.7$	ESR	0.25	2.1	0.04
	$(P = 0.005)$	Intercept	B = 13.8	3.1	0.002
		Excluded			
		Age	−0.01	−0.1	0.97
		Sex	0.08	0.7	0.50
		Disease duration	0.03	0.2	0.83
		Education	−0.04	−0.3	0.78
		Income	−0.08	−0.7	0.51
		Pain	0.03	0.3	0.79
		Disability	0.12	0.9	0.37

TABLE IV
Determinants of clinical depression in RA patients, as analysed by a stepwise logistic regression model with depression/no depression as the dependent variable

Dependent	Model	Independent	B	SE B	P	Odds ratio
		Included				
Depression	Model $\chi^2 = 9.3$	HAQ-Disability	1.3	0.49	0.007	3.8
	$(P = 0.002)$	Unmarried	0.7	0.36	0.04	2.1
	$R^2 = 0.22$	Intercept	−3.0	0.9	0.001	
		Excluded				
		Age			0.97	
		Sex			0.83	
		Disease duration			0.24	
		Urban/rural			0.51	
		Education			0.56	
		Income			0.28	
		Pain			0.43	
		ESR			0.35	

of clinical depression in patients with the same level of disability, while a one-unit increase in HAQ-Disability increased the odds of clinical depression by 3.8 in patients with similar marital status. By regressing the values of clinical depression predicted from the model on the observed values, it was found that marital status and disability together predict 22% of clinical depression ($R^2 = 0.22$). To enhance our understanding of the relationship between these three variables, we calculated from the logistic equation the probabilities of clinical depression at different levels of the two independent variables (Table V).

Finally, we decided to test the effects of the demographic and disease variables included in the regression analysis on the differences in depression scores between RA and OA patients. The analysis of covariance was first repeated with the demographic variables (marital status, urban/rural residence, education, income) included as covariates together with age, sex and disease duration. Their contribution was not significant however, and the difference between RA and OA patients remained significant ($P = 0.001$), as in Table I. Next, pain and disability were each entered separately as covariates with the aforementioned variables in the analysis of covariance. Pain also was not a significant contributor to the difference, but HAQ-Disability was found to have a significant effect ($P = 0.01$) and the differences between RA and OA patients in depression scores were no longer significant ($P = 0.06$).

TABLE V
The probability* of clinical depression at different levels of HAQ-Disability in married and unmarried RA patients

	HAQ			
	0	1	2	3
Married	0.05	0.10	0.40	0.71
Unmarried	0.09	0.27	0.57	0.83

*Calculated from the logistic equation.

DISCUSSION

Both our samples of consecutive RA and OA outpatients may not be representative of either disease as seen in the community. However, recruitment of hospital patients is viewed as an advantage when studying a clinical issue like depression [7]. At the same level of clinical interest, it would seem best to interview patients who come voluntarily to the clinic [34]. In this regard, directly and completely collected clinical

data like ours would offer an advantage over data collected by telephone or mail, and then used for a diagnosis of depression.

Apart from the sample, the method used for estimating depression is critical. Criterion contamination was demonstrated for items of both commonly used criteria [14, 15] and depression scales [10–14]. Deletion of such items or the revision of existing criteria in a particular study is not justified empirically [6]. Some researchers [7] try to get around the problem by using psychological measures recommended specifically for rheumatic patients such as the AIMS-Depression scale [35]. However, it is important to remember that this six-item scale was developed as part of the AIMS to assess the overall impact of RA. Although it reflects to a degree the mood of the patient, its ability to measure a multidimensional construct like depression and to distinguish clinically depressed from non-depressed individuals have not yet been examined in a clinical setting. The developers of the original AIMS have been wise in renaming the revised depression items 'mood' in the AIMS2 [36], which while measuring an important outcome aspect, does not presume a clinical diagnosis.

Our approach to the problem was to 'screen' patients first before subjecting them to a psychiatric interview. We used a scale that was validated in RA patients, being not affected by disease-related items [29]. The 100% agreement between cut-off scores and a clinical diagnosis of depression according to the more conservative DSM-III-R criteria suggests that the scale has high specificity for predicting clinical depression. Our prevalence figure for depression in RA patients (23%) is also similar to the 20% figure believed to be the true prevalence of depression in RA when assessed independently of disease-related items [15].

Our results demonstrate that depressive symptoms are significantly higher in RA patients than in OA patients, with and without control for the effects of age, sex, disease duration and the demographic variables. One disease factor that may explain such a difference is disability, which was the only covariate which made the difference in depression scores between RA and OA patients non-significant.

Although we can conclude with confidence that RA patients have more depressive symptoms than OA patients, we cannot conclude the same with clinical depression. This is probably because of the relatively small OA sample (type II statistical error). The same OR we obtained would be significant if the sample size is doubled. There are few researchers who have addressed this difference between RA and OA patients before. Zaphiropoulos and Burry [3] found that depression is significantly more common in RA patients than in a heterogeneous group of controls with painful locomotor disorders, while Hawley and Wolfe [7], in a longitudinal study, found that depression is not more common in RA than in seven other rheumatic disease groups including OA, concluding that the notion that RA patients are more depressed should be abandoned. However, a limitation in that study is the use of the six-item AIMS depression scale as the only measure of depression. Moreover, the individual patient score represented the mean score of a number of visits over up to 10 yr, and it was recently shown in a 4 yr study of depression in RA that while 15–17% of patients were depressed in at least 1 yr, only 4% were depressed every year [8]. Therefore, the data of Hawley and Wolfe [7] really represent the average mood of patients over a number of years and cannot be looked on as a point or period prevalence of clinical depression in RA and other rheumatic patients.

Other than the differences between RA and OA patients, we explored the variables that may contribute to depressive symptoms and clinical depression in the RA group. In the regression model for depressive symptoms, the ESR was the only disease variable that had a significant but modest contribution (6%). The ESR was found before to be significantly correlated with depressive symptoms [37] as well as a significant, albeit weak, predictor of depression in RA patients [4]. Our data reveal that depressive symptoms are largely unrelated to disease activity in RA patients.

More important than the disease variables in predicting depressive symptoms in our RA sample, however, were sociodemographic variables such as being unmarried and urbanicity. Being unmarried was similarly found by others to be a significant predictor of depression in RA [8]. The inclusion of urbanicity as a predictor variable of depressive symptoms in RA patients has not been attempted before, although rural RA patients were shown to be more satisfied with life and mobile than urban patients [38].

In our study, being unmarried, an urban residence, and a high ESR together predicted 20% of the variance of depressive symptoms in RA. Therefore, a large amount of depressive symptoms remain unexplained by clinical and demographic variables as found before [4, 34]. This disagrees with the study of Newman *et al.* [17], who predicted 44% in the variance of depression by psychosocial and clinical variables using hierarchical regression. We would like to note, however, that we used a stepwise technique, including only significant predictors. An R^2 approaching one (nearly 100% of variance explained) can be achieved by forced entry of a large number of intercorrelated independent variables with few variables achieving significance [32]. In the study of Newman *et al.* [17], 18 independent variables were used hierarchically, many of which would be highly intercorrelated (functional grade and disability; morning stiffness, ESR and articular index). Moreover, depression was measured by the Beck Depression Inventory, which was later shown to include six disease-related items [12], and therefore we believe that the amount of variance explained in that study was spurious.

The relationship between disability and depression was examined before in several studies. A review of these studies concluded that the relationship is bi-directional, complex and possibly not linear [39]. Our study provides some evidence for this in RA

patients. While HAQ-Disability was neither significantly correlated with depressive symptoms nor a significant predictor of them, the clinically depressed RA patients showed significantly higher disability scores (mean = 1.9) than non-depressed patients (mean = 1.2) ($P < 0.01$). Also, higher disability was a significant predictor of clinical depression in the logistic regression ($P < 0.01$). This shows that in comparison with the ESR, for example, disability scores are not predictive of small variations in depression scores, but when patients reach a certain (threshold) level of disability, they have a much higher probability of being clinically depressed. To illustrate the relationship further, we have calculated the probability of clinical depression at different levels of disability in married and unmarried patients (Table V). It can be seen that the difference in the probability of depression is more pronounced when the HAQ score increases from one to two than when it increases from zero to one. Marital status and disability together were able to predict 22% of clinical depression in our sample of RA patients.

We finally conclude that although RA patients definitely have more depressive symptoms than OA patients, our data cannot establish with confidence that the prevalence of clinical depression is higher in RA patients. The notion that RA patients have more depression than OA patients need not be abandoned, however, before being investigated further using valid measures of depression. Further research is also needed to unravel aspects of the complex relationship between disability and depression in RA patients, and the mediators of such a relationship.

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