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Predictors of Depression in a Multiethnic Cohort of Patients with Rheumatoid Arthritis

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Abstract

Objective—Patients with rheumatoid arthritis (RA) who experience depression have worse health outcomes. This study identifies predictors of depression in an ethnically and racially diverse population of patients with RA.

Methods—Patients with RA in a prospective cohort at San Francisco General Hospital out-patient rheumatology clinic were included if they were ≥ 18 years of age, met the American College of Rheumatology classification criteria for RA, had a Health Assessment Questionnaire (HAQ) score collected, and had the RA-specific Disease Activity Scale 28 performed by a rheumatologist. The outcome variable was a depression score measured by the Patient Health Questionnaire-9 (PHQ-9), a self-report questionnaire validated to correlate with a diagnosis of major depression.

Results—Three hundred and forty-nine clinical visits for 172 patients were included in the analysis. Forty percent of patients scored ≥ 10 on the PHQ-9 during at least one clinic visit which corresponds to a symptom severity of at least moderate depression. The mean PHQ-9 score was 7, corresponding to symptom severity of mild depression. In the multivariate analysis, higher HAQ scores were associated with depression, and Asians have lower depression scores compared to Hispanic, white, and black subjects.

Conclusion—Identifying associated predictors of depression in a diverse population of patients with RA can help guide treatment which should include preventing disability and decreased function as well as targeting depressive symptoms more specifically in patients with RA.

Background

Of the 1.3 million adults in the United States (0.6%) with rheumatoid arthritis (RA)[1, 2], most will develop progressive functional limitation and physical disability[3]. Depression, a frequent disorder among patients with RA, is often unrecognized or under-treated[4, 5]. The prevalence of major depressive disorder is 13–42% in patients with RA[6–13], at least double that in the general population.

In patients with RA, poor clinical characteristics and function are associated with subsequent depression. There are more RA related physician visits and hospitalizations in patients with depression as well as negative health and functional outcomes and increased health service utilization[14]. Patients with rheumatologic diseases and depression are less likely to be adherent with their medications[15] and more likely to discontinue anti-TNF-α therapy when compared to non-depressed patients[16], which may then have a negative effect on their health

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status. Furthermore, comorbid depression is an independent risk factor for death in patients with RA, with a hazard ratio of 2.2 (95% CI 1.2–3.9, p = 0.01)[17, 18].

Studies have shown that disability and decreased function as measured by the Health Assessment Questionnaire (HAQ) are associated with depression in Caucasian populations[19–22] but have not addressed potential predictors of depression in an ethnically diverse group of patients with RA. A recent study identified Hispanics with RA as more likely to have symptoms of depression than non-Hispanic subjects[23]; however, important covariates such as disease activity and disease duration were not taken into account. Furthermore, patients of other race/ethnicity (African-Americans, Asians) were not included in this study for comparison. Since concomitant depressive symptoms are known to worsen health outcomes and confer independent risk for mortality in RA, it would be useful to discover associations between depression and rheumatoid arthritis in diverse populations.

Our objective is to identify demographic and disease-related predictors of depressive symptoms in a socioeconomically and ethnically distinct population of patients with RA at an urban, public hospital. The independent effects of race/ethnicity on depressive symptoms were of special interest in this multiethnic cohort of patients with RA.

Subjects and Methods

Study population

Study subjects are patients from the University of California, San Francisco (UCSF) RA Cohort at San Francisco General Hospital (SFGH) outpatient rheumatology clinic. SFGH is the major provider to the poor, minority, and uninsured population of the City and County of San Francisco. Patients were consecutively enrolled as they presented for their initial or return visits between 11/06–12/08, and the data were gathered at regular clinical visits. Patients were included in the analysis if they were ≥ 18 years of age, met the American College of Rheumatology (ACR) classification criteria for RA[24], had a depression score measured by the Patient Health Questionnaire-9 (PHQ-9), had the RA-specific Disease Activity Scale 28 (DAS 28) performed by a rheumatologist at the same clinical visit, and had a Health Assessment Questionnaire (HAQ) score collected within 6 months before or after the depression measure. The UCSF Committee on Human Research approved the study. Written informed consent was obtained and patients were interviewed in the language of their choice (English, Cantonese, Mandarin, or Spanish).

Assessment of clinical data

In addition to sociodemographic characteristics (age, gender, self-reported ethnicity) and disease duration, a wide range of clinical data was collected at enrollment and at regular intervals thereafter during routine clinical encounters. Disease activity was assessed with the DAS 28, calculated at each clinic visit. The DAS 28 includes scores of tender and swollen joint counts, patient global disease activity rating scale, and erythrocyte sedimentation rate (ESR)[25, 26]. Functional limitation was evaluated with the 20-item HAQ score[27]. The HAQ is a reliable and valid assessment of functional limitations in RA. HAQ scores collected within 6 months of the depression scores were included since HAQ scores are generally stable over a one-year period among patients with RA[28–30]. The HAQ has been translated and validated in Mandarin/Cantonese[31] and Spanish[32]. Treatment-related variables were prednisone use and other disease-modifying anti-rheumatic drugs (DMARDs) such as methotrexate, hydroxychloroquine, sulfasalazine, azathioprine, leflunomide, anti-TNFα medications, or other biologic medications.

Ascertainment of depression

The outcome variable was depressive symptoms measured by the PHQ-9. This self-report depression questionnaire yields a 27-point scale that correlates with a diagnosis of major depression[33, 34]. The PHQ-9 is composed of ten items, corresponding to the nine diagnostic criteria of major depressive disorder, plus one item to assess functional impairment secondary to depression. A score of 5–9 is consistent with mild depressive symptoms, 10–14 moderate, 15–19 moderate-severe, and ≥ 20 is severe depressive symptoms. The PHQ-9 has established reliability and validity in Caucasian, African-American, Asian, and Hispanic populations[35, 36]. To classify patients with at least moderate symptoms of depression, we used a clinically useful cut-point of 10, which distinguishes between no/mild and moderate/severe depressive symptoms for the univariate analysis. The term “depressed” will be used from this point forward to refer to participants meeting this threshold of symptom severity.

Data Collection

Study research staff who were not the patient’s physician administered standardized questionnaires (PHQ-9, HAQ) and interviews in English, Spanish, or Chinese. Research staff and treating physicians were blind to PHQ-9 scores and HAQ scores, as scores were tabulated independently

Statistical analysis

The analysis accommodated the repeated measures from an individual’s multiple clinical encounters and was conducted using STATA, version 10.0 (Statacorp, College Station, Texas). The unit of analysis is a subject’s clinical visit in which the patient had a PHQ-9 collected. A total of 46 visits (11%), involving 22 patients were excluded because of incomplete information. Most of these visits were dropped because a component of the DAS 28 was not collected and therefore could not be calculated.

Differences in baseline characteristics between depressed and non-depressed subjects were assessed using a two-sided *t*-test with equal variance or the Pearson’s chi-square test, as appropriate. For the multivariate analysis, we used generalized estimating equations (GEE) with robust standard errors to assess associations with depression scores while taking into account within-subject correlations between measurements over time and to account for the influence of potential confounding covariates. The PHQ-9 score was used as a continuous outcome for the multivariate analysis. Interpretation of the coefficient using repeated measures analysis with robust standard errors is the same as for linear regression[37]. For the multivariate analysis, independent variables were determined *a priori* with a forward inclusion strategy. Variables were selected based on face validity and biologic plausibility to obtain a minimally confounded estimate of their effect and identify important independent predictors of depression in patients with RA. Given the modest-sized data set and the number of predictors, bootstrapping (re-sampling of subjects not observations) was employed to check the reliability of the standard errors and confidence intervals.

Results

Three hundred and forty-nine clinical visits for 172 patients were included in the analysis. The average number of outpatient rheumatology clinic visits per patient was 2 (range 1–5). In this study, 89% percent of included patients were female with a mean age of 52 years (Table 1). In regard to race/ethnicity, 54% of the patients were Latino/Hispanic, 34% were Asian/Pacific Islander, 7% were African-American, and 4% were Caucasian. Eighty-four percent of patients were rheumatoid factor (RF) positive and 81% were anti-cyclic citrullinated peptide (CCP) positive. Eighty-three percent of patients were on a DMARD, 24% were on anti-TNFα medications, and 62% of patients were taking prednisone with a

mean dose of 4mg. Fifteen percent of patients were prescribed an anti-depressant medication. The mean HAQ score was 1.25, and the mean DAS 28 was 4.1 indicative of farily high levels of functional impairment and disease activity, respectively.

With regard to the prevalence of depressive symptoms in our cohort, 40% of patients scored ≥ 10 on the PHQ-9 during at least one clinic visit (n=138) which corresponds to a symptom severity of at least moderate depression. The mean PHQ-9 score was 7, corresponding to symptom severity of mild depression.

In univariate analyses of baseline data, there were no statistically significant differences between depressed and non-depressed patients with regards to age, ethnicity, disease duration, antibody status or prednisone treatment and dosage. However, depressed participants had higher HAQ and DAS 28 scores, and were less likely to be treated with a DMARD (Table 1).

After adjusting for age, gender, race/ethnicity, disease duration and activity, prednisone treatment, and RA disease-modifying medications in a GEE model (Table 2), higher HAQ scores were significantly associated with higher PHQ-9 scores. A clinically meaningful change in HAQ score is .24[38] so the GEE coefficient, which represents associations with a 1-point difference in HAQ, was divided by 4. With each one .24 unit increase in the HAQ score there was an increase in PHQ9 score of .98 (95% CI .73, 1.3). Asian ethnicity was associated with lower depression scores, specifically a decrease in PHQ9 score of 1.6 (95% CI −3.0, −.28), as compared to the reference group, Hispanic/Latinos (Table 2). The confidence intervals from this multivariate model remained similar when bootstrapping was employed. The effects of HAQ score and race/ethnicity on PHQ-9 scores did not differ when modeled for possible interactions.

Discussion

This is the first study evaluating predictors of depressive symptoms in an underserved, ethnically diverse cohort of patients with rigorously defined rheumatoid arthritis that includes measures of functional disability and disease status as measured by the DAS 28. Several conclusions stem from our data.

First, the prevalence of clinically significant depressive symptoms in an urban, multiethnic sample of patients with RA is 40%. While within the range of previously reported results (13–42%), this cohort appears to be on the higher end of the spectrum compared to Caucasian populations with RA and depression.

Second, those with more disability are at greater risk for depression. These findings in a previously unstudied population corroborate with results in the literature. Given the high prevalence of depression in our subjects and the strong association with decreased function, this raises the question if depression is a product of increased disability as measured by the HAQ. A mean HAQ score of 1.25 is well above the previously reported mean HAQ of .71 in a population-based cohort of patients with RA[28]. Why is there a higher mean HAQ score in our study sample? The most obvious answer is that health disparities in the RA cohort at SFGH associated with race/ethnicity and socioeconomic status lead to worse outcomes[39], in this case a higher mean HAQ score. Identifying pathways and mechanisms by which socioeconomic status and race/ethnicity affect HAQ scores would provide more options for intervention to eliminate disability and subsequent depression in patients with RA.

Third, it is surprising that disease activity as measured by swollen, tender joints, patient global disease activity scale, and the ESR is not associated with higher depression scores given previous research in this area[40, 41]. In the multivariate analysis, the upper limit for the

GEE coefficient of the DAS 28 is .45. The standard deviation for the DAS 28 in the cohort is 1.4 and a two-point change in the DAS 28 would be quite large clinically. Therefore, the maximum change in the depression score associated with a two-point increase in DAS 28 is only .9, which is quite a small clinical change. This suggests that it is not the acute pain or clinical manifestations of RA disease activity but more the long-term disability and joint damage associated with arthritis that leads to depression in patients with RA.

Lastly, Asians in the SFGH RA cohort had lower depression scores compared to Latino/Hispanic, White/Caucasian, and Black/African-American subjects. To our knowledge, this is the first study to find an association between depressive symptoms and Asian race/ethnicity in patients with RA. This finding is supported by previous literature other than that specific to RA which substantiates that there is a low level of reporting depressive symptoms in Asians[42] owing to the stigma of mental illness, implications of "weakness of character," and factors that may protect against depression such as stoicism and cultural support systems[43].

There are limitations to this study. Like other chronic conditions, assessing depressive symptoms in patients with RA can be difficult. It is well understood that somatic symptoms of depression (e.g., fatigue or decreased energy) overlap with symptoms of RA. Consequently, there is a risk that depression in RA may be overestimated[44]. Conversely, there is also some evidence to suggest that using conventional assessments of depression does not overestimate depression in similar chronic conditions[45]. In our approach to the assessment of depressive symptoms we chose the PHQ-9 as a measure for depression since it was developed for primary care, is in widespread use for a range of acute and chronic medical conditions, and was developed to closely parallel the diagnostic symptoms of Major Depressive Disorder[33, 34, 46].

Our study design was cross-sectional and therefore causality between greater HAQ scores and the associated, increased depressive symptoms cannot be established. Future studies should examine this relationship in a prospective manner. Also, as a cross-sectional study, there is a limit to the information that can be produced about the waxing and waning nature of depressive symptoms over time. The high prevalence of depressive symptoms in this cohort is either because they occur more frequently or because the depressive symptoms last longer.

Another potential limitation to this study is that 'detection bias' or 'medical surveillance bias' could influence the association between depressive symptoms and clinical predictors if depressed patients with RA were less likely to attend clinical appointments. To investigate this possibility, we compared the mean number of clinical visits of patients with and without depressive symptoms and found no significant difference.

Finally, the question of how applicable our results are to other RA patients must be asked. The high percentage of subjects with positive anti-CCP (81%), which portends a poor prognosis, is greater than reported in other clinical cohorts. Furthermore, our study population has relatively high disease activity with a mean DAS 28 of 4.1, and a mean HAQ score of 1.25. The fact that our study population has more erosive and disabling disease is likely a reflection of referral practices to a public, urban hospital clinic as well as our precise definition of RA for entry into the study. While our cohort was skewed toward more severe disease and included an ethnically diverse patient population, we believe that the heterogeneity of our cohort is a benefit and not a limitation.

Given that patients in this cohort are comprised of minorities who struggle with poverty and often have poor education, future studies should directly compare this population to those with higher socioeconomic status to determine associations between depression, disease activity, disability and health disparities in patients with RA. Since depression is known to

confer risk of poor health outcomes, identifying associated predictors of depression in all patients with RA, not just Caucasians, is useful. The above findings can help guide treatment to include preventing disability and decreased function as well as targeting depressive symptoms more specifically in a vulnerable population of patients with rheumatoid arthritis.

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References

1. Helmick CG, Felson DT, Lawrence RC, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part I. Arthritis Rheum. Jan; 2008 58(1):15–25. [PubMed: 18163481]
2. Lawrence RC, Helmick CG, Arnett FC, et al. Estimates of the prevalence of arthritis and selected musculoskeletal disorders in the United States. Arthritis Rheum. May; 1998 41(5):778–799. [PubMed: 9588729]
3. Yelin E, Meenan R, Nevitt M, Epstein W. Work disability in rheumatoid arthritis: effects of disease, social, and work factors. Ann Intern Med. Oct; 1980 93(4):551–556. [PubMed: 7436187]
4. Newman S, Mulligan K. The psychology of rheumatic diseases. Baillieres Best Pract Res Clin Rheumatol. Dec; 2000 14(4):773–786. [PubMed: 11092801]
5. Nagyova I, Stewart RE, Macejova Z, van Dijk JP, van den Heuvel WJ. The impact of pain on psychological well-being in rheumatoid arthritis: the mediating effects of self-esteem and adjustment to disease. Patient Educ Couns. Jul; 2005 58(1):55–62. [PubMed: 15950837]
6. Nakajima A, Kamitsuji S, Saito A, et al. Disability and patient's appraisal of general health contribute to depressed mood in rheumatoid arthritis in a large clinical study in Japan. Mod Rheumatol. 2006; 16(3):151–157. [PubMed: 16767553]
7. Frank RG, Beck NC, Parker JC, et al. Depression in rheumatoid arthritis. J Rheumatol. Jun; 1988 15(6):920–925. [PubMed: 3418641]
8. Abdel-Nasser AM, Abd El-Azim S, Taal E, El-Badawy SA, Rasker JJ, Valkenburg HA. Depression and depressive symptoms in rheumatoid arthritis patients: an analysis of their occurrence and determinants. Br J Rheumatol. Apr; 1998 37(4):391–397. [PubMed: 9619889]
9. Wright GE, Parker JC, Smarr KL, Johnson JC, Hewett JE, Walker SE. Age, depressive symptoms, and rheumatoid arthritis. Arthritis Rheum. Feb; 1998 41(2):298–305. [PubMed: 9485088]
10. Pincus T, Griffith J, Pearce S, Isenberg D. Prevalence of self-reported depression in patients with rheumatoid arthritis. Br J Rheumatol. Sep; 1996 35(9):879–883. [PubMed: 8810672]
11. Dickens C, McGowan L, Clark-Carter D, Creed F. Depression in rheumatoid arthritis: a systematic review of the literature with meta-analysis. Psychosom Med. Jan–Feb; 2002 64(1):52–60. [PubMed: 11818586]
12. Smedstad LM, Moum T, Vaglum P, Kvien TK. The impact of early rheumatoid arthritis on psychological distress. A comparison between 238 patients with RA and 116 matched controls. Scand J Rheumatol. 1996; 25(6):377–382. [PubMed: 8996472]
13. Lowe B, Willand L, Eich W, et al. Psychiatric comorbidity and work disability in patients with inflammatory rheumatic diseases. Psychosom Med. May–Jun; 2004 66(3):395–402. [PubMed: 15184703]
14. Katz PP, Yelin EH. Prevalence and correlates of depressive symptoms among persons with rheumatoid arthritis. J Rheumatol. May; 1993 20(5):790–796. [PubMed: 8336304]

15. Julian LJ, Yelin E, Yazdany J, et al. Depression, medication adherence, and service utilization in systemic lupus erythematosus. Arthritis Rheum. Jan 29; 2009 61(2):240–246. [PubMed: 19177526]
16. McNamara D. Depression Interferes with Anti-TNF Therapy. Rheumatology News. June.2007 6(6)
17. Ang DC, Choi H, Kroenke K, Wolfe F. Comorbid depression is an independent risk factor for mortality in patients with rheumatoid arthritis. J Rheumatol. Jun; 2005 32(6):1013–1019. [PubMed: 15940760]
18. Pincus T. Is mortality increased in patients with rheumatoid arthritis? J Musculoskeletal Med. 1988; 5:27–46.
19. Katz PP, Yelin EH. The development of depressive symptoms among women with rheumatoid arthritis. The role of function. Arthritis Rheum. Jan; 1995 38(1):49–56. [PubMed: 7818571]
20. Katz PP, Yelin EH. Activity loss and the onset of depressive symptoms: do some activities matter more than others? Arthritis Rheum. May; 2001 44(5):1194–1202. [PubMed: 11352254]
21. Covic T, Adamson B, Spencer D, Howe G. A biopsychosocial model of pain and depression in rheumatoid arthritis: a 12-month longitudinal study. Rheumatology (Oxford). Nov; 2003 42(11): 1287–1294. [PubMed: 12810932]
22. Smedstad LM, Vaglum P, Moum T, Kvien TK. The relationship between psychological distress and traditional clinical variables: a 2 year prospective study of 216 patients with early rheumatoid arthritis. Br J Rheumatol. Dec; 1997 36(12):1304–1311. [PubMed: 9448592]
23. Escalante A, del Rincon I, Mulrow CD. Symptoms of depression and psychological distress among Hispanics with rheumatoid arthritis. Arthritis Care Res. Jun; 2000 13(3):156–167. [PubMed: 14635289]
24. Arnett FC, Edworthy SM, Bloch DA, et al. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. Arthritis Rheum. Mar; 1988 31(3):315–324. [PubMed: 3358796]
25. Prevoo ML, van 't Hof MA, Kuper HH, van Leeuwen MA, van de Putte LB, van Riel PL. Modified disease activity scores that include twenty-eight-joint counts. Development and validation in a prospective longitudinal study of patients with rheumatoid arthritis. Arthritis Rheum. Jan; 1995 38(1):44–48. [PubMed: 7818570]
26. Balsa A, Carmona L, Gonzalez-Alvaro I, Belmonte MA, Tena X, Sanmarti R. Value of Disease Activity Score 28 (DAS28) and DAS28-3 compared to American College of Rheumatology-defined remission in rheumatoid arthritis. J Rheumatol. Jan; 2004 31(1):40–46. [PubMed: 14705217]
27. Fries JF, Spitz PW, Young DY. The dimensions of health outcomes: the health assessment questionnaire, disability and pain scales. J Rheumatol. Sep–Oct; 1982 9(5):789–793. [PubMed: 7175852]
28. Sokka T, Kautiainen H, Hannonen P, Pincus T. Changes in Health Assessment Questionnaire disability scores over five years in patients with rheumatoid arthritis compared with the general population. Arthritis Rheum. Oct; 2006 54(10):3113–3118. [PubMed: 17009231]
29. Ward MM, Leigh JP, Fries JF. Progression of functional disability in patients with rheumatoid arthritis. Associations with rheumatology subspecialty care. Arch Intern Med. Oct 11; 1993 153(19):2229–2237. [PubMed: 8215726]
30. Wolfe F, Choi HK. Benchmarking and the percentile assessment of RA: adding a new dimension to rheumatic disease measurement. Ann Rheum Dis. Nov; 2001 60(11):994–995. [PubMed: 11602461]
31. Koh ET, Seow A, Pong LY, et al. Cross cultural adaptation and validation of the Chinese Health Assessment Questionnaire for use in rheumatoid arthritis. J Rheumatol. Sep; 1998 25(9):1705–1708. [PubMed: 9733449]
32. Esteve-Vives J, Batlle-Gualda E, Reig A. Spanish version of the Health Assessment Questionnaire: reliability, validity and transcultural equivalency. Grupo para la Adaptacion del HAQ a la Poblacion Espanola. J Rheumatol. Dec; 1993 20(12):2116–2122. [PubMed: 8014941]
33. Spitzer RL, Williams JB, Kroenke K, et al. Utility of a new procedure for diagnosing mental disorders in primary care. The PRIME-MD 1000 study. Jama. Dec 14; 1994 272(22):1749–1756. [PubMed: 7966923]

34. Kroenke K, Spitzer RL, Williams JB. The PHQ-9: validity of a brief depression severity measure. J Gen Intern Med. Sep; 2001 16(9):606–613. [PubMed: 11556941]
35. Huang FY, Chung H, Kroenke K, Delucchi KL, Spitzer RL. Using the Patient Health Questionnaire-9 to measure depression among racially and ethnically diverse primary care patients. J Gen Intern Med. Jun; 2006 21(6):547–552. [PubMed: 16808734]
36. Wulsin L, Somoza E, Heck J. The Feasibility of Using the Spanish PHQ-9 to Screen for Depression in Primary Care in Honduras. Prim Care Companion J Clin Psychiatry. Oct; 2002 4(5): 191–195. [PubMed: 15014707]
37. Vittinghoff, E. Regression methods in biostatistics : linear, logistic, survival, and repeated measures models. New York: Springer; 2005.
38. Drossaers-Bakker KW, de Buck M, van Zeben D, Zwinderman AH, Breedveld FC, Hazes JM. Long-term course and outcome of functional capacity in rheumatoid arthritis: the effect of disease activity and radiologic damage over time. Arthritis Rheum. Sep; 1999 42(9):1854–1860. [PubMed: 10513799]
39. Adler NE, Rehkopf DH. U.S. disparities in health: descriptions, causes, and mechanisms. Annu Rev Public Health. 2008; 29:235–252. [PubMed: 18031225]
40. Odegard S, Finset A, Mowinckel P, Kvien TK, Uhlig T. Pain and psychological health status over a 10-year period in patients with recent onset rheumatoid arthritis. Ann Rheum Dis. Sep; 2007 66(9):1195–1201. [PubMed: 17392351]
41. Tander B, Cengiz K, Alayli G, Ilhanli I, Canbaz S, Canturk F. A comparative evaluation of health related quality of life and depression in patients with fibromyalgia syndrome and rheumatoid arthritis. Rheumatol Int. Jul; 2008 28(9):859–865. [PubMed: 18317770]
42. Kleinman A, Eisenberg L, Good B. Culture, illness, and care: clinical lessons from anthropologic and cross-cultural research. Ann Intern Med. Feb; 1978 88(2):251–258. [PubMed: 626456]
43. Parker G, Gladstone G, Chee KT. Depression in the planet's largest ethnic group: the Chinese. Am J Psychiatry. Jun; 2001 158(6):857–864. [PubMed: 11384889]
44. Estlander AM, Takala EP, Verkasalo M. Assessment of depression in chronic musculoskeletal pain patients. Clin J Pain. Sep; 1995 11(3):194–200. [PubMed: 8535038]
45. Ellis GK, Robinson JA, Crawford GB. When symptoms of disease overlap with symptoms of depression. Aust Fam Physician. Aug; 2006 35(8):647–649. [PubMed: 16894444]
46. Spitzer RL, Kroenke K, Williams JB. Validation and utility of a self-report version of PRIME-MD: the PHQ primary care study. Primary Care Evaluation of Mental Disorders. Patient Health Questionnaire. Jama. Nov 10; 1999 282(18):1737–1744. [PubMed: 10568646]

Table 1

Baseline Characteristics of SFGH RA Cohort by Depression

Characteristic	All Subjects n = 172	Subjects with PHQ-9 < 10 at any visit n = 103	Subjects with PHQ-9 ≥10 at any visit n = 69	*p*-Value
Gender – n (%)				.68
Female	153 (89)	92 (89)	61 (88)	
Male	19 (11)	11 (11)	8 (12)	
Age – mean ± SD	52 ± 13	51	53	.26
Ethnicity – n (%)				.17
Latino/Hispanic	93 (54)	55 (53)	37 (54)	
Black/African American	12 (7)	8 (8)	4 (6)	
Asian/Pacific Islander	58 (34)	34 (33)	25 (36)	
White/Caucasian	7 (4)	6 (6)	1 (1)	
American Indian/Other	2 (1)	0 (0)	2 (3)	
DAS 28*	4.1 ± 1.4	4.0	4.3	.04
Disease Duration (year)	7.5 ± 8.0	6.9	8.4	.08
HAQ¶ score	1.25 ± .81	1.1	1.4	< .0001
Antibody Positive				
RF	144 (84)	87 (84)	58 (84)	.92
Anti-CCP	139 (81)	83 (81)	57 (83)	.66
DMARD† – no (%)	143 (83)	90 (87)	54 (78)	.03
Methotrexate – no (%)	104 (61)	66 (64)	39 (56)	.11
Prednisone – no (%)	107 (62)	65 (63)	41 (60)	.65
Prednisone Dose (mg)	4 ± 4.8	4.1	4.3	.76
Anti-TNF – no (%)	41 (24)	25 (24)	17 (25)	.84

*Disease Activity Scale 28 (DAS 28) includes tender and swollen joint counts, patient global rating scale, and erythrocyte sedimentation rate

¶Health Assessment Questionnaire

†Includes any disease-modifying anti-rheumatic drug other than prednisone (i.e. methotrexate, plaquenil, sulfasalazine, azathioprine, leflunomide, anti-TNFα medications, and rituximab)

Table 2

Multivariate Model of Predictors of Depression

Characteristic	GEE Coefficient (95% CI)	P-value
Female Gender	−.41 (−2.6, 1.7)	.70
Age - years	−.31 (−.09, .02)	.27
Ethnicity		
Latino/Hispanic	Reference	
Black/African American	−.20 (−2.7, 2.4)	.88
Asian/Pacific Islander	−1.6 (−3.0, −.28)	.02
White/Caucasian	.37 (−2.1, 2.8)	.77
American Indian/Other	−1.8 (−6.1, 2.6)	.43
DAS 28*	.08 (−.31, .45)	.70
Disease Duration (year)	−.02 (−.10, .05)	.50
HAQscore¶	.98 (.73, 1.3)	< .0001
DMARD†	−.76 (−2.6, 1.1)	.43
Prednisone	−.42 (−1.7, .83)	.51

*Disease Activity Scale 28 (DAS 28) includes tender and swollen joint counts, patient global rating scale, and erythrocyte sedimentation rate

¶Health Assessment Questionnaire score

†Includes any disease-modifying anti-rheumatic drug other than prednisone (i.e. methotrexate, plaquenil, sulfasalazine, azathioprine, leflunomide, anti-TNFα medications, and rituximab)