Rheumatology

Venlafaxine Treatment of Fibromyalgia

Kemal Sayar, Gokhan Aksu, Ismail Ak, and Mehmet Tosun

BACKGROUND: Although the pathophysiology of fibromyalgia is unknown, central monoaminergic transmission may play a role. Antidepressants have proved to be successful in alleviating symptoms of fibromyalgia. Medications that act on multiple neurotransmitters may be more effective in symptom management.

OBJECTIVE: To assess the efficacy of venlafaxine, a potent inhibitor of both norepinephrine and serotonin reuptake, in the treatment of patients with fibromyalgia.

METHODS: Fifteen patients with fibromyalgia were assessed prior to and after treatment with fixed-dose venlafaxine 75 mg/d. Before initiation of pharmacotherapy, patients were interviewed with the Structured Clinical Interview for Axis I disorders in the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition. The study lasted for 12 weeks, and patients were evaluated in weeks 6 and 12. The primary outcome measures were the Fibromyalgia Impact Questionnaire (FIQ) total score and pain score. The anxiety and depression levels of the patients were measured with the Beck Depression, the Beck Anxiety, the Hamilton Anxiety, and the Hamilton Depression scales.

RESULTS: There was a significant improvement in the mean intensity of pain ($F = 14.3$; $p = 0.0001$) and in the disability caused by fibromyalgia ($F = 42.7$; $p = 0.0001$) from baseline to week 12 of treatment. The depression and anxiety scores also decreased significantly from baseline to week 12. The improvement in the FIQ scores did not correlate with the decrease of scores in both patient- and physician-rated depression and anxiety inventories. Change in pain scores also was not correlated with the change in depression and anxiety scores.

CONCLUSIONS: Venlafaxine was quite promising in alleviating the pain and disability associated with fibromyalgia. This effect seems to be independent of its anxiolytic and antidepressant properties. Blockade of both norepinephrine and serotonin reuptake might be more effective than blockade of either neurotransmitter alone in the treatment of fibromyalgia.

KEY WORDS: antidepressants, fibromyalgia, venlafaxine.

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Fibromyalgia is a syndrome of chronic musculoskeletal pain with typical symptoms of pain and stiffness, tenderness over specific trigger points, fatigue, and sleep disturbances.[1] The prevalence of fibromyalgia is estimated to be 2% in the US, and it was found to be 5% in a Turkish community study.[2,3] It is more common in females, and the incidence increases with age.[4] The role of psychological factors in the pathogenesis of fibromyalgia is a much debated issue. Whether excessive levels of depression and anxiety in patients with fibromyalgia is the cause or the result of the disorder is a matter of interpretation. It has also been reported that the prevalence of depression is no higher in patients with fibromyalgia than in patients with rheumatoid arthritis or healthy controls.[5]

Although the pathophysiology of fibromyalgia is unknown, central monoaminergic transmission may play a role in its etiology. Two recent meta-analyses of trials of antidepressants have shown tricyclic antidepressants to be moderately effective in the treatment of patients with fibromyalgia.[6,7] A systematic review showed that antidepressants improved sleep, fatigue, pain, and well-being, but not trigger points. Only 1 study revealed a correlation between symptom improvement and depression scores.[7] The effects

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This study was independently conducted. Wyeth Pharmaceuticals, Turkey, covered the cost of the statistical procedures used in this research.

of serotonin-reuptake inhibitors in fibromyalgia also showed discrepant findings. Placebo-controlled studies involving citalopram and fluoxetine revealed conflicting results.[8-12] The only study assessing the efficacy of venlafaxine, a potent inhibitor of both norepinephrine and serotonin reuptake, was open-label and disclosed a positive response in the majority of fibromyalgia patients. In this study, the presence of lifetime psychiatric disorders, particularly depressive and anxiety disorders, predicted a positive response to venlafaxine.[13] Our trial was designed to assess the efficacy of the norepinephrine and serotonin-reuptake inhibitor venlafaxine in fibromyalgia patients and to seek any possible correlation of its efficacy with psychological measures.

Methods

SUBJECTS

The study was conducted between December 2001 and April 2002 at the outpatient clinics of Karadeniz Technical University (KTU) Medical School, a university hospital based in the city of Trabzon in northeastern Turkey. All consecutive patients diagnosed as having fibromyalgia according to American College of Rheumatology (ACR) criteria in the outpatient clinics of physical therapy and rehabilitation department were interviewed.[1] Patients with current suicidal thoughts, severe heart disease (congestive heart failure or coronary heart disease), or a debilitating neurologic condition were excluded from the study, as well as patients using psychotropic agents (antidepressants, anxiolytics, antipsychotics) and analgesics (including nonsteroidal antiinflammatory drugs and acetaminophen) within the last month. Eighteen potential subjects were excluded from the study and 20 were found to be eligible. All patients provided informed consent and none refused to participate. There was no sign of hepatic or renal impairment in our patients. All the eligible patients were women. Through the course of therapy, 5 patients left the study and 15 patients completed it. The procedures followed were in accordance with the ethical standards of KTU Medical School and the Helsinki declaration of 1975, as revised in 1983.

PROCEDURE

All patients were diagnosed as having fibromyalgia according to the operational criteria proposed by the ACR.[1] At the initial assessment, the study group was evaluated by using the Structured Clinical Interview for Axis I Disorders (SCID) in the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition.[14,15] The subjects were given the following scales in addition to the sociodemographic data form: the Beck Depression[16] and Anxiety[17] Inventories, the Hamilton Depression[18] and Anxiety[19] Scales, the Fibromyalgia Impact Questionnaire (FIQ),[20] and the Short Form-36. The intensity of the pain was recorded with a visual analog scale (VAS) of 100 mm length by patients.[21] All patients were able to complete the questionnaires independently. With FIQ and VAS pain being the primary outcome measures, 4 other scales were also given to patients to investigate the much-debated association between psychological distress and symptom severity. Self-report scales (e.g., Beck Depression and Anxiety) were administered to patients, as well as the Hamilton rating scales for depression and anxiety, which were rated by physicians. The subjects were given a fixed dose of venlafaxine 75 mg/d after the initial assessment, and therapeutic efficacy was assessed at weeks 6 and 12 of treatment. No change was made in the dose of the medication from the beginning to the end of the study. The patients were not initiated on any new treatments for fibromyalgia, and they provided informed consent.

STATISTICS

Repeated-measures ANOVA was used to compare the measures before and after treatment. The Bonferroni method was used to detect which groups showed a significant difference. The impact of current or past psychiatric disorder on the measures was assessed with Mann–Whitney U-test. Pearson correlation analysis was performed to identify a possible association with the change in depression and anxiety scores and the change in pain and disability scores. Whether these relations were statistically significant was investigated with 2 different correlation analyses for every possible pair of variables; $p < 0.05$ was regarded as significant.

MEASURES

The 100-mm VAS was used with anchors of "no pain" and of "pain as bad as it could be." Most studies that compare VAS with numerical and verbal ratings conclude that the VAS or numerical ratings are statistically preferable to the verbal rating scales.[21]

The FIQ is a self-report instrument composed of 19 items.[20] The first 10 items comprise a physical functioning scale; each item is rated on a 4-point Likert-type scale. On items 11 and 12, subjects indicated the number of days that they felt well or missed work because of fibromyalgia symptoms. Items 13 through 19 are 10-cm visual analog scales along which subjects rated the difficulty in performing their job responsibilities, pain, fatigue, morning tiredness, stiffness, anxiety, and depression. All subscores, with the exception of the 2 work-related scores, were summed to yield the total score of fibromyalgia impact, which ranges from 0 (no impact) to 80 (maximum impact). FIQ is widely used in patients with fibromyalgia to evaluate both the clinical severity of the disease and the efficacy of different interventions. It has been found to be valid and reliable in Turkish fibromyalgia patients.[22] The Beck Depression Inventory is a 21-item self-report questionnaire that assesses severity of depression.[16] Individuals are asked to rate themselves on a 0–3 spectrum (0 = least, 3 = most) with a score range of 0 to 63. The total score is a sum of all items. It was shown to be valid and reliable in Turkish.[23]

The Beck Anxiety Inventory is a 21-item self-report questionnaire.[17] Each item is rated on a 4-point Likert scale ranging from 0 (not at all) to 3 (severely, I could barely stand it). The total score ranges from 0 to 63. It was shown to be valid and reliable in Turkish.[24]

The levels of depression and anxiety were also assessed by the psychiatrists using the Hamilton Anxiety[19] and Depression[18] scales. These scales have been demonstrated to be valid and reliable in Turkish population studies.[25,26]

Results

Within the first week of treatment, 5 patients dropped out due to adverse effects of the medication. The most commonly reported adverse effects were nausea, irritability, insomnia, anorexia, and headache. One patient reported a worsening of the pain after initiation of treatment. Half of all patients suffered from 1 or more adverse effects. Fifteen patients completed the study. These patients had a mean ± SD duration of disease of 3.7 ± 2.8 years and a mean age of 37.7 ± 7.8 years. Fourteeen subjects were married. Five had graduated from primary school, 4 from secondary school, 4 from high school, and 2 were university graduates. Two of the patients in the study group reported a past history of antidepressant treatment. Of these 15 women, 9 did not have any current psychiatric disorder, but 5 were diagnosed as having generalized anxiety disorder and 1 as having bipolar disorder. Of the 9 patients without a current psychiatric disorder, 3 were found to have a past history of major depressive disorder according to the SCID. The intensity of pain did not decrease significantly from baseline to the sixth week of treatment, but showed a significant improvement in the mean intensity of pain ($F = 14.3$; $p = 0.0001$) and the disability caused by fibromyalgia ($F = 42.7$; $p = 0.0001$) from baseline to week 12 of treatment.

The depression and anxiety scores also decreased significantly from baseline to week 12 (Table 1).

There was no difference in the perceived pain scores of patients with or without a current or past psychiatric disorder. The Beck Anxiety Inventory scores decreased significantly in the sixth week of treatment from baseline and decreased further at the 12-week treatment. Anxiety levels as reported by the Beck Anxiety Inventory dropped steadily with treatment ($F = 17.6$; $p = 0.0001$). The Beck Depression Inventory scores did not show a significant decrease in week 6, but decreased significantly from baseline by week 12. There was a significant decrease of Beck Depression Inventory scores from week 6 to 12 ($F = 8.6$; $p = 0.002$). The Hamilton Anxiety Scale scores also showed a steady and significant decrease during treatment. The differences between baseline measure and in treatment measures were significant ($F = 23.5$; $p = 0.0001$). The Hamilton Rating Scale for Depression scores also showed a steadily decreasing pattern in which differences were significant ($F = 21.6$; $p = 0.0001$). The FIQ scores decreased significantly during both study periods. There was a steady and significant decrease in the FIQ scores with treatment ($F = 42.7$; $p = 0.0001$). Neither the improvement in the anxiety or depression measures nor the improvement on fibromyalgia-related disability were influenced by the presence of a current or past psychiatric disorder. Patients who had current or past psychiatric disorders did not differ in their improvement rate compared with those without that history. The improvement in the FIQ scores did not correlate with the decrease of scores in both patient- and physician-rated depression and anxiety inventories. Change in pain scores also was not correlated with the change in depression and anxiety scores. The r values are presented in Tables 2 and 3.

Discussion

We found venlafaxine to significantly improve the FIQ total score; moreover, patients receiving venlafaxine had a significantly decreased severity of pain. We wanted to determine whether this improvement was dependent on the psychological relief afforded by this particular medication. This effect was independent of the changes in anxiety and depression scores. A randomized, double-blind, crossover study of 19 patients compared the independent and combined efficacy of fluoxetine 20 mg/d and amitriptyline 25 mg/d with placebo in the treatment of fibromyalgia.[11] Compared with the placebo group, the fluoxetine group showed significant improvements on scores in the FIQ and on VASs for pain, global well-being, and sleep disturbances. The amitriptyline group showed similar improvements. When fluoxetine and amitriptyline were used together, there was even greater improvement in these variables. Blockade of both norepinephrine and serotonin reuptake seemed to be more effective than blockade of either neurotransmitter alone. This study gave us the inspiration that a dual-action antidepressant with both serotonergic and noradrenergic properties might be effective in the treatment of fibromyalgia.

In a similar vein, Dwight et al.[13] found venlafaxine to alleviate the symptoms of fibromyalgia in some patients. An open 8-week trial of venlafaxine in 11 patients resulted in the reduction of fibromyalgia symptoms in 6 patients. The presence of chronic psychiatric disorders, particularly depressive and anxiety disorders, predicted a positive response to venlafaxine. There was no correlation between current Axis I disorders and treatment response. To our knowledge, our study is the second that has investigated the efficacy of the serotonin and norepinephrine reuptake inhibitor ven-

Table 1. Scale Scores Before and After Treatment[a]

	Treatment Time			p Value	
Scale	Baseline	6 wk	12 wk	Baseline–6 wk	6–12 wk
Pain	58.8 ± 10.2	51.2 ± 14.6	37.4 ± 12.5	0.117	0.034
HAS	21.6 ± 12.9	12.8 ± 6.4	8.1 ± 4.3	0.006	0.000
HRSD	11.2 ± 5.4	6.7 ± 3.7	5.9 ± 3.0	0.001	0.186
BAI	27.3 ± 12.5	18.8 ± 9.0	15.0 ± 8.2	0.002	1.000
BDI	17.0 ± 4.9	18.4 ± 5.2	10.5 ± 4.6	1.000	0.023
FIQ	55.2 ± 8.3	46.6 ± 8.8	39.4 ± 10.9	0.001	0.0001

BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory; FIQ = Fibromyalgia Impact Questionnaire; HAS = Hamilton Anxiety Scale; HRSD = Hamilton Rating Scale for Depression.
[a]Mean ± SD in 15 patients.

Table 2. Correlation Between Change in Pain Scores and Anxiety and Depression Measures[a]

Score Change Pain	Treatment Time	
	Baseline–6 wk	6–12 wk
HAS	0.37	–0.03
HRSD	–0.17	–0.19
BAI	0.15	0.51
BDI	–0.09	–0.02

BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory; HAS = Hamilton Anxiety Scale; HRSD = Hamilton Rating Scale for Depression.
[a]r values; $p > 0.05$.

Table 3. Correlation Between FIQ Scores and Anxiety and Depression Measures[a]

Score Change FIQ	Treatment Time	
	Baseline–6 wk	6–12 wk
HAS	–0.02	–0.09
HRSD	0.02	0.13
BAI	–0.05	0.18
BDI	0.37	0.25

BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory; FIQ = Fibromyalgia Impact Questionnaire; HAS = Hamilton Anxiety Scale; HRSD = Hamilton Rating Scale for Depression.
[a]$p > 0.05$.

lafaxine in fibromyalgia patients. In conjunction with the 2 studies cited above, we propose that both norepinephrine and serotonin may play a role in the pathophysiology of fibromyalgia. We found a significant decrease from baseline in all measures of anxiety and depression at the end of the treatment. Given the high comorbidity of depression with fibromyalgia, one might speculate that venlafaxine has exerted its effect by alleviating psychological distress. This proved to be untrue in our study group where changes in the pain and FIQ scores were not correlated with the changes in the anxiety or depression scores. This particular finding is pointing toward an efficacy of venlafaxine in patients with fibromyalgia independent of its antidepressant and anxiolytic properties.

Our results differ from those of Dwight et al.[13] regarding the impact of past psychiatric disorders on treatment outcome. We found neither past nor current psychiatric disorders to predict treatment response to venlafaxine. There was no difference in the pain or FIQ scores in patients with or without a past or current psychiatric disorder. This is also in line with the finding that venlafaxine exerted its effect independent of its antidepressant properties. Antidepressants may also have effects in patients with pain regardless of the presence of a depressive syndrome.[27]

We should mention several caveats of our study. First, this was an open trial; it was neither controlled nor blinded, and the placebo response was therefore not possible to elicit. We did not elaborate a tender point measurement. Whether there was any decrease in tender points in response to treatment could have provided additional data, although in studies of antidepressant use in fibromyalgia, tender points seem to have improved only minimally despite improvement in measures of pain, global well-being, and sleep.[6,7] Five of 20 patients (25%) withdrew due to adverse effects of the drug, and this number is comparable to the one reported by Dwight et al. (4 of 15). This drop-out rate may be due to our fixed-dose regimen. Had we started the medication in smaller amounts, we might not have missed some of those patients. Finally, this sample size is too small to draw conclusions regarding which factors predicted treatment response.

Summary

We found venlafaxine to be quite promising in alleviating the pain and disability induced by fibromyalgia. This finding is in favor of the idea that medications that act on multiple neurotransmitters may prove to be more effective in symptom management.[28] Blockade of both norepinephrine and serotonin reuptake might therefore be more effective than blockade of either neurotransmitter alone in the treatment of fibromyalgia. This result should be corroborated further with a placebo-controlled, double-blind study.

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References

1. Wolfe F, Smythe HA, Yunus MB, Bennett RM, Bombardier C, Goldenberg DL, et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. Report of the Multicenter Criteria Committee. Arthritis Rheum 1990;33:160-72.
2. Wolfe F, Ross K, Anderson J, Russell IJ, Hebert L. The prevalence and characteristics of fibromyalgia in the general population. Arthritis Rheum 1995;38:19-28.
3. Cebi A. [The prevalence and sociodemographic characteristics of fibromyalgia in Trabzon region] Turkish. (dissertation). Trabzon: Karadeniz Technical University Medical School, 2001.
4. Goldenberg DL. Fibromyalgia syndrome a decade later: what have we learned? Arch Intern Med 1999;159:777-85.
5. Ahles TA, Khan SA, Yunus MB, Spiegell DA, Masi AT. Psychiatric status of patients with primary fibromyalgia, patients with rheumatoid arthritis and subjects without pain: a blind comparison of DSM-III diagnoses. Am J Psychiatry 1991;148:1721-6.
6. Arnold LM, Keck PE Jr, Welge JA. Antidepressant treatment of fibromyalgia. A meta-analysis and review. Psychosomatics 2000;41:104-13.
7. O'Malley PG, Balden E, Tomkins G, Santoro J, Kroenke K, Jackson JL. Treatment of fibromyalgia with antidepressants: a meta-analysis. J Gen Intern Med 2000;15:659-66.
8. Norregaard J, Volkmann H, Danneskiold-Samso B. A randomized controlled trial of citalopram in the treatment of fibromyalgia. Pain 1995; 61:445-9.
9. Anderberg UM, Marteinsdottir I, von Knorring L. Citalopram in patients with fibromyalgia — a randomized, double-blind, placebo-controlled study. Eur J Pain 2000;4:27-35.
10. Wolfe F, Cathey MA, Hawley DJ. A double-blind placebo controlled trial of fluoxetine in fibromyalgia. Scand J Rheumatol 1994;23:255-9.
11. Goldenberg DL, Mayskiy M, Mossey C, Ruthazer R, Schmid C. A randomized, double-blind crossover trial of fluoxetine and amitriptyline in the treatment of fibromyalgia. Arthritis Rheum 1996;39:1852-9.
12. Arnold LM, Hess EV, Hudson JI, Welge JA, Berno SE, Keck PE Jr. A randomized, placebo-controlled, double-blind, flexible-dose study of fluoxetine in the treatment of women with fibromyalgia. Am J Med 2002; 112:191-7.
13. Dwight MM, Arnold LM, O'Brien H, Metzger R, Morris-Park E, Keck PE Jr. An open clinical trial of venlafaxine treatment of fibromyalgia. Psychosomatics 1998;39:14-7.
14. First MB, Spitzer RL, Gibbon M, Williams JBW. Structured Clinical Interview for the DSM-IV Axis I Disorders — patient edition (SCID-I/P, version 2.0). New York: Biometrics Research Department, New York State Psychiatric Institute, 1995.
15. First MB, Spitzer RL, Gibbon M, Williams JBW: DSM-IV Eksen I Bozukluklari Icin Yapilandirilmis Klinik Gorusme. [Translated by A Corapcioglu]. Ankara: Hekimler Yayin Birligi, 1995.
16. Beck AT, Waed CH, Mendelson M. An inventory for measuring depression. Arch Gen Psychiatry 1961;4:561-71.
17. Beck AT, Epstein N, Brown G, Steer RA. An inventory for measuring clinical anxiety: psychometric properties. J Consult Clin Psychol 1988; 56:893-7.
18. Hamilton M. A rating scale for depression. J Neurol Neurosurg Psychiatry 1960;23:56-62.
19. Hamilton M. The assessment of anxiety disorders by rating. Br J Med Psychol 1959;32:50-65.
20. Burckhardt CS, Clark SR, Bennett RM. The Fibromyalgia Impact Questionnaire: development and validation. J Rheumatol 1991;18:728-33.
21. Hujkisson HC. Visual analog scales. In: Handbook of psychiatric measures. Washington, DC: American Psychiatric Press, 2000:601-3.
22. Sarmer S, Ergin S, Yavuzer G. The validity and reliability of the Turkish version of the Fibromyalgia Impact Questionnaire. Rheumatol Int 2000; 20:9-12.

23. Hisli N. [The reliability and validity study of the Beck Depression Inventory in a Turkish sample] Turkish. Psikoloji Dergisi 1988;6:118-22.
24. Ulusoy M, Sahin NH, Erkmen H. Turkish version of the Beck Anxiety Inventory: psychometric properties. J Cognitive Psychother 1998;12: 163-72.
25. Yazici MK, Demir B, Tanriverdi N, Karaagaoglu E, Yolac P. [Hamilton anxiety scale, inter-rater reliability and validity study] Turkish. Turk Psikiyatri Dergisi 1998;9:114-7.
26. Akdemir A, Turkcapar MH, Orsel SD, Demirergi N, Dag I, Ozbay MH. Reliability and validity of the Turkish version of the Hamilton Depression Rating Scale. Compr Psychiatry 2001;42:161-5.
27. Eberhard G, von Knorring L, Nilsson HL, Sundequist U, Bjorling G, Linder H, et al. A double-blind randomized study of clomipramine and maprotiline in patients with idiopathic pain syndromes. Neuropsychobiology 1988;19:25-34.
28. Miller LJ, Kubes KL. Serotonergic agents in the treatment of fibromyalgia syndrome. Ann Pharmacother 2002;36:707-12. DOI10.1345/aph.1A184

EXTRACTO

TRASFONDO: Aunque se desconoce la patofisiología de la fibromialgia, la transmisión monoaminérgica central puede ejercer un rol importante en su etiología. Los antidepresivos han sido efectivos en aliviar los síntomas de la fibromialgia. Fármacos que actúan sobre múltiples neurotransmisores pudieran ser más efectivos en manejar los síntomas.

OBJETIVO: Para evaluar la eficacia de venlafaxina, un inhibidor potente de la recaptación de norepinefrina y serotonina, en el manejo de pacientes con fibromialgia.

MÉTODOS: Se evaluaron 15 pacientes con fibromialgia antes y después del tratamiento con 75 mg diario de venlafaxina en dosis fija. El estudio duró 12 semanas y los pacientes fueron evaluados en las semanas 6 y 12 de tratamiento. Las medidas primarias de respuesta fueron la marca en el Cuestionario de Impacto de Fibromialgia (CIF) y la marca en la escala de dolor. Los niveles de ansiedad y depresión de los pacientes fueron medidos con la Escala de Depresión de Beck, la Escala de Ansiedad de Beck, y las Escalas de Depresión y Ansiedad de Hamilton.

RESULTADOS: Hubo mejoría significativa en la intensidad promedio del dolor ($F = 14.3$, $p = 0.0001$) y la incapacidad causada por la fibromialgia ($F = 42.7$, $p = 0.0001$) desde el inicio hasta la duodécima semana de tratamiento. Las marcas de ansiedad y depresión también bajaron significativamente entre el inicio y la semana duodécima de tratamiento. No hubo correlación entre la mejoría en el CIF y la clasificación del paciente o el médico del nivel de depresión o ansiedad. Tampoco hubo correlación entre el cambio en el nivel de dolor y los cambios en el nivel de ansiedad y depresión.

CONCLUSIONES: La venlafaxina promete ser buena en aliviar la incapacidad y el nivel del dolor causado por la fibromialgia. El efecto parece ser independiente de sus propiedades ansiolíticas y antidepresivas. En el manejo de fibromialgia, el bloqueo simultáneo de la recaptación de norepinefrina y de serotonina pudiera ser más efectiva que el bloqueo individual de cualquiera de estos neurotransmisores.

Mitchell Nazario

RÉSUMÉ

HISTORIQUE: La physiopathologie de la fibromyalgie n'est pas clairement connue, toutefois la transmission monoaminergique centrale pourrait être impliquée dans son étiologie. L'efficacité de certains antidépresseurs pour atténuer les symptômes de la fibromyalgie a été démontrée. Les agents touchant plusieurs neurotransmetteurs pourraient mieux contrôler les symptômes de la fibromyalgie.

OBJECTIF: Évaluer l'efficacité de la venlafaxine, un puissant inhibiteur de la recapture de la norépinéphrine et de la sérotonine, pour le traitement de la fibromyalgie.

SITE DE L'ÉTUDE: Clinique ambulatoire de psychiatrie d'un hôpital universitaire du nord-est de la Turquie (Karadeniz Technical University Medical School).

MÉTHODOLOGIE: Un groupe de 15 patients fibromyalgiques a été évalué avant et après un traitement à dose fixe de venlafaxine à dose quotidienne de 75 mg par jour. Les participants potentiels étaient identifiés à partir de la population diagnostiquée avec une fibromyalgie (selon les critères du Collège américaine de rhumatologie) à la clinique de physiothérapie ou la clinique de réadaptation. Avant de débuter la thérapie avec l'antidépresseur, une entrevue était conduite avec le patient en utilisant le guide d'entrevue clinique structurée (Structured Clinical Interview) pour les désordres de l'axe 1 selon le DSM IV. La durée de l'étude était de 12 semaines. Les participants étaient évalués lors de la 6e et de la 12e semaine de traitement. Les critères cliniques principaux d'efficacité étaient le score total ainsi que le score de douleur obtenus au questionnaire d'impact sur la fibromyalgie (FIQ: Fibromyalgia Impact Questionnaire). Les critères cliniques secondaires étaient les niveaux d'anxiété et de dépression mesurés avec les échelles de Beck et de Hamilton pour l'anxiété et la dépression.

RÉSULTATS: Une amélioration significative a été observée à la 12e semaine, comparativement à la valeur de départ, pour l'intensité moyenne de la douleur ($F = 14.3$; $p = 000.1$) ainsi que pour l'incapacité causée par la fibromyalgie ($F = 42.7$; $p = 0.0001$). Les scores de dépression et d'anxiété étaient aussi significativement diminués à la 12e semaine. L'amélioration du score total d'impact sur la fibromyalgie (FIQ) n'a pas montrée de corrélation avec la diminution des scores de dépression et d'anxiété obtenus; et ceci, tant pour les scores mesurés par les patients que ceux mesurés par le médecin. Le changement du score de douleur n'était pas non plus en corrélation avec les scores de dépression et d'anxiété.

CONCLUSIONS: L'efficacité de la venlafaxine pour soulager la douleur et l'incapacité causée par la fibromyalgie, mesurée dans cette étude, s'avère prometteuse. Cet effet semble indépendant des propriétés anxiolytiques et antidépressives de la venlafaxine. Le blocage simultané de la recapture de la norépinéphrine et de la sérotonine pourrait traiter plus efficacement la fibromyalgie que le blocage d'un seul des 2 neurotransmetteurs.

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