

THE MMPI-2 NEUROTIC TRIAD SUBSCALES AND DEPRESSION LEVELS AFTER PHARMACOLOGICAL TREATMENT IN PATIENTS WITH DEPRESSIVE DISORDERS - CLINICAL STUDY

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SUMMARY

Background: Affective disorders provide for one third of the main causes of psychiatric inpatient care, both in male and female subjects. An early diagnosis of the disease with precise identification of the character of its particular symptoms are key important factors for the efficacy of treatment. The goal of the study was an identification of possible associations between scores of the neurotic triad in the MMPI-2 test (hypochondria - Hs, depression - D, hysteria - Hy), evaluated at initial hospitalization period with remission degree assessed by the Hamilton Depression Rating Scale (HDRS), following eight weeks of treatment with SSRI.

Subjects and methods: A group of 50 subjects took part in the study. The MMPI-2 test and HDRS were used in the study. The HDRS was performed at the therapy onset and reapplied after 8 weeks of its continuation. The MMPI-2 test was applied at the beginning of treatment.

Results: Higher scores in Hs ($p=0.007$), D ($p=0.021$) and Hy scales ($p=0.001$) are associated with the higher degree of depression, measured by the HDRS at the therapy onset. The highest performance in Hs scale ($p=0.003$) and Hy scale ($p=0.001$) evaluated on admission, was associated with the highest depression level after pharmacological treatment.

Conclusion: The higher the degree of hypochondria and hysteria symptoms, measured by the MMPI-2 test at the onset of therapy in patients with depressive disorders, the higher severity of depression is being found after 8 weeks of therapy with SSRI agents, measured by the HDRS scale.

Key words: depressive disorder - MMPI - pharmacotherapy

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INTRODUCTION

Affective disorders constitute one third of the main causes for psychiatric inpatient care, regarding both male and female subjects. Every year, approximately 100 million people all over the world demonstrate symptoms of depression, which means that depressive disorders affect at least 15% of the adult female and 10% of the adult male population (Talarowska et al. 2009). An early diagnosis of the disease with precise identification of the character of its particular symptoms and the disease course in each individual patient are key factors for the efficacy of administered medical treatment (Klonsky & Bertelson 2000, Biles 2005).

According to the data, presented by the World Health Organisation, as many as 95% of all psychiatric patients report various physical ailments, depression being one of prevailing disease entities in this group of patients. In turn, almost 2/3 of patients with depressive disorders experience physical pain (Garcia-Cebrian et al. 2006, Krebs & Bair 2008) and in approximately 90-99% of the patients, at least one physical symptom is present (fatigue in 85%, sleep disorders in 78%, appetite changes in 58%, generalized pain in 49% and sexual dysfunction in 48%) (Mustapha 2005). Furthermore, it may be concluded that as many as 69% of patients with later diagnosed depression attend a GP for more or less intense physical complaints (Lerman et al. 2010). The

most recent advances in neurobiological research provide increasing evidence that inflammatory and neuroprogressive processes play a significant role in depression. Preclinical and clinical studies on depression highlight an increased production of inflammatory markers, such as interleukin (IL)-1, IL-6, tumor necrosis factor- α and interferon- α and γ . In animal models, acute and chronic administration of cytokines or cytokine inducers trigger depressive symptoms (Maes 1999). By contrast, Pols & Battersby (2008) observed an inverse relationship, namely that among patients with somatic symptoms (including 300 examined subjects), 50% fulfilled the diagnostic criteria of depression, while various degrees of anxiety are identified in the remaining 35%. It should be kept in mind that intensive somatic symptoms (including pain) accompany not only the so-called masked depression but they occur together with typical depressive disorders as well. In the former group, out of the above-mentioned ones - somatic symptoms come into prominence and are thus relatively early identified both by the patient himself/herself and by his/her physician. In these cases, the symptoms which are characteristic for depressive disorders, such as depressed mood, disturbed circadian rhythms, the feeling of guilt or loss of body weight are less frequent and less enhanced, while physical complaints overlap the clinical picture of the base disease, impeding the diagnosis (Mihaila 2004).

The Minnesota Multiphasic Personality Inventory (MMPI-2) test by S. Hathaway and J. McKinley is a psychological tool, used in the diagnostics of various disorders, and is also helpful in determining the mode of planned psychotherapeutic interventions. When applying the MMPI-2 test profilogram during assessment of depression symptoms, there are multiple scales and subscales for disposal, characterised by various degrees of diagnostic accuracy, e.g., the clinical depression scale (D), the Harris-Lingoes subscales of depression (D1, D2, D3, D4 and D5), the depression content scale (DEP), the components of content scales, concerning depression (DEP1, DEP2, DEP3, DEP4), selected critical questions by Koss-Butcher and Lachara-Wrobel or Wiener-Harmon's scale of symptoms of obvious (O-D) and subtle (S-D) depression. Regarding patients with depression, those scales are also diagnostic, revealing patient's concentration on somatic and physical functioning of his/her body, including the hypochondria scale (Hs) and the hysteria scale (Hy). The depression, hypochondria and hysteria scales constitute the 'neurotic triad' (Hathaway & McKinley 1943, Jones 2001, Biles 2005). High scores in all the three scales are associated with an excessive concentration on somatic health status, frequent complaints of physical ailments, lack of energy, sleep problems, impaired attention, concentration and low self-esteem, diffidence and

pessimism. Subjects in this group react to difficult situations with strong somatic symptoms. They are nervous, impatient and live under constant tension (Kucharski 2002, Talarowska et al. 2010).

The aim of this study was to identify possible associations between scores in the neurotic triad in the MMPI-2 test (hypochondria, depression, hysteria), measured at the time of hospital admission and remission degree assessed by the Hamilton Depression Rating Scale (HDRS) following 8 weeks of pharmacological treatment with selective serotonin reuptake inhibitors (SSRI), in a group of patients diagnosed with depressive disorders.

SUBJECTS AND METHODS

Subjects

The reported study comprised 50 subjects (women n=30; men n=20), aged 20-62. The studied groups did not differ significantly in terms of gender ($p>0.05$). Education was measured by the number of years of completed school education (years at school). Considering the Polish education system, the education period ≤ 9 years was considered as primary education, 10-12 years - secondary and >12 years - higher education. See Table 1 for the demographic characteristics of the studied group and for data on the disease course.

Table 1. Demographic characteristics of the studied group and the data on disease course

Variable		N	%	M	SD	range
Gender	F	30	60.00	-	-	-
	M	20	40.00	-	-	-
Age	-	-	-	44.12 yrs	12.39 yrs	20-62 yrs
Education	Primary	19	38	-	-	-
	Secondary	23	46	-	-	-
	High	8	16	-	-	-
Education period	-	-	-	11.66 yrs	2.73 yrs	9-17 yrs
Disease	Duration of the disease	-	-	8.06 yrs	8.73 yrs	2-30 yrs
	Number of hospitalization episodes for depressive disorders	-	-	2.56	1.85	1.00-8.00
	Number of depression episodes	-	-	7.05	7.54	1.00-16.00

Patients were selected for the study according to the inclusion diagnostic criteria of ICD-10 (F32, F33) (1993). Patients with the diagnosis of severe depressive episode with psychotic symptoms (F32.3), other depressive episodes (F32.8), recurrent depressive disorder, current episode severe with psychotic symptoms (F33.3), recurrent depressive disorder, currently in remission (F33.4) and other recurrent depressive disorders (F33.8) were not enrolled in the study. The presence of mental disorders, other than depressive episode, and the diagnosis of somatic diseases and injuries of the central nervous system (CNS), were also regarded as exclusion criteria. Brain imaging techniques

(computer tomography and magnetic resonance) were performed in order to assess changes in the CNS. The study group included subjects, hospitalized for the first time for depressive episode and depression treatment-naïve, as well as those, treated for many years before and with multiple hospitalisation episodes in their history, the latter admitted for various degrees of health deterioration. All the subjects from the DD group were examined during the course of their hospitalization. In assessing the presence of organic changes in the CNS the imaging studies (computed tomography and magnetic resonance imaging) were used. In all the included subjects, case history was obtained prior to

main study procedure, using the standardized Composite International Diagnostic Interview (Patten 1997). Additionally, the number of depression episodes, the disease duration periods and the number of hospitalization episodes were recorded in each patient.

No evaluations of the intellectual functions of the enrolled patients were carried out prior to the psychological examination. However, on the basis of medical records and anamnesis, it was established that none of the participants had been diagnosed with mental disability or any of the analyzed intellectual deficits.

During hospitalization, all the patients received anti-depressant pharmacotherapy (monotherapy), including SSRI (Selective Serotonin Reuptake Inhibitors) group: 25 patients received fluoxetine, the onset dose (20 mg/day, the maximum dose: 60 mg/day, the mean dose (M): 35 mg/day, SD 9.5), 8 patients received sertraline (the onset dose: 50 mg/day, the maximum dose: 200 mg/day, M 130 mg/day, SD 14.7), 12 patients were administered citalopram (the onset dose: 20 mg/day, the maximum dose: 40 mg/day, M 31 mg/day, SD 4.3), 5 patients received paroxetine (the onset dose: 20 mg/day, the maximum dose: 60 mg/day, M 32 mg/day, SD 3.9). The specified agents were administered in therapeutic doses as defined by Taylor, Paton and Kerwin (2007).

Although 66 patients were initially enrolled in the study, the data represent the results of those patients (n=50) who achieved remission after 8 weeks of pharmacotherapy treatment. 10 patients dropped out from the study group due to deterioration in mental state or lack of improvement, 6 due to serious adverse effects (2 - dizziness and headache, 2 - nausea and other gastrointestinal symptoms, 2 - growing agitation and anxiety).

Methods

Neurotic triad of MMPI-2. The Polish version of the Minnesota Multiphasic Personality Inventory by S. Hathaway and J. McKinley, adapted by T. Kucharski (MMPI-2) (2002), was used in the study in paper- and-pencil form. The result of the study in its standard version is a scoring profile, comprising three control scales and results of the following ten (10) clinical scales: Hypochondria (Hs), Depression (D), Hysteria (Hy), Psychopathic Deviate (Pd), Masculinity/Femininity (Mf), Paranoia (Pa), Psychasthenia (Pt), Schizophrenia (Sc), Mania (Ma), Social Introversion (Si) (Moser et al., 2007). Results, obtained in the three scales of the, so-called, neurotic triad: hypochondria (Hs), depression (D), hysteria (Hy) were analyzed.

Reliability evaluation. Gough Dissimulation Index was used as a validity indicator for MMPI testing. It is calculated from the raw test results: the results of the F scale - results in the K scale. The scale of F detects deviant and unusual/atypical ways of responding. High results in F scale may indicate the severity of perceived life difficulties and reveal simulation tendencies. The K

scale is designed to detect defensiveness and measures the willingness to disclose personal information. A high score reflects an uncooperative attitude and reluctance to disclose personal information (Kucharski 2002).

Severity of depression. The severity of depression was assessed using the 21-item Hamilton Depression Rating Scale (HDRS) (Hamilton 1960, Moonseong et al. 2007). The HDRS consists of items evaluating depressed mood, psychomotor agitation, inhibition, the sense of guilt, sleep and appetite disorders, anxiety symptoms, suicidal thoughts and self-criticism. The questionnaire was designed by Hamilton in 1960 and has been and still is one of the most frequently used tools to evaluate the degree of depression symptoms, especially their dynamics during the episode. Cronbach a coefficient of reliability, calculated for the scale, was - on the average - 0.70 (Bagby et al. 2004), of sensitivity 0.78 and of specificity 0.75 (Aben et al. 2002). The severity of depressive symptoms were assessed using a four-point scale (for questions 1-13) and at two-point scale (for questions 14-15). The scores, obtained from the 4 last questions, were not included into the total result. Depressive symptom intensity levels were classified by the grades, specified in the study by Demyttenaere and De Fruyt (2003): <7 - no depressive symptoms, 8-12 - mild depressive symptoms, 13-17 - moderate depressive symptoms, 18-29 - severe depressive symptoms, >30 - very severe depressive symptoms (the authors based their interpretation of results on the 17-item version of the scale). The HDRS scale is also used for evaluation of clinical improvements after applied pharmacological therapy. Mental status improvement and the efficacy of applied therapy were evaluated in two aspects: the response to therapy and disease remission. The response to therapy was defined as $\geq 50\%$ reduction of depression symptoms vs. the base level, while the HDRS score <8 was regarded as disease remission.

Rater and interrater training

The HDRS was performed at the therapy onset (on admission) and after 8 weeks of its continuation. The MMPI-2 test was applied at the beginning of the pharmacological treatment. All the patients were examined on admission, i.e., at the symptomatic phase, before or shortly after previous antidepressant drug regime modification. The study by the above-mentioned tests was in each case performed by the same person: MMPI-2 test was performed and its results were evaluated by the same psychologist, while HDRS evaluation was performed by the same physician-psychiatrist.

Data analysis

Statistical analysis of the collected material employed descriptive methods, as well as a statistical conclusion. In order to describe the studied group of patients, structural indexes were calculated in the

qualitative analysis of characteristics. In order to estimate the average values for the quantitative characteristics, arithmetic means (M) were calculated. Standard deviation (SD) was adopted as the measure of scatter of results.

The Lilliefors (Kolmogorov-Smirnov) test for normality was used to evaluate distribution normality of the studied variables. The t-test for paired groups was used to evaluate differences in the degree of depressive disorders, both on admission (HDRS-I) and after 8 weeks of the therapy continuation (HDRS-II). The relationships between MMPI-2 performance levels, evaluated on admission, depression degree, assessed by the HDRS before and after eight (8) weeks of pharmacological treatment, were expressed as Pearson's correlation coefficients. Student's-t test was used to evaluate differences in the neurotic triad performance levels in the patients with remission and in those without remission on the day of discharge. Statistical analyses were performed using the STATISTICA Program v. 8, and the p value for statistical significance was: $p < 0.05$.

Ethics

An informed, written consent for participation in the study was obtained from each subject, according to the protocol, approved by the Bioethical Committee of the Medical University of Łódź (No RNN/603/08/KB).

RESULTS

On admission, 2 subjects met the Hamilton Depression Rating Scale score criteria for mild depression episode, 9 for moderate one and 39 for severe depression episode. On the day of discharge, 26 subjects did not meet the HDRS criteria for depressive disorder, 20 met the HDRS criteria for mild depression and 4 for moderate one (see Figure 1). Figure 2 demonstrates the final evaluation of therapy efficacy in the HDRS scale (see Figure 2).

In the study group, 10 patients were diagnosed with depressive episode (F32), and 40 with recurrent

depressive disorder (F33). Due to the small number of patients in the first group, no separate analysis was performed. This group included all antidepressants-naïve patients.

The mean values of results in the studied group for Hs, D and for Hy scale are presented in Table 2. Statistically significant differences were found in the intensity of depression symptoms, measured by the HDRS in the examined group on therapy onset (HDRS-I) vs. the examination results after 8 weeks of treatment (HDRS-II) ($p < 0.001$) (see Table 2). The mean Gough indicator values showed cooperative attitude and willingness to disclose personal information of subjects from the study group, allowing valid and reliable interpretation of the test results (see Table 2).

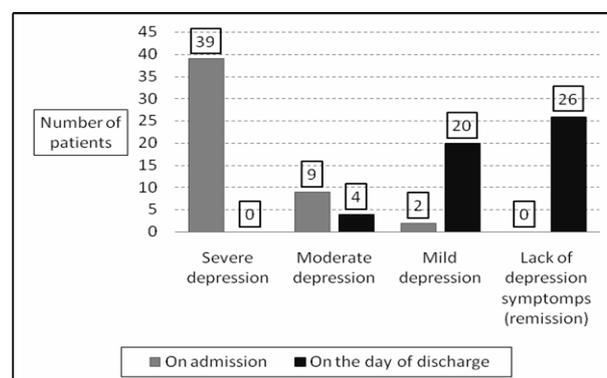


Figure 1. Severity of depression symptoms in the study group (n=50) on admission and discharge

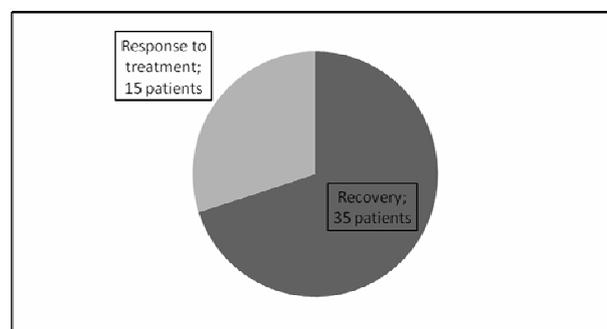


Figure 2. Evaluation of therapy efficacy in the study group (n=50) by the HDRS scale

Table 2. Scores of particular MMPI-2 scales in the study group and the degree of depression symptoms, measured by HDRS at the onset of pharmacological therapy and after 8 weeks of its continuation

Variable	Min.	Max.	M	SD	t	p
Hypochondria scale	43.00	93.00	74.89	14.67		
Depression scale	50.00	99.00	79.85	11.17	-	-
Hysteria scale	45.00	99.00	74.45	13.18		
Gough Index	-14.00	34.00	8.32	10.76		
HDRS-I**	10.00	48.00	23.64	7.75	15.22	<0.001*
HDRS-II***	1.00	15.00	6.82	4.09		
Difference between HDRS-II and HDRS-I	1.00	44.00	16.82	7.82	-	-

* p - statistically significant, $p < 0.05$; **HDRS-I - the degree of depression symptoms, measured by HDRS at the onset of pharmacological treatment; ***HDRS-II - the degree of depression symptoms, measured by HDRS after 8 weeks of pharmacological therapy continuation.

No statistically significant differences were observed in the neurotic triad performance levels between the patients with remission on the day of discharge (HDRS <7 score) and those without remission on the day of discharge: Hs (t(48)=0.639, ns), D (t(48)=0.523, ns), Hy (t(48)=0.326, ns). However, the patients in the former group obtained lower score values in each scale of the neurotic triad vs. those in the latter group (see Table 3).

Statistical analysis (see Table 4) revealed significant relationships between HDRS scores before and after 8 weeks of pharmacotherapy and MMPI-2 scales before the administered treatment. Higher scores in Hs (p=0.007), D (p=0.021) and Hy scales (p=0.001) are

associated with the higher degree of depression, measured by the HDRS at the therapy onset. The highest performance in Hs scale (p=0.003) and Hy scale (p=0.001) evaluated on admission, was connected with the highest depression level after pharmacological treatment. Also the difference between HDRS-II and HDRS-I results negatively correlates with both above-mentioned scales. The lower Hs (p=0.048) and Hy (p=0.031) degrees before pharmacotherapy, the higher the difference between HDRS-II and HDRS-I, what indicated better health improvement of the examined patients.

Table 3. Differences in performance levels of the scales in the MMPI-2 test neurotic triad between the patients with remission on the day of discharge (HDRS < 7 score) and those without remission (HDRS > 7 score)

Variable	HDRS <7 points		HDRS >7 points		t	p
	M	SD	M	SD		
Hypochondria scale	73.96	14.826	75.96	14.761	-0.471	0.639
Depression scale	78.88	11.978	80.96	10.337	-0.644	0.523
Hysteria scale	72.69	14.246	76.43	11.866	-0.992	0.326

*p - statistically significant, p<0.05; **HDRS - Hamilton Depression Rating Scale

Table 4. The Pearson's correlation coefficient for scales of the MMPI-2 neurotic triad and HDRS values

Variable	HDRS-I**		HDRS-II***		Difference between HDRS-II and HDRS-I	
	Pearson's correl. coef.	p	Pearson's correl. coef.	p	Pearson's correl. coef.	p
Hypochondria (Hs) High scores reflect undefined physical problems, concern for own health, concentration on invented somatic problems, lack of energy, dissatisfaction, sleep problems, complaining, claiming attitude.	0.464	0.007*	0.378	0.003*	-0.281	0.048*
Depression (D) High scores reflect depressive mood, low self-esteem and the feeling of being inappropriate, worrying, dissatisfaction with life status, withdrawal	0.326	0.021*	0.072	0.262	-0.112	0.439
Hysteria (Hy) High scores mean little insight into life problems and emotions, numerous somatic fears, sleep problems, negation, claiming approach, self-concentration.	0.471	0.001*	0.438	0.001*	-0.305	0.031*

* p - statistically significant, < 0.05; **HDRS-I - the degree of depression symptoms, measured by HDRS at the onset of pharmacological treatment; *** HDRS-II - the degree of depression symptoms, measured by HDRS after 8 weeks of pharmacological therapy continuation

DISCUSSION

The presented results come from the first attempt of evaluating possible correlations between MMPI-2 test results and the efficacy of anti-depression therapy. The degree of depression, hypochondria and hysteria at the beginning of the therapy positively correlated with the degree of depression symptoms, measured by the HDRS before pharmacotherapy. The degree of hypochondria

and hysteria symptoms at the beginning of therapy positively correlated with the degree of depression symptoms after 8 weeks of therapy continuation. Also the difference between HDRS-II and HDRS-I results (indicating the degree of improvement) negatively correlated with both above-mentioned scales (see Table 4). Despite the fact that no significant differences were demonstrated in the MMPI-2 performance between the patients with and without remission, still the patients in

the former group obtained lower results in each of the three scales of the neurotic triad (see Table 3). Therefore, a conclusion may be considered that the higher the degree of hypochondria and hysteria scale symptoms at therapy onset, the higher degree of depression symptoms (measured by HDRS) after 8 weeks of pharmacological therapy with SSRI agents. No such correlations were observed with regards to the depression scale.

These results are identical with the view which dominates in literature reports, namely that out of all the MMPI-2 test scales, the depression scale is the most accurate one for nosological and differential diagnosis of depressive disorders (Nelson et al. 1996, Greenblatt & Davis 1999, Baqby et al. 2005). Some authors indicate, however, a higher diagnostic value of content scale components with regards to depression - DEP (Gross 2002). According to Klonsky & Bertelson (2000), higher scores in the depression scale are obtained both by patients with depressive disorders and those with identified dysthymia. However, only in the former group, are high scores additionally observed in the hypochondria and hysteria scales, which indicates a higher number of somatic symptoms in those patients vs. those with dysthymia. As demonstrated in the studies by Slesinger (2001), Hs, D and Hy scores are also elevated in depressive patients, who complain of enhanced pain sensations, when compared to depressive patients without such symptoms. Moreover, patients with the diagnosis of masked depression obtain higher scores in Hs and Hy scales, while patients with diagnosed depressive disorders present with higher scores in the depression scale (Mihaila 2004). According to Grossardt et al. (2009), pessimistic, anxious, and depressive personality traits (measured by MMPI) were associated with increased all-cause mortality in both men and women. These associations remained significant even when personality was measured early in life (ages 20-39 years). Those findings suggest that personality traits, related to neuroticism, are associated with an increased risk of all-cause mortality, even when they are measured early in life. Personality is linked to a variety of health problems. In the study by Cardoni (2009), the relationship between elevations on MMPI clinical scales and back surgery outcome was assessed. Further, moderator effects (such as treatment components, differences in outcome measures, gender and age of patient, and location of injury) were analyzed to determine the impact of potential pre-existing factors on surgical outcome. Hypochondria, Depression, and Hysteria did result in medium-sized correlations with treatment outcome.

Our results may also be supported by the relationships, observed between depression and somatic sensations, including pain, which are extensively reported in literature. The majority of patients with depressive disorders come to a GP mainly for enhanced physical sensations (Lerman et al. 2010, Teh et al. 2010). Patients with depression evaluate their pain

sensations as more intensive than patients with pain alone and the higher is the intensity of pain sensation, which those patients report, the higher are their depression symptoms (Bair et al. 2008, Ehnavall et al. 2009, Fister 2009). Chronic pain, which accompanies depressive disorders, complicates the course of therapy and is associated with lower efficacy of anti-depressant treatment (Greco et al. 2004, Hush 2009). Pain symptoms are associated with various depression degrees, while pain alleviation may significantly improve depression control (DeVeugh-Geiss et al. 2010). The presented results also demonstrated a necessity to evaluate somatic symptoms among patients with depressive disorders. Such an evaluation may influence the efficacy of applied therapy.

The results of numerous studies indicate lower efficacy of SSRI agents in treatment of pain symptoms (headaches, pains in the course of fibromyalgia, neuropathic pains in the course of diabetes, arthralgias, idiopathic pains of various localisations) vs. tricyclic anti-depressants (TCAs) or drugs from the group of serotonin-norepinephrine reuptake inhibitors (SNRI) (Saper et al. 2001, Briley 2004, Moja et al. 2005, Mallinckrodt et al. 2007). Rahimi et al. (2008) evaluated the efficacy of SSRIs for the management of irritable bowel syndrome (IBS) by the meta-analysis technique. SSRIs do not significantly improve abdominal pain, abdominal bloating or IBS symptoms. In the studies by DeVeugh-Geiss AM et al. (2010) when compared to patients with no pain at baseline, those with severe pain were less likely to achieve remission and partial response. Patients with early pain improvement were more likely to achieve remission (data from the Randomized Trial Investigating SSRI Treatment (ARTIST)). Also in the studies of Perahia et al. (2009), among patients, treated for depressive disorders with agents of the SSRI group (HDRS score >15), in whom enhanced somatic symptoms were observed, the improvement of health was rather slight. Our results are conformable with those in the above reports. The drugs chosen from the SSRI group turned out to be less effective among the patients who reported multiple somatic complaints before the onset of pharmacotherapy. It should be emphasized that not only objectively diagnosed somatic diseases but also a subjective evaluation of their intensity by the patient himself/herself is of key importance for remission of depressive disorders. It should also be kept in mind that the degree of somatic complaints is significantly reduced during the first month of pharmacological therapy and the mood improves gradually in the course of a few subsequent months of continued therapy (To et al. 2005, Tylee & Gandhi 2005), while an enhanced intensity of unpleasant somatic sensations (including pain) significantly increases the risk for recurrence of affective disorders.

Since HDRS, as well as other, commonly known scales, used for evaluation of depressive symptoms,

does not include a sufficient number of questions, which would facilitate the diagnosis of somatic symptoms, it seems appropriate to perform a detailed assessment of all the symptoms in depressive disorders, including the MMPI-2 test, prior to selection of pharmacological therapy.

LIMITATIONS

The number of patients in the study group may be perceived as a limitation for the reported study, as the number of patients in any study group may affect the statistical value of methods. What is more, the correlations found by the authors are relatively weak. In consideration of such limitations, the authors suggest that one should interpret the presented results with some caution, emphasizing, however, that the reported study, despite its character of a preliminary report, unequivocally demonstrates its key issue, i.e., the correlations between personality traits, measured by MMPI, and depression levels after pharmacological therapy. The obtained results are, however, important for pharmacological therapy of depression and can become a useful prompt for subsequent studies on this issue.

CONCLUSION

The higher the degree of hypochondria and hysteria symptoms, measured by the MMPI-2 test at the onset of therapy in patients with depressive disorders, the higher is the severity of depression found after 8 weeks of therapy with SSRI agents, measured by the HDRS scale.

REFERENCES

1. Aben I, Verhey F, Lousberg R, Lodder J & Honig A: Validity of the Beck Depression Inventory, Hospital Anxiety and Depression Scale, SCL-90, and Hamilton Depression Rating Scale as screening instruments for depression in stroke patients. *Psychosomatics* 2002; 43: 386-393.
2. Bair M, Wu J, Damush T, Sutherland J & Kroenke K: Association of depression and anxiety alone and in combination with chronic musculoskeletal pain in primary care patients. *Psychosom Med* 2008; 70: 890-897.
3. Baqby RM, Ryder AG, Schuller DR & Marshall MB: The Hamilton Depression Rating Scale: has the gold standard become a lead weight? *Am J Psychiatry* 2004; 161: 2163-2177.
4. Baqby RM, Marshall MB, Basso MR, Nicholson R, Bacchiochi J & Miller L: Distinguishing bipolar depression, major depression, and schizophrenia with the MMPI-2 Clinical a Content Scales. *J Per Assess* 2005; 84: 89-95.
5. Biles L: A taxometric analysis of the MMPI/MMPI-2 depression scales. A Dissertation Presented to the Faculty of Pacific Graduate School of Psychology Palo Alto, 2005.
6. Briley M: Clinical experience with dual action antidepressants in different chronic pain syndromes. *Hum Psychopharmac* 2004; 19:21-25.
7. Cardoni M: Utility of the MMPI as a predictor for back treatment outcome: a meta-analysis. The University of Tulsa, 2009.
8. Demyttenaere K & De Fruyt J: Getting what you ask for: on the selectivity of depression rating scales. *Psychother Psychosom* 2003; 72:61-70.
9. DeVeauugh-Geiss AM, West SL, Miller WC, Sleath B, Gaynes BN & Kroenke K: The adverse effects of comorbid pain on depression outcomes in primary care patients: results from the ARTIST trial. *Pain Med*. 2010; 11:732-741.
10. Ehnvall A, Mitchell P, Hadzi-Pavlovic D, Malhi G & Parker G: Pain during depression and relationship to rejection sensitivity. *Acta Psychiatr Scand* 2009; 119: 375-382.
11. Fister K: A combined primary care intervention works for pain and depression. *Br Med J (International edition)* 2009; 338: 1355-1361.
12. Garcia-Cebrian A, Gandhi P, Demyttenaere K & Peveler R: The association of depression and painful physical symptoms-a review of the European literature. *Eur Psych* 2006; 21: 379-388.
13. Greco T, Eckert G & Kroenke K: The outcome of physical symptoms with treatment of depression. *J Gen Intern Med* 2004; 19: 813-818.
14. Greenblatt RL & Davis WE: Differential diagnosis of PTSD, schizophrenia, and depression with the MMPI-2. *J Clin Psychol* 1999; 55: 217-223.
15. Gross K: The incremental validity of the MMPI-2 and demographic variables in assessing major depression. Palo Alto: Pacific Graduate School of Psychology, 2002.
16. Grossardt BR, Bower JH, Geda YE, Colligan RC & Rocca WA: Pessimistic, anxious, and depressive personality traits predict all-cause mortality: the Mayo Clinic cohort study of personality and aging. *Psychosom Med* 2009; 71: 491-500.
17. Hamilton M: A rating scale for depression. *J Neurol, Neurosurg Psychiatry* 1960; 23: 56-62.
18. Hathaway SR & McKinley JC: The Minnesota Multiphasic Personality Inventory. Minneapolis: University of Minnesota Press, 1943.
19. Hush J: Combined pain self-management and antidepressant therapy are effective in patients with chronic musculoskeletal pain with depression. *Aust J Physiother* 2009; 55:208-209.
20. ICD-10 Classification of Mental & Behavioural Disorders. Geneva: World Health Organization, 1993.
21. Jones A: An examination of the MMPI-2 Wiener-Harmon subtle subscales for D and Hy: Implications for parent scale and neurotic triad interpretation. *J Pers Assess* 2001; 77:105-121.
22. Klonsky ED & Bertelson AD: MMPI-2 clinical scale differences between dysthymia and major depression. *Assessment* 2000; 7:143-149.
23. Krebs EE & Bair MJ: Advancing the science of primary care pain medicine. *Pain Med* 2008; 9: 490-492.
24. Kucharski T: Selected issues connected with polish adaptation of the MMPI-2 and the MMPI-A. Toruń: CPKiROZ, 2002.
25. Lerman SF, Zvia R & Golan S: Distinguishing affective and somatic dimensions of pain and depression: a confirmatory factor analytic study. *J Clin Psychol* 2010; 66:456-465.

26. Maes M: Major depression and activation of the inflammatory response system. *Adv Exp Med Biol*. 1999; 461:25-46.
27. Mallinckrodt C, Prakash A, Houstin J, Swindle R, Detke M & Fava M: Differential antidepressant symptom efficacy: placebo-controlled comparisons of duloxetine and SSRIs (fluoxetine, paroxetine, escitalopram). *Neuropsychobiol* 2007; 56:73-85.
28. Mihaila E: Impression management and self-deception positivity in the identification of depression with the MMPI-2. Palo Alto: Pacific Graduate School of Psychology, 2004.
29. Moja P, Cusi C, Sterzi R & Canepari C: Selective serotonin re-uptake inhibitors (SSRIs) for preventing migraine and tension-type headaches. *Cochrane Database System Revision* 2005; 3: CD002919.
30. Moonseong H, Murphy CF & Meyers BS: Relationship between the Hamilton Depression Rating Scale and the Montgomery-Åsberg Depression Rating Scale in depressed elderly. *Am J Ger Psychiatry* 2007; 15: 899-905.
31. Mustapha A: Depression can cause severe physical symptoms. *Br J Nur* 2005; 14:482.
32. Nelson LD, Pham D & Uchiyama C: Subtlety of the MMPI-2 depression scale: A subject laid to rest? *Psychol Assess* 1996; 8:331-333.
33. Patten S: Performance of the Composite International Diagnostic Interview Short Form for Major Depression in community and clinical samples. *Chronic Dis Can* 1997; 3:109-112.
34. Perahia DG, Quail D, Desaiyah D, Montejo AL & Schatzberg AF: Switching to duloxetine in selective serotonin reuptake inhibitor non- and partial-responders: effects on painful physical symptoms of depression. *J Psychiatr Res*. 2009; 43:512-518.
35. Pols RG & Battersby MW: Coordinated care in the management of patients with unexplained physical symptoms: depression is a key issue. *Med J Aust* 2008; 188: 133-137.
36. Rahimi R, Nikfar S & Abdollahi M: Selective serotonin reuptake inhibitors for the management of irritable bowel syndrome: A meta-analysis of randomized controlled trials. *Arch Med Sci* 2008; 1: 71-76.
37. Saper JR, Lake AE & Tepper SJ: Nefazodone for chronic daily headache prophylaxis: an open-label study. *Headache* 2001; 41: 465-474.
38. Slesinger D: Minnesota Multiphasic Personality Inventory-2: Correlates in a chronic pain population. Virginia Consortium for Professional Psychology Old Dominion University; 2001.
39. Talarowska M, Florkowski A, Galecki P, Wysokiński A & Zboralski K: Cognitive functions in depression. *Psych Pol* 2009; XLIII: 31-40.
40. Talarowska M, Florkowski A, Zboralski K & Galecki P: Differences in the course of depressive disorders among women and men measured by MMPI-2. *Psych Pol* 2010; XLIV: 319-328.
41. Taylor D, Paton C & Kerwin R: The Maudsley prescribing guidelines. London: Informa Healthcare, 2007.
42. Teh FC, Zaslavsky AM, Reynolds III CF & Cleary PD: Effect of depression treatment on chronic pain outcomes. *Psychosom Med* 2010; 72: 61-67.
43. To S, Zepf R & Woods A: The Symptoms, Neurobiology, and Current Pharmacological Treatment of Depression. *J Neurosci Nurs* 2005; 37: 102-107.
44. Tylee A & Gandhi P: The importance of somatic symptoms in depression in primary care. *Prim Care Comp J Clin Psychiatry* 2005; 7: 167-176.

List of abbreviations used in the paper:

- MMPI-2 - The Minnesota Multiphasic Personality Inventory (2)
HDRS - Hamilton Depression Rating Scale
SSRI - selective serotonin reuptake inhibitors
ICD – 10 – International Classification of Diseases - 10
D – depression scale
Hs - hypochondria scale
Hy - hysteria scale
CNS - central nervous system
M – mean
SD – standard deviation
HDRS-I - degree of depressive disorders on admission
HDRS-II – degree of depressive disorders after 8 weeks of the therapy continuation

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