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**Obsessive-compulsive disorder
– psychopathology and therapy¹**

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Summary

Both clinical observations and therapy outcome studies of patients diagnosed with obsessive-compulsive disorder (OCD) suggest that this ICD-10 category is heterogeneous. It most probably includes three separate kinds of disorder with a similar clinical pattern but different pathomechanisms. Psychopathological analyses of 62 OCD patients and preliminary results of their treatment are presented.

Key words: obsessive-compulsive disorder, psychotherapy outcome

Introduction

Both clinical observations and therapy outcome studies of patients diagnosed with obsessive-compulsive disorder (OCD) suggest that this ICD-10 category is heterogeneous. It most probably includes three separate kinds of disorder with a similar clinical pattern but different pathomechanisms. Psychopathological analyses of 62 OCD patients and preliminary results of their treatment are presented.

Obsessive-compulsive disorder (OCD) is at present a focus of particular attention due to proposals of neurosurgical treatment. This is one of reasons why there is a need for a verification of views on OCD specificity and its underlying pathomechanisms, as well as indications to treatment.

In the ICD-10 classification the term “obsessive-compulsive or anankastic neurosis” was replaced by the “obsessive – compulsive disorder” categorized among neurotic disorders (while in the DSM-IV it is contained in the section of anxiety disorders). Its “essential feature is recurrent obsessive thoughts or compulsive acts...” which are “...repeated again and again in a stereotyped form” and “almost invariably distressing” – mostly because of their violent, obscene or senseless content, but also due to their effect impairing various aspects of the individual’s functioning and activity. They are recognised as the individual’s own even though they are involuntary and often repugnant. Experiences and behaviours of obsessive-compulsive quality are associated with a feeling that one is forced to surrender to them, being unable to

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resist – although at the same time there must be at least one thought or act that the individual still attempts to resist, which is one of the main OCD diagnostic criteria [1]. Compulsions are associated with tension and anxiety, manifested sometimes in autonomous symptoms, the feeling of depersonalisation, hypochondriac complaints, etc. [1, 2, 3].

Obsessive thoughts, especially in the early stages of the illness, are strongly correlated with depressive symptoms. The latter are less frequent in chronic than in acute forms of OCD. On the other hand, in affective disorders – above all, in aggravation of recurring depressive episodes – obsessive thoughts and ruminations occur very often. This justifies hypotheses including OCD in the so-called “affective disorders spectrum” [4]. In affective disorders similarly as e.g. in schizophrenia, Gilles de la Tourette’s syndrome, or organic mental disorder, the presence of obsessive thoughts and compulsive acts is regarded as “a part of the condition”.

An alternative explanation of compulsion concurrence with other symptoms is to assume that two separate disorders, especially affective and obsessive-compulsive – can coexist independently from each other [5, 6]. Like the first hypothesis, this promotes pharmacological treatment of OCD patients with successive generations of drugs used in affective disorders.

However, the ICD-10 assumptions and relevant diagnostic criteria raise many doubts. The ICD-10 classification of mental disorders is limited to the description of their observable aspects, i.e. symptoms above all. The descriptions “carry no theoretical implications, and they do not pretend to be comprehensive statements about the current state of knowledge of the disorders” [1, p. 2]. According to ICD-10 authors, “Instead of following the neurotic – psychotic dichotomy, the disorders are now arranged in groups according to major common themes or descriptive likeness, which makes for increased convenience of use” [1, p. 3]. Therefore, it is not a classification of diseases, but rather of “disorders”, and this “not an exact term... is used here to imply the existence of a clinically recognizable set of symptoms or behaviour associated in most cases with distress and with interference with personal functions” [1, p.5].

Such assumptions, seemingly logical and reflecting the awareness that our knowledge of psychopathology is limited, nevertheless are regarded by many clinicians as leading only to diagnostic difficulties and misunderstandings. The more so, that – as pointed out by Sartorius in the Preface to the ICD-10 – the classification criteria for many disorders were a compromise agreement between “all the major traditions and schools of psychiatry represented” (p. VII). Not surprisingly then such criteria work poorly in the clinical practice. Seeking a compromise between divergent opinions of scientists is not the best way to discover the truth. What’s worse, the results of these endeavours in the case of the ICD-10 are exceptionally inconsistent. Descriptive criteria are mixed up with etiopathogenic ones (e.g. the category: “reaction to severe stress and adjustment disorder”); diagnostic categories are not clearly separated from each other, and various ICD-10 revisions present different psychopathological patterns. The “guidelines for diagnosing mental disorders by the primary care staff” [7] are particularly odd, where guidelines for doctors differ from those for the remaining primary care staff members. The reality the latter are expected to perceive has little in common with what is to be seen and classified by psychiatrists, although not only the disorders in question, but also the patients are the same.

It has been emphasized that ICD-10 is not a set of descriptions of nosological units. The main purpose of this classification clearly stated in the outline of its principles is to facilitate general medical statistics (and not to systematize the existing knowledge about mental disorders). Moreover, an instrumental and exclusively administrative role of the ICD-10 is explicitly stated in various revisions of the classification. Despite all that, they are commonly regarded as obligatory manuals of contemporary psychopathology. The suggestion that the state-of-the-art knowledge is presented in this classification comes not only from the ICD-10 wording referring to “diagnostic guidelines” or “reliable diagnosis”, but also from the users’ expectation that information about advances in medical sciences should be received from the WHO. The misunderstanding results in not very accurate diagnoses and what’s worse – in erroneous therapeutic decisions. This pertains also to the obsessive-compulsive disorder.

Perhaps in response to inadequacy of the ICD-10, attempts are made to develop other ways of clustering and categorizing mental disorders, as reflected in coining of such terms as the “obsessive-compulsive disorder spectrum” [4, 8, 9]. However, these cannot have a major impact on the development of knowledge, since there is an obligation to use the “official” language of psychiatry.

One can speculate that solutions proposed by the contemporary classification systems reflect cognitive nihilism, or in other words a belief that the phenomenon of mental disorders can be neither described nor comprehended. This would explain the preference for creating a system of notions with a sole purpose: just to enable the communication between psychiatrists and other health care staff - instead of attempting to name mental disorders possibly adequately and accurately. However, abandoning such attempts has serious practical consequences – including the already mentioned proposals of neurosurgical treatment of OCD patients who do not respond either to psycho- or pharmacotherapy.

The very term “obsessive-compulsive disorder” suggests there is a single (and homogeneous) disorder classified among neurotic disorders according to the ICD-10. Categorisation of compulsions into this group is apparently appropriate. It seems probable that most patients with obsessions and compulsions predominating in the clinical pattern of their symptoms suffer from causes described as ‘neurotic’ or in other words ‘psychogenic’. What’s more, compulsive thoughts and acts, occasionally even of great intensity, occur also in patients suffering from a variety of other neurotic disorders (according to the ICD-10 criteria). However, it is not so obvious that it is a single disorder and that symptoms of compulsive thoughts and activities are always of neurotic nature.

Clinical observations indicate that very many individuals diagnosed with OCD suffer from other than neurotic conditions [6]. In consequence, a view is quite often expressed that the obsessive-compulsive disorder should be excluded from the category of neurotic disorders; even distinguishing OCD as a separate classification unit is considered unjustified by some authors [10]. Perhaps one could agree with the view that the term “obsessive-compulsive disorder” became an overused category containing a variety of conditions that manifest themselves in a similar way, but are of different nature. This might be one of the main reasons not only of discrepancies between results of epidemiological studies, but also of diagnostic difficulties and unsuccessful therapeutic efforts [11].

Obviously, it is difficult to differentiate between a compulsive thought or behavioural act of neurotic character and seemingly the same symptom of a psychotic process from the schizophrenia spectrum or a mood disorder. Nevertheless, even if we give up analysing quality of the phenomenon called obsessive-compulsive disorder (such as those carried out in the past by e.g. Jaspers), the group of symptoms classified as “obsessive-compulsive” can be hardly regarded as being qualitatively homogeneous.

E.g. ritual behaviours (which are less associated with depression than compulsive thoughts) represent an evidently different quality than compulsive avoidance of certain even non-threatening situations. Some of these symptoms – e.g. “magical obsessions” or compulsive activities in the form of rituals (related with a more severe course of the disorder [12]) – are of a different quality than repetitive checking whether some activity was correctly performed, despite the similar aspect of the stereotypy.

Unfortunately, instead of introspective seeking to establish such particular qualities through defining the differences and similarities between various mental acts, the language in which these experiences are expressed became the focus of attention. As a result, some subtle, but extremely important differences were lost, such as these e.g. between compulsive thoughts of anxious content and compulsively recurring phobic reactions, anxiety states, anxious expectations, hypochondriac and supervalent thoughts, or obsessive thoughts associated with the object of a specific phobia.

Even if coining of such notions as “obsessive phobias” or “compulsive fears” is assumed justifiable, it can be easily seen that they find a common denominator for anxiety associated with objects often encountered in the individual’s environment and the continuous presence of obsessive (e.g. aggressive) anxiety-evoking impulses [3, 6]. Recognition of such specificity may turn out to be useful in the differential diagnosis, as same as keeping in mind that perseverations are usually associated with lesions in the CNS [10].

Perhaps the views formerly present in psychopathology were right that a differentiation should be made between the rumination phenomenon and behaviours of ritual character, as well as between magical thinking and obsessively recurring thoughts with the resulting compulsion to perform activities in a certain order. Consideration was also given to the role of contradiction between confirmation and negation present in the phenomenon of compulsive doubts, or to the experience of “compulsion”. E.g. the compulsion of regarding something as truth was differentiated from that to drive-directed behaviour, and the compulsion in the obsessive-compulsive disorder - from that in eating disorders [13], etc. Observations concerning the difference between compulsive anxious ideas resulting in passivity and compulsive doubts leading to slowing down or desisting from activity, etc. have been lost.

Only some of the phenomena classified into the obsessive-compulsive category seem to be of an evidently “neurotic quality”. As noted earlier, such compulsions and obsessions occur in various neurotic disorders, not only in these diagnosed as obsessive-compulsive disorder. Compulsive thoughts and behaviours belong to the symptomatology present in almost all (over 80%) patients with neurotic disorders [14]. Like other neurotic functional disorders, these symptoms may predominate in the clinical pattern for some time, being replaced in turn by phobic anxiety, generalized anxiety, anxiety-depressive symptoms, or somatoform (especially - hypochondriac)

disorders, etc. This is nothing unusual – all neurotic disorders, especially those with a marked severity and exacerbated course, generally include dysfunctions of the emotional sphere, activity of body organs, and behaviour. Some of the dysfunctions periodically move to the foreground (or are selectively noted by the diagnostician).

According to an over a century old supposition, the patient's symptom in a disguised way informs about the area of his helplessness (as does any "sign" associating the denoted with the denoting symbol in a specific, individual way). If it is true, then not surprisingly changes occurring in the areas of the patient's helplessness lead to a change in the type of his symptoms, including compulsion [12]. On the other hand, its stability due to a specific personality dysfunction results in persistence of the same symptoms over many years [15]. Persistence of the disorder and invariability of the clinical pattern of compulsion cannot be then a clear-cut diagnostic criterion and (contrary to popular beliefs) is not an evidence of an organic underpinning of OCD.

Neurotic disorders are almost always associated with personality dysfunctions. In OCD anankastic personality features have been described particularly often. Nowadays the features of histrionic, dependent, avoidant or schizoid personality are recognised even more often. Moreover, the onset or aggravation of compulsion has been found to be associated with saliency of personality traits related to evaluation, morality or tendency to keeping everything in order – i.e. with a more rigid personality structure. Perhaps such rigidity acts as a counterbalance to drive-directedness, revealing itself in symptoms, mostly obsessive. In OCD patients meticulousness resulting from their fear of committing an error can be frequently seen, as well as a tendency to anxious ruminations, uncertainty, timidity, but also irritability and impulsiveness. Perhaps it is timidity that leads to the tendency noted in some patients - to continually think over their behaviour. This gave rise to the views emphasizing relationship between timid personality lacking self-confidence and the presence of obsessive thoughts and compulsive behaviours. Meticulousness due to rigidity of norms with the resulting sense of dutifulness that can be seen in some patients' attitude to their tasks and their job performance may be a manifestation of overcompensation of such uncertainty [16, 17, 18].

Numerous observations suggest that the type of personality dysfunction is related to the type of compulsion. This can be seen e.g. in patients suffering from depressive symptoms concurrent with obsessive thoughts. Their personality is often predominated by rigidity of norms and requirements leading to a proneness to guilt feelings. Moreover, the existence of such relationships is confirmed by the observation that contrasting compulsions are associated with aggressive features, hypochondriac – with infantile psychological traits, while obsessive doubts and compulsive acts – with psychasthenic traits [10]. The latter can be associated with the individual's specific, typically psychasthenic self-evaluation of his behaviour, i.e. a sense of unfinished activity². Obsessive ideas seem to be more frequent in people with histrionic personality, while obsessive thoughts – in patients with anankastic traits. This may point to a relationship between the type of symptoms and specificity of helplessness resulting from such personality traits. At the same time, it may explain the discrepancies or even contradictions in descriptions of OCD patients' personality traits.

² P. Janet.

Helplessness determined by personality traits may lead to a contrast – described by Kretschmer – between the sexual sphere and ethical requirements. This phenomenon is relatively often seen in OCD patients. Although sexuality has gained cultural legitimisation in the past few decades, this does not mean that circumstances evoking fear of sexual drive manifestation have disappeared. Impulse drive restricted by external bans may be realized despite and even against these bans, if only the individual's personality is equipped with appropriate coping mechanisms. However, nowadays – as same as in the late 19th century, when Freud was creating his theses – cognitive schemata instilled by upbringing are extremely often a source of guilt feelings resulting from sexual needs manifestation, which leads to personality disorders being the cause of helplessness in this sphere of life.

At present conflicts in this sphere are often expressed in compulsive thoughts and activities connected with dirt and cleanliness, obsessive thoughts about the risk of contracting AIDS (frequently being a phobia and a hypochondriac disorder at the same time), as well as in blasphemous sexually-laden thoughts. Their existence explains not only the compulsion of performing acts that reduce guilt feelings but also disturbances in sex life development frequent in patients with OCD symptoms [10].

In blasphemous thought symptoms particularly salient are manifestations of the conflict between impulse drive on the one hand and on the other - bans resulting from rigid norms and restrictions related to sexual life. The conflict is often enhanced by the patient's fear of losing love and respect of important others he is dependent on. The dependency is expressed e.g. by a tendency to an apparent submission to others. This is often associated with a strong ambivalence of love-hate feelings both towards self and others. The ambivalence – often revealed by the psychotherapeutic process – is visible e.g. in the individual's seemingly aloof attitude toward his environment.

The presently considerable prevalence of neurotic thoughts and compulsive activities of blasphemous nature [19] seems to be due not only to the standpoint of the Church in the matters of sexuality, but also to the common acceptance of aggression. Perhaps compulsions that express fear of manifesting aggression (or hide such contents) develop in individuals who for some reason have not submitted to the contemporary cultural legitimisation of brutality and assertiveness. The OCD definition quoted earlier suggests that most patients with this disorder do not accept their own aggression, and the function of their compulsions as indicated by the content is to prevent e.g. their harming themselves or somebody else.

A frequent symptom in OCD patients, also those with blasphemous thoughts, is their compulsive checking various devices, as well as their own activities or thoughts. It resembles the striving for control over an uncontrolled part of "self". A motive of control – or on the contrary, of a lack of control - can be found not only in compulsive checking, but also in repetitive behaviours, the compulsion of counting, tidying up, collecting things in fear of throwing away something potentially important or useful, etc. The need for control over the individual's own feelings is especially salient in ritual activities that are to restore the "lost" sense of having everything under control, and to quieten anxiety at the same time.

Compulsive thoughts and activities focused on tidying up that result from the patient's inability to accept any lack of symmetry or order in the environment often reveal both his helplessness in the situation of "disordered life" and a rigid system of

norms and demands from himself. The onset of such symptoms may be due to disruption of the order hitherto existing in the individual's life, e.g. by circumstances that require a change of occupation or lifestyle, or by some tragic event. OCD symptoms may have then the nature of a reactive stress-related or adjustment disorder. An apparent OCD onset following a period of "depression" seems to be frequently recognized in such cases, while in reality it is a bereavement reaction. This leads to an unjustified associating OCD with affective disorder.

Neurotic compulsive syndromes are often accompanied by depressed mood, but if we assume their relationship with affective disorders, we seem to be misled by appearances. The very presence of compulsions (more than of any other symptoms) is quite sufficient to be a source of anger with oneself and of depressed mood resulting from guilt. Perhaps this is why an exacerbation of compulsive symptoms is so often noted to be associated with depressed mood aggravation. The presence of OCD, and especially the experience of compulsion justifies also panic attacks in an individual fearing that he may lose control over his behaviour, as well as anxiety-laden worrying, as same as that occurring in the generalized anxiety disorder.

Thus, there are no sufficient reasons to suppose that in neurotic compulsive syndromes pharmacological treatment may be more effective than an appropriate psychotherapeutic management [20]. However, it is particularly difficult to undertake such appropriate – and in consequence, efficacious - psychotherapeutic intervention, when helping the patient requires that the therapist should be independent from the commonly held attitudes and values (that he not infrequently shares). Perhaps that is one of factors contributing to the limited effectiveness of psychotherapy in the treatment of OCD.

On the other hand, pharmacotherapy outcome data are not too impressive either. Symptom relief is usually short lasting, in very many OCD patients no improvement is attained, and even a deterioration of the condition may occur. Thus, on the grounds of the existing literature it is difficult to reliably evaluate effectiveness of antidepressants or SSRI in the treatment of OCD.

Perhaps the considerable frequency of therapeutic failure in clinical groups in the study (about 40% of "non-responders") is due to their non-homogeneity. Medication efficacy estimates are probably lowered by the subgroup of patients with OCD of neurotic nature. It is such OCD patients that do not respond to drug treatment or manifest only transitory, placebo-type effects [6, 21, 22]. This supposition seems to be corroborated by numerous case reports as well as data indicating similar (at least apparently) efficacy of drugs with very different mechanisms of action [22].

Pharmacotherapy may be hypothesised to bring good effects in cases of OCD symptoms with underlying biological mechanisms. One of the facts supporting the hypothesis is a higher OCD prevalence rate among first-degree relatives of patients suffering from this disorder [21].

A study by Winid suggests that cerebral dysfunctions are very frequent in the population of OCD patients. In the group he had under observation pathological EEG was found in as many as 50% of the patients, in numerous cases the features of "characteropathy" were present, and in 10% of cases organic brain damage was diagnosed [10]. The latter might include both atrophies and neoplastic processes. Of course, such disturbances should not be identified with dysfunctions in the areas of activity of

particular cerebral structures or their connections (neuronal networks), diagnosed by means of functional neuroimaging techniques [5]. A marked lability of this activity and even its normalization after psychotherapy suggests a functional nature of such changes [6].

Research findings concerning the OCD neurochemical substrate (e.g. the “serotonin theory”) [22], as well as the results of outcome studies on pharmacotherapy aimed at neurotransmission correction (especially with regard to cognitive functions) are not clear. Nevertheless, pharmacotherapy is presently believed to be the most effective form of treatment that can be offered to OCD patients. However, more and more authors seem to share the view that the treatment outcome consists mostly in an improvement of the patients’ quality of life, often unrelated to any symptom relief [23].

It seems that psychotherapy outcomes in OCD patients should be interpreted likewise. Great differences in the frequency of psychotherapy successfulness may also result from non-homogeneity of the groups treated.

Some reports evidence a significant efficacy of psychotherapy – e.g. Winid noted improvement in as many as 76% of the treated patients [10]. Even a short-term group psychotherapy course may lead to a significant decrease in compulsive symptom severity and to strengthening or activation of mature defence mechanisms [24]. Moreover, usefulness of psychotherapy in OCD patients is evidenced by its post-treatment improvement sustaining longer than that after pharmacotherapy [20]. However, in the literature scepticism prevails and most authors believe that psychotherapy is successful in few OCD patients only – the rates of no improvement after psychotherapy are similar to these reported in pharmacotherapy outcome studies.

Most outcome studies evaluating psychotherapy in patients diagnosed with OCD deal with behavioural methods, such as exposure or reaction withholding (ERP). These are expected to eliminate compulsion symptoms (especially rituals) or significantly reduce their severity through decreasing the intensity of the mechanism of “undoing” in patients with predominance of compulsive behaviours [24]. Cognitive techniques turn out to be more successful in obsessive thoughts [20, 25]. The relationship between psychotherapy outcome and the type of compulsion symptoms has been noted also in our research. Namely, psychotherapy turned out to be more effective in the treatment of obsessive thoughts of aggressive and sexual content than for compulsive activities. There is consensus in literature that psychotherapy of OCD requires from the therapist a particularly flexible approach to the patient and an understanding of his or her specific difficulties related to participation in the therapeutic situation, often appearing like resistance [26].

Material and method

A part of a larger study aimed at an analysis of OCD psychopathology is presented in what follows. The fragment below deals with the treatment outcome evaluation.

Participants in the study were 62 patients diagnosed with OCD, 30 men and 32 women aged 19 to 58 years, treated in the years 1990-2003 on the outpatient basis in day hospital. The intense comprehensive therapy included first and foremost group psychotherapy (3 hours daily), supported by individual psychotherapy (at least one session per week) and by pharmacotherapy tailored to the individual needs. Psycho-

therapy was based on an integrative approach with predomination of psychodynamic and cognitive elements, applied accordingly to the patient's diagnosed problems underlying the development of his/her disorder. In the course of the treatment provided 5 or 3 days a week, for the average time of 10 weeks, the patient participated in about 160 psychotherapy sessions.

In the study we evaluated, among other things, outcome of such an intense therapy and the effect of supportive pharmacotherapy (with Clomipramine mostly – in 21% of cases, and SSRI's – in 13% of the whole patient group). The frequency of symptom relief in patients without any signs and symptoms of organic brain disorder was compared with therapy outcome in those in whom pre-treatment organic change tests (Bender, Benton, Graham-Kendall) and other examinations (e.g. EEG) had evidenced changes in the CNS.

To measure therapy outcome, pre-treatment scores in the psychopathological symptomatology of the Symptom check-list "O" (global score) and the Neurotic Disintegration Index on the Cattell's 16 PF test were compared with these obtained on treatment completion. Categories of change (recovery, major improvement, slight improvement, no change, slight deterioration and major deterioration) were defined in accordance with the principles published in 1989 [27]. Significance of inter-group differences was assessed using significance tests for comparison of two structure indicators from the Statistica 5.0 package, assuming the statistical significance level at $p < 0.05$.

Results

Preliminary results of the treatment outcome analysis in terms of symptom change are presented in Table 1.

Category of symptomatic change	A No organic changes	B Organic changes in CNS	Statistical significance of difference between A and B	C Medication in group A	D Medication in group B	Statistical significance of difference between C and D	Medication jointly (A+B)
Major improvement and recovery	26 (68.4%)	9 (37.5%)	$p = 0.056$	11 (42.3%)**	7 (77.7%)	n.s.	18 (51.4%)
Including "recovery"	23 (60.5%)	9 (37.5%)	n.s.	9 (39.1%)	7 (77.7%)	n.s.	16 (50%)
Slight improvement	3 (7.9%)	5 (20.8%)	n.s.	1 (33.3%)	2 (40%)	n.s.	3 (37.5%)
No change	1 (2.6%)	1 (4.2%)	n.s.	0	1 (100%)	n.s.	1 (50%)
Deterioration – slight or major	3 (7.9%)	5 (20.8%)	n.s.	1 (33.3%)	5 (100%)	$p < 0.05$	6 (75%)
Missing data*	5 (13.2%)	4 (16.7%)	n.s.	4 (80%)	0	n.s.	4 (44.4%)
Total	38 (61.3%***)	24 (38.7%)	$p < 0.05$	17 (44.7%)	15 (62.5%)	n.s.	32 (51.5%)

* most often – discontinuation of treatment (drop out)

** when calculating percentages of patients receiving medication, the number of patients in the relevant category of change was assumed as 100%

*** percent of the whole group studied

It follows from Table 1 that patients without organic disorders predominated, but this information is of limited value because of patient selection in the course of qualification to treatment. Even though patients who required special treatment due to e.g. their condition severity were not admitted, nevertheless it turns out that only 60% of the population treated could be diagnosed with undoubtedly neurotic disorders, with no organic underpinning. This subgroup as compared to patients with organic CNS changes turned out also to benefit more from treatment – among the former there is a higher percentage of those with major symptomatic improvement and fewer cases of deterioration (with the difference approaching the statistical significance level of 0.05).

Introduction of pharmacotherapy considerably less often contributed to symptom amelioration in the subgroup without organic changes than in those with CNS changes. At the same time, pharmacotherapy did not prevent deterioration in the latter subgroup (and in the former either). On the other hand, the frequency of a lack of symptomatic change and of dropping out from treatment was similar among patients free from organic disorders and in those in whom such changes had been found.

Personality changes resulting from treatment in day hospitals are shown in Table 2.

Category of personality change	A No organic changes	B Organic changes in CNS	Statistical significance of difference between A and B	C Medication in group A	D Medication in group B	Statistical significance of difference between C and D	Medication jointly (A+B)
Major improvement and recovery	11 (28.9%)	9 (37.5%)	n.s.	4 (36.4%) **	5 (55.5%)	n.s.	9 (45.0%)
Including "recovery"	9 (23.7%)	6 (25.0%)	n.s.	3 (33.3%)	2 (33.3%)	n.s.	5 (33.3%)
Slight improvement	8 (21.1%)	2 (8.3%)	n.s.	3 (37.5%)	2 (100%)	n.s.	5 (50.0%)
No change	3 (7.9%)	3 (12.5%)	n.s.	2 (66.7%)	3 (100%)	n.s.	5 (83.3%)
Deterioration – slight or major	5 (13.2%)	1 (4.2%)	n.s.	1 (20.0%)	1 (100%)	n.s.	2 (33.3%)
Missing data*	11 (28.9%)	9 (37.5%)	n.s.	7 (63.6%)	4 (44.4%)	n.s.	11 (55.0%)
Total	38	24	$p < 0.05$	17 (44.7%)	15 (62.5%)	n.s.	32 (51.6)

* most often – missing data, or discontinuation of treatment (drop out)

** when calculating percentages of patients receiving medication, the number of patients in the relevant category of change was assumed as 100%

Table 2 suggests that the presence or absence of organic changes seems to be of no particular importance for the attainment of favourable personality change. The ratios of patients with such changes are similar irrespective whether they were provided with pharmacotherapy or not, same as the ratios of deterioration and no change cases. Since data on personality change in as many as 20 patients (32%) are missing, possibility of interpretation is rather limited. However, the considerable frequency of favourable personality change in subgroup B (equal to the frequency of symptomatic improvement) seems astonishing. The small effect of pharmacotherapy on the frequency of personality change (in both subgroups) is less surprising.

The results presented above seem to confirm that obsessive-compulsive disorder is diagnosed in the presence of at least two types of symptoms – firstly, of neurotic nature, and secondly, apparently similar ones but due to the central nervous system damage evidenced by the organic changes test scores.

Treatment outcomes in terms of change in the symptom severity and prevalence in these two groups suggest that patients without organic lesions are more susceptible to psychotherapy, while in those with organic brain damage significant effects of pharmacotherapy can be seen. Features of CNS damage as evidenced by organic change test scores were found in about 39% of the treated patients with the diagnosis of OCD. Symptom relief due to pharmacotherapy seems to be more marked in the latter group (major symptomatic improvement could be ascribed to psychotherapy in 2 patients from this group) than in those with OCD of neurotic (purely “psychogenic”) nature. The probability of symptom amelioration was not increased by the associated treatment (i.e. psychotherapy supported with medication).

Discussion and conclusions

The review of psychopathology research literature and of outcome studies in OCD patients as well as our own research findings indicate that at least three variations of this disorder should be distinguished differing in some subtle features of the clinical pattern and in their etiopathogenic determinants. The neurotic OCD with compulsive thoughts and activities in the foreground is only one of them. There are no data that would warrant assumption that its prevalence is higher than that of other OCD variations.

Another kind of OCD includes compulsive syndromes resulting from neurotransmission disturbance (e.g. prior to the onset of a paranoid syndrome). In some of these patients the course of OCD is similar to that of the schizophrenic process [28]. (It is in such circumstances that a similarity of obsessive thoughts to hallucinations can be seen). This form of OCD is often referred to as “pseudoneuroses”. Perhaps compulsive thoughts and activities serve then as a mechanism of coping with a global dysfunction resulting from neurotransmission disturbance.

Literature suggests that it is such patients who respond well to neurotransmission-correcting medication (like do many patients with affective disorders). They constitute most probably about 20% of the described groups of OCD patients treated with psychotropic drugs. It is these patients who provide the crucial argument to justify pharmacological treatment of this disorder.

Compulsive syndromes due to lesions in the CNS (e.g. in the basal ganglia), or to structural cerebral abnormalities [29] that result in secondary brain dysfunctions seem to be still another category. The knowledge about such an impairment of cerebral functions is rather limited yet and the relationship between cerebral dysfunction and the onset of compulsive symptoms is not clear [30, 31, 32]. Particular importance is ascribed to the frontal lobes and to their connections with other regions of the CNS [33, 34]. Research in this area gives rise to interpreting compulsive thoughts and activities as symptoms of “corticostriatal dysfunction” [25, 33]; in accordance with this concept the presence of such symptoms is regarded as evidencing an enhanced activity in the limbic-frontal-basal areas of the brain [30, 31, 35, 36]. One can hardly

resist a reflection that descriptions of dysfunction in the communication between cortical areas and the limbic system structures seem to be analogous to these of conflicts between the sphere of drives and the system of norms, or between the sphere of emotions and “instincts” on the one hand and “intellect” on the other [37].

Nevertheless, also in such disorders the primary treatment seems to be pharmacotherapy, and not psychotherapy. Of course, if the dysfunction of the CNS is caused by atrophic or neoplastic processes, marked and permanent changes can be hardly expected to arise that way. In the case of an atrophic process no corrective effects can be expected either from any neurosurgical intervention aimed at elimination of rigid, pathological pathways and at restoration of the neuronal communication flexibility [34].

Since numerous studies on neurosurgical intervention in mental disorders have brought equivocal results, they do not allow for clear-cut recommendations to be made to this type of treatment in obsessive-compulsive disorder. Limited efficacy and many side effects of such interventions warrant proceeding with special caution [31, 33, 38, 39, 40], particularly in diagnosing the type of disorder on the grounds of predominating symptoms, without regard to a variety of factors underpinning the development of compulsions.

In view of these comments it seems obvious that psychosurgical management may be at present taken into account only in very special OCD cases. These special cases include undoubtedly a neoplastic process or status post cranio-cerebral trauma, while any remaining ones require careful consideration of the determinants and nature of symptoms in individual cases.

Neither ineffectiveness of psychotherapeutic and pharmacological treatment methods nor the long-term character of OCD symptoms seem to be sufficient arguments to warrant neurosurgical intervention, since treatment failure may be (and frequently is) due to diagnostic errors. As regards psychotherapy, failure most probably results also from erroneous treatment directions being, as it has been already mentioned, a source of particular difficulty to therapists. These may belong among factors responsible for the relatively small efficacy of treatment (especially regarding personality change) in day hospitals, which will be investigated in further studies. Nevertheless, preliminary results suggest that either psychotherapy or pharmacotherapy may be selectively recommended in particular cases, depending on whether the features of neurotransmission disturbance and brain damage are found in the clinical picture.

The review of the literature and the authors' preliminary research findings indicate non-homogeneity of the group of patients diagnosed with obsessive-compulsive disorder (OCD). The group is differentiated, among other things, by specific features of their symptoms. Selection of experimental patient groups diagnosed using the ICD-10 criteria is unjustified, and this may explain discrepant results of psycho- and pharmacotherapy outcome studies. Psychotherapy is most probably more effective than pharmacotherapy in the treatment of OCD of neurotic nature, while pharmacotherapy – in cases of compulsions due to organic changes in the CNS. The latter include either neurotransmission disturbances (where pharmacotherapy is recommended), or anatomical abnormalities, e.g. neoplastic process (it is in such cases only that neurosurgical intervention may be considered).

References

1. *The ICD-10 classification of mental and behavioural disorders. Clinical descriptions and diagnostic guidelines*. Kraków-Warszawa: IPiN, UMW "Vesalius"; 2000.
2. Aleksandrowicz JW. *Neurotic, personality, and behavioural disorders in adults (according to the ICD-10)*. Collegium Medicum UJ, Cracow 1997.
3. Kępiński A. *Obsessive-compulsive neurosis* In: Kępiński A. *Psychopathology of neuroses*. Warszawa: PZWL; 1973, 58–76.
4. McElroy SL, Phillips KA, Keck PE. *Obsessive compulsive spectrum disorder*. *J. Clin. Psychiatr.* 1994, 55: 33–53.
5. Lopes AC, Soares KVS, Del Porto JA. *Surgery for obsessive-compulsive disorder*. [Protocol] The Cochrane Library, 2003, Vol 1.
6. Rabe-Jabłońska J. *Obsessive-compulsive disorder*. *Przewodnik Lekarza*. 2003; 3: 121–126.
7. ICD-10. *Zaburzenia psychiczne w praktyce podstawowej opieki zdrowotnej*. Warszawa: IPiN; 1999.
8. Bienvenu OJ, Samuels JF, Riddle MA, Hoehn-Saric R, Liang KY, Cullen BA, Grados MA, Nestadt G. *The relationship of obsessive-compulsive disorder to possible spectrum disorders: results from a family study*. *Biological Psychiatry* 2000, 48 (4): 287–293.
9. Pacan P, Kantorska-Janiec M, Kiejna A. *Trichotillomania*. *Psychiatr. Pol.* 1998, 32 (6): 799–805.
10. Winid B. *Zespół natręctw* (Thesis for the title of doktor habilitowany) Kraków, 1970.
11. Stein MB, Forde DR, Anderson G, Walker JR. *Obsessive-compulsive disorder in the community: an epidemiological survey with clinical reappraisal*. *Am. J. Psychiatry* 1997, 154 (8): 1120–26.
12. Skoog G, Skoog I. *A 40-year follow-up of patients with obsessive-compulsive disorder*. *Arch. Gen. Psychiatry* 1999, 56 (2): 121–27.
13. Bulik CM, Tozzi F, Anderson C, Mazzeo SE, Aggen S, Sullivan PF. *The relation between eating disorders and components of perfectionism*. *Am. J. Psychiatry* 2003, 160 (2): 366–8.
14. Aleksandrowicz JW. *Częstość występowania objawów nerwicowych*. *Psychiatr. Pol.* 2000, 34: 5–20.
15. Mataix-Cols D, Rauch SL, Baer L, Eisen JL, Shera DM, Goodman WK, Rasmussen SA, Jenike MA. *Symptom stability in adult obsessive-compulsive disorder: data from a naturalistic two-year follow-up study*. *Am. J. Psychiatry* 2002, 159 (2): 263–8.
16. Bogetto F, Barzegà G, Bellino S, Maina G, Ravizza L. *Obsessive-compulsive disorder and personality dimension: a study report*. *Eur. J. Psychiatry* 1997, 11 (3): 156–161.
17. Baer L, Jenike MA, Ricciardi JN et al. *Standardized assessment of personality disorders in OCD*. *Arch. Gen. Psychiatry* 1990, 47 (8).
18. Black DW, Yates WR, Noyes R et al. *DSM-III personality disorder in obsessive compulsive study volunteers: a controlled study*. *J. Personal. Dis.* 1989, (3): 58–62.
19. Aleksandrowicz JW. *Zmiany częstości występowania objawów nerwicowych*. *Psychiatr. Pol.* 2001, 35 (3): 351–377.
20. Bryńska A, Wolańczyk T. *Metody psychoterapeutyczne stosowane w leczeniu zaburzenia obsesyjno-kompulsyjnego u dzieci i młodzieży*. *Psychiatr. Pol.* 1998, 32 (6): 723–738.
21. Erzegovesi S, Cavallini MC, Cavedini P, Diaferia G, Locatelli M, Bellodi L. *Clinical predictors of drug response in obsessive-compulsive disorder*. *J. Clin. Psychopharmacol.* 2001, 21 (5): 488–92.
22. Ackerman DL, Greenland S. *Multivariate meta-analysis of controlled drug studies for obsessive-compulsive disorder*. *J. Clin. Psychopharmacol.* 2002, 22 (3): 309–17.
23. Tenney NH, Denys DA, van Megen HJ, Glas G, Westenberg HG. *Effect of a pharmacological intervention on quality of life in patients with obsessive-compulsive disorder*. *Int. Clin. Psychopharmacol.* 2003, 18 (1): 29–33.

24. Albuher R, Abelson J, Nesse RM. *Defense mechanism changes in successfully treated patients with obsessive-compulsive disorder*. Am. J. Psychiatry 1998, 155 (4): 58–559.
25. Silva P de, Rachman S. *Nerwica natręctw*. Warsaw: PWN; 1994.
26. Winid B. *Uwagi o postępowaniu psychoterapeutycznym w nerwicy natręctw*. Neurologia, Neurochirurgia i Psychiatria Polska Rok XII, 12, (6): 911–916.
27. Aleksandrowicz JW, Pawelec B, Sikora G. *Model oceny efektów terapii nerwic - wskaźniki i kryteria zmian*. Psychoterapia 1989, 66 (1): 53–61.
28. Price LH, Rasmussen SA, Eisen JL. *The natural history of obsessive-compulsive disorder*. Arch. Gen. Psychiatry 1999, 56 (2): 131–32.
29. Berthier ML, Kulisevsky JJ, Gironell A, Lopez OL. *Obsessive-compulsive disorder and traumatic brain injury: behavioral, cognitive, and neuroimaging findings*. Neuropsychiatry Neuropsychol. Behav. Neurol. 2001, 14 (1): 23–31.
30. Baxter LR, Schwartz JM, Bergman KS, et al. *Caudate glucose metabolic rate changes with both drug and behavior therapy for obsessive-compulsive disorder*. Arch. Gen. Psychiatry 1992, (49): 681–9.
31. Biver F. *Changes in metabolism of cerebral glucose after stereotactic leukotomy for refractory obsessive-compulsive disorder: a case report*. J. Neurol. Neurosurg. Psychiatry 1995, 58 (4): 502–505.
32. McGuire PK, Bench CJ, Frith CD, Marks IM, Frackowiak RSJ, Dolan RJ. *Functional anatomy of obsessive-compulsive phenomena*. Brit. J. Psychiatry 1994, 164: 459–68.
33. Irle E, Exner C, Thielen K, Weniger G, Ruther E. *Obsessive-compulsive disorder and ventromedial frontal lesions: clinical and neuropsychological findings*. Am. J. Psychiatry 1998, 155 (2): 255–263.
34. Swayze VW. *Frontal leukotomy and related psychosurgical procedures in the era before antipsychotics (1935-1954): a historical overview*. Am. J. Psychiatry 1995, 152 (4): 505–515.
35. Baxter LR, Schwartz JM, Mazziotta JC, et al. *Cerebral glucose metabolic rates in non-depressed patients with obsessive-compulsive disorder*. Am. J. Psychiatry 1988, 145: 1560–63.
36. Machlin S, Harris G, Pearson G, et al. *Elevated medial-frontal cerebral blood flow in obsessive-compulsive patients: SPECT study*. Am. J. Psychiatry 1991, 148: 1240–2.
37. Schulz P, Steimer T. *Psychotropic medication, psychiatric disorders, and higher brain functions*. Dialogues Clin. Neurosci. 2000, 2 (3): 177–182.
38. Snaith RP. *Surgery for mental illness has been proved effective*. BMJ. 1997, 314: 7073–75.
39. Baer L, Rauch S, Ballantine T, Martuza R, Cosgrove R, Cassem E, Giriunas I, Manzo P, Dimino C, Jenike M. *Cingulotomy for intractable obsessive-compulsive disorder: pro-spective long-term follow-up of 18 patients*. Arch. Gen. Psychiatry 1995, 52 (5): 348–92.
40. Krzyżowski J. *Natręctwa, obsesje i kompulsje*. Warszawa; 2003.

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