

Journal Abstracts

A review of the Depressive Personality

By: Phillips, K.A. et al. *Am. J. Psychiat.*, **147**: 7, 380-837, 1990.

A depressive type of personality disorder has been described by both German phenomenologists and psychoanalysts and has been included in standard international nosologic systems (ICD-9), it has never been recognized in DSM.

This literature review, identifies and explores the issues relevant to the possible inclusion of such a category - on axis II personality disorder linked to axis I depressive disorder - in the upcoming DSM-IV.

Comment :

The inclusion of new diagnostic items add to the diagnostic armamentarium of psychiatrists. Though it carries the risk of adding more confusion and imprecision.

The depressive personality disorder was said to be responsive to antidepressant medications. Is it a hopeful step for treatment of personality disorders or this diagnosis needs a careful re-evaluation?

M. GHANEM

Epidemiology of obsessive-compulsive disorder during childhood and adolescence

By M. Flament, *Encephale*, **XVI**: 311-317, 1990.

Although obsessive - compulsive disorders are rarely found in young subjects seen in infantile psychiatry, anamnestic studies have shown that more than one third of obsessive patients seen at adult age agree that the onset of their disorder dates back to when they were under 15 years old.

Over 5,500 high school teenagers were asked to fill in a questionnaire dealing with different aspects of mental pathology and 356 of them were selected, on the basis of their answers, to be included in further clinical examination. The current prevalence rate of this disorder was found to be $1 \pm 0.5\%$ of the general adolescent population, and the life time prevalence rate was $1.9 \pm 0.7\%$.

Adolescents showed the same clinical characteristics as adults except for the absence of male predominance. 75% of obsessive patients also presented other associated disorders, most often major depression, anxiety and/ or bulimia, but only 20% of them had already consulted mental health professionals.

This study shows that obsessive compulsive disorder during adolescence is more frequent than expected, but often remains undiagnosed and untreated.

Comment :

It seems that the discovery of recent efficient treatment for obsessive disorder, notably fluvoxamine and fluoxetine (drugs with specific predilection for serotonin), has led to an increased interest in the disorder and a tendency for clinicians to over diagnose it.

M. GHANEM

The obsessive-compulsive syndrome: reflection of left lateralized frontal-caudate dysregulation?

By P. Flor-Henry;
L'Encephale, **XVI**: 325-9, 1990.

Neuropsychological studies of the obsessional syndrome reveal bilateral frontal dysfunction. Early onset is characteristic of males, who have a more chronic course than females, and who are more often non-dextral. Hypermetabolic activ-

ity of the frontal and caudate, bilaterally, is found in most PET investigations. Given the frequency with which basal ganglia disease leads to obsessive compulsive phenomena (CF Sydenham's chorea, Gilles de la Tourette) and frontal lobe inertia, a perturbation of frontal caudate regulatory motor and ideational sub-systems in the obsessional syndrome appears probable. Bilateral caudate atrophy in CT scan has been reported. Further, EP investigations, both somatosensory and auditory, implicate the left hemisphere in obsessions; both somatosensory and auditory, implicate the left hemisphere in obsessions; together with reduced P 300 latencies and during imaginal flooding there is increased left frontal hemisphere flow (*r* CBF). Single case studies document the relationship of obsessions to left frontal-left caudate unilateral pathology. It is suggested that lateralized dysregulation of the left fronto-caudate network is the major cerebral determinant of obsessive compulsive states.

Comment:

Attempts to localise psychiatric disorders to a lobe, hemisphere or even to a dysregulation of function of cybernetic loops are still going on. Can this approach be helpful in the unhiding of the mystery? The answer will be left to the future.

M. GHANEM

Acute Dystonia Induced by Clomipramine Therapy

By RN. Chithiramohan, C. G. Ballard and L. G. Measey, *Irish Journal of Psychological Medicine*, 7: 141-142, 1990.

The authors presented a case showing inducement of acute dystonia by clomipramine therapy and discussed the possible explanations.

The case was a 36 year old married school teacher, with symptoms of depression, of two months duration, fulfilling

the DSM-III R criteria of Major affective disorder, she was given 75 mg clomipramine and presented 5 days later with symptoms of an acute dystonia and an oculogyric crisis, torticollis and lead-pipe rigidity. Clomipramine was discontinued and she was given 10 mg procyclidine I.V. which improved torticollis and oculogyric crisis, but rigidity persisted. On the 6th day after stoppage of clomipramine, choreo-athetoid movements were observed in Rt hand & leg and persisted for 5 days. Most of her investigation including NMR showed no abnormality. She was given Dothiepin 75Y nocte, then increased to 75Y bd with good improvement. After 6 months of follow-up, there was no recurrence of extra pyramidal or depressive symptoms.

In the literature of acute dystonia induced by anti-depressant medication, only 8 cases have been reported.

Possible explanations for the phenomena, includes.

1) Concurrent administration of neuroleptics (positive in two of the 8 cases reported).

(2) A recent course of ECT, which does have short term effects on dopaminergic system (reported in one case).

(3) Toxicity is a likely precipitating factor.

(4) It might be possible in the case reported with "Nomifensine" that it is related to the Da re-uptake defects.

(5) Increased serotonergic activity of Raphe nuclei projections on nigral cells, with resultant deleterious effects on Da turnover, might be implicated.

(explains how extra pyramidal effects could occur without dystonia).

(6) Choreo-athetoid movement might be related to concurrent problem with GABAergic system.

Comment :

The rarity of case reports and presence of other variables limited consideration of these unknown side effects as a phenomenon. However, they pave the way for further research studying the mechanism

and sites of action of antidepressants, which seem more complex than what has been thought before

T. ASSAAD

Anti-depressants in 'Depressed' Schizophrenic Inpatients.

By Mark S. Kramer, Wolfgang H. Vogel, Celeste Di Johnson; Donna Ann Dewey; Patricia Sheves; Steven Cavichia; Patrick Little; Robert Schmidt; Ivakimes.

Arch. J. Psychiat., Vol. 46, October 1989.

It is well known that a high incidence of depressive symptoms and syndromes occurs throughout the course of schizophrenia.

Depressive syndromes in schizophrenic patients have been described in various contexts:

1) As features of the prodroma of acute psychotic episodes, that continue to manifest throughout the acute and active psychoses in about 50% of the patients.

(2) As features of the "post-psychotic" phase, where the depressive syndrome appears in approximately 25% of patients after or during the resolution of the acute psychotic episode.

(3) In some instances, the depressive syndrome is thought to be induced by neuroleptic treatment.

In this study, Fifty-eight actively psychotic inpatients, who initially met criteria for long-standing schizophrenia and subsequently met criteria for a current episode of schizo-affective disorder (mainly schizophrenic) with a depressive syndrome, and who scored at least 30 (mean = 55, SEM = 1.6) on the Brief Psychiatric Rating Scale, and 17 (mean = 23, SEM = 0.7) on the Hamilton Rating Scale for depression, were treated for 5 weeks with haloperidol and benzodiazepines.

Those patients who consistently scored greater than 17 on the Hamilton Rating Scale for depression were randomly as-

signed for following double-blind treatment groups for 4 weeks: adjunctive amitriptyline, desipramine or placebo.

Adjunctive amitriptyline or desipramine showed no significant therapeutic advantage, when compared with haloperidol and placebo, on the Brief Psychiatric Rating scale or the Hamilton Rating Scale for Depression.

After 4 weeks of combined therapy, patients receiving adjunctive antidepressants, as compared with those receiving adjunctive placebo, showed greater score on the Brief Psychiatric Rating Scale, hallucinatory behaviour item and on the thinking disturbance factor, than patients receiving placebo.

These results suggest that adjunctive anti-depressants are not indicated for the treatment of depressive symptoms in actively psychotic schizophrenic inpatients, as it might retard the rate of resolution of psychosis.

T. ASSAAD

Serum Immunoglobulins in tardive dyskinesia Implications for pathogenesis of the syndrome

By Hanafy Youssef.

British Journal of psychological medicine 1990; 7: 145-146

In searching for the factors associated with vulnerability to tardive dyskinesia, it has been proposed that organic brain dysfunction may predispose to the emergence of the syndrome.

The course of tardive dyskinesia is characterized by repeated reactivation and remission. This course, while not pathognomonic of immune disorder, resembles the course of other disorders of immunologic processes such as "multiple sclerosis", "systemic lupus erythematosus" "nephritis".

The relative preparation of different immunoglobulins is of importance in the diagnosis of certain diseases of immune

pathology. This study assesses the immunoglobulin finding in schizophrenic patients with and without tardive dyskinesia.

Levels of immunoglobulins were measured in 32 patients having schizophrenia with tardive dyskinesia and 34 schizophrenic patients without tardive dyskinesia. The duration of neuroleptic treatment in years was similar in the two groups. The serum immunoglobulins concentration (mg%) were found to be significantly different in the two groups; IGA and IGM levels were higher in patients with tardive dyskinesia. Possible explanations:-

The level of neuroleptic agents given to chronic schizophrenics may be sufficient to cause cell destruction.

Phenothiazine and butyrophenone drugs alter immune response, which suggests involvement of dopamine receptors as non-specific immune modulators. Drugs can act as haptens, bind to tissue protein, and are capable of inducing an immune response.

However, it seems that the drug level of neuroleptic is not essential for the emergence of T.D in schizophrenic patients.

Age - specific - changes occur in the immune system in man: both the cellular immunity, and delayed type hypersensitivity reactions are impaired and there is an increase in serum immunoglobulin and auto-antibodies.

The differences in immunoglobulins in the two aging groups in this study may indicate a physiological change which was accelerated at the brain level in tardive dyskinesia group, and this may lead to the chronic deteriorating course of negative symptoms and intellectual impairment.

Comment :

It is unclear whether the increase in immunoglobulin is due to T.D., the schizophrenic illness itself or, the aging process, the drugs used or all these factors combined together.

The limited material and the only once estimation of IG levels limit the interpretation of the results.

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