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AMANTADINE AND DEPRESSION

T.J. Huber[1] *, D.E. Dietrich[1], H.M. Emrich[1]. [1]*Department of Clinical Psychiatry, Medical School Hannover, Hannover, Germany*

Since its introduction for the treatment and prophylaxis of influenza infection in 1963, amantadine has also been proved effective in a variety of neuropsychiatric disorders: it not only alleviates rigor, tremor and bradykinesia in drug-induced Parikinsonism and Parkinson's disease, but also improves vigilance in the latter disorder as well as in patients having suffered traumatic head injury and dementia processes. It improves fatigability in multiple sclerosis and has been used successfully in cocaine withdrawal.

Effects of amantadine on symptoms of affective disorders have been demonstrated in several trails administering it for varying purposes. Additionally, animal studies as well as clinical trials in men have shown a direct antidepressant activity of amantadine, which is presented here.

Amantadine appears to work through several pharmacological mechanisms, of which none could be identified as the one chief mode of action. It is a dopaminergic, noradrenergic and serotonergic substance, blocks monoaminoxidase A and NMDA-receptors and seems to raise beta-endorphin/beta-lipotropin levels. Amantadine has also recently been shown to have antiviral effects on Borna disease virus, which is suspected to possibly cause depressive disorders. All of these actions could constitute an antidepressant property, and it is suggested that amantadine might work as an antidepressant not through one, but through several mechanisms thought to be related to antidepressant activity.

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AMANTADINE TO AUGMENT ANTIDEPRESSIVE MEDICATION IN BORNA DISEASE VIRUS (BDV) INFECTED PATIENTS WITH AFFECTIVE DISORDERS

D.E. Dietrich[1] *, L. Bode[2], C.W. Spannhuth[1], T.J. Huber[1], H. Ludwig[3], H.M. Emrich[1]. [1]*Dept. of Clinical Psychiatry, Medical School Hannover;* [2]*Dept. of Virology, Robert Koch-Institute, Berlin;* [3]*Institute of Virology, Free University of Berlin, Berlin, Germany*

Amantadine is known to have multiple pharmacological effects on the CNS, e.g., influence on the dopamine-, noradrenaline-, serotonin-system and NMDA-receptors. In addition, immune modulating as well as antiviral actions have been described. Amantadine was therefore mainly used for the therapy and prophylaxis of influenza infection, Parkinson-syndromes and to improve vigilance in disordered patients. The use of amantadine in the therapy of depressive disorders was also described, but high-lighted by the discovery of its antiviral effect on Borna Disease Virus (BDV), which may be causally related to subtypes of affective disorders.

BDV is well known as pathogenic in certain animal species. Symptoms in infected animals range from inapparent or subclinical manifestations to fatal neurologic disorders with neurobehavioral and/or emotional disturbances.

Psychiatric diseases were considered to be potentially associated with human BDV infections, since BDV-antibodies were detected in humans, and, recently, human strains of BDV were isolated from patients with recurrent mood disorders. However, the knowledge of the influence of BDV on the development and progress, e.g., of depressive episodes is still limited.

This report focuses on the use of amantadine to augment antidepressive therapy in BDV-infected patients (n = 20) with depressive episodes in bipolar and major depressive disorders with a special emphasis to clinical experiences.

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BIPOLAR II VERSUS UNIPOLAR ATYPICAL DEPRESSION IN PRIVATE PRACTICE OUTPATIENTS

F. Benazzi. *Department of Psychiatry, Public Hospital Morgagni 47100 Forlì, Italy*

Objective: Differences between bipolar and unipolar atypical depression are not well studied. Aim of this study was to compare bipolar II with unipolar atypical depression in private practice outpatients

Method: Consecutive atypical depressed outpatients were interviewed with the Comprehensive Assessment of Symptoms and History, the Montgomery Asberg Depression Rating Scale, and the Global Assessment of Functioning scale.

Results: No significant differences were found between bipolar II (n = 38) and unipolar (n = 23) atypical depression on age at baseline/onset, gender, duration of illness, severity, psychosis, comorbidity, chronicity, and recurrences. Findings disagree with some reported differences between bipolar disorders and unipolar disorder (age at onset, female/male ratio, recurrences, comorbidity). Results support the view, based mainly on the better response to MAOI than to TCA, that atypical depression may be different from other forms of depression.

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BIPOLAR VERSUS UNIPOLAR PSYCHOTIC DEPRESSION IN PRIVATE PRACTICE OUTPATIENTS

F. Benazzi. *Department of Psychiatry, Public Hospital Morgagni 47100 Forlì, Italy*

Objective: To compare bipolar with unipolar psychotic depression in private practice outpatients.

Method: 48 consecutive psychotic depressed outpatients were interviewed with the Comprehensive Assessment of Symptoms and History, the Montgomery Asberg Depression Rating Scale (MADRS), the Global Assessment of Functioning (GAF) scale, and the Brief Psychiatric Rating Scale (BPRS).

Results: 43.7% had bipolar I/II disorder, 56.2% had major depressive disorder. Of the variables investigated (age, duration of illness, severity, recurrences, atypical features, chronicity, gender, comorbidity, hallucinations, delusions) only depression severity, measured by MADRS and BPRS, was significantly greater in bipolar than in unipolar psychotic depression.

Conclusions: Bipolar psychotic depression was similar to unipolar psychotic depression on variables reported to distinguish bipolar from major depressive disorder (age, gender, recurrences, atypical features, comorbidity).

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CLINICAL EXPERIENCE WITH GABAPENTIN IN PATIENTS WITH BIPOLAR DISORDER: RESULTS OF AN OPEN LABEL STUDY

P.L. Cabras*, J. Hardoy, C. Hardoy. *Department of Neurological and Psychiatric Sciences, Unit of Psychiatry, University of Florence, Italy*

Objectives: This study was carried out to evaluate the efficacy, tolerability and safety of gabapentin (GBP) used as adjunctive treatment in patients with bipolar disorder with acute symptoms and used as unique mood-stabilizer as prophylactic treatment.

Methods: Fifty patients fulfilling DSM IV diagnostic criteria for bipolar disorder underwent a 24-week, open trial treatment with

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