The aim of this study was to examine the long-term efficacy and safety of a monotherapy with quetiapine or sodium valproate (VPA) in patients with rapid cycling bipolar disorder.

This open-label trial was conducted at three German centers. A sample of 38 remitted or partly remitted bipolar patients with rapid cycling (quetiapine n = 22; VPA n = 16) were treated with quetiapine or VPA (flexible-dose design) up to 12 months. Analyses were based on the ITT-LOCF principle.

41 % of the patients with quetiapine and 50 % with VPA completed the trial. According to the Clinical Global Impression Scale responder rates tended to be higher for quetiapine than for VPA: i.e. 43 % vs. 25 % (depression), 48 % vs. 36 % (mania), and 43 % vs. 19 % (improvement in both mania and depression). There were no differences found between the treatment groups evaluating the HRSD, MADRS and YMRS. In contrast, Life Chart Method data showed that patients being treated with quetiapine had significantly less depressive days than patients on VPA whilst they did not differ in the number of days with manic symptoms. The incidence of adverse events, especially of orthostatic dysregulation and sedation was higher in the quetiapine group.

Quetiapine may be more effective than VPA regarding depressive symptoms and as effective as VPA in the treatment of manic symptoms in the long-term treatment of rapid cycling bipolar disorder. The side effect profile of quetiapine tends to be less favorable than the one of VPA.

P0328

Hematologic toxicity with Sodium Valproate in bipolar affective disorder and comorbid Behcet's syndrome

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Objectives: Behçet's Disease(BD) is a primary vasculitis with wide variety of symptoms, in which psychiatric disorders as Bipolar Affective Disorder(BPD) are seen. In such cases; as in all comorbidities, the importance of preferring appropriate treatment is emphasized.

We present a case of hematologic toxicity associated with a valproate level of 116 mg/l in of BPD and BD comorbidity.

Case: The 38 years old male patient, who was diagnosed as BPD 10 years ago and BD 3 years ago, was hospitalized for mixed episode with psychotic features. His routine laboratory parameters were in normal ranges. Olanzapine 10mg/d and valproate 500mg/d were started and Valprote was titrated up to 1000 mg/d. At the 8th day of medication he complained of fatigue and somnolence. By the following 6 days a decrease in the hematologic values as: haemoglobin:15,0/11,4g/dl, hematocrit:%42/32, platelets:334000/202000 microg/ml was noticed. He had no active symptoms of BD. The liver enzymes were moderately high and because of the hepatoxicity with valproate was reported in previous studies, Valprote was stopped and Olanzapine was continued. The hematological and biochemical parameters normalized in two weeks.

The decrease of hematological parameters could not be explained by BD itself or any other organic pathology and normalized after cessation of valproate. Thus a hematotoxicty associated by valproate was considered.

Conclusions: Although the effects of the comorbidity of BPD-BD to hemototoxicity are not clear exactly, this case underlies the importance of careful monitoring the Valprote levels and associated parameters in BPD particularly if comorbidity is present.

P0329

Carbamazepine induced bicytopenia, three years later- case report

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Carbamazepine was the first anticonvulsant widely used in psychiatry, first for the treatment of bipolar disorder and later for other psychotic disorder (adjunctive). Some of the side effects, which usually occur at the beginning of the therapy with this medicament, are hematological changes which consider transitory leucopenia (10%), persistent leucopenia (2%) and rarely thrombocytopenia. Just these side effects were registered in patient described in this paper.

26 year old men diagnosed as schizophrenia (according to ICD X criteria) was administrated carbamazepine as adjuvant therapy because difficulties in behavior control, seven years ago in dosage of 400mg daily. After four years of therapy, on routine complete blood count (CBC) checking decreased number of white blood cells (2.9 white cells/mm3) and platelets (110000/mm3) were registered. Carbamazepine was excluded from therapy immediately, and from that time on patient is under a regular control of hematologist who diagnosed Bicytopenia (Leucopenia and Thrombocytopenia) and prescribed multivitamin therapy. After three years of these changes, CBC normalized. This entire time patient did not have any symptom which could consider immunological trouble or problem with blood coagulation.

P0330

Psychophysiological and neurobehavioral effects of endogenous Lithium

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Backgrounds and Aims: Lithium occurs naturally in food and water. While low environmental concentrations in drinking water are associated with mental illnesses and behavioral offences, at therapeutic dosages it is used to treat psychiatric disorders, partly by facilitating serotonergic (5-HT) neurotransmission. However, as little is known about the physiological role of nutritional lithium for neurobiological functioning and emotional processing in the general population, endogenous lithium concentrations were hypothesized to be associated with measurable effects on emotional liability and the loudness dependence (LD) that is proposed one of the most valid indicators of 5-HT neurotransmission.

Methods: Auditory evoked potentials (AEP) of healthy volunteers with either high or low lithium serum concentrations were recorded using multi-channel EEG. Emotional liability was assessed using the Brief Symptom Inventory (BSI).

Results: Serum lithium concentrations varied widely, low levels correlating with symptoms of Somatization. While there were no significant correlations between LD and lithium concentrations, LD correlated positively with Paranoid Ideation, and in the high-lithium group inversely with further aspects of emotional liability (Depression, Psychological Distress).

Conclusions: Effects of low levels of endogenous lithium are associated with an increase in emotional liability, and high levels

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