Results and Conclusion: We compare our one-year experience with treatment of outpatients with anxiety disorders. After a short time therapy the effect was significant – the improvement of the of the clinical symptoms and successful come back to every day life activities. Patients, that have finished their treatment by alliance, were without benzodiazepins and treatment without relevant psychotherapy. Combination of psychopharmacotherapy and psychotherapy results in decreasing of clinical symptoms of anxiety disorders.

P46.10

Synthesis of endogenous ethanol during treatment of alcoholic dependence

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Decrease of basic level of blood endogenous ethanol is observed at patients suffering alcoholic dependence especially in the period of abstinence. The level of alcoholic motivation is in the reverse dependence with the content of blood endogenous ethanol. Precursor of endogenous ethanol is pyruvic acid from which acetylated aldehyde is formed and quickly reduced to endogenous alcohol. Sufficient concentrations of lactate inhibit conversion of sodium piruvate into lactate and direct the reaction to formation of endogenous ethanol. The patients were treated per oral with sodium pyruvate in combination with phosphotiamine, magnesium sulfate and potassium lactate. This treatment normalized metabolic processes disturbed as a result of alcohol abuse and restored synthesis of endogenous ethanol. The use of dichotomous emotiogenous test revealed more marked influence of sodim pyruvate on posterior parts of both celebral hemispheres and predominantly correcting influence on the structures of right cerebral hemisphere.

P46.11

Creatine kinase levels during treatment with atypical antipsychotic agents

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Marked elevations of serum creatine kinase (SCK) have been associated with treatment with both typical and atypical neuroleptic agents.

Objectives. The purpose of this paper is to report preliminary data concerning the frequency of SCK elevation in patients treated with atypical neuroleptics in comparison with a group receiving typical neuroleptic drugs.

Methods. Before the initiation of neuroleptic therapy (clozapine [n=11], olanzapine [n=9] and risperidone [n=13], haloperidol [n=6] and perphenazine [n=8]) CK levels in peripheral blood was determined. Blood sample for CK determination was also collected at weeks: +1, +2, +3, +4, +8, +12. Treatment compliance was periodically assessed using the reports of nursing staff.

Results: About 4% (n=2, one with clozapine and one with perphenazine) of patients was found with elevated SCK levels above upper normal limits (290 IU/L and 345 IU/L).

Conclusions: The rate of frequency of SCK elevation in patients treated with neuroleptics that was found in our sample is compatible with previous reports (2–10%). In the present paper, the role of atypical as well as typical antipsychotic agents in producing SCK elevations, its' magnitude and pathophysiological significance and the ways of its' further study will be discussed.

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P46.12

Alteration of hormone status during the treatment of risperidon

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Objective: To study of hormone status peculiarities of the patient's who under risperidon therapy.

Method: Enzyme immunoassay, clinical-psychopathological.

Results: There was examined 16 patients (8 men and 9 women) with diagnosis of paranoid schizophrenia that had anti-psychotic treatment of risperidon during three weeks in the dose of 4 mg a day. The taking of blood for estimation of hormone level (prolactin, testosterone, T3, T4, TSH) made three times: before the administration of risperidon, on 10 and 21 day of it's taking. The blood took from 9 to 10 A.M. on an empty stomach.

31% of patients has shown the rise of TSH level (1,23 μcIU/μl before the beginning of therapy and 3,43 μcIU/μl on 21 day of it). At the same time there was no significant changes of T3 and T4 levels.

All of the men from the study group have shown noticeably increase of prolactin level (1496 IU/l) on the 10 day of beginning risperidon therapy in comparison with the basic level (343 μIU/l). On 21 day of therapy the prolactin level has already high (1639 μIU/l). Testosterone level during the treatment has decreased with 19% of men (from 17 nmol/l before therapy to 7 nmol/l on 21 day of therapy). The women before therapy had mean level of prolactin 420 μIU/l, on 10 day 2130 μIU/l, on 21 day 3150 μIU/l and there was no significant changes of they testosterone levels.

Conclusions: Risperidon therapy brings about the noticeably increase of prolactin level. Re-duction of testosterone level of part of male patients could be explain by the influence of hyperprolactinaemia which get broken pulsate secretion of FSH. The fact that some kind of pa-tients have the rise of TSH could be explained by risperidone's influence on serotoninergic and dophaminergic systems which exert significant influence on the TSH's secretion.

https://doi.org/10.1016/S0924-9338(02)80818-5 Published online by Cambridge University Press