

**Two cases of the neuroleptic malignant syndrome.
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Neuroleptic malignant syndrome was described by Dr. Delei in 1960 and it is caused by negative side-effect of the neuroleptic and anti-psychotic drugs. Neuroleptic malignant syndrome's rate is about 3-27% and mortality rate is around 40%. Neuroleptic malignant syndrome's pathogenesis are dysfunction of the central, dopamine and sympathetic nervous system and skeleton muscle membrane and it diagnosticate by antecedent anamnesis, past drug, life and family history and clinical presentation. Treatment program includes muscle relaxant and dopaminergic drugs, psychotropic activity decreasing medicals, plasmapheresis, hormonotherapy, brain anti-oedema, nosotropic and routine i.v. infusion therapy and etc.

Key words: neuroleptic malignant syndrome, critical patient.

Neuroleptic malignant syndrome presents vital function disorder that is caused by harmful effect of the neuroleptic and antipsychotic drugs. The disease was described in 1960 by Dr. Delei, when Phenothiazin was used for the first time. The disease is quite rare and sometimes it finishes with fatal outcomes. Its frequency in patients treated by neuroleptic drug is about 3-27%. Neuroleptic malignant syndrome's pathogenesis are dysfunction of the central, dopamine and sympathetic nervous system and skeleton muscle membrane and it diagnosticate by antecedent anamnesis, past drug, life and family history and clinical presentation.

The disease begins harmfully. The main symptom is fever. For the first time temperature is mild pyrexia, but gradually it gets higher and reaches febrile. There exist catatonic disorders -stupor, catalepsies, negativism and tachycardia. Attention should be paid to the existence of the catatonic and psychopathologic disorders showed by acute delirium and hallucination syndromes.

It must be mentioned that at neuroleptic malignant syndrome neuroleptic loading cessation markedly improves disease pattern.

Neuroleptic malignant syndrome's treatment standard is complex and includes skeleton muscle relaxant to remove the generalized rigidity of the muscles: dantrolen - 2-3 mg/kg, dopaminergic drugs – bromocriptine - 2, 5 mg. Psychotropic activity decreasing benzodiazepine, barbiturate and midazolam are also used as well as routine i.v. infusion therapy, hormonotherapy, cardiotoxic, brain anti-oedema and etc. There is neuroleptic malignant syndrome treatment little experience in the clinical medicine because of its rarity, so, it must be very interesting to describe every case of this disease. Let's describe 2 cases that were treated in The Critical Care Medicine Institute's clinical unit. In both cases the beginning of the disease was associated with taking antipsychotic drugs.

The first critical patient was 39 years old and severe chronic alcoholic. He had refused nutrition and at the same time otic and vision hallucinations were developed and in psychiatric outpatient center he was being treated for delirium tremens, where he was taking seduxen, haloperidol and neuroleptic – clopixon-depot, i.m. 200 mg.. After drug loading there were gradually appeared changes in extra-pyramid system like neck and limb muscles' rigidity, dysarthria and accompanied acute respiratory failure. There were also discovered hyperemia, high fever about 39°C, respiratory tract infection – bilateral bronchopneumonia, conjugated hallucinations, uncoordinated hyperkinesia and 6 point cerebral coma by Glasgow scale. The patient underwent baseline clinical examination and routine laboratory multiphasic testing. There were moderate hypercoagulation and leukocytosis with deviation to the left. Acid-base balance markers were supervised and strictly corrected. Standard intravenous infusion was used, as well as water exchange and

electrolyte supporting, brain anti-edematous and antibacterial therapy, parenteral and enteral feeding about 28 - 45 Kcal/Kg /per 24 hr.

The second critical patient was 17 years old teen. She had refused nutrition and otic and vision hallucinations had developed. There were expressed hyperemia and hyperthermia -39°C with subsequent subfebrility. The patient was hospitalized in psychiatric clinical unit, where she was taking tiftazin and leponex (clopazin-25 mg). After six days treatment she was admitted to the Critical Care Medicine Institute and long since had tremor, muscles rigidity and respiratory failure. The patient underwent baseline clinical examination and routine laboratory multiphasic testing. There were leukocytosis, trombocytosis and anemia. CPC was about 40 unit/l. Cerebrospinal fluid and spinal cord were intact. Virus and immune research also was done. CMV, EBV, (VCA), VZV were positive. T-general-34%, T-active-10%, T-helper-26%, T-suppressor-8%, T-lymphocyte-10 %. There were cerebral mild atrophic changes by nuclear magnetic resonance research.

In both cases the beginning of the disease was association with antipsychotic drugs' reception. In the first case it was clopixol-depot and in the latter case - leponex. In both cases the disease began in strict forms and antipsychotic drugs were stopped. Treatment was addressed towards making vital functions in order. For reduction of the hyperthermia and psycho-motor excitement benzodiazepines and barbiturates were used. There were also realized hemotransfusion, hormono- and antibacterial therapy and both patients underwent plasmapheresis from the first day of the treatment which was done by sessions for 6 days: during each session about 1000,0 ml of the blood was refined. In either case standard intravenous infusion therapy was used, as well as water exchange and electrolyte supporting, antiedematous glycerin about 1 mg/kg/24hr and parenteral and enteral feeding about 28 - 45 Kcal/Kg /per 24 hr. From the first days of the treatment progenitor precursors' committing therapy was done (1,2,3). After complex treatment patients health status considerably improved.

In the first case treatment's duration reached 25 patient-days and cost of the treatment was equal to 8825 USD. In either case treatment lasted 16 patient-days and had cost 5700 USD.

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ავთვისებიანი ნეიროლევსიური სინდრომის ორი შემთხვევა.
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ავთვისებიანი ნეიროლევსიური სინდრომი გამოწვეულია ნეიროლევსიური და ანტიფსიქოზური პრეპარატების უარყოფითი გვერდითი ეფექტით. დაავადება პირველად აღწერა ჯ. დელეიმ 1960 წელს. ავთვისებიანი ნეიროლევსიური სინდრომის სიხშირე ნეიროლევტიკებით მკურნალობისას 3-27%, ხოლო სიკვდილიანობა 40%. ავთვისებიანი ნეიროლევსიური სინდრომის პათოგენეზია ცენტრალური, დოფამინური და სიმპატიკური ნერვული სისტემების დისფუნქცია, ცვლილებები ჩონჩხის კუნთების მემბრანებში და სხვა. დაავადება ამოიცნობა ანამნეზისა და კლინიკური სურათის საფუძველზე. მკურნალობის სტანდარტი მოიცავს ჩონჩხის კუნთების რელაქსანტებს; ალგზების კუპირებისთვის იყენებენ ბენზოდიაზეპინს და ბარბიტურატებს; რეკომენდებულია ჰორმონოთერაპია, საჭიროებისას ფილტვების ხელოვნური ვენტილაცია და თავის ტვინის შეშუპების საწინააღმდეგო თერაპია. ავთვისებიანი ნეიროლევსიური სინდრომი იშვიათი დაავადებაა და მისი დიაგნოსტიკისა და მკურნალობის თაობაზე ექიმებს მცირე გამოცდილება აქვთ. ამდენად, თითოეული შემთხვევის აღწერა მნიშვნელოვანია. მოყვანილია ავთვისებიანი ნეიროლევსიური სინდრომის ორი შემთხვევის აღწერა, რომელსაც საქართველოს კრიტიკული მედიცინის ინსტიტუტში ჰქონდა ადგილი.