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Clinical case of ataxia of toxic origin

Raskurazhev A.A., Kuznetsova P.I., Tanashyan M.M.

Research Center of Neurology
80, Volokolamskoe Sh., Moscow, 125367, Russian Federation

ABSTRACT

A clinical case of ataxia development associated with accidental phenobarbital overdose is presented. Clinical manifestations and differential diagnosis are described; the results of laboratory examinations for this pathology are presented. In conclusion, the importance of collecting anamnestic data is emphasized.

Key words: ataxia, clinical neurology, toxicology.

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Клинический случай атаксии токсического генеза

Раскуражев А.А., Кузнецова П.И., Танашян М.М.

Научный центр неврологии
Россия, 125367, г. Москва, Волоколамское шоссе, 80

РЕЗЮМЕ

Представлен клинический случай развития атаксии на фоне случайно передозировки фенобарбитал-содержащего препарата. Описаны клинические проявления, дифференциальная диагностика, приведены результаты лабораторных обследований при данной патологии. В заключение делается акцент на важность сбора анамнестических сведений.

Ключевые слова: атаксия, клиническая неврология, токсикология.

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✉ *Kuznetsova Polina I.,* e-mail: angioneurology0@gmail.com.

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INTRODUCTION

Ataxia is a clinical syndrome of incoordination, which may result from damage to the cerebellum and related pathways [1]. Among the main manifestations of ataxia are gait disturbances and dysmetria, often associated with dysarthria and nystagmus. According to the classification [2], three major groups of ataxia can be defined: acquired, hereditary, and non-hereditary degenerative. The first group is comprised of a vast array of disorders including toxic, paraneoplastic, immune-mediated, and vitamin deficiency disorders. This article presents an unusual case of subacute ataxia of toxic origin due to phenobarbital exposure.

CLINICAL CASE

Patient D., a 32-year-old male, a dentist, had an appointment with a neurologist at the Research Center of Neurology where he complained of slurred speech, dizziness, unsteadiness and "staggering" gait (the patient himself described it "as if I were drunk"). Three days prior to the visit, he was admitted to one of the city hospitals with the specified symptoms. Upon admission, a computed tomography of the brain was performed. A CT scan with no pathological changes was obtained, and the patient was diagnosed with a transient ischemic attack in the vertebrobasilar basin. Subsequently, the patient left the medical facilityon his own. The symptoms developed gradually in the course of several weeks with no obvious etiological factor.

On examination, the patient was conscious, though drowsy. Systolic blood pressure was 100 mm Hg, diastolic – 60 mm Hg; heart rate was 64 bpm. A neurologic examination revealed moderate dysarthria, severe horizontal and vertical nystagmus, dysmetria during finger-to-nose test, and dysdiadochokinesia. Kinetic tremor, muscle hypotonia, and hyporeflexia were also noted. The gait was unsteady, wide-based.

Magnetic resonance imaging of the brain showed no signs of ischemic or degenerative pathology; the results of ultrasound examination were within normal values.

The patient denied the use of alcohol or any narcotic substances. However, when clarifying the anamnesis, it turned out that during the previous month the patient had problems with sleep, for which he was taking the drug "Valocordin" (Table 1).

Table 1

Composition of the "Valocordin" drug

Substance	Content, mg per 1 ml of the drug
Phenobarbital	18.40
Ethyl bromisovalerianate	18.40
Menthol oil	1.29
Hop oil	0.18
Ethanol	469.75

Due to its sedative properties, this drug is frequently used as a mild sleeping medicine with a standard dose amounting to 10–20 drops (0.25–0.5 ml). In the described case, the patient used 20–25 ml of "Valocordin" every other night for 4 weeks.

Subsequent blood and urine tests were performed (Table 2), which demonstrated a significant increase in phenobarbital excretion. On this basis, it was suggested that the patient had phenobarbital intoxication with ataxia being the main clinical manifestation.

The patient was prescribed intravenous saline infusions, as well as oral activated charcoal for the next 10 days. On the 2nd day of the therapy, a significant improvement was observed, on the 8th day the patient had made a complete recovery.

Table 2

Laboratory findings in patient D., 32 years old

Parameter	Value	Reference
Complete blood count		
Hemoglobin, g/L	151	131–173
Erythrocytes, × 10^{12}/L	5.06	4.3–5.7
Hematocrit, %	44.3	39–49
Platelets, × 10^{9}/L	386	180–320
Leukocytes, × 10^{9}/L	7.29	4.5–11.3

Table 2 (continued)

Parameter	Value	Reference
ESR, mm/h	17	0–15
Hemostasis		
APTT, s	31.9	25.1–36.5
PTI, %	98	70–130
Fibrinogen, g/L	5.11	1.8–4.0
Antithrombin III, %	153	75–125
Blood chemistry		
Creatinine, mcmol/L	68.0	62–106
Glucose, mmol/L	5.0	4.1–5.9
ALT, U/L	18.6	< 41
AST, U/L	28.3	< 40
CPK, U/L	211	< 190
Bilirubin, mcmol/L	3.1	< 24
C-reactive protein, mg/L	83.97	< 5
Uric acid, mcmol/L	468.0	202.3–416.5
Excretion of barbiturates in urine		
Amobarbital, ng/mL	0.0	0–200
Butalbarbital, ng/mL	0.0	0–200
Pentobarbital, ng/mL	0.0	0–200
Secobarbital, ng/mL	0.0	0–200
Phenobarbital, ng/mL	501,538.9	0–200

Note. ESR – erythrocyte sedimentation rate, APTT – activated partial thromboplastin time, PTI – prothrombin index, ALT - alanine aminotransferase, AST – aspartate aminotransferase, CPK – creatine phosphokinase.

CONCLUSION

Ataxia is a common neurological syndrome and a frequent reason for seeking medical care. Nevertheless, a broad spectrum of underlying pathologies is associated with this condition, which often presents difficulties in differential diagnosis. The described case underlines the necessity of a detailed and thorough patient interview, which was proven to be of vital importance in this particular patient. Although ataxia is listed in the "Side effects" section of the phenobarbital instructions for use, a search for relevant publications in the PubMed database did not reveal any reports of ataxia resulting from accidental overdose of phenobarbital drugs.

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Authors information

Raskurazhev Anton A., Cand. Sci. (Med.), Researcher, 1st Neurological Department, Research Center of Neurology, Moscow, Russian Federation. ORCID 0000-0003-0522-767X.

Kuznetsova Polina I., Cand. Sci. (Med.), Researcher, 1st Neurological Department, Research Center of Neurology, Moscow, Russian Federation. ORCID 0000-0002-4626-6520.

Tanashyan Marine M., Dr. Sci. (Med.), Professor, Corresponding Member of RAS, Head of the 1st Neurological Department, Deputy Director for Scientific Work, Research Center of Neurology, Moscow, Russian Federation. ORCID 0000-0002-5883-8119.

(✉) **Kuznetsova Polina I.,** e-mail: angioneurology0@gmail.com.

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