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Р.Ф. Ахмадуллина[1], М.А. Кутлубаев[2]

[1] — ГБУЗ «Республиканская клиническая больница им. Г.Г. Куватова», отделение неврологии, Уфа, Россия
[2] — ФГБОУ ВО «Башкирский государственный медицинский университет» Министерства здравоохранения России, кафедра неврологии, Уфа, Россия

СЛУЧАЙ ИНТОКСИКАЦИИ ТАЛЛИЕМ

R.F. Akhmadullina[1], M.A. Kutlubaev[2]

[1] — G.G. Kuvatov Republican Clinical Hospital, Department of Neurology, Ufa, Republic of Bashkotostan, Russia
[2] — Bashkir State Medical University, Department of Neurology, Ufa, Russia

Case of thallium intoxication

Резюме

Соединения таллия крайне токсичны. Механизмы их токсичности связаны со снижением активности ферментов, участвующих в метаболизме глюкозы и нарушением синтеза макроэргов. Интоксикация таллием появляется через 3-4 часа после его попадания в желудочно-кишечный тракт (ЖКТ) в виде диспептических явлений, через 2-5 суток –появляются симптомы поражения нервной системы в виде сенсорной полинейропатии с нейропатическим болевым синдромом. Через 2-3 недели развиваются дерматологические осложнения: алопеция, ангидроз, глоссит. Антидотом является гексацианоферрат калия.

Представляем собственное клинической наблюдение. Женщина, 38 лет, поступила в стационар с подострой сенсорно-моторной полинейропатией, гепатитом и алопецией. Лабораторные исследования продемонстрировали незначительное повышение уровня печеночных ферментов и тенденцию к снижению уровня калия в крови. Стимуляционная электронейромиография выявила сенсорно-моторную полинейропатию по типу аксонопатии. Учитывая полисистемность поражения, проводилась дифференциальная диагностика между аутоиммунными заболеваниями и острой интоксикацией. Анализ крови на маркеры системных заболеваний соединительной ткани дал отрицательный результат. Токсикологическое исследование сыворотки и мочи выявили резкое повышение уровня таллия. Введение гексацианоферрата калия дало положительный результат. Осмотр пациентки через год показал полное разрешение моторных и сенсорных симптомов, возобновление роста волос.

Интоксикации таллием в настоящее время встречаются крайне редко и представляют трудности для своевременной диагностики. При токсических полинейропатиях необходимо проводить дифференциальную диагностику с интоксикациями другими тяжелыми металлами (свинец, ртуть, кадмий), а также аутоиммунными процессами. Своевременное введение антидота позволяет вывести соединения таллия из организма и получить выраженным положительный клинический эффект.

Ключевые слова: *таллий, интоксикация, полинейропатия, алопеция*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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Abstract

Thallium compounds are extremely toxic. Their mechanisms of toxicity are associated with reduced activity of enzymes involved in glucose metabolism and impaired synthesis of high-energy compounds. Symptoms of thallium intoxication appear 3-4 hours after its ingestion in the form of dyspeptic phenomena. Within 2-5 days, symptoms of nervous system damage appear in the form of sensory polyneuropathy with neuropathic pain syndrome. After 2-3 weeks, dermatological complications develop: alopecia, anhidrosis, glossitis. The antidote is potassium hexacyanoferrate.

Clinical case. A 38-year-old woman was admitted to the hospital with subacute sensorimotor polyneuropathy, hepatitis, and alopecia. Laboratory tests showed a slight increase in liver enzyme levels and a tendency towards decreased blood potassium levels. Stimulatory electroneuromyography revealed sensorimotor polyneuropathy of the axonal type. Given the multisystem involvement, a differential diagnosis was conducted between autoimmune diseases and acute intoxication. A blood test for markers of systemic connective tissue diseases was negative. Toxicological analysis of serum and urine revealed a sharp increase in thallium levels. Administration of potassium hexacyanoferrate yielded a positive result. A follow-up examination of the patient one year later showed complete resolution of motor and sensory symptoms and regrowth of hair.

Thallium intoxications are currently extremely rare and present challenges for timely diagnosis. In cases of toxic polyneuropathies, differential diagnosis should include intoxication with other heavy metals (lead, mercury, cadmium) as well as autoimmune processes. Timely administration of the antidote facilitates the elimination of thallium compounds from the body and yields a pronounced positive clinical effect.

Key words: *thallium, intoxication, polyneuropathy, alopecia*

Conflict of interests
The authors declare no conflict of interests

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Conformity with the principles of ethics
The patient consented to the publication of laboratory and instrumental research data in the article «Case of thallium intoxication» for the journal «The Russian Archives of Internal Medicine» by signing an informed consent

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GIT — gastrointestinal tract, ATP — adenosine triphosphate, ENMG — electroneuromyography, ECG — electrocardiography, VAS — visual analogue scale, BP — blood pressure, HR — heart rate, RR — respiratory rate, MRI — magnetic resonance imaging, SCTD — systemic connective tissue disorders

Introduction

Thallium is a heavy metal, which is a component of over 20 rare minerals. Thallium does not occur in nature in its pure form; however, synthetically produced thallium salts were previously used in some countries in agriculture as components of pesticides, as well as in medicine and cosmetology. The widespread use of thallium compounds stopped in mid-1970s; however, there are still clinical cases of thallium intoxication [1]. The literature even contains reports of mass thallium poisoning [2].

Thallium salts are highly toxic compounds, which are colourless and odourless. They can penetrate the body during direct skin contact, by inhalation (via lungs), and as a result of swallowing (via gastrointestinal tract (GIT)). Nowadays, poisoning with thallium compounds can occur as a part of occupational intoxication of chemical lab staff, with thallium-contaminated food or biologically active supplements, as well as in case of intentional malicious thallium poisoning [3].

The mechanisms of thallium toxicity are associated with reduced activity of enzymes involved in glucose metabolism and impaired synthesis of adenosine triphosphate (ATP) and other high-energy compounds. It causes impaired ATP-dependent Na/K-ion channel functioning and oedema, swelling and death of excitable tissues. Thallium also modifies riboflavin bioavailability and impairs ATP synthesis. Other mechanisms of thallium toxicity include disruption of the protein keratin through cleavage of cysteine-mediated disulphide bonds; inhibition of protein synthesis due to ribosomal damage, particularly involving the 60S subunit; and demyelination of nerve fibres in both the central and peripheral nervous systems [4].

Clinical manifestations of thallium poisoning can be seen 3–4 hours after swallowing as dyspepsia. Two to five days later, symptoms of nervous system damage appear: sensory polyneuropathy with neuropathic pain syndrome, ataxy, trembling; and the pathological process often involves cranial nerves. Two to three weeks later, dermatological complications develop: hair loss, anhidrosis, glossitis. Examination can reveal Aldrich-Mees lines on nails. Other signs of thallium intoxication include sleep disturbances, acute symptomatic cramps, and headache. Severe cases can result in a coma [5].

Diagnosis is established if thallium is found in body fluids, mostly in urine, and also in hair. In addition, a standard panel of laboratory tests is performed, along with electrocardiography (ECG) and stimulation electroneuromyography (ENMG) [5].

If ingested, thallium intoxication is treated with potassium hexacyanoferrate (Berlin blue), while activated carbon is less efficient. Haemodialysis helps reducing thallium concentration in blood [5].

At present, thallium poisoning is extremely rare, and healthcare professionals often have a low index of suspicion for this condition. This slows down diagnosis and targeted therapy, resulting in severe complications

and even death. Below is a case study of severe thallium intoxication with predominant peripheral nervous system involvement.

Case Study

Female patient U., 38 years old, with higher education, employed, was admitted to the Neurology Ward of G. G. Kuvatov Republican Clinical Hospital (Ufa) on December 5, 2023.

The patient complained of marked leg pain (anteroexternal thigh area, back of shins, and bottom of feet), 7–8 points on the visual analogue scale (VAS); leg weakness, making it difficult to walk; massive hair loss; recurrent abdominal heaviness; unstable stool; tearfulness; sleep disturbances.

She felt ill two weeks ago, when she noticed thigh pain; next day, pain spread to shins and feet; at the same time, she felt stabbing pain in her heart and lump in the throat. One week later, she noticed intensive hair loss.

She was diagnosed with autonomic instability and underwent outpatient treatment at the place of her residence. Since pain in her legs was worsening, the patient was hospitalised to the local district hospital with lumbosacral osteochondrosis (muscular irritation syndrome, attack phase). The therapy (non-steroidal anti-inflammatory drugs, muscle relaxants, vitamins B) was not efficient, and the patient was referred to G. G. Kuvatov Republican Clinical Hospital (Ufa).

The patient denied any chronic diseases.

Upon admission, the patient had hardly any hair left on the hairy part of her head. Positive hair pull test. Growth of the eyebrows and axillary hair was preserved. The skin was pale, with no rashes. The patient had an asthenic body build; body mass index: 17.95 kg/m2; blood pressure (BP): 110/70 mm Hg; heart rate (HR): 85 beats per minute. Cardiac rhythm was regular, heart sounds were normal, and no murmurs were detected. Vesicular breath sounds were present throughout all lung fields, with a respiratory rate of 18 breaths per minute. The abdomen was soft, with moderate tenderness on palpation in the epigastric and pylorobulbar regions. The liver and spleen were not enlarged.

Assessment of neurological status: cranial nerves are intact. A fine postural tremor of the hands was present. Arm muscle strength: 5 points; leg muscle strength: 3 points (proximal), 4 points (distal). Muscular hypotonia in legs was observed. Deep tendon reflexes in the upper extremities were brisk and symmetrical. In the lower extremities, patellar reflexes were absent, and Achilles tendon reflexes were diminished, more prominently on the left. Marked hyperesthesia of the feet was noted. Thigh and shin muscles were painful on palpation. Deep sensation was moderately diminished in lower extremities. No pathological reflexes were observed. There was no bladder or bowel dysfunction. The straight leg raise (Lasegue) test was positive at 60° bilaterally. The patient was emotionally labile.

Bloodwork showed slightly elevated erythrocyte sedimentation rate of 24 mm/h (reference value for women is up to 20 mm/h), CRP — 29.75 mg/L (up to 5 mg/L), ALT — 169.2 U/L, AST — 59.8 U/L (up to 31 U/L). Potassium and sodium levels were at the lower level of reference values: 3.62 and 138 mmol/L, respectively. Thyroid hormone levels were unremarkable. Urinalysis demonstrated leukocyturia (15 cells/μL). Spinal fluid composition was unchanged.

ECG showed sinus rhythm, HR 85 beats per minute; diffuse abnormalities in myocardium repolarisation were observed. Abdomen ultrasound revealed diffuse changes in the liver and deformed gall bladder. Magnetic resonance imaging (MRI) of thoracic and lumbar spine demonstrated degenerative-dystrophic changes, which could not be explained by the present neurological symptoms. Stimulation electroneuromyography showed signs of polyneuropathy, mostly with axonal involvement.

Since the patient had sensorimotor polyneuropathy syndrome, hair loss and liver involvement, differential diagnosis included systemic connective tissue disorder (SCTD) and intoxication with an unknown substance.

The patient was examined by a rheumatologist; her blood was tested for antibodies against double-stranded DNA, C3 and C4 component of the compliment, antinuclear antibody, and anti-mitochondria antibodies. Clinical and laboratory test results ruled out autoimmune diseases.

Biomaterials were tested for toxic microelements: thallium (hair) 1947.58 μg/kg (reference value: below 5 μg/kg), thallium (serum) 127.24 μg/L (below 0.05 μg/L), thallium (urine) 2020.98 μg/L (below 1 μg/L), thallium (nails) 12018.89 μg/kg (below 5 μg/kg). Concentrations of other microelements were within the normal range.

Given the clinical representation and results of toxic microelement tests, thallium intoxication was established. The patient was transferred to Acute Intoxication Ward at another medical institution. Therapy included sodium thiosulfate, unitiol, potassium hexacyanoferrate. Once potassium hexacyanoferrate was administered, the patient noted significant improvement of her condition.

At the 3-month follow-up examination, the patient's condition had improved, with resolution of the pain syndrome, improved gait (she no longer required assistance for walking), and restoration of scalp hair growth. The patient still complained of her leg muscles getting tired very quickly when walking for long distances; numb feeling in her feet; and emotional instability. Clinically,

Figure 1. *Mee's lines on finger nails in 3 months after intoxication*

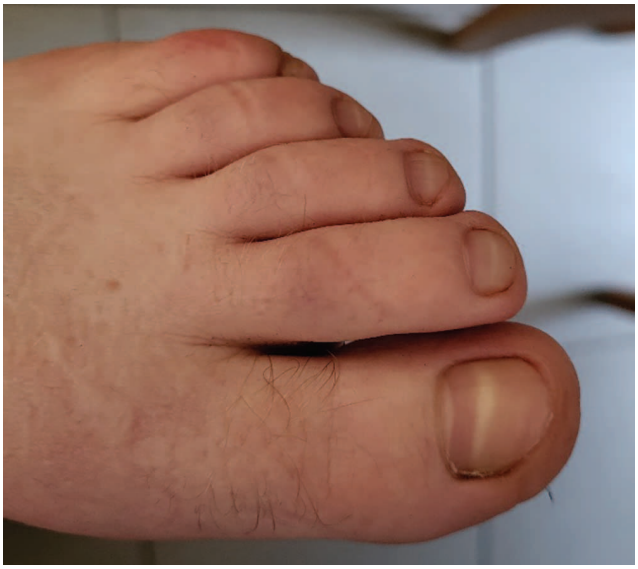

Figure 2. *Mee's lines on toe nails in 3 months after intoxication*

reduced muscle strength in the feet persisted, graded as 4/5, along with autonomic disturbances involving the skin of the feet (increased sweating and coldness to the touch). White transverse lines on the nails of fingers and toes (Aldrich-Mees lines, see Fig. 1 and 2) are worth noting.

Assessment using the Hospital Anxiety and Depression Scale (HADS) revealed subclinical anxiety (score: 10 points). Cognitive functions were normal, as demonstrated by Montreal Cognitive Assessment (29 points). Repeat measurement of thallium levels performed 1.5 months after admission showed concentrations of 10.99 μg/L in urine and 0.509 μg/L in blood serum. Following specific antidote therapy, thallium blood and urine concentrations dropped approximately 180–250 times, but remained 10 times the reference value.

At the 1-year follow-up examination, signs of peripheral autonomic dysfunction in the lower extremities persisted, along with increased emotional lability. A daytime anti-anxiety medication was prescribed, resulting in a favourable clinical response. Sensorimotor polyneuropathy resolved. Aldrich-Mees lines disappeared. The route by which thallium entered the patient's body is still unknown.

Discussion

At present, thallium compounds are hardly used in domestic settings, therefore thallium intoxication is extremely rare. In this clinical case, the patient exhibited multisystem manifestations at the time of hospital admission, and the differential diagnosis primarily included systemic connective tissue diseases and toxic exposures; accordingly, investigations were performed as part of a toxicological screening workup. Toxic polyneuropathy and hair loss are more common for patients undergoing chemotherapy; in this patient, this option was ruled out. Accordingly, it was decided to assess biological specimens for the presence and concentrations of toxic trace elements.

Toxic polyneuropathy can develop after intoxication with heavy metals — lead, arsenic, cadmium, and thallium. Lead-mediated polyneuropathy is characterised by asymmetrical involvement of motor fibres, as well as kidney damage and microcytic anaemia. In earlier years, this polyneuropathy was common; however, abandonment of lead compounds from industrial and domestic use resulted in drastic reduction in incidence of this condition. Arsenic poisoning is primarily characterised by gastrointestinal manifestations, followed by the development of polyneuropathy resembling acute inflammatory demyelinating polyneuropathy. Cadmium poisoning is characterised by polyneuropathy together with internal organ damage, encephalopathy and anosmia (Table 1) [7].

A retrospective analysis demonstrated that the patient had symptoms of acute thallium poisoning. The most common clinical signs are axonal polyneuropathy with pain syndrome and hair loss. Laboratory test results revealed moderate liver damage, as well as tendency to hypokalemia, which is also typical of thallium intoxication. Specific antidote therapy resulted in prompt favourable effect.

Figure 3 shows a diagnostic and therapy algorithm for thallium poisoning.

Table 1. *Differential diagnosis of toxic polyneuropathies caused by heavy metal poisoning*

	Lead Poisoning	Arsenic Poisoning	Cadmium Poisoning	Thallium Poisoning
Source of Exposure	Battery production, lead dust, old paint.	Semiconductors, pesticides, contaminated water/soil.	Battery production, pigments, soldering.	Electronics manufacturing, in the past — pesticides, rodenticides.
Type of Polyneuropathy	Predominantly **motor**. Primarily affects wrist extensors ("wrist drop"). Chronic, slowly progressive.	Predominantly **sensorimotor with neuropathic pain**. Acute or subacute course.	Predominantly **sensory or sensorimotor**.	Severe **sensorimotor polyneuropathy with neuropathic pain**. Rapid progression.
Pathognomonic Signs	Lead line on gums (Burton's line). Microcytic anemia with basophilic stippling. Abdominal pain («lead colic»), constipation.	Mees' lines (leukonychia) — white transverse stripes on nails. Skin manifestations (hyperkeratosis, melanosis). GI involvement resembling cholera (vomiting, diarrhea).	Proteinuria (renal tubular damage). Emphysema, osteomalacia. Olfactory disturbances.	**Hair loss (alopecia)** 2-3 weeks after poisoning. Mental disorders.
Laboratory Diagnostics	Blood (lead in whole blood).	Urine (24-hour urine for arsenic). Hair/nails (in chronic poisoning).	Urine (cadmium in urine). Blood (reflects recent exposure).	Urine (thallium in urine). Blood (in acute phase).
Antidotes	Ethylenediaminetetraacetic acid (EDTA); D-penicillamine	Unithiol; D-penicillamine	Ethylenediaminetetraacetic acid (EDTA)	Potassium hexacyanoferrate (II) («Prussian blue»)

Comparison with previously published reports of thallium poisoning showed that the clinical case presented here is representative of the typical manifestations of this condition. However, unlike cases of more severe poisoning, this patient did not have any signs of cranial nerve involvement manifesting as visual and oculomotor disorders, nystagmus. The patient did not have clear manifestations of nephropathy, whereas high thallium concentrations in the body cause acute kidney damage, which leads to poor outcomes [4].

Conclusion

This clinical case demonstrates the importance of timely diagnosis of thallium intoxication. Acute multisystem involvement affecting the peripheral nervous system, the skin and its appendages, as well as the liver and kidneys, should prompt screening for thallium intoxication, even in the absence of a history of exposure to chemical agents. Once thallium intoxication has been confirmed, immediate therapy with potassium hexacyanoferrate should be initiated.

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Кутлубаев М.А.: формирование идеи и структуры статьи, редактирование, утверждение финального варианта рукописи

Author Contribution:

All the authors contributed significantly to the study and the article, read and approved the final version of the article before publication.

Akhmadullina R.F.: Data collection and analysis, preparation and writing of the article text

Kutlubaev M.A.: Conceptualization and design of the article, editing, final approval of the manuscript

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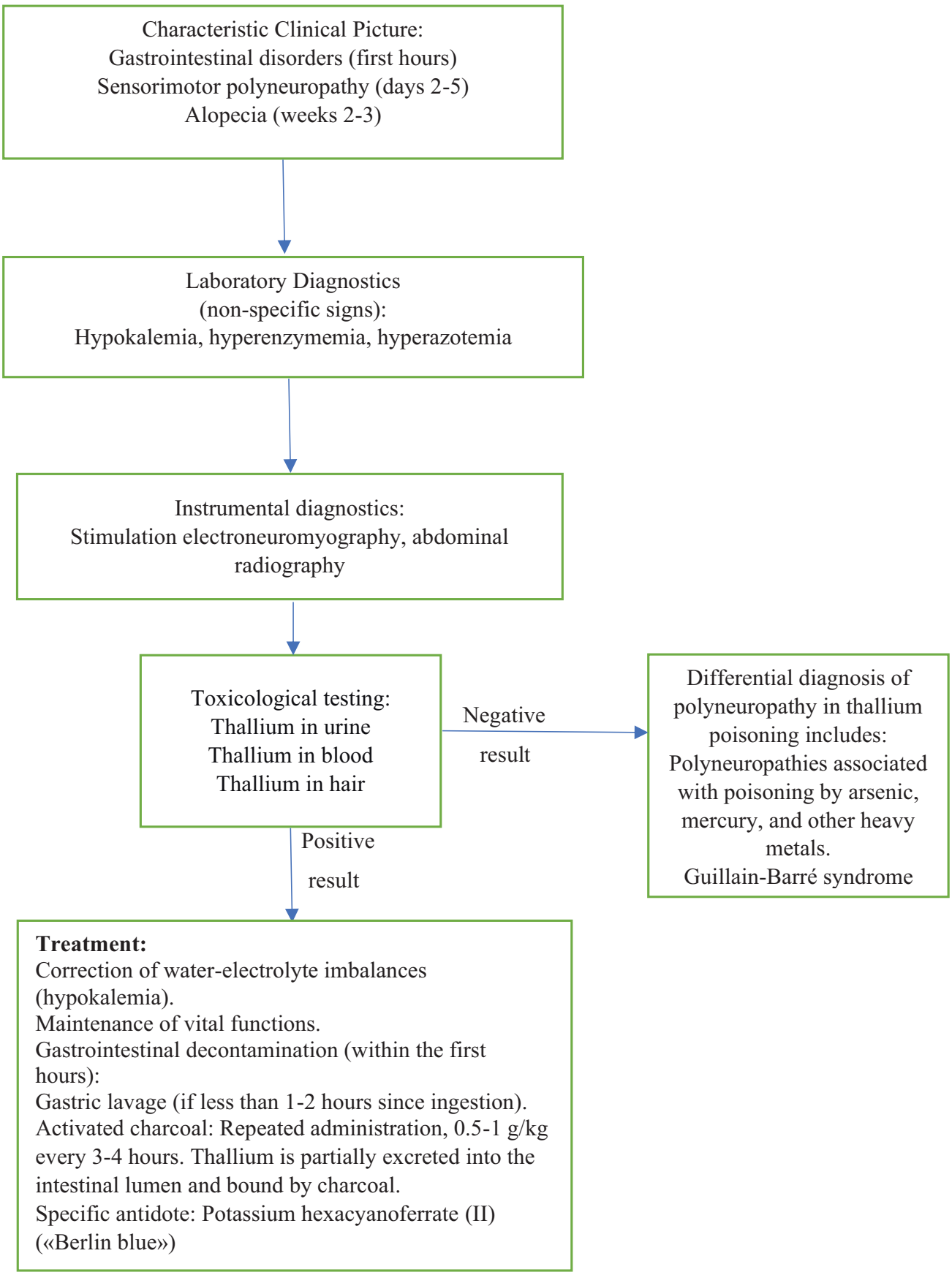

Figure 3. *Diagnostic and Treatment Algorithm for Thallium Poisoning*

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Информация об авторах:

Ахмадуллина Регина Фуатовна — врач- невролог, ГБУЗ Республиканская клиническая больница им. Г.Г. Куватова, Уфа, E-mail: regina.akhmadullina17@gmail.com, ORCID ID: https://orcid.org/0009-0000-2608-3578

Кутлубаев Мансур Амирович — д.м.н., доцент, заведующий кафедрой неврологии ФГБОУ ВО «Башкирский государственный медицинский университет» Минздрава России, Уфа, E-mail: E-mail: mansur.kutlubaev@yahoo.com, ORCID ID: https://orcid.org/0000-0003-1001-2024

Authors Information:

Regina F. Akhmadullina — Neurologist, Republican Clinical Hospital named after G.G. Kuvatov, Ufa, Russia. E-mail: regina.akhmadullina17@gmail.com; https://orcid.org/0009-0000-2608-3578

Mansur A. Kutlubaev — MD, PhD, Associate Professor, Head of the Department of Neurology, Bashkir State Medical University, Ufa, Russia; E-mail: mansur.kutlubaev@yahoo.com, https://orcid.org/0000-0003-1001-2024

Автор, ответственный за переписку / Corresponding author