DOI 10.29254/2077-4214-2024-2-173-246-253
UDC 616.746/748-008-071

[1,2]Synhaivskyi A. M., [1]Delva M. Yu., [1]Purdenko T. Y., [2]Pushko O. O.,
[1]Tarianyk K. A., [1]Sylenko H. Ya., [1]Hryn K. V.

LIMB-GIRDLE MUSCULAR DYSTROPHY, TYPE 2A (CALPAINOPATHY): CLINICAL OBSERVATION

[1]Poltava State Medical University (Poltava, Ukraine)
[2]Municipal Enterprise «M.V. Sklifosovskyi Poltava Regional Clinical Hospital of the Poltava Regional Council» (Poltava, Ukraine)

s000572@vnmu.edu.ua

The article illustrates a case study derived from our clinical observations, focusing on a patient diagnosed with limb-girdle muscular dystrophy, specifically type 2A, commonly known as calpainopathy. This is a group of rare genetic diseases of non-inflammatory genesis, which are based on a primary deficiency of muscle fibers due to their atrophy. To date, more than 30 different genetic forms of limb-girdle muscular dystrophies have been identified. The article outlines the clinical manifestations and diagnostic criteria associated with this condition. The disease has an autosomal recessive type of inheritance, accounting for about 30% of all cases, and occurs as a result of a point mutation in the CAPN3 gene (15q15.1 - q21.1). The normal product of the CAPN3 gene is the proteolytic protein calpain-3, which probably ensures cytoskeletal stability. Calpain-3 is the only member of the calpain family that is present in skeletal muscle and is involved in the regulation of various tissue processes. There are 2 clinical forms of calpainopathies 2A: Leyden-Möbius type is characterized by initial weakness of the pelvic girdle muscles, which eventually spreads to the shoulder girdle, while in Erb type, weakness first manifests itself in the shoulder girdle muscles, and after a few years - in the pelvic girdle muscles. In order to diagnose calpainopathy, the doctor should be guided by the anamnesis, clinical presentation, results of laboratory, functional and medical genetic studies, and histological examination of the affected muscle.

Currently, there is no specific treatment for calpainopathy. Treatment is symptomatic in nature, aimed at normalizing the patient's general condition, restoring lost functions and possibly slowing down the rate of disease progression. Dynamic patient monitoring and symptom management improve the possibility of extending the duration and quality of life.

Key words: *limb-girdle muscular dystrophy, calpainopathy, diagnosis, treatment.*

Connection of the publication with planned research works.

The article is a part of the research project «Optimization of diagnostics, prognosis and prevention of neuropsychological disorders in organic diseases of the nervous system», state registration number 0120U104165.

Introduction.

Limb-girdle muscular dystrophy (LGMD) is a group of rare genetic diseases of non-inflammatory genesis, which are based on a primary deficiency of muscle fibers due to their atrophy. LGMD is clinically manifested by progressive muscle weakness, muscle atrophy, and motor disorders, with onset ranging from early childhood to adulthood, and varying course [1, 2].

In the middle of the 19th century, Duchenne de Boulogne and William Gowers described several patients of different genders and ages, whose clinical symptoms were later termed «Duchenne myodystrophy» [1]. The German physicians Ernst Viktor von Leyden (1876) and Paulus Julius Möbius (1879) were the first to identify isolated cases of adult patients with atrophy of the pelvic girdle and thigh muscles. In 1884, Wilhelm Heinrich Erb described a childhood form of proximal muscle weakness, and in 1891 he coined the term «progressive muscular dystrophy» [3].

The term LGMD itself was proposed by J.N. Walton and F.J. Nattras in 1954 [4], but without clear guidelines for its definition. In 2017, a new system for the definition and classification of this pathology was presented at the 229th European Neuromuscular Center International Workshop [5]. Today, LGMD is recognized as a genetic disease that leads to progressive, predominantly proximal muscle weakness, with elevated serum creatine kinase and dystrophic changes detected by histological examination of the affected muscle and confirmed by genetic testing [1, 5, 6].

To date, there are more than 30 genetically determined forms of LGMD, which are divided into two types. Type 1 is autosomal dominant (AD-LGMD), in which 2 copies of a gene have the same mutation, they are numbered from 1 to 8, and type 2 is autosomal recessive (AR-LGMD), in which one copy of a mutated gene in a cell causes a disorder, they are numbered from 1 to 24 [5]. About 10% of the forms are dominant and 90% are recessive [7, 8]. AR-LGMD has a juvenile onset and a rapid increase in symptoms, while AD-LGMD is more likely to debut after the age of 20 and has a gradual progressive course [9].

In the population, the prevalence of LGMD varies, on average, from 4-7 cases per 100 thousand people, depending on geographic and ethnic characteristics [2, 10]. Unfortunately, there are no accurate statistics on the prevalence of these diseases in Ukraine, as no relevant epidemiological data have been collected.

The nature of the clinical manifestations of LGMD is determined by mutations in various genes responsible for the synthesis of specific proteins necessary for normal muscle function and recovery [11].

Calpainopathy, or AR-LGMD type 2A, is caused by a point mutation in the CAPN3 gene (15q15.1 – q21.1). The normal product of the CAPN3 gene is the proteolytic

protein calpain-3, which probably ensures cytoskeletal stability. Calpain-3 is the only member of the calpain family that is present in skeletal muscle and is involved in the regulation of various tissue processes [12].

To date, there is no clear data on the function of the calpain-3 protein, so the pathogenetic mechanisms of AR-LGMD type 2A development are not yet fully explained [13].

Calpainopathy is the most common type of LGMD and accounts for approximately 30% of all LGMD [14]. AR-LGMD type 2A is manifested by progressive symmetrical (in 25% of cases, asymmetrical lesions are observed) weakness and atrophy of the proximal muscle groups – the shoulder and pelvic girdles. The debut of calpainopathies varies widely (from 2 to 60 years), but usually symptoms begin to develop during the first 20 years of life. It is known that the earlier the disease begins, the faster its progression is expected [14, 15, 16, 17].

For an accurate clinical diagnosis of calpainopathy, it is important to establish the sequence of involvement of the relevant muscles in the pathological process. The type of calpainopathy in which weakness of the pelvic girdle muscles is initially observed and eventually spreads to the shoulder girdle is called the Leyden-Möbius type. The type of calpainopathy in which weakness first manifests itself in the muscles of the shoulder girdle, and after a few years – in the muscles of the pelvic girdle, is called the Erb type [1, 3, 18, 19].

The primary early clinical indicator that sets calpainopathies apart from other types of LGMD is the distinctive toe walking observed in children, often identified prior to the onset of muscle weakness [12]. Daytime activities such as rising from the floor, running, jumping, or engaging in physical pursuits are notably compromised during early childhood, yet the capability to walk typically remains intact until middle childhood [19]. When attempting to rise from the floor, children commonly resort to the Gowers technique [1].

Calpainopathy manifests with notable atrophy of the scapular muscles, often resulting in the characteristic "wing-shaped scapulae," alongside weakness observed in the biceps brachii, hip adductors, as well as flexors and extensors of the hips and knees. Notably, the muscles of the face and neck remain unaffected [12]. While pseudohypertrophy of the calf muscles is infrequent in calpainopathy patients, joint contractures progressively emerge in the later stages of the condition, affecting various joints including the ankles, knees, hips, elbows, and interphalangeal joints [20].

The heart muscle is not affected, but patients present with dysfunction of the cardiac conduction system [19, 20, 21]. Cardiomyopathy in patients with calpainopathy is quite rare, as the calpain-3 protein is not expressed in the heart muscle [13, 22, 23].

Individuals diagnosed with AR-LGMD type 2A commonly exhibit degenerative and dystrophic alterations in the spine, characterized by hyperlordosis of the lumbar spine and the presence of scoliosis. Importantly, cognitive function typically remains unaffected in these patients [3].

During the advanced stages of calpainopathy, impairment of the diaphragm and respiratory muscles becomes apparent, resulting in hypoventilation and indications of respiratory insufficiency [24, 25]. Although respiratory complications in calpainopathy tend to be milder compared to other forms of muscular dystrophy, they nonetheless diminish both the quality and longevity of the patient's life [10].

The aim of the study.

To present a clinical case of a patient with calpainopathy to attract the attention of family doctors, neurologists, physical therapists, pediatricians, traumatologists and other specialists.

Object and methods of the research.

Complaints, medical and life history, objective and neurological examination, laboratory, neuroimaging and neurophysiological methods of examination of a patient with calpainopathy.

The study was conducted in accordance with the principles of the Helsinki Declaration of Human Rights, the Council of Europe Convention on Human Rights and Biomedicine. Written informed consent was obtained for the study and the collection and processing of patient data.

Research results.

Patient A, aged 29, underwent treatment at the Neurology Department of the Municipal Enterprise "M. V. Sklifosovskyi Poltava Regional Clinical Hospital of the Poltava Regional Council" in the summer of 2023. Upon admission, he presented with complaints of limb weakness, particularly in the lower extremities, muscle wasting in the shoulders, buttocks, and thighs, swelling in the lower legs, significant gait disturbances, and difficulty with activities such as climbing stairs and rising from bed.

His medical history reveals a five-year duration of illness, initially characterized by progressive weakness in the proximal leg muscles, followed by the onset of similar weakness in the arms about 1.5 years ago. Over the past two years, progressive deterioration in walking ability has been noted, prompting his first medical consultation.

Family history indicates that the patient is the third child in the family, with two healthy older sisters. He attained developmental milestones appropriately, received vaccinations according to the schedule, and denies any history of surgeries, injuries, or chronic illnesses. No motor disorders were observed during childhood. It is noteworthy that the patient's parents are third-degree relatives.

Upon objective examination, the patient presents with an asthenic physique and pale pink skin and mucous membranes. Vital signs are within normal limits, with a body temperature of 36.7°C, respiratory rate of 17 per minute, and SpO2 of 97%. Auscultation reveals vesicular breathing in the lungs with no wheezing, while blood pressure is 125/80 mm Hg, and pulse is 78 beats per minute, both with satisfactory filling and tension. Cardiac sounds are rhythmic and sound, and the abdomen is soft and non-tender upon palpation, with normal physiological discharges.

Neurologically, oculomotor skills are intact, with mild nasolabial asymmetry and a midline tongue. Reflexes of the soft palate and pharyngeal wall are preserved, and there is no impairment in speech or swallowing. However, there is reduced muscle tone predominantly in the proximal extremities, especially the legs, with a lower Barre "+" test. Strength in the proximal upper extremities, specifically the deltoid muscles and biceps, is di-

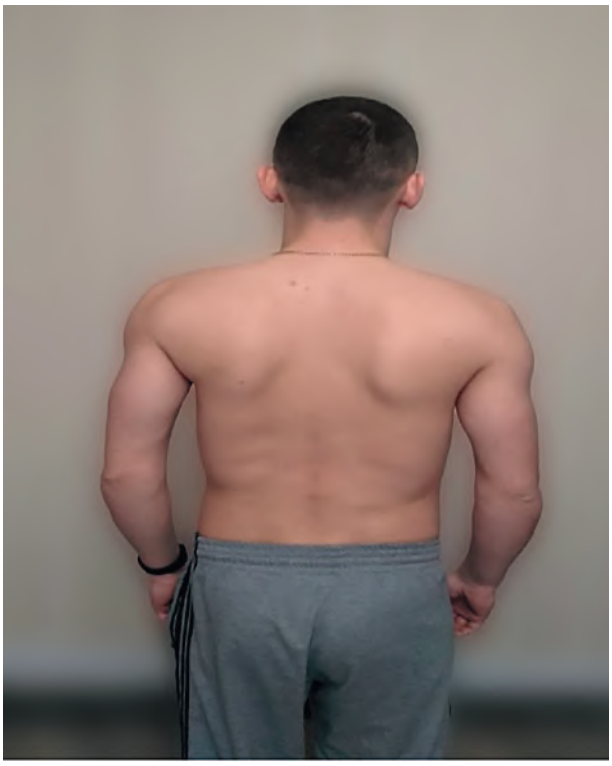

Figure 1 – Wing-like scapulae (2018).

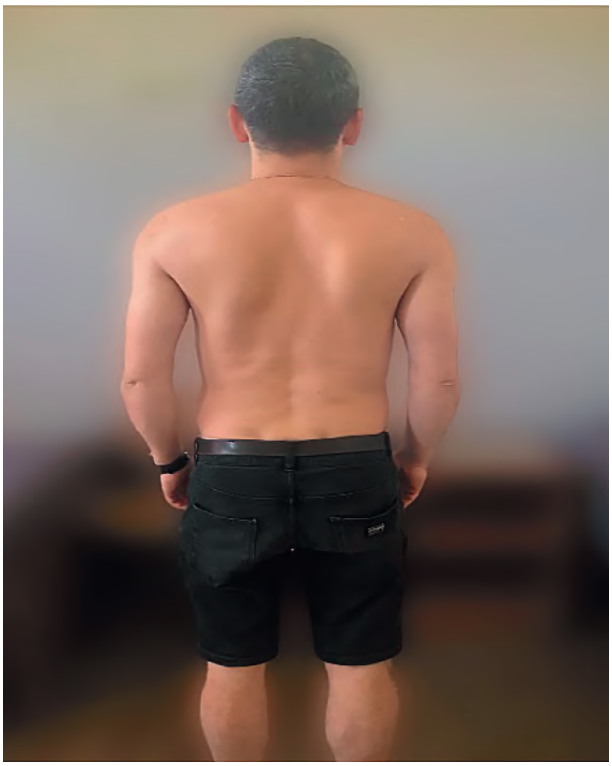

Figure 2 – Wing-like scapulae (2023).

minished to 4.0 points, while strength in the proximal lower extremities, including the thigh adductor muscles, hip flexors and extensors, and quadriceps, is reduced to 3.0 points. Muscle hypotrophy is evident in the scapular muscles, particularly on the right, resulting in "wing-like" scapulae **(fig. 1, 2)**. Additionally, hypotrophy affects proximal muscles of the extremities, with notable involvement of the biceps brachii **(fig. 3)** and right gluteus maximus **(fig. 4, 5)**. Pseudohypertrophy of the calf muscles is also observed **(fig. 6)**.

Arm reflexes: flexor-elbow D=S decreased, extensor-elbow D=S of moderate intensity, carpi radial D=S decreased; leg reflexes: knee D=S decreased, Achilles D=S of moderate intensity; abdominal reflexes D=S vigorous. No pathological reflexes were elicited. Both superficial and deep sensations are intact. The patient demonstrates satisfactory performance on coordination tests and maintains stability in Romberg's position. However, his gait is notably impaired, resembling a «duck-like» walk, and he experiences difficulties with activities such as climbing stairs and rising from a squatting position, with a positive Gowers' sign. There is no evidence of myotonic phenomena. Spinal examination reveals scoliotic curvature **(fig. 7)** and hyperlordosis of the lumbar spine **(fig. 8)**, with no signs of tension. Range of motion in the joints is preserved,

Figure 3 – Hypotrophy of the biceps brachii.

Figure 4 – Atrophy of the thigh muscles.

Figure 5 – Atrophy of the thigh muscles.

Figure 6 – Pseudohypertrophy of the calf muscles.

and there are no pelvic organ dysfunctions. The patient's cognitive functions are within normal limits.

Additional laboratory investigations revealed normal results for complete blood count, urine analysis, blood glucose, coagulation profile, thyroid hormones, troponin T, and D-dimer levels. However, the biochemical blood test showed elevated levels of creatine phosphokinase (6941.7 U/l), alanine aminotransferase (155.4 U/l), and aspartate aminotransferase (119.4 U/l), while other parameters remained within normal ranges.

Tests for alpha-galactosidase activity (Fabry disease) and alpha-1,4-glucosidase activity (Pompe disease) yielded normal findings.

Electrocardiography revealed sinus rhythm with a normal position of the electrical axis of the heart and a heart rate of 64 beats per minute, along with early ventricular repolarization syndrome.

Echocardiography demonstrated no enlargement of the heart cavities, preserved contractility of the left ventricular myocardium with a left ventricular ejection fraction of 57%, and unremarkable valves.

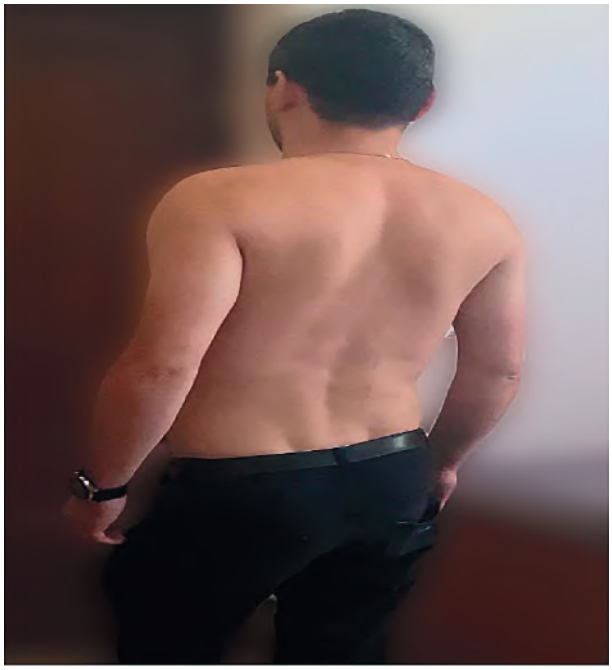

Figure 7 – Scoliosis of the spine.

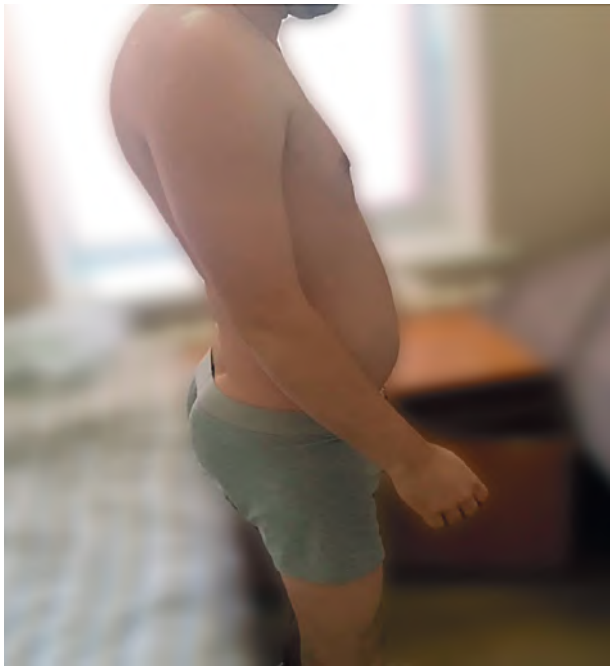

Figure 8 – Hyperlordosis of the lumbar spine.

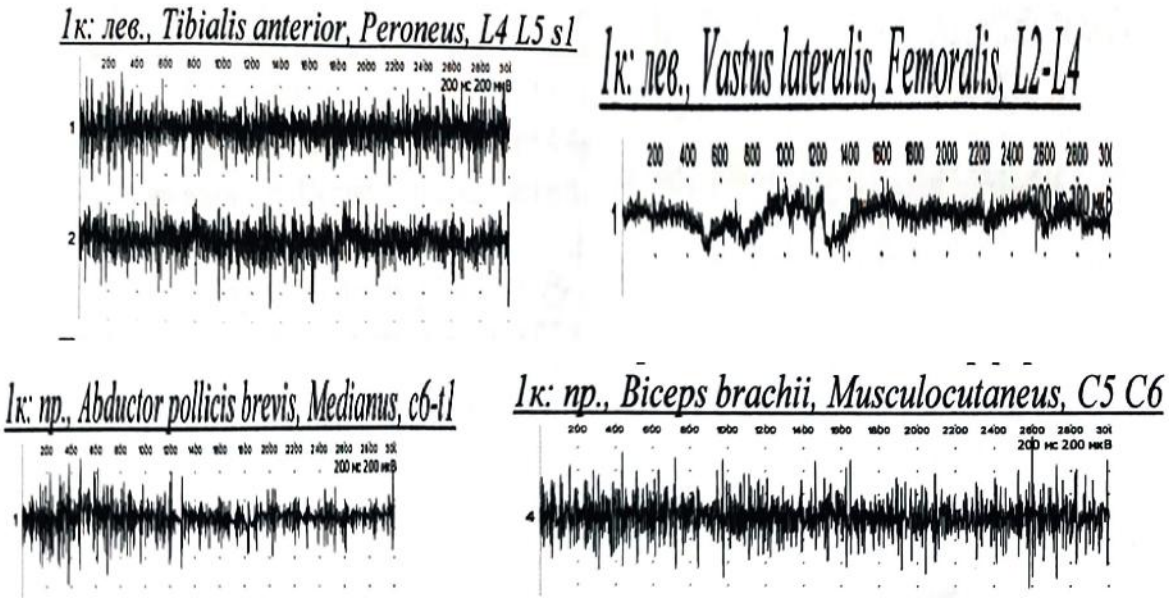

Figure 9 – Data from electromyography.

Electromyography of the upper and lower extremities indicated a primary muscular involvement of the pelvic and shoulder girdle muscles, characterized by changes in motor unit potentials consistent with a myopathic pattern. Additionally, there was evidence of impaired conduction along the peripheral nerves of both the upper and lower extremities, suggestive of a root nature of dysfunction **(fig. 9).**

MRI of the brain revealed no abnormalities in signal characteristics of the brain substance, with non-dilated ventricles and undisturbed midline structures. Convexional subarachnoid spaces were within normal limits, and satisfactory pneumatization was observed in the frontal sinuses, lattice bone, main sinuses, and temporal pyramids.

Following consultations with an endocrinologist and cardiologist, no evidence of endocrine pathology or cardiovascular disease was detected.

Based on the patient's clinical presentation, medical and family history, as well as results from instrumental and laboratory tests, the diagnosis of Limb-girdle muscular dystrophy, type 2A (calpainopathy) with peripheral proximal tetraparesis (mild in the arms, moderate in the legs), accompanied by a myopathic syndrome and moderate impairment of walking function, was established.

Further confirmation of the diagnosis occurred during consultation at the medical genetic center of the Municipal Enterprise "Regional Clinical Hospital for Rehabilitation Treatment and Diagnostics with Regional Centers for Family Planning and Human Reproduction, Medical Genetics of the Poltava Regional Council," where the diagnosis of limb-girdle muscular dystrophy, type 2A (calpainopathy), in the clinical form of Leyden-Möbius, was affirmed.

Treatment during the patient's stay in the neurological department included neurometabolic and antioxidant therapy, general strengthening measures, vitamin supplementation, and physiotherapy.

Discussion of the research results.

This observation reveals the main aspects of calpainopathies in the context of LGMD and emphasizes the need for timely diagnosis and genetic testing of this pathology in order to develop an individual treatment strategy [6, 26].

As one can observe, the history of the disease, objective and neurological examination play a leading role in diagnostics. It is necessary to pay attention to the time of onset of the disease [14], the symmetry of the involvement of the relevant muscle groups and the nature of walking [12], the presence of contractures in the joints and degenerative-dystrophic changes in the spine [20].

In our patient, the onset of the disease was at the age of 24, the symptom-complex that forms the clinical presentation consists of peripheral proximal tetraparesis, myopathic syndrome, which coincides with the literature data on the debut and course of calpainopathy, Leyden-Möbius type [1, 4, 10, 12, 15].

Laboratory and instrumental research methods help confirm the diagnosis. According to the literature, a significant increase in serum creatine phosphokinase is detected in calpainopathy, indicating a dystrophic process in the muscles. The activity of other «muscle enzymes» (alanine aminotransferase, aspartate aminotransferase, lactate dehydrogenase) also increases [27]. We observed these changes in the form of increased creatine phosphokinase, aspartate aminotransferase and alanine aminotransferase in our patient.

The electromyogram of our patient shows characteristic changes in the potential of motor units of the myopathic type, which confirms the primary lesion of the pelvic and shoulder girdle muscles (these changes are characteristic of myopathies) and is consistent with the literature data on calpainopathy. Biopsy of the affected muscles can reveal dystrophic and denervation changes and the presence of a specific mutant protein that disrupts its function [28, 29].

However, for the effective management of this pathology, further research on the molecular mechanisms of calpainopathy is needed to develop new therapeutic approaches and improve the quality of life of patients with this disease [10, 13, 14].

Conclusions.

At present, there is no specific treatment for calpainopathy, therefore, therapy is symptomatic, aimed at normalizing the patient's general condition, restoring lost functions, and possibly slowing the rate of progression. Dynamic monitoring of the patient and timely management of symptoms increases the possibility of extending life expectancy and improving its quality.

Prospects for further research.

Identification and analysis of clinical cases of LGMD 2A in order to improve diagnostic, therapeutic and rehabilitation measures.

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КІНЦІВКОВО-ПОЯСНА М'ЯЗОВА ДИСТРОФІЯ, ТИП 2А (КАЛЬПАЇНОПАТІЯ): ВЛАСНЕ КЛІНІЧНЕ СПОСТЕРЕЖЕННЯ

Сингаївський А. М., Дельва М. Ю., Пурденко Т. Й., Пушко О. О., Таряник К. А., Силенко Г. Я., Гринь К. В.

Резюме. Метою дослідження було представлення клінічного випадку пацієнта з кальпаїнопатією з метою привертання уваги у сімейних лікарів, неврологів, фізичних терапевтів, педіатрів, травматологів та інших спеціалістів.

Об'єкт і методи. Скарги, анамнез захворювання і життя, об'єктивний та неврологічний огляд, лабораторні, нейровізуалізаційні та нейрофізіологічні методи дослідження

Результати. Пацієнт А., 29 років, при госпіталізації скаржився на слабкість у кінцівках, особливо в ногах, схуднення м'язів плечей, сідниць і стегон, збільшення в об'ємі гомілок, виражене порушення ходьби, труднощі при підйомах сходами та при вставанні з ліжка. Хворіє близько 5-ти років, коли почав відмічати слабкість у проксимальних м'язах ніг, яка поступово наростала; близько 1,5 роки тому з'явилась слабкість у проксимальних м'язах рук. Останні 2-ва роки відмічає прогресуючу зміну ходи. За медичною допомогою звернувся вперше. Хронічні захворювання заперечує. Рухові порушення в дитинстві не визначались. Відомо, що батько та мати пацієнта є родичами у третьому поколінні. У неврологічному статусі: зниження м'язового тонусу в проксимальних відділах кінцівок, переважно в ногах. Проба Барре «+» нижня. Сила в проксимальних відділах верхніх кінцівок, а саме у дельтоподібних м'язах та біцепсах, знижена до 4,0 балів. Сила в проксимальних відділах нижніх кінцівках, а саме, у привідних м'язах стегна, згиначах та розгиначах стегна, квадрицепсах знижена до 3,0-х балів. М'язова гіпотрофія лопаткових м'язів, більше справа, з формуванням «крилоподібних (виступаючих) лопаток». Також відмічається м'язова гіпотрофія проксимальних м'язів кінцівок із переважним ураженням біцепсів плечей, сіднично-стегнових м'язів, більше справа. Псевдогіпертрофія литкових м'язів. Чутливість збережена. Хода утруднена, «качина». Відмічаються труднощі при підніманні сходами, при вставанні після присідання, позитивний симптом Говерса. Сколіотичні явища хребта, гіперлордоз поперекового відділу хребта. Когнітивні функції в нормі. У біохімічному аналізі крові: значне підвищення креатинфосфокинази, аланінамінотрансферази та аспартатамінотрансферази, інші показники – в межах норми. МРТ головного мозку: речовина головного мозку без змін сигнальних характеристик. На електронейроміограмі виявлено характерні зміни потенціалу рухових одиниць за міопатичним типом. Аналізи на активність альфа-галактозидази та альфа-1,4-глюкозидази: норма. Консультація ендокринологом та кардіологом: даних за патологію на час огляду не виявлено. Медичним генетиком поставлений діагноз: кінцівково-поясна м'язова дистрофія, тип 2А (кальпаїнопатія).

Враховуючі вищеописані дані пацієнту встановлено діагноз: Кінцівково-поясна м'язова дистрофія, тип 2А (кальпаїнопатія) з периферичним проксимальним тетрапарезом (легким у руках, помірним у ногах), міопатичним синдромом, помірним порушенням функції ходьби.

Пацієнту було проведено нейрометаболічну, антиоксидантну, загальнозміцнювальну терапії, вітамінотерапію та фізіотерапевтичне лікування.

Висновки. На сьогодні не існує специфічного лікування кальпаїнопатії, тому терапія має симптоматичний характер, направлена на нормалізацію загального стану пацієнта, відновлення втрачених функцій та ймовірне сповільнення швидкості прогресування. А динамічне спостереження за пацієнтом і вчасний менеджмент симптомів поліпшує можливість продовження тривалості життя та покращення його якості.

Ключові слова: кінцівково-поясна м'язова дистрофія, кальпаїнопатія, діагностика, лікування.

LIMB-GIRDLE MUSCULAR DYSTROPHY, TYPE 2A (CALPAINOPATHY): CLINICAL OBSERVATION

Synhaivskyi A. M., Delva M. Yu., Purdenko T. Y., Pushko O. O., Tarianyk K. A., Sylenko H. Ya., Hryn K. V.

Abstract. The aim of the study was to present a clinical case of calpainopathy, intending to capture the attention of a broad spectrum of medical professionals including family doctors, neurologists, physical therapists, pediatricians, traumatologists, and other specialists.

Object and methods. We conducted a comprehensive assessment including patient complaints, medical and life history, objective and neurological examinations, as well as utilizing laboratory analyses, neuroimaging, and neurophysiological methodologies.

Results. Patient A, aged 29, presented with complaints of limb weakness, predominantly in the legs, accompanied by weight loss in the muscles of the shoulders, buttocks, and thighs. Additionally, he reported increased volume of the lower legs, significant difficulty in walking, climbing stairs, and rising from bed. Symptoms had manifested approximately five years prior, initially with weakness in the proximal leg muscles, progressively worsening over time. About 1.5 years ago, weakness extended to the proximal arm muscles. Within the last two years, a noticeable alteration in gait emerged. Seeking medical attention for the first time, he denied any chronic conditions and had no history of childhood motor impairments. Neurological examination revealed reduced muscle tone in the proximal limbs, particularly the legs, along with a lower positive Barre test. Strength assessments indicated diminished muscle power, graded at 4.0 in the proximal upper extremities (deltoid and biceps muscles) and 3.0 in the proximal lower extremities (adductor muscles of the thigh, hip flexors and extensors, and quadriceps). Muscle hypotrophy was observed in the scapular muscles, more pronounced on the right side, resulting in wing-like scapulae. Additionally, there was hypotrophy in the proximal limb muscles, notably affecting the biceps brachii and gluteus maximus, predominantly on the right side, accompanied by pseudohypertrophy of the calf muscles. Sensory functions remained intact. The patient's gait was described as challenging, «duck-like», with difficulties in stair climbing and rising from a squatting position, along with a positive Gowers' sign. Spinal examination revealed scoliotic features and hyperlordosis of the lumbar spine. Cognitive functions were not impaired. Biochemical blood analysis revealed elevated levels of creatine phosphokinase, alanine aminotransferase, and aspartate aminotransferase, while other parameters remained within normal limits. Brain MRI showed no abnormalities in signal characteristics. Electroneuromyography demonstrated characteristic changes indicative of myopathic motor unit potentials. Tests for alpha-galactosidase and alpha-1,4-glucosidase activity returned normal results. Consultations with an endocrinologist and cardiologist revealed no pathological findings. A medical geneticist ultimately diagnosed the patient with limb-girdle muscular dystrophy, type 2A, commonly known as calpainopathy.

Taking into account the above data, the patient was diagnosed with the following: Limb-girdle muscular dystrophy, type 2A (calpainopathy) with peripheral proximal tetraparesis (mild in the arms, moderate in the legs), myopathic syndrome, moderate impairment of walking function.

The patient underwent neurometabolic, antioxidant, general strengthening therapy, vitamin therapy and physiotherapy.

Conclusions. To date, there is no specific treatment for calpainopathy, therefore, therapy is symptomatic, aimed at normalizing the patient's general condition, restoring lost functions and possibly slowing the rate of progression. Dynamic monitoring of the patient and timely management of symptoms improves the possibility of extending life expectancy and improving its quality.

Key words: limb-girdle muscular dystrophy, calpainopathy, diagnosis, treatment.

ORCID and contributionship:
Synhaivskyi A. M.: https://orcid.org/0009-0001-1461-6899 ABDE
Delva M. Yu.: https://orcid.org/0000-0001-5648-7506 AEF
Purdenko T. Y.: https://orcid.org/0000-0002-3561-4331 ABDE
Pushko O. O.: https://orcid.org/0000-0001-7309-4798 AEF
Tarianyk K. A.: https://orcid.org/0000-0003-4606-5398 DE
Sylenko H. Ya.: https://orcid.org/0000-0002-6225-0174 BD
Hryn K. V.: https://orcid.org/0000-0002-8759-3560 BD

Conflict of interest:
The Authors declare no conflict of interest.

Corresponding author
Synhaivskyi Andrii Mykhailovych
Municipal Enterprise «M.V. Sklifosovskyi Poltava Regional Clinical Hospital of the Poltava Regional Council»
Ukraine, 36000, Poltava, 23 Shevchenko str.
Tel.: +380931351234
E-mail: s000572@vnmu.edu.ua

A – Work concept and design, **B** – Data collection and analysis, **C** – Responsibility for statistical analysis, **D** – Writing the article, **E** – Critical review, **F** – Final approval of the article.

Received 13.01.2024
Accepted 01.05.2024

DOI 10.29254/2077-4214-2024-2-173-253-262
UDC 616.12-008.331.1:616.1-06]-085.225

Stanovska L. V., Krynytska I. Ya.

ASSOCIATION OF ACHIEVEMENT/FAILURE OF THE TARGET LEVEL OF BLOOD PRESSURE AND ADHERENCE TO ANTIHYPERTENSIVE TREATMENT IN OUTPATIENTS WITH ISOLATED ARTERIAL HYPERTENSION AND IN COMBINATION WITH CARDIOVASCULAR COMORBIDITY

I. Horbachevsky Ternopil National Medical University (Ternopil, Ukraine)
krynytska@tdmu.edu.ua

This study aimed to assess adherence to antihypertensive treatment in outpatients with isolated hypertension and cardiovascular comorbidity and to determine the possible relationship between achievement/non-achievement of target blood pressure (TBP) and adherence to antihypertensive treatment. The study included 140 outpatients with hypertension who were divided into three groups: isolated hypertension (n=60), hypertension + coronary heart disease (CHD) (n=35), and hypertension + CHD + chronic heart failure (CHF) (n=45). A high proportion of people with low adherence to antihypertensive treatment was found among outpatients with both isolated hypertension and hypertension and cardiovascular comorbidity (in the group with isolated hypertension - 60.00%; in the group with hypertension + CHD - 65.71%; in the group with hypertension + CHD + CHF - 66.67%). At the same time, in the group of hypertension + CHD, 69.57% of patients with low adherence to treatment were female (p=0.030)). When analysing the relationship between achievement/non-achievement of TBP and adherence to antihypertensive treatment, statistically significant differences were found both in patients with isolated hypertension and patients with hypertension in combination with cardiovascular comorbidity. Thus, the proportion of patients with low adherence to antihypertensive treatment among patients who did not achieve TBP was 76.92% (p<0.001) in the group with isolated hypertension and 87.50% in the group with hypertension+CHD (p=0.030).

Key words: *arterial hypertension, cardiovascular comorbidity, target blood pressure level, adherence to antihypertensive treatment.*