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CASE REPORT

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Multiminicore disease as an important cause of toe walking: case report and literature review

ABSTRACT

Toe walking belongs to gait deformities, which are challenging in diagnosing the underlying disease. Among different neurological and developmental causes of toe walking, one must remember congenital myopathies, among which multiminicore disease (MmD) represents a significant percentage and may cause life-threatening situations in the process of surgical treatment. Based on the presented case and a literature review, the spectrum of this particular congenital myopathy, the importance of muscle biopsy in the diagnostic process of toe walkers, and the risk concerning anaesthesia in children suspected of congenital myopathies are presented.

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Key words: toe walking; malignant hyperthermia; muscle myopathy; vulpius procedure; muscle biopsy

INTRODUCTION

Toe walking is a gait abnormality whereby equinus deformity of the foot is present during most or all of the phases of a gait cycle [1–3]. Its idiopathic form is characterised by the lack of definitive aetiology [1, 2]; however, in most cases, it affects children who present the characteristic gait pattern from the outset of walking. Even more puzzling are toe walkers who develop this disorder at preschool or school age. An accurate diagnosis is crucial in these cases to help reduce the anxiety of parents and child. The various neurological and developmental causes of toe walking include congenital myopathies, of which multiminicore disease (MmD) is one of the examples. We present the case of a young male patient with a progressive form of toe walking and scoliosis who was diagnosed with MmD. Based on this case, we elucidate the spectrum of this particular congenital myopathy, the importance of muscle biopsy when searching for the reasons behind idiopathic toe walking, and the risks concerning the use of anaesthesia in children suspected of having a congenital myopathy.

CASE PRESENTATION

A 13-year-old boy with MmD was admitted to our department with progressive toe walking accompanied by scoliotic deformity of the spine. Medical history revealed that the first symptoms were noted by the boy's mother when he was 5 years old. In 2010, neurological and orthopaedic examination revealed essential tremors that escalated after emotional excitement, flattening of the back and bilateral hamstrings, and digastric hypertension. Psychological consultation divulged attention-deficit disorder. At that time, the patient presented obvious difficulties with climbing stairs and tiptoeing, and minor tremor abnormalities were confirmed on electroencephalogram (EEG). Conservative treatment lasting 18 months did not diminish the symptoms. The child's psychomotor development did not

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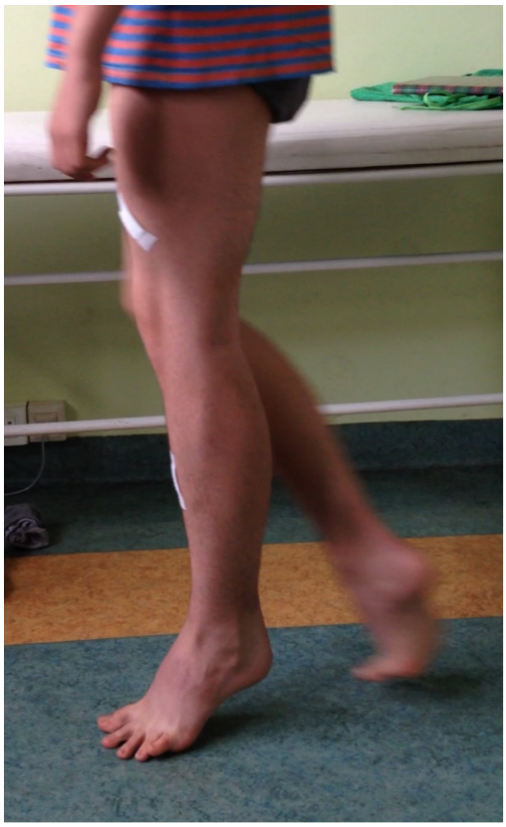

Figure 1. Toe walking in the clinical setting

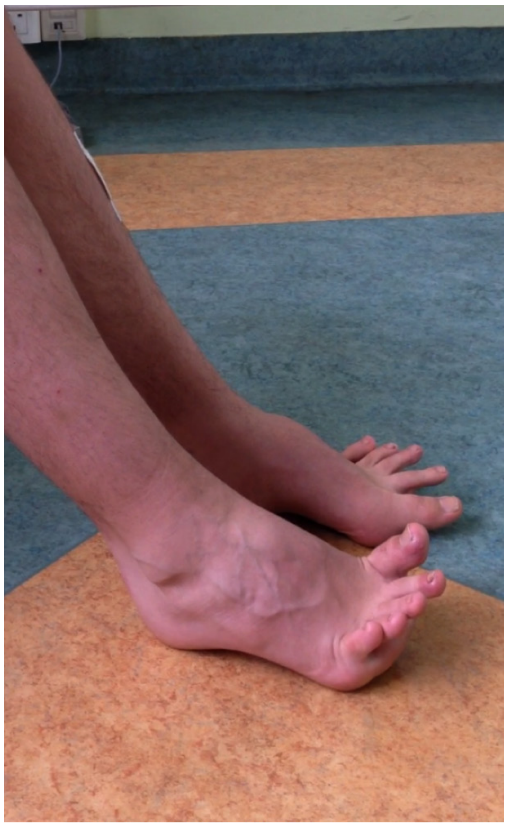

Figure 2. Assessment of selectivity of movement around the foot. While checking the selectivity of dorsiflexors of the foot (left and right), the patient presented simultaneous dorsiflexion in the contralateral foot as proof of decreased selectivity

deviate from the average (e.g., he started to sit independently at 6 months and walk independently at 12 months of age).

A thorough clinical evaluation of the patient revealed numerous clinical signs: bilateral equinus gait (Fig. 1), deficit of dorsiflexion of the foot in the ankle joint in both lower limbs, decreased selectivity of the foot and flexors and extensors of phalanges (Fig. 2), thoracic scoliosis with shallowing of the spinal curvature in the sagittal plane, muscle force asymmetry with distinctive lower limb muscle weakness, dominance of back extensors relative to abdominal muscularity, and neurological signs (tremor in the upper limbs at rest, decreased knee and Achilles reflexes).

Laboratory tests showed a significantly increased creatine kinase level (exceeding triple the norm). In contrast, electromyography (EMG) examination showed good conduction in peroneal, tibial, and femoral nerves and neurogenic disorders in vastus lateralis bilaterally. Genetic testing excluded Duchenne and Becker dystrophies.

Based on the whole history, clinical examination, and additional diagnostic tests, the patient was scheduled for surgical management of toe walking consisting of gastrocsoleus recession with gastrocnemius muscle biopsy. He was also referred for brace treatment of the scoliosis. During surgery, the patient developed bradypnoea with bradycardia for unknown reasons (he underwent a spinal block with no muscle relaxants or anaesthetic gases). In the postoperative period, he suffered delusions and hallucinations, which resolved within 1 day. Postoperative neurological assessment did not reveal any other serious abnormalities.

Histopathological evaluation with optical and electron microscopy confirmed MmD with the partial involvement of muscle fibres (detailed below).

During the 18-month post-surgical follow-up, the patient's gait improved significantly, while the use of a brace slowed the progression of spinal deformity.

HISTOPATHOLOGICAL FINDINGS

Using routine light and transmission electron microscopy (TEM) we evaluated the muscle biopsy taken from the gastrocnemius muscle of the presented patient. The samples, which were oriented transversely to the fibers' long axis, showed fibers of thinner diameter.

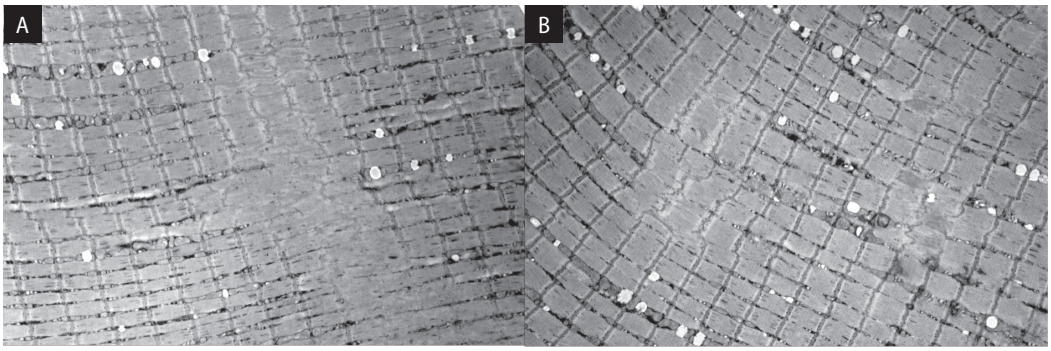

Figure 3. Image of muscle fibers in electron microscopy (magnification 7500x). **A.** In the presented here muscle fiber, abnormalities of the "Z" band are seen in 9 sarcomeres located in the central area of the cell. "Z" bands are broadened and irregular, and some impinge on the structure of all bands forming the sarcomere; **B.** This muscle fiber has centrally located abnormal sarcomeres. Part of it has the "Z" band changes described above. The loss of all typical sarcomere structures without "Z" bands is also located centrally

Some of the thinner fibers had centrally located nuclei. In several other cells, irregularities of the sarcomere structure were spread throughout different parts of the cell's body.

The syndrome is characterized by the presence of specific structural abnormalities of "Z" bands, which distinguish this disorder from other myopathies. The sarcomeres are thinner, grouped few together, and located between those with regular structures.

"Z" bands were broadened, irregular, and cogged, occupying spaces of other bands. The aforementioned lesions were present only in a small number of the muscle cells (Fig. 3).

Table 1. Summary of common factors causing toe walking

Etiological factors of toe walking
Cerebral palsy
Congenital muscular dystrophy (e.g. Becker, Duchenne)
Congenital myopathies
Limb-length discrepancy
Real idiopathic cases (shortening of the calcaneal tendon)
Spina bifida
Global developmental delay
Charcot-Marie-Tooth disease

DISCUSSION

Patients presenting with progressive toe walking are relatively common in paediatric orthopaedic practice. While the natural history and optimal treatment for early-onset idiopathic toe walkers are poorly documented [4], recommendations for patients with late-onset symptomatic equinus during walking treatment are even less established [5]. Most of these patients will be suspected of one of the forms of muscular dystrophy. However, based on the other associated signs, differentiation could be made between muscular dystrophies and other neuromuscular disorders. The summary of the most common etiological factors of toe walking is found in Table 1.

We present the case in which MmD was diagnosed based on a biopsy in a patient presenting with the problem of progressive foot drop while walking. It may seem to be an accidental finding. However, since we began to take samples for electron microscopy (EM) examination while doing Achilles tendon lengthening procedures (since 2014) in this group of patients, we have found already three patients with congenital myopathies, including two with MmD and one diagnosed with central core disease (CCD). Taking into account the fact that less than 10 patients have been operated on because of toe walking in our department between the years 2014–2016 (excluding congenital neurological deficits such as cerebral palsy or spina bifida as well as congenital limb deformities such as clubfoot), the percentage of patients with congenital myopathies is considerable.

MmD, according to the Online Mendelian Inheritance in Man (OMIM) web base, is an inherited neuromuscular disorder defined pathologically by the presence of multiple areas of reduced mitochondrial oxidative activity running along a limited extent of the longitudinal axis of the muscle fiber, so-called "minicores" [6]. As it is a neuromuscular disorder, it is characterised by multiple orthopaedic manifestations. All the orthopaedic clinical signs and full manifestations of the disease are summarised in Table 2.

Table 2. Summary of orthopaedic manifestations of the multiminicore disease (MmD) (with their references)

Orthopaedic clinical signs	References
Proximal and axial muscles group involvement	Ferreiro [7, 13], Jungbluth [8, 11, 15], Zhou [12], Osada [14], Sharma [16], Estournet [17], Cullup [18], Kava [19], Jungbluth [22], Jungbluth [23], Monnier [24, 25]
Scoliosis	Ferreiro [7, 13], Jungbluth [8, 11, 15], Zhou [12], Osada [14], Sharma [16], Estournet [17], Cullup [18], Trevesa [21], Jungbluth [22, 23], Monnier [24, 25]
Spinal rigidity	Jungbluth [8, 11, 15], Ferreiro [13], Cullup [18]
Kyphosis, hyperlordosis	Jungbluth [8, 15], Ferreiro [13]
Joints hyperlaxity	Ferreiro [7], Zhou [12], Jungbluth [8, 15], Trevesa [21], Monnier [25]
Distal weakness, predominantly affecting the hands (intrinsic muscles)	Jungbluth [8, 15], Zhou [12], Osada [14], Sharma [16], Cullup [18], Mitsuhashi [20]
Patellar and knee luxation	Ferreiro [7]
General arthrogryposis	Ferreiro [7], Jungbluth [11, 15], Sharma [16], Trevesa [21], Monnier [25]
Presence of tightened joints (hip, knee, ankle, elbow, wrist and finger)	Ferreiro [7, 13], Cullup [18], Monnier [25]
Toe walking	Jungbluth [15], Mitsuhashi [20]
Club feet	Jungbluth [8, 15]
Thorax deformity	Ferreiro [7, 13]
Achilles contractures	Jungbluth [8, 15], Cullup [18]
Muscles pain	Cullup [18]

Table 3. Comparison of patient's clinical signs with those typical for multiminicore disease (MmD)

Patient's clinical symptoms	Typical signs for MmD
Late onset	Early onset
Toe walking	Toe walking is a rare sign, feet deformations (club feet) after birth
Distal muscles weakness	Axial and proximal muscles weakness
Scoliosis with thoraco-lumbar junction kyphotic deformity,	Scoliosis, hyperlordosis, hyperkyphosis and other spinal deformity problems
Bilateral vastus lateralis muscles neurogenic impairment	Muscles neurogenic impairment is typical symptom
Intention tremor, tremor at rest	Only one case with essential tremor
Bradycardia with bradypnoe during the surgery	Malignant hyperthermia
Increased level of creatine kinase	Normal levels of creatine kinase

Although four typical patterns of MmD have been described recently (classic form; moderate form, with hand involvement; antenatal form, with arthrogryposis multiplex congenita; ophthalmoplegic form), none of these matched the spectrum of clinical signs in presented here patients [7–10].

MmD disease is predominantly recognizable by its axial and proximal muscle weakness with hypotonia, respiratory difficulty, high occurrence of scoliosis, and absence of marked limb contractures in patients who develop signs mainly at birth or during infancy [7–10]. In the presented case, the patients main complaints were: progressive deformation of the gait expressed by toe walking with fixed equinus contracture and progressive scoliotic deformation of the spine. The axial muscles presented better function than the peripheral ones, which is different from previously described [7, 8, 11–19]. What is remarkable is that difficulties with walking and other clinical signs were noticed by the child and his parents no sooner than the seventh year of life, while in the literature, in most cases, especially in the classic form of MmD, first signs appear in the newborn or infants. Cases of MmD with foot equines deformity have been recently described by Cullup et al. and Mitsuhashi et al.; however, based on the reviews of most of the cases of MmD published, it is not a typical sign of the disease [15, 18, 20]. On the contra-

ry, the presence of the spine deformity, which was diagnosed in our patient, is classified as one of the characteristics of the disease [7, 8, 11–18, 21–24]. Additionally, rest and intention tremors were noted while a thorough clinical examination had been carried out (although very slightly expressed), which, to our knowledge, has not been yet described in any of the patterns of the disease.

Typical for patients with MmD, high levels of creatine phosphokinase (over 800 IU/L, N: 55–370 IU/L) are present, while other laboratory findings, including aminotransferases and alkaline phosphatase, are in the normal range.

What requires a mention is the fact that during anaesthesia the, episode of bradycardia and respiratory disorders occurred in our patient. MmD is linked with malignant hyperthermia, which is a condition that develops due to a hypercatabolic state. It presents as a very high temperature, tachycardia, and hyperpnoe (with increased carbon dioxide production and increased oxygen consumption), mixed acidosis, muscle rigidity, and rhabdomyolysis [26, 27]. Typically this condition may be triggered by certain groups of drugs used commonly in general anaesthesia, namely volatile anesthetic gases such as halothane, sevoflurane, desflurane, isoflurane, or enflurane, as well as the depolarizing muscle relaxants — suxamethonium and decamethonium [25, 27]. However, in our patient, none of those mentioned above drugs had been used because he was anaesthetised with the use of the spinal (subarachnoid) block. Moreover, the signs he presented during and after the episode did not match the features of malignant hyperthermia. The neurological consultation, magnetic resonance imaging (MRI) examination, and laboratory diagnostics did not find the cause of this kind of disorder. Errors during anaesthesia had also been ruled out. Thus, the answer to the reason for the episode of bradycardia remains unknown. This should, however, lead us to take extraordinary measures while introducing anaesthesia to the patient with recognized or suspected congenital myopathies or dystrophies.

MmD is linked to mutations in particular genes: the RYR1 and SEPN1. Still, our patient has yet to be tested genetically. However, it is questionable whether patients with one of the congenital myopathies diagnosed based on the histopathologic (including EM) findings need to run expensive genetic tests to confirm the diagnosis. We believe that only in doubtful cases must the full panel of gene mutations be checked.

Muscle biopsy is an inestimable method of providing a definitive diagnosis of a wide range of myopathies and denervating disorders and gives essential information on the stage and course of the disease, methods of treatment, as well as prognosis [28]. In most of these cases, EM is an inseparable part of the examination in the process of identifying the disorder. The main surgical techniques used for fixed equinus of the foot are Z-plasty of the Achilles tendon or different fashions of the gastrocnemius muscle recession [29–31]. In each lengthening technique, accessibility to the gastrocnemius or soleus muscle belly is sufficient to reach it using a clamp or grasper. However, one must remember that the biopsy site in these cases is far from the insertion of these muscles, which results in a small amount of nerve branches in the samples. In cases of an unclear histopathological diagnosis, the surgeon may be obligated to take a small-sized biopsy of the skin from the insertion site. We recommend doing a biopsy and lengthening procedures in one stage, not as separate procedures.

CONCLUSIONS

1. Patients presenting with either early or late onset toe walking of a progressive nature should be checked for the presence of congenital myopathies.
2. If MmD is suspected, all precautions must be taken during the surgical procedures, especially from the anaesthesiology team, since there is a risk of malignant hyperthermia reactions and others of unknown character.
3. Muscle biopsy for EM examination is an integral part of the follow-up to surgical muscle lengthening procedures in cases where the final diagnosis has not been established.

ETHICS STATEMENT

On behalf of, and having obtained permission from all the authors, we declare that the material has not been published in whole or in part elsewhere; the paper is not currently being considered for publication elsewhere; and all relevant ethical safeguards have been met in relation to patient or subject protection, or animal experimentation. We testify to the accuracy of the above on behalf of all the authors.

AUTHOR CONTRIBUTIONS

B.M. proposed the case description and the literature review, searched through the literature, revised patient's data and summarized them, participated in interpreting the results, and drafted the manuscript. M.P. created the research design, searched through the literature, collected patient data, participated in interpreting results, and drafted the manuscript. M.S. collected patient data, took part in the interpretation of results, and revised the manuscript critically. M.R. took part in drafting the manuscript. M.J. took part in interpreting the results and revised the manuscript critically. All authors read and approved the final version of the article.

CONFLICT OF INTEREST

We declare no known conflicts of interest associated with this publication, and there has been no financial support for this work that could have influenced its outcome. We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further confirm that all have approved the order of authors listed in the manuscript. We prove that we have given due consideration to the protection of intellectual property associated with this work and that there are no impediments to publication, including the timing of publication, concerning intellectual property. In so doing we confirm that we have followed the regulations of our institutions concerning intellectual property.

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None declared.

CONSENT

The patient has given their informed consent for the case report to be published.

References

1. Pomarino D, Ramírez Llamas J, Pomarino A. Idiopathic Toe Walking: Tests and Family Predisposition. Foot Ankle Spec. 2016; 9(4): 301–306, doi: 10.1177/1938640016630056, indexed in Pubmed: 26872463.
2. Oetgen ME, Peden S. Idiopathic toe walking. J Am Acad Orthop Surg. 2012; 20(5): 292–300, doi: 10.5435/JAAOS-20-05-292, indexed in Pubmed: 22553101.
3. Jóźwiak M, Ławniczak D, Manikowska F, et al. [Idiopathic toe walking (ITW)--normal or pathological?]. Chir Narzadow Ruchu Ortop Pol. 2010; 75(1): 24–29, indexed in Pubmed: 20496774.
4. Dietz F, Songsak K. Idiopathic Toe Walking: To Treat or not to Treat, that is the Question. Iowa Orthop J. 2012; 32: 184–188.
5. Scher DM, Mubarak SJ. Surgical prevention of foot deformity in patients with Duchenne muscular dystrophy. J Pediatr Orthop. 2002; 22(3): 384–391, indexed in Pubmed: 11961461.
6. Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. MIM Number: #255320: 22/05/2015. http://omim.org/.
7. Ferreiro A, Estournet B, Chateau D, et al. Multi-minicore disease--searching for boundaries: phenotype analysis of 38 cases. Ann Neurol. 2000; 48(5): 745–757, indexed in Pubmed: 11079538.
8. Jungbluth H, Sewry C, Brown SC, et al. Minicore myopathy in children: a clinical and histopathological study of 19 cases. Neuromuscul Disord. 2000; 10(4-5): 264–273, doi: 10.1016/s0960-8966(99)00125-x, indexed in Pubmed: 10838253.
9. Ferreiro A, Fardeau M. 80th ENMC International Workshop on Multi-Minicore Disease: 1st International MmD Workshop. 12-13th May, 2000, Soestduinen, The Netherlands. Neuromuscul Disord. 2002; 12(1): 60–68, doi: 10.1016/s0960-8966(01)00237-1, indexed in Pubmed: 11731287.
10. Nadaj-Pakleza A, Fidziańska A, Ryniewicz B, et al. Multi-minicore myopathy: a clinical and histopathological study of 17 cases. Folia Neuropathol. 2007; 45(2): 56–65, indexed in Pubmed: 17594595.
11. Jungbluth H. Multi-minicore Disease. Orphanet J Rare Dis. 2007; 2: 31, doi: 10.1186/1750-1172-2-31, indexed in Pubmed: 17631035.
12. Zhou H, Lillis S, Loy RE, et al. Multi-minicore disease and atypical periodic paralysis associated with novel mutations in the skeletal muscle ryanodine receptor (RYR1) gene. Neuromuscul Disord. 2010; 20(3): 166–173, doi: 10.1016/j.nmd.2009.12.005, indexed in Pubmed: 20080402.
13. Ferreiro A, Quijano-Roy S, Pichereau C, et al. Mutations of the selenoprotein N gene, which is implicated in rigid spine muscular dystrophy, cause the classical phenotype of multiminicore disease: reassessing the nosology of early-onset myopathies. Am J Hum Genet. 2002; 71(4): 739–749, doi: 10.1086/342719, indexed in Pubmed: 12192640.
14. Osada H, Masuda K, Seki K, et al. Multi-minicore disease with susceptibility to malignant hyperthermia in pregnancy. Gynecol Obstet Invest. 2004; 58(1): 32–35, doi: 10.1159/000077658, indexed in Pubmed: 15056993.
15. Jungbluth H, Beggs A, Bönnemann C, et al. 111th ENMC International Workshop on Multi-minicore Disease. 2nd International MmD Workshop, 9-11 November 2002, Naarden, The Netherlands. Neuromuscul Disord. 2004; 14(11): 754–766, doi: 10.1016/j.nmd.2004.07.007, indexed in Pubmed: 15482962.
16. Sharma MC, Gulati S, Sarkar C, et al. Multi-minicore disease: a rare form of myopathy. Neurol India. 2007; 55(1): 50–53, doi: 10.4103/0028-3886.30427, indexed in Pubmed: 17272900.

17. Estournet B, Barois A, Bataille J. Multi minicore disease myopathy: 20 cases. Neuromus Disord. 1997; 7(6-7): 431, doi: 10.1016/s0960-8966(97)87180-5.
18. Cullup T, Lamont PJ, Cirak S, et al. Mutations in MYH7 cause Multi-minicore Disease (MmD) with variable cardiac involvement. Neuromuscul Disord. 2012; 22(12): 1096–1104, doi: 10.1016/j.nmd.2012.06.007, indexed in Pubmed: 22784669.
19. Kava MP, Hawkins C, Millan L, et al. C.P.5 Three siblings with multi-minicore myopathy not associated with SEPN1 or RYR1 mutations. Neuromusc Disord. 2012; 22(9-10): 842, doi: 10.1016/j.nmd.2012.06.133.
20. Mitsuhashi S, Nonaka I, Wu S, et al. Distal myopathy in multi-minicore disease. Intern Med. 2009; 48(19): 1759–1762, doi: 10.2169/internalmedicine.48.2425, indexed in Pubmed: 19797833.
21. Treves S, Anderson AA, Ducreux S, et al. Ryanodine receptor 1 mutations, dysregulation of calcium homeostasis and neuromuscular disorders. Neuromuscul Disord. 2005; 15(9-10): 577–587, doi: 10.1016/j.nmd.2005.06.008, indexed in Pubmed: 16084090.
22. Jungbluth H, Zhou H, Hartley L, et al. Minicore myopathy with ophthalmoplegia caused by mutations in the ryanodine receptor type 1 gene. Neurology. 2005; 65(12): 1930–1935, doi: 10.1212/01.wnl.0000188870.37076.f2, indexed in Pubmed: 16380615.
23. Monnier N, Marty I, Faure J, et al. Null mutations causing depletion of the type 1 ryanodine receptor (RYR1) are commonly associated with recessive structural congenital myopathies with cores. Hum Mutat. 2008; 29(5): 670–678, doi: 10.1002/humu.20696, indexed in Pubmed: 18253926.
24. Monnier N, Ferreiro A, Marty I, et al. A homozygous splicing mutation causing a depletion of skeletal muscle RYR1 is associated with multi-minicore disease congenital myopathy with ophthalmoplegia. Hum Mol Genet. 2003; 12(10): 1171–1178, doi: 10.1093/hmg/ddg121, indexed in Pubmed: 12719381.
25. Rosenberg H, Davis M, James D, et al. Malignant hyperthermia. Orphanet J Rare Dis. 2007; 2: 21, doi: 10.1186/1750-1172-2-21, indexed in Pubmed: 17456235.
26. Litman RS, Rosenberg H. Malignant hyperthermia: update on susceptibility testing. JAMA. 2005; 293(23): 2918–2924, doi: 10.1001/jama.293.23.2918, indexed in Pubmed: 15956637.
27. Groom L, Muldoon SM, Tang ZZ, et al. Identical de novo mutation in the type 1 ryanodine receptor gene associated with fatal, stress-induced malignant hyperthermia in two unrelated families. Anesthesiology. 2011; 115(5): 938–945, doi: 10.1097/ALN.0b013e3182320068, indexed in Pubmed: 21918424.
28. Patel HP, Cooper C, Sayer AA(2012). Percutaneous Muscle Biopsy: History, Methods and Acceptability. in Muscle Biopsy, Dr. Challa Sundaram (Ed.). ISBN: 978-953-307-778-9, InTech. http://www.intechopen.com/books/muscle-biopsy/percutaneous-muscle-biopsy-history-methods-andacceptability doi: 10.5772/30714.
29. Downey MS, Banks AS. Gastrocnemius recession in the treatment of nonspastic ankle equinus. A retrospective study. J Am Podiatr Med Assoc. 1989; 79(4): 159–174, doi: 10.7547/87507315-79-4-159, indexed in Pubmed: 2732918.
30. Downey MS. Ankle equinus. In: Banks AS, Martin DE. ed. Comprehensive textbook of foot surgery. Lippincott Williams & Vilkins, Baltimore 2001: 715–760.
31. Hemo Y, Macdessi SJ, Pierce RA, et al. Outcome of patients after Achilles tendon lengthening for treatment of idiopathic toe walking. J Pediatr Orthop. 2006; 26(3): 336–340, doi: 10.1097/01.bpo.0000217743.44609.44, indexed in Pubmed: 16670545.