Genij Ortopedii. 2022. Vol. 28, no. 6. P. 858-866.

Review article

https://doi.org/10.18019/1028-4427-2022-28-6-858-866

A contemporary view of Blount's disease (literature review)

E.L. Strokova, N.Yu. Pakhomova, A.A. Korytkin, V.V. Kozhevnikov, A.F. Gusev✉, A.M. Zaidman

Novosibirsk Research Institute of Traumatology and Orthopedics n.a. Ya.L. Tsivyan, Novosibirsk, Russian Federation

Corresponding author: Arkady F. Gusev, agusev@niito.ru

Annotation

Introduction Blount's disease is a severe pediatric pathology of the musculoskeletal system. The condition is characterized by impaired growth and development of the proximal medial tibia and epiphysis, leading to varus deformity of the knee joint. Blount's disease is not just a cosmetic defect, but a serious orthopaedic condition accompanied by a gait disorder in a child. Patients with Blount's disease need surgical correction followed by long-term rehabilitation and can develop recurrence. The etiology of Blount's disease is unknown. Varus deformity of the knee joint is diagnosed in children all over the world, but studies on this pathology are few. There is a paucity of publications in the modern Russian literature reporting the pathology. **The objective** was review the literature on the classification, diagnosis, etiology and treatment of Blount's disease. **Material and methods** Electronic databases of PubMed, Scopus, eLibrary were used to source literature on the topic. **Results** Blount's disease has been shown to be characterized by disordered growth of the medial aspect of the proximal tibial physis and epiphysis that results in a three-dimensional deformity of the lower limb. In recent years, significant progress has been made in the diagnosis and surgical treatment of the cohort of patients. The etiology of Blount disease is unknown, and it is currently thought to result from a multifactorial combination of hereditary, humoral, biomechanical, and developmental factors. **Conclusion** Genetic predisposition has been postulated in the development of Blount's disease in many studies. Multiple factors such as ethnicity, obesity and early walking age are thought to be the contributing elements to this disease. To understand the key factor of the disease, further study of the hereditary nature of this pathology is necessary.

Keywords: Blount's disease, Erlacher-Blount-Biesin disease, varus deformity of lower limb, etiology and treatment of Blount's disease

For citation: Strokova E.L., Pakhomova N.Yu., Korytkin A.A., Kozhevnikov V.V., Gusev A.F., Zaidman A.M. A contemporary view of Blount's disease (literature review). *Genij Ortopedii*, 2022, vol. 28, no. 6, pp. 858-866. DOI: 10.18019/1028-4427-2022-28-6-858-866.

INTRODUCTION

Blount's disease is asymmetric disordered growth and development of the medial aspect of the proximal tibial physis resulting in a three-dimensional lower-limb deformity [1, 2]. The disease is characterized by progressive varus (O-shaped) deformity of the knee joint in a child (Fig. 1). Valgus (X-shaped) deformity can be observed in some cases [1, 3].

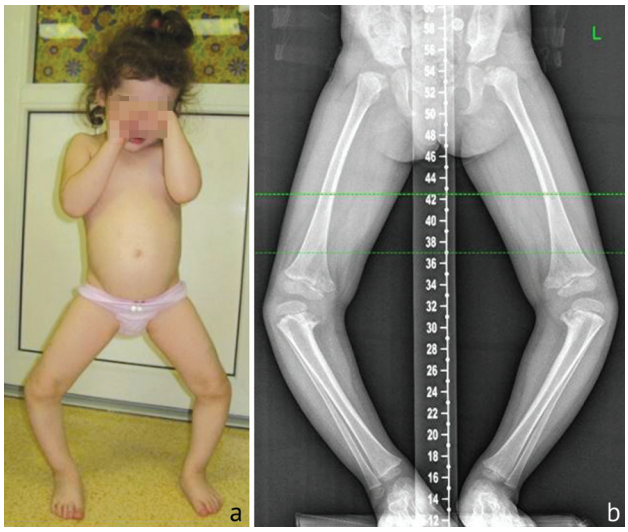

Fig. 1 Appearance (*a*) and radiograph of the lower limbs (*b*) of a patient with bilateral Blount's disease aged 3 years 1 month [4]

First manifestations of the disease can be detected as the child starts walking [5]. Patients are diagnosed with radiographic changes in the proximal tibia in early stages. The effect of secondary changes in the femur on varus deformity is not so significant and increases in elder children with a late onset of the disease [6]. The lateral tibia and fibula develop normally. Concomitant valgus deformity of the ankle may occur in combination with tibial torsion in Blount disease [7]. Deformity of the knee joint can affect one or both limbs [8]. Bilateral involvement occurs in about 50 % of cases [9]. Patients with unilateral involvement can develop a relative limb shortening and impaired gait. Knee ligament instability and compensatory valgus deformity of the distal femur can be noted in some cases [7].

The severity of the disease can vary from injury to the articular cartilage to the knee deformity, premature arthritis and limb length discrepancy [7]. Absence of timely treatment and deformity progression can result in the knee dysfunction [7, 10, 11]. The prognosis of Blount disease in these cases is often severe, particularly in the infantile form due to the development of premature medial proximal tibial epiphysiodesis at about 6 to 8 years of age [7]. Irreversible asymmetric epiphysiodesis makes conservative treatment ineffective. The condition is characterized by a

gradually progressive deformity of the knee, early degenerative changes in the knee joint with difficult ambulation in some cases [7]. Overweight is the main risk factor for disease progression [12]. Patients with Blount's disease require surgical correction followed by long-term rehabilitation; recurrence can occur. The etiology of Blount's disease is unknown. There is a paucity of publications in the Russian scientific literature describing Blount's disease.

The objective was review the Russian and foreign literature on the classification, diagnosis, etiology and treatment of Blount's disease.

MATERIAL AND METHODS

Databases of electronic resources PubMed, Scopus, eLibrary were searched for scientific publications including the following keywords: "Blount's disease", "Erlacher-Blount-Beezin's disease", "lower-limb varus deformity", "etiology and treatment of Blount's disease". The review included articles written in Russian, English and French. The search depth was 20 years, from 2002 to 2022. The review included publications that explored main parameters and general characteristics of Blount's disease in the historical aspect starting from 1922 when the disease was identified as a separate nosological entity. Of the articles identified, 80 full-text manuscripts were selected for analysis. The choice was determined by the fundamental nature of the work on diagnostic methods, etiology and treatment of Blount's disease. The articles selected corresponded to II-III levels of evidence.

RESULTS AND DISCUSSION

Prevalence and classification

Blount's disease ranks second after rickets among lower-limb deformities in children [13]. The prevalence of Blount's disease varies, and the available data suggest significant geographic differences in incidence and prognosis. The prevalence is less than 1 % in young children in the United States [14]. In the Caribbean, the prevalence of early-onset pathology is estimated at 1 in 1200 live births. A typical patient with Blount's disease for this area is an Afro-Caribbean child or a black African under the age of 3 years who is overweight and walks early (before 10 months) [15]. The combination of Blount disease and obesity is less common in other ethnic groups [15, 16, 17]. Significant differences are also observed in the gender of Blount disease. A multicenter study conducted in 2003 in Japan showed that the disease was more common in girls [18]. Other studies provide opposite data: Blount's disease is more common in boys than in girls [9, 19].

The first clinical and radiographic description of a single patient was by Erlacher in the German literature in 1922 [20]. In 1937, W.P.Blount provided detailed histologic and radiographic descriptions of 13 original cases of tibia vara and coined the term “osteochondrosis deformans tibiae” [9]. In the Russian literature, the disease was first described by A.P. Biezin and was called "subepiphyseal osteochondropathy of the tibia" [21]. Erlacher-Blount-Biesin's disease/syndrome has been a common term to define the pathology for several decades and the term Blount's disease is commonly used in modern literature. Blount's disease is coded as Q68.4 "Congenital curvature of the tibia and fibula" in the international classification of diseases ICD-10. Then Blount’s disease was re-classified into three age-onset groups: (1) infantile: through three years of age, (2) juvenile: four to ten years of age, and (3) adolescent: 11 years or older before skeletal maturity [4, 22]. There is no consensus on the classification of Blount's disease. A number of authors combine the juvenile and adolescent forms into a separate group with a late onset [17], while others consider it inappropriate to isolate the juvenile form and classify it as a late-detected infantile form that was not previously diagnosed [23, 24]. Consdering different treatment strategies and clinical and radiological characteristics, the identification of two main forms – infantile and adolescent - is the most appropriate [4, 7].

A meta-analysis of the demographic data of patients with Blount's disease showed that there are differences between the early and late forms of the disease based on asymmetry, race and gender. The early form of the disease is characterized by severe deformity of the tibia, bilateral involvement, and is more common in females [25, 26]. Patients with a late onset of the disease have a unilateral involvement including additional hip deformities [27]. Late onset tibia vara is more prevalent in Blacks, Hispanics, and Scandinavians than other populations and nationalities [17, 26].

In 1964, A. Langenskiold and E.B. Riska suggested a radiographic classification based on a study of patients in Sweden [19]. The classification was based on a degree of epiphyseal depression and metaphyseal fragmentation of the proximal medial tibial epiphysis [19, 22, 28, 29]. The system offered by A. Langenskiold described six progressive radiographic stages of the childhood type of Blount's disease, which are widely used as factors that would determine the prognosis [30]. Early stages (I-III) have a high potential for spontaneous resolution and complete recovery and late stages (IV-VI) have a greater potential for deformity progression and correlate with major surgery and the risk of recurrence after corrective surgery [31-35]. The

classification was confirmed by many radiographic studies performed in Blount disease between 1952 and 1963 [19, 22, 28, 29]. Despite the errors (intra- and inter observer differences in measurements, relapses at stages II and III), the A. Langenskiold system is still used by many orthopaedic surgeons [31, 36]. Langenskiold's classification was simplified in the following decades to improve the clinical and radiological correlations [15, 37], and the FDF (Fort-de-France) classification based on MRI scanning was often employed in practice [38]. A new modified 3-step (types A, B, and C) classification system based on the morphology of the metaphyseal/epiphyseal tilt of the tibia was developed for infantile Blount disease to address the recurrence [39]. The classification system correlated better with treatment outcomes than the traditional A. Langenskiold grading system and was reliable system for predicting relapses [40].

Research methods and differential diagnostics

Deformity of the lower limbs in childhood is a common manifestation. Blount's disease, rickets, tibial hemimelia, bone dysplasia and physiological bowing of the lower limbs should also be considered in the differential diagnosis in young children [41]. Varus deformity of the knee joints (genu varum) is a normal physiological process in a child under 2 years of age [42]. Physiological deformity of the proximal medial metaphysis of the tibia can be accompanied by a radiological sign in the form of a slight medial metaphyseal beak (MMB), which is difficult to distinguish from Blount disease [1]. Based on one radiograph at the first visit to the orthopaedic surgeon, it is difficult to distinguish between physiological deformity and Blount disease. Inadequate differential diagnosis of physiological bowing and early onset of Blount disease can lead to irreversible consequences including limb length discrepancy, progressive deformity, disturbed gait and premature arthritis of the knee joint [42]. A retrospective study showed that Blount's disease was diagnosed in 20 children out of 69 children aged 2-4 years who presented with O-shaped deformity of the lower limbs of unknown etiology. The rest 49 children developed self-correction of physiological bowed legs [12]. Early and accurate distinction between these two conditions is of great importance for timely treatment of Blount's disease. No treatment is normally required for physiological bowed legs [1]. Radiography is the standard method for verifying Blount's disease. Anteroposterior screening radiography of the lower limbs, standard anteroposterior and lateral radiographs of the knee joint are used for early diagnosis of the disease. In early-stage infantile Blount disease, they show medial varus malalignment of the tibial metaphysis, delayed medial ossification of the epiphyseal ossification centre, broadening and contour irregularity of the medial metaphysis producing a beak-like protuberance, and lateral subluxation of the tibia [7].

The angle of the medial metaphyseal beak is a potential radiographic parameter for the differential diagnosis of the disease and physiological bowed legs (Fig. 2). The medial metaphyseal beak in Blount's disease measures 122 degrees or over (downward), which corresponds to Langenskiold stage II on the anteroposterior (AP) radiograph of the knee joint [1] (Fig. 3). The final diagnosis of Blount's disease is based on progressive bowing of the knee joints with characteristic radiographic changes in the proximal medial tibia [1].

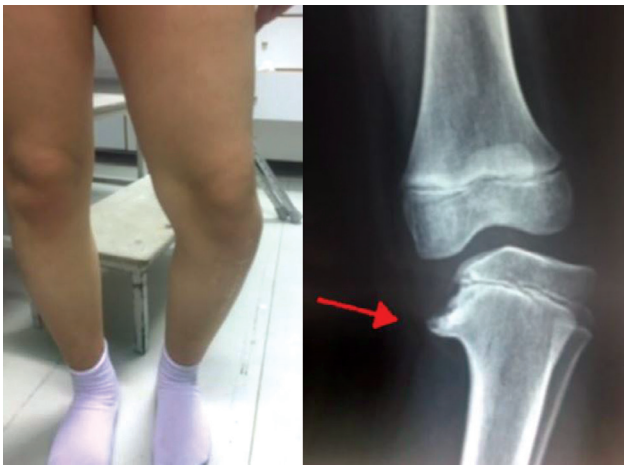

Fig. 2 Clinical manifestations of Blount disease with the red arrow indicating the medial metaphyseal beak (MMB) [42]

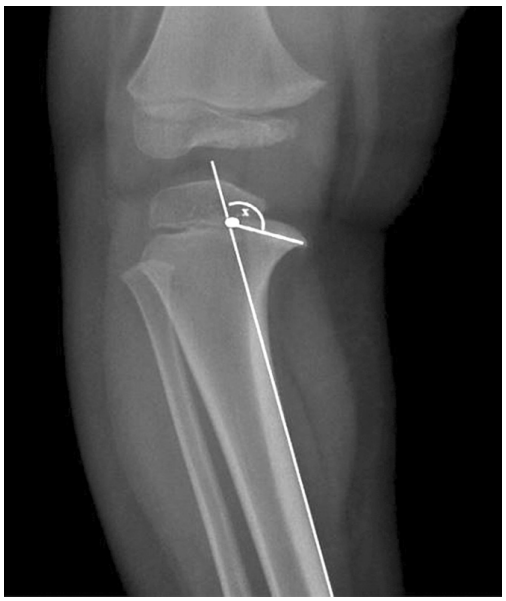

Fig. 3 The angle of the medial metaphyseal beak (MMB) (x) is formed by two lines: line 1 is drawn parallel to the medial cortex of the proximal tibia, line 2 is drawn from the point of intersection of the first line with the proximal tibial metaphysis to the most distal point of the medial metaphyseal beak [1]

The standard screening radiograph is most common method for Blount disease. However, the initial changes in the proximal growth zone of the tibia may not be visualized on conventional radiographs in some cases and computed tomography (CT) of the knee joints can be recommended for accurate diagnosis. CT is practical for identifying early manifestations of the disease and for early differential diagnosis. In addition to that, CT is reliable for assessment of the proliferative layer of the growth zone and diagnosis of premature synostosis of the proximal growth zone of the tibia and of complete elimination of the dysplasia nidus after deformity correction [43].

Magnetic resonance imaging (MRI) can be informative in delayed or delayed forms of Blount's disease seen in children older than four years. MRI allows assessment of tendons, ligaments, menisci, cartilage and vascularisation (if needed) of the main components of the joint [44]. MRI is effective in detecting cartilage changes of the tibial epiphysis, in particular, obtaining more accurate data than measurements made with radiographs [17, 45, 46]. The structure of the meniscus and articular surface of the affected knee was explored in patients with Blount's disease using MRI. The studies showed an increased height and width of the medial meniscus, an increased thickness of the chondroepiphysis of the proximal medial tibia, and a higher frequency of abnormal signals in the posterior horn of the medial meniscus. The authors suggested that the structural changes were designed to compensate for the decrease in the height of the ossified portion of the medial proximal tibia [35]. Histological studies demonstrated an impaired enchondral ossification on the medial side of the proximal tibial physis, disorganized bone and cartilage structures slowing down replacement of cartilage tissue with bone in Blount cases [47]. Impaired columnar structure of the growth zone and replacement of cartilage and bone between the epiphysis and metaphysis with fibrous tissue were reported in severe cases [9, 22]. Varus deformity of the distal femur could be observed in children, adolescents, and young adults with Blount's disease [48].

Etiology and pathogenesis of Blount's disease

Since the first publication of Blount's disease, different hypotheses on the aetiology are proposed but no consensus exists [47]. The etiology of Blount's disease is unknown [10]. A variety of hypotheses was postulated in these articles with most research in the field of increased mechanical pressure (obesity, early walking age) and race (descend). Blount's disease most likely has a multifactorial origin with influence of genetic and racial predisposition, increased mechanical pressure on the growth plate as a consequence of obesity or early walking age and possibly also nutrition. Most papers focus only on one hypotheses of Blount's disease occurrence and all are characterized as low level of evidence. In the Americas and Caribbean, Blount disease chiefly affects black obese children [7], the question of the relationship of the diseases remains open. There is no consensus on the role of obesity in the formation of knee deformity. However, most researchers establish a direct link between the conditions and define Blount's disease as a developmental disorder associated with childhood obesity [12, 35, 49, 50]. The associations of Blount disease with black ethnicity and obesity in other parts of the world are less clear, and the prognosis is often less severe [7].

Familial inheritance for Blount disease was explored in West Africa, in the Republic of Ghana. The results of the sequencing revealed no genetic predisposition to this disease [51]. Many researchers tend to believe that Blount's disease has a multifactorial origin and is associated with mechanical overload in genetically susceptible individuals. Predisposing factors for the disease include genetic and racial predisposition, obesity, tall stature, early walking, African American origin, or a combination of the above factors [7, 10, 47]. The association of pathology with vitamin D deficiency has also been reported for early and late forms [52, 53]. It can be concluded that Blount's disease is the result of a multifactorial combination of genetic, humoral, biomechanical and developmental factors [10, 47]. Further research on especially genetic predisposition is needed to provide more insight in this factor of Blount's disease [47].

The pathophysiological mechanisms of the disease are unclear [7]. The most common biomechanical hypothesis explains the development of Blount's disease by excessive mechanical stress in combination with specific factors of genetic susceptibility. With physiological deformity, excess weight in a child increases the load on the bones and joints, on the posteromedial portion of the knee, in particular, and there is a gradual suppression of growth in the medial tibia, slowing down of ossification and destruction of stabilizing ligament systems. Compressive forces have a greater effect on the tibia rather than the femur due to the limited growth potential and a weaker three-dimensional tibial structure [54]. However, the biomechanical hypothesis cannot explain severe cases of early-onset Blount disease in non-obese children.

Treatment of Blount's disease

Blount's disease can develop in different ways. The disease can be treated conservatively in some cases and does not require surgical intervention. Surgical treatment can be offered for a progressing pathological process and most effective options must be identified [10]. Early-onset disease can resolve spontaneously. Regression

may occur spontaneously, even in Afro-Caribbean children. A retrospective study by Laville et al. of patients in the Reunion Island found that one-third of stage I infantile cases healed spontaneously [37]. In a Japanese study of 46 limbs with infantile Blount disease, spontaneous healing was observed for 22 limbs [32]. An early diagnosis and treatment of this disease can have a great functional impact and lead to very good health outcomes [2].

There is no consensus on the optimal approach to the treatment of Blount disease [55]. The treatment option depends on the severity of the deformity as classified by A. Langenskiold and is based mainly on radiographic findings. Conservative treatment is used in most cases of infantile type stage I or stage II, while surgery is indicated for stage III or IV of the disease [18]. Early treatment of childhood Blount disease involves fixation, although this method in stage I or II is a controversial treatment with variable outcomes [3, 32, 56, 57]. A KAFO orthosis is usually used for fixation. Orthopedic treatment can be practical if provided for one year and initiated before three years of age in non-obese children [3]. Surgical treatment is recommended if orthotic correction fails [58]. A variety of surgical interventions are usually used for deformity progression. The goal of surgical treatment is to restore the congruence of the articular surfaces, restore the normal position of the limb, equalize the limb length and prevent recurrence. Statistical analysis shows that surgical correction before the age of 4 years can lead to complete recovery in 80 % of patients with a progressive early onset form of the disease [15-17]. Osteotomy of the proximal tibia (Fig. 4) for angular deformity correction and realigning the articular surface is the historical standard of surgical treatment of Blount disease [35, 59-61].

Corrective osteotomy can provide accurate correction, simultaneous elimination of all components of the deformity and unload the medial epiphyseal portion [39, 62, 63]. However, overcorrection with the osteotomy can lead to persistent valgus deformity in some cases or greater malalignment. Adverse events of the operations include trauma, delayed consolidation, vascular disorders, compartment syndrome, peroneal nerve palsy, infection and deformity relapses [39, 62, 63]. Corrective osteotomy performed at Langenskiold stage 3 disorder in children older than 3 years is accompanied by a high recurrence rate. Recurrence can result from an initially successful operation [17, 64-66]. The recurrence of varus deformity in Blount disease ranges between 30 and 100 % and can be reduced with the initial operation performed at an early age [65]. The patient's age and the stage of the pathology can be prognostic for deformity recurrence [67]. Surgical treatment can be recommended for patients aged four years and younger with progressive clinical and radiological signs of Blount's disease Langenskiold stage I or II to reduce the percentage of relapses [64, 68, 69]. Greater stage of the disease is the most important prognostic factor [59, 62].

Temporal hemiepiphysiodesis has been developed in recent years as a new approach to the treatment of childhood Blount's disease with controlled growth modulation [70, 71] (Fig. 5).

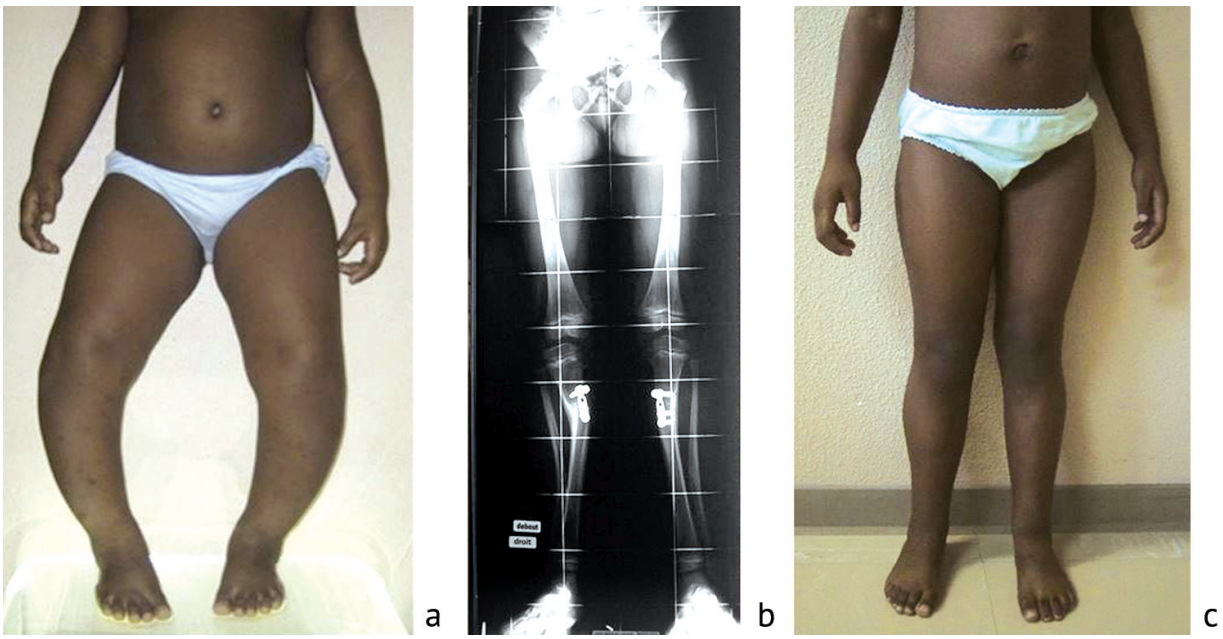

Fig. 4 Stage I Blount disease with early onset in a 31/2-year-old child (FDF classification) treated with bilateral valgus and derotation osteotomies showing (*a*) preoperative appearance of the lower limbs; (*b*) long-standing radiograph; (c) appearance of the lower limbs at 1 year of surgery [7]

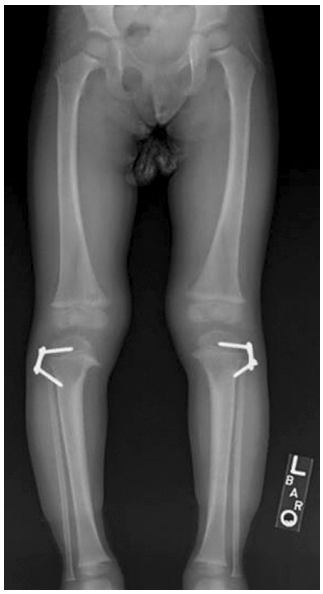

Fig. 5 Long-standing film of a child with infantile Blount's disease at 10 months of bilateral lateral hemiepiphysiodesis [8]

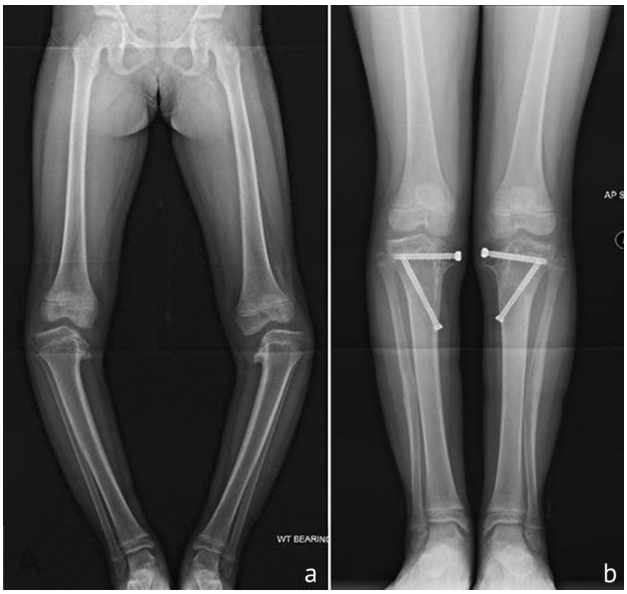

Fig. 6 Long standing films (AP) of a girl with bilateral late infantile Blount's disease (*a*) performed preoperatively at the age of 8 years; (*b*) at 2 years of surgical correction (at the age of 10 years) [78]

The method is gaining popularity and is a viable treatment option for stage 1 and 2 disease due to the relative safety and minimal invasiveness [72-74]. Guided growth can be achieved either with a tension band or with screws designed to temporarily arrest normal growth in the lateral tibia and allow growth to take place in the medial tibia. The angulation correction rate is 100 % with no need for corrective osteotomy. However, there is a recurrence rate of 33 % with the treatment and the deformity can be successfully corrected with an osteotomy or with the method described above in the cases [55]. Despite the significant spread of temporary hemiepiphysiodesis, a significant number of poor results has been reported with the approach of surgical treatment for Blount's disease [75-77].

A late-presenting and recurrent infantile Blount disease (IBD) is a surgical challenge [78]. IBD is relatively rare and is characterized by instability of the knee joint. Medial elevation osteotomy combined with lateral proximal tibial epiphysiodesis and metaphyseal tibial realignment osteotomy is used for restoration of lower limb alignment in a high proportion of cases (Fig. 6). Obesity and residual instability are associated with an increased risk of poor alignment.

There is no universally accepted treatment for infantile forms of Blount's disease. The treatment of this complex pathology must be adapted to each individual case. Successful surgical treatment would be dependent on the early age of the patient (3 to 7 years with the best results reported at the age of 4 years, early stages of the disease (prior to symptoms of synostosis) and complete restoration of the limb axis and mild hypercorrection [23, 63, 79]. Patients treated for the condition in childhood can develop residual anomalies of the knee joint in adulthood and premature osteoarthritis [7].

Treatment of Blount's disease in adolescents and young adults is complicated. Ilizarov external fixation or Taylor Spatial Frame (TSF) can be used for correction of the proximal tibia deformity in Blount disease in this cohort of patients with a low risk of soft tissue complications [80]. The lack of timely treatment of the disease in adulthood can lead to progressive varus deformity and concomitant deformity of the distal femur and/or tibia, limb length discrepancy and a severe articular deformity [10]. Untreated Blount disease in adults is rarely encountered [7].

CONCLUSION

A review of the modern literature on Blount disease revealed that the century-old history of exploring the condition as a separate nosological entity failed to provide insight into the development of this complex musculoskeletal pathology in children and adolescents. The etiology and pathogenetic mechanisms of the pathological process remain unclear a century after the first clinical and radiological description of patients with impaired growth and development of the proximal medial tibia and epiphysis leading to a three-dimensional deformity of the lower limbs. In recent years, progress has been made in the diagnosis and surgical treatment of this cohort of patients. The statistical analysis show that surgical correction under 4 years of age leads to a

complete recovery in 80 % of cases in children with a progressive form and early onset of the disease. Most studies have shown that Blount's disease is the cause of a multifactorial combination of genetic, humoral, biomechanical and developmental factors with genetic susceptibility having a significant role. Further study of the hereditary nature of the pathology is needed to understand the key factor of the disease.

REFERENCES

1. Wongcharoenwatana J., Kaewpornsawan K., Chotigavanichaya C., Eamsobhana P., Laoharojanaphand T., Musikachart P., Ariyawatkul T. Medial metaphyseal beak angle as a predictor for Langenskiöld Stage II of Blount's disease. *Orthop. Surg*., 2020, vol. 12, no. 6, pp. 1703-1709. DOI: 10.1111/os.12760.
2. Vasiliadis A.V., Maris A., Gadikoppula S. Tibia vara or Blount's disease: Why an early diagnosis and treatment are important? *Clin. Pract*., 2020, vol. 10, no. 1, pp. 1222. DOI: 10.4081/cp.2020.1222.
3. Sabharwal S., Sabharwal S. Treatment of infantile Blount disease: An update. *J. Pediatr. Orthop*., 2017, vol. 37, no. Suppl. 2, pp. S26-S31. DOI: 10.1097/BPO.0000000000001027.
4. Vilenskii V.A., Fomylina O.A., Trofimova S.I., Zakharian E.A., Pozdeev A.A. Lechenie patsientov s rannimi stadiiami infantilnoi formy bolezni Blaunta [Treatment of patients with early stages of the infantile form of Blount's disease]. *Sovremennye Problemy Nauki i Obrazovaniia*, 2020, no. 2. (in Russian) DOI: 10.17513/spno.29639.
5. Murthy D.T.S., De Leucio A. *Blount Disease*. Treasure Island (FL), StatPearls Publishing LLC. 2022. Available at: https://www.ncbi.nlm.nih.gov/books/NBK560923/.
6. Gordon J.E., Heidenreich F.P., Carpenter C.J., Kelly-Hahn J., Schoenecker P.L. Comprehensive treatment of late-onset tibia vara. *J. Bone Joint Surg. Am*., 2005, vol. 87, no. 7, pp. 1561-1570. DOI: 10.2106/JBJS.02276.
7. Janoyer M. Blount disease. *Orthop. Traumatol. Surg. Res*., 2019, vol. 105, no. 1S, pp. S111-S121. DOI: 10.1016/j.otsr.2018.01.009.
8. Robbins C.A. Deformity reconstruction surgery for Blount's disease. *Children* (Basel), 2021, vol. 8, no. 7, pp. 566. DOI: 10.3390/children8070566.
9. Blount W.P. Osteochondrosis deformans tibiae. *J. Bone Joint Surg*., 1937, vol. 19, no. 1, pp. 1-29.
10. Birch J.G. Blount disease. *J. Am. Acad. Orthop. Surg*., 2013, vol. 21, no. 7, pp. 408-418. DOI: 10.5435/JAAOS-21-07-408.
11. Natoli R.M., Nypaver C.M., Schiff A.P., Hopkinson W.J., Rees H.W. Total knee arthroplasty in patients with Blount disease or Blount-like deformity. *J. Arthroplasty*, 2016, vol. 31, no. 1, pp. 124-127. DOI: 10.1016/j.arth.2015.07.014.
12. Scott A.C., Kelly C.H., Sullivan E. Body mass index as a prognostic factor in development of infantile Blount disease. *J. Pediatr. Orthop*., 2007, vol. 27, no. 8, pp. 921-925. DOI: 10.1097/BPO.0b013e3181558cd4.
13. Babin E.A. *Bolezn Erlakhera-Blaunta (klinika, diagnostika, lechenie). Diss. kand. med. nauk* [Erlacher-Blount disease (clinic, diagnosis, treatment). Cand. med. sci. diss.]. M., 2008. (in Russian)
14. Smith C.F. Tibia vara (Blount's disease). *J. Bone Joint Surg. Am*., 1982, vol. 64, no. 4, pp. 630-632.
15. Catonné Y. *La maladie de Blount* [Blount's disease]. Paris: Cahiers d'Enseignement de la Société Française d'Orthopédie et Traumatologie. Expansion Scientifique Française, 1997, pp. 147-163. (in French)
16. Herring J. Disorders of the leg. *Tachdjian's Pediatric Orthopaedic*s, 3rd Ed. Philadelphia: W.B. Saunders Company. 2002. Vol. 2, Chapter 21.
17. Sabharwal S. Blount disease. *J. Bone Joint Surg. Am*., 2009, vol. 91, no. 7, pp. 1758-1776. DOI: 10.2106/JBJS.H.01348.
18. Inaba Y., Saito T., Takamura K. Multicenter study of Blount disease in Japan by the Japanese Pediatric Orthopaedic Association. *J. Orthop. Sci*., 2014, vol. 19, no. 1, pp. 132-140. DOI: 10.1007/s00776-013-0489-8.
19. Langenskiold A., Riska E.B. Tibia vara (Osteochondrosis deformans tibiae): a survey of seventy-one cases. *J. Bone Joint Surg. Am*., 1964, vol. 46, pp. 1405-1420.
20. Erlacher P. Deformierende prozesse der epiphysengegend bei kindern [Deforming processes of the epiphyseal area in children]. *Arch. Orthop. Unfallchir*., 1922, vol. 20, pp. 81-96. (in German). Available at: https://doi.org/10.1007/BF02597575.
21. Biezin A.P. *Detskaia khirurgiia* [Pediatric Surgery]. M., Meditsina, 1964, 334 p. (in Russian)
22. Thompson G.H., Carter J.R. Late-onset tibia vara (Blount's disease). Current concepts. *Clin. Orthop. Relat. Res*., 1990, no. 255, pp. 24-35.
23. Filatov S.V. *Subepifizarnaia osteokhondropatiia bolshebertsovoi kosti u detei (kliniko-rentgenologicheskoe issledovanie). Diss. kand. med. nauk* [Sub-epiphyseal osteochondropathy of the tibia in children (a clinical-and-radiological study). Cand. med. sci. diss.]. L., 1972. (in Russian)
24. Herring J.A. *Tachdjian's Pediatric Orthopaedics: From the Texas Scottish Rite Hospital for Children E-Book*. Elsevier Health Sciences, 2020.
25. Sabharwal S., Lee J. Jr., Zhao C. Multiplanar deformity analysis of untreated Blount disease. *J. Pediatr. Orthop*., 2007, vol. 27, no. 3, pp. 260-265. DOI: 10.1097/BPO.0b013e31803433c3.
26. Rivero S.M., Zhao C., Sabharwal S. Are patient demographics different for early-onset and late-onset Blount disease? Results based on meta-analysis. *J. Pediatr. Orthop. B*, 2015, vol. 24, no. 6, pp. 515-520. DOI: 10.1097/BPB.0000000000000211.
27. Myers T.G., Fishman M.K., McCarthy J.J., Davidson R.S., Gaughan J. Incidence of distal femoral and distal tibial deformities in infantile and adolescent Blount disease. *J. Pediatr. Orthop*., 2005, vol. 25, no. 2, pp. 215-218. DOI: 10.1097/01.bpo.0000149858.04659.d8.
28. Langenskiöld A. Tibia vara: osteochondrosis deformans tibiae. Blount's disease. *Clin. Orthop. Relat. Res*., 1981, no. 158, pp. 77-82.
29. Langenskiöld A. Tibia vara. A critical review. *Clin. Orthop. Relat. Res*., 1989, no 246, pp. 195-207.
30. Du Plessis J., Firth G.B., Robertson A. Assessment of the reliability and reproducibility of the Langenskiöld classification in Blount's disease. *J. Pediatr. Orthop. B*, 2020, vol. 29, no. 4, pp. 311-316. DOI: 10.1097/BPB.0000000000000692.
31. Stricker S.J., Edwards P.M., Tidwell M.A. Langenskiöld classification of tibia vara: an assessment of interobserver variability. *J. Pediatr. Orthop*., 1994, vol. 14, no. 2, pp. 152-155. DOI: 10.1097/01241398-199403000-00004.
32. Shinohara Y., Kamegaya M., Kuniyoshi K., Moriya H. Natural history of infantile tibia vara. *J. Bone Joint Surg. Br*., 2002, vol. 84, no. 2, pp. 263-268. DOI: 10.1302/0301-620x.84b2.11821.
33. Erkus S., Turgut A., Kalenderer O. Langenskiöld classification for Blount disease: is it reliable? *Indian J. Orthop*., 2019, vol. 53, no. 5, pp. 662-664. DOI: 10.4103/ortho.IJOrtho_679_18.
34. Khanfour A.A. Does Langenskiöld staging have a good prognostic value in late onset tibia vara? *J. Orthop. Surg. Res*., 2012, vol. 7, pp. 23. DOI: 10.1186/1749-799X-7-23.
35. Sabharwal S. Blount disease: an update. *Orthop. Clin. North Am*., 2015, vol. 46, no. 1, pp. 37-47. DOI: 10.1016/j.ocl.2014.09.002.
36. Bradway J.K., Klassen R.A., Peterson H.A. Blount disease: a review of the English literature. *J. Pediatr. Orthop*., 1987, vol. 7, no. 4, pp. 472-480.
37. Laville J.M., Wiart Y., Salmeron F. Can Blount's disease heal spontaneously? *Orthop. Traumatol. Surg. Res*., 2010, vol. 96, no. 5, pp. 531-535. DOI: 10.1016/j.otsr.2010.03.015.
38. Janoyer M., Jabbari H., Rouvillain J.L., Sommier J., Py G., Catonné Y., Colombani J.F. Infantile Blount's disease treated by hemiplateau elevation and epiphyseal distraction using a specific external fixator: preliminary report. *J. Pediatr. Orthop. B*, 2007, vol. 16, no. 4, pp. 273-280. DOI: 10.1097/01.bpb.0000210591.35652.84.

39. LaMont L.E., McIntosh A.L., Jo C.H., Birch J.G., Johnston C.E. Recurrence after surgical intervention for infantile tibia vara: assessment of a new modified classification. *J. Pediatr. Orthop*., 2019, vol. 39, no. 2, pp. 65-70. DOI: 10.1097/BPO.0000000000000933.
40. Jardaly A.H., Conklin M., Strom S.F., Wall K.C., Gilbert S. Prognostic reliability of a new classification system for Blount's disease. *Cureus*, 2020, vol. 12, no. 5, pp. e8353. DOI: 10.7759/cureus.8353.
41. Brooks W.C., Gross R.H. Genu varum in children: diagnosis and treatment. *J. Am. Acad. Orthop. Surg*., 1995, vol. 3, no. 6, pp. 326-335. DOI: 10.5435/00124635-199511000-00003.
42. Delniotis I., Leidinger B., Kyriakou A., Galanis N. Blount's disease: The importance of early diagnosis and early treatment. *Clin. Case Rep*., 2019, vol. 7, no. 7, pp. 1454-1455. DOI: 10.1002/ccr3.2214.
43. Diachkova G.V., Chelovechkova A.A., Korabelnikov M.A. The possibilities of the knee zone CT in the diagnosis and evaluation of Erlacher-Blaunt disease treatment. *Genij Ortopedii*, 2004, no. 2, pp. 26-29. (in Russian)
44. Gill K.G., Nemeth B.A., Davis K.W. Magnetic resonance imaging of the pediatric knee. *Magn. Reson. Imaging Clin. N. Am*., 2014, vol. 22, no. 4, pp. 743-63. DOI: 10.1016/j.mric.2014.07.010.
45. Sabharwal S., Wenokor C., Mehta A., Zhao C. Intra-articular morphology of the knee joint in children with Blount disease: a case-control study using MRI. *J. Bone Joint Surg. Am*., 2012, vol. 94, no. 10, pp. 883-890. DOI: 10.2106/JBJS.K.00956.
46. Ho-Fung V., Jaimes C., Delgado J., Davidson R.S., Jaramillo D. MRI evaluation of the knee in children with infantile Blount disease: tibial and extra-tibial findings. *Pediatr. Radiol*., 2013, vol. 43, no. 10, pp. 1316-1326. DOI: 10.1007/s00247-013-2686-1.
47. Banwarie R.R., Hollman F., Meijs N., Arts J.J., Vroemen P., Moh P., Staal H.M. Insight into the possible aetiologies of Blount's disease: a systematic review of the literature. *J. Pediatr. Orthop. B*, 2020, vol. 29, no. 4, pp. 323-336. DOI: 10.1097/BPB.0000000000000677.
48. Firth G.B., Ngcakani A., Ramguthy Y., Izu A., Robertson A. The femoral deformity in Blount's disease: a comparative study of infantile, juvenile and adolescent Blount's disease. *J. Pediatr. Orthop. B*, 2020, vol. 29, no. 4, pp. 317-322. DOI: 10.1097/BPB.0000000000000722.
49. Jardaly A., McGwin G. Jr., Gilbert S.R. Blount disease and obstructive sleep apnea: an under-recognized association? *J. Pediatr. Orthop*., 2020, vol. 40, no. 10, pp. 604-607. DOI: 10.1097/BPO.0000000000001591.
50. Jardaly A.H., Hicks J.W., Doyle J.S., Conklin M.J., Gilbert S.R. Co-occurrence of Blount's disease and Legg-Calvé-Perthes disease: is obesity a factor? *J. Pediatr. Orthop. B*, 2022, vol. 31, no. 2, pp. e180-e184. DOI: 10.1097/BPB.0000000000000888.
51. Jansen N., Hollman F., Bovendeert F., Moh P., Stegmann A., Staal H.M. Blount disease and familial inheritance in Ghana, area cross-sectional study. *BMJ Paediatr. Open*, 2021, vol. 5, no. 1, pp. e001052. DOI: 10.1136/bmjpo-2021-001052.
52. Güven A, Hancılı S, Kuru Lİ. Obesity and increasing rate of infantile Blount disease. *Clin. Pediatr*. (Phila), 2014, vol. 53, no. 6, pp. 539-543. DOI: 10.1177/0009922813518424.
53. Lisenda L., Simmons D., Firth G.B., Ramguthy Y., Kebashni T., Robertson A.J. Vitamin D status in Blount disease. *J. Pediatr. Orthop*., 2016, vol. 36, no. 5, pp. 59-62. DOI: 10.1097/BPO.0000000000000607.
54. Grover J.P., Vanderby R., Leiferman E.M., Wilsman N.J., Noonan K.J. Mechanical behavior of the lamb growth plate in response to asymmetrical loading: a model for Blount disease. *J. Pediatr. Orthop*., 2007, vol. 27, no. 5, pp. 485-492. DOI: 10.1097/BPO.0b013e318070cb9b.
55. Griswold B.G., Shaw K.A., Houston H., Bertrand S., Cearley D. Guided growth for the treatment of infantile Blount's disease: is it a viable option? *J. Orthop*., 2020, vol. 20, pp. 41-45. DOI: 10.1016/j.jor.2020.01.007.
56. Richards B.S., Katz D.E., Sims J.B. Effectiveness of brace treatment in early infantile Blount's disease. *J. Pediatr. Orthop*., 1998, vol. 18, no. 3, pp. 374-380.
57. De Miranda Luzo M.C., Montenegro N.B., Massa B.S., de Angeli L.R., Cordeiro F.G., Guarniero R. Management of infantile Blount's disease with molded orthoses: a new perspective. *Acta Ortop. Bras*., 2016, vol. 24, no. 2, pp. 85-89. DOI: 10.1590/1413-785220162402153725.
58. Zionts L.E., Shean C.J. Brace treatment of early infantile tibia vara. *J. Pediatr. Orthop*., 1998, vol. 18, no. 1, pp. 102-109.
59. Ferriter P., Shapiro F. Infantile tibia vara: factors affecting outcome following proximal tibial osteotomy. *J. Pediatr. Orthop*., 1987, vol. 7, no. 1, pp. 1-7.
60. McCarthy J.J., Ranade A., Davidson R.S. Pediatric deformity correction using a multiaxial correction fixator. *Clin. Orthop. Relat. Res*., 2008, vol. 466, no. 12, pp. 3011-3017. DOI: 10.1007/s11999-008-0491-1.
61. Alsancak S., Guner S., Kinik H. Orthotic variations in the management of infantile tibia vara and the results of treatment. *Prosthet. Orthot. Int*., 2013, vol. 37, no. 5, pp. 375-383. DOI: 10.1177/0309364612471369.
62. Eamsobhana P., Kaewpornsawan K., Yusuwan K. Do we need to do overcorrection in Blount's disease? *Int. Orthop*., 2014, vol. 38, no. 8, pp. 1661-1664. DOI: 10.1007/s00264-014-2365-3.
63. Tenilin N.A., Bogosian A.B., Karataeva D.S. 40-letnii opyt primeneniia korrigiruiushchikh osteotomii pri lechenii bolezni Blaunta [40 years of experience with corrective osteotomies in the treatment of Blount's disease]. *Vestnik Travmatologii i Ortopedii im. N.N. Priorova*, 2012, no. 3, pp. 3-8. (in Russian)
64. Chotigavanichaya C., Salinas G., Green T., Moseley C.F., Otsuka N.Y. Recurrence of varus deformity after proximal tibial osteotomy in Blount disease: long-term follow-up. *J. Pediatr. Orthop*., 2002, vol. 22, no. 5, pp. 638-641.
65. Gonzàlez-Herranz P. Recurrence of varus deformity after proximal tibial osteotomy in Blount disease: long-term follow-up. *J. Pediatr. Orthop*., 2005, vol. 25, no. 1, pp. 130. DOI: 10.1097/00004694-200501000-00028.
66. Jones J.K., Gill L., John M., Goddard M., Hambleton I.R. Outcome analysis of surgery for Blount disease. *J. Pediatr. Orthop*., 2009, vol. 29, no. 7, pp. 730-735. DOI: 10.1097/BPO.0b013e3181b768d9.
67. Schoenecker P.L., Meade W.C., Pierron R.L., Sheridan J.J., Capelli A.M. Blount's disease: a retrospective review and recommendations for treatment. *J. Pediatr. Orthop*., 1985, vol. 5, no. 2, pp. 181-186.
68. Hayek S., Segev E., Ezra E., Lokiec F., Wientroub S. Serrated W/M osteotomy. Results using a new technique for the correction of infantile tibia vara. *J. Bone Joint Surg. Br*., 2000, vol. 82, no. 7, pp. 1026-1029. DOI: 10.1302/0301-620x.82b7.10507.
69. Synder M., Vera J., Harcke H.T., Bowen J.R. Magnetic resonance imaging of the growth plate in late-onset tibia vara. *Int. Orthop*., 2003, vol. 27, no. 4, pp. 217-222. DOI: 10.1007/s00264-003-0467-4.
70. Danino B., Rödl R., Herzenberg J.E., Shabtai L., Grill F., Narayanan U., Gigi R., Segev E., Wientroub S. The efficacy of guided growth as an initial strategy for Blount disease treatment. *J. Child. Orthop*., 2020, vol. 14, no. 4, pp. 312-317. DOI: 10.1302/1863-2548.14.200070.
71. Khanfour A.A. Blount's disease – an up-to-date insight with contemporary treatment guidelines deduced from critical analysis of a large 146 surgical case series. *J. Pediatr. Orthop. B*, 2021, vol. 30, no. 3, pp. 239-249. DOI: 10.1097/BPB.0000000000000769.
72. Scott A.C. Treatment of infantile Blount disease with lateral tension band plating. *J. Pediatr. Orthop*., 2012, vol. 32, no. 1, pp. 29-34. DOI: 10.1097/BPO.0b013e31823db034.
73. Kenis V.M., Klychkova I.Iu., Melchenko E.V., Ivanov S.V., Sapogovskii A.V. Korrektsiia deformatsii nizhnikh konechnostei u detei s pomoshchiu metoda upravliaemogo rosta [Correction of deformities of the lower extremities in children using the method of guided growth]. *Vestnik Travmatologii i Ortopedii im. N.N. Priorova*, 2013, no. 4, pp. 50-55. (in Russian)
74. Heflin J.A., Ford S., Stevens P. Guided growth for tibia vara (Blount's disease). *Medicine* (Baltimore), 2016, vol. 95, no. 41, pp. e4951. DOI: 10.1097/MD.0000000000004951.
75. Oto M., Yılmaz G., Bowen J.R., Thacker M., Kruse R. Adolescent Blount disease in obese children treated by eight-plate hemiepiphysiodesis. *Eklem. Hastalik. Cerrahisi*., 2012, vol. 23, no. 1, pp. 20-24.

76. Burghardt R.D., Herzenberg J.E., Strahl A., Bernius P., Kazim M.A. Treatment failures and complications in patients with Blount disease treated with temporary hemiepiphysiodesis: a critical systematic literature review. *J. Pediatr. Orthop. B*, 2018, vol. 27, no. 6, pp. 522-529. DOI: 10.1097/BPB.0000000000000523.
77. Fan B., Zhao C., Sabharwal S. Risk factors for failure of temporary hemiepiphysiodesis in Blount disease: a systematic review. *J. Pediatr. Orthop. B*, 2020, vol. 29, no. 1, pp. 65-72. DOI: 10.1097/BPB.0000000000000603.
78. Maré P.H., Thompson D.M., Marais L.C. The medial elevation osteotomy for late-presenting and recurrent infantile Blount disease. *J. Pediatr. Orthop*., 2021, vol. 41, no. 2, pp. 67-76. DOI: 10.1097/BPO.0000000000001722.
79. Blount W.P. Tibia vara, osteochondrosis deformans tibiae. *Curr. Pract. Orthop. Surg*., 1966, vol. 3, pp.141-156.
80. Saw A., Phang Z.H., Alrasheed M.K., Gunalan R., Albaker M.Z., Shanmugam R. Gradual correction of proximal tibia deformity for Blount disease in adolescent and young adults. *J. Orthop. Surg*. (Hong Kong), 2019, vol. 27, no. 3, 2309499019873987. DOI: 10.1177/2309499019873987.

The article was submitted 07.10.2022; approved after reviewing 17.10.2022; accepted for publication 19.10.2022.

Information about the authors:

1. Elena L. Strokova – Candidate of Biological Sciences, EZavyalova@niito.ru, https://orcid.org/0000-0002-5789-6982;
2. Natalya Yu. Pahomova – Candidate of Medical Sciences, Associate Professor, NPahomova@niito.ru, https://orcid.org/0000-0002-9575-4096;
3. Andrey A. Korytkin – Candidate of Medical Sciences, andrey.korytkin@gmail.com, https://orcid.org/0000-0001-9231-5891;
4. Vadim V. Kozhevnikov – Candidate of Medical Sciences, vadim-barnaul@bk.ru, https://orcid.org/0000-0003-2556-3347;
5. Arkady F. Gusev – Candidate of Medical Sciences, agusev@niito.ru, https://orcid.org/0000-0003-1572-0089;
6. Alla M. Zaydman – Doctor of Medical Sciences, Professor, Honored Scientist, zaydmanam@gmail.com, https://orcid.org/0000-0002-6613-1615.