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ABSTRACT

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THE KNEE JOINT IS THE ACHILLES' HEEL OF A CHILD'S BODY

The study objective was to draw practicing physicians' attention to the risk of knee joint disorders in children during intensive growth to ensure timely detection and treatment.

Materials and Methods. We analyzed the results of studies on the diseases of the knee joint carried out by modern scientists over the past ten years. We also took into account the clinical observations of children receiving inpatient treatment at the regional children's hospital. The examination methods were radiography, computer tomography, magnetic resonance imaging, and ultrasound.

Results and Discussion. During growth, a child's knee joint is subjected to enormous loads and is sensitive to external and internal factors. The knee joint plays a dominant role in the limb's longitudinal growth; the growth zones grow so intensively that they can be compared to the work of a "bone nuclear reactor." The bones of the skeleton grow unevenly and intermittently. The peak height velocity is observed at 10–14 years in girls and at 13–18 years in boys; then, height velocity slows down, and after 19 years, height growth almost stops.

During the period of intensive growth, bones are very sensitive to internal and external factors of shape formation (growth hormones, physiological loads, radiation, vibration, injuries, hypothermia, metabolic disorders, etc. Therefore, this part is most sensitive to various endogenous and exogenous factors, which is manifested by various dysplastic changes and diseases.

Conclusions. During the intensive growth of the child, the knee joint is most vulnerable to various endogenous and exogenous negative factors, which is manifested by various diseases. Osteogenic sarcoma, fibrous cortical defect, and juvenile osteocartilaginous exostoses most often affect the weakest place of

the knee joint – the metaphyses of the femur and tibia (Achilles' heel).

Keywords: knee joint, osteogenic sarcoma, fibrous cortical defect, juvenile osteocartilaginous exostoses.

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КОЛІННИЙ СУГЛОБ – «АХІЛЛЕСОВА П'ЯТА» ДИТЯЧОГО ОРГАНІЗМУ

Мета дослідження – звернути увагу практичних лікарів на ризик захворювань в ділянці колінного суглоба у дітей в період інтенсивного росту з метою їх своєчасного розпізнавання та лікування цих недуг.

Матеріали та методи. Здійснено аналіз результатів досліджень сучасних науковців за останні десять років присвячених захворюванням колінного суглоба. Враховані також наші клінічні спостереження дітей, які знаходилися на стаціонарному лікуванні у обласній дитячій лікарні. Методами обстеження були рентгенографія, комп'ютерна томографія, магнітно-резонансна томографія, ультразвукова діагностика.

Результати дослідження та їх обговорення. В процесі росту на колінний суглоб дитини припадають величезні навантаження, і тому він реагує на зовнішні та внутрішні фактори. Саме колінний суглоб має домінуючий вплив на ріст кінцівки в довжину, зони росту настільки інтенсивно працюють, що їх можна порівняти з роботою «кісткового атомного реактора». Ріст кісток скелета іде нерівномірно, стрибкувато. Особливо інтенсивний ріст у дівчаток з 10–14 років, а у хлопчиків з 13–18 років, потім відбувається сповільнення росту, і після 19 років ріст у довжину майже припиняється.

В період інтенсивного росту кістки дуже чутливі до внутрішніх та зовнішніх факторів формоутворення (гормонів росту, фізіологічних навантажень, опромінення, вібрації, травм, переохолодження, розладів обміну речовин тощо). Тому ця частина найбільш вразлива до різноманітних ендогенних та екзогенних негативних впливів, що проявляється різноманітними диспластичними змінами та хворобами.

Висновки. Колінний суглоб в процесі інтенсивного росту дитини є дуже вразливим до різноманітних ендогенних та екзогенних негативних впливів, що проявляється різноманітними хворобами. Остеогенна саркома, фіброзний кортикальний дефект, юнацькі кістково-хрящові екзостози найчастіше уражають найслабше місце колінного суглоба – метафізи кісток стегна та гомілки (ахіллесова п'ята).

Ключові слова: колінний суглоб, остеогенна саркома, фіброзний кортикальний дефект, юнацькі кістково-хрящові екзостози.

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INTRODUCTION / ВСТУП

The knee joint is made up of the distal end of the femur, the proximal end of the tibia, and the largest sesamoid bone of the human body, the patella. The distal end of the femur gradually expands into two condyles: medial (larger) and lateral (smaller), with an intercondylar fossa between them. The femoral condyles have articular surfaces for articulation with the tibia and patella. On the lateral surfaces of the femur, there are bony protrusions slightly above the articular surfaces of the condyles: the medial and lateral epicondyles, to which ligaments are attached. The patella is located in front of the distal femoral epiphysis. The shape of the bone resembles a convex lens with a large rounding on top and a slightly pointed end on the bottom. It is the largest sesamoid bone. The patella is located within the tendon of the quadriceps femoris muscle; it increases the strength of the quadriceps muscle and protects the joint from injuries. With the help of a ligament, which is a continuation of the quadriceps femoris tendon, the patella is attached to the tibial tubercle. It is well palpable under the skin. Its front surface is rough, and the back has an articular surface that contacts the femur [1, 2].

To ensure functional activity, bone components are supplemented with muscles, tendons, and cartilage. Supination of the lower leg (as well as pronation) is possible only to the degree of bending in the knee joint. Supinators of the lower leg are located on the lateral side of the joint: biceps femoris; lateral gastrocnemius head. Therefore, the pronator muscles of the lower leg are much stronger than the supinator muscles [3, 4].

A child's knee joint is subjected to enormous loads during growth and is sensitive to external and internal factors [5, 6]. The knee joint plays a dominant role in the limb's longitudinal growth; the growth zones grow so intensively that they can be compared to the work of a "bone nuclear reactor" [7, 8].

The morphogenesis of the knee joint is extremely complex and not fully understood. Most scientists noticed that various diseases most often manifest in this anatomical part of children [9, 10]. The bone growth plates of the knee joint are one of the largest in the child's body. It remains a mystery how exactly bones grow in length. This is a complex and poorly studied process in which a number of genes participate indirectly via the so-called morphogenetic proteins. The process of osteogenesis is extremely delicate and sensitive to any negative factors, so most scientists are looking for an answer to the question – what role the bone growth plates of the knee joint play in the development of a number of diseases [11, 12, 13].

Knee joint diseases can lead to various morphological changes and destruction in the joint structure [14]. The main morphological changes observed in knee joint diseases include the following [15]:

- Cartilage wear lesions: This is one of the typical signs of arthrosis. Cartilage tissue in the knee joint undergoes degenerative changes, becomes thinner, and loses its smoothness. This causes pain and limitation of movement and creates a risk of further damage to the joint [16].
- Osteophytes: Osteophytes are bony projections that can develop around the knee joint due to cartilage wear lesions. Osteophytes can lead to pain and limitation of movement [17].
- Inflammation of the synovial membranes: Pathological changes can affect the membranes surrounding the knee joint, such as the synovial membrane. Inflammation of these membranes leads to accumulation of fluid in the joint (joint effusion) and irritation of joint tissues [18].
- Tendon injuries: Wear-and-tear degeneration of the joint can cause irritation and damage to the tendons attached to the knee joint. This can cause pain and limitation of movement [19].
- Bone defects: In severe cases of knee joint disorders, bone defects may develop [20].

These morphological changes can be detected on X-ray examination, MRI (magnetic resonance imaging), or other imaging methods, as well as during surgical intervention for the knee joint disorder [21].

STUDY OBJECTIVE was to draw practicing physicians' attention to the risk of knee joint disorders in children during intensive growth with the aim of their timely detection and treatment.

Some disorders may develop without noticeable symptoms, while others may only become apparent after a certain period of time. It is important to take this into account during medical monitoring.

MATERIALS AND METHODS. We analyzed the results of studies on the diseases of the knee joint carried out by modern scientists over the past ten years. We also took into account the clinical observations of children receiving inpatient treatment at the regional children's hospital. The examination methods were radiography, computer tomography, magnetic resonance imaging, and ultrasound.

RESULTS AND DISCUSSION. The bones of the skeleton grow unevenly and intermittently. The peak height velocity is observed at 10–14 years in girls and at 13–18 years in boys; then, height velocity slows down, and after 19 years, height growth almost stops [22]. During the period of intensive growth, bones are susceptible to internal and external factors of shape formation (growth hormones, physiological loads, radiation, vibration, injuries, hypothermia, metabolic disorders, etc.) [23].

The distal femoral metaphysis is extremely active during this period, with a branched network of blood vessels ensuring intensive longitudinal bone growth.

Gross specimens of this part of the bone reveal the distal part abundantly covered with pores for blood vessels (Fig. 1, Fig. 2).

Figure 1 – A gross specimen of femur. The proximal and distal surface of the growth zones of the distal femoral metaphysis. Bone "reactor" for longitudinal bone growth. A huge area of blood supply

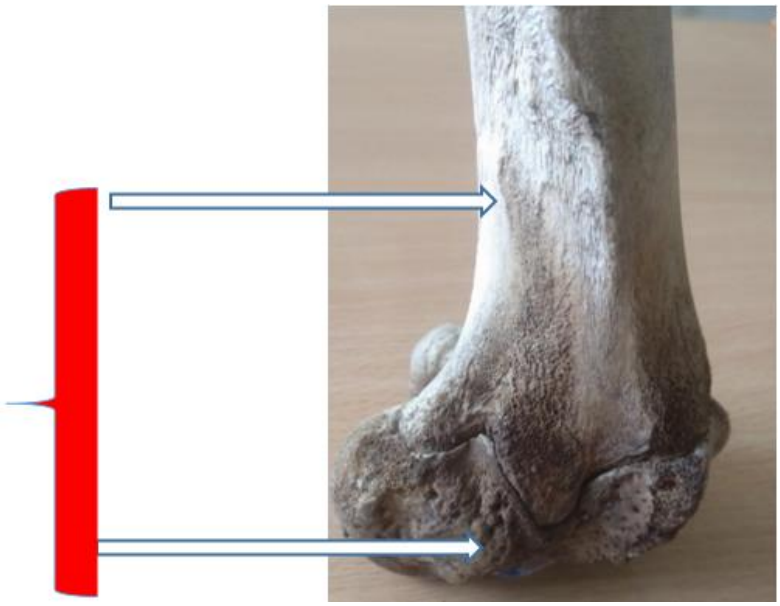

Figure 2 – A gross specimen of femur. Distal femoral portion. The epiphysis, metaepiphysis, and metaphysis are abundantly permeated with a network of holes for intensive blood supply

Therefore, this part is most vulnerable to various endogenous and exogenous negative factors, which is manifested by various dysplastic changes and diseases.

Further, we will consider the three most common diseases that are of great importance for an orthopedic practice in pediatrics.

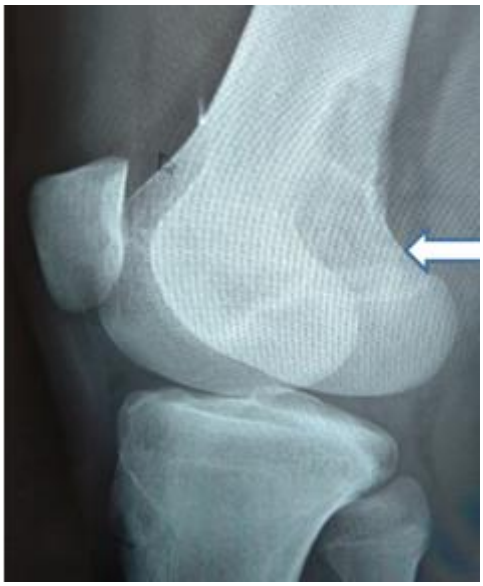

Figure 3 – A 16-year-old boy P. Lateral knee X-ray. FCD of the distal femoral metaphysis

For the proximal end of the tibia, the following location of the FCD is characteristic: medial part – 19.8% and lateral part – 11%. FCD is believed to be present in 30% of children and have an asymptomatic course (Fig. 4).

Sometimes, FCDs are multiple; they simultaneously affect the distal femoral metaphysis and the proximal tibial metaphysis and are often combined with osteochondropathy of the tibial tuberosity (Fig. 5).

This combination of FCD and osteochondropathy remains a big mystery for scientists. Dysplastic changes in connective tissue (cartilage and bones) may have common causes, but manifest themselves in different ways at different stages of the morphogenesis in the developing organism (Fig. 6).

FCD is not a neoplasm and, according to the definition of WHO, belongs to the group of developmental anomalies. It is poorly understood why foci of embryonic tissue are not replaced by normal bone tissue in time.

X-ray examination showed the variability of FCD, which gave a reason to determine the stages of the disease.

Stage A – a small oval lumen with clear edges adjacent to the growth zone.

Fibrous cortical defect (FCD) is located in the cortical layer of the knee bones (90% of cases). According to the published data, FCD is most often localized in the distal metaphysis of the femur (60%): medial condyle – 47.3% and lateral condyle – 12% of cases, respectively (Fig. 3).

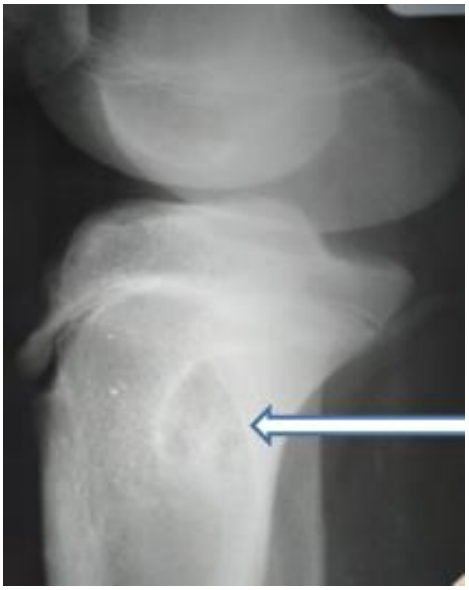

Figure 4 – A 15-year-old boy K. Lateral knee X-ray. FCD of the proximal tibial metaphysis

Stage B – in the growth process, the focus of FCD translocates towards the metaphysis, with clear contours resembling grapes.

Stage C – partial calcification in the form of a bone island.

Stage D – complete calcification of the lesion.

FCD resembles a non-ossifying fibroma (NOF); the difference is only in size. FCD: small intracortical foci (average size of 22 mm). NOF: located eccentrically, in the medullary zone, and of large size.

The main method of FCD examination is a two-dimensional X-ray. Including CT, MRI, and biopsy for examination is not advisable. From a medical point of view, this is not justified while a child is exposed to much higher doses of ionizing radiation. The typical location of the lesion, age-related features of the bone structure, past history, asymptomatic course of the disease, accidental detection of the lesion will help to diagnose this disease.

The main treatment is conservative with dynamic X-ray control. As a rule, FCDs disappear, replaced by normal bone tissue in the growth process. Large FCDs (more than half the perimeter of the bone) are subject to operative treatment when there is a risk of a pathological fracture.

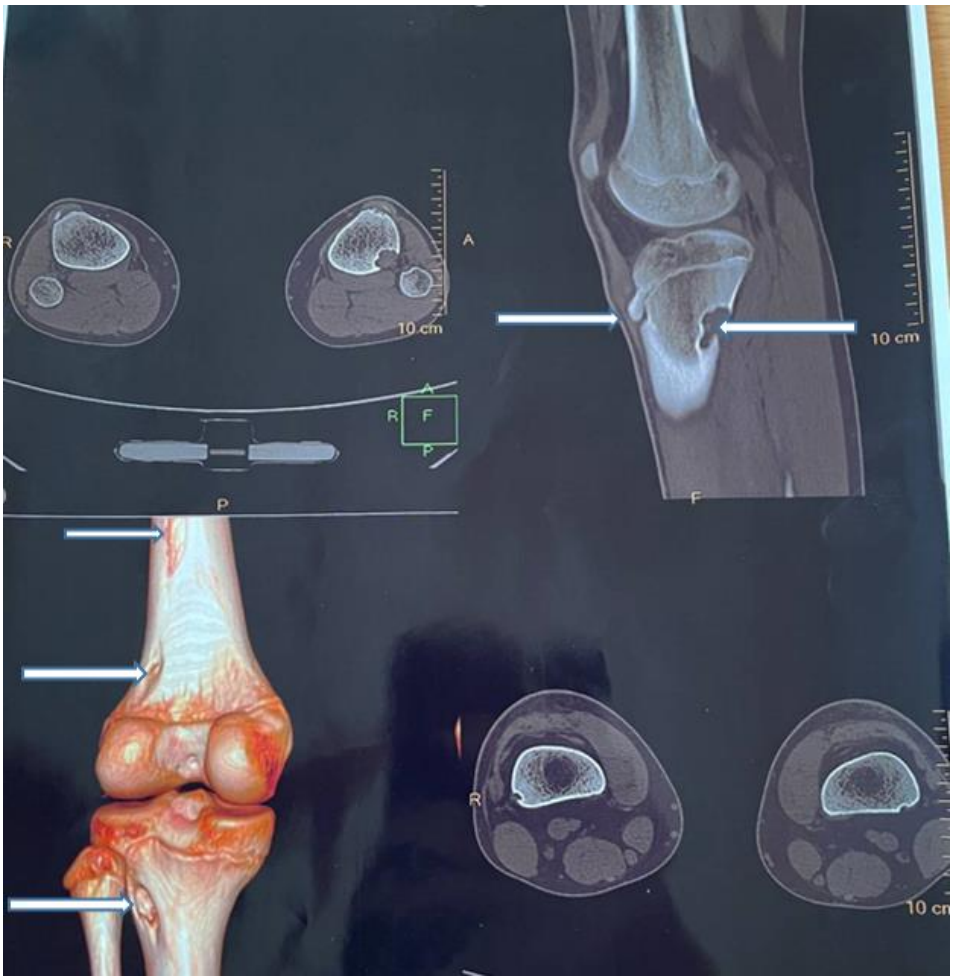

Figure 5 – MRI of the right knee. A 17-year-old boy. Massive FCD of the proximal tibia along the outer contour 2 x 1.5 cm. Spindle-shaped FCD of the distal femur portions at different levels 2 x 0.5 cm. Osteochondropathy of the right tibial tubercle (Osgood-Schlatter disease)

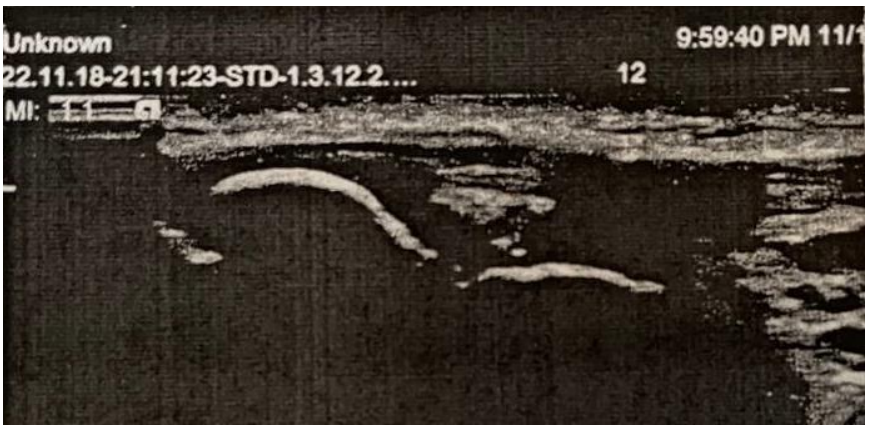

Figure 6 – Ultrasound of the right knee. Boy, 17 years old. Multiple femoral and tibial FCDs. Osteochondropathy of the right tibial tubercle (Osgood-Schlatter disease)

Osteogenic sarcoma (OS) mainly affects bones of enchondral origin and is localized mainly in long tubular bones [24]. The most frequent localization of OS is the distal metaphysis of the femur and the proximal metaphysis of the tibia (75% of cases).

OS is an extremely malignant form of primary malignant bone tumor in children. It develops from bone tissue cells and elements of mesenchymal undifferentiated cells, which, under normal conditions, participate in the development of

connective, cartilage, and bone tissue. The pathogenesis of OS is unknown. According to age, children with OS were distributed as follows: 6 children under 5 years, 27 children of 5–10 years, and 72 children of 10–15 years. Among these, boys were twice the number of girls.

The main clinical features of OS are rapid tumor growth, significant movement disorders, rapid spread of metastases along arterioles, pronounced pain syndrome, rapid wasting (cancer cachexia), signs resembling acute hematogenous osteomyelitis (high temperature, leukocytosis, anemia, increased ESR, and toxic granulation of leukocytes). Only isolated cases of OS metastasis to other bones were described; as a rule, it was a monostotic tumor. The basic method of OS diagnosis is X-ray examination revealing a triad of signs: bone destruction, cancer-associated periostitis, and spicules that grow into the surrounding tissues. CT and MRI are used when necessary (Fig. 7).

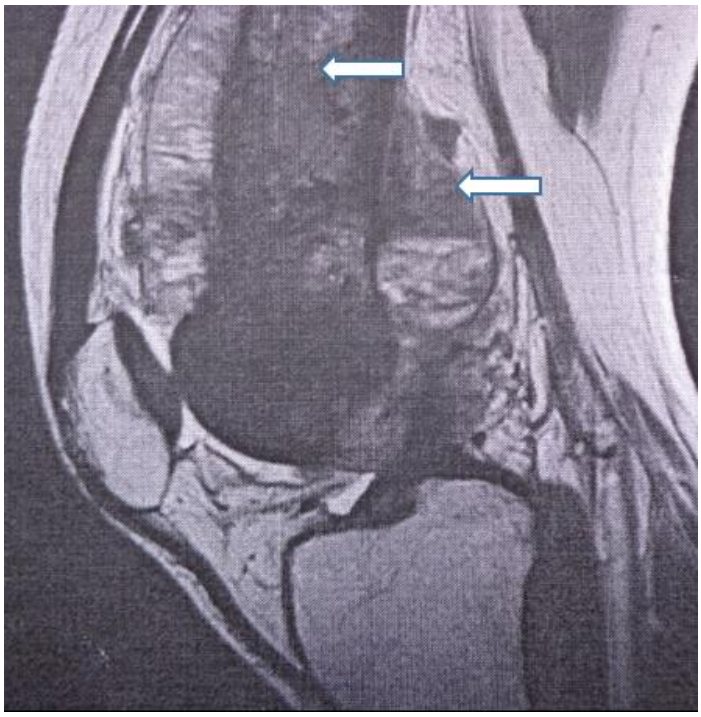

Figure 7 – MRI of the knee joint in a 17-year-old girl. Osteogenic sarcoma of the distal femoral end. Metaphysis and epiphysis are almost destroyed. Cancer-associated periostitis. Spicules that grow into the surrounding tissue

Treatment of OS includes three components: chemotherapy, radiation therapy, and surgical treatment. The treatment outcome depends on the child's age and the time since the onset of the disease. Mortality from OS is high. Cancer immunotherapy proposed by 2018 Nobel Prize laureates, immunologists James Ellison and Tasuku Honjo, is promising. They developed a fundamentally new approach to cancer therapy, namely a method of inhibiting the negative regulation of the immune system.

Juvenile osteocartilaginous exostoses (exostotic chondrodysplasia, Keith's syndrome, JE). It is a hereditary disease with a dominant type of inheritance. This is a defect in the epiphyseal cartilage development manifested by extra bone growth extending outward in the epiphyses and metaphyses of tubular bones. These lesions can be single or multiple. Most children were diagnosed with JE at the age of 9–12. The shape, size, and growth rate of JE can be different.

The largest number of single JE is localized in the distal portion of the femur and the proximal portion of the tibia and accounts for more than 70% of cases. Localization of multiple JE most often occurs in the distal femoral metaphysis and the proximal tibial metaphysis (70–80% of cases).

Thus, most often, JE is observed in the knee joint. More rarely, JE can be observed in other bones: fibular bone, humerus, foot bones, hands, ribs, pelvic bones, skull, clavicle, forearm bones. This can be explained by the fact that the distal metaphysis of the

femur and the proximal metaphysis of the fibula have the greatest growing power. As a result of JE, primary and secondary deformities and complications are observed due to the pressure of the tumor on the surrounding tissues, nerves, and vessels. If JE is large, secondary symptoms may develop: nerve compression, painful contractures, paresis, paralysis, skin sensitivity disorders, and vascular lesions.

There are three groups of large JEs.

1. Bulging of the bone.

2. JE pressing on the adjacent bone which causes its distortion.

3. Disorders of normal bone growth due to malfunction of the growth zone; bones grow crooked.

These individual manifestations can be isolated or combined. Small, isolated JEs can have an asymptomatic disease course and be diagnosed accidentally.

X-ray examination. In the initial stages, JEs are located near the bone growth zones near the metaphysis. As the child grows, JE gradually translocates from the epimetaphysis towards the diaphysis. The further JE is located from the growth zone, the longer the disease lasts. Linear JEs have a dense bone stem with normal cortical and cancellous bone tissue, clear contours, and no destruction or periosteal reaction (Fig. 8).

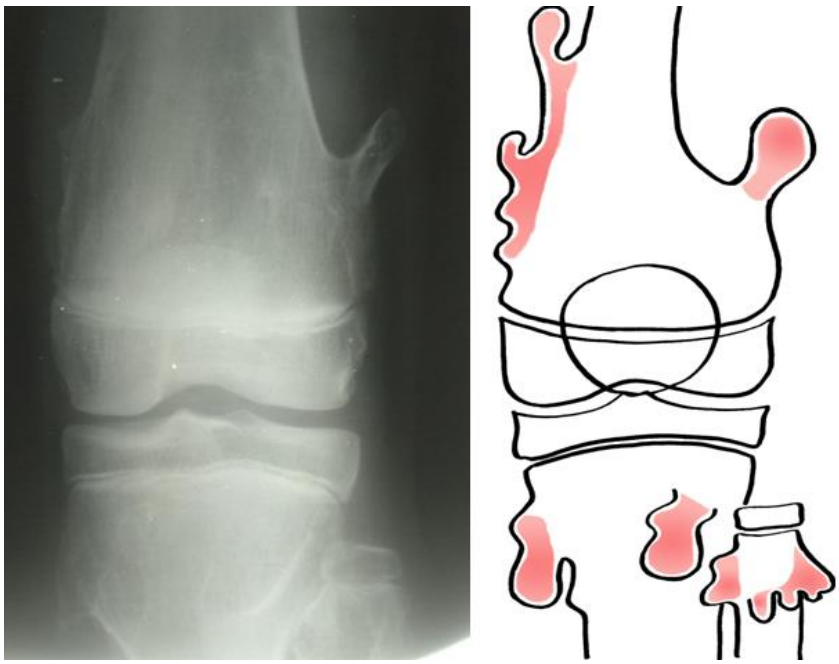

Figure 8 – Knee X-ray and a schematic representation of multiple juvenile osteocartilaginous exostoses of the knee joint in a 15-year-old boy M.

Valgus deformity of the knee joint. Short stature, shortening of limbs. Bilateral ulnar clubhand. S-shaped scoliosis of the thoracic spine. Ball-shaped JEs resemble cauliflower, with clear contours and no signs of bone tissue destruction. JE size on radiographs is always smaller than the clinical parameters because the top of the exostosis is covered with a layer of cartilage that is invisible to X-rays (Fig. 9).

Treatment of JE depends on the size, number of lesions, and location. Single, small JEs may be treated by non-surgical procedures. Operative treatment is required for JEs that cause pressure on blood vessels, nerves, adjacent bones, muscles, or tendons (Fig. 10).

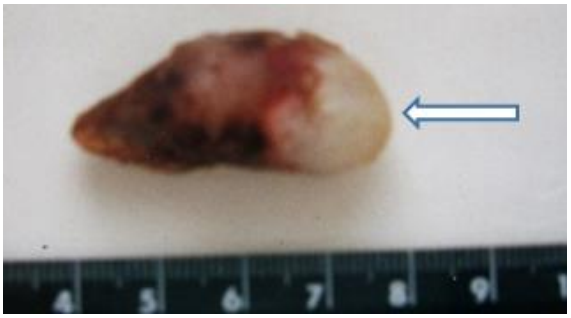

Figure 9 – A gross specimen of a juvenile osteocartilaginous exostosis removed from the proximal tibial metaphysis in a 14-year-old girl. The top of the exostosis is covered with cartilage (arrowed)

Relative indication for surgical treatment is for cosmetic purposes, especially in adolescent girls with JEs on the front surface of the tibia.

Gene editing is a promising method for correcting the human genome for the treatment of genetic and oncological diseases by introducing genes that can help the immune system attack cancer cells.

Gene therapy is a special type of medical treatment that involves introducing healthy genes into a human cell to replace defective or missing genes.

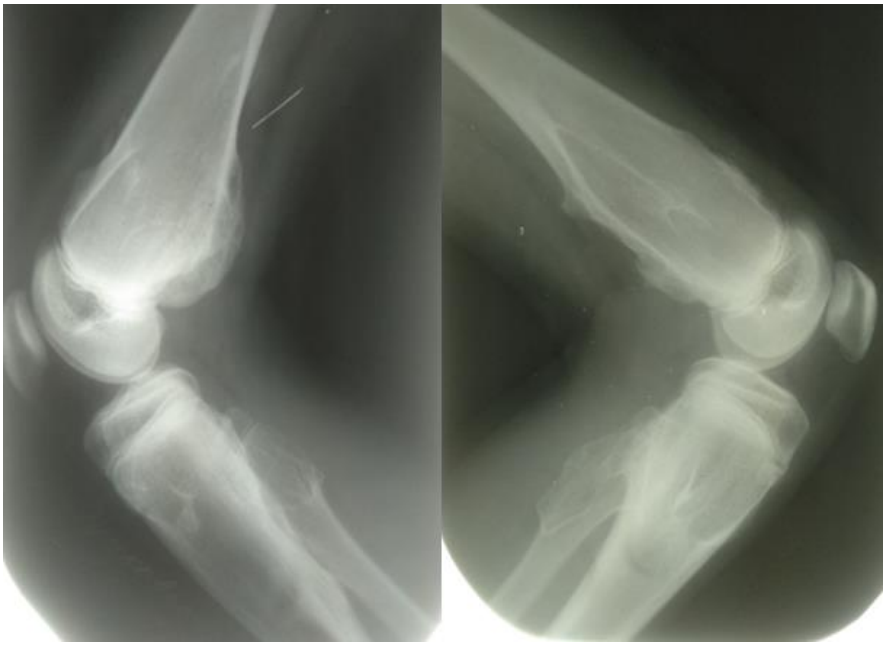

Figure 10 – Lateral knee X-ray. Multiple juvenile osteocartilaginous exostoses of the metaphyses of the knee in a 15-year-old boy M, causing gait disorders

From 2022, thanks to gene therapy (CRISPR – Clustered Regularly Interspaced Palindromic Repeats), the diseases that earlier were considered incurable, such as sickle cell anemia, hemophilia, non-Hodgkin's lymphoma, multiple myeloma, and Duchenne muscular dystrophy can be treated.

We hope that thanks to gene therapy, the treatment of OS, FCD, and JE, which most often affect the knee joint, will be successful, and its preventive potential will be used long before the first clinical signs of the disease.

CONCLUSIONS / ВИСНОВКИ

During the intensive growth of the child, the knee joint is most vulnerable to various endogenous and exogenous negative factors, which is manifested by various diseases.

Osteogenic sarcoma, fibrous cortical defect, and juvenile osteocartilaginous exostoses most often affect the weakest place of the knee joint – the metaphyses of the femur and tibia, which is the Achilles' heel of the child's skeleton.

PROSPECTS FOR FUTURE RESEARCH / ПЕРСПЕКТИВИ ПОДАЛЬШИХ ДОСЛІДЖЕНЬ

An in-depth study of knee joint diseases will make it possible to detect these disorders in children on time and help to prescribe adequate therapeutic treatment.

CONFLICT OF INTEREST / КОНФЛІКТ ІНТЕРЕСІВ

The authors declare no conflict of interest.

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None.

AUTHOR CONTRIBUTIONS / ВКЛАД АВТОРІВ

All authors substantively contributed to the drafting of the initial and revised versions of this paper. They take full responsibility for the integrity of all aspects of the work.

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