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Treatment approaches to flexion contractures of the knee

Diz fleksiyon kontraktüründe tedavi yaklaşımları

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The knee is the most affected joint in children with cerebral palsy. Flexion contracture of the knee is the cause of crouch gait pattern, instability in stance phase of gait, and difficulties during standing and sitting, and for daily living activities. It may also cause patella alta, degeneration of the patellofemoral joint, and stress fractures of the patella and tibial tubercle in young adults. Children with cerebral palsy may even give up walking due to its high energy demand in the adult period. The purpose of this article is to review the causes of the knee flexion contractures, clinical and radiological evaluations, and treatment principles in children with cerebral palsy. The biomechanical reasons of knee flexion deformity are discussed in detail in the light of previous studies and gait analysis data.

Key words: Cerebral palsy; child; contracture; gait; hip joint; knee joint; muscle, skeletal; tendons.

Beyin felcinde en çok etkilenen eklem diz eklemidir. Diz fleksiyon kontraktürü, çömelme pozisyonunda yürümeye, basma fazında stabilite kaybına, ayakta durma ve oturmada ve günlük yaşam aktivitelerinde zorluklara yol açar. Ayrıca, genç erişkin dönemde, patella alta, patellofemoral eklem dejenerasyonu, patella ve tüberküler kırıklarına da neden olabilir. Bu çocuklar ileri yaşlarda, yüksek enerji gerektirdiği için yürümeden bile vazgeçebilirler. Bu yazıda, beyin felçli çocuklarda diz fleksiyon kontraktürlerinin nedenleri, klinik ve radyolojik değerlendirmeler ve tedavi prensipleri gözden geçirildi. Ayrıca, yapılan çalışmalar ve yürüme analizi verileri ışığında diz fleksiyon kontraktürünün biyomekaniksel nedenleri ayrıntılı olarak tartışıldı.

Anahtar sözcükler: Beyin felci; çocuk; kontraktür; yürüyüş; kalça eklemi; diz eklemi; kas, iskelet; tendon.

Hamstring spasticity is the most common problem in cerebral palsy (CP). If left untreated, it results in knee flexion contractures.[1] Untreated flexion contractures lead to gradual deformation of the femoral condyles. In literature, crouch gait is reported in 69% of the general CP population, being 74% in diparetic and 88% in quadriparetic patients.[2]

Knee flexion contracture in children with CP causes crouch gait and excessive energy consumption during walking, making daily activities difficult such as standing, reaching to objects, and standing up from a chair. Quadriceps muscle works excessively as a result of knee flexion contracture. This increases the loading on several joints, especially on the patellofemoral joint and becomes an important problem causing anterior knee pain and stress fractures of the patella and tibial tubercle.[3-5]

Together with other common neuromuscular and musculoskeletal problems in CP patients, knee flexion contracture makes functional activities more difficult. A detailed evaluation is necessary to decide the appropriate treatment.[5]

The aim of this article is to assess the reasons of knee flexion contractures in children with CP, analyze assessment methods, overview compensatory mechanisms related to deformities, and discuss treat-

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ment alternatives for detected problems for selecting the most appropriate treatment for the patient.

Causes of knee flexion contracture

Knee flexion contracture may develop *(i)* after hip flexion contracture and increased anterior pelvic tilt, or due to *(ii)* hamstring spasticity or contracture, *(iii)* gastrocnemius tightness, *(iv)* triceps surae weakness following surgery, or *(v)* posterior capsule contracture that develop over time.[6-10]

Hamstring spasticity or contractures

Medial and lateral hamstrings attached to the proximal tibia are knee flexors and hip extensors.[11] Three-dimensional gait analysis studies showed prolonged medial hamstring muscle activity resulting in increased hip extensor muscle strength.[9] It has long been believed that children with crouch gait have hamstring spasticity and this has been the focus of several studies. Many have shown that hamstring lengths are usually normal in these children.[5,6,9,12] Even though the length of the hamstring is normal, it appears contracted when pelvic tilt increases. Hamstring release in this condition will further increase the pelvic tilt. Thus, decision for hamstring release operation should be made after clinical tests and three-dimensional gait analysis which provides dynamic length measurement of hamstring muscles.[5,9,12,13]

Long-standing knee flexion contracture imposes a heavy burden on knee extensor muscles that results in patella alta. This makes knee extension by quadriceps difficult during the stance phase. Therefore, no matter how effective knee extensors are, the lever arm of the knee will not be sufficient for full knee extension. In severe cases, this condition gives rise to patella and tibial tubercle fractures.[4]

Knee flexion contracture following hip flexion contracture and increased anterior pelvic tilt

Anterior pelvic tilt and knee flexion are usually increased in children with CP while standing or during the stance phase of gait.[5,7,9,13] Normally, ground reaction force (GRF) passes from the center of the hip and knee while standing. In children with crouch gait, the GRF passes from anterior of the hip and posterior of the knee because of hip flexor tightness (Fig. 1). Lumbar lordosis is increased. To compensate lumbar lordosis, superior part of the trunk moves back and the knee is flexed.[5] This crouching posture at the hip and knee leads to knee flexion contracture over time. Increased knee flexion associated with increased anterior pelvic tilt contributes to knee flexion contracture in time. As anterior pelvic tilt continues to increase, knee flexion during standing and walking will increase. Usually this might be perceived as an increase in knee flexor tightness clinically, necessitating hamstring lengthening.

Gastrocnemius tightness

The gastrocnemius muscle is the primary ankle plantar flexor and knee flexor.[10] As knee flexion increases, flexion moment of the gastrocnemius at the knee gradually increases. There is selective motor deficit in the gastrocnemius muscle of children with CP and spasticity dominates. For this reason, it is usually stretched, causing early heel rise, toe walking, and heel valgus during the stance phase of gait.[5] Gastrocnemius causes toe walking distally and this forms an extra proximal moment pulling the knee to flexion.[10] This moment directly affects acceleration of the knee to flexion at the end of the stance phase and contributes to maximum flexion of the knee during

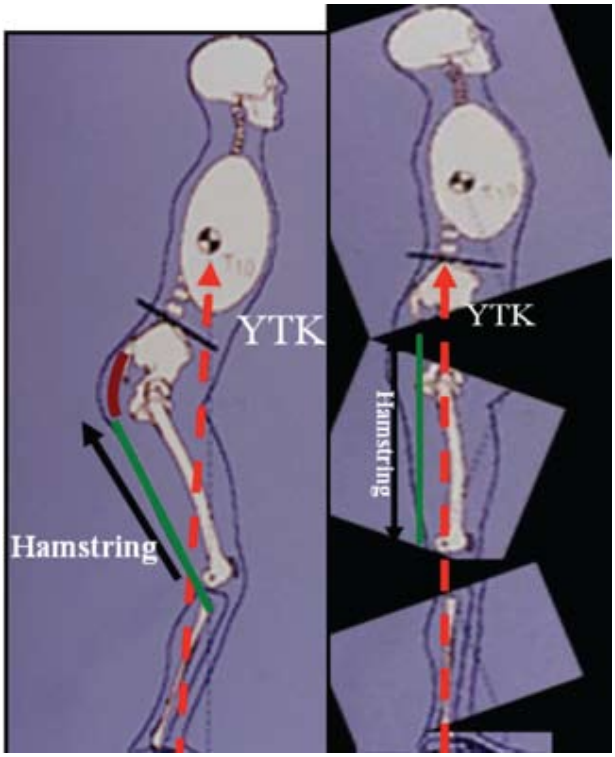

Fig. 1. Location of the ground reaction force (GRF) in crouch posture.

the swing phase.[14] Gastrocnemius-soleus complex is the most important part of plantar flexion-knee extension couple which provides adequate knee extension during the stance phase.[5] While gastrocnemius muscle stretch occurring in the proximal contributes to increased knee flexion, it loses its important role in extension during the stance phase and becomes a knee flexor.[15]

Postoperative triceps surae weakness

Soleus generates 40% to 50% of the total force needed to straighten the body during the first one-third of gait cycle.[5] This activity enables knee flexion by producing a moment against the GRF passing from the anterior of the ankle. This plantar flexor effect on the ankle and extensor effect on the knee is called plantar flexion-knee extension couple. Thus, extra muscle activation for knee extension during the stance phase is eliminated. In spastic diplegia and quadriplegia, the gastrocnemius and hip flexor muscles stretch while the soleus, vastus muscles, and gluteus maximus elongate. Any procedure that weakens the soleus such as selective dorsal rhizotomy or Achilles tendon release results in deficiency of the soleus to retract the tibia. As a result, ankle dorsiflexion and knee flexion increase during the stance phase, leading to the crouch gait pattern and knee flexion contracture.[5]

Posterior knee capsule tightness

Knee flexion contracture leads to shortening and thickening of the posterior capsule and shortening of the sciatic nerve.[16] For this reason, particularly in fixed knee contractures of 30° to 40°, it is suggested that hamstring release be combined with posterior capsule release.[5,16]

Assessment methods of knee flexion contracture

Knee flexion contractures are assessed clinically and by laboratory and imaging studies.

Clinical assessment methods

Patients on their mothers' lap or walking with the help of an assistive device (walker, canadian crutch, cane) should be observed for lower extremity position and activity. Posterior pelvic tilt, lumbar kyphosis and forward bending of the trunk while sitting give clues about possible hamstring contracture and/or trunk hypotonicity.

In order to determine the underlying cause of knee flexion contracture, hamstring muscles should first be assessed. The popliteal angle never exceeds 50° throughout the life. However, it may increase up to 90° in CP. Thus, in the first instance, the popliteal angle and hamstring contracture should be assessed (Fig. 2). During this test, the physician observes whether hamstring spasticity and dynamic contracture are present by increasing the speed of passive knee extension.[9] Measurement of the hamstring length is important to distinguish between normal and contracted muscle lengths (Fig. 2b). The popliteal angle is measured both unilaterally and bilaterally. Hamstring shift is calculated by subtracting unilateral popliteal angle from bilateral popliteal angle (Fig. 2a, b).

Unilateral popliteal angle is measured while the patient's existing lumbar lordosis is preserved and the contralateral hip is in neutral position. It gives information on functional hamstring contracture. However, bilateral popliteal angle is measured by

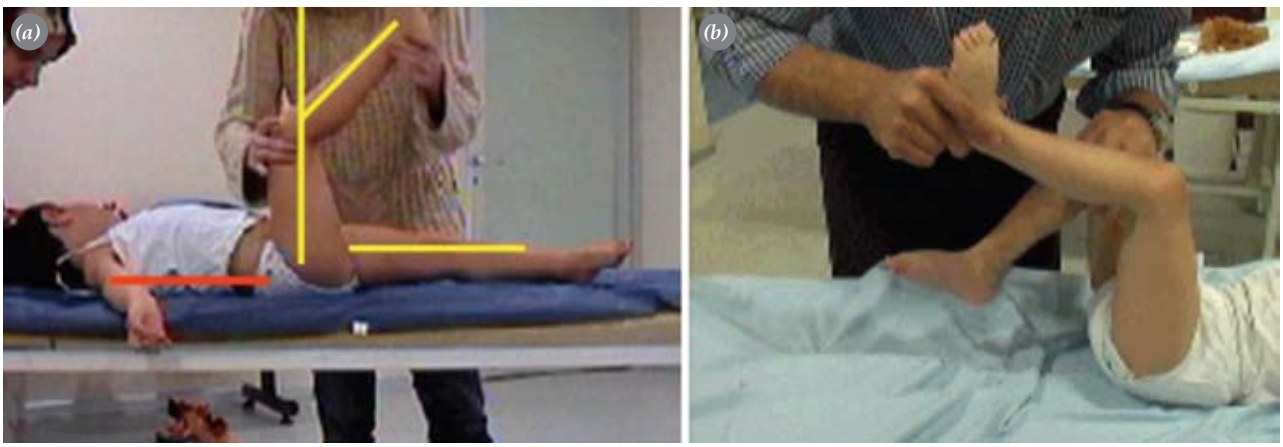

Fig. 2. Determination of the **(a)** popliteal angel and **(b)** hamstring shift.

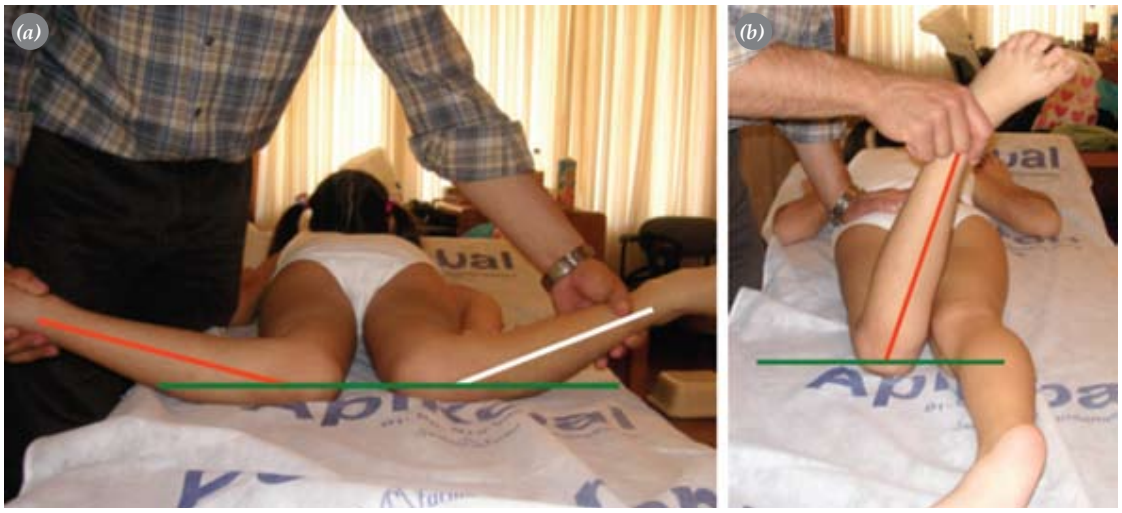

Fig. 3. **(a)** Increased internal rotation and **(b)** decreased external rotation of the hip in femoral anteversion.

bringing the contralateral hip to flexion until the ASIS (anterior superior iliac spine) and PSIS (posterior superior iliac spine) are vertical. Then, ipsilateral popliteal angle is measured, which provides information about true hamstring contracture (Fig. 2b). The difference between the two angles shows if hamstring length is normal or not. If the difference is greater than normal, this means that hamstring tightness decreases with posterior rotation of the pelvis, suggesting normal hamstring length. In general, anterior pelvic tilt is increased in patients with spastic diparesia and quadriparesia, with a greater hamstring shift. If the clinician determines functional hamstring contracture without bringing pelvic tilt to normal and performs hamstring release, this will further decrease hip extensor moment of hamstrings, increase pelvic tilt, and as a result of contracted hip flexors, end up with knee flexion during gait.[10,13,17] Delp et al.[13] found that every 1° increase in pelvic anterior tilt increased bilateral popliteal angle by 2°. Thus, a difference of greater than 20° between unilateral and bilateral popliteal angles often suggests hip flexor contracture, weak abdominal muscles and/or weak hip extensor muscles. Considering the difficulties in clinical measurement of dynamic hamstring length, evaluation of the patient in gait analysis laboratory is necessary.[10]

Clinically gastrocnemius-soles tightness should be assessed separately like the hamstring muscles, because the soleus muscle is usually normal or extended in most CP patients.[7] The primary role of the soleus is to control the forward movement of the tibia during the mid-stance phase, thereby enabling knee extension. Radical soleus stretching exercises and surgical procedures such as Achilles tendon release that cause excessive lengthening of the soleus weakens the muscle, resulting in increased knee flexion during the stance phase and knee flexion contracture. The Silfverskiöld test performed under anesthesia is the most appropriate method to identify gastrocnemius contracture and excessive soleus length. But, the positioning of the mid and front segments of the feet should not interfere with the test. Thus, the subtalar joint is brought to as much neutral or varus position as possible during the test.[10]

Delay in walking and hip flexion cause high femoral anteversion in children with CP. With increased anteversion, the child walks in internal rotation and pelvic tilt increases. Increased femoral anteversion is associated with kinetic and kinematic changes that result in crouch posture. Therefore, correction of rotational deformities is first line treatment of crouch posture in CP patients if orthopedic procedures are considered (Fig. 3).[5]

Clinically video-based observational gait analysis (VBOGA) may help understand the influence of knee flexion contracture on walking. The clinical application of this method can be made by a single specialist experienced in VBOGA. It has been shown that observation of the gait in slow motion and evaluation of the gait in fewer phases (dividing the stance phase to three parts only) with a simple evaluation form increase reliability.[18,19] Even though

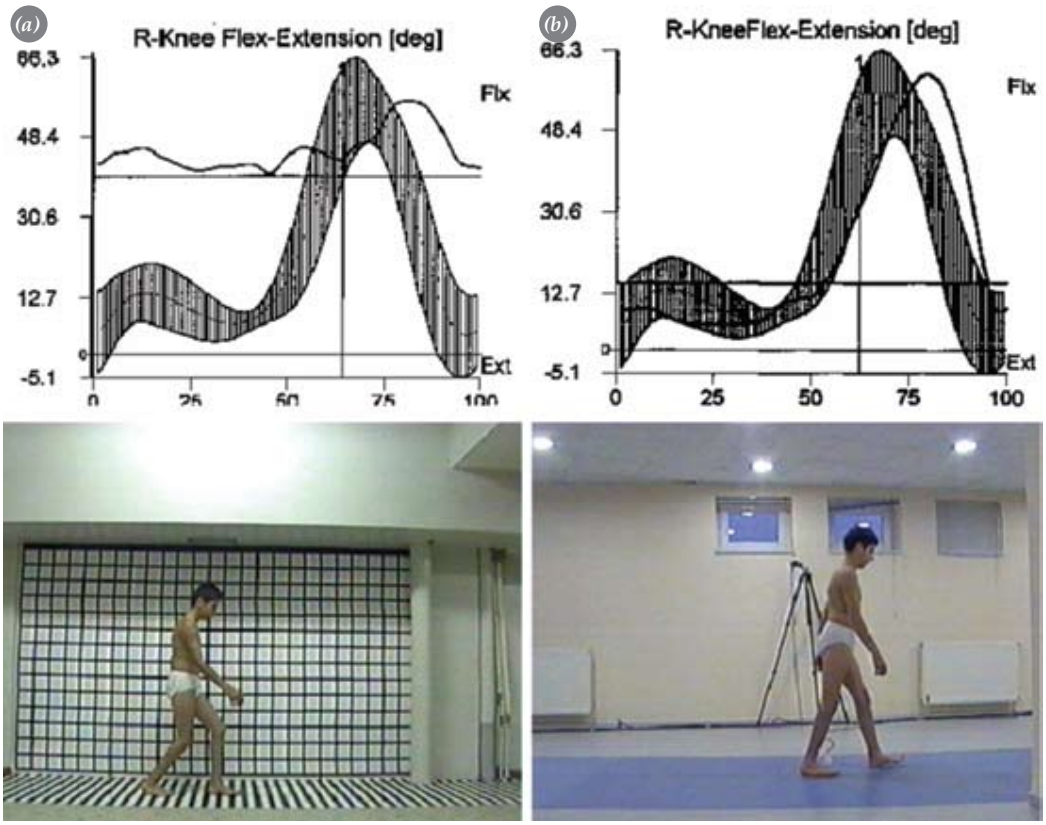

Fig. 4. **(a)** Crouch gait with increased hip and knee flexion, anterior pelvic tilt, and ankle dorsiflexion (preoperative). **(b)** Gait is close to normal (postoperative).

detailed and time consuming, Perry's observational gait analysis evaluation form developed in 1992 is still used for gait assessment.[3,19]

Computerized gait analysis

In CP patients with knee contracture, kinetic analysis of gait shows increased knee flexion in the stance phase and, despite minimal movement of the knee in the swing phase, an increase in the knee extensor moment during the loading phase, and increased quadriceps activity, depending on the severity of contracture. This is because the extensor muscles are trying to bring the knee to extension.[5] Knee flexion contracture is not examined in the laboratory solely in the knee context, investigation of its effects on other joints is helpful for the treatment.

In spastic diplegia and quadriplegia, usually crouch gait is seen (Fig. 4). Foot dorsiflexion and knee flexion are increased due to soleus weakness and/or femoral anteversion and there is a continuous increase in the knee extensor moment. EMG shows increased hip and knee extensor muscle activity and high energy consumption.[5] With addition of ankle plantar flexion during the stance phase to this picture, the knee is flexed in the beginning of the stance phase.

Management of knee flexion deformities

The goals of treatment should be as follows:

1. Decrease knee flexion during gait,
2. Increase stride length,
3. Decrease patellofemoral joint load, and
4. Increase strength (durability).

In principle, the pelvis, hip, knee, and ankle should be assessed as a whole.

1. Rotational deformities (femoral anteversion, tibial torsion, varus-valgus-adductus deformities of the feet, hip subluxation, etc.) should be corrected.
2. Shortened muscles should be lengthened (care should be given to biarticular muscles).
3. Elongated muscles should be shortened.

4. Fixed joint contractures should be corrected.
5. Ground reaction orthosis may be needed.

In mild knee flexion contractures, immobilizer, angle adjustable KAFO, and botulinum toxin A injection can be used in children younger than 5 years age whose popliteal angle is smaller than 60 degrees.

Botulinum toxin A injection should be used in carefully selected patients. Corry et al.[20] showed that anterior pelvic tilt increased after botulinum toxin A injection to hamstring muscles in 10 CP patients with crouch gait. Care should be taken not to cause isolated hamstring weakness. As the hamstring is a hip extensor, its excessive weakness increases anterior pelvic tilt.

If knee flexion contracture is between 10 to 30 degrees, surgery is necessary in patients older than 10 years. Hamstring lengthening and if necessary posterior knee capsulotomy operations can be performed. Gradual correction with casting may be rarely necessary. No deformity has developed in femoral condyles.[1]

Indications of hamstring lengthening

1. Popliteal angle is greater than 50° under anesthesia and knee flexion is greater than 20° while standing,
2. Fixed knee contracture is greater than 5°-10°,
3. Having difficulty sitting and standing without a wheelchair,
4. Disappearance of lumbar kyphosis while sitting with hamstring relaxation.

Semitendinosus, semimembranosus, and long head of the biceps femoris are knee flexors and hip extensors. Semitendinosus and semimembranosus lengthening may correct the popliteal angle separately by 10 to 15 degrees. Excessive hamstring lengthening should be avoided because it causes anterior pelvic tilt and stiff knee gait. If the biceps femoris is not lengthened intramuscularly, it may lead to tibial external rotation.

Should we use passive hamstring stretching?

Instead of long-term aggressive hamstring stretch exercises, functional stretching and relaxation methods can be more effective in relaxing the child's tight muscles and whole body during play.[21]

If fixed knee flexion contracture is greater than 30°, the distal ends of the femoral condyles may become flattened, disrupting the articular surface of the tibiofemoral joint. In this situation, distal femoral extension osteotomy is a better option than capsular release.[1] Capsular release in the presence of flattened femoral condyles results in decreased slide/roll behavior of the tibiofemoral joint, turning the knee joint a hinge joint rather than a sliding one around the condyles. Hamstring lengthening, patellar tendon plication, and distal tibial tubercle transfer may be performed in the same session after supracondylar osteotomy. Since distal femoral extension will cause femoral shortening, the development of sciatic nerve palsy will be much rarer. Supracondylar closing wedge extension osteotomy of the femur is an effective and safe procedure for the correction of knee flexion contracture in adult patients with spastic diparesis. One advantage of this operation is femoral shortening and relief of neurovascular structures. Sciatic nerve neuropathy and vascular insufficiency are rare (Fig. 5).[9]

Temporary growth arrest of anterior femoral epiphysis is a new method used in the management of knee flexion contractures (Fig. 6).[9] This method should be used in patients around 13 years of age,

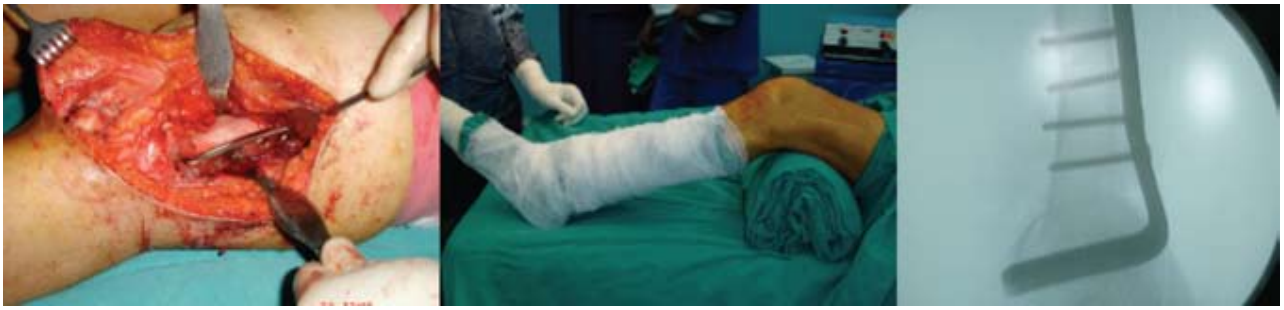

Fig. 5. Preoperative and postoperative images of a patient undergoing supracondylar femoral osteotomy and patellar tendon plication.

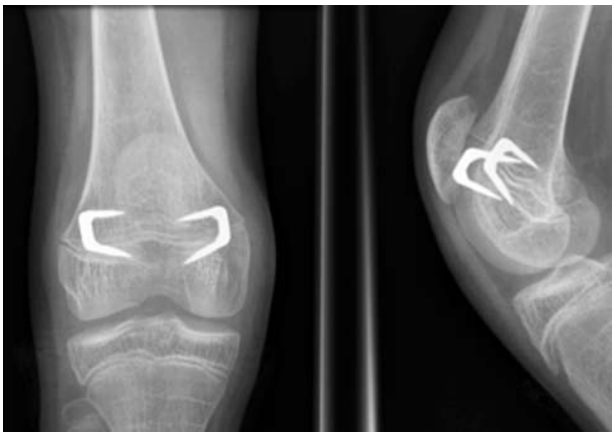

Fig. 6. Temporary growth arrest of anterior femoral epiphysis.[23]

with 5 to 20 degrees of flexion contracture. Genu recurvatum deformity develops in patients less than 13 years of age and deformity correction is incomplete in patients older than 14 years.

Results and problems

Potential problems related to treatment include:

1. Recurrence of knee flexion deformity.
2. Increases in postoperative lumbar lordosis and anterior pelvic tilt should be avoided. If present, hip flexion contracture should also be corrected.
3. In the presence of quadriceps spasticity or if the hamstrings are too weak, stiff knee gait or genu recurvatum will ensue. Distal rectus femoris transfer will solve the problem.
4. Partial correction of knee flexion deformity.
5. Crouch gait may develop due to excessive triceps lengthening. Solution is ground reaction AFO usage.
6. Sciatic nerve lesion.

As a principle, the pelvis, hip, knee, and ankle should be assessed as a whole. Rotational deformities should be corrected initially. Muscle length should be balanced, fixed joint contractures should be corrected, and recurrence of fixed contractures should be avoided by using ground reaction orthoses.

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