Femur Deformities

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Femoral deformities are the main subject of the Ilizarov surgery. They can be congenital or acquired. Many different methods can be used to correct these deformities. Acute or gradual correction and internal or external fixation can be chosen according to etiology of disease and experience of the surgeon. We will mention mainly our experience.

This book mentions about many of the femoral deformities in different sections. In our chapter we will mention mainly the subjects that are not discussed in other chapters.

28.1 Proximal Femur Deformities

28.1.1 Coxa Vara

The line that connects the center of the femoral head and the tip of the trochanter major makes an angle between the mechanical axis of the femur about 90° (90 \pm 5). This angle is called lateral proximal femoral angle (mLPFA) and normally it's 90°. If it is less than 85°, deformity is called coxa valga; if it is more than 95°, the deformity is called coxa vara (Fig. 28.1) [19, 20].

Another definition can be made with the angles between femoral neck midline and corpus. This angle is about 120° and less is called coxa vara deformity. Incidence is 1/25,000 without any distinguish of sex and ethnicity. Thirty percent of the case are bilateral. Hoffa first described the term of developmental coxa valga in 1905 and classified as developmental, congenital, dysplastic, or acquired [1–9].

Another terms for developmental coxa vara are congenital coxa vara, infantile coxa vara, and cervical coxa vara. The hips are normal at birth. Because of the cartilage defect on femoral neck, trochanter major enlarges, neck shortens, and angle between femoral neck and corpus decreases. The physis gets vertical in time with development and varus deformity reveals (Fig. 28.2).



Fig. 28.1 Coxa valga (a) and coxa vara (b) deformities

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Fig. 28.2 Characteristic x-ray view of coxa vara

Developmental coxa vara is a continuing process. Deformity must be followed until patient's skeletal maturation stops. After early surgeries deformity can reemerge. Due to the excessive advance of the deformity, growing plate medially positioned around femoral neck can be injured which augments the deformity more [4–7].

On radiological views typically angle between femur neck and corpus reduces and neck shortens and, because of the fact that great trochanter remains the same, relatively enlarges.

Vertically positioned growth plate, short extremity, enlargement of the proximal physis, triangleshaped bone fragment medially positioned around the neck and radiolucent lines both medially and laterally, short neck, and decreased anteversion angle are other findings (Fig. 28.2) [5–8].

Decreased angulation between femoral neck and corpus can be seen on PFFD and congenital short femur. However, deformities are more severe with increased shortness, three-planar deformity, and soft tissue contractures on PFFD (Fig. 28.3).

Fairbank classifies coxa vara in three subgroups: congenital, developmental, and acquired. Congenital reasons are proximal focal femoral deficiency (PFFD) and congenital short femur.



Fig. 28.3 Short femoral neck, increased anteversion angle, and radiolucent lines both positioned medially and laterally with triangle-shaped bone fragment around the neck

Acquired reasons are slipped capital femoral epiphysis (SCFE), sequelae after osteonecrosis of the femoral head which can be seen after trauma, infections, septic necrosis, Legg–Calve–Perthes disease, developmental hip disorder, postreduction osteonecrosis, pathological bone diseases, osteogenesis imperfecta, fibrous dysplasia, rickets with renal osteodystrophia, osteopetrosis, and other bone-weakening conditions [4].

On bilateral situations, skeletal dysplasia (cleidocranial dysostosis, metaphyseal dysostosis, Jansen-type and Kozlowski-type dysplasia, spondylometaphyseal dysplasia) must be taken into consideration for differential diagnosis. On pathogenesis there is an enchondral ossification defect on the medial side of the neck which causes increase on varus deformity [5–9].

Patients with developmental coxa vara might feel the need to have medical examination because of slight limping, owing itself to the weakness on abductor muscles and limb length discrepancies. If the pathology is monolateral, Trendelenburg gait is common.

Particular lines and angles are described on AP radiographs which are neck–corpus angle, head–corpus angle (angle between long axis of femur and perpendicular line to the physis), and Hilgenreiner-physeal angle (angle between Hilgenreiner line and physis line). Hilgenreinerphyseal angle is the most important angle for prognosis. A study of Weinstein et al. gives the normal value of this angle of $16^{\circ} (0-25) [13, 17]$. For developmental coxa vara if this angle is more than 60°, the deformity can get worse; however, if the angle is less than 45°, progression of deformity is not expected even regression can occur. If H-E angle is between 45 and 60°, the patient must be followed up closely [13, 17]. There is no primary treatment for the developmental coxa vara because of the unknown etiology. Nevertheless, secondary deformities are the primary concern. On a horizontal physis while compressive forces are positioned medially, tensile forces are positioned laterally. In coxa vara compressive forces are normally expected to make sheading forces because of more vertically positioned physis. As a result, the medial side of the femoral neck doesn't elongate because the expansion of the lateral side and tip of the trochanter major elevate and deformity increases [13]. Because of the shortness on the neck of the femur, abductor distance and compressive forces on femoral neck expand which leads more bending on femur [1-9].

Diagnosis is important on developmental coxa vara (DCV). The goal for treatment is to obtain normal varus angle, normal head-corpus angle, turning abnormal sheading forces into compression forces on physis. Consequently, normal ossification and union are acquired. The secondary goal is to obtain normal muscle tone for abductor muscle group which corrects biomechanics. Asymptomatic patient that has normal H-E angle must be investigated for skeletal dysplasias and must be evaluated for limb length discrepancies if it's unilateral. Periodic x-ray imagining must be performed until skeletal maturation. If H-E angle is between 45 and 59, more frequent serial imaging is necessary. If the patients are symptomatic on this group, deformity gets worse or H-E angle gets more than 60° surgical intervention and becomes substantial [13–17]. If patients

become symptomatic and have limping or Trendelenburg gait or progressive deformity with more than 60° of H-E angle, $90-100^{\circ}$ of or less femur neck–corpus angle need surgery. Conservative approaches such as bed rest, abduction splints, traction, and exercise are useless in that condition. For surgery, proximal femur valgization, flexion, and derotation osteotomy must be performed. With valgization of femur, anteversion is increased, and derotation is necessary for distal femur [7–11].

On literature besides the osteotomies for valgization described by Langenskiold and Pauwels (Fig. 28.4), Borden also described another form of valgization osteotomy which is performed at subtrochanteric region. Early-age osteotomies come with the disadvantage of recurrence because of remodeling capacity and failure for fixation of proximal femur [10, 11]. On the other hand, with the delayed osteotomies, acetabular dysplasia may increase. For these reasons the optimal timing for surgery is the time when sufficient ossification is detected. The most secure fixation can be obtained with closed-wedge osteotomy and plate fixation. Besides, external fixation can be also applied with subtrochanteric osteotomy. External fixators have certain advantages of gradual correction options. Osteotomies performed from a distance to CORA come with the disadvantage of translations while correction, named as osteotomy rule 2 described according to Paley. This translation remodels on the maturation, remodeling process of the patient, and doesn't pose an obstacle to our treatment (Fig. 28.5).

With the coxa vara deformity, valgus deformity can be developed in lower extremity (Fig. 28.6)

At this point valgization will be performed on the hip which exerts difficulties on walking because of the increase of total valgus. With the preoperative anatomical and surgical planning performed on orthoroentgenogram, necessity for distal osteotomies and controlled corrections (epiphysiodesis, gradual correction with external fixator) must be determined (Fig. 28.7).



Fig. 28.4 (a) Postoperative x-ray for bilateral Pauwels valgization osteotomy during the childhood. Osteotomy fixation is done with plates. (b) Subtrochanteric osteot-

Proximal focal femoral deficiency is a disorder of femur with shortness of femoral neck and shaft with discontinuity. It is a rare congenital disorder with partial absence of proximal femur, and abnormal ossification PFFD is 15% bilateral, and 50% of patients have additional congenital abnormalities. Not only the hips and the femur but also the knee joint is affected; it is also a reason for coxa vara

omy for the coxa vara deformity in adolescence period. Fixation is done with external fixator. Expected translation according to osteotomy rule 2

deformity. Subject will be discussed in another chapter [16].

Some pathologies, tumors (fibrous dysplasia), metabolic disorders (rickets), and repetitive pathological fractures (osteogenesis imperfecta) can cause coxa vara also. The aim of the treatment of such pathologies is to correct hip biomechanics and prevent secondary complications (Figs. 28.8, 28.9, and 28.10).



Fig.28.5 Compensatoire valgus deformity on lower extremity due to coxa vara for the patient with epiphyseal dysplasia. MAD is valgus although that we have coxa vara deformity on the hips



Fig. 28.7 Postoperative x-ray of a patient that undergone a surgery of valgization osteotomy for the right hip joint, hemiepiphysiodesis on distal femur, and correction with external fixators at proximal tibia with peroneal nerve release. This patient's valgus deformity originated from distal femur is expected to be corrected with the effect of hemiepiphysiodesis gradually. But for early weight-bearing deformity at proximal, tibia is corrected with external fixation. In case of correction of more than 20-degree valgus deformity, we suggest peroneal nerve release



Fig. 28.8 Coxa vara due to rickets



Fig. 28.9 Coxa vara due to osteogenesis imperfect. Intramedullary rodding corrected the diaphyseal deformity but not the proximal femoral deformity



Fig. 28.10 Coxa vara due to fibrous dysplasia. Internal mechanical support is needed to correct and keep it

28.2 Coxa Valga

When the angle between femoral neck and shaft exceeds 135° , the deformity is called coxa valga. Angle between the mechanical axes and tip of the trochanter major, called mLPFA, is less than 85° . Normally this angle is about 90° (Fig. 28.1) (varies between 85 and 95°) [20].

Coxa valga can be acquired or congenital. A femoral neck fracture, metabolic bone disease, multiple exostosis, osteogenesis imperfecta, Paget's disease, fibrous dysplasia, cerebral palsy, or developmental hip dysplasia can cause a coxa valga deformity (Fig. 28.11).

With a varization osteotomy that enables correction at the intertrochanteric region, compression forces spread to a wider area at the joint, and concentric reduction is obtained for the hip joint (Fig. 28.12).

If we obtain more concentric reduction with the abduction internal rotation x-ray of the hip joint, varization derotation osteotomy can be per-



Fig. 28.11 Bilateral coxa valga deformity on a patient with cerebral palsy, with increase in anteversion



Fig. 28.12 Bilateral varization, derotation osteotomy for coxa valga. As the CORA of the deformity in at the physis, with an osteotomy at the trochanteric region we need translation not to have osteotomy rule 3

formed. Because of the improvement on the joint consistency and extended circulation due to hip region osteotomy, further arthrosis can be prevented (Fig. 28.13).

At clinical practice coxa valga is presented with increase anteversion angle which leads to intoeing. At these cases for protecting the biomechanical features of the hip, derotation procedure must be added to varization osteotomy. Until 8–10 years old, the hips are tent to get external rotation posture. If the deformity is unilateral or physical examination on prone position reveals 80° or more of internal rotation deformity and on regular examinations, no progress is



Fig. 28.13 A patient with coxarthrosis at the right hip joint due to developmental hip dysplasia treated with varization, derotation, and translation osteotomy

detected clinically; surgical intervention can be planned earlier. Otherwise it is much proper to wait until 10 years old according to our experiences (Fig. 28.14).

For the treatment of osteoarthritis for dysplastic hip joints, Pauwels 1 (varization) and Pauwels 2 (valgization) osteotomies can be performed. If the hip joint is reducted on the abduction x-ray images, varization osteotomy, however, for the adduction x-rays, is the best option. If there is no consistency in both situations, the best option is translation osteotomy described by Mc Murray that translates distal fragment medially that allows the mechanical axis to cross more medially which eventually decreases the forces to the hip with adductor and iliopsoas loosening. Intertrochanteric femoral osteotomies (varus, valgus, flexion, extension, translation) that improve the joint surface consistency and that increase contact area cartilage of the hip, replace joint rotation center medially.



Fig. 28.14 A patient with bilateral valgus deformity with increase anteversion deformity treated with derotation osteotomy. Fixation is performed with intramedullary nailing on the right side and plate on the left side

The degree of derotation performed during these osteotomies is determined according to lower extremity rotational profile obtained with clinical examination under anesthesia [12–14].

28.3 Slipped Capital Femoral Epiphysis

Usually it is a problem for pubertal ages. Classically, when a 12–14-year-old adolescent comes with limping, slipped capitis femoris is first diagnosis to rule out. Hypertrophic zone of the growth plate leads migration of the proximal side of the femoral head superiorly and anteriorly because of the shearing forces. Femoral head remains inside acetabulum. In fact it is not the epiphysis which slips but the distal part of the physis. By the way it cause a huge deformity. It is more common on the left

side, African-Americans, and male sex. Obesity and endocrinopathies are probably main factors. On endocrinopathies 50% of patients are bilateral. If the patient weight is less than 50 percentile and younger than 10 years old, endocrinologic reasons must be investigated especially hypothyroidism, hypogonadism, renal osteodystrophy, and growth hormone therapy. HLA-DR4 phenotype is common for this patients [13, 17].

Patients usually have medial pain on the leg and knee. External rotation deformity, shortness of 1-2 cm, limping, and reduced range of motion are the main findings.

SCFE is classified according to stability of the growth plate, onset of the complaints, and radiology. While acute clinical situation is less than 3 weeks, chronic is more than 3 weeks. Acute SCF on chronic base is defined as slip of the femoral head after 3 weeks of mild chronic discomfort. If patient can weight bear to the affected side, it is called stable; if not, it is called unstable. Classification can be made according to the difference of the head-corpus angle between two hips. If the difference is less than 30° , it is called mild, $30-50^{\circ}$ is moderate, and more than 50° is called as severe SCFE. Clinical diagnosis is made according to AP and frog leg x-rays. At the upper side of the neck of the femur, tangential line to the neck cortex, called as Klein's line, doesn't contact with the head of the femur on the AP view of the hip joint. It is an another diagnostic feature used for especially mild cases [17] (Fig. 28.15).

Purpose of the emergency treatment is to stop the progression. Meanwhile, complications such as chondrolysis or avascular necrosis must be prevented. Extensive forces for deformity correction aren't recommended on acute situations. If the deformity correction isn't obtained or there is a chronical phase underlying, in situ pinning with one screw will be adequate. Surgery must be performed according to structure of the deformity. Experience is also important in proper fixation of the femur physis. In proper fixation can increase the risk for chondrolysis. Fixation technique should be different from what is done with collum femoris fracture



Fig. 28.15 SCFE on the right hip. Klein's line doesn't cross the physis on the right side while left is normal



Fig. 28.16 Lateral x-ray of the R hip after the surgery for the SCFE. An incorrect fixation which is unable to hold the physis and has risks for chondrolysis. Mostly, we do not need three screws as trauma surgeon has the experiences from collum femoris fractures

(Fig. 28.16). A trauma surgeon can easily miss the epiphysis and can penetrate the joint with screw.

Screw must be placed on anterolateral–posterior medial direction. Otherwise, missing of the physis and penetration of screw to joint can be seen. Proper direction for screw is from anterior to posterior in femoral neck. Prophylactic pinning for contralateral side is recommended for patients with endocrinopathies and patients younger than 10 years old.

On chronic phase the goal is to increase the range of motion and maintain the joint structure and correction of the deformity. The key point for correction of the deformity is determining the CORA with proper analysis and making osteotomy at the CORA. Because of the nature of the intracapsular deformity, there is a great risk for avascular necrosis for the correction. Surgical interventions described are three-planar intertrochanteric osteotomy described by Southwick and subcapital osteotomy described by Ganz. With the technique of safe dislocation and intracapsular osteotomy described by Ganz, it is possible to reach CORA and correct the deformity. With these techniques, femoroacetabulary impingement can be prevented. Major complication of SCF is chondrolysis and avascular necrosis that is especially seen after the hard tries for correction (Fig. 28.17).

Later phases of the disease that subchondral collapse are seen; pain gets worse; joint movements are restricted; and mainly internal rotation is restricted more.

Radiological findings that determine prognosis are collapse of the cartilage, magnitude of the necrotic area, subsidence at the femoral head, and involvement of the acetabulum. As referred on many studies, more than 50% of involvement is the primary reason of failure of the intertrochanteric osteotomies. The main principle for these osteotomies is to maintain the consistency of the joint at the superior side that allows transition of the weight. According the finding seen on MRI, we can decide the best osteotomy that will bring the intact surface of the femoral head to the face in acetabulum. In other words, we should try to bring the weight-bearing surface of the acetabulum on intact surface of the femoral head. For that reason on varus osteotomy, lateral side of the femur head, and for the valgus osteotomy medial side of the femur head must be intact. While flexion osteotomy can be performed with intact anterior region on sagittal plane, intact posterior region indicates extension osteotomy. Also biplanar correction can be performed (Fig. 28.18).



Fig. 28.18 Valgization and extension osteotomy for the hip in a case of SCFE. Because of the osteotomy performed from a different place other than CORA, deformity is corrected with translation



Fig. 28.17 Avascular necrosis after treatment of closed pinning of a SCFE case with hard reduction. Postoperative x-ray shows a well acute correction of the deformity

Proximal femoral osteotomies decrease the motion on the hip joint. For this reason patient needs up to 70° of flexion. Fixed external rotation contractures are contraindications. The fixation materials such as external fixators or plates depend on the surgeon for these osteotomies. External fixators have some advantages such as postoperative manipulation and progressive correction. A careful preoperative planning and template preparation will prevent complications and mistakes on internal fixation. It must be considered that proximal femur osteotomies can redirect the mechanical axis of lower extremity. Sometimes additional correction procedures can be added for distal femur or proximal tibia to obtain proper mechanical axis of lower extremity. In summary template preparation is essential and highly recommended [12].

28.4 Distal Femur Deformities

The distal femur deformities can be grouped as frontal plain, sagittal plain, rotational, and shortening deformities. Main complains are the functional defects, pain, limping, decrease in activity, and increase in risk of osteoarthritis.

If the mechanical lateral femoral distal angle (m LDFA) is not in the range of $85-90^{\circ}$, we call it as frontal plain deformity. If it is less than 85° , we call as valgus deformity, and if it is more than 90° , we call as varus deformity. If the posterior distal femoral angle is not between the range of $79-87^{\circ}$, we call it as sagittal plain deformity. Rotational abnormalities are decided after the physical examination where we determine the rotation profile of the lower extremity [19, 20].

Not every deformities are operated. The main indications for the operations in frontal plain are more than 15 mm deviation in mechanical axis, more than 10° of varus–valgus deformities, and more than 2–3 cm of shortening (Figs. 28.19, 28.20, and 28.21). In case of sagittal plain adaptation of the knee joint is an important factor for the decision. In case of flexion deformity of the knee that cause over activity of quadriceps and hyperextension deformity that cause posterior soft tissue laxity, we suggest to correct the deformity [13].



Fig. 28.19 Lateral deviation of the mechanical axis because of the distal femoral deformity, mLDFA 78

Fig. 28.20 Deformity described in Fig. 28.19 is planned to correct by an open-up osteotomy. Circular-type external fixator is preferred. Hinges are placed medially and motor unit laterally



Fig. 28.21 (a) Radiological result of the treatment. Before and after the removal of the external fixator. (b) Valgus deformity at the distal femur can also be corrected

with internal fixation with close-up osteotomy technique with acceptance of up to 1-1.5 cm of shortening. In both methods mechanical axis is corrected



Fig. 28.22 Correction of distal femoral valgus and rotational deformity with monoplanar external fixator

To find out the CORA of the deformity, malalignment test described by Paley should be performed. After the finding of the CORA, we should decide to make and open-wedge or closed-wedge osteotomy according to the limb length discrepancy. If we have the shortening, we should prefer open-wedge osteotomy, and if we need to shorten the extremity, we should prefer closed-wedge osteotomy to correct the deformity. In case of valgus deformities, if we have more than 20° of deformity or in revision case, we have to decompress the peroneal nerve both around the fibular head peroneal fascia and between the anterior and anterolateral compartment and intermuscular septum [21].

Correction of the deformity with an external fixator has some advantages. First, you can continue to correction after the operation. Second, there is no hardware inside that needs to take out. But it has also some disadvantages: pin care and lack of the comfort. Therefore we can use the monolateral external fixator to increase the comfort. In case of the monolateral fixator, surgeon has to have experience on external fixators. Angle of the Schanz screws should be well calculated to correct the deformity on three plains. In case of the monolateral external fixator applied laterally, it is possible to correct the frontal plain residual deformity after the operation with swivel clamps, but sagittal and rotational deformity cannot be corrected after the operation (Fig. 28.22).

Sagittal plain deformity of the femur also affects lower extremity biomechanics. Most of the sagittal plain deformities result in deformity in the knee joint as the clinical presentation. X-ray taken with the knee joint in full extension on the lateral plain and application of malalignment tests reveal the reason of the knee deformity; it is a soft tissue contracture or bone deformity. Bone deformity can originate from tibia or femur. Full extension for these patients is important to walk. Flexion



Fig. 28.23 Knee flexion contracture case with a 40-degree flexion deformity. As there is no deformity on sagittal plain (mPDFA and mPPTA are in normal ranges), we can talk about a pure soft tissue contracture in that case

deformity of femur or tibia can be corrected with extension osteotomy. Gradual correction of the deformity especially if it is more than 20°, with external fixator, will prevent vascular and nerve injuries that can be seen in case of acute correction (Figs. 28.23 and 28.24). But in case of the soft tissue contracture and intraarticular pathology, we have to correct the deformity from the center of rotation of the knee joint. In such case soft tissue releases should be performed before the application of external fixator. In mild soft tissue contractures, it is possible to correct the defor-



Fig. 28.24 In pure soft tissue contracture of the knee joint, hinges are placed to the center of rotation of knee joint and motor units placed posteriorly. Distraction is limited as 1 mm on the soft tissue at the posterior part of the knee (vascular and nerve tissues)

mity with a supracondylar femur extension osteotomy. Theoretically, such a procedure will prevent the last flexion of the knee as much as correction angle maintained with supracondylar femur osteotomy. In mild deformities, less than $15-20^{\circ}$, it is not clinically significant [18].

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