Genu Varum in Children: Diagnosis and Treatment

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Abstract

Genu varum is a relatively common finding in children. Physiologic bowing, which is seen most often, has a well-documented favorable natural history. Idiopathic tibia vara is the most common of the pathologic conditions that are associated with bowed legs; treatment strategies vary with the patient's age and the stage of disease and deformity. Genu varum may also accompany systemic conditions, such as achondroplasia, vitamin D–resistant rickets, renal osteodystrophy, and osteogenesis imperfecta—all of which can result in short stature. Indications for intervention are not always well defined. A rare disorder, focal fibrocartilaginous dysplasia, usually requires no treatment. Standing radiographs of the entire lower limbs are necessary for surgical planning, as the deformity can sometimes affect the distal femur rather than the proximal tibia. Restoration of the mechanical axis of the limb is the principal goal of treatment; the particular type of internal fixation is of secondary importance.

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Genu varum, known colloquially as bowlegs, is relatively common in children and is a frequent cause of parental concern. In the vast majority of cases, genu varum will correct with growth. A small number of children have pathologic conditions that may result in functional and cosmetic problems if left untreated. In this article, we will review pertinent factors in the assessment of genu varum, associated conditions, and treatment options.

Assessment

History

A thorough history will often distinguish the relatively infrequent pathologic genu varum from the much more common physiologic variety. A family history of short stature or similar varus alignment should be sought; the grandparents may be a good source for this information. The ages at attainment of various developmental milestones, such as sitting independently, pulling to stand, and walking, should be determined. It is useful to establish whether the parents consider the deformity to be progressive. The positional sleeping and sitting habits of the child are also of interest.

Physical Examination

After routine documentation of the height and weight and determination of their percentiles for age, the patient's pelvis, knees, and feet should be examined carefully. Shortening of the limbs relative to the trunk, especially rhizomelic shortening, suggests a dwarfing condition. In ambulatory children, the appearance while standing and during gait provides the most information. Both limbs should be examined in the frontal and sagittal planes for asymmetry and alignment. It should then be determined whether the deformity is a gradual bowing or an abrupt angulation. If the deformity is angular, its location is identified in the distal femur, the knee, or the proximal tibia. Obliquity of the popliteal crease, if present, is a useful sign; distal femoral varus will produce obliquity of the popliteal crease, while deformity more distal in the extremity will not.

Passive rotation of the hips and motion of the knee are noted. Ligamentous stability of the knee is assessed, with particular attention to the lateral ligamentous complex. A dynamic component of the deformity or lateral thrust at the knee during the stance phase of gait indicates laxity of the lateral ligamentous complex. Torsion of the tibia should also be routinely assessed; determination of the thigh-foot angle and evaluation of the bimalleolar axis, as described by Staheli et al,[1] are useful

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in torsional assessment. Serial photographs of the standing child at the initial and follow-up evaluations serve as an inexpensive method of documenting any progression of the deformity.

Radiographs

We believe radiographs are unnecessary in a young child of normal stature with physical findings compatible with physiologic bowing. When there is a localized deformity or the child is short, full-length standing radiographs (hip to ankle) should be obtained with the knees pointing straight forward. When internal tibial torsion is present, the technician often attempts to externally rotate the leg (and the knee) to point the foot straight forward; however, rotation affects the tibiofemoral and metaphyseal-diaphyseal angles and tends to minimize the degree of deformity.[2] In addition to the angular deformity present, the physes of the femur and tibia should be carefully assessed.

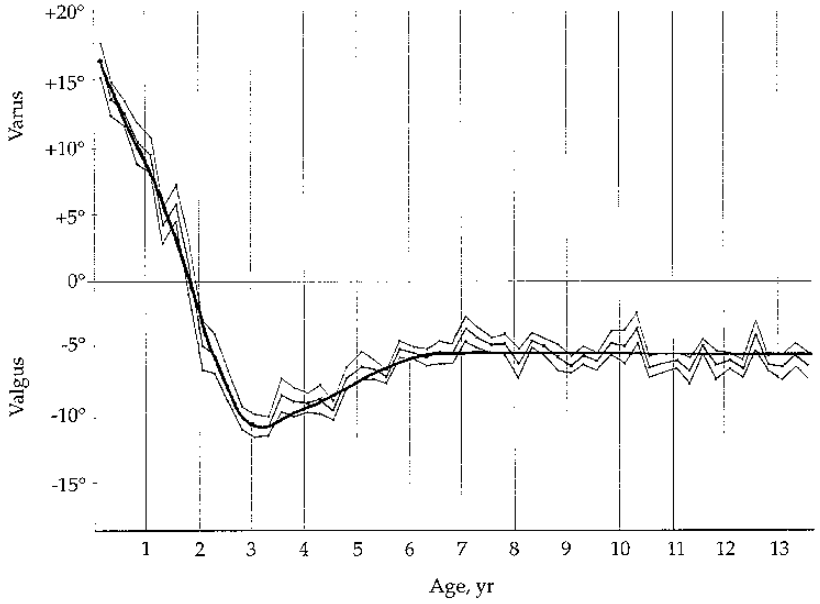

Fig. 1 Graph illustrating the development of the tibiofemoral angle in children during growth, based on measurements from 1,480 examinations of 979 children. Of the lighter lines, the middle one represents the mean value at a given point in time, and the other two represent the deviation from the mean. The darker line represents the general trend. (Adapted with permission from Salenius P, Vankka E: The development of the tibiofemoral angle in children. J Bone Joint Surg Am 1975;57:259-261.)

Physiologic Genu Varum

Physiologic genu varum is by far the most common cause of bowlegs in a toddler. The natural history of the changing angular relationship between the femur and the tibia in children is required knowledge for any orthopaedist with a pediatric practice. Development of the tibiofemoral angle follows a predictable sequential pattern. Infantile genu varum progresses to excessive genu valgum, followed a gradual correction to adult physiologic valgus alignment (Fig. 1). Genu varum is greatest at 6 months of age. Correction to neutral alignment is often complete by 18 months of age. Heath and Staheli[3] report that persistence of genu varum beyond 2 years of age is abnormal, and Salenius and Vankka[4] state that neutral alignment may not be reached until 22 to 24 months of age. Even pronounced physiologic genu varum greater than 30 degrees can correct with continuing growth.[3] Overcorrection to excessive genu valgum is maximal at 4 years of age; the valgus angulation averages 8 degrees. Correction to physiologic valgus is usually complete by 5 or 6 years of age.[4]

Early walking has been documented in black children,[5] and this may be a factor in a tendency toward increased physiologic bowing. Internal tibial torsion is frequently found in association with physiologic genu varum; if physiologic, it corrects concomitantly with the genu varum.

Radiographically, physiologic genu varum is characterized by bowing of the entire limb. On the standing anteroposterior radiograph, both the distal femur and the proximal tibia will have some varus bowing without an acute angular component. The physis will appear normal without medial physeal changes. There may be equal beaking of both the distal femoral and the proximal tibial metaphyses.

The treatment of physiologic genu varum is periodic observation and examination, together with education and reassurance of the parents. Occasionally, spontaneous correction of the physiologic genu varum will be delayed. We believe this happens more often in children who habitually sleep or sit with their legs rotated beneath them, as this seems to counteract the normal unwinding effect of weight-bearing in correcting tibial torsion and genu varum.

Reassurance of anxious parents or other relatives of a child with physiologic bowing is not always easy to achieve. We find that giving a copy of a graph depicting the nor-

mal progression of genu varum to genu valgum in early childhood, along with the orthopaedist's explanation of the graph, is extremely helpful. The parents can then show the graph to other concerned relatives. In addition, the American Academy of Orthopaedic Surgeons has produced a video on common lower-limb problems in children ("Growing Out of It: Torsional Deformities in Children" [No. 29-074]), which can be viewed independently by the family.

Tibia Vara

History

Tibia vara, often referred to as Blount's disease, is characterized by an abrupt varus deformity of the proximal tibia. It is the most frequent cause of pathologic genu varum. Unlike physiologic genu varum, Blount's disease is progressive and rarely corrects spontaneously. There are two predominant types of tibia vara—infantile and late-onset, or adolescent—which are distinguished by the age at onset and the distinctive clinical presentation. While the two types are similar in the histologic appearance of the proximal tibial physes, the exact etiologies remain somewhat unclear.

Tibia vara is classified as infantile when the onset occurs before 5 years of age. Idiopathic infantile tibia vara is more often seen in black, female, and obese children and in children who begin to walk earlier than usual. Involvement is bilateral in 80% of patients and is associated with a greater degree of internal tibial torsion than in the adolescent form. There may be difficulty in distinguishing early infantile tibia vara from physiologic genu varum.

Children in whom significant tibia vara develops after 6 years of age are thought to have the late-onset form, which is less common than the infantile type. Late-onset tibia vara is more often unilateral and has a greater prevalence in black, male, and obese children and teenagers. In his study of the natural history of tibia vara in Finnish children, Langenskiöld[6] reported more rapid progression of the varus deformity in the infantile type than in the late-onset type. The prevalence of morbid obesity in adolescents has increased in the United States,[7] and there appears to be an associated increase in the incidence of late-onset tibia vara.

Children in whom pathologic tibia vara later develops are born with normal alignment of the femur and tibia; the deformity results from a subsequent growth disturbance of the proximal tibia. Tibia vara does not occur in children who do not walk. While the exact etiology of infantile tibia vara remains unclear, an association between tibia vara and walking at an early age and obesity in infancy suggests that mechanical forces are at least partially causative. Early weight-bearing and obesity result in greater compressive forces across the medial tibial physis than in infants who are not obese and who begin walking at about 1 year of age. The compressed posteromedial physis responds with slower growth in this region, producing a progressive varus deformity of the proximal tibia.

In the late-onset type, there is usually a mild preexisting varus deformity, which is thought to be a factor in the development of a progressive varus deformity. When mild genu varum persists into adolescence, increased body weight and physical activity repetitively traumatize the posteromedial physis, causing medial growth suppression.[8]

Cook et al[9] performed a two-dimensional finite-element analysis of the effect of varus angulation and increased body weight on resultant forces on the medial proximal tibial physis and found that in older children, lesser degrees of varus were necessary to produce medial physeal growth retardation. In a 2-year-old, 20 degrees of varus could produce medial tibial physeal growth inhibition; in a 5-year-old of normal weight, as little as 10 degrees of varus angulation could result in growth inhibition.

Assessment

Examination of the child with tibia vara is notable for an angular varus deformity discernible just below the knee. In morbidly obese children, the acute angulation of the tibia may be hidden by their excessive soft tissue. In contrast, the young child with physiologic genu varum will have a more gentle curvature of the entire extremity. An inwardly directed thigh-foot angle due to internal tibial torsion may accompany either of these conditions, but is more severe in infantile tibia vara. A lateral thrust, indicating laxity of the lateral ligamentous complex, may be seen in children over the age of 3 with tibia vara. This laxity is thought to exacerbate the dynamic forces across the physis during gait and is not seen in physiologic bowing.

Pathology

The histopathologic findings in the physes are the same whether the child has infantile or late-onset tibia vara.[8,10] The physeal disruption is similar to that found in slipped capital femoral epiphysis, which may suggest a common etiology. Disorganized physeal cartilage is present, with disruption of the normal columnar architecture of the physis, which is most evident in the resting zone. Islands of densely packed, unusually hypertrophic cartilage cells are seen. Both fibrovascular and cartilaginous reparative tissue can be found at the physeal-metaphyseal junction.

Radiographs

The characteristic radiographic appearance of tibia vara is not usually present until the age of 2 years. The radiographic classification developed by Langenskiöld[6] is most often used to stage the infantile forms of the disease (Fig. 2). The potential for recovering growth after treatment is thought to be directly related to the stage of the disease, although this staging may be more pertinent in a retrospective review than prospectively.

Medial fragmentation of the proximal tibial metaphysis is the earliest abnormal radiographic finding. Later, medial physeal depression and varus angulation of the metaphysis develop, with beaking of the proximal tibial metaphysis. In very late stages of Blount's disease, the medial physis develops an osseous bridge between the epiphysis and the metaphysis.

As with the use of many classification systems, there is considerable interobserver variation, and the staging is not precise. However, the system does provide the user with some notion of the natural history of progression of the condition. Whether a given tibia demonstrates stage III or stage IV changes is less important than is the recognition that tibia vara is well established and the treatment that might be appropriate for stage I or II is no longer appropriate.

In late-onset tibia vara, the radiographic changes are less dramatic. The growth plate shows less irregularity and rarely forms the physeal bone bridge that may be seen in the infantile form. The epiphysis is less deformed, producing less articular incongruity. Overgrowth of the lateral femoral condyle is common, and distal femoral varus may be predominant.[11]

Levine and Drennan[12] have popularized measurement of the metaphyseal-diaphyseal angle of the proximal tibia for the early differentiation of infantile tibia vara from physiologic genu varum. This angle is formed by a line drawn perpendicular to the tibial diaphysis and a line drawn between the medial and lateral aspects of the tibial metaphysis (Fig. 3). A metaphyseal-diaphyseal angle greater than 11 degrees is strongly associated with subsequent development of tibia vara. In their study, Levine and Drennan found that 29 of 30 affected limbs with a metaphyseal-diaphyseal angle over 11 degrees developed advanced radiographic changes consistent with the diagnosis of tibia vara, with a false-positive rate of only 3%. In contrast, tibia vara developed in only 3 of 59 limbs with metaphyseal-diaphyseal angles of less than 11 degrees.

Feldman and Schoenecker[2] found the metaphyseal-diaphyseal angle to be somewhat less reliable in younger patients. On linear-regression analysis of the use of a metaphyseal-diaphyseal angle of 11 degrees or more as a basis for deciding treatment, they found a false-negative rate of 9% and a false-positive rate of 33%.

With increasing age, the metaphyseal-diaphyseal angle is more reli-

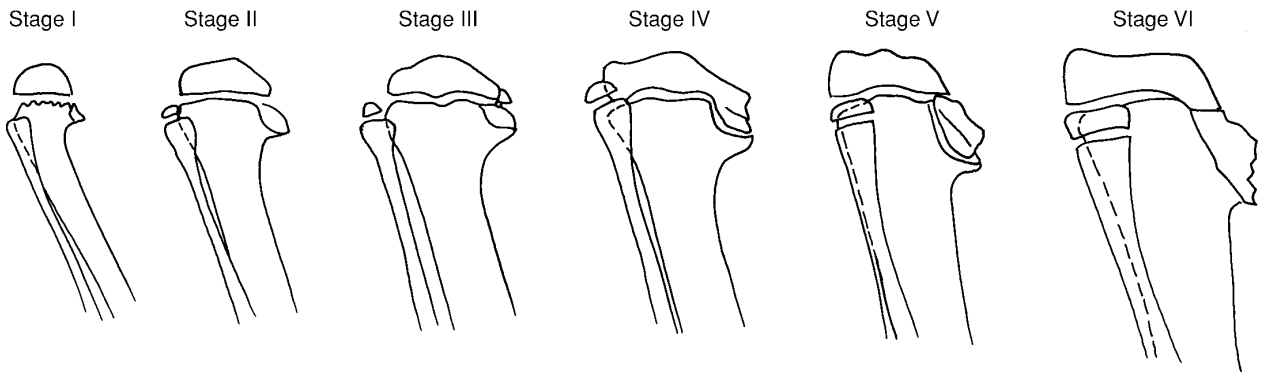

Fig. 2 The six stages of tibia vara, as described by Langenskiöld.[6] Stage I (seen in children up to age 3 years) is characterized by medial and distal beaking of the metaphysis and irregularity of the entire metaphysis. Stage II (seen in children aged 2½ to 4 years) is characterized by a sharp lateromedial depression in the ossification line of the wedge-shaped medial metaphysis. Complete restoration is common in this stage. Stage III (seen from ages 4 to 6 years) is characterized by deepening of the metaphyseal beak, which gives the appearance of a step in the medial metaphysis. Stage IV (seen from ages 5 to 10 years) is characterized by enlargement of the epiphysis, which occupies the medial metaphyseal depression. Restoration is still possible in this stage. Stage V (seen from ages 9 to 11) is characterized by a cleft in the epiphysis, which gives the appearance of a double epiphysis; the articular surface of the medial tibia is deformed, sloping distally and medially from the intercondylar region. Stage VI (seen from ages 10 to 13) is characterized by closure of the medial proximal tibial physis, with a normal lateral physis. Langenskiöld described his findings on the basis of his observations of Finnish children; changes in African-American children tend to occur at a younger age.

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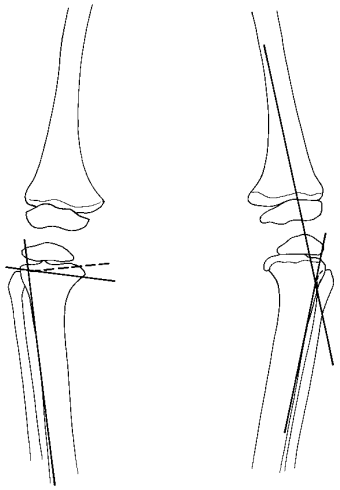


Fig. 3 Determination of the metaphyseal-diaphyseal (MD) and tibial-femoral (TF) angles.

able, since it tends to increase in magnitude in patients who have Blount's disease and decrease in magnitude in patients with physiologic genu varum. Rotation can have a small but significant effect on the radiographic measurement of the metaphyseal-diaphyseal angle. Henderson et al[13] compared radiographs obtained with and without rotation and found a difference of 2.8 ± 1.2 degrees in the metaphyseal-diaphyseal angles that were measured. When attempting to distinguish physiologic genu varum from tibia vara in young patients, this amount of measuring error can be misleading if one is relying on the metaphyseal-diaphyseal angle alone for the diagnosis. If there is doubt regarding the radiographic findings, we believe a further period of observation is indicated, rather than initiating treatment on the basis of blind adherence to arbitrary radiographic measurements.

Treatment

There are still no generally accepted criteria for initiation of treatment in infantile tibia vara. Although Levine and Drennan[12] reported that a metaphyseal-diaphyseal angle of 11 degrees could be used as a basis for treatment, others have recommended observation of children aged less than 24 months with metaphyseal-diaphyseal angles of as much as 16 degrees.[2] Persistent internal tibial torsion, lateral thrust during stance phase in gait, and posterolateral instability are additional findings that may influence a decision to initiate early treatment.

There is certainly a place for observation of the young child with abnormal clinical and radiographic findings before initiating brace or surgical treatment. A child with a metaphyseal-diaphyseal angle of less than 9 degrees is obviously at minimal risk for tibia vara. If the angle is greater than 16 degrees, treatment probably should be initiated. Children with metaphyseal-diaphyseal angles between 9 and 16 degrees are generally treated if there has been no tendency toward correction after 24 months of age.

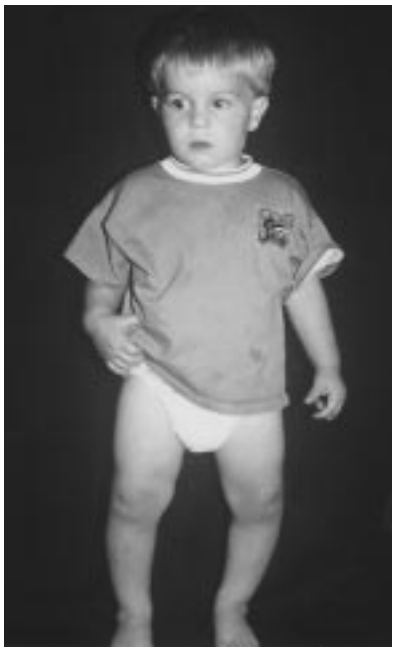
While early tibia vara will correct without bracing in some children (Fig. 4), bracing has often been recommended as the initial treatment of children with Langenskiöld stage I or II tibia vara. The device usually prescribed is a knee-ankle-foot orthosis with a single medial upright secured at the upper thigh and ankle. A knee hinge is not used, but this does not prevent the child from sitting. The ankle is left free. A strap at the knee applies a corrective valgus force. The brace is worn nearly full-time, especially during walking, to minimize the valgus stress at the knee. The effectiveness of the brace is thought to be related to the relief of weight-bearing stresses on the medial physeal region of the proximal tibia. Brace treatment is reported to be successful in 50% to 80% of the patients treated.[14,15] The brace is worn until the deformity has been corrected and reconstitution of medial physeal growth is present on radiographs, which usually takes about 1 year. Thus, bracing is usually not a viable option for children over the age of 3.[16] Factors such as patient age, stage of the disease, family compliance, and brace fit can have an effect on the success of bracing. Studies controlling for all these variables have not yet been reported. However, it seems that bracing is a reasonable first treatment option when a decision is made to start treatment of early tibia vara in a 2- or 3-year-old child.

Children who are too old for bracing and children in whom tibia vara has progressed despite bracing are best treated with a proximal tibial valgus osteotomy. The goal of the osteotomy is to restore the mechanical axis of the lower extremity. The osteotomy is performed below the tibial tubercle apophysis and is combined with a fibular osteotomy.

Ideally, the osteotomy is done before the child is 4 years old. Residual internal tibial torsion can be corrected at the same time. If osteotomies are first done in older children, repeat osteotomies are more often needed. Ferriter and Shapiro[17] retrospectively analyzed factors affecting the outcome of 77 proximal tibial osteotomies performed on 25 patients with tibia vara and found a 76% rate of deformity recurrence in children operated on after the age of 4.5 years. In younger children, the rate of recurrence of varus deformity was 31%.

Loder and Johnston[14] reported lower rates of recurrent deformity after valgus tibial osteotomy. Prognostic factors associated with a higher rate of recurrence in their older patients included morbid obesity and more severe disease (Langenskiöld stages IV, V, or VI).

Efforts to improve the results of tibial osteotomy as treatment of infantile tibia vara in children older than 4 years include physeal-bar re-



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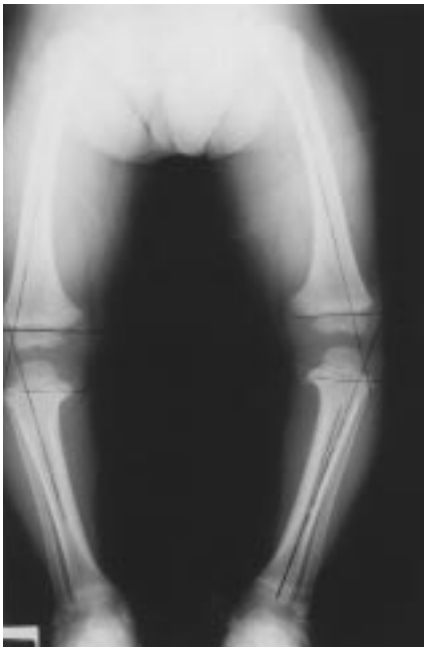

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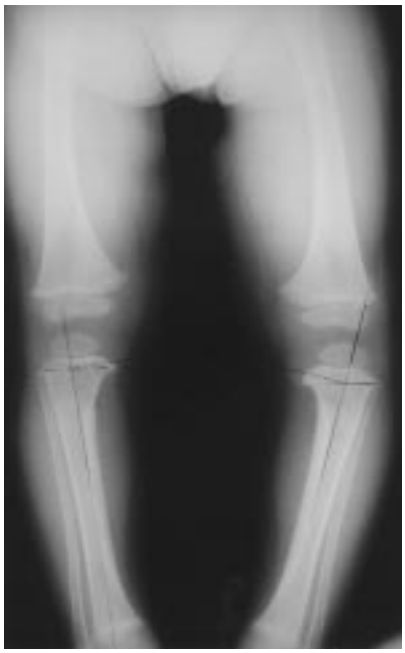

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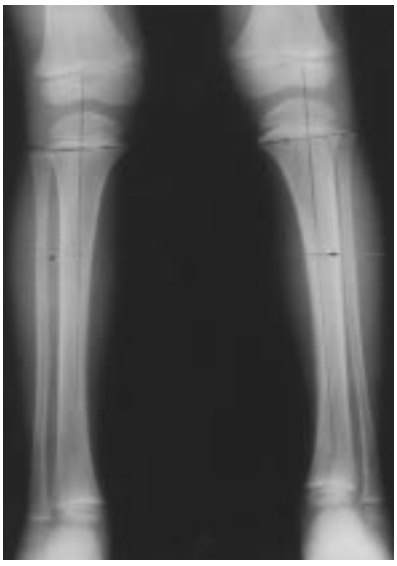

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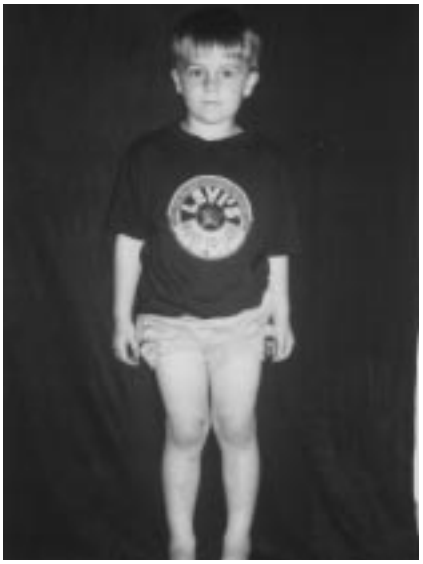


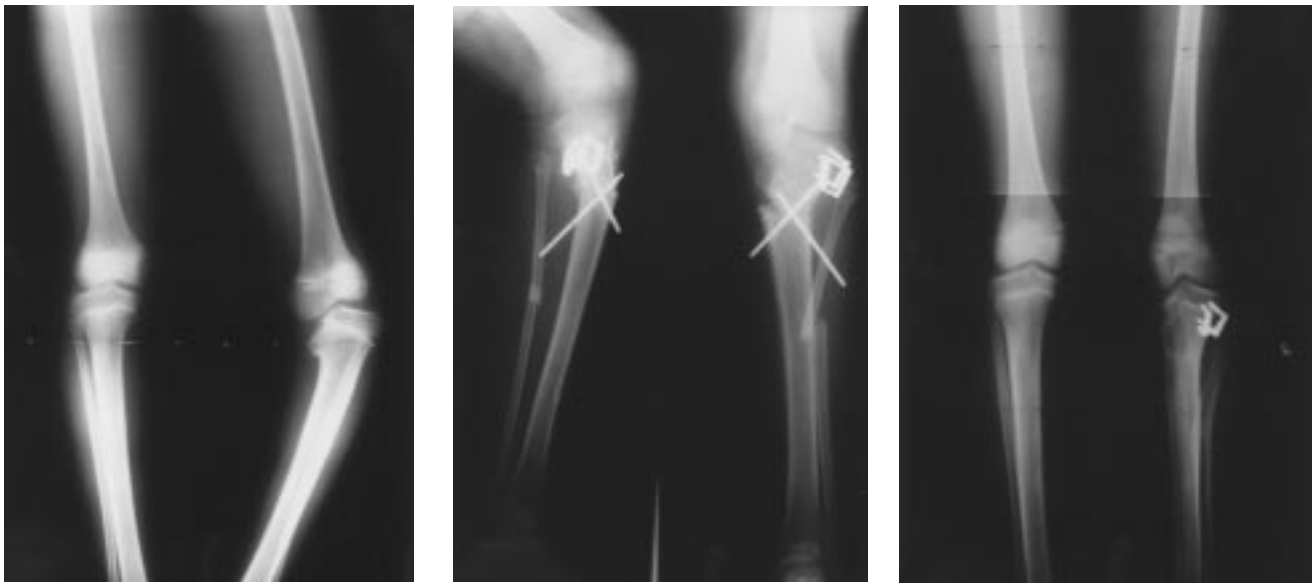
E

Fig. 4 **A,** Clinical appearance of a healthy boy, aged 1 year 9 months, being evaluated for infantile tibia vara. **B,** Initial radiograph shows medial tibial physeal beaking and metaphyseal-diaphyseal angles of 17 degrees on the left and 12 degrees on the right. Observation without bracing was elected at that time. **C,** Radiograph obtained at age 2 years 3 months shows persistent medial tibial beaking and irregularity of the proximal tibial physes, greater on the left than the right. **D,** Normal tibial alignment was seen at age 4 years 10 months. **E,** Clinical appearance at age 4 years 10 months.

section and lateral proximal tibial hemiepiphysiodesis (Fig. 5). These procedures were developed with an understanding that recurrence of the deformity is largely due to the lack of reconstitution of growth of the medial proximal tibial physis.

Physeal bridges are more common in children with infantile tibia vara who are older than 5 years. The presence of a physeal bridge may be impossible to ascertain on routine radiographs. Computed tomography or magnetic resonance imaging, with thin sections obtained through the physis, can be helpful in detecting the presence and size of the osseous bridge.

Physeal-bridge resection is a difficult operative procedure due to the deformity of the physis, and the poorest results have been associated with involvement of more than 30% of the physis.[16] Fat, cartilage, Silastic, and methylmethacrylate have all been used as spacers to prevent bridge recurrence after surgical resection. Fa-



A B C

Fig. 5 **A,** Radiograph of a 7-year-old girl with unilateral varus deformity and severe depression of the medial tibial physis. **B,** A corrective closing-wedge proximal tibial osteotomy was performed, with stapling of the lateral proximal tibial physis. **C,** On radiograph obtained at 2-year follow-up examination, alignment is normal, with minimal leg-length discrepancy.

vorable outcomes in small series of patients have been reported.[14,18] Actual growth, however, is difficult to determine from the published radiographs, and our personal experience with this procedure has been poor. A rim of viable physis surrounding the excised portion is necessary if growth is to recover after partial physeal resection. It may not be possible to determine on gross examination at the time of surgery whether the remaining physeal rim is biologically active.

The use of lateral hemiepiphysiodesis of the proximal tibial physis is an attractive treatment alternative in the older child with tibia vara at risk for redevelopment of varus deformity after proximal tibial osteotomy alone. A formal ablation of the lateral tibial physis or simple stapling can be done (Fig. 5). When done unilaterally, a limb-length discrepancy is predictable in younger patients; however, treatment of this inequality may not be needed.[15]

In advanced forms of infantile tibia vara (Langenskiöld stage IV or greater), resultant deformity of the tibial epiphysis, as well as the physis, produces articular incongruity. Restoration of normal articular anatomy by elevation of the depressed medial epiphysis and physis has been reported to reconstruct the joint architecture, generally in combination with a valgus tibial osteotomy to restore the alignment of the lower limb.[19] Medial elevation combined with proximal tibial osteotomy (and occasionally distal femoral osteotomy) has been utilized with success by Schoenecker et al.[15]

For patients with late-onset tibia vara, the indication for treatment has been defined arbitrarily as varus alignment greater than approximately 10 degrees. The goal of surgery is correction of the mechanical axis to prevent the development of medial knee-compartment osteoarthrosis. Young adults with tibia vara have a high incidence of accelerated symptomatic degenerative changes of the knee, which is related to the degree of varus malalignment.[20]

Treatment of the adolescent with tibia vara often involves increased technical problems due to morbid obesity. Complications are more common than in the treatment of infantile tibia vara. These complications include difficulty in the exposure and performance of the osteotomy and failure of osteotomy fixation. For these reasons, Henderson et al[21] have proposed lateral proximal tibial hemiepiphysiodesis as a primary procedure, reserving osteotomy for those cases in which more conservative procedures have failed. The importance of standing radiographs of the entire lower limbs has recently been emphasized.[11] We believe such radiographs are necessary for proper preoperative planning and postoperative assessment. The literature is replete with techniques for the performance of the tibial osteotomy in late-onset tibia vara, including various types of internal and external fixation. Regardless of the method of fixation chosen, the goals of

surgery are unchanged: correction of the mechanical axis and leveling of the knee joints.

Differential Diagnosis

Vitamin D–Resistant Rickets

Progressive genu varum often develops in children with untreated hypophosphatemic rickets, a sex-linked inherited disorder due to vitamin D resistance that results in defective bone mineralization. Children with this disorder typically present with bilateral lower-limb angular deformities. The diagnosis should be considered if the child is relatively short, because height in affected children is usually in the lower 10th percentile. The bowing is due to a combination of varus of the distal femur and varus of the proximal tibia.

Medical treatment of this type of rickets includes oral phosphates and some form of vitamin D replacement. Surgical measures to correct the deformity are often unsuccessful when adequate medical control of the rickets has not been achieved before surgery. In that situation, it may be best to wait until skeletal maturity to realign the mechanical axis.[22] When only partially treated, this condition may be difficult to distinguish from physiologic bowing, but children with rickets typically are older. Massive doses of a vitamin D preparation can restore a normal radiographic appearance to the epiphysis; however, normal growth will not be restored unless phosphate replacement is also adequate. Phosphate replacement therapy has to be administered at regular intervals, and patient compliance with this strict dosage schedule may be poor.

Renal Failure and Renal Osteodystrophy

Children who are in renal failure or who have renal osteodystrophy have a high incidence of growth disturbance in both the proximal and distal ends of the tibia. The physes in these children have been shown to exhibit many of the same pathologic changes found in tibia vara and slipped capital femoral epiphysis, particularly disorganized growth plates at the physeal-metaphyseal junction.[23] Deformity results when eccentric forces occur across the weakened physis. Because renal failure occurs more commonly in older children who have already achieved physiologic valgus alignment, valgus deformity is encountered most often at the knee. Younger children who have retained physiologic varus alignment may undergo exaggeration of preexisting genu varum. Deformities secondary to renal disease are usually bilateral, with a gentle curve of the extremity due to simultaneous involvement of both the distal femoral and proximal tibial physes.

Rickets and renal osteodystrophy may be easily distinguished from tibia vara on the basis of their radiographic appearance. In both, physeal cupping and widening occur at both the distal femoral and proximal tibial physes. Marked osteopenia and thinning of cortical bone are also present.

Orthopaedic treatment of angular lower-limb deformities resulting from renal disease is wisely postponed until the renal status has stabilized in response to medical treatment or renal transplantation. Correction of genu varum or valgum with osteotomy will be short-lived unless the abnormal bone metabolism resulting from the renal disease has been reversed.

Metaphyseal Chondrodysplasia

Metaphyseal chondrodysplasia, which results from abnormal chondroblast function and chondroid production, is a very rare cause of genu varum. A number of metaphyseal dysplasias can lead to bowing, among them the Jansen and Schmid types. The more severe Jansen type has an autosomal-dominant inheritance and is characterized by mental retardation, short-limb dwarfism, exophthalmia, hypercalcemia, and long-bone bowing. The more mild Schmid type, which is also transmitted by autosomal-dominant inheritance, is characterized by normal intellect and normal laboratory findings. As lower-extremity bowing does occur with this condition, it may be difficult to distinguish from rickets. Even though the physes are widened and cupped in the Schmid type, the epiphyses are normal, and the presence of short stature should be helpful in arriving at the correct diagnosis.

Achondroplasia

Genu varum is a frequent finding in achondroplasia, a rhizomelic dwarfing condition due to abnormal endochondral bone formation. At birth, lower-limb alignment is relatively normal. However, with growth, the spontaneous correction to genu valgum does not occur. Instead, genu varum tends to increase throughout childhood and adolescence, largely due to overgrowth of the fibula in relation to the tibia. In addition, the growth of the proximal tibial metaphysis may be asymmetrical. Radiographically, the proximal fibular physis is superior to the proximal tibial physis. Although the tibial metaphysis is enlarged, the epiphysis remains normal.

Children with achondroplasia rarely have knee pain, and functional indications for surgical correction of bowlegs are not well defined. Treatment options include proximal fibular epiphysiodesis and tibial osteotomy. A fibular epiphysiodesis must be done early in childhood to prevent the development of progressive genu varum. For established genu varum, proximal tibial valgus

osteotomy is most often used. An accompanying distal tibial osteotomy and concomitant tibial lengthening have also been advocated by some. The role of lengthening of short limbs in this condition is still unsettled. Bracing is ineffective, in part because of the joint laxity commonly present.

Osteogenesis Imperfecta

Osteogenesis imperfecta results from a defect in type I collagen and produces varying degrees of skeletal fragility. In the more severe forms, multiple fractures of the lower extremities are common. The femur is most frequently fractured, followed by the tibia. Repeated fractures often lead to bowing and torsional deformities of the lower extremity. The distal third of the femur is a common location of these fractures, usually associated with anterolateral angulation at the fracture site. Residual deformity after fracture is common, and the varus angulation often increases as a result of repeated fractures. Radiographs demonstrate diffuse osteopenia, occasionally accompanied by evidence of fracture healing at multiple locations.

In cases of mild deformity, bracing can be used for support and prophylaxis against repeat fractures. Occasionally in more severe cases, pronounced bowing is present from birth, and ambulation will not be possible unless correction is undertaken early. There are a number of options for the surgical management of varus deformity secondary to osteogenesis imperfecta; selection is dependent on the age of the patient and the nature of the anatomic deformity.

Focal Fibrocartilaginous Dysplasia

Focal fibrocartilaginous dysplasia is a rare cause of unilateral genu varum.[24,25] It affects the proximal medial tibia, and the resultant deformity is more properly termed tibia vara. The deformity is usually apparent to the parents before the child is 18 months of age. Radiographs show a characteristic cortical lucency with surrounding sclerosis in the proximal medial tibial metaphysis and varus angulation. The condition usually corrects by age 4 with growth. Surgical correction of the deformity is usually not needed.[25]

Less Common Causes

Any disorder that can affect the proximal tibial or distal femoral growth plate has the potential for causing genu varum. For example, infantile osteomyelitis with abscess formation can generate uneven subsequent growth, with resultant deformity. Another such disorder is physeal growth disturbance secondary to trauma or sepsis. The distal femur is a relatively common site of growth disturbance following physeal fracture. Physeal fractures of the proximal tibia are much less common. Management of physeal growth disturbances is complex and is beyond the scope of this article.

Principles of Evaluation and Treatment

The following are a few principles that will help the orthopaedist in the evaluation and treatment of the child with genu varum:

(1) Genu varum is physiologic until the age of 18 to 24 months, and treatment is unnecessary.

(2) In a child with normal stature and findings compatible with physiologic bowing, radiographic documentation is unnecessary. If documentation of the condition is desired, photographs are less expensive and just as valuable.

(3) If radiographs are deemed necessary, full-length standing films of the entire lower limbs are required for the evaluation of the mechanical axis and the site of deformity.

(4) Shortness of stature should signal the likelihood that a constitutional disorder is the cause of genu varum.

(5) Idiopathic tibia vara is the most common pathologic cause of bowlegs in the child. Bracing may be effective in the early stages, but this has not been established by prospective controlled clinical trials.

(6) Surgical correction of tibia vara can be guided by the principle that reestablishing a normal mechanical axis in the early stages will allow normal growth to occur. In older children, resumption of normal growth cannot be assumed, and measures to slow later tibial physeal growth may also be needed.

(7) There are various types of internal and external fixation, all of which are satisfactory. The particular type of fixation used for surgical treatment of tibia vara is less important than reestablishment of the mechanical axis.

(8) Treatment of genu varum secondary to constitutional disorders must be tailored on an individual basis.

Conclusion

Although genu varum is fairly common in children, considerable changes in evaluation and treatment approaches have occurred over the past decade. Further refinements can be expected in the coming years, perhaps including a clearer concept of the etiology of tibia vara, a better grasp of the role of bracing in infantile tibia vara, and a more complete understanding of the effects of treatment (both positive and negative) in constitutional disorders such as achondroplasia.

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