Blount Disease in an Adolescent Male Athlete: A Case Report

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Published: 2023
Journal of the International Academy of Neuromusculoskeletal Medicine
Volume 20, Issue 1

ABSTRACT

Objective: The purpose of this case report is to describe the clinical presentation, radiographic evaluation, physiological and pathological differentials, and surgical management of progressive symptomatic tibia vara (Blount disease).

Clinical Features: A 13-year-old male sought evaluation and care for knee pain at an outpatient chiropractic clinic. The chiropractor identified varus bowing of the involved leg and exaggerated lateral movement at the knee in the heel strike phase of the patient's gait. Radiographic evaluation revealed a medial proximal tibial slope, increased metaphyseal-diaphyseal angle, and metaphyseal beaking of the involved leg, consistent with Blount disease.

Intervention and Outcome: The patient was evaluated in two orthopedic clinics for management of Blount disease. An outpatient surgical intervention with tension-band plating was performed to arrest lateral tibial growth. The patient was weight-bearing at the time of release, with minimal pain management interventions, and with few activity restrictions.

Conclusion: Physiological tibia vara is common in young children and must be differentiated from pathological tibia vara by clinicians and radiologists through assessment of risk factors, clinical presentation, radiographic assessment, and monitoring of progression. Early detection and treatment of pathological tibia vara is essential to limit the

progression and subsequent complications of undetected or untreated Blount disease.

Key Words: Blount Disease, Tibia Vara, Bowed Legs, Lateral Thrust, Osteotomy, Epiphysiodesis, Metaphyseal Beak

INTRODUCTION

Bilateral and symmetrical outward bowing of the legs (tibia vara) is a common and expected physiological finding in toddlers under 2 years of age that tends to regress with increased age and skeletal development.[1] When the bowing deformities are persistent or progressive, continued monitoring through early adolescence is vital to distinguish between physiological tibial vara and the pathological tibia vara differentials of dietary vitamin D deficiency and vitamin D-resistant rickets, renal osteodystrophy, proximal tibial metaphyseal fibrocartilaginous defects, skeletal dysplasia, and physeal changes due to trauma, infection, or radiation therapy.[2] Early detection allows appropriate interventions before irreversible changes to the physis occur that could ultimately result in long-term patient complications.

Searches of Index to Chiropractic Literature (ICL), PubMed, PubMed Central, MEDLINE Complete, Academic Search Premier, Alt HealthWatch, Audiobook Collection (EBSCOhost), CINAHL Complete, eBook Collection (EBSCOhost), Library, and Information Science & Technology Abstracts yielded no case studies, reports, or research articles of Blount disease or tibia vara and chiropractic evaluation or intervention.

CASE PRESENTATION

A 13-year-old white male multi-sport athlete sought care in an outpatient chiropractic clinic for evaluation of persistent left knee pain. Starting at age 10, the patient experienced 2-3 knee pain episodes per year, located at the region of the lateral tibial condyle and fibular head, which was attributed by the parents to growing pains or possible Osgood-Schlatter disease (tibial tubercle apophysitis). Each episode of knee pain was managed successfully with 1-2 chiropractic treatments. Shortly after turning 13, the patient's knee pain became persistent. The chiropractor observed unilateral outward bowing and a lateral thrust in the patient's gait; bilateral AP and lateral knee radiographs were performed.

Findings of the bilateral knee radiographs included left proximal medial tibial physeal irregularity, with depression of the adjacent medial epiphyseal articular surface and the medial metaphysis, and a medial metaphyseal beak (**Figure 1** arrow); subtle widening and irregularity of the right proximal medial tibial physis; diaphyseal-metaphyseal angle of Drennan at 21° on the left and 9° on the right (normal <11°; **Figure 1** angle); left anterior tibial bowing was not identified. A diagnosis of Blount disease was made.

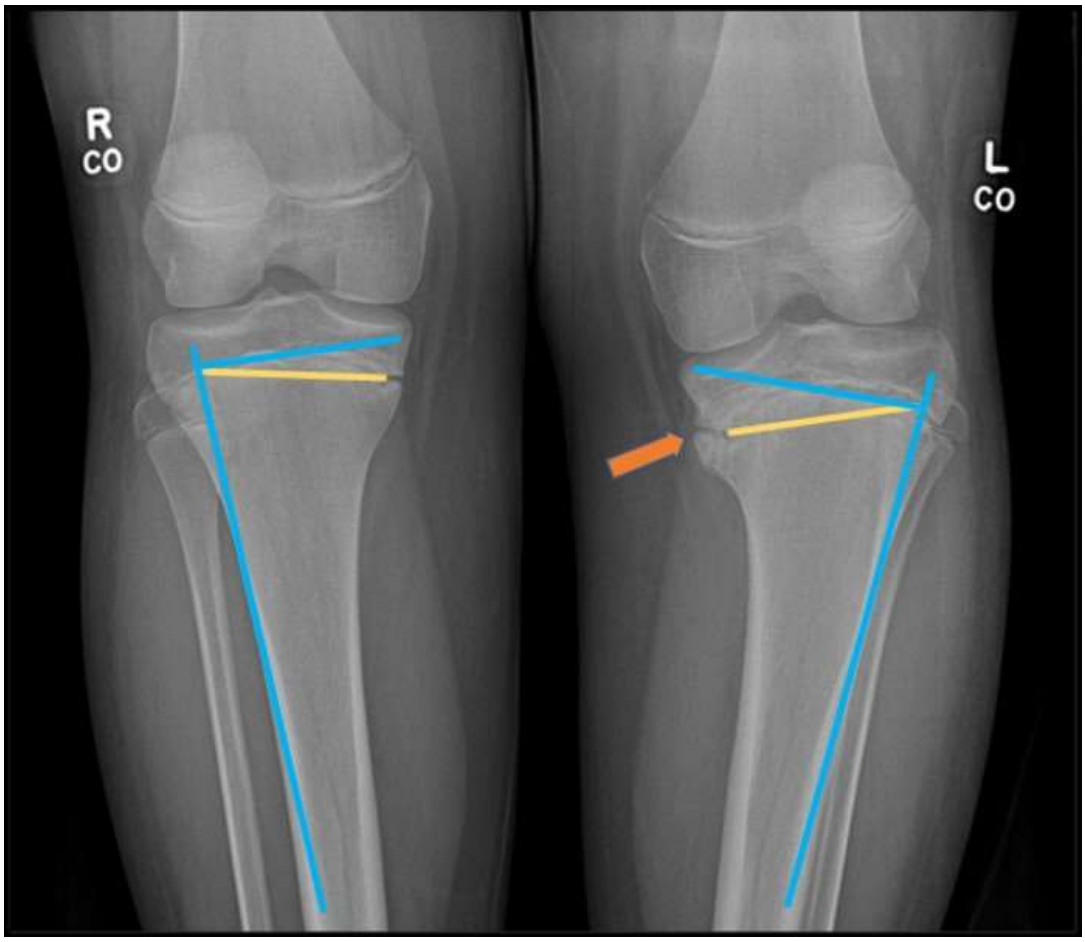

Figure 1: Anteroposterior radiograph of the bilateral knees of a 13-year-old male with left medial proximal tibial slope, increased metaphyseal-diaphyseal angle (MDA) and metaphyseal beaking (arrow).

The chiropractor referred the patient to an orthopedic clinic where surgical correction with an osteotomy was recommended. The patient and his parents were concerned about the extensive nature of this intervention and the projected 6 to 8-month recovery period. They sought consultation with a university-based orthopedic clinic, during which a lateral tension band hemi-epiphysiodesis with plate and screw fixation was recommended. The patient underwent this surgical correction approximately 8 weeks following the initial diagnosis of Blount disease and was discharged the same day (**Figure 2**). He was weight-bearing at the time of discharge, was off pain medication after 1 week, and was restricted from sports participation for 3 weeks. No rehabilitation program was prescribed.

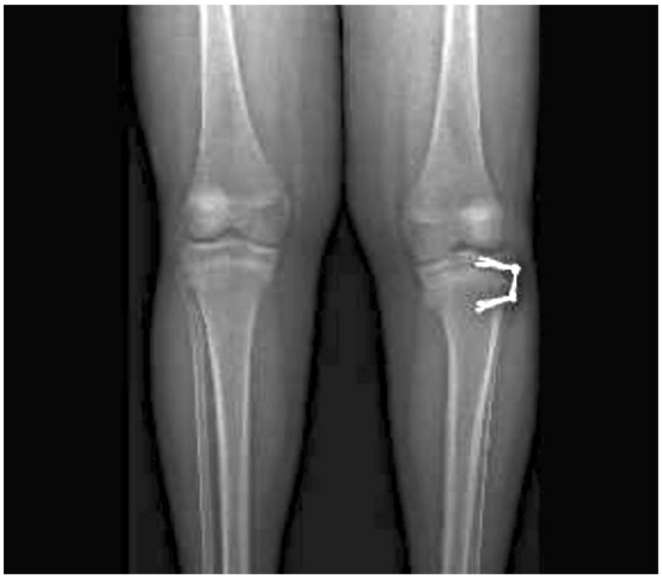

Figure 2: Post-surgical (same day) anteroposterior radiographs of the bilateral knees of a 13-year-old male following tension-band plate placement at the left lateral tibia.

The patient underwent post-surgical evaluations at one month and 6 months and is scheduled for a 1-year evaluation. A second surgery to remove the plate will occur when the genu varum is effectively reduced. The patient is not restricted from any activity unless the activity results in pain.

DISCUSSION

Concerns of bowing deformities of the lower extremities in children may prompt their parents or caregivers to seek evaluations by chiropractic, pediatric, and orthopedic physicians. From the onset of walking to approximately age 2, physiological tibia vara or bowing is typical, owing to the expected growth and development of the tibia's coronal and transverse planes. Between ages 2 and 3, the knock-knee appearance of exaggerated tibial valgus is commonly seen, followed by lessening of the angulation around ages 6 to 7 to the typically slight valgus alignment seen in adults.[1]

Pathological causes of pathological lower leg bowing include Blount disease (most common), dietary vitamin D deficiency and vitamin D-resistant rickets, renal osteodystrophy, proximal tibial metaphyseal fibrocartilaginous defects, skeletal dysplasia,[1] and physeal changes due to trauma, infection, or radiation therapy.[2] In this patient's case, the unilateral presentation and the lack of cupping and fraying of the metaphyseal margins ("paintbrush metaphysis"), the absence of metaphyseal widening, and the lack of transverse lucent lines with adjacent sclerosis (Looser zones or pseudo fracture lines) did not support a radiographic diagnosis of vitamin D deficiency or vitamin-resistant rickets.

The progressive proximal tibial multiplanar varus deformity now known as Blount disease was first described as "tibia vara" in 1922 by Phillip Erlacher.[3] In 1937, Walter Putnam Blount reported on 28 cases, including 13 patients within his practice, in his article Tibia

Vara: Osteochondrosis Deformans Tibiae, published in The Journal of Bone and Joint Surgery.[4,5]

Blount disease is an acquired developmental growth disorder of unknown pathophysiology of the posteromedial proximal tibial physis and epiphysis,[6] leading to progressive genu varum deformity. The clinical presentation of the condition is commonly described in two forms: Infantile and Adolescent. Infantile Blount disease is identified as a varus deformity in children 2-5 years of age; adolescent or late-onset Blount disease develops in children 10 and older. In 1984, Thompson described a juvenile classification for patients 4 to 10 years of age, which is a less commonly utilized categorization today[6] and is considered a classification of untreated, poorly treated, or recurrent Blount disease.[7] The estimated prevalence of the infantile form of Blount disease in the United States is approximately 1%.[1]

Distinguishing between physiological and early-onset pathological forms of persistent tibia vara can be achieved through review of risk factors, clinical findings, and radiographic features. The risk factors for infantile or early-onset Blount disease include obesity; black African, African American, and Afro-Caribbean race; and ambulation prior to age 10 months.[1] Infantile Blount disease onset is typically prior to age three, is present bilaterally but asymmetrically in greater than 50% of cases, and is more prevalent in females.[3] Delayed growth at the posteromedial region of the proximal tibial physis causes flexion, internal rotation, varus deformity of the tibia,[1] and leg length discrepancy of approximately 1 cm.[7] The results of permanent epiphysiodesis (premature closure of the physis) mark a watershed stage of the disease in which the clinical features will include tibia varus and a gait disturbance of lateral (varus) thrust, amplifying the appearance of the bowed leg(s) during the initial phase of weight-bearing.

The risk factors of late-onset Blount disease are similar to those of early-onset Blount disease and comprise obesity, Afro-Caribbean race, and possible pre-existing tibia vara in children older than 10. Radiographic features include delayed physeal ossification at the posteromedial tibia, with widening of the medial tibial epiphyseal-diaphyseal growth center, and increased likelihood of medial femoral condyle hypertrophy and compensatory ankle valgus than in the early-onset form. The tibial deformities are less pronounced, with varus deformity occurring first, followed by medial tibial rotation and anterior tibial bowing; epiphysiodesis is rarely identified (**Table 1)**.[7]

	Infantile Blount Disease Characteristics	Adolescent Blount Disease Characteristics
Age of onset	1-3 years	10 or older
Obesity as a risk factor	30-60%	90%
Race as a risk factor	Black	Black
Sex as a risk factor	Female	Male
Distribution	Usually bilateral	Usually unilateral
Degree of varus deformity	Often >25 degrees	Rarely >30 degrees
Degree of epiphyseal angle	Often >25 degrees	Rarely >30 degrees
Recurrence after osteotomy	Common	Rare

Table 1: Distinguishing characteristics of infantile and adolescent Blount disease.[7]

Standing long-leg (full) anteroposterior radiographs are standard in the initial assessment of suspected pathological tibia vara.[8] The radiographic features of early-onset Blount disease are most evident after age 2.5, permitting differentiation from other causes of tibia vara.[7] The metaphyseal-diaphyseal angle (MDA), also known as the Angle of Drennan, is determined as the angle between a line perpendicular to the long axis of the lateral tibial cortex and a line in the transverse plane of the proximal tibial metaphysis (**Figure 1**).[8] A Drennan angle greater than 16° is considered highly indicative of pathological tibia vara, with an estimated 95% chance of progression. Metaphyseal-diaphyseal angles less than 10° have high likelihood (95%) of natural resolution of the bowing deformity. Drennan angles between 11-16° require close observation for the progression of tibia vara.[8]

Radiographic features and a classification system of infantile Blount disease were described in 1964 by Langenskiold and Riska.[1] Langenskiold's six stage radiographic classification system (**Table 2**), along with the Fort-de-France classification by Catonne,[7] is still used today by orthopedic surgeons to guide management decisions and predict outcomes,[9] and is based on progression of proximal tibial varus deformity, prominent osseous projection of the medial metaphysis, and varying epiphyseal findings ranging from absence to osseous fragmentation; in severe cases, physeal bony bars are seen.[10]

Langenskiold and Riska Infantile Blount Classification System		
Stage 1:	Ages 2-3	Irregular metaphyseal ossification line, slowed medial epiphyseal growth, and medial metaphyseal osseous projection (beak)
Stage 2:	Ages 2.5-4	Acute slope of the medial tibial ossification line, medial metaphyseal osseous beak, delayed growth of the medial epiphysis
Stage 3:	Ages 4-6	Increased depression of the metaphyseal beak, development of metaphyseal depression, increased wedging, and continued delayed growth of the medial epiphysis
Stage 4:	Ages 5-10	Growth center narrowing, epiphyseal enlargement, increased depth of metaphyseal depression, depression of the medial epiphysis into the medial metaphysis
Stage 5:	Ages 9-11	Separation of the bony epiphysis into two segments, partially doubled epiphyseal ossification line, medial articular slope
Stage 6:	Ages 0-13	Medial tibial growth arrest with ossification of the physis, normal lateral tibial growth

Table 2: Langenskiold and Riska six stage classification system of Blount disease[10]

Examination with magnetic resonance imaging (MRI) allows a more accurate evaluation of the cartilaginous tibial angle, the ligaments, the menisci, and the physeal blood supply. Evidence of homogeneous blood supply and absent signal changes in the physis support conservative management options.[7]

Few conservative non-operative treatments for early-onset Blount disease are available and their efficacy is disputed.[7] Orthotic treatment with a year or more of full leg bracing to limit knee movement and to apply valgus pressure to the knee may be appropriate and effective

for non-obese children younger than 3 years of age,[7] and is most efficacious in Langenskiold stage I or II disease. If orthotic treatment is not successful, surgical correction with osteotomy before the child reaches age 4 is indicated.[11]

Two primary surgical options are employed in the treatment of both early- and late-onset Blount disease: osteotomy and placement of tension band screw plates. There are several forms of osteotomy procedures, e.g., oblique, Z, V, inverted V, dome, closing and open wedge forms,[12] all of which remove bone from the tibia and/or elevate a portion of the tibia, changing its proximal articular surface angle, thereby reducing the varus load.[11]

In the 2006 review article *Guided growth: 1933 to the present*, Stevens advocated for tension-band plates (TBPs) as an alternative surgical intervention to osteotomies for leg length discrepancies and angular deformities.[13] The placement of a plate adjacent to the epiphysis and metaphysis through which one epiphyseal and one metaphyseal screw are threaded creates a focal hinge at the margin of the growth center, arresting the growth without violating the physis. Tension band plates are currently the most used implants for growth arrest. Complications of TBP interventions include early growth plate closure, screw breakage, and failure to complete normalization of the mechanical axis, with screw breakage in obese patients being the most common of all TBP complications.[14] Osteotomy surgery complications comprise peroneal nerve paresthesia, septic arthritis, osteomyelitis, misalignment, and union failure.[15]

Approximately 80% of patients receiving surgical re-alignment prior to age 4 achieve a full recovery.[16] Patients older than age 4 at the time of surgery, patients with late-onset Blount disease, patients with Langenskiold stages 5 and 6, and surgical overcorrection resulting in 15 degrees or less of valgus increase the likelihood of tibia vara recurrence.[17] While obesity has been established as the most-strongly associated risk factor for the degree of deformity in Blount disease, obesity has not been associated with treatment failure.[18]

CONCLUSION

This case report describes the clinical presentation, radiographic findings, risk factors, and complications of Blount disease, a pathological form of tibia vara. This case report also describes the necessity of thorough investigation and timely referral for surgical intervention to limit complications of delayed diagnosis and delayed treatment of Blount disease.

LIMITATIONS

As this report describes a single patient's clinical presentation, diagnostic evaluations, and treatment, generalization of this report's content to other individuals with similar clinical presentations should be avoided.

CONSENT

Written informed consent was obtained from the patient and parent/legal guardian for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

COMPETING INTERESTS

The authors declare they have no competing interests.

ACKNOWLEDGEMENTS

The authors wish to thank the patient and his parents for permission to share this information about Blount disease.

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