The Association between Idiopathic Clubfoot and Increased Internal Hip Rotation

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Abstract Clinical observation suggests the coexistence of increased internal hip rotation in limbs with clubfoot, thereby providing an additional, and perhaps overlooked, site of deformity to account for an intoeing gait in these limbs. Furthermore, assuming a genetic basis exists for exaggerated femoral and/or acetabular anteversion, which are the possible cause(s) for increased internal hip rotation, this association could provide another key to the multifactorial etiology of clubfoot. We asked whether such an association exists and retrospectively reviewed 114 children (178 clubfeet). We then tested for an association between clubfoot and increased internal hip rotation. These rotational measurements were compared with published normative data on torsion in children. In cases of unilateral clubfoot, an additional analysis compared the rotational

profiles of the affected and unaffected extremities. Increased internal hip rotation occurred more frequently in limbs with idiopathic clubfoot. In patients with unilateral clubfoot, the affected extremities manifested greater internal hip rotation than the unaffected extremities, whereas the latter showed no difference in internal hip rotation compared with normative values. Clinical evaluation of intoeing in children with a history of clubfoot should include a rotational profile to determine the level(s) of deformity and guide therapeutic intervention.

Level of Evidence: Level III, prognostic study (case control study). See Guidelines for Authors for a complete description of levels of evidence.

Introduction

Clubfoot, the most common congenital deformity of the musculoskeletal system, is believed to be etiologically multifactorial [2, 5, 7, 8, 10, 14, 26, 34]. This means it is the result of the interaction of several genes and, to some extent, environmental factors. The etiologic genes have not been specifically identified and only some environmental associations such as maternal smoking [14, 29] and familial ligament laxity [24] have been elucidated. The identification of genetic and environmental associations should help determine the etiology.

Studies investigating idiopathic clubfoot have reported that other musculoskeletal abnormalities commonly occur in conjunction with this foot deformity. For instance, numerous studies document the association between tibial shortening, internal tibial torsion, and idiopathic clubfoot [15, 18, 27, 31], leading some investigators to conclude idiopathic clubfoot is part of a pathologic condition affecting the entire distal lower extremity. However,

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clinical observation by the senior author (VSM) over a period of more than two decades suggests the coexistence of increased internal hip rotation in limbs with clubfoot. Furthermore, anecdotal comments exist in the pediatric orthopaedic literature suggesting clubfoot may be associated with more proximal abnormalities, specifically increased femoral anteversion [22]. These anecdotal observations, if they were to withstand scrutiny, would broaden our understanding of idiopathic clubfoot as part of a condition that affects the entire lower extremity. The interaction of the genes that cause tibial torsion, limb shortening, and/or femoral or acetabular anteversion with the gene or genes that code for clubfoot could be among the factors necessary to create a clubfoot.

Additionally, placing idiopathic clubfoot in this larger context has clinical implications. It calls attention to deformities at multiple levels of the lower extremity, the manifestations of which may obscure or compound one another. For instance, increased femoral anteversion, a proximal limb deformity, creates inward rotation of the limb and foot that to the casual observer might appear to represent residual or recurrent clubfoot deformity when none exists.

We were unable to find any clinical studies evaluating hip rotational profiles in children with clubfoot. In the orthopaedic literature and in conversations among many orthopaedists, greater than average internal hip rotation combined with less than average external hip rotation has generally been equated with femoral anteversion, despite the fact that rotational motion at the hip joint does not necessarily accurately represent the torsional shape of the femur [1, 3, 4, 9, 19, 21, 28, 32, 33]. This is in contrast to the clinical assessment of tibial torsion in which the torsional shape of that bone is measured directly by comparing the axis of the foot to that of the femur when the knee is flexed 90° and the ankle is in neutral dorsiflexion. The assumption that increased internal hip rotation equals femoral anteversion ignores acetabular anteversion, which can produce the same rotational variation [32]. Since clinical torsional assessment cannot differentiate between the two, we have chosen in this article to discuss our findings in terms of increased internal hip rotation as a proxy for femoral and/or acetabular anteversion. The exact site(s) of deformity is not relevant to the purposes, findings, and at least some of the implications of this study.

In 1992, before computed tomography techniques for assessing torsion were perfected, Ruwe et al. reported that CT underestimated femoral anteversion and that clinically determined anteversion more accurately matched the femoral anteversion determined intraoperatively with metallic implants and fluoroscopy [28]. They did not study limbs with clubfoot. Using CT, Cuevas de Alba et al. in 1998 reported no difference in anatomic femoral anteversion

between normal limbs and those with clubfoot [6]. They did not measure acetabular version nor did they report the clinical rotational hip profiles of their subjects.

Fortunately, the literature has established normative data for the rotational hip profiles of children against which the rotational hip profiles of children with idiopathic clubfoot may be compared [32]. Moreover, several studies have established the reproducibility of the routine clinical assessment of torsional alignment that is most often used by the pediatric orthopaedist [1, 3, 4, 9, 19, 21, 28, 32, 33]. This information helps to justify the use of the physical exam, in contrast to imaging studies, because it is reproducible, quick, inexpensive, avoids radiation exposure, and measures what we observe clinically.

We tested the following hypotheses: (1) increased internal hip rotation exists in extremities affected by clubfoot when compared with normative data on hip rotation in children; and (2) the extremity ipsilateral to a clubfoot manifests increased internal hip rotation compared with the contralateral, unaffected extremity in cases of unilateral clubfoot.

Materials and Methods

We identified 203 patients (350 clubfeet) in a patient database between 1985 and 2005 by a systematic search using International Classification of Diseases, 9th revision codes. Our selection criteria included patients aged 3 to 18 years of age with a diagnosis of idiopathic clubfoot as documented in the medical records. All patients had been treated by serial casting and, in many cases, by surgical release. Most of the early patients in the series underwent conventional open surgical procedures, although most of the patients in the last decade of the study were treated using the Ponseti nonsurgical method [25]. We obtained all data in this single-center study by retrospective chart review of orthopaedic notes written by the senior author (VSM), who established the diagnosis of clubfoot and performed the rotational hip examinations. The torsional assessment is being reported as the magnitude of internal hip rotation, in degrees, rather than as femoral anteversion. As noted above, clinical torsional measurements cannot differentiate between femoral and acetabular anteversion, so we view increased internal hip rotation as a proxy for femoral and/or acetabular anteversion. We excluded patients from the study using the following criteria in an attempt to focus on cases of idiopathic clubfoot and to minimize any confounding clinical factors: (1) teratologic equinovarus deformity, including arthrogryposis, cerebral palsy, congenital constriction bands, myelomeningocele, and other neurologic diseases or syndromes; and (2) other limb deformities such as developmental dysplasia of the

hip, femoral or tibial fractures, and other pathologic conditions of the knee and hip. Of the 203 patients who met the inclusion criteria, 114 patients with 178 clubfeet had rotational profiles in the medical records (Fig. 1). Sixty-four patients (128 feet) had bilateral involvement, 25 patients had unilateral right clubfoot, and 25 had unilateral left-sided deformity.

We collected hip rotation measurements with the patient in the prone position and with the knees flexed 90°; all measurements were made by one investigator (VSM) using visual estimates. While maintaining a level pelvis, the hips were internally and externally rotated to the position maintained by gravity alone while ensuring relaxation of the hip and knee musculature (Fig. 2). With 0° defined as perpendicular with the examination table and 90° as parallel with the table, both internal and external hip rotation were determined and recorded for both extremities.

Fig. 1 The distribution of participants by age ($n = 114$) is shown.

Fig. 2 The clinical assessment of internal hip rotation is demonstrated, showing increased internal hip rotation ipsilateral to the right clubfoot.

We compared clinical rotational hip data with the age-matched normative data published by Staheli et al. [32]. The mean and confidence intervals for internal and external rotation for these extremities were then plotted with the age-matched normative data. The group individual normative internal rotation values by age were compared by t-test with the rotation values of 64 children with bilateral clubfeet and 50 with unilateral clubfeet. For participants with unilateral clubfoot, we compared the values for the hip ipsilateral to the clubfoot with those for the contralateral (unaffected) limb to determine relative torsional variation using a paired t-test. The mean internal hip rotation ipsilateral to the clubfoot was 62.4° (SD 14.7°) with a range of 15°–85°. For the contralateral unaffected limb, the mean internal hip rotation was 50.6° (SD 15.0°) with a range of 15°–80° with no significant difference in variance ($p = .52$ and $.73$ respectively). The incidence of a difference in internal hip rotation greater than or equal to 10° as compared to the nonaffected limbs was tested with the chi square test in the sample of 50 children with unilateral clubfeet. We performed all analyses completed with SPSS version 13.0 (SPSS Inc, Chicago, IL).

Results

The mean internal rotation of the hips ipsilateral to both unilateral and bilateral clubfeet was greater ($p = 0.001$) than that of the average age-matched normative data (Fig. 3). Conversely, the mean external rotation of these hips was less ($p = 0.001$) than the average age-matched normative control data. When considered separately, the hips of patients with bilateral clubfeet had greater ($p = 0.001$) mean internal rotation than the matched normative data (Fig. 4).

When comparing the affected limbs of patients with unilateral clubfeet, we observed more ($p = 0.001$) internal hip rotation than in the normative controls (Fig. 5) and more ($p = 0.001$) than in the contralateral (nonclubfoot) limbs (Fig. 6). Compared to the contralateral limb, a greater percentage of patients with unilateral clubfoot (41 of 50, or 82%) had greater than or equal to ten degrees of increased internal hip rotation than those with less than ten degrees difference (9 of 50, or 18%) ($p = 0.02$). The amount of excessive internal rotation in the hip ipsilateral to a clubfoot was clinically apparent but rarely dramatic, usually only 10° to 20°, making careful assessment important. Only 26 of 178 cases (15%) of extremities with clubfeet had internal rotation of the hip beyond two standard deviations that, according to Staheli's normative data, is 80°.

Finally, the mean internal hip rotation of the contralateral nonclubfoot limb was similar to ($p = 0.14$) that of the normative data (Fig. 7).

Fig. 3 There was increased internal rotation of hips ipsilateral to a clubfoot ($n = 178$, bilateral and unilateral) sorted by age and compared with age-matched normative data [32]. Open circles = mean measurements for all bilateral and unilaterally affected limbs in study patients by age group; dashed lines = average and two standard deviations for study patient data; solid circles = mean measurements for age-matched normative controls; solid lines = average and two standard deviations for age-matched normative control data.

Fig. 5 There was increased internal rotation of hips ipsilateral to a unilateral clubfoot ($n = 50$) sorted by age and compared with age-matched normative data [32]. Open circles = mean measurements for all unilaterally affected limbs in study patients by age group; dashed lines = average and two standard deviations for study patient data; solid circles = mean measurements for age-matched normative controls; solid lines = average and two standard deviations for age-matched normative control data.

Fig. 4 There was increased internal rotation of hips in limbs with bilateral clubfeet ($n = 128$) sorted by age and compared with age-matched normative data [32]. Open circles = mean measurements for all bilateral clubfoot limbs in study patients by age group; dashed lines = average and two standard deviations for study patient data; solid circles = mean measurements for age-matched normative controls; solid lines = average and two standard deviations for age-matched normative control data.

Fig. 6 There was increased internal rotation of hips ipsilateral to a unilateral clubfoot ($n = 50$) sorted by age and compared with the contralateral nonaffected side. Open circles = mean measurements for hips ipsilateral to a unilateral clubfoot by age group; dashed lines = average and two standard deviations for hips ipsilateral to a unilateral clubfoot; solid circles = mean measurements for hips in contralateral nonaffected side by age group; solid lines = average and two standard deviations for hips in contralateral nonaffected side.

Fig. 7 There was no difference in internal rotation of hips in the contralateral nonaffected limbs (in patients with unilateral clubfoot; $n = 50$) sorted by age and compared with age-matched normative data [32]. Open circles = mean measurements for contralateral nonaffected limbs (in unilateral cases) by age group; dashed lines = average and two standard deviations for contralateral nonaffected limbs (in unilateral cases); solid circles = mean measurements for age-matched normative controls; solid lines = average and two standard deviations for age-matched normative control data.

Discussion

Clubfoot is a common congenital deformity that is considered multifactorial in etiology. Some of the factors are genetic and others are environmental. Identification and investigation of all apparent associations with clubfoot should help to determine the complex etiology. Over the past two decades, the senior author (VSM) has observed that, based on physical examination, the hip ipsilateral to a clubfoot has greater than normal internal rotation. We tested, and confirmed, the hypotheses that increased internal hip rotation exists in extremities affected by clubfoot when compared with normative data on hip rotation in children; and the extremity ipsilateral to a clubfoot manifests increased internal hip rotation compared with the contralateral, unaffected extremity in cases of unilateral clubfoot.

A potential shortcoming of the approach used in this study is the fact that it is a retrospective chart review. Eighty-nine patients could not be included because no rotational profile had been recorded in the medical record and they did not present for further examination. It is possible that the exclusion of these patients could introduce selection bias into our study. However, selection bias could not account for the fact that 41 of 50 subjects (82%) with

unilateral clubfoot manifested a greater than 10° increase in internal hip rotation when comparing the extremity with clubfoot with their unaffected extremity.

Another potential shortcoming of the study is the fact that the hip rotation measurements were made by a single examiner. Several studies [3, 4, 19] have demonstrated that variability by one tester, as in our study, is less than inter-examiner variability, especially for a specific population. Luchini and Stevens specifically cautioned against inter-examiner reproducibility (not intraexaminer reproducibility) when focusing on the torsional exam in children younger than 3 years of age, a difficult population to assess and one that was, therefore, excluded from the present study [20].

The reliability of our measurements, when compared with the published normative data, was confirmed by our finding that there was no difference in internal hip rotation between the contralateral nonaffected limb in unilateral cases and the age-matched normative control data.

Clinical examination, rather than imaging studies, was used to assess hip rotation, because the exact site(s) of deformity, whether femoral or acetabular, was less important to us than the observation that an association between idiopathic clubfoot and increased internal hip rotation exists. The literature documents the reproducibility of the clinical assessment of torsion to within 4° , regardless of the specific clinical method used by the individual investigator, in multiple studies spanning four decades [1, 3, 4, 9, 19, 21, 23, 28, 32, 33]. Our finding of greater than 10° of relative internal hip rotation in an extremity with a clubfoot compared with an unaffected limb notably falls beyond this measurement, thereby increasing confidence in the validity of our data. Other studies support the assertion that physical examination is more clinically valid than imaging modalities such as CT and MRI in evaluating torsion [12, 16, 28, 33].

The association between clubfoot and femoral anteversion (not internal hip rotation) was investigated by Cuevas de Alba et al. in 1998 using CT measurements on a sample population of 47 children in which there were 70 clubfeet [6]. They found no difference in femoral anteversion between the normal limbs and those with clubfoot. If this is true, it suggests that our clinical observations are the result of acetabular anteversion, an anatomic feature not measured in the CT study of Cuevas de Alba et al. [6], but one that can account for greater internal and less external hip rotation than normal. The combination of femoral and acetabular version results in the hip rotational profile that is measured and clinically apparent. Ruwe et al. reported the clinical examination more accurately matched femoral anteversion measured intraoperatively, using metallic implants and fluoroscopy, than those measured with either preoperative radiographic techniques or CT [28]. That study was performed in 1992, perhaps before the CT

technique was perfected. Regardless of the reason for inaccuracy of the CT in that study, the important observation was that the clinical determination of femoral anteversion was within 4° of the anteversion measured intraoperatively. More recent studies [16] suggest a CT scan is no more reliable than the 4° mean error produced by the physical examination. The radiation exposure, expense, and inconvenience of obtaining the information by CT are, therefore, hard to justify. Although multiple studies assert that MRI is more accurate, Tamari et al. concede the clinical examination “had a good level of association” and is the preferred clinical screening tool [12, 33].

Unlike CT, physical examination is reproducible, quick, inexpensive, avoids radiation exposure, and measures what we observe clinically [28]. Using the prone torsional profile examination, the preferred screening tool used in the clinical setting to assess hip rotation, we found an increase in internal hip rotation in extremities with clubfoot deformity compared with published normative values. The differences are clinically apparent, but rarely extreme. Moreover, we show that in patients with unilateral clubfoot, the extremity ipsilateral to the foot deformity manifests increased internal hip rotation when compared with the unaffected extremity with an average difference between affected and unaffected sides of greater than 10°. Previously published literature describes idiopathic clubfoot as a deformity of the distal extremity with associated tibial shortening and internal tibial torsion [15, 18, 31]. Our findings are the first to suggest idiopathic clubfoot is more than a distal extremity problem. The whole limb manifests abnormal clinical features.

We did not attempt to establish normative data for the population of children with a clubfoot deformity, but did call attention to the fact that these children often have deformities at multiple levels in the lower extremity. A 10° increase in internal hip rotation may have clinical importance when combined with internal rotation from a clubfoot that manifests residual adductus of the forefoot or inversion of the hindfoot. However, of more importance is identifying the true site(s) of intoeing when the clubfoot is well-corrected. The senior author has been consulted, after the fact, on several cases in which an adequately corrected clubfoot had undergone an inappropriate deformity-producing operation on the foot or tibia for the indication of intoeing that was, in fact, the result of increased internal hip rotation. Evaluation of any foot deformity, particularly a clubfoot deformity, must include evaluation of the entire limb.

The association we found between idiopathic clubfoot and increased internal hip rotation has implications for the genetic basis of clubfoot. Guidera et al. reviewed the multifactorial intrauterine factors that contribute to lower extremity torsional deformities [13]. The consensus in the

literature is that intrauterine molding accounts for most torsional abnormalities and once compressive forces are removed after birth, a gradual decrease of these deformities is usually observed [11, 17, 31]. However, Staheli and others concede that genetic factors may account for more pronounced or persistent torsional abnormalities [8, 10, 14, 30]. It is unknown if a single gene or multiple factors are involved. As previously noted, a number of investigators have proposed that idiopathic clubfoot has a genetic basis and is multifactorial in origin [2, 5, 7, 8, 26, 34]. The association we found between increased internal hip rotation and idiopathic clubfoot suggests that the genetic basis of femoral and/or acetabular anteversion, which are the likely cause(s) for increased internal hip rotation, may play a role in the etiology of idiopathic clubfoot. The converse is not suggested by this study.

Idiopathic clubfoot has traditionally been viewed as part of a constellation of deformities that affects the distal part of the extremity. Our data suggest a more proximal deformity is often present in the form of increased internal hip rotation, suggesting the entire extremity is involved in the underlying disease process. Clinical evaluation of intoeing in children with a history of clubfoot, whether or not corrected and regardless of type of correction, should include a careful rotational profile to determine the level of deformity, or deformities, and to guide therapeutic intervention.

References

1. Ashton BB, Pickles B, Roll JW. Reliability of goniometric measurements of hip motion in spastic cerebral palsy. *Dev Med Child Neurol.* 1978;20:87–94.
2. Barker S, Chesney D, Miedzybrodzka Z, Maffulli N. Genetics and epidemiology of idiopathic congenital talipes equinovarus. *J Pediatr Orthop.* 2003;23:265–272.
3. Boone DC, Azen SP. Normal range of motion of joints in male subjects. *J Bone Joint Surg Am.* 1979;61:756–759.
4. Boone DC, Azen SP, Lin CM, Spence C, Baron C, Lee L. Reliability of goniometric measurements. *Phys Ther.* 1978;58:1355–1390.
5. Cowell H, Wein B. Genetic aspects of club foot. *J Bone Joint Surg Am.* 1980;62:1381–1384.
6. Cuevas de Alba C, Guille JT, Bowen JR, Harcke HT. Computed tomography for femoral and tibial torsion in children with clubfoot. *Clin Orthop Relat Res.* 1998;353:203–209.
7. de Andrade M, Barnholtz JS, Amos CI, Lochmiller C, Scott A, Risman M, Hecht JT. Segregation analysis of idiopathic talipes equinovarus in a Texan population. *Am J Med Genet.* 1998;79:97–102.
8. Dietz F. The genetics of idiopathic clubfoot. *Clin Orthop Relat Res.* 2002;401:39–48.
9. Ekstrand J, Wiktorsson M, Oberg B, Gillquist J. Lower extremity goniometric measurements: a study to determine their reliability. *Arch Phys Med Rehabil.* 1982;63:171–175.
10. Engell V, Damborg F, Andersen M, Kyvik KO, Thomsen K. Club foot: a twin study. *J Bone Joint Surg Br.* 2006;88:374–376.

11. Fabry G, Cheng LX, Molenaers G. Normal and abnormal torsional development in children. *Clin Orthop Relat Res.* 1994; 302:22–26.
12. Guenther KP, Tomczak R, Kessler S, Pfeiffer T, Puhl W. Measurement of femoral anteversion by magnetic resonance imaging—evaluation of a new technique in children and adolescents. *Eur J Radiol.* 1995;21:47–52.
13. Guidera KJ, Ganey TM, Keneally CR, Ogden JA. The embryology of lower-extremity torsion. *Clin Orthop Relat Res.* 1994; 302:17–21.
14. Honein MA, Paulozzi LJ, Moore CA. Family history, maternal smoking, and clubfoot: an indication of a gene-environment interaction. *Am J Epidemiol.* 2000;152:658–665.
15. Hutchins PM, Rambicki D, Comacchio L, Paterson DC. Tibiofibular torsion in normal and treated clubfoot populations. *J Pediatr Orthop.* 1986;6:452–455.
16. Jaarsma RL, Bruggeman AW, Pakvis DF, Verdonschot N, Lemmens J, Avan Kampen A. Computed tomography determined femoral torsion is not accurate. *Arch Orthop Trauma Surg.* 2004; 124:552–554.
17. Katz K, Naor N, Merlob P, Wielunsky E. Rotational deformities of the tibia and foot in preterm infants. *J Pediatr Orthop.* 1990; 10:483–485.
18. Krishna M, Evans R, Sprigg A, Taylor JF, Theis JC. Tibial torsion measured by ultrasound in children with talipes equinovarus. *J Bone Joint Surg Br.* 1991;73:207–210.
19. Low JL. The reliability of joint measurement. *Physiotherapy.* 1976;62:227–229.
20. Luchini M, Stevens DB. Validity of torsional profile examination. *J Pediatr Orthop.* 1983;3:41–44.
21. Mayerson NH, Milano RA. Goniometric measurement reliability in physical medicine. *Arch Phys Med Rehabil.* 1984;65:92–94.
22. McSweeney A. A study of femoral torsion in children. *J Bone Joint Surg Br.* 1971;53:90–95.
23. Murphy SB, Simon SR, Kijewski PK, Wilkinson RH, Griscom NT. Femoral anteversion. *J Bone Joint Surg Am.* 1987;69:1169–1176.
24. Olshan AF, Schroeder JC, Alderman BW, Mosca VS. Joint laxity and the risk of clubfoot. *Birth Defects Res A Clin Mol Teratol.* 2003;67:585–590.
25. Ponseti IV. *Congenital Clubfoot: Fundamentals of Treatment.* New York: Oxford University Press; 1996.
26. Rebbeck T, Dietz F, Murray J, Buetow K. A single-gene explanation for the probability of having idiopathic talipes equinovarus. *Am J Hum Genet.* 1993;53:1051–1063.
27. Reikeras O, Kristiansen LP, Gunderson R, Steen H. Reduced tibial torsion in congenital clubfoot: CT measurements in 24 patients. *Acta Orthop Scand.* 2001;72:53–56.
28. Ruwe PA, Gage JR, Ozonoff MB, DeLuca PA. Clinical determination of femoral anteversion. A comparison with established techniques. *J Bone Joint Surg Am.* 1992;74:820–830.
29. Skelly AC, Holt VL, Mosca VS, Alderman BW. Talipes equinovarus and maternal smoking: a population-based case-control study in Washington State. *Teratology.* 2002;66:91–100.
30. Staheli LT. Torsional deformity. *Pediatr Clin North Am.* 1977;24:799–811.
31. Staheli LT. Lower positional deformity in infants and children: a review. *J Pediatr Orthop.* 1990;10:559–563.
32. Staheli LT, Corbett M, Wyss C, King H. Lower-extremity rotational problems in children. Normal values to guide management. *J Bone Joint Surg Am.* 1985;67:39–47.
33. Tamari K, Tinley P, Briffa K, Breidahl W. Validity and reliability of existing and modified clinical methods of measuring femoral and tibiofibular torsion in healthy subjects: use of different reference axes may improve reliability. *Clin Anat.* 2005; 18:46–55.
34. Wang JH, Palmer RM, Chung CS. The role of major gene in clubfoot. *Am J Hum Genet.* 1988;42:772–776.