

Clinical and statistical assessment of aetiological factors of genu varum in children

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Abstract

Genu varum (bow leg) represents one of the most common lower limb deformities in children. Its aetiology is divided into two main categories, physiologic and pathologic. It poses a source of concern for the parents. In physiologic genu varum, the condition is self-limiting and usually it needs just clinical follow up. In pathologic cases it is liable for progression with time predisposing for mechanical lower limb problems as well as joint complications. In the physiologic variety, it is important to establish the diagnosis so as to avoid unnecessary treatment. In this study we assessed the

aetiologic factors affecting the incidence of this condition. The results point that the physiologic type is still the commonest in our locality. The second commonest cause was found to be nutritional rickets while other causes were uncommon or even extremely rare. These results are contrary to the common belief that most of the cases in our locality are attributed to rickets. The purpose behind the study is to clarify the aetiologic contribution of various conditions involved so that to base the subsequent management on solid ground.

Introduction

Genu varum (bow-leg) represents one of the most common lower limb deformities in children(1,2,3,4). It is defined as medial deviation of the vertical axis of the leg in relation to that of the thigh (1,2). Clinically, it is assessed in standing position using a goniometer by measuring the angle between the axis of the femur and that of the tibia while the patella is facing forward(4).

Also in this context, it is characterized by increment in the intercondylar distance so that it is more than 6 cm(4). Radiologically, it is assessed with better accuracy using the same axes, again in standing position(2). Very frequently the child is brought by parents who are concerned about his/her cosmetic appearance in absence of any other symptoms. The aetiology of this condition is

divided into two main categories, physiologic and pathologic(5). The physiologic type represents a benign deviation from the normal change in the vertical alignment of the lower limb that occurs during growth and

in most of cases it needs just clinical follow up (6,7) . It's one of the major tasks of the orthopaedist or the pediatrician to differentiate between these two so as to avoid unnecessary treatment.

Physiologic genu varum: The normal newborn child has genu varum(8). This is because of intrauterine position ("Buddha position") which implies hip and knee flexion and internal rotation of the foot and leg. This causes contracture at the medial knee capsule. This alignment increases with growth reaching a maximum tibiofemoral alignment of 10-15 degrees at the age of 12-14 months(8). From this time, the varus alignment starts to decrease as the capsular contracture stretches so that a neutral one is attained at the age of 18 months. After this time the alignment changes into valgus with a maximum amount is reached at the age of 4 years which is about 8 degrees(8). From this time the valgus decreases so that the adult alignment of 6 degrees is reached at the age of 6-8 years(8). Physiologic genu

varum results from variation of this pattern. It is defined as a medial deviation of the tibiofemoral alignment of more than 10 degrees seen after the age of 18 months in otherwise normal child(7,8). The condition is typically bilateral although not necessarily symmetrical. The child is usually an early walker (before the age of 12 months)(9). Radiologically, it is characterized by normal physis and medial bowing of the proximal tibia and usually of distal femur. Sometimes there is delayed ossification of the medial part of the epiphysis as well as flaring of the metaphysis of the proximal tibia and distal femur(2). It may be associated with internal tibial torsion. It usually presents after walking. The fate of this condition is spontaneous resolution(7,8).

Pathologic genu varum: It includes the following entities:

1- Metabolic bone disease: This encompasses conditions associated with defective bone mineralization (10) . These include endocrine disorders, rickets and renal osteodystrophy (10) . Rickets is the commonest by far(11) . The common endocrinopathies involved in this issue are hypothyroidism , hyperparathyroidism, hypopituitarism and hypercortisolism (including exogenous steroid-induced)(10). Rickets includes multiple types of disorders sharing the common feature of lack of vitamin D effect(11,12). The commonest type in developing countries is the nutritional rickets while the hypophosphataemic rickets (vitamin D resistant rickets) represents the commonest type in the developed world (11). Other uncommon types include hypophosphatasia, vitamin D dependent rickets and rickets secondary to hepatic or renal disease(12,13). Vitamin D is obtained from two sources, namely, the skin via the solar effect on 7-dehydrocholesterol (the major source) and the intestine as dietary intake (minor source, mainly meat, fish and dairy)(11,12). It needs activation by hydroxylation in liver and kidney(11,12). Physiologically, it acts on the intestine to increase the absorption of calcium and phosphate and on the bone where it has 2 effects: induction of osteoclastic activity and promotion of osteoid mineralization(11,12). Regardless of the type, the radiological features of rickets are the same(2,3). They

include decreased cortical thickness, flaring and fraying of the metaphysis, widening of the physis and thickening of the epiphysis (2,3). Biochemical changes common to all types of rickets include diminished serum calcium and phosphate and raised level of alkaline phosphatase (except in the rare entity of hypophosphatasia)(12). In vitamin D deficiency, 25-OH D level is decreased(12,13). The calcium phosphate product is normally about 3mmol/L; in rickets this is diagnostically less than 2.4 mmol/L(14,15). Renal osteodystrophy occurs in patients with advanced chronic renal failure due to hypocalcaemia, hyperphosphataemia, secondary hyperparathyroidism, defective hydroxylation of vitamin-D and Aluminum retention(10). Radiologically this condition is characterized by osteopenia as well as osteosclerosis giving the spine what is called the rugger-jersey appearance(10,2). Biochemically, it is characterized by hypocalcaemia, hyperphosphataemia, raised alkaline phosphatase and parathyroid hormone(10).

2- Infantile tibia vara (Blount`s disease) :it results from growth retardation in the medial aspect of the proximal tibial physis and epiphysis leading to progressive genu varum(16). The exact aetiology is not fully understood. It is suggested that it is genetic (autosomal dominant with variable penetrance) or developmental in origin(16,17). Pathologically,

there is necrosis of the medial side of the epiphysis of the proximal tibia and sometimes the distal femur(16,17). Clinically the picture is quite similar to physiologic genu varum with two vital differences: the patient is often obese and there is usually a lateral thrust during walking(16). Radiological signs develop after the age of 18 months(2). They include varus angulation at the metaphyseal-diaphyseal junction, widening and irregularity of the medial physis, medial sloping and irregular ossification of the epiphysis and medial beaking of the metaphysis(2). The metaphyseal-diaphyseal angle was thoroughly studied and it was found that when it is less than 10 degrees, it rules out Blount's disease with 95% accuracy, while when it is more than 16 degrees, it confirms Blount's disease with 95% accuracy(19). Langenskiold put a classification system Blount's disease consisting of 6 stages with increasing severity (18). This system is very useful clinically because it can predict the appropriate treatment. For stages 1 through 3, they can be treated by osteotomy whereas stages 4, 5 and 6 which are characterized by physeal arrest with possible bony bar need complex reconstructive procedures(18). This condition is a progressive one and once diagnosis is made, active treatment should be started. According to clinical studies, corrective osteotomy should be completed before the age of 4-8 years(18).

3- Physeal arrest: This follows either trauma or infection(20). Physeal injuries account for 20-25% of all fractures in children(20). They are classified according the Salter-Harris system into 5 types (recently, a 6th type have been added by Rang) (20,21). The commonest is type 2 (about 75%)(20). Physeal growth affection occurs mostly with unreduced displacement specially with types 3 and 4(20). Physeal growth affection also is seen in the rare type 5 which is characterized by crushing of the physis (20). Growth arrest results in either asymmetrical growth when the bone bar is formed either medially or laterally or in an intraarticular incongruence when the bar is central (21). Radiologically, physeal arrest is characterized by deformed metaphysis, narrowing of the physis at one side, and asymmetrical Harris lines (21).

4- skeletal dysplasias : This term encompasses a heterogenous group of uncommon congenital conditions characterized by abnormal bone growth and/or modeling (22). Each one of these conditions has a characteristic mode of inheritance, clinical picture and XR signs (22). They are classified according to the site of bone affection into epiphyseal, physeal, metaphyseal, diaphyseal and mixed types (22). Those which are associated with genu varum include achondroplasia, osteogenesis imperfecta, fibrous dysplasia, multiple epiphyseal dysplasia, metaphyseal chondrodysplasia, pseudoachondroplasia and focal fibrocartilaginous dysplasia (22,23).

The child is typically short (below the fifth percentile). Only plain XR is needed to confirm the diagnosis (2).

Persistence of genu varum beyond the age of 2 years is abnormal and is due to either severe physiologic bowlegs which is considered as the most common cause or a pathologic condition(24). Below the age of 2 years, XR is not indicated, unless there are clinical signs of pathologic bone affection(24). The presence of rotational malalignment in the form of internal tibial torsion exaggerates the appearance of leg bowing(24). The pathologic type occurs either

uni- or bilaterally and is characterized by a lateral thrust due to varus instability and is progressive with time; on the other hand, physiologic variety is almost always bilateral and usually symmetrical, shows no lateral thrust and tends to resolve with growth(24,25). Children with physiologic genu varum and Blount's disease tend to be early walkers(24). For purpose of grading, genu varum in general is divided according to the amount of the tibiofemoral angle into mild (less than 10 degrees), moderate (between 10 and 25 degrees) and severe (more than 25 degrees)(25).

Patients and methods

Patients included in this study are those below the age of 14 years with genu varum proved clinically and radiologically. We excluded children below 2 years having genu varum on a background of the normal physiologic change in the tibiofemoral alignment. So, patients were received and full history and physical assessment were effected. This included the age, sex, the time when the deformity was first observed and how it changed with time, the age of walking, any other abnormalities seen by the parents, history of trauma or infection, the nutritional history including the breast and formula feeding. Clinical assessment included measurement of the stature and the leg length, observing the gait, evaluation of the deformity

while the patient is standing with the patellae facing forward by using a goniometer and by measuring the intercondylar distance with checking the level of the deformity (femoral, upper tibial or lower tibial) and whether it is smooth or abrupt, being bilateral and whether it is symmetrical or not. Also vital in the evaluation is the state of the ligaments of the knee which when laxen cause lateral thrust. The epiphyses of the long bones were also palpated for any enlargement (rickets). The torsional alignment was also assessed using the thigh foot angle.

XR was taken for the patient under the following indications. 1-genu varum persisting

beyond the age of 2 years. 2-the condition is unilateral or asymmetrical. 3-the angulation is acute in the proximal tibial metaphysis. 4-the possibility of a pathologic condition on clinical ground. Once the condition is confirmed to be physiologic with age below 2 years it was excluded from the statistics. The XR taken is a full length standing bilateral anteroposterior view from the hip to ankle with the patellae pointing forward. The focus of radiologic assessment includes the site and amount of the deformity, the status of the metaphyses, physes and epiphyses of the long bones, the metaphyseal-diaphyseal angle (less than 11

degrees in physiologic genu varum and more than 11 degrees in Blount's disease) and the features of rickets, Blount's disease and skeletal dysplasias emphasized above. Biochemical assays were performed in suspected metabolic disorders and included serum calcium, phosphate and alkaline phosphatase. When the diagnosis was set on a solid ground then the case was labeled accordingly. However, in equivocal cases, the case was followed on regular intervals until the diagnosis settles on the base of subsequent evaluation, XRs, investigations and progression.

Results:

During a period of 32 months (from 1, December, 2009 to 31, July, 2012) we received 567 patients with genu varum in an out-patient setting. All of these cases presented as the leg deformity being the chief complaint. Out of the total number, 243 patients were discarded from the study as being less than 2 years with bilateral affection due to the normal developmental change in the lower limb axes mentioned above. Those whom were included (324 patients) were those with physiologic genu varum more than 2 years age or those with proved pathologic affection of whatever age. These patients were followed up for a period of time which ranged from 6-18 months (mean 8 months) at regular intervals of 1.5-2 months.

The aim of the follow up was to document the progression of the condition clinically and radiologically so as to confirm the diagnosis and observe the response to treatment. In some cases the diagnosis set at the initial presentation was changed according to subsequent evidences. Out of the number mentioned, 62 patients missed follow up before the definite diagnosis was made; so the final number included was 262. Males constituted 148 (114 females). The age of presentation ranged from 11 months to 6.2 years with the majority of patients presented in the age group of 18-24 months. The condition was bilateral in 231 and unilateral in 31 patients. All the cases of physeal arrest were unilateral. The

distribution of patients according to cause of genu varum is illustrated in table-1.

Table-1: The distribution of cases of genu varum according to the aetiology.

| Diagnosis | Number of patients | Percentage |
|--------------------|--------------------|---------------|
| Physiologic | 139 | 53.1% |
| Metabolic | 101 | 38.5% |
| Skeletal dysplasia | 11 | 4.2% |
| Blount`s disease | 2 | 0.8% |
| Physeal arrest | 9 | 3.4% |
| Total | 262 | 100.0% |

Figure-1 illustrates the sex incidence of physiologic and pathologic genu varum.

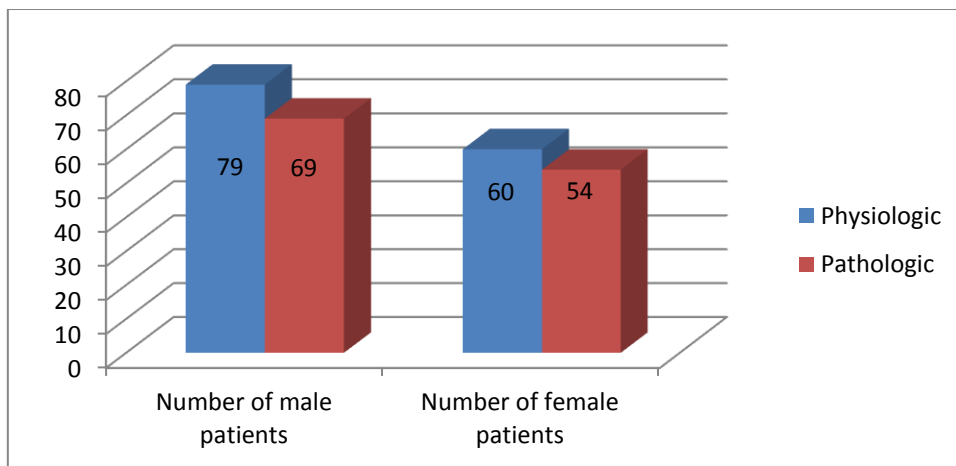


Figure-1 the sex distribution of cases of genu varum in both the physiologic and pathologic categories.

The various causes of metabolic bone disease associated with genu varum are illustrated in table-2.

Table-2: The distribution of cases of pathologic genu varum according to the various causes.

| Diagnosis | Number of patients | Percentage |
|---------------------------|--------------------|------------|
| Nutritional rickets | 88 | 87.13% |
| Hypophosphataemic rickets | 4 | 3.96% |

| | | |
|-----------------------------|-----|--------|
| vitamin-D dependent rickets | 1 | 0.99% |
| Endocrine disorders | 2 | 1.98% |
| Renal osteodystrophy | 2 | 2.97% |
| Steroid-induced | 4 | 2.97% |
| Total | 101 | 100.0% |

As table-2 shows, the commonest cause for metabolic rickets is the nutritional deficiency of vitamin D. The diagnosis was made after XR and biochemical analysis and confirmed by clinical, radiological and biochemical response to vitamin D supplements. It was shown that calcium and phosphate levels reverted to normal after 7 days and XR changes appeared after 2-4 weeks after therapy started. However, correction of lower limb deformity was slow and took a period ranged between 16-26 months. The mode of milk feeding was assessed in patients of nutritional rickets and it was found that out of 88 patients, 27 (30.6%) were kept on regular formula feeding, while 61 (69.4%) were kept on breast feeding with or without infrequent formula feeding. We had 4 cases of hypophosphataemic rickets including 2 cases

of 2 female siblings. These cases were characterized by severe XR changes, irresponsiveness to repeated vitamin D administration, and normal calcium levels with low phosphate levels. In all these cases, high doses of phosphorus were given with appropriate biochemical and radiologic response. The only case of vitamin D dependent rickets belonged to a 5 years old male patient with positive family history and severe genu varum, short stature and alopecia, a phenotype which agrees with this congenital condition transmitted as autosomal recessive. A survey of the nutritional history of patients of nutritional rickets revealed that 63 of them were mostly dependent on breast feeding. The distribution of the mode of feeding in patients of nutritional rickets is illustrated in the chart-1.

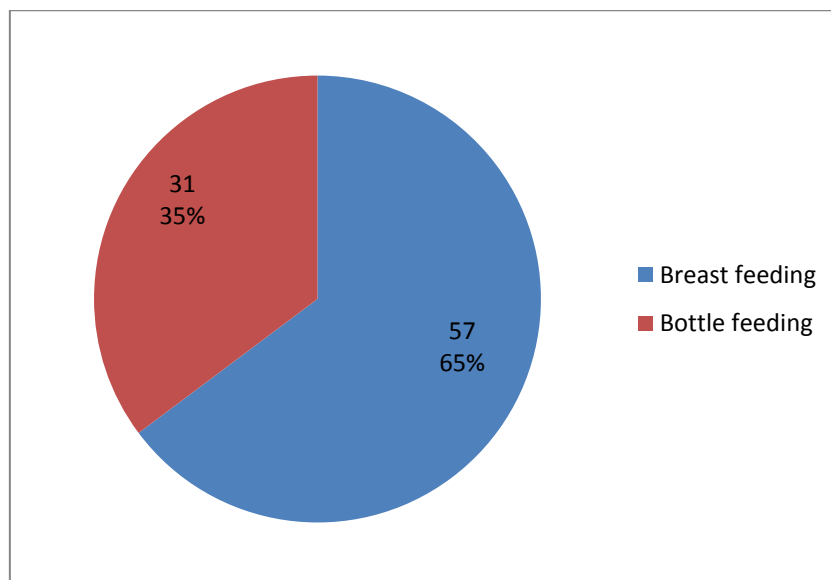


Figure-2: The mode of milk feeding in patients with genu varum due to nutritional rickets.

There were 2 cases of genu varum secondary to endocrinopathy. Both of them were due to congenital hypothyroidism (cretinism) and both were referred by a pediatrician for skeletal evaluation. The 4 cases of steroid-induced metabolic bone disease belonged to 4 cases of asthma treated with steroids for a long term. They all showed osteopenia and osteonecrosis was present in one case. The condition showed improvement on stoppage of steroid and triple therapy (calcium, vitamin D and bisphosphonates).

We had only 2 cases of Blount's disease proved on application of XR criteria. Both cases presented before the age of 2 years with no initial XR changes. Clinically both

patients were obese males with bilateral affection and lateral thrust on walking. A full blown picture appeared after the age of 2.5 years.

History of walking was analyzed and it was found that 61 (43.8%) patients with physiologic genu varum were early walkers (before the age of 12 months). This is in contrast to patients with rickets, where it was found that only 16 (17.2%) were early walkers. Both cases of Blount's disease walked before the age of 12 months.

The distribution of patients according to the severity scale mentioned earlier for patients with physiologic genu varum, nutritional rickets and Blount's disease is illustrated in table-3

Table-3: the distribution of cases genu varum according to the severity

| Diagnosis | Mild Number(%) | Moderate Number(%) | Severe Number(%) | Total |
|--------------------------|-------------------|-----------------------|---------------------|-------|
| Phsiologic genu varum | 17(12.2%) | 65(46.7%) | 57(41.1%) | 139 |
| Nutritional rickets | 11(12.5%) | 37(42.0%) | 40(45.5%) | 88 |
| Blount`s disease | 0(0.0%) | 2(100%) | 0(0.0%) | 2 |

The cases of physiologic rickets showed good prognosis with majority of cases improved over time. The time needed for the tibiofemoral angle to become zero (neutral alignment) ranged from 24-35 months (mean 27 month). In majority of cases, the neutral alignment was achieved

before reaching the age of 4.5 years. Factors which appeared to be related to the prognosis included the severity at initial presentation, the onset of walking, the nutritional status, the body weight and the general activity of the child with the prognosis being good in the hyperactive child.

Discussion

The final number of patients included in the study was 262. This number included physiologic cases with an age equal or more than 2 years as well as proved pathologic cases regardless of the age. The prime goal of the study is to define the real incidence of pathologic cases in our locality and find out the size of the problem of providing unnecessary treatment on a base of faulty diagnosis of a disease status. We found that there was no significant sex difference in the

incidence of genu varum as the overall male to female ratio was 1.3:1 with approximate ratios within the physiologic and pathologic varieties (see figure-1). The very slight male preponderance may be attributed to a cultural interest in the welfare of male children. The age of presentation was found to be more than 11 months as the deformity becomes more evident when the child starts to stand and walk. Most of the patients presented in the age group of 18-24 months

as the natural history of the physiologic and pathologic conditions causing genu varum make them more evident at this age group. The condition was mostly bilateral reflecting the systemic nature of the aetiology in the majority of cases; however, where the pathology was local, the condition was unilateral as in all cases of physal arrest (9 cases). Otherwise, we believe that even in the seemingly unilateral cases, the condition was just discrepant bilateral affection. Table-1 shows that the incidence of physiologic genu varum is 53.1%. This figure implies that the contribution of physiologic genu varum in the overall incidence of this problem is less than that encountered in the developed world (6,7). This can be explained easily by referring to both tables-1 and 2 where we can find that the excess cases of genu varum on the pathologic side can be attributed to the increased incidence of nutritional rickets. This type of metabolic bone affection had become uncommon in the developed world mainly due to formula feeding fortified with vitamin D as well as vitamin D supplements provided for the pregnant females (7,9); in these parts of the world, the commonest variety of rickets seen is the hypophosphataemic type (10). This is substantiated by our finding that 69.3% of patients with nutritional rickets were mainly dependable on breast feeding. The overall incidence of nutritional rickets in this study was found to be 33.5% in

apposition to 53.1% incidence of physiologic variety. So, it is quite clear that it is still in our locality that the commonest type of genu varum is the physiologic in contrast to the over-diagnosis of rickets with the subsequent unnecessary treatment with vitamin D. Another common misbelief is the overdiagnosis of Blount's disease as table-1 shows that we had only 2 cases (0.8%). As was emphasized previously, this condition, once diagnosed, needs active treatment and surgery is usually required to prevent progression. The diagnosis is easily set on the base of clinical examination, strict XR features, and the follow up of the case. The rarity of this condition in our locality may be explained by an underlying racial and genetic predisposition.

The history of early walking was found to be a useful clinical sign despite being not constant. In physiologic genu varum, 43.8% gave such a history in contrast to 17.2% in cases of rickets. This can be a trigger for revision of the diagnosis in assumed rickets cases. Early walking may be also considered as an aetiologic factor in physiologic genu varum as weight bearing can induce bowing of the limb.

The cases of physiologic and nutritional rachitic genu varum showed no difference in the severity of the condition as seen in table-3 which clarifies that in both conditions, the

majority of patients had moderate to severe affection. Accordingly, it is not feasible to use severity as a differential diagnostic measure between these two conditions.

Follow up of cases was useful in establishing the diagnosis as in many cases the clinical and radiologic changes were quite subtle initially. This includes mild cases of rickets which showed at first presentation features similar to physiologic genu varum as the bone density was not markedly affected to make the right diagnosis. The recommendation in this context is to assess fully the clinical picture and to send for biochemical analyses.

ملخص البحث:

ان الجنف الانسي للساق يعتبر من اكثر التشوهات شيوعا في الاطراف السفلى للأطفال. تنقسم العوامل المسببة لهذه الحالة بصورة عامة الى نوعين تتمثل بالسبب الفسلجي والأسباب المرضية علما ان النوع الفسلجي يعتبر اكثر حصولا وعادة لا يحتاج الى اكثر من المتابعة السريرية للمريض. في الحالات التي يكون فيها هذا التشوه مرضيا فان الحالة تكون مرشحة للتطور نحو الأسوء وتحتاج الى تداخل علاجي لمنع حصول مضاعفات تؤثر على سلامة المفاصل والطرف السفلي ككل. ان الهدف من اجراء هذه الدراسة هو تقييم حجم تأثير كل عامل في المعدل الكلي لحصول هذه الحالة المرضية في منطقتنا ومن خلال ذلك يمكن للطبيب الذي يتعامل مع هذه الحالة ان يكون لديه تقدير مسبق لمدى مساهمة كل من هذه العوامل وصولا الى تجنب تقديم العلاج الطبي والجراحي الى حالات لا تستدعي ذلك. لقد توصلنا من خلال الدراسة ان السبب الفسلجي هو الاكثر حصولا تلتها حالات الكساح الناجمة عن لنقص الغذائي لفيتامين د. ان هذه النتيجة تناقض الاعتقاد السائد ان معظم حالات الجنف الانسي للساق تكون مرضية.

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