Global Advanced Research Journal of Medicine and Medical Science (ISSN: 2315-5159) Vol. 3(4) pp. 076-079, April 2014 Available online http://garj.org/garjmms/index.htm Copyright © 2014 Global Advanced Research Journals

# Full Length Research Paper

# Frequency of benign hypermobility syndrome in females with knee pain

Oguntona SA\* and Adelowo OO

Rheumatology Unit, Department of Medicine, Olabisi Onabanjo University, Sagamu, Ogun State, Nigeria.

Accepted 07 April, 2014

Joint hypermobility is an uncommon recognized aetiology for musculoskeletal complains. A prospective study of 124 females was carried out to assess the occurrence and importance of joint hypermobility in adult females presented with knee pain to the rheumatology clinic. Eight (6.5%) had joint hypermobility at three or more locations (greater than or equal to 5 points on a 9 point scale). This result shows that joint hypermobility is common and is significantly associated with knee osteoarthritis.

**Keywords:** Hypermobility, young females, Knee pain, Nigeria.

## INTRODUCTION

Benign joint hypermobility syndrome (BJHS) is a connective tissue disorder with hypermobility in which musculoskeletal symptoms occur in the absence of systemic rheumatologic disease. The primary clinical manifestations of BJHS are hypermobility and pain in multiple joints.

Benign hypermobility is poorly reported among black populace. It is however common condition seen among black Africans. It is sometimes associated with serious joint complaints (Beighton et al., 1973). Kirk first coined the term hypermobility to describe the occurrence of hyperextensibility and hyper-flexibility seen among certain persons (Kirk et al., 1967). The hyperextensibility is however not attributable to hereditary connective tissue disease such as Ehler-Danlos or Marfan syndrome.

There are noticeable racial differences in joint mobility among normal populations. Indians are more mobile than blacks and blacks in turn are more mobile than Caucasians (Finsterbush and Pogrund, 1982; Larsson et al., 1993). Females are generally more mobile than males in the same age group and mobility decreases with advanced age (Seckin et al., 2005).

Benign hypermobility is diagnosed using Beighton hypermobility score with a maximum of 9 and a minimum of 5 scores (Bulbena et al., 1992). Sometimes score less than 5 may also be indicative of benign hypermobility syndrome (BHS). Clinical presentation such as disc herniation, hernia, visceral hernia and joint dislocation are also included as part of the diagnostic criteria (Grahame, 2000).

The treatment of BHS consists of explanation, muscle strengthening exercise, joint protection and symptomatic treatment with medications as needed (Russek, 2000). Bracing is sometimes necessary for the knees.

<sup>\*</sup>Corresponding Author E- mail: oguntonasa@yahoo.com Phone: 08035534449

**Table 1.** Demography and associated joint findings.

Total number of patients	124	Percentage(%)
Age range of patients	20-35 years	
Mean age	26 years	
Numbers with significant Beighton's index	8	6.5
Number with joint effusion	4	3.2
Number with bilateral knee pain	20	16.1

Table 2. Showing level of acute phase reactant (ESR).

ESR	Number of patients	Percentage
Less than 20mm/hr	120	96.8
Greater than 20mm/hr	4	3.2

#### **METHODOLOGY**

## **Patients**

All young females that presented at the rheumatology clinic with complaints of non-traumatic knee pain were examined. Joint movement was assessed clinically. The study was carried out over a five year period (Jan. 2006-Dec. 2010). The Beighton criteria for the diagnosis of hypermobility was used. Persons with a minimum score of 5 was considered hypermobile.

The following joint movements were considered and if more than three of the joint pairs are positive generalized hypermobility is present-

- . Apposition of the thumb to the flexor aspect of the forearm
- . Passive dorsiflexion of the little finger metacarpophallangeal joint to 90<sup>0</sup>
  - . Hyperextension of the elbow beyond 90°.
  - . Hyperextension of the knee beyond 90°.
- . Forward flexion with the hands flat on floor and knee extended

Exclusion criteria included traumatic knee pain, presence of other inflammatory arthritis and the heritable connective tissue disorders. The physical examination was made with particular attention to any of the stigmata of heritable connective tissue disorders, such as high arched palate, ocular and cardiac lesions, hyper-elastic skin or arachnodactyly.

Anti-inflammatory agents were used where necessary.

#### Study site

The study was carried out in a University Teaching Hospital in the South West Nigeria.

#### **RESULTS**

Total of 124 young females were studied. Eight (6.5%) patients fulfilled the Beighton's index for BJHS. Pain was bilateral in 20 patients and 4 patients had associated joint effusion.

The physical appearance of the hypermobile patients was normal. They do not show other features of heritable connective tissue disorders. The acute phase reactant was generally normal except transient elevation in patients with joint effusion. Table 1 above shows Demography and associated joint findings, while table 2 above shows level of acute phase reactant (ESR).

# DISCUSSION

Benign joint hypermobility syndrome (BJHS) is relatively common in the general population, but reports of musculoskeletal complaints are infrequent. Most symptoms are mild and self-limiting and so patients may not seek medical attention. The overall incidence of musculoskeletal complaints due to hypermobility is likely to be much greater than recognized.

The studied patients were diagnosed clinically. Presently, the diagnosis of benign hypermobility syndrome is essentially clinical and it is diagnosed using Beighton hypermobility score with a maximum of 9 and a minimum of 5 scores (Bulbena et al., 1992).

Hypermobility syndrome is used for situations in which joint laxity is associated with musculoskeletal complaints. In this studied patients, the mean joint score in the hypermobile group was 6. It should however be noted that a higher joint score did not always accompany severe complaints or a more widespread pattern of symptoms (Bridges et al., 1992).

Eight patients representing 6.5% were hypermobile in this study. Among studies examining the prevalence of generalized hypermobility in patients referred to Rheumatologists, one study found that hypermobility occurred in 66% of school children with arthralgia of unknown etiology (Seckin et al., 2005). Another study showed a similar prevalence of hypermobility in children; however, there was no association between hypermobility and the development of arthralgia (Gedalia and Brewer, 1993).

Four (3.2%) of the studied patients had joint effusion. Sometimes, patients with BJHS have an effusion from meniscal and cartilage irritation but joint aspirate usually shows a non-inflammatory pattern (Everman and Robin, 1998).

Hypermobile joints should be looked for in young people who present with joint pain, because it is a common unrecognized cause of pain. Different literatures have supported this. Carter and Wilkinson (1964) found excessive motion in at least four joint pairs in 7 percent of 285 English school children (Carter and Wilkinson, 1964). Sutro (1947) shown that three or more joint pairs were hypermobile in 4 percent of 435 adult orthopaedic out patients. Sutro (1947) describe thirteen young adults with pain and effusion in the knees or ankles developing in the course of military training. The family history was negative and the majority of them were unaware of being hypermobile (Sutro, 1947).

Benign joint hypermobility must be differentiated from other causes of joint laxity. Generalized joint laxity is also a feature of the hereditary connective tissue disorders, Marfan's syndrome, Ehlers-Danlos syndrome, and osteogenesis imperfecta (McKusick, 1966). Several rare diseases may show this feature, including the disorder of amino acid metabolism, homocystinuria (Schimke et al., 1965) and hyperlysinaemia (Ghadimi et al., 1965).

Patients with benign hypermobility often lead normal life unlike other causes of joint laxity. The prognosis for patients with BJHS is generally good owing to the syndrome's non-progressive nature and decreased joint laxity and symptoms that occur with age. However, patients need to be aware of the potential sequelae that have an increased frequency associated with BJHS. These sequelae include acute ligament and soft tissue injury, overuse injury, joint instability, possible increase in fractures and scoliosis, and increased frequency of uterine and rectal prolapse. In addition, these patients may be predisposed to osteoarthritis from years of excessive joint motion<sup>18</sup>. Despite these sequelae, patients should remain as active as possible.

Normally, proprioceptive sensory feedback is utilized by the central nervous system for conscious appreciation of the position and alteration of proprioceptive activity. This feedback mechanism is defective in hypermobility syndrome. Hall et al reported that hypermobility syndrome patients have poorer proprioceptive feedback than controls and that the reduced sensory feedback may

lead to biomechanically unsound limb positions being adopted (Hall et al., 1995). Such a mechanism may allow acceleration of degenerative joint conditions, and may account for the increase prevalence of such conditions seen with hypermobility syndrome subjects (Hall et al., 1995).

Lewkonia et al considered that the additional stresses imposed upon a joint by ligamentous laxity predispose sufferers to premature articular degeneration (Lewkonia and Ansell, 1983). These stresses are likely to result from biomechanically disadvantageous loading conditions being adopted, resulting in microtrauma. The proprioceptive deficit is a possible mechanism for such a situation to arise, and could explain the increase incidence of osteoarthritis reported clinically.

The management of these patients commenced with reassurance as to the absence of serious disease. They were encouraged to avoid activities which precipitate symptoms where possible. Joint protection and symptomatic treatment with non-steroidal anti-inflammatory drugs were prescribed where necessary.

The purpose of this paper is to describe a group of patients with joint laxity and musculoskeletal complaints, drawing attention to the hypermobility syndrome as an explanation for the aetiology of their musculoskeletal pain.

# **ACKNOWLEDGEMENT**

The authors wish to acknowledge Dr. S.A. Edunjobi and other staff Nurses who made the data collection possible. Authors acknowledge the great help received from the scholars whose articles are cited and included in references of this manuscript. The authors are also grateful to authors / editors / publishers of all those articles, journals and books from where the literature for this article has been reviewed and discussed.

#### **REFERENCES**

Beighton P, Solomon L, Soskolne CL (1973). Articular mobility in an African population. Ann. Rheum. Dis. 2: 413-418.

Bridges AJ, Smith E, Reid J (1992). Joint hypermobility in adults referred to rheumatology clinics. Ann. Rheum. Dis1. 793-796.

Bulbena A, Duro JC, Porta M, Faus S, Vallescar R, Martin–Santos R (1992). Clinical assessment of hypermobility of joints: assembling criteria. J. Rheumatol. 19:115–122

Carter C, Wilkinson J (1964). Persistent joint laxity and congenital dislocation of the hip. J. Bone Joint Surg. 46: 40-45.

Everman DB, Robin NH (1998). Hypermobility syndrome. Pediatr. Rev. 19: 111–117.

Finsterbush A, Pogrund H (1982). The hypermobility syndrome. Clin. Orthop. Relat. Res. 168:124–127.

Gedalia A, Brewer EJ (1993). Joint hypermobility in pediatric practice—a review. J. Rheumatol. 20: 371–374.

Ghadimi H, Binnington VI, Pecora P (1965). Hyperlysinemia associated with retardation New Engl. J. Med 273: 723-729.

Grahame R (2000). The revised (Brighton 1998) criteria for the diagnosis of benign joint hypermobility syndrome (BJHS). *J. Rheumatol.* 27:1777–1779.

- Grahame R, Pyeritz RE (1995). Marfan syndrome: joint and skin manifestations are prevalent and correlated. Br. J. Rheumatol. 34:126-131.
- Hall MG, Ferrell WR, Sturrock RD, Hamblen DL, Baxendale RH (1995). The effect of hypermobility syndrome on knee joint proprioception. *Br. J. Rheumatol.* 34: 121–125.
- Kirk JA, Ansell BM, Bywaters EG (1967). The hypermobility syndrome. Musculoskeletal complaints associated with generalized joint hypermobility. Ann. Rheum. Dis. 26: 419-425.
- Larsson LG, Baum J, Mudholkar GS, Kollia GD (1993). Benefits and disadvantages of joint hypermobility among musicians. N. Engl. J. Med. 329:1079–1082.
- Lewkonia RM, Ansell BM (1983). Articular hypermobility simulating chronic rheumatic disease. Arch. Dis. Child. 58: 988-992.
- McCormack M, Briggs J, Hakim A, Grahame R (2004). Joint laxity and the benign joint hypermobility syndrome in student and professional ballet dancers. J. Rheumatol. 31:173–178.

- Russek LN (2000). Examination and treatment of a patient with hypermobility syndrome. Phys. Ther. 80: 386–398.
- Schimke RN, McKusick VA, Huang T, Pollack AD (1965). Homocystinuria J. Amer. med. Ass. 193:711-719.
- Seckin U, SonelTur B, Yilmaz O, Yagci I, Bodur H, Arasil T (2005). The prevalence of joint hypermobility among high school students. Rheumatol. Int. 25: 260–263.
- Seckin U, SonelTur B, Yilmaz O, Yagci I, Bodur H, Arasil T (2005). The prevalence of joint hypermobility among high school students. Rheumatol. Int. 25:260–263.
- Sutro CJ (1947). Hypermobility of bones due to "overlengthened" capsular and ligamentous tissues; a cause for recurrent intra-articular effusions. Surgery 21: 67-76.