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## Osteoarthritic Deformities in the Knee Joint

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### Abstract:

**Objective:** To determine the correlation between knee joint deformities and duration of osteoarthritis (OA).

**Methods:** A prospective randomized study was enrolled 100 patients, in Sulaimany City, in Iraqi-Kurdistan region, with radiographic finding of unilateral or bilateral OA of the knee joints; age, gender, obesity, family history, and duration of the OA were taken in consideration. Secondary OA were excluded from this study.

**Results:** out of 100 patients, 21 had deformity (21%), of which 17 patients (17%) had varus, 2 patients (2%) had valgus, and other 2 patients (2%) had fixed flexion deformity.

OA durations were ranging from 1 to 15 years; therefore we categorized the patients according to the durations of OA into 3 groups (1-5, 6-10, and 11-15). From 1 to 5 years there were 72 patients, of these 9 patients had deformity (12.5%), 63 patients with out deformity. From 6 to 10 years there were 17 patients, of these 5 patients had deformity (29.41%), 12 patients with out deformity. From 11 to 15 years there were 11 patients, of these 7 patients had deformity (63.64%), 4 patients with out deformity.

**Conclusion:** There is significant positive correlation between deformities and duration of the knee OA.

**Key words:** Osteoarthritis, knee, and deformities.

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### Introduction:

#### Osteoarthritis:

(O A, osteoarthrosis) is by far the most common joint disorder in the United States and throughout the world, and is one of the leading causes of disability and pain in the elderly [1]. Pathologically, it may be defined as a condition of synovial joints characterized by focal loss of articular cartilage with proliferation of new bone and remodeling of joint contour. Inflammation is not a prominent feature. This non-inflammatory condition arises from degenerative changes and progressive loss of cartilage with resultant hypertrophic changes in surrounding bone [2] OA was previously thought to be a normal consequence of aging, thereby leading to the term degenerative joint disease. However, it is now realized that osteoarthritis results from a complex interplay of multiple factors, including joint integrity, genetic predisposition, local inflammation, mechanical forces, and cellular and biochemical processes [3]. OA has traditionally been subdivided by etiology into idiopathic and secondary forms:

#### Idiopathic OA:

Idiopathic OA can be categorized into localized and generalized forms of the disease. Localized OA most commonly affects the hands (Heberden's and Bouchard's nodes in the distal and proximal interphalangeal joints respectively). Feet, knee, hip, and spine are also affected. Other joints are less commonly involved, and include the shoulder, temporomandibular, sacroiliac, ankle, and wrist joints. Generalized OA consists of involvement of three or more joints [3].

#### Secondary OA:

Specific conditions may cause or enhance the risk of developing osteoarthritis. These include: Trauma, Congenital or developmental disorders, inflammatory conditions like Calcium pyrophosphate dihydrate deposition disease (CPPD) rheumatoid arthritis, gouty arthritis, and septic arthritis. Other metabolic and endocrine disorders like osteonecrosis, Paget's disease of bone, diabetes mellitus, acromegaly, hypothyroidism, neuropathic (Charcot) arthropathy [4].

#### Epidemiology and risk factors:

Although the disease commonly affects the cervical and lumbar spine, most epidemiologic studies report that it has a predilection for weight-bearing joints in the leg and certain joints in the hand. Multiple risk factors have been linked to osteoarthritis in epidemiological studies including:

#### Age:

The prevalence of OA in all joints correlates strikingly with age. One-third of people aged 65 years and older have knee OA that is evident by radiograph [5].

#### Female gender:

In those less than 55 years, the distribution in men and women is similar; in older individuals, OA of interphalangeal joints and the thumb base is more common in women. Similarly, radiographic evidence of knee OA, and especially of symptomatic knee OA, is more common in women than in men [6]. Innumerable studies have found that female sex is associated with an increased risk of osteoarthritis [7, 8]. The relative risk of developing osteoarthritis for

women has been estimated to be 2.6 after adjustment for age, and weight [8].

#### **Obesity:**

Population-based studies of OA consistently have shown that over weight people are at greater risk of developing knee OA than average-weight controls. Obese women are four to five times more likely to have knee OA than persons of average weight. Weight reduction is likely to lessen the symptoms of knee OA [9].

#### **Injury and occupations:**

Major acute knee injuries, including cruciate ligament and meniscal tears, are common causes of knee OA. Osteoarthritic changes have been reported in up to 89 % of people after meniscectomy [10].

#### **Developmental deformities:**

Anatomic abnormalities of the knee and the hip that are present at birth or that develop during childhood may result in accelerated or premature OA. These abnormalities include genu varum, genu valgum, unequal leg length, hypermobility, developmental dislocation of the hip (DDH), slipped capital femoral epiphysis, Legg-Calve-Perthes disease, and acetabular dysplasia [4].

#### **Amputation:**

Amputation of one lower extremity usually increases the amount of weight borne on the unaffected limb, even when prosthesis is used. This may account for the increased prevalence of osteoarthritis of the contralateral knee, which has been observed in unilateral amputees [11, 12].

#### **Sex hormones:**

OA occurs more frequently in women over the age of 50 years than in age-matched men. Epidemiologic studies of women who take estrogen replacement therapy report that these women are less likely to develop OA than women not taking estrogen [13].

#### **Genetic susceptibility:**

Many studies have demonstrated that genetic factors influence the incidence of OA. Heritability of primary OA of the hands has been reported to be as high as 65 % (14). In fact, twin and familial studies have shown OA to be a multigenic trait, with several genes involved [15].

#### **Racial differences:**

There is conflicting evidence as to whether African-Americans have different rates of OA than Caucasians. The incidence of knee OA could be higher in African-American women [16].

#### **Bone mineral density:**

Women with moderate to severe evidence of osteoarthritis had higher bone mineral density measurements than those without osteoarthritis. This inverse association between osteoporosis and osteoarthritis was supported by large cross-sectional studies which controlled for the effects of estrogen replacement therapy [17].

#### **Other systemic factors:**

Low vitamin D and vitamin C intakes are associated with increased risk of knee OA progression [4].

#### **Patients & Methods:**

The study enrolled 100 patients with OA of one or both knee joints. The patients were collected from July 2006 to January 2007, in Sulaimany Governorate in Iraqi-Kurdistan region, at Rheumatology and Medical Rehabilitation center. The age of the patients were ranged from 40 to 82 years. The patients' diagnoses were based on the revised criteria of the American College of Rheumatology (ACR) for classification of idiopathic OA of the knee [18]. We take history and physical examination, with a weight-bearing (standing) antero-posterior and lateral radiography of both knee joints for all of the patients.

Secondary OA due to developmental deformities and trauma were not included.

Inflammatory conditions were excluded by doing laboratory investigations [Erythrocyte Sedimentation Rate (ESR), and C-reactive protein (CRP)] were done for all the patients.

Goniometer was used for detection of deformities, by measurement of angle between femur and tibia for detection of varus or valgus deformities, and measuring the degree of extension of the knee for detection of flexion deformities.

Body Mass Index (BMI) was used for assessment of weight of the patients.

#### **Statistical analysis:**

Statistical analysis were performed for correlation between the results, by using the Chi-square test, t-tests, and the results were analyzed, and assessed by mean value, with significant p – value (< 0.05), and mean standard deviation value depending on the protocol data on excel sheet analysis.

#### **Results:**

##### **Age and sex distributions:**

Among 100 patients of knee OA, 71 patients (71%) were female, 29 patients (29%) were male (Table 1), and female: male ratio was 2.45: 1. The age of the patients range from 40 to 82 years [mean age 62.42, mean standard deviation (MSD) = ± 9.88].

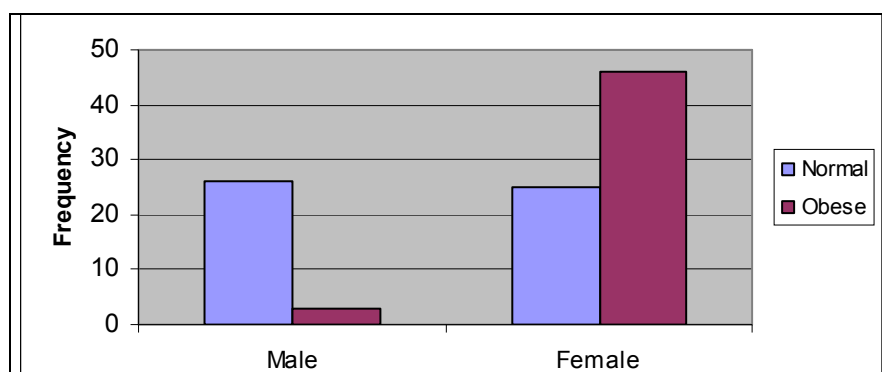
**Table 1. Age and sex distributions.**

Age	Male	Female	Total
40 - 49	0	9	9
50 - 59	5	24	29
60 - 69	10	19	29
70 - 79	11	18	29
80 - 89	3	1	4
<b>Total:</b>	<b>29</b>	<b>71</b>	<b>100</b>

**Weight distributions according to BMI:** 49 patients were obese with high BMI > 30 kg/m<sup>2</sup> of which 46 64.78 % in the females and 10.3 % in the males.

patients were females; meanwhile 3 were males (Figure1). Rate of obesity was (10%) had deformity (9 varus and 1 fixed flexion deformity). P-value=0.011 (Table 2).

**Family history of OA:** 26 patients had family history of OA in the first degree relatives, 10 of them



**Figure 1. Obesity and sex distributions**

**Table 2. Family history and deformities.**

Deformities	Family history		Total
	positive	negative	
present	10	11	21
absent	16	63	79
<b>Total</b>	<b>26</b>	<b>74</b>	<b>100</b>

P-value = 0.011

**Distribution of deformities:**

21 patients (21%) were presented with deformity; 17 patients had varus deformity (10 females, 7 males), 2 patients (females) had valgus deformity, and 2 patients (males) had fixed flexion deformity (Figure 2).

**Site of OA, crepitus, and effusion:**

61 patients (61%) had bilateral OA, 18 patients (18%) had right side OA, 21 patients (21%) had left side OA. 87 patients (87%) had crepitus. 9 patients (9%) had effusion.

**Disease duration and Deformities:**

(Table 3) OA durations were ranging from 1 to 15 years; therefore we categorized the patients according to durations of OA into 3 groups (1-5, 6-10, and 11-15).

From 1 to 5 years there were 72 cases, of these 9 cases had deformity (12.5%), 63 cases with out deformity. From 6 to 10 years there were 17 cases, of these 5 had deformity (29.41%), 12 with out deformity. From 11 to 15 years there were 11 cases, of these 7 had deformity (63.64%), 4 with out deformity (Figure3). P-Value=0.00035.

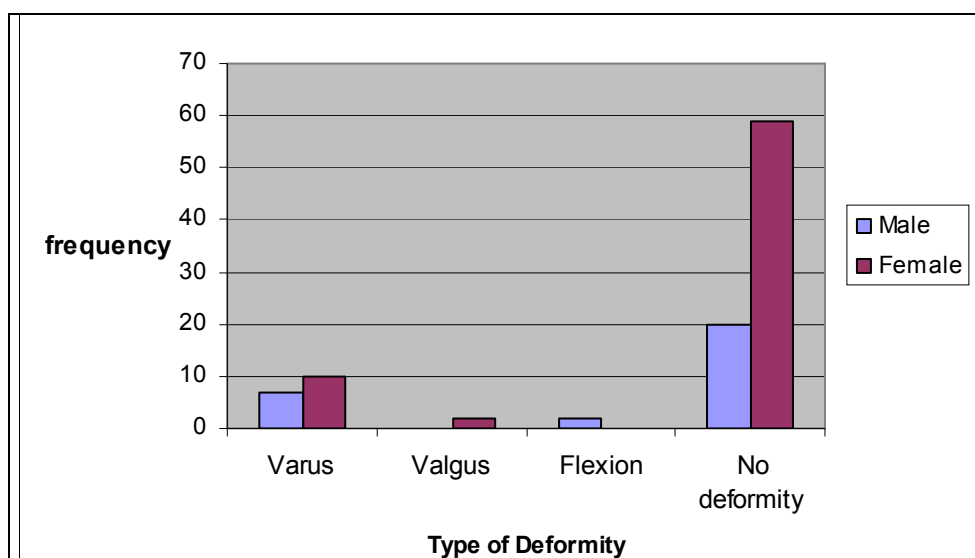


Figure 2. Deformities and sex distributions

Table 3. Disease Duration and Deformities.

deformities	Duration (years)			
	1-5	6-10	11-15	Total
present	9	5	7	21
absent	63	12	4	79
<b>Total</b>	72	17	11	100

P value=0.00035

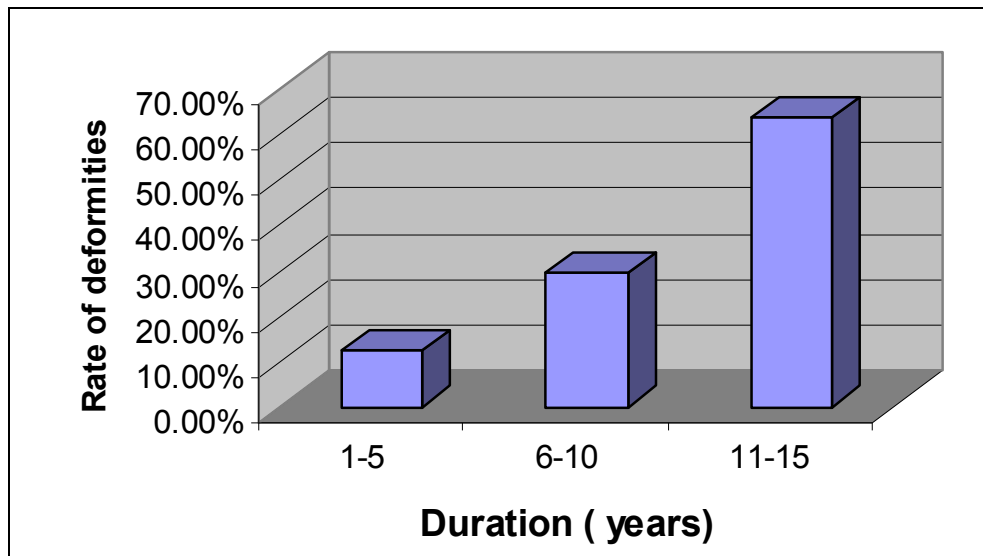


Figure 3. Rate of Deformities and duration of O.A

#### Discussion:

OA is the most common musculoskeletal disorder affecting the synovial joints. The disease process is characterized by degenerative structural remodeling of joint cartilage and of the underlying subchondral bone, which often results in significant pain and disability. OA is by far the most common joint disorder in the United States and throughout the world, and is one of the leading causes of disability and pain in the elderly<sup>[1]</sup>.

Advanced age is one of the strongest risk factors associated with OA. Prevalence of OA increases with age in both sexes<sup>[5]</sup>. In our patients mean age was 62.45 and 62% of the patients were above 60 years.

The knee joint in women is particularly susceptible to this insidious disease. The relative risk of developing OA for women has been estimated to be 2.6 after adjustment for age, and weight<sup>[8]</sup>. 71 of our patients (71%) were females, 29 patients (29%) were males, and female: male ratio was 2.45:1. Among 100 OA patients, 49 cases (49%) were obese, 46 of them were females, and 3 cases were males, which mean a close relation between body weight and OA, this strong relation is also mentioned by authors<sup>[7, 8, 9]</sup>, whom considered obesity to be a strongest modifiable risk factor for knee OA which is more common among females. Obese women are four to five times more likely to have knee OA than persons of average weight<sup>[9]</sup>. We found that the rate of obesity in the females was 64.78 %, while rate of obesity in the males was 10.3%.

Both the tibiofemoral and patellofemoral compartments of the knee appeared to be influenced by genetic factors<sup>[14]</sup>. In fact, twin and familial studies have shown a multigenic trait, with several

genes involved<sup>[15]</sup>. We found that 26 (26%) of our patients had positive family history of OA in first degree relatives.

Granlink JM. and coworkers et al.<sup>[19]</sup> claim that, medial compartment narrowing, which leads to varus deformity (bow-legged), due to loss of articular cartilage in medial compartment, by weight bearing effect, occurs more frequently than lateral compartment narrowing, which leads to valgus deformity (knock-kneed), and these correlate with progression and duration of OA<sup>[19]</sup>. Varus-valgus laxity may precede the development of knee OA and become worse with progression of the disease<sup>[20]</sup>. On physical exam, more than 80% of women had patello-femoral crepitus on flexion and extension of their knees, the most common physical finding in patients with knee OA; and 17% had either a varus or valgus deformity of the knee<sup>[20]</sup>. These findings are in agreement with our study, in which 87 patients (87%) had crepitus, 21 patients were presented with deformities, 17 patients (17%) had varus deformity, 2 patients (2%) had valgus deformity, and other 2 patients (2%) had fixed flexion deformity. Rate of deformities was 12.5% within 1-5 years, 29.41% within 6-10 years, and 63.64% within 11-15 years (P-value = 0.00035).

From the above results we concluded that there is highly significant positive correlation between deformities and duration of the knee OA.

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