The Effect of Body Weight & Smoking on the Risk of Osteoarthritis of the Knee Joint

Zina H. Abdul-Qahar; FICMS Chemical Pathology *, Ikhlas K. Hameed; FICMS Chemical Pathology **, Zina A. Rajab; M.Sc. Histocytopathplogy ***.

*Lecturer. Department of Biochemistry, College of Medicine/ University of Baghdad.
**Lecturer. Department of Biochemistry, Al-Kindi Medical College/ University of   Baghdad.
***Department of Cytology, Teaching Laboratories, Medical City-Baghdad.

Abstract

Background: Knee osteoarthritis is common in old adults. Determination of risk factors of knee osteoarthritis may help in its prevention and modification of treatment. The objective of this study is to determine the significance of some risk factors like obesity and smoking on knee osteoarthritis.

Material & methods: The study included 132 outpatient attendants (102 females & 30 males) aged (53±9) years. Height and weight were measured for calculation of Body Mass Index; smoking habits were registered by questionnaire. Hemoglobin level, Erythrocyte Sedimentation Rate & C - reactive protein was measured for all participants.

Results: The number of patients with knee osteoarthritis was 61(46%), of these; 39 patients (64%) were obese including 13 patients who were smokers.

The mean & standard deviation of Hemoglobin & Erythrocyte Sedimentation Rate were 12±1 & 24±16 respectively, while C - reactive protein was positive in 29 (22%) of participants.

Smoking habit was significantly higher in patients with knee osteoarthritis (p<0.01) and in knee osteoarthritis obese patients (p<0.01) when compared to non- knee osteoarthritis subjects and obese non- knee osteoarthritis subjects; respectively.

Conclusion In accordance with the literature, this study found a strong association between Body Mass Index and knee osteoarthritis risk. In addition, the increase in the prevalence of smoking in radiographically confirmed knee osteoarthritis was observed suggesting that smoking habit may be a potentially modifiable environmental risk factor for knee osteoarthritis.

Keywords: Knee osteoarthritis, smoking, obesity, Body Mass Index.

الملخص

الخلفية و الهدف من الدراسة: مرض الفصال العظمي هو مرض شائع لدى كبار السن لذلك دراسة عوامل الخطورة قد يساعد على الوقاية منه و تحسين طرق علاجه. الهدف من الدراسة هو بيان تأثير بعض عوامل الخطورة مثل السمنة و التدخين و علاقتها بمرض الفصال العظمي لمفصل الركبة.

المواد والطرق: تضمنت الدراسة 132 شخص (102 اناث و 30 ذكور) متوسط اعمارهم (53±9) سنة .تم قياس الطول و الوزن لحساب معامل كتلة الجسم اضافة لسؤال المشاركين عن عادات التدخين لديهم. لجميع المشاركين تم قياس نسبة الهيموكلوبين و سرعة ترسب الكريات الحمراء و البروتين التفاعلي-C.

النتائج: عدد المرضى المصابين بالسمنة كان 92 (69٫7 %) حيث ان معامل كتلة الجسم لديهم (33±5) كغم\م²، وكان(42٫4%) منهم يعانون من مرض الفصال العظمي لمفصل الركبة من ضمنهم(13) مريضاً كانوا من المدخنين. عدد المدخنين كان اكثر في مجموعة المرضى الكلي مقارنة بالأصحاء (p<0.01) و بقيت هذه العلاقة قوية لدى المرضى الذين يعانون من السمنة (p<0.01).

الأستنتاج: وجدت الدراسة علاقة قوية بين السمنة و الأصابة بمرض الفصال العظمي لمفصل الركبة. بالاضافة الى إن زيادة إنتشار عادة التدخين لدى مرضى الفصال العظمي لمفصل الركبة المشخص شعاعياً يدل على ان التدخين قد يكون من عوامل الخطورة الممكن تلافيها أو تعديلها للوقاية من مرض الفصال العظمي لمفصل الركبة.

Introduction

Osteoarthritis (OA) is defined as a gradual loss of articular cartilage, combined with thickening of the subchondral bone, bony outgrowths (osteophytes) at the joint margins, and mild, chronic nonspecific synovial inflammation [1]. The central pathological feature of Knee Osreoarthritis (KOA) was considered to be merely due to cartilage destruction but this concept has evolved, and today OA is generally regarded as a disease that may affect the whole joint (bone, muscle, ligaments& synovium) [2].

Obesity and osteoarthritis are two commonly encountered problems that can lead to a significant physical and emotional disability that is why improved understanding of its contribution to pathogenesis of OA will hopefully lead to improved treatment and subsequent amelioration of this important risk factor for OA [3, 4].

Obesity is a state of excess adipose tissue mass, and its pathophysiology seems simple; a chronic excess of nutrient intake relative to level of energy expenditure [5].

The current working hypothesis is that adipokines, cytokines & other factors produced and released by White Adipose Tissue (WAT) in obese subjects are responsible for a chronic subclinical pro-inflammatory state [6].

Circulating leptin levels appear to be one of the best biological markers of obesity& hyperleptinemia is closely associated with several risk factors related to obesity syndrome [7]. Regarding KOA; its levels are elevated in synovial fluid of patients and correlate with their Body Mass Index (BMI). Leptin and its receptor are also expressed in osteoarthritic chondrocytes [8].

The recent studies on KOA have focused more on evaluation of biochemical markers in serum and/or synovial fluid of knee joint; such as, leptin, MMPs, TIMPs, toxic oxygen radicals, and others [9-11].

Smoking is accepted as one of the major risk factors for many conditions such as cancer, diabetes and cardiovascular diseases [12] In contrast; smoking is reported to have a negative association with risk of developing certain conditions such as ulcerative colitis [13], Alzheimer's disease [14], and Parkinson's disease [15].

Smoking is associated with the elevated risk of back pain [16] and rheumatoid arthritis [17]. Controversially, smoking has been reported to have a protective association with osteoarthritis (OA) [18, 19]. It is unclear what mechanisms account for this phenomenon or, indeed, if any exist [19]. In contrast, some studies report that smoking may be associated with a greater risk both of cartilage loss and knee pain in OA [20, 21]. However, the effect of smoking on the pathogenesis and progression of symptomatic knee osteoarthritis, one of the leading causes of disability in elderly people, has been unclear. [20]

Objective

The objective of this study is to determine the significance of some risk factors like obesity and smoking on KOA in Iraqi individuals.

Material and Methods

This study included one hundred and thirty two subjects (102 females& 30 males) whom age ranged from (40-70) years old; all were examined in the outpatient clinic in Medical City, Baghdad Teaching Hospital (Rheumatology& Rehabilitation Consultation Department) during the period from June 2007 till December of the same year.

All attendants suffered from knee joint pain were examined and KOA cases were

diagnosed clinically and radiologically according to the Kellgren&Lawrence Score System [22]. Cases diagnosed as rheumatoid arthritis, infection or traumas were excluded from the study.

Height and weight were measured for calculation of Body Mass Index (BMI) using the formula BMI equals weight/Height^2 in (kilogram/meter^2) [5].

Smoking habits were registered with questioning, smokers had an average of 40-50 cigarettes/day. Blood was drown for the assessment of Hemoglobin level (Hb), Erythrocyte Sedimentation Rate (ESR) & C - reactive protein (CRP) for all participants.

All data were tabulated using SPSS for descriptive analysis showing correlations through cross tabulation and chi-square, $p<0.05$ was considered statistically significant.

Results

Mean and standard deviation of the age of study subjects were (53 ± 9) years while of Hb and ESR were (12 ± 1) mg/dl & (24 ± 16) mm/hour respectively. CRP was positive in 29 (22%) of subjects; 26 (20%) of them were diagnosed as KOA cases.

The distribution of all study subjects with relation to their KOA, Obesity and smoking habits is shown in table (1).

Table 1. Distribution of Study Subjects

KOA	Obese		Non-Obese		Total
	Smoker	Non-smoker	Smoker	Non-smoker	
With KOA	13	26	4	18	61
Without KOA	5	48	2	16	71
Total	18	74	6	34	132

KOA cases were 61 (46.2%), thirty nine of them (64%) were obese with their BMI (33 ± 5) kg/m^2. (Figure 1).
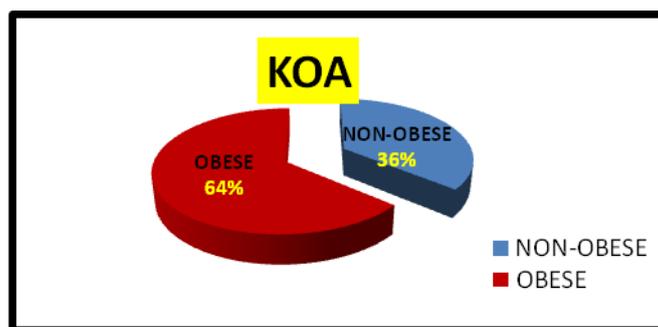

Figure 1. Percentage of Obese & Non-obese KOA Cases

Smoking habit was significantly higher in patients with KOA ($p<0.01$) and in KOA obese patients ($p<0.01$) when compared to non-KOA subjects and obese non-KOA subjects; respectively. CRP and ESR were significantly higher in KOA patients, in both p value was <0.001. Table (2).

Figure (2) shows distribution of smokers among total participants whether diagnosed as KOA cases or not, it clarifies that the number of smokers is higher among KOA patients compared to those not diagnosed as having KOA.

Table 2. Distribution of statistical data (Smoking, crp & esr) among study groups

characteristics	KOA	Non-KOA	p value
Mean±SD of age(year)	54±8	52±9	Not Significant
No. and % of smokers	17 - 27.9%	7 – 9.8%	<0.01
No. and % of obese smokers	13 – 33%	5 – 9.4%	<0.01
No. and % of positive CRP	26 – 22%	3 – 2%	<0.001
ESR (mm/hr)	30±16	18±13	<0.001
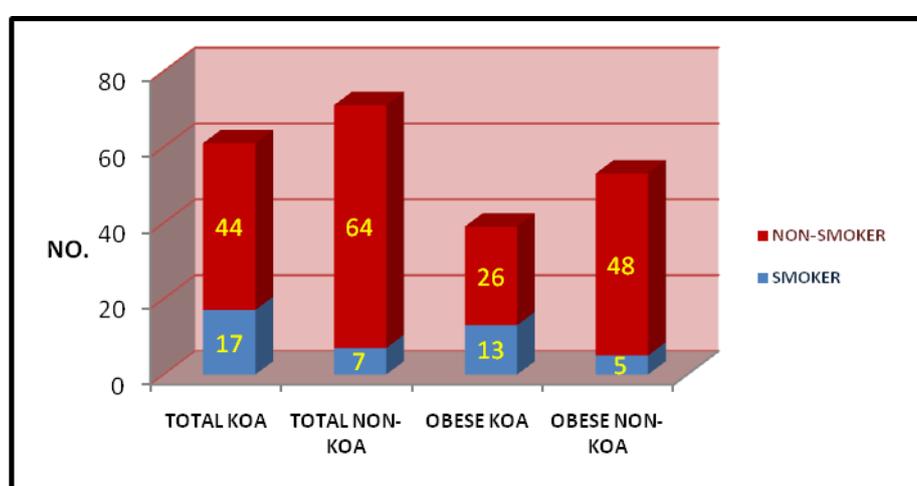
Figure 2. Number of Smokers among study subjects

Discussion

In this study most of patients with KOA were obese (64%), the explanation of this finding may be that stress and amount of force on the weight bearing joints are increased in overweight subjects and this additional physical load could cause cartilage breakdown leading to osteoarthritis. *Felson DT* has found that overweight persons have a higher bone density, which could be a risk factor. [23]

The increased risk of osteoarthritis associated with overweight and obesity was suggested by previous studies. [2, 4, 8, 9, 24, 25]

The structural changes of osteoarthritis are usually age-related and have long been regarded as irreversible. New MRI techniques has enabled us understand the natural history of osteoarthritis and the effects of noval risk factors such as smoking, lipids, leptin, vitamin D deficiency, and inflammation that have been associated with early knee structural changes [8, 10, 26].

There are suggestions of a contribution of environmental factors to KOA [27-29], smoking has been reported to have a protective association with osteoarthritis (OA) by *Anderson and colleagues* [18, 19] and the report from the Framingham study was one of the first to generate this hypothesis [30]. The protective effect of smoking in OA observed in some epidemiological studies is likely to be false. It may be caused by selection bias, often in a hospital setting where control subjects have smoking-related conditions and studies that are not primarily designed

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to investigate smoking. Critical appraisal of such studies is needed [19, 28, 31].

In this study, there was higher incidence of smoking habit in KOA cases compared to normal subjects. In spite of adjustment of the results for age, body mass index, it was found that the smokers were at increased risk of KOA than those who did not smoke; this is agreed by previous studies [20, 21, 32].

These findings raise concern about the negative effects of smoking on chondrocyte function in articular cartilage. In addition, the deleterious effect of smoking may be greatest when cartilage is already damaged. Other theories to explain the cartilage damage, according to the investigators, increased carbon monoxide levels in arterial blood, adding to tissue hypoxia, which could impair cartilage repair and increased oxidant stress, which contributes to cartilage loss [20, 21, 27, 29].

Nitric oxide (NO) is an important mediator of oxidative stress. Chondrocytes have long been known to express inducible nitric oxide synthase (NOS), and now a greater role for NO in the pathogenesis of OA has been recognized. Increased production of NO and associated molecules has been noted in OA joints and specifically in chondrocytes [32, 33].

Our study has limitations; first is the small sample size, second is the inability to determine the risk of smoking on cartilage loss in KOA patients therefore our ability to deal with all possible confounders in analyses was limited, especially given the number of potential mechanisms by which cigarette smoke may have an adverse effect on articular cartilage.

The finding that cigarette smoking plays a role in the progression of knee osteoarthritis is important, because it is a potentially modifiable risk factor. Few studies have examined the association between smoking and symptomatic knee osteoarthritis [20]. To the best of our knowledge, this the first study in Iraq assessing the effect of smoking as a risk factor to KOA.

Conclusion

In accordance with the literature, this study found a strong association between BMI and knee osteoarthritis risk. In addition, the increase in the prevalence of smoking in radiographically confirmed knee OA was observed suggesting that smoking habit may be a potentially modifiable environmental risk factor for KOA.

Recommendations

Despite the study limitations, its findings are provocative and suggest the need for further study about the metabolic effects of smoking on knee osteoarthritis with larger sample size enabling assessment of gender based difference regarding smoking habit and KOA incidence.

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