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Original Research Article

PREVALENCE OF KNEE OSTEOARTHRITIS AMONG EGYPTIAN DIABETIC TYPE 2 PATIENTS

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ABSTRACT

Osteoarthritis is characterized by cartilage degeneration or degradation of one or more joints. It is a common condition to affect synovial joints, the single most important cause of locomotor disability and a major challenge to health care. Previous studies have suggested that diabetic patients are at higher risk of developing rheumatic disorders. Some have reported a correlation of osteoarthritis with longer diabetes mellitus duration and poor glycemic control. Furthermore, the presence of peripheral neuropathy in diabetes mellitus patients may increase the risk of aggressive forms of osteoarthritis. Some studies found that type 2 diabetes increases incidence of knee osteoarthritis and predicts the development of severe osteoarthritis independent of age and Body Mass Index. This paper gives data about prevalence and severity of osteoarthritis in diabetic type 2 patients.

KEY WORDS: Diabetes Mellitus, osteoarthritis, prevalence, peripheral neuropathy, poor glycemic control, longer diabetes mellitus duration.

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INTRODUCTION

Osteoarthritis (OA) is a heterogeneous disorder for which the exact cause is unknown, the condition is thought to consist of a group of overlapping distinct diseases which may occur in response to a variety of different biological and mechanical factors including metabolic, genetic or hereditary predisposition, age, physical factors such as obesity, and environmental factors [1,2].

Osteoarthritis is a slowly evolving articular disease characterized by the gradual development of joint pain, stiffness and limitation of motion. The term of degenerative joint disease or osteoarthrosis may be more precise because degeneration of cartilage is the most prominent pathologic change [3].

Pain is the predominant symptom of knee OA with the pain being generally related to joint use and with relief at rest. As OA progresses, pain may become more persistent and can appear also at rest and during the night [4].

The mechanism of pain production in OA is not clear. The disease process may affect all intra capsular and periarticular tissues of the synovial joint loading to many possible sources of pain. The articular cartilage is aneural and a vascular tissue. However, it has a rich sensory innervations exists in other joint tissues [5]. It

has been suggested that several processes in bone or/and subchondral bone such as elevated intraosseus pressure, bone marrow oedema, structural changes, and periosteal stretching may associate with the joint pain [6]. On other hand, the capsular mechanoreceptors may be stimulated by intra-articular hypertension, and the ischemia caused by mild synovitis may activate nociceptors [6].

The term Diabetes Mellitus (DM), describes a metabolic disorder of multiple aetiologies characterized by chronic hyperglycaemia with disturbance of carbohydrate, fat, and protein metabolism resulting from defects in insulin secretion, insulin action, or both [7]. It has been estimated that by the year 2030, there will be 8.6 million adults with diabetes in Egypt, making it the country with the tenth largest population of diabetics in the world [8].

The effects of diabetes include long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, heart, and blood vessels [9,10]. As modern therapeutics have helped to decrease the mortality and morbidity of DM, increased musculoskeletal symptoms may be discovered as these patients lead longer and more active lives. It is important to recognize the various joint, soft tissue and bone manifestations of diabetes which may be very debilitating. Many of these rheumatologic manifestations of DM have been reviewed in recent years [11-13]. In general, these are syndromes commonly or uniquely associated with DM [11-13], including adhesive capsulitis, shoulder hand syndrome, limited joint mobility, Dupuytren's Disease, neuroarthropathy, and hyperostosis [13]. Arthropathies associated with DM includes osteoarthritis, gout, calcium pyrophosphate deposition arthropathy and migratory osteolysis of hip and knee [13].

It has been suggested that type 2 diabetes is important risk factor for development of OA [14]. Diabetes affects cartilage metabolism and osteophyte formation of knee joint [14].

As DM is one of highly prevalent diseases in Egypt [8], the study of the prevalence of knee OA among diabetic patients and the determination of the risk imposed by diabetes to develop knee OA will be useful for better understanding of knee OA among Egyptian population.

MATERIALS AND METHODS

The study included: One hundred type 2 diabetic patients selected from Diabetes Clinic and Physical Medicine, Rheumatology and Rehabilitation Clinic of The Main University Hospital. In addition one hundred age and sex matched non-diabetic subjects constituted the control group.All participants did not inquired initially about knee pain or having knee OA. Participants who have renal disease were excluded. The research topic was explained to the participating individuals, each participant signed an informed consent.

The following data were recorded for each case: History and demographic data, clinical data: Complete clinical examination of the knee joint was done. The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) [15,16] was included for evaluation of patient activities and participation. Radiological: Radiographic abnormality of the knee joint was assessed using plain X-Ray of both knees antero-posterior and lateral views while standing and the disease severity was assessed by using the Kellgren and Lawrence global scale [17]. Laboratory methods including: Hb A1C levels were measured in diabetic patients.

RESULT

Table 1: Frequency of knee OA among the studied groups.

	Diabetic		Non diabetic		Total	
	No.	%	No.	%	No.	%
With OA	76	76	53	53	129	64.5
Without OA	24	24	47	47	71	35.5
χ^2(p)	11.55*(0.001*)					
Total	100	100	100	100	200	100

χ^2: Chi square test
*: Statistically significant at p d" 0.05

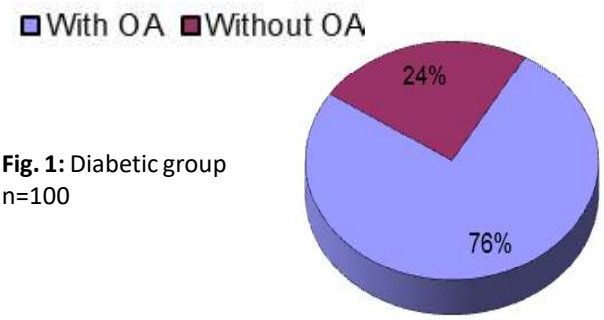

Fig. 1: Diabetic group n=100

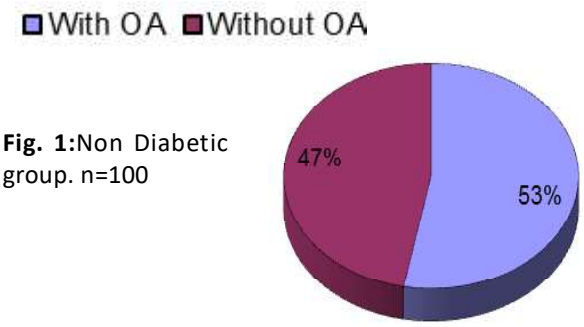

Fig. 1: Non Diabetic group. n=100

Table 2: Tenderness of knee joint among the studied groups (Ritchie Articular Index).

	Diabetic OA (n = 76)		Non Diabetic OA (n = 53)		Total (n = 129)	
	No.	%	No.	%	No.	%
Tenderness						
0	1	1.3	12	22.6	13	10.1
1	15	19.7	20	37.7	35	27.1
2	25	32.9	11	20.7	36	27.9
3	35	46.1	10	14	45	34.9
χ^2 (^{MC}p)	26.084* (<0.001*)					
Total	76	100	53	100	129	100

χ^2: Chi square test
MC: Monte Carlo test
*: Statistically significant at p ≤ 0.05

Table 3: Kellgren-Lawrence grading of knee OA among the studied groups.

	Diabetic OA (n = 76)		Non Diabetic OA (n = 53)		Total (n = 129)	
	No.	%	No.	%	No.	%
Kellgren-Lawrence grading of knee OA						
Grade 1	7	9.2	15	28.3	22	17.1
Grade 2	30	39.5	34	64.2	64	49.6
Grade 3	25	32.9	3	5.7	28	21.7
Grade 4	14	18.4	1	1.9	15	11.6
χ^2 (^{MC}p)	28.517* (0.001*)					
Total	**76**	**100**	**53**	**100**	**129**	**100**

χ^2: Chi square test
MC: Monte Carlo test
*: Statistically significant at p ≤ 0.05

Table 4: Pain scoring (by WOMAC osteoarthritis index) among the studied groups of knee OA.

	Diabetic OA (n = 76)		Non Diabetic OA (n = 53)		Total (n = 129)	
	No.	%	No.	%	No.	%
Pain						
Mild	4	5.3	26	49.1	30	23.3
Moderate	35	46.1	27	50.9	62	48.1
Severe	37	48.7	0	0	37	28.7
χ^2 (^{MC}p)	51.709* (0.001*)					
Total	**76**	**100**	**53**	**100**	**129**	**100**

χ^2: Chi square test
MC: Monte Carlo test
*: Statistically significant at p ≤ 0.05

Table 5: Knee stiffness (by WOMAC osteoarthritis index) among the studied groups of knee OA.

	Diabetic OA (n = 76)		Non Diabetic OA (n = 53)		Total (n = 129)	
	No.	%	No.	%	No.	%
Stiffness						
None	0	0	2	3.8	2	1.6
Mild	5	6.6	21	39.6	26	20.2
Moderate	45	59.2	30	56.6	75	58.1
Severe	26	34.2	0	0	26	20.2
χ^2 (^{MC}p)	37.952* (0.001*)					
Total	**76**	**100**	**53**	**100**	**129**	**100**

χ^2: Chi square test
MC: Monte Carlo test
*: Statistically significant at p ≤ 0.05

Table 6: Difficulty performing daily activities (by WOMAC osteoarthritis index) among the studied groups of knee OA.

	Diabetic OA (n = 76)		Non Diabetic OA (n = 53)		Total (n = 129)	
	No.	%	No.	%	No.	%
Difficulty performing daily activities						
Mild	2	2.6	19	35.8	21	16.3
Moderate	36	47.4	26	49.1	62	48.1
Severe	38	50	8	15.1	46	35.7
χ^2 (^{MC}p)	31.852* (0.001*)					
Total	**76**	**100**	**53**	**100**	**129**	**100**

χ^2: Chi square test
MC: Monte Carlo test
*: Statistically significant at p ≤ 0.05

DISCUSSION

The present study was conducted on two groups; the first group consisted of 100 diabetic type 2 patients with primary knee OA. The second group was 100 non diabetic patients with primary knee OA as a control group; matched regarding age and sex. All patients were subjected to complete knee clinical examination. Disease severity was assessed by radiological Kellgren Lawrence grading system. Western Ontario and McMaster University Osteoarthritis Index was used to assess severity of self-reported physical function limitation, pain, and stiffness. In diabetic group, laboratory parameters included Hb A1C in diabetic patients.

The results of the present study revealed no significant difference between the diabetic and non-diabetic patients as regard to age, sex, body mass index and occupation. These findings exclude these variables as effectors on pain severity, joint tenderness, progression of knee OA and the radiological findings.

The prevalence of knee OA among type 2 diabetic patients in current study was significantly higher compared to general population (76% in diabetics compared to 54% non-diabetics).

Nieves-Plaza et al in 2013 [18] reported that DM patients were more likely to have OA of hands or knees than were non-diabetic subjects. DM patients had 2.18 fold increased risk of hand or knee OA compared to non-diabetic subjects. Also, Hussin et al in 2013 [19] observed that there may be a possible association between diabetes mellitus type 2 and development of osteoarthritis of the knee joint. Louati et al in 2015 [20] reported that the prevalence of osteoarthritis in type 2 diabetic patients was significantly higher than the estimated prevalence in the general population. In a large study on osteoarthritis including 1026 patients, the mean fasting glucose concentration was higher in subjects with osteoarthritis than in subjects without OA [21].

Hyperglycemia is suggested to be the main trigger of joint degradation in OA. Local increased concentration of glucose may alter the cartilage matrix by increasing the formation of AGEs [22,23] that can activate chondrocytes and synoviocytes to produce pro-degradative and pro-inflammatory mediators and modify the quality of the subchondral bone. Alterations in the function of the glucose transporters at the surface of chondrocytes participate in the perpetuation of the deleterious process. Hyperglycemia induces a low-grade systemic inflammation state that aggravates the OA process [24]. Also, the neurotoxicity of hyperglycemia leads to a neuromuscular deficiency, which may also worsen OA by destabilizing the joint [25].

Tenderness of osteoarthritic knees may be relevant, among other causes, to synovitis and/ or associated anserine bursitis; both of which were found to be more prevalent among diabetic patients [26-29].

Doyle et al in 1981 [30] suggested that joint tenderness seemed to be reflection of inflammation in osteoarthritis. Thus, the more joint tenderness in the diabetic OA patients of this study might have been due to more inflammation in the diabetic OA compared to the non-diabetic group.

The studied clinical manifestations and radiologic features showed evidences of increased severity of knee OA in diabetic compared to non-diabetic patients. The observed increased narrowing of joint space among diabetic knee OA patients can be explained in the view of the diabetes-relevant increased activity of pro-inflammatory cytokines incriminated in increased rates of cartilage destruction [13,14].

Eymard et al in 2015 [31] found that type 2 DM was a predictor of radiologic joint space reduction in men with established knee OA.

Schett et al in 2013 [32] also found that type 2 diabetes is a strong predictor for the development of clinical and radiologic severe OA. This finding is independent of age and BMI and suggests that longstanding diabetes per se is detrimental for knee and hip joints, leading to progressive destruction and joint failure. Also, they stated that arthroplasties due to severe symptomatic hip/ knee OA were performed in diabetic subjects.

In addition, Cimmino et al in 1990 [21] found that poor glycemic control increase risk and severity of OA.

As regard WOMAC scaling of pain, stiffness and difficulty performing daily activities we found that more severe pain, stiffness and difficulty performing daily activities were much higher present in diabetic OA group than non-diabetic OA group. Also much higher incidence of mild pain, stiffness and difficulty performing daily activities were encountered among the non-diabetic group.

In agreement with current study Schett et al in 2013 [26] found that clinical symptoms of OA by total WOMAC score (pain, stiffness and difficulty performing daily activities) showed significantly more severe OA symptoms in subjects with type 2 diabetes than in controls. Moreover, WOMAC subscales for joint pain and function were more pathologic in type 2 diabetic patients than controls. Also, Schett et al in 2013 [26] stated that the pain subscales of the WOMAC and KOOS scores exhibit particularly pronounced associations with type 2 diabetes.

Sensory polyneuropathy obviously does not offset the high burden of pain in OA among diabetic subjects. Moreover, Berenbaum in 2011 [24] found that pain.

CONCLUSION

Knee OA is more prevalent in diabetic population. Knee OA in diabetic patients is associated with more severe symptoms and signs that reflect increased inflammation and cartilage degeneration. The results of this study provide probable partial explanation for the clinically observed severity of knee OA among diabetic patients.

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ABBREVIATIONS

DM - Diabetes Mellitus
OA - Osteoarthritis
AGEs - Advanced glycation end products.
ACR - American College of Rheumatology
BMI - Body Mass Index
WOMAC - Western Ontario and McMaster Universities Osteoarthritis Index
KOOS - Knee injury and Osteoarthritis Outcome Score
Hb A1C - Glycosylated hemoglobin

Conflicts of interest: None

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