THE EFFECT OF SOME ANTHROPOMETRIC MEASUREMENTS AND SERUM LACTATE DEHYDROGENASE ON KNEE OSTEOARTHRITIS

Rana Kareem Al- Saady[a] and Salah Mohammed Fizea[a]*

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Osteoarthritis (OA) is the most common degenerative joint disorder that ultimately results in the progressive destruction of articular cartilage. The occurrence of knee osteoarthritis (OA) increases with obesity and is more common in women compared with men. Thirty patients (20 females, 10 males) clinically diagnosed with knee OA admitted to Baghdad teaching hospital between January 2015-June 2015. Patients between 35-70 year of age and 30 healthy subject (20 females, 10 males) ages matched. The results showed that increased body mass index (BMI) is a well-recognized risk factor for knee osteoarthritis, and the effect of obesity is a stronger predictor of developing knee OA symptoms in women than men. There was also a significant difference between lactate dehydrogenase (LDH) in serum patients and control (p<0.01). This study supported a positive association between systemic BMI and OA. Furthermore, serum LDH activity could be of diagnostic value for identifying osteoarthritis.

*Corresponding author
E-Mail: salahmf79@yahoo.com
[a] Department of Chemistry, College of Education for Pure Science, Ibn Al-Haitham, University of Baghdad, Iraq.

Introduction

Osteoarthritis (OA) has been characterized by progressive articular cartilage loss and osteophyte formation. Although OA was long considered to be due only to an imbalance between loss of cartilage and an attempt to repair cartilage matrix, it is now known that OA, at least in the knee, is a heterogeneous disease involving all the articular tissues including cartilage, subchondral bone, menisci, and periarticular soft tissues such as the synovial membrane. Synovitis is often present and is considered to be secondary to the alterations in other joint tissues. Yet, findings indicate that synovial inflammation could be a component of even the early events leading to the clinical stage of the disease.[1]

Lactate dehydrogenase (LDH) is mainly an intracellular enzyme. It is responsible for interconversion of pyruvate and lactate in the cells. Its levels are several times greater inside the cells than in the plasma and are an enzyme that is expressed at higher levels when cells are distressed and damaged. Elevating LDH is a possible indication of disease progression.[2]

LDH helps facilitate the process of turning sugar into energy for cells to use. In inflammatory conditions like (rheumatoid arthritis) RA and OA, LDH may be released into the bloodstream causing its levels to increase and higher levels of LDH in the blood indicate acute or chronic cell damage.[3]

OA affects joint cartilage, adjacent skeleton, and surrounding soft tissue[4] and may affect most joints. Major risk factors for OA include age and obesity. The mechanism concerning the association between Body mass index (BMI) and knee OA is not clearly understood. BMI is an established risk factor for knee OA.[5]

The mechanism of the association between BMI and knee OA traditionally was thought to be purely biomechanical, with the excess weight inducing deleterious effects on the joints. This makes the differing associations between knees OA with BMI surprising because the forces from body weight pass through the hips as well as the knees, although the different morphology of the joints might explain different abilities to withstand adverse mechanical loading. However, recent advances in adipose biology have suggested the possibility that other factors may affect the joints. Patterns of distribution of adipose tissue within the body and associations with metabolic syndrome are now known, and adipocytokines are secreted by and related to adipose tissue. The adipocytokine, leptin, is not related to metabolic syndrome but also has direct effects on chondrocytes.[6]

The present study aims to investigate the effect of other anthropometric measures on knee OA and estimation of LDH value compared with control.

Experimental

Thirty patients (20 females, 10 males, between 35 and 70 year of age) clinically diagnosed with knee OA, attending rheumatology outpatient clinic in Baghdad teaching hospital between January 2015-June 2015, were selected. Healthy subjects (20 females, 10 males) of similar age group were selected as control. The OA was diagnosed based on physical examination, laboratory results and radiological findings. Blood was collected in a red vial and was centrifuged to separate serum from it and stored until the assay time.

Measurement of BMI

BMI was calculated in kg m^2 for the male and female groups, classifying as normal (<25 kg m^2), overweight (25 and <30 kg m^2) and obese (30 kg m^2 or more). Waist and hip was measured for every patient and control according to world health organization (WHO) criteria.[7]

Table 1. Demographic features and anthropometric measurements of the patent and control groups.

Characteristics	Male (patient)	Male (control)	p-value	Female (patient)	Female (control)	*p*-value
Number	10	10	--	20	20	-
Age (Years)	48.85±11.0	46.7 ± 10.2	0.34*	44.84±7.17	42.33±7.55	0.63*
BMI (Kg m^2)	29.9 ± 2.0	23.9±2.1	0.001	34.37±2.50	25.39±0.50	0.001
WHR	0.80± 0.04	0.70±0.04	0.001	0.88±0.05	0.72±0.04	0.001

* Not significant

Assay of serum lactate dehydrogenase

The concentration of serum LDH levels were determined by using kits from Randox, U.K.

Statistical Analysis

Values were calculated as mean ± SD and the statistical analysis was done using SPSS software and Microsoft Excel 2010. The *p*-value of less than 0.01, 0.001 was considered as statistically significant.

Results and discussion

The demographics of the present study are shown in Table 1. Twenty of the patients were female and ten were male, while twenty of the control patients were female and ten were male. The control groups had lower BMI and WHR and were younger than the knee OA.

The mean levels of serum LDH of the patient group is 253±55.7, whereas that of the control group is 161.4±23.9. This indicates that there is highly significant difference between the patients and control groups (p = 0.01).

Obesity is of particular interest amongst the risk factors for knee osteoarthritis. The effect of obesity on osteoarthritis is associated with the greater load that is observed in the lower extremities. It is possible that an excessive body mass for prolonged periods may increase the risk of subsequently developing knee OA and worsening of the disease.[8] In present study we have observed that being overweight or obese has a strong association with knee OA. High BMI is a well-known risk factor for knee OA and overweight has been found to precede the disease in the knee. Contact stress in knee-joint cartilage is a significant predictor of developing symptoms that are interpreted to indicate the presence of knee OA. Obesity has been consistently shown to be higher risk factor for knee OA for in the case of women than in the case of men.

In their large case-control study, Holliday et al investigated by using BMI the effects of obesity and other anthropometric measurements associated with obesity on severe knee OA and HOA (Hip osteoarthritis).[9] It has been observed that higher WC (waist circumference) and HC hip circumference values were associated with the risk of development of knee and hip OA, however, this association is lower in comparison to that with the BMI. When only women were evaluated, the HOA risk was observed to be associated with the waist: hip ratio.[10]

LDH is an enzyme that helps facilitate the process of turning sugar into energy for cells to use. In inflammatory conditions like RA, OA, LDH may be released into the bloodstream causing the levels to increase and higher levels of LDH in the blood indicate acute or chronic cell damage.[11]

Veys et al had shown that cases of rheumatoid arthritis had high LDH activity both in cell-free fluid and in cellular material.[12] In our study, we have observed a significant increase in serum LDH level in patients with OA in comparison with that in control groups.

From previous reports, degenerative joint diseases are deemed to be associated with increased LDH activity in the synovial fluid. In order to verify the distribution of LDH, Eveline et al have made a study to examine healthy and degenerative stifle joints for the goal of clarifying the origin of LDA in synovial fluid through many technical means, such as transmission electron microscopy (TEM), immunolabeling and enzyme cytochemistry. And then all techniques corroborated the presence of LDH in chondrocytes and in the interterritorial matrix of degenerative stiff joints. Whereas LDH is retained in healthy cartilage due to permeability limitations, it is released into synovial fluid through abrasion as well as through unrestricted diffusion as a result of degradation of collagen and increased water content in degenerative joints.[13]

Other studies have LDH levels in serum and synovial fluid of osteoarthritic patients.[14,15] LDH is an enzyme that catalyses the conversion of pyruvate to lactate and was found to be elevated in the synovial fluid of osteoarthritic joints.

Conclusion

These results indicate that obesity is a significant risk factor for the development of knee OA, and that the association is stronger for women than for men. LDH have become key targets in the development of the diagnosis and treatment of osteoarthritis, and significant progress has been made over the decades.

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