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Original Article

Serum level of adropin in primary knee osteoarthritis patients and its relation to ultrasonographic findings

Rheumatology

Nourhan M. Nooh[1], Soad A. El Sawy[1], Hemmat A. Elabd[1], Samar M. Abd Elhamid[2]

[1] Rheumatology and Rehabilitation Department, Faculty of Medicine for Girls, Cairo, Al-Azhar University, Egypt.

[2] Clinical Pathology Department, Faculty of Medicine for Girls, Cairo, Al-Azhar University, Egypt.

ABSTRACT

Background: Knee osteoarthritis (KOA) represents a chronic, multifaceted condition characterized by degenerative afflictions predominantly targeting the knee joint, a commonly implicated site in osteoarthritic pathologies. Adropin, a peptide hormone encoded by the energy homeostasis associated (ENHO) gene, that exhibits extensive expression across multiple tissues, notably in the liver, brain, heart, kidneys, pancreas, coronary arteries, and umbilical veins. Furthermore, adropin is discernible within a broad spectrum of bodily fluids, including plasma, serum, and exocrine secretions like colostrum and milk, indicating its pervasive role and systemic relevance in physiological processes.

Objective: Measurement of Serum Adropin level in knee osteoarthritis patients and its relationship with clinical manifestations, different radiological grading and ultrasonographic findings.

Methodology This case control study has been conducted on 40 patients with varying grades of knee osteoarthritis were compared to 40 age and sex-matched healthy people as controls. The serum adropin level has been measured in both groups using the enzyme linked immunosorbent assay (ELISA) technique.

Results: As regards adropin level, there were statistically significant differences between patient group with a mean of (266.12±65.75) compared with the control group with a mean of (582.63±129.73, $p < 0.001$). There was a negative correlation between adropin level "pg/ml" and Western Ontario and McMaster Universities (WOMAC) grading with p-value (< 0.05). There was negative correlation between adropin level "pg. /ml "and Kellgren Lawrence (KL) grading ($r = - 0.909$, $p < 0.05$).

Conclusion: Serum adropin concentrations were markedly reduced in patients with KOA relative to control subjects, demonstrating a specificity of 87.5% and a sensitivity of 90%. There exists a negative association between serum adropin levels and several clinical indicators: Kellgren Lawrence (KL) grading scale, cartilage thickness, the Western Ontario and McMaster Universities (WOMAC), and visual analogue scale (VAS). Consequently, serum adropin measurements may serve as both a sensitive and specific diagnostic biomarker for knee osteoarthritis

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Corresponding author: Nourhan M. Nooh, Rheumatology and rehabilitation department, faculty of medicine for girls, Cairo, Al-Azhar University, Egypt. **Tel:** 01080731518. **E-mail:** norhan.mostafaz123@gmail.com

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INTRODUCTION

Knee osteoarthritis (KOA) is a multifactorial, chronically debilitating disorder that predominantly afflicts the knee joint, a frequent target within osteoarthritic conditions. The pathophysiological alterations observed in KOA transcend mere passive degeneration or attritional damage; rather, they represent active modifications driven by disequilibrium between damage to and repair of articular tissues. This condition is typified by a metabolic dysregulation involving the anabolic and catabolic factors secreted by chondrocytes [1]

Knee osteoarthritis predominantly manifests in individuals aged 50 and above. It represents a chronic, progressive articular disease marked by clinical symptoms such as pain, joint deformation, and diminished mobility, frequently culminating in disability. In the context of an accelerating demographic shift towards an older population and an increasing incidence of obesity, KOA is recognized as the eleventh leading cause of disability on a global scale and is ranked 38th among factors that negatively impact life expectancy [2].

In the diagnostic process for KOA, clinicians primarily rely on physical assessments and radiological imaging

techniques. The predominant method employed is plain radiography, utilizing the Kellgren Lawrence (KL) classification system, which is noted for its high specificity albeit limited sensitivity. Additionally, ultrasound imaging has proven effective in identifying early-stage alterations indicative of KOA. Currently, there are no specific laboratory tests available that can definitively diagnose KOA in its initial stages [3].

Adropin, a peptide hormone, is synthesized under the regulation of energy homeostasis associated (ENHO) gene and is prominently expressed across a spectrum of tissues, notably the liver, brain, heart, kidneys, pancreas, coronary arteries, and umbilical veins. Furthermore, this hormone is detectable in an array of body fluids including plasma, serum, and secretions such as colostrum and milk, underscoring its widespread physiological distribution and functional significance [4]. Adropin regulates energy balance, glucose, lipid and protein metabolism. It has anti-inflammatory response, improves endothelial cell survival and can regulate endothelial nitric oxide (eNO) which exerts a protective effect against KOA [5]. Decreased adropin level leads to increase inflammatory mediators as TNF-alpha and IL-1 which plays a vital role in OA [6]..Diminished levels of adropin are linked with a variety of pathological conditions including insulin resistance related to obesity, acute myocardial infarction, gestational diabetes mellitus , endothelial disorders, non-alcoholic fatty liver disease, rheumatoid arthritis, and osteoarthritis [7]. The purpose of this study was the measurement of Serum Adropin level in knee osteoarthritis patients and to find its relationship with clinical manifestations, different radiological grading and ultrasonographic findings.

PATIENT AND METHODS

Study design and patients:

This case control study was carried out on 80 subjects attended the Rheumatology and Rehabilitation department's outpatient clinic at Al-Zahraa University Hospital. Forty patients with varying grades of knee OA, 5 of them were males and 35 were females as primary OA more common in females and their ages ranged from 50 to 65 years. Another 40 apparently healthy individuals with the same age and sex were included for comparison.

Inclusion criteria were knee OA patients aged 50 to 65 years' old who met the 2016 ACR Criteria for diagnosis of knee osteoarthritis.

Exclusion criteria were secondary knee osteoarthritis as infective arthritis, crystal deposition induced arthritis and inflammatory arthritis, malignancy, liver diseases, cardiovascular and respiratory diseases, systemic infections, autoimmune disease, diabetes, current pregnancy, breast feeding, consumption of alcohol, current acute or chronic infection, patient on oral glucocorticoid therapy > 10 mg daily.

All participants underwent a comprehensive evaluation, which included a detailed medical history, clinical examination encompassing general and locomotor assessments, pain assessment using the WOMAC score, and imaging studies such plain X-rays and ultrasonographic scans of the knee joint. KL classification according to plain x-ray showing grade 1 (doubtful joint space narrowing), grade 2 (definite osteophytes), grade 3 (as 2 plus minimal sclerosis) and grade 4 (as grade 2 plus severe sclerosis and definite deformity of bone ends).

Laboratory investigations: general tests: CBC and ESR. Kidney function tests: serum creatinine levels and blood urea, Liver function tests: serum ALT and AST levels and CRP were measured by Cobas Integra 400 auto-analyzer, using commercial kits supplied by Roche Diagnostics, Germany. Rheumatoid factor (RF) assay by latex agglutination method.

Special test; estimation of Adropin serum levels by ELISA. According to the manufacturer's instructions using Technology company, (China). Human Adropin ELISA Kit (Catalog Number 201-12-3107), provided by Sun Red Technology Company, based in China.

Diagnosis of knee osteoarthritis according to ACR 2016 criteria:

Domain I: Mechanical pain localized to the knee. Presence of tenderness upon palpation of knee bones. Audible crepitus during knee movement. Synovial fluid analysis consistent with osteoarthritis characteristics.

Domain II: Onset of symptoms occurring between the ages of 40 and 50 years. Enlargement of knee bone structures. Presence of osteophytes as detected in knee radiographs or evidence from knee MRI indicative of osteoarthritis.

In the context of OA diagnostic protocols, a score of three points out of a possible ten with at least one of these points derived from **Domain II-** is required for a confirmed diagnosis, assuming all preliminary criteria are met. Exclusion criteria for the diagnosis encompass several key indicators: 1) moderate to severe knee synovitis, 2) the presence of a hot or erythematous knee, 3) clinical history or examination findings indicative of internal knee derangement. Symptoms characteristic of KOA include pain that escalates with physical activity or exercise and diminishes upon resting. Additionally, the presence of synovial fluid that is clear and retains normal viscosity, with a WBC count below 2000/mm^3 and PMN percentage under 25%, should not distract from the diagnostic process, especially if osteophytes are evident in knee radiography [8].

The study received ethical approval from the Medical Ethics Committee of the Faculty of Medicine for Girls at Al Azhar University. Comprehensive counseling was provided to all participants involved in the study, and informed consent was duly obtained.

Statistical analysis

The data underwent analytical processing through the

utilization of the SPSS software suite (IBM Corp., 2017 edition, IBM SPSS Statistics for Windows, Version 25.0, Armonk, NY: IBM Corp). Quantitative data were summarized and analyzed using descriptive statistical measures such as mean values and standard deviations (± SD), while qualitative data were evaluated using frequency distributions and proportional analyses. The Chi-Square test was utilized to explore and ascertain the statistical associations between categorical variables. Correlation analysis was conducted to ascertain the strength and nature of the association between two quantitative variables. All statistical tests employed a two-sided approach, and the level of significance considered for this study was set at $p < 0.05$.

RESULTS

As regards demographic data; in patient group, there were 40 patients 5 males (12.5%), 35 were females (87,5%) with range of age (50-65). In control group, there were 40 apparently healthy people 3 were males (7.5%), 37 were females (92.5%) with age matched. According to BMI, patient group with mean (27.83±4.15) versus (26.07±3.13) in control group as shown in table (1).

Table (2) shows clinical data among patient group according to WOMAC, VAS, KL classification. As for WOMAC, 13 patients (32.5%) had mild affection with scoring between (0- 24), 17 patients (42.5%) had moderate affection with scoring (24- 48) and 10 patients (25.0 %) had severe affection with scoring (48-72) with a mean of (47) SD range of (27 – 66). According to VAS score, in patient group (40) patient, total vas score from 10 there was median score of 6, SD (5-8). For Kellegren - Lawrance classification, 12 patients (30%) were class 1, 15 patients (37.5%) were class 2, 7 patients (17.5%) were class 3 and 6 patients (15.0%) were class4 among patient group.

A markedly significant disparity was discerned between the control group and the patient group concerning the thickness of Lt and Rt middle cartilage. Furthermore, a statistically significant difference was identified between the patient group and the control group in the context of Rt and Lt medial cartilage thickness. In addition, an exceptionally significant divergence was detected between the control group and the patient group regarding the thickness of the Rt and Lt lateral cartilage. As regards medial and lateral cartilage degeneration grading, there was highly significant difference between patient group and control group, (p-value < 0.001 for all) as displayed in table 3.

In the context of knee effusion, a highly statistically significant disparity was evident between the control and the patient group, denoted by a p-value of less than 0.001. There was highly statistically significant difference between patient group 24 patients (60,0%) had no power Doppler grade 0, 9 patients (22.5%) grade 1, 6 patients (15.0%) grade 2, 1patient (2.5%) grade 3 and control group with p-value (<0.001). For synovial hypertrophy grading, there was highly statistically significant difference between patient group 18 patients (45.0%) had no synovial hypertrophy grade 0, 16 patients (40.0%) grade 1, 6 patients (15.0%) grade 2, and control group with p-value (<0.001) as shown in table 4.

Pertaining to osteophyte protrusion, a statistically significant distinction was identified between the patient group and the control group, with the difference being underscored by a p-value of less than 0.001. Regarding meniscal tears, there was also a statistically significant difference, with 5 patients (12.5%) in the patient group exhibiting meniscal tears compared to the control group, evidenced by a p-value of less than 0.05. Laboratory evaluations indicated a statistically significant variance between the patient and control groups across several parameters, evidenced by a p-value of less than 0.05, with the exception of CRP levels, where the difference did not reach statistical significance, reflected by ap-value exceeding 0.05. Pertaining specifically to adropin levels, there was a highly statistically significant differences between patient group with a mean of (266.12±65.75) compared with control group with a mean of (582.63±129.73) with p –value (< 0.001) as shown in table 5.

Table (1): Characteristics of patients and controls

Demographic data	Patients group n = 40	Control group n = 40	p-value
Age "years"			
Mean ± SD	55.20±9.11	50.83±6.13	0.001*
Range	50-65	50-65	
Sex (no.%)			
Male	5 (12.5%)	3 (7.5%)	0.456
Female	35 (87.5%)	37 (92.5%)	
BMI [wt/ (ht) ^{2}]			
Mean ± SD	27.83±4.15	26.07±3.13	0.036*
Range	20.36-35.4	20.36-31.2	

Using: t-Independent Sample t-test for Mean ± SD; Using: x^2: Chi-square test for number (%) or Fisher's exact test, when appropriate, *: Significant p-value (< 0.05)

Table (2): Clinical data among patient group according to (WOMAC grading, VAS, KL classification)

Other Clinical	Patient group n = 40
VAS	
Median (IQR)	6 (5-8)
Range	3-10
WOMAC Grading (no. %)	
Mild (0-24)	13 (32.5%)
Moderate (24-48)	17 (42.5%)
Severe (48-72)	10 (25.0%)
WOMAC score	
Median (IQR)	47 (27-66)
Range	20-80
KL classification (no,%)	
Class 1	12 (30.0%)
Class 2	15 (37.5%)
Class 3	7 (17.5%)
Class 4	6 (15.0%)

IQR: Interquartile range. Chi-square test for number (%) or Fisher's exact test, *: Significant p-value (< 0.05)

Table (3): Comparison between patient group and control group according to RT, LT cartilage thickness and cartilage degeneration grading

	Patients group n = 40	Control group n = 40	p- value
Cartilage thickness RT knee			
Rt middle	2.01±0.37	2.92±0.18	0.001*
Rt medial	1.25±0.15	1.79±0.10	0.001*
Rt lateral	1.54±0.22	1.98±0.11	0.001*
Cartilage thickness Lt knees			
Lt middle	2.13±0.36	2.89±0.13	0.001*
Lt medial	1.35±0.32	1.78±0.10	0.001*
Lt lateral	1.45±0.26	1.98±0.10	0.001*
Cartilages Degeneration Grading			
- **Medial cartilage degeneration Grading**			
Grade 0	0 (0.0%)	33 (82.5%)	0.001*
Grade 1	5 (12.5%)	7 (17.5%)	
Grade 2A	15 (37.5%)	0 (0.0%)	
Grade 2B	12 (30.0%)	0 (0.0%)	
Grade 3	8 (20.0%)	0 (0.0%)	
- **Lateral cartilage degeneration Grading**			
Grade 0	0 (0.0%)	36 (90.0%)	0.001*
Grade 1	5 (12.5%)	4 (10.0%)	
Grade 2A	17 (42.5%)	0 (0.0%)	
Grade 2B	13 (32.5%)	0 (0.0%)	
Grade 3	5 (12.5%)	0 (0.0%)	

Using: t-Independent Sample t-test Using: x^2: Chi-square test for Number (%) or Fisher's exact test, when appropriate, *: Significant p-value (< 0.05)

An inverse correlation was noted between adropin levels (pg/ml) and WOMAC grading, which was statistically significant with a p-value of less than 0.05. Additionally, there was a significant negative correlation between adropin levels (pg/ml) and the KL grading scale, indicated by a correlation coefficient of -0.909 and a p-value of less than 0.05. Furthermore, a negative relation was also observed between adropin levels (pg/ml) and both Medial and Lateral cartilage degeneration grading, with this association reaching high statistical significance, as evidenced by a p-value of less than 0.001, as depicted in figure 1.

There was a higher mean value of Adropin in Mild WOMAC grading, followed by moderate and severe,

with p-value (p<0.001). As regards adropin level and KL classification, there was statistically significant higher mean value of Adropin in Class 1, followed by Class 2, then Class 3 and Class 4 about KL classification, with p-value (p<0.001). According to cartilage degeneration grading and adropin level, there was a negative correlation between adropin "pg/ml" with Medial and Lateral cartilage degeneration grading, with p-value (p<0.001) as shown in table 6.

ROC curves were utilized to assess the prognostic utility of adropin as a biomarker for predicting knee osteoarthritis in the study participants, demonstrating a robust predictive value as indicated by the substantial AUC. The determination of the optimal threshold for adropin was identified at a value less than 363.8, exhibiting a specificity of 87.5%, sensitivity of 90%, along with a PPV of 87.8%, and a NPV of 89.7%. The diagnostic efficacy of this measure was further validated by an AUC of 0.949, with a standard error of 0.0471, and a 95% CI spanning from 0.876 to 0.986. The statistical significance was marked by a Z statistic of 5.234 with a p-value of less than 0.001, as illustrated in figure (2).

Table (4): Comparison between patient group and control group according to Effusion, power Doppler and synovial hypertrophy

	Patients group n = 40	Control group n = 40	p-value
Effusion			
No	9 (22.5%)	40 (100.0%)	0.001*
Grade 1	14 (35.0%)	0 (0.0%)	
Grade 2	10 (25.0%)	0 (0.0%)	
Grade 3	7 (17.5%)	0 (0.0%)	
Power Doppler			
Grade 0	24 (60.0%)	40 (100.0%)	0.001*
Grade 1	9 (22.5%)	0 (0.0%)	
Grade 2	6 (15.0%)	0 (0.0%)	
Grade 3	1 (2.5%)	0 (0.0%)	
Synovial hypertrophy			
Grade 0	18 (45.0%)	40 (100.0%)	0.001*
Grade 1	16 (40.0%)	0 (0.0%)	
Grade 2	6 (15.0%)	0 (0.0%)	

Using: x^2: Chi-square test for Number (%) or Fisher's exact test, when appropriate, *: Significant p-value (< 0.05)

Table 5: Comparison between patient group and control group according to laboratory finding and Ultrasound

	Patients group n = 40	Control group n = 40	p-value
Laboratory finding			
Adropin (pg/ml	266.12±65.75	582.63±129.73	0.001*
Hb (g/dl)	11.55±0.96	12.02±0.83	0.021*
WBCs × 10^3 /uL	6.94±1.67	6.21±1.29	0.032*
ESR (mm/h)	17.50±3.95	11.28±2.24	0.001*
CRP (mg/L)	3.02±0.79	2.65±0.73	0.032*
Other ultrasound findings			
Osteophytes	22 (55.0%)	0 (0.0%)	0.001*
Protrusion	13 (32.5%)	0 (0.0%)	0.001*
Meniscial tear	15 (12.5%)	0 (0.0%)	0.021*
Meniscial degeneration	13 (32.5%)	0 (0.0%)	0.001*

Using: t-Independent Sample t-test for Mean ± SD; Using: x^2: Chi-square test for Number (%) or Fisher's exact test, *: Significant p-value (< 0.05)

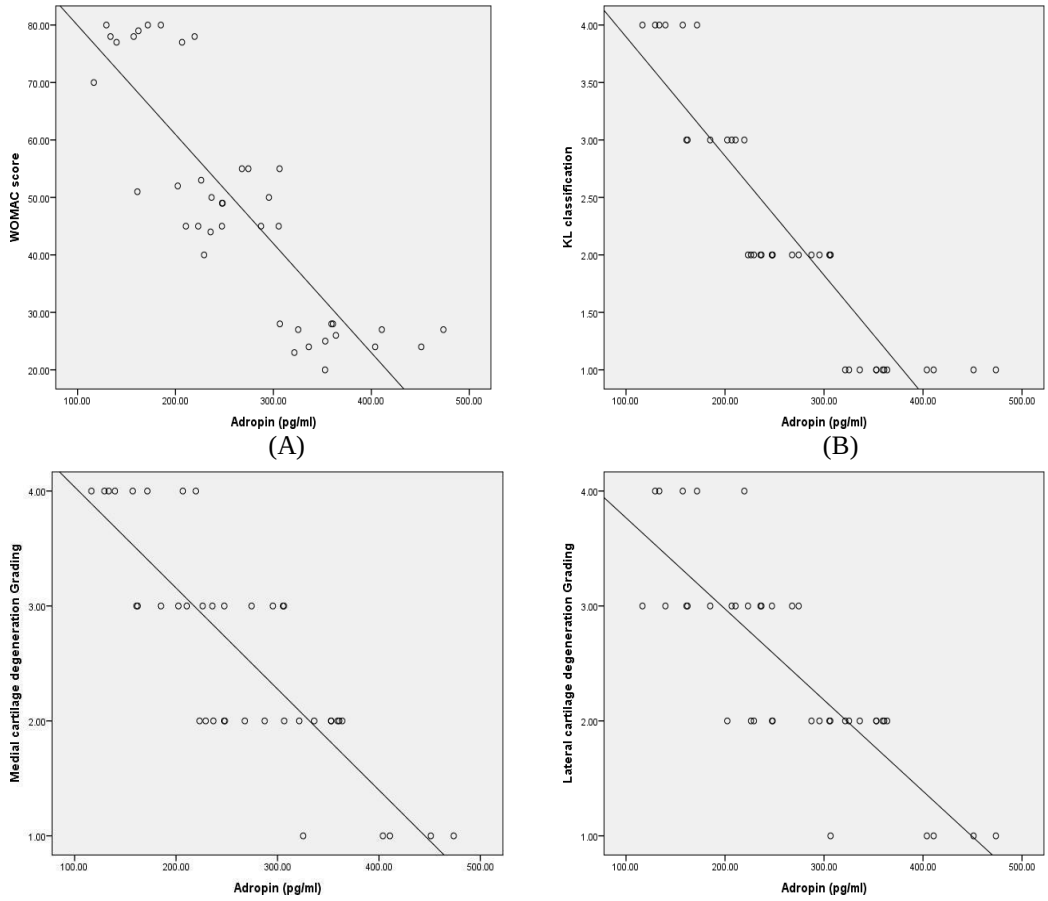

Figure (1): Negative correlation between adropin "pg/ml" and (A) WOMAC score and (B) KL classification and (C) and medial cartilage degeneration grading and (D) lateral cartilage degeneration grading

Table (6): Association between Adropin (pg/ml) level with WOMAC grading, KL classification and cartilage degeneration grading in patient group

	Adropin (pg/ml) Mean ± S D	**p-value**
WOMAC Grading		
Mild	370.6 2 ± 50.38	0.001*
Moderate	247.34 ± 38.99	
Severe	162.18 ± 33.95	
KL classification		
Class 1	375.96 ± 48.63	0.001*
Class 2	262.49 ± 31.14	
Class 3	192.48 ± 23.53	
Class 4	141.40 ± 19.97	
Cartilage Degeneration Grading		
- Medial cartilage		
Grade 1	412.90 ± 56.78	0.001*
Grade 2A	299.56 ± 53.60	
Grade 2B	234.35 ± 52.58	
Grade 3	159.33 ± 37.39	
- Lateral cartilage		
Grade 1	409.16 ± 64.13	0.001*
Grade 2A	301.18 ± 53.00	
Grade 2B	205.18 ± 49.26	
Grade 3	162.30 ± 36.37	

Using: One-way Analysis of Variance test was performed for Mean ± SD and multiple comparison between groups through Post Hoc test: Tukey's test. Different capital letters indicate significant difference at ($p<0.05$) among means in the same row, *: Significant p-value (< 0.05)

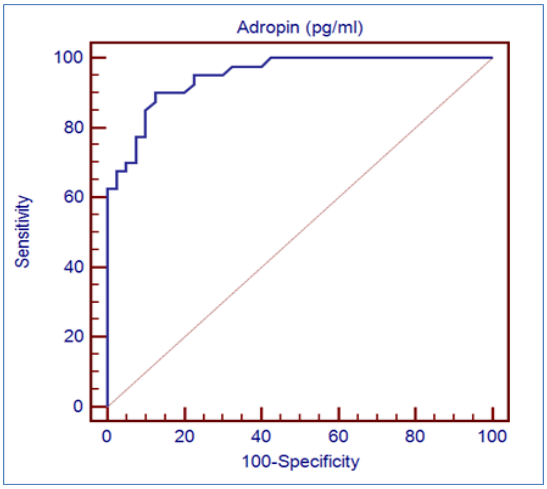

Figure (2): The receiver operating characteristic (ROC) curve between serum adropin level of knee osteoarthritis patients and controls

DISCUSSION

Osteoarthritis (OA) is a progressively debilitating joint disorder, principally marked by the degeneration of articular cartilage, which leads to pain, stiffness, and limited joint mobility. The exact etiology of osteoarthritis is multifactorial and not fully understood [9]. In our study, it was found that the ages of the studied groups ranged from 50-65 years. This is in agreement with McCarthy et al. [10], who stated that most of the adult population suffering from KOA are over 45 years old. Oxidative stress due to aging plays a key role in the catabolic- anabolic imbalance that causes progressive matrix destruction of cartilage leads to OA [10].

Our patient group comprised 5 males (12.5%) and 35 females (87.5%). Meanwhile, the control group with 3 males (7.5%) and 37 females (92.5%), showing an insignificant difference between the two groups. As supported by Tosi et al.(2019), who attributed this higher prevalence to hormonal, neural, and mechanical events in the joint [11]. In our study, 34 (85%) had bilateral knee affection, while 6 (15%) had unilateral side affection. Bilateral knee affection may have the highest prevalence, as unilateral affection often progresses into bilateral disease, particularly in patients with advanced disease [12]. As for the WOMAC grading, 13 patients (32.5%) had mild affection with scores between 0 and 24, 17 patients (42.5%) had moderate affection with scores between 24 and 48, and 10 patients (25.0%) had severe affection with scores between 48 and 72, with a median 47 (27-66). This coincides with the results of Alnahdi et al. [13] who attributed physical function limitations in KOA to muscle impairments, not limited to quadriceps muscles but also involving the hamstrings and muscles of the hip joint.

Regarding ultrasound findings, as for Rt and Lt cartilage thickness, significant differences were observed between the patient and control groups. For example, regarding the Rt middle cartilage thickness, there was a highly significant difference between the patient group and the control group with a p-value < 0.001. Similar results were reported by Pane et al., who compared cartilage thickness in osteoarthritis patients and a control group, finding significantly smaller cartilage thickness in the osteoarthritis group [14]. Additionally, a pronouncedly significant disparity was observed between control group and the patient group, ($p< 0.001$). This finding is consistent with the research conducted by Singh et al. [15] which explored the associations between ultrasonographic observations and the symptomatic manifestations of knee OA.

We found a statistically significant difference regarding osteophytes, protrusion, meniscal tear, and meniscal degeneration between the control and patient groups. Similarly, there were significant differences in protrusion, meniscal tear, and meniscal degeneration between the two groups. Singh et al. [15], also reported prevalent imaging findings on ultrasonography, such as osteophytes and meniscal extrusion [15]. Regarding power Doppler, our study showed significant differences between the control and patient groups. This aligns with findings by Vries et al. [16] who reported on power Doppler ultrasound findings in osteoarthritic patients.

In our study, we observed a highly statistically significant difference in serum adropin levels between the patient and control groups. Adropin, primarily associated with energy homeostasis and vascular protection, has been linked to various disorders characterized by low-grade chronic inflammation, including OA [7]. Additionally, our study revealed a negative correlation between adropin levels and KL grading. Our study has reported correlations between adropin levels and disease severity, with lower adropin levels associated with more advanced OA stages [17].

In our study, we observed a negative correlation between adropin levels and both Visual Analog Scale VAS and WOMAC. This finding aligns with previous research indicating a relationship between adropin levels and OA severity. In contrast, Serban et al. [18] presented divergent results, demonstrating a substantial

correlation between the pain subscale of the WOMAC and the KL grading system. Singh et al. [15] and Roemer et al. [19] identified positive correlations between the VAS, the WOMAC, and knee OA. Conversely, Naredo et al. [20] discovered a strong association between WOMAC scores and pain experienced both during knee motion and at rest.

CONCLUSION

Our investigation disclosed that serum adropin concentrations were notably diminished patients with KOA when juxtaposed with control subjects, demonstrating a specificity of 87.5% and a sensitivity of 90%. There exists an inverse relationship between serum adropin levels and the KL grading scale, cartilage thickness, WOMAC, and VAS. Consequently, serum adropin measurements may serve as a sensitive and specific diagnostic marker for KOA. Future research endeavors should prioritize prospective cohort studies with large sample sizes to provide more robust evidence regarding the relationship between serum adropin levels and OA severity and explore the potential synergistic effect of serum adropin level with other biomarkers. More efforts should be made to control for potential confounding variables as comorbidities, medication use and lifestyle factors to minimize bias and enhance the validity of study results. Standardization of Assay methods for adropin measurement to minimize errors and variability.

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الملخص العربي

مستوى الأدروبين في الدم في مرضى خشونة الركبه الأولية وعلاقته بنتائج التصوير بالموجات فوق الصوتية

نورهان مصطفى عبد الغفار[1]، سعاد أحمد الصاوى[1]، همت أحمد العبد[1]، سمر محمد عبد الحميد[2]

[1] قسم الروماتيزم والتأهيل، كلية طب بنات، القاهرة، جامعة الأزهر، جمهورية مصر العربية.

[2] قسم الباثولوجيا الإكلينيكية، كلية طب بنات، القاهرة، جامعة الأزهر، جمهورية مصر العربية.

ملخص البحث

الخلفية: خشونة الركبة من أحد الأمراض المزمنة متعددة الأسباب، تتميز بفقدان الغضروف التدريجي والإضطراب اللاحق في بيولوجيا الخلايا الغضروفية. الأدروبين هو هرمون الببتيد المشفر بواسطه انهو جين و يبلغ مستوى التعبير عن الأدروبين بشكل أساسى في أنسجه الكبد والدماغ والقلب والكلى والبنكرياس والشرايين التاجية والأوردة السرية وأيضا في البلازما والمصل وسوائل الجسم مثل اللبأ والحليب، مما يدل على دوره المنتشر وأهميته النظامية في عملية فسيولوجية ، ويمكنه تنظيم أكسيد النيتريك البطانى الذى يمارس تأثيرا وقائيا ضد خشونة الركبة.

الهدف: قياس مستوى الأدروبين في الدم لدى مرضى خشونةمفصل الركبة وارتباطه بالأعراض والتصوير الإشعاعي ونتائج الموجات فوق الصوتية.

الطرق: في هذه الدراسة تمت مقارنة 40 مريضا بدرجات متفاوته من خشونة مفصل الركبه مع 40 شخص ممن يتطابقون مع العمر والجنس الذين يتمتعون بصحة جيدة كعناصر ضابطة. تم قياس مستوي الأدروبين في كلا المجموعتين باستخدام تقنية مقايسة الممتز المناعي المرتبط بالإنزيم.

النتائج: كان هناك انخفاض ذو دلاله إحصائية في مستوي الأدروبين في مجموعه خشونة مفصل الركبه بمتوسط (65.759±266.12) مقارنه ب (129.73±582.63) في المجموعه الضابطه، وكانت هناك علاقه سلبيه بين مصل الأدروبين ومؤشر خشونه المفاصل في جامعات غرب اونتاريو وجامعه ماكماستر، وقياس كيلجرين لورانس.

الإستنتاجات: انخفض تركيز الأدروبين في الدم بشكل ملحوظ في المرضى الذين يعانون من خشونة مفصل الركبة بالنسبة إلى الأشخاص الخاضعين للمراقبة، ويرتبط مستواه بشكل سلبي مع مؤشر خشونه المفاصل في جامعات غرب اونتاريو وجامعه ماكماستر، وقياس كيلجرين لورانس. ونتيجة لذلك، قد تكون قياسات الأدروبين في الدم بمثابة العلامات الحيوية التشخيصية الحساسة والمحددة لالتهاب مفاصل الركبة.

الكلمات المفتاحية: خشونة مفصل الركبة، الأدروبين، الموجات فوق الصوتية.

الباحث الرئيسي:

الاسم: نورهان مصطفى عبد الغفار، قسم الروماتيزم والتأهيل، كلية طب بنات، القاهرة، جامعة الأزهر، جمهورية مصر العربيه.

الهاتف : 01080731518

البريد الالكتروني: norhan.mostafaz123@gmail.com