RESEARCH ARTICLE

SARCOPENIA VERSUS GONARTHROSE: RELATION BETWEEN THE BODY COMPOSITION AND THE STRUCTURAL SEVERITY OF THE KNEE OSTEOARTHRITIS

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Abstract

Background: Sarcopenia is initially defined as the involuntary loss of age-related muscle mass. Body composition has been rarely studied during osteoarthritis.

The aim: of this study was to determine the relationship between the body composition and the structural severity of the knee osteoarthritis.

Methods: We enrolled 100 patients with knee osteoarthritis of all stages. Lean mass and fat mass were measured by absorptiometry (DXA). The skeletal mass index (SMI) was defined as appendicular lean mass /height squared, and the sarcopenia in our patients was defined according to the International Working Group on Sarcopenia (IWGS) by a threshold lower than 5.67 kg/m². Pain and function have been assessed using the WOMAC, and the radiographic severity was evaluated by knee x-rays according to the Kallgren-Lawrence classification.

Results: Two groups were defined by severity based on the Kellgren-Lawrence classification; mild knee osteoarthritis as group 1 (stage 1 and 2 n = 45); and severe knee osteoarthritis as group 2 (stage 3 and 4 n =55). The mean body fat and lean mass were 39.68±7.44Kg and 38.89±4.5Kg in group 1 and 2 respectively. The fat and lean mass indices were 15.698±3.07Kg/m² and 15.66±1.93Kg/m² respectively. Our study found that sarcopenia is related to the severity of knee osteoarthritis (p<0.001). In univariate analysis and in multivariate analysis, decreased lean mass (sarcopenia) was associated with history of fracture (p <0.05); Sarcopenia was not associated with WOMAC pain.

Conclusion: Our study found that the low lean mass index was significantly associated to the radiographic severity of knee OA.

Introduction:-

Background: Sarcopenia is a mysterious entity, whose boundaries are blurred and in motion. It is defined as the involuntary loss of muscle mass in old age. This term finds its etymology in the Greek “sarx”, the flesh, and “penia”, the loss. The pathophysiology is incompletely understood, but clearly reveals similarities with osteoarthritis: both diseases are linked and one aggravates the other. Patients with knee osteoarthritis have a high prevalence of sarcopenia.
Main symptom of osteoarthritis is pain that affects muscle activity and contraction, as well as the muscle weakness gives joints instability and degeneration of cartilage.[1][2] Recent studies have highlighted the role of the dynamic compression in the transport of solute and cartilage constructs like this [3][4], the quadriceps wasting was associated with increased risk for knee osteoarthritis [5], also, the increase in fall reported with osteoarthritis of the lower extremity has been explained by disorder offunctional capacity, notably the decrease in muscle strength and muscle weakness.[6] Body composition has been rarely studied during osteoarthritis. The aim of this study was to evaluate the relationship between structural severity of osteoarthritis and sarcopenia and whether this association is influenced by pain and functional discomfort.

Methods:-
Subjects:
One hundred women with knee osteoarthritis were recruited from the rheumatology department of the Mohamed V Military Hospital (HMIMV). Written informed consent was obtained from all of the participants.

This was a cross-sectional study conducted from November 2017 to February 2018. The inclusion criteria included knee osteoarthritis on standard radiography imaging at all stages. We excluded subjects with diseases known to affect bone metabolism; neoplasia, kidney disease, hyperparathyroidism, chronic inflammatory rheumatism, gastrectomy, intestinal resection, recent hyperthyroidism or hyperparathyroidism, recent severe immobilization or patients taking drugs known to influence bone metabolism (corticosteroids (more than 3 months) or hormone therapy). A change in weight> 5% in the last 3 months, knee surgery or corticosteroid infiltration or viscosupplementation in the last 6 months were also excluded. Socio-demographic and health information about each participant was collected, including age, sex, ethnicity, and comorbid conditions.

Body composition parameters measurements:
BMI was calculated with the formula: weight (in kg)/[height (in m²)]. Pain was assessed through the WOMAC pain and the WOMAC function. The severity of osteoarthritis was assessed using knee x-rays requested to stage knee osteoarthritis according to the classification of Kellgren and Lawrence, in 4 grades. Body composition was measured by dual-energy X-ray absorptiometry (DXA) (GE Healthcare Lunar Prodigy): total mass, total lean mass(kg), total fat mass (kg) and percent fat mass (%) were collected. Lean mass index (LMI: total lean mass/height²), appendicular lean mass (ALM: arms LM/legs LM), appendicular lean mass index (ALMI: appendicular lean mass/height²), skeletal muscle mass index (SMI: ALM/height²) were calculated.

The patients were divided into two groups based on the presence or absence of sarcopenia. This study defined sarcopenia according to the International Working Group on Sarcopenia (IWGS), as skeletal mass index (SMI) <5.67 kg/m² for female by a threshold value ASMI = appendicular skeletal muscle mass index <5.67 kg/m².

Statistical analysis:
Descriptive statistics were presented in mean ± standard deviation (SD), and frequency, as appropriate. Univariate and multivariate logistic regression analyses were used to assess the relationship between body composition measures and knee OA. To compare patients with sarcopenia and without, and the severity of knee osteoarthritis chi-square test was used firstly. All analyses used p <0.05 as the threshold for statistical significance. All statistical analyses were carried out using SPSS software.

Results:-
Among the 100 women enrolled into the present study, 31 (31%) were diagnosed with sarcopenia. The patients were divided into two groups based on the presence or absence of sarcopenia, and we compared patient characteristics, between the groups (Table 1). No differences were observed between the study groups in BMI, WOMAC pain, WOMAC function and T-score. Women in the sarcopenia group were younger than those in the group without sarcopenia (59 ± 6.1 vs 58.2 ± 5.4 years, P 0.01).

After adjustment, sarcopenia remained significantly and positively associated with severity of knee OA (chi-square test, p<0.001), and history of fracture (chi-square test, P = 0.031). While diabetes, dyslipidemia and menopause were not found to be significantly associated with sarcopenia (p>0.05)(table 2).
On uni-variable and multivariate analysis, only knee osteoarthritis was significantly associated with sarcopenia. (p<0.0001) (table 3)

**Discussion:**

Our study show that sarcopenia is positively associated with the severity of knee OA. Sarcopenia was first described in 1988 as the age-related loss in skeletal muscle mass, and several studies showed that the prevalence of osteoarthritis increases among the aged.

Menopause accelerates the aging changes of muscle mass, that may accelerate sarcopenia. [7] In addition, pathophysiological and histological similarities and clinical coexistence of these diseases have been demonstrated. Both osteoarthritis and sarcopenia are the consequence of inability to regenerate tissues and organs, rather structural unit damage (chondrocytes and myocytes), involving an inflammatory state, or a production of free radicals and cellular apoptosis.

A statistical association between decrease in lower limb lean mass and osteoarthritis of the knee in women has been shown [8][9]. It is known that bed rest leads to activity limitation, that contributes to loss of muscle mass.[10] and to a neuronal loss, affecting alpha motor neurons. This contribute to sarcopenia development, as changes are correlated with a disorder of the neuromuscular junction, caused by increased production of several cytokines.[11]

Indeed, sarcopenia has been described as presenting in chronic disease states and especially associated with the systemic inflammation of these diseases, in particular, elevated levels of TNF-α, IL-6, IL-1, and CRP, also observed in acute flares of knee osteoarthritis. This interplay between sarcopenia and osteoarthritis explained along with the increasing stresses on joints by muscle weakness that can contribute to cartilage deterioration.[12][13].

R. Youssef et al, conclude that quadriceps atrophy induced severe cartilage damage[14]

There were no differences between the two groups of sarcopenia vs. no sarcopenia; concerning the WOMAC pain and the WOMAC function. As could be expected, our patients have chronic forms of arthritis, and receive analgesic treatment.

A prospective study has evaluated the potential role of self-reported joint pain by participants who have a radiographic osteoarthritis (ROA), in sarcopenia progression and falls risk in older adults. It concluded that knee and hip pain contribute to weakness of the knee extensor mainly related to the progression of sarcopenia and increased the risk of falls. [15] .

The prevalence of osteoarthritis is high in elderly subjects[16][17]. The results of our study highlight the need to undergo therapeutic exercise intervention as a means to alleviate the symptoms of OA, as well as to improve their muscle strength. The EULAR recommendations specify an isometric exercise for both. Aerobic activity, stretching and exercise are also recommended.[18][19]

To fight obesity and to conserve lean body mass prevent the musculoskeletal disorders in aging. [20] Some authors have shown that an increase of skeletal muscle mass acts as a protector from the development of pain and OA [21][22]. Therefore, hormonal therapy increases muscle mass in sarcopenia [23]. However, testosterone has unavoidable side effects such as cardiovascular disorders [24].

One research group has found an efficacious administration paradigm using selective androgen receptor modulator to combat sarcopenia .[25] Suppression of the Myostatin-based pathway, referred to as growth differentiation factor (TGF) by a myostatin binding protein, follistatin, is attributed to a muscle hypertrophy [26]. Also, follistatin normally acts to inhibit other TGF to regulate muscle size, like activin and Growth differentiation factor 11 (DFG11). [27] Both GDF11 and Myostatin are negative regulators of skeletal muscle mass.[28]

It has been demonstrated that the percentage of apoptotic cells collected from elderly subjects was increased versus young individuals, which, aging is associated with an increase or decrease of enzymatic antioxidant defense [29], occurring with a decrease in antioxidants [30]. Other researches verified that Green tea (Camellia sinensis) contains potential antioxidant, which activates the proliferation and differentiation of cells in aged rats.[31] hypothesis that may be a potential therapeutic target for sarcopenia. Also, A double-blind, placebo-controlled trial of the
angiotensin-converting enzyme ACE inhibitor showed that this treatment was associated with significant improvements in walking distances in treatment groups compared to control, the ACE inhibitors to improved muscle strength in older people.

Conclusion:-
This study showed an independent association between sarcopenia and osteoarthritis of the knee. The pathophysiology of these diseases is similar and early treatment of sarcopenia may prevent the occurrence of knee osteoarthritis.

Abbreviation:
DXA: Dual-energy X-ray absorptiometry; OA: Osteoarthritis; SMI: skeletal mass index; IWGS: International Working Group on Sarcopenia; WOMAC: Western Ontario and McMaster Universities Arthritis Index; LMI: Lean mass index; ALM: appendicular lean mass; ALMI: skeletal muscle mass index; TGF: Transforming Growth Factor; GDF11: Growth differentiation factor 11; ACE: angiotensin converting enzyme; BMI: body mass index; SD, standard deviation; Ns: not significant.

Ethics Approval and Consent to Participate:
A subjects’ written consent was obtained according to the Declaration of Helsinki and the study was approved by our local ethics committee (Military Hospital Mohammed V, Rabat).

Author information

Consent for Publication:
Not applicable.

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The authors declare that they have no competing interests.

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