

Prevalence and Clinical Characteristics of Diabetic Myopathy in Type 2 Diabetes Mellitus

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ABSTRACT

Background: Diabetic myopathy is an under-recognized complication of Type 2 Diabetes Mellitus (T2DM) that significantly contributes to physical disability. The objective was to estimate the frequency and clinical characteristics of myopathy in T2DM patients using clinical evaluation and electromyography (EMG).

Methods: This cross-sectional study evaluated 600 patients with T2DM (duration >5 years) at a tertiary care center. Comprehensive assessments included clinical history, anthropometric measurements, biochemical profiles, and EMG. Myopathy was diagnosed based on clinical features and confirmed through neurophysiological findings. Diabetic myopathy was diagnosed based on clinically demonstrable muscle weakness confirmed by EMG evidence of a myopathic pattern after exclusion of alternative neuromuscular disorders.

Results: Myopathy was identified in 85 patients, yielding a frequency of 14.2%. The condition peaked in the 61-70 age group (41.2%) and was more prevalent in males (57.6%). Key correlates included obesity (52.9%) and prolonged disease duration (>15 years). Muscle weakness was a universal finding (100%), with a marked predominance of proximal involvement (61.2%) over distal patterns. Notably, the condition was largely painless (94.1%) with an absence of muscle tenderness. Biochemical analysis revealed poor glycemic control (mean HbA1c 10.33%) and near-normal CK-NAC levels (mean 90.04 U/L), reflecting a non-inflammatory metabolic etiology.

Conclusion: Diabetic myopathy affects a significant proportion of patients with long-standing T2DM. It typically presents as a painless, proximal weakness driven by chronic metabolic derangement. Incorporating musculoskeletal screening into routine diabetic management is essential for early detection and the implementation of interventions to preserve motor function and independence.

Keywords: Type 2 Diabetes Mellitus, Diabetic Myopathy, Electromyography, Sarcopenia, Proximal Myopathy, Metabolic Complications, Neurophysiology

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INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) remains one of the most formidable global health challenges of the 21st century, traditionally characterized by its microvascular and macrovascular sequelae. [1-3] While the clinical focus has historically centered on nephropathy, retinopathy, and cardiovascular disease, an emerging paradigm recognizes the skeletal muscle as a critical target of end-organ damage. Often termed diabetic myopathy-this complication manifests as a progressive decline in muscle mass (sarcopenia), reduced muscular strength, and impaired functional endurance.[4] Given that skeletal muscle accounts for approximately 40% of total body mass and serves as the primary site for insulin-mediated glucose disposal, its dysfunction creates a deleterious feedback loop that exacerbates metabolic instability and increases mortality.[5]

The pathogenesis of diabetic myopathy is multifactorial and involves metabolic derangement, impaired skeletal muscle microvascular function, oxidative stress, and mitochondrial dysfunction.[4,6] Chronic hyperglycaemia and insulin resistance promote the accumulation of advanced glycation end products (AGEs), oxidative stress, mitochondrial dysfunction, and lipid peroxidation within skeletal muscle, thereby impairing muscle regeneration and contractile function.[4,7] These factors collectively impair the regenerative capacity of satellite cells the myogenic precursors responsible for muscle repair while promoting myosteatosis (fatty infiltration) and reduced capillary density.[8]

Despite its clinical significance, diabetic myopathy remains significantly under-recognized. Unlike established complications, it lacks universally standardized diagnostic criteria, often leading to its misclassification as general frailty or diabetic neuropathy.[7] Diagnosis currently relies on a composite of clinical history characterized by painless proximal muscle weakness together with standardized functional assessments, including hand-grip strength and the Timed Up and Go (TUG) test, which are recommended for evaluating muscle strength and physical performance in individuals with sarcopenia and related muscle disorders.[9] While advanced imaging like MRI can detect structural fatty replacement or rare myonecrosis, and electromyography (EMG) is essential to exclude neuropathic mimics, these tools are rarely employed for routine screening.[10] Although diabetic myopathy has increasingly been recognized as a clinically important complication of diabetes, available literature remains limited. Giha HA et al. [7] described diabetic tendino-myopathy as an under-recognized manifestation of diabetes, while Sharma V et al. [11] evaluated endocrine myopathies more broadly without specifically characterizing diabetic myopathy in Indian patients. To our knowledge, few Indian studies have systematically assessed the prevalence and clinical characteristics of diabetic myopathy using electromyographic confirmation in patients with long-standing Type 2 Diabetes Mellitus. Therefore, the present study was undertaken to address

this important knowledge gap. Consequently, there is an urgent need to quantify the prevalence of this condition to improve early intervention strategies. Therefore, the present study estimated the frequency of myopathy among T2DM patients, providing a clearer understanding of its burden and clinical presentation.

MATERIALS AND METHODS

Study Design and Setting: This cross-sectional study was conducted in the Department of Medicine at Sri Guru Ram Das Institute of Medical Sciences and Research, Amritsar, Punjab. The study protocol was approved by the Institutional Ethics Committee (SGRD/IEC/2024-363) and the investigation spanned from July 2024 to December 2025. All participants provided written informed consent in their native language prior to enrolment.

As the exact prevalence of diabetic myopathy in the target population is variable or unknown, a conservative anticipated prevalence of 50% was utilized to calculate the sample size. This proportion yields the maximum variance and ensures the study is sufficiently powered for any true underlying prevalence. Assuming a 50% prevalence, a 95% confidence level, and a strict absolute precision (margin of error) of 4.0%, the calculated minimum required sample size was 600 based on the standard proportion sample size formula.

Participant Selection: A total of 600 patients presenting to the Outpatient (OPD), Inpatient (IPD), and Emergency Departments were screened. Patients were eligible if they were over 25 years of age and had a confirmed diagnosis of Type 2 Diabetes Mellitus (T2DM) for a duration exceeding five years. T2DM was defined according to the American Diabetes Association (ADA) criteria [12]: Glycated Haemoglobin (HbA1C) $\geq 6.5\%$, Fasting Plasma Glucose (FPG) ≥ 126 mg/dl, or a 2-hour post-prandial glucose ≥ 200 mg/dl during an Oral Glucose Tolerance Test (OGTT).

Exclusion Criteria: Patients with Type 1 diabetes mellitus or gestational diabetes were excluded. Individuals receiving myotoxic medications (statins, amiodarone, fibrates, or zidovudine), those with recent musculoskeletal trauma, and patients with conditions known to affect muscle function, including electrolyte disorders, inflammatory myositis, muscular dystrophies, and endocrine or metabolic myopathies, were also excluded.

Data Collection and Clinical Assessment: Sociodemographic and clinical data were collected using a structured proforma. Body Mass Index (BMI) was calculated for all participants. Clinical examination included assessment of muscle strength, atrophy, and tenderness. Patients presenting with muscle weakness and functional limitation were evaluated for possible myopathy.

As no standardized diagnostic criteria for diabetic myopathy are currently available, diagnosis was based on the presence of clinically evident muscle weakness together

with electromyographic findings suggestive of myopathy, after exclusion of other neuromuscular, inflammatory, endocrine, metabolic, and drug-induced causes of muscle dysfunction.

Electromyographic Evaluation: Electromyography (EMG) was performed to confirm the diagnosis of myopathy. Muscles were examined for spontaneous activity at rest, including fibrillation potentials and positive sharp waves. During voluntary contraction, myopathic changes were identified by low-amplitude, short-duration motor unit action potentials with early recruitment. These findings helped distinguish myopathic from neuropathic disorders.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean $\pm$ SD, while categorical variables were presented as frequencies and percentages. Comparisons between patients with and without diabetic myopathy were performed using the independent samples t-test for continuous variables and the Chi-square test for categorical variables. Among patients with diabetic myopathy, proximal and distal involvement groups were compared using the independent samples t-test. Associations between categorical variables were assessed using the Chi-square test or Fisher's exact test, as appropriate. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 600 patients with long-standing Type 2 Diabe-

tes Mellitus were screened for eligibility during the study period. Two patients were excluded because of incomplete clinical records, resulting in 598 participants available for final analysis (Figure 1)). Among them, 85 patients (14.2%) fulfilled the predefined criteria for diabetic myopathy, while 513 patients did not demonstrate evidence of myopathy. Of the 85 diagnosed cases, 52 (61.2%) exhibited predominantly proximal muscle involvement and 33 (38.8%) showed distal involvement.

This prevalence suggests that approximately one in seven patients with chronic T2DM exhibits significant skeletal muscle involvement, a complication that remains clinically under-recognized. The demographic profile of the affected subset ($n=85$) revealed a peak prevalence in the seventh decade of life, with 41.2% of cases occurring in the 61-70 years age group. A male predominance was observed, accounting for 57.6% of the myopathic cohort. High Body Mass Index (BMI) was a significant clinical correlate, with 52.9% of patients classified as obese and 35.3% as overweight. The manifestation of myopathy was strongly associated with disease chronicity; the majority of cases occurred in patients with a T2DM duration of 16-20 years (41.2%) or 21-25 years (37.6%) (Table 1). Notably, no patient with diabetic myopathy had a diabetes duration below 10 years, despite the study inclusion criterion requiring a minimum disease duration of five years. Although patients aged ≥ 25 years were eligible for enrolment, no cases of diabetic myopathy were identified among participants younger than 40 years. Thus, all patients diagnosed with diabetic myopathy were aged ≥ 40 years.

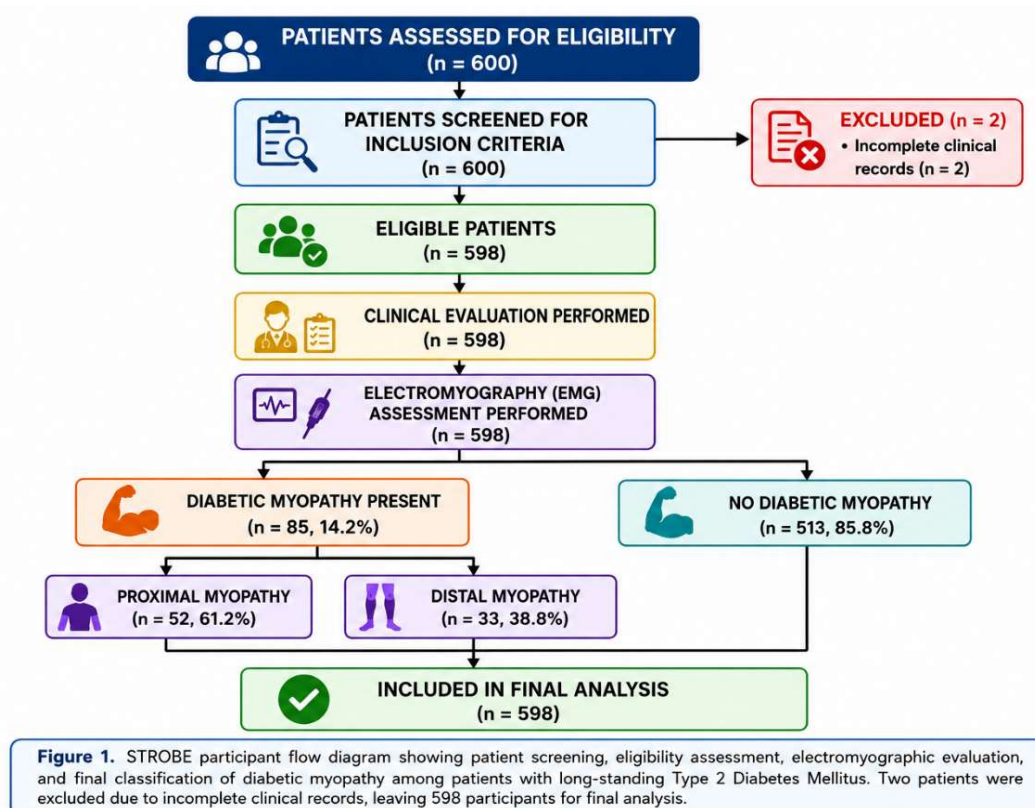


Figure 1: STROBE participant flow diagram

MI categories were defined according to the World Health Organization classification: normal ($<25 \text{ kg/m}^2$), overweight ($25\text{-}29.9 \text{ kg/m}^2$), and obesity ($\geq 30 \text{ kg/m}^2$) [13] (Table 1). All patients diagnosed with diabetic myopathy had HbA1c levels $\geq 8\%$. This finding was observed during analysis and was not part of the study inclusion criteria or diagnostic definition.

Table 1: Socio-Demographic and Clinical Characteristics of Patients with Diabetic Myopathy (n=85)

Characteristic	Cases (%)
Gender	
Male	49 (57.6)
Female	36 (42.4)
Age Cohort (Years)	
40-60	27 (31.8)
61-70	35 (41.2)
>70	23 (27.1)
Glycemic Control	
HbA1c $<8.0\%$	0 (0)
HbA1c $\geq 8.0\%$	85 (100)
BMI Status	
Normal ($<25 \text{ kg/m}^2$)	10 (11.8)
Overweight ($25\text{-}29.9 \text{ kg/m}^2$)	30 (35.3)
Obese ($\geq 30 \text{ kg/m}^2$)	45 (52.9)
Disease Duration	
10-20 Years	41 (48.3)
>20 Years	44 (51.7)

Table 2: Clinical Phenotype and Anatomical Distribution of Myopathy

Clinical Feature	Cases (%)
Primary Symptom	
Muscle Weakness	85 (100)
Anatomical Pattern	
Proximal Involvement	52 (61.2)
Distal Involvement	33 (38.8)
Associated Symptoms	
Myalgia (Pain)	5 (5.9)
Muscle Tenderness	0 (0)
EMG Confirmation	
Myopathic Potentials*	85 (100)

EMG = Electromyography

Table 3: Biochemical and Anthropometric Parameters According to Anatomical Pattern

Parameter	Proximal (n=52) Mean $\pm$ SD	Distal (n=33) Mean $\pm$ SD	p-value
BMI (kg/m^2)	30.65 $\pm$ 4.14	29.18 $\pm$ 3.86	0.102
HbA1c (%)	10.37 $\pm$ 1.18	10.25 $\pm$ 1.19	0.654
CK-NAC (U/L)	93.17 $\pm$ 42.32	85.12 $\pm$ 33.93	0.393
Duration of Diabetes (yrs)	21.12 $\pm$ 4.43	21.39 $\pm$ 5.48	0.807
Age (Years)	64.39 $\pm$ 9.28	64.39 $\pm$ 11.01	0.997

BMI=Body Mass Index; HbA1c = Glycated Hemoglobin; CK-NAC=Creatine Kinase-N-Acetylcysteine Activated; SD = Standard Deviation. p-values were calculated using the independent samples t-test.

Regarding anatomical distribution, proximal muscle involvement was more prevalent (61.2%) than distal involvement (38.8%), typically presenting as a painless functional decline (Table 2). Analysis of the symptomatic profile revealed that muscle weakness was the universal clinical hallmark, present in 100% (n=85) of the diagnosed cases. In contrast, sensory symptoms were notably rare; myalgia (muscle pain) was reported by only 5.9% (n=5) of the cohort, while the remaining 94.1% experienced a painless progression of weakness (Table 2). Furthermore, muscle tenderness was completely absent across the study group (0%), underscoring that diabetic myopathy in this population typically manifests as a non-inflammatory, painless functional decline.

The cohort demonstrated a profound degree of metabolic derangement and disease chronicity, regardless of the anatomical distribution of muscle involvement. The overall mean HbA1c was $10.33\pm 1.7\%$, and the average duration of diabetes was 21.23 ± 4.86 years, underscoring the link between long-term glycemic instability and myogenesis failure. When stratified by anatomical pattern, patients with proximal involvement exhibited a higher mean BMI ($30.65\pm 4.14 \text{ kg/m}^2$) and elevated serum CK-NAC levels ($93.17\pm 42.32 \text{ U/L}$) compared to those with distal involvement. Notably, the mean age (64.39 years) remained identical across both groups, suggesting that the clinical phenotype whether proximal or distal is likely driven by metabolic severity and disease duration rather than age-related senescence. These findings indicate that while the systemic burden of T2DM is consistent, those with proximal weakness may present with a more pronounced metabolic and biochemical profile (Table 3).

DISCUSSION

The present study demonstrated a prevalence of diabetic myopathy of 14.2% among patients with long-standing Type 2 Diabetes Mellitus (T2DM). This finding supports the growing recognition that skeletal muscle is an important target of chronic diabetic complications, alongside established microvascular manifestations such as nephropathy and retinopathy.[1,2] Patients with diabetic myopathy were significantly older than those without myopathy (64.8 ± 9.9 vs. 56.5 ± 11.8 years, $p<0.001$; Table 4). This observation is consistent with the concept that age-related sarcopenia and chronic metabolic stress may act synergistically to impair muscle structure and function in individuals with Type 2 Diabetes Mellitus.[4,5] In addition, male sex was more common among patients with diabetic myopathy than among those without myopathy (56.5% vs. 46.0%, $p=0.008$). Although previous studies have described sex-related differences in patients with Type 2 Diabetes Mellitus [14,15], evidence specifically evaluating sex distribution in diabetic myopathy remains limited.

A large proportion of patients with diabetic myopathy in the present study were overweight or obese (88.2%). As BMI was not compared between patients with and without myopathy, this finding should be interpreted as a

Table 4: Comparison of Patients with and without Diabetic Myopathy

Variable	Myopathy (n=85)	No Myopathy (n=513)	p-value
Age (years), Mean±SD	64.8 ± 9.9	56.5 ± 11.8	<0.001
Male Sex, n (%)	48 (56.5)	236 (46.0)	0.008
Female Sex, n (%)	37 (43.5)	277 (54.0)	
HbA1c (%), Mean ± SD	10.33 ± 1.17	8.35 ± 1.67	<0.001

p-value calculated using Chi-square test.

Table 5: Association Between HbA1c Category and Anatomical Pattern

HbA1c Level	Proximal (P)(%)	Distal (D)(%)	Predominated Pattern
8.2-9.5	11(55.0)	9(45.0)	P>D→ Proximal
9.6-11	28(59.6)	19(40.4)	P>D→ Proximal
>11	12(70.6)	5(29.4)	P>>D→ Strongly Proximal

Chi-square = 0.994 & p = 0.608

characteristic of the affected cohort rather than evidence of an independent association. Nevertheless, excess adiposity may contribute to skeletal muscle dysfunction through intramyocellular lipid accumulation, oxidative stress, mitochondrial impairment, and low-grade inflammation.[16,17] Furthermore, patients with diabetic myopathy exhibited significantly higher HbA1c levels than those without myopathy ($10.33 \pm 1.17\%$ vs. $8.35 \pm 1.67\%$, $p<0.001$). This finding highlights the potential role of poor glycaemic control in the development of muscle dysfunction. Chronic hyperglycaemia promotes the formation of advanced glycation end-products (AGEs), which adversely affect muscle elasticity, contractility, and regenerative capacity.[7]

The predominance of proximal muscle involvement (61.2%) observed in this study is consistent with the typical pattern described in metabolic myopathies. Clinically, most patients presented with painless muscle weakness, while myalgia was uncommon and muscle tenderness was absent. In addition, serum CK-NAC levels remained within or near the normal range despite clinically significant weakness. These findings support the concept that diabetic myopathy is primarily a metabolic rather than an inflammatory or necrotizing muscle disorder.[7,11] Comparison of patients with proximal and distal involvement revealed no significant differences in BMI, HbA1c, CK-NAC levels, duration of diabetes, or age (Table 3), suggesting that both anatomical patterns may represent different clinical manifestations of the same underlying systemic process. Similarly, although more than half of the affected patients had diabetes duration exceeding 20 years, the absence of comparative data from patients without myopathy precludes conclusions regarding disease duration as an independent risk factor. Analysis according to HbA1c categories showed a numerical predominance of proximal involvement across all glycaemic ranges; however, this trend did not reach statistical significance ($p=0.608$; Table 5). Therefore, larger

studies are required to determine whether worsening glycaemic control influences the anatomical pattern of muscle involvement. The limited relationship between CK-NAC levels and clinical severity further emphasizes the importance of comprehensive clinical assessment and electrophysiological evaluation. Routine biochemical markers alone may underestimate functional muscle impairment, and therefore should be interpreted in conjunction with clinical findings and electrophysiological studies.[18]

LIMITATIONS

The study has several limitations. Its tertiary-care setting and consecutive sampling may have introduced referral bias, with preferential inclusion of patients with more severe or longstanding disease, limiting generalisability. The lack of universally accepted diagnostic criteria for diabetic myopathy limits comparison with previous studies; diagnosis was based on clinical evaluation and electromyographic findings after excluding recognised causes of muscle weakness. Nerve conduction studies and objective functional measures were not routinely performed, so concomitant diabetic peripheral neuropathy and functional impairment may have been under-recognised. Type 2 Diabetes Mellitus was diagnosed using established clinical criteria and documented physician diagnosis. As autoimmune antibody and C-peptide testing were not routinely available, LADA cannot be completely excluded, although patients with known Type 1 Diabetes Mellitus were excluded.

FUTURE DIRECTIONS

Further studies should explore the longitudinal course of diabetic myopathy through prospective follow-up of patients with Type 2 Diabetes Mellitus. Large multicentre investigations involving diverse populations would help establish more reliable prevalence estimates and improve the external validity of findings. The use of advanced imaging modalities, including magnetic resonance imaging (MRI) and dual-energy X-ray absorptiometry (DXA), may facilitate more accurate assessment of muscle mass and composition. Future research should also aim to develop uniform diagnostic criteria and examine the effectiveness of interventions such as improved glycaemic control, structured physical activity, and rehabilitation programmes in preserving or restoring muscle function.

CONCLUSION

Diabetic myopathy was observed in a considerable proportion of patients with long-standing Type 2 Diabetes Mellitus, emphasizing its importance as a potentially under-recognized complication of diabetes. The condition was most commonly associated with painless proximal muscle weakness, poor glycaemic control, and creatine kinase levels that remained within or close to the normal range, supporting a predominantly metabolic rather than inflammatory mechanism.

Patients with diabetic myopathy were generally older and had significantly higher HbA1c levels than those without myopathy. These findings highlight the need for careful evaluation of unexplained muscle weakness in individuals with diabetes. Early identification and appropriate management, including optimization of glycaemic control, may help reduce disability and improve functional outcomes.

Individual Author's Contribution: PS conceived and coordinated the study, supervised data collection, performed the statistical analysis, and prepared the initial manuscript draft. RKS and DK contributed to the study design, patient recruitment, and data acquisition. APS assisted with methodological planning and study implementation. PKG contributed to data interpretation, critical revision of the manuscript, and preparation of the final version. All authors reviewed and approved the final manuscript.

Availability of data: The data that support the findings of this study are available from the corresponding author on reasonable request.

Declaration of Non-use of generative AI Tools: This article was prepared without the use of generative AI tools for content creation, analysis, or data generation. All findings and interpretations are based solely on the authors' independent work and expertise.

REFERENCES

- Viigimaa M, Sachinidis A, Toumpourleka M, Koutsampasopoulos K, Alliksoo S, Titma T. Macrovascular Complications of Type 2 Diabetes Mellitus. *Curr Vasc Pharmacol*. 2020;18(2):110-116. DOI: <https://doi.org/10.2174/1570161117666190405165151> PMID:30961498
- Goldney J, Sargeant JA, Davies MJ. Incretins and microvascular complications of diabetes: neuropathy, nephropathy, retinopathy and microangiopathy. *Diabetologia*. 2023;66(10):1832-1845. DOI: <https://doi.org/10.1007/s00125-023-05988-3> PMID:37597048 PMCID:PMC10474214
- Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N, et al. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas, 9th edition. *Diabetes Res Clin Pract*. 2019;157:107843. DOI: <https://doi.org/10.1016/j.diabres.2019.107843> PMID:31518657
- D'Souza DM, Al-Sajee D, Hawke TJ. Diabetic myopathy: impact of diabetes mellitus on skeletal muscle progenitor cells. *Front Physiol*. 2013 Dec 20;4:379. DOI: <https://doi.org/10.3389/fphys.2013.00379> PMID:24391596 PMCID:PMC3868943
- Nishikawa H, Asai A, Fukunishi S, Nishiguchi S, Higuchi K. Metabolic Syndrome and Sarcopenia. *Nutrients*. 2021 Oct 7;13(10):3519. DOI: <https://doi.org/10.3390/nu13103519> PMID:34684520 PMCID:PMC8541622
- Horton WB, Barrett EJ. Microvascular Dysfunction in Diabetes Mellitus and Cardiometabolic Disease. *Endocr Rev*. 2021 Jan 28;42(1):29-55. DOI: <https://doi.org/10.1210/endrev/bnaa025> PMID:33125468 PMCID:PMC7846151
- Giha HA, Sater MS, Alamin OA. Diabetes mellitus tendino-myopathy: epidemiology, clinical features, diagnosis and management of an overlooked diabetic complication. *Acta diabetologica*. 2022;59(7):871-883. DOI: <https://doi.org/10.1007/s00592-022-01860-9> PMID:35291027
- Careccia G, Mangiavini L, Cirillo F. Regulation of Satellite Cells Functions during Skeletal Muscle Regeneration: A Critical Step in Physiological and Pathological Conditions. *Int J Mol Sci*. 2024; 25(1):512. DOI: <https://doi.org/10.3390/ijms25010512> PMID:38203683 PMCID:PMC10778731
- Chen LK, Woo J, Assantachai P, Auyeung TW, Chou MY, Iijima K, et al. Asian Working Group for Sarcopenia: 2019 Consensus Update on Sarcopenia Diagnosis and Treatment. *J Am Med Dir Assoc*. 2020;21(3):300-307.e2. DOI: <https://doi.org/10.1016/j.jamda.2019.12.012> PMID:32033882
- Hannaford A, Paling E, Silsby M, Vincenten S, van Alfen N, Simon NG. Electrodiagnostic studies and new diagnostic modalities for evaluation of peripheral nerve disorders. *Muscle Nerve*. 2024;69(6):653-669. DOI: <https://doi.org/10.1002/mus.28068> PMID:38433118
- Sharma V, Borah P, Basumatary LJ, Das M, Goswami M, Kayal AK. Myopathies of endocrine disorders: A prospective clinical and biochemical study. *Ann Indian Acad Neurol*. 2014;17(3):298-302. DOI: <https://doi.org/10.4103/0972-2327.138505> PMID:25221399 PMCID:PMC4162016
- American Diabetes Association Professional Practice Committee. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2022. *Diabetes Care*. 2022 Jan 1;45(Suppl 1):S17-S38. DOI: <https://doi.org/10.2337/dc22-S002> PMID:34964875
- Obesity: preventing and managing the global epidemic. Report of a WHO consultation. *World Health Organ Tech Rep Ser*. 2000;894:i-xii, 1-253. PMID: 11234459.
- Al-Mukhtar SB, Fadhil NN, Hanna BE. General and gender characteristics of type 2 diabetes mellitus among the younger and older age groups. *Oman Med J*. 2012;27(5):375-382. DOI: <https://doi.org/10.5001/omj.2012.94> PMID:23074547 PMCID:PMC3472577
- Kautzky-Willer A, Harreiter J, Pacini G. Sex and Gender Differences in Risk, Pathophysiology and Complications of Type 2 Diabetes Mellitus. *Endocr Rev*. 2016;37(3):278-316. DOI: <https://doi.org/10.1210/er.2015-1137> PMID:27159875 PMCID:PMC4890267
- Razi O, de Moraes C, Zamani N, Saeidi A, Hadjicharalambous M, Hackney AC, et al. From Obesity to Muscle Insulin Resistance: The Mediating Roles of Intramyocellular Lipids, Inflammation, and Oxidative Stress. *Diabetes Metab Res Rev*. 2025 Oct;41(7):e70094. DOI: <https://doi.org/10.1002/dmrr.70094> PMID:41057221
- Chen M, Wang Y, Deng S, Lian Z, Yu K. Skeletal muscle oxidative stress and inflammation in aging: Focus on antioxidant and anti-inflammatory therapy. *Front Cell Dev Biol*. 2022;10:964130. DOI: <https://doi.org/10.3389/fcell.2022.964130> PMID:36111339 PMCID:PMC9470179
- Giannini M, Charles AL, Evrard C, Blaess J, Bouchard-Marmen M, Debrut L, et al. Sarcopenia assessed by DXA and hand-grip dynamometer: a potential marker of damage, disability and myokines imbalance in inflammatory myopathies. *Rheumatology*. 2024;63(9):2503-2514. DOI: <https://doi.org/10.1093/rheumatology/keae207> PMID:38544289