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THE MUSCULOSKELETAL COMPLICATIONS OF DIABETES MELLITUS

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ABSTRACT

Diabetes mellitus (DM) is a progressive metabolic disorder with increasing global prevalence and a wide variety of complications. While clinical attention focuses mostly on its vascular pathologies, musculoskeletal disorders remain under-recognized and poorly managed. This review provides an overview of musculoskeletal complications associated with both type 1 and 2 diabetes, including bone fragility, Charcot neuroarthropathy, diffuse idiopathic skeletal hyperostosis, osteoarthritis, adhesive capsulitis, diabetic amyotrophy, and diabetic muscle infarction. The pathophysiology of these conditions is complex, involving impaired glucose levels, insulin resistance, accumulation of advanced glycation end-products, chronic inflammation process, and vascular changes. Preventive strategies, early diagnosis, and adjusted management are crucial to mitigate the impact of these complications on diabetic patients, their mobility, and quality of life. This article aims to highlight the importance of musculoskeletal complications and raise awareness of the significance of integrated management of diabetes.

Objective: This review aims to describe several musculoskeletal complications of diabetes mellitus.

Materials and Methods: A review of the available literature from the PubMed, Scopus, and Web of Science databases published within the last 20 years.

KEYWORDS

Diabetes Mellitus, Diabetes Complications, Skeletal Complications, Musculoskeletal Complications

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1. Introduction

Diabetes mellitus (DM) is a progressive metabolic disorder characterized by elevated blood glucose levels, resulting from either inadequate insulin production or impaired insulin action. Its prevalence has increased dramatically over the last decades from 630 million adults worldwide living with diabetes in 1990 to approximately 828 million in 2022 and has become a significant global public health concern [1,2].

The classification of diabetes includes two main types, gestational diabetes, hybrid forms, and other special forms [3]. This classification system consider a range of factors that typically vary between individuals with type 1 and type 2 diabetes, including age at onset, level of insulin resistance (IR), presence of metabolic syndrome (MS), extent of pancreatic β-cell dysfunction, presence of autoantibodies related to β-cell destruction, blood C-peptide concentrations, and the necessity of insulin therapy for survival (Table 1) [4].

Effective management of diabetes requires a multipronged approach. Lifestyle modifications have a significant impact both on the prevention of the disease and on further management, so patient education is a foundational layer. The primary treatment strategy for type 1 diabetes typically involves frequent self-monitoring of blood glucose levels combined with multiple daily subcutaneous insulin injections [5]. Type 2 diabetes is usually managed by monotherapy or combinatorial drug therapies including insulin secretagogues, biguanides, insulin sensitizers, alpha-glucosidase inhibitors, incretin mimetics, amylin antagonists, and sodium-glucose co-transporter-2 (SGLT2) inhibitors [6].

Impaired glucose levels in diabetes predispose to a broad spectrum of complications. Neurophathy, retinopathy, and kidney disease are among the most common vascular complications seen in both chronic type 1 diabetes and in patients with either newly diagnosed or existing type 2 diabetes [7,8]. Compared to life-threatening microvascular and macrovascular complications, joint, bone, and muscle disorders in individuals with diabetes have been relatively under-recognized. In this article, we aim to depict possible musculoskeletal complications in patients with diabetes.

Table 1. Differences between type 1 and type 2 diabetes [4]

Feature	Type 1 Diabetes (T1D)	Type 2 Diabetes (T2D)
Age at onset	Typically early onset; younger individuals	Usually older age at onset
Level of insulin resistance (IR)	Generally low IR	High IR
Presence of metabolic syndrome (MS)	Rare in classical T1D	Common; often associated with MS
Extent of pancreatic β-cell dysfunction	Severe; near-total β-cell loss; absolute insulin deficiency	Progressive β-cell dysfunction secondary to IR
Presence of autoantibodies	Present (markers of autoimmune β-cell destruction)	Absent
Blood C-peptide concentrations	Very low or undetectable	Preserved or relatively higher levels
Necessity of insulin therapy for survival	Immediate and lifelong insulin requirement	Not required initially; may need insulin later as disease progresses

2. Methods

A literature search was conducted using the medical database PubMed, Google Scholar and National Library of Medicine. Articles were retrieved using the keywords: „diabetes mellitus", „diabetes complications", „musculoskeletal complications", „skeletal complications" in appropriate configurations.

3. Results

3.1 Skeletal and joint complications

3.1.1. Bone fragility

Impaired glucose level affects bone metabolism, causing cellular abnormalities, matrix interactions, immune and microvascular changes, and musculoskeletal maladaptation leading to increased bone fragility [9,10].

In individuals with type 1 diabetes, both bone mass and bone strength reduction lead to as much as a fivefold increase in lifetime fracture risk [9]. In contrast, type 2 diabetes is associated with an elevated fracture risk despite normal bone mass [9].

The pathophysiology is multifaceted and complicated. In type 1 diabetes, low levels of insulin-like growth factor (IGF-1) and the insulin-like growth factor-binding protein 3 (IGFBP-3) lead to diminished function of osteoblasts, resulting in apparent osteoporosis, while in type 2 diabetes mellitus advanced glycation end products (AGEs) are key mediators of bone metabolism disturbances through inducing the receptor expression of osteoclasts [10,11].

Guidelines for diabetes patients with reduced bone density aren't specific; fall-prevention and pharmacological management are the same as for osteoporotic patients [10]. An adequate intake of protein, calcium, and vitamin D combined with physical activity should be advised [9]. Glucose-lowering medications with neutral/beneficial skeletal effects should help in reducing the risk of bone fractures [12].

3.1.2. Charcot's neuroarthropathy

Charcot neuroarthropathy (CN) is a chronic, destructive condition affecting bones, joints, and soft tissues, mostly in patients with peripheral neuropathy, characterized by a sterile inflammatory process accompanied by painful or painless damage of limbs [13,14]. CN is most commonly caused by diabetes mellitus, affects approximately 0.1% to 0.4% of individuals with diabetes, and, based on various studies, from 29% to 35% of patients with peripheral neuropathy [13,15].

CN is characterized by progressive neuropathy, deformity, and joint destruction, which can lead to significant morbidity and functional impairment [14,16]. If not treated adequately, it may progress to repeated fractures and dislocations, ultimately resulting in severely deformed joints [14,16]. While the foot is the most affected joint in individuals with diabetes-related Charcot neuroarthropathy, other sites such as the knee, wrist, hip, and spine have also been reported [13].

Classification or staging of CN can be based on the anatomical location of the damage- Sanders-Frykberg classification, the common affected regions - Brodsky-Trepman classification, and the clinical and radiological signs- Eichenholtz (Table 2). The last one is the most used in literature and is helpful in diagnosis and selection of appropriate treatment [13,17].

Table 2. The modified Eischenscholtz classification [10,17]

Stage	Clinical finding	Radiological findings
0 (prodromal)	Warmth, swelling, erythema	Normal radiograph
I (development)	Warmth, swelling, erythema, ligament laxity	Osteopenia, fragmentation, joint subluxation or dislocation
II (coalescence)	Decreased warmth and swelling, decreased erythema	Absorption of debris, sclerosis, fusion of larger fragments
III (reconstruction)	Absence of warmth and swelling, joint deformity	Consolidation of deformity, joint arthrosis, fibrous ankyloses, rounding and smoothing of bone fragments

Charcot neuroarthropathy should be considered in patients with longstanding diabetes and peripheral neuropathy who present with unilateral warmth, edema, and erythema of the foot or ankle [10,17]. A thorough differential diagnosis is essential to exclude other potential causes, including cellulitis, septic arthritis, osteomyelitis, gout, osteoarthritis, and rheumatoid arthritis [10,17].

The first-line treatment of Charcot neuroarthropathy is early and prolonged off-loading—typically with a cast or removable devices —to reduce inflammation, prevent bone necrosis and deformity, and allow for osseous healing [11,13,14]. Immobilization should be maintained until clinical signs of inflammation resolve, particularly localized edema and temperature differences compared to the contralateral limb [11,13,14]. In some cases, CN can be managed surgically [18].

3.1.3. Diffuse idiopathic skeletal hyperostosis (DISH)

DISH is a condition characterized by calcification of the spine and peripheral entheses primarily affecting the anterior longitudinal ligament [11,19]. Recent studies have shown the connection between DISH and diabetes, with 5-50% higher prevalence than in the general population [10]. Radiological findings include ossifications along the anterior side of the spine, involving at least 4 vertebral bodies [20]. Most commonly, DISH is localized in the thoracic spine [19]. Clinically, DISH can manifest as back pain, stiffness, functional impairment, dysphagia, or neurological deficits, but it can also be asymptomatic [10,19,20]. Management strategies include activity modification, physiotherapy or bracing, nonsteroidal anti-inflammatory drugs (NSAIDs), and bisphosphonates [19].

3.1.4. Osteoarthitis (OA)

OA is a major cause of global disability, mostly affecting the hip and knee joints [21,22]. Although established risk factors for the development and progression of osteoarthritis include advanced age, female sex, history of joint trauma, and obesity, numerous studies have also reported a significant association between osteoarthritis and diabetes [21,23]. Glucose impairment and chronic hyperglycemic state contribute to cartilage destruction and joint inflammation through increased oxidative stress and the accumulation of AGEs in the joint, leading to matrix stiffening, impaired mechanical cushioning, and enhanced inflammatory responses [21,23]. OA manifests clinically as joint pain, difficulty with physical activity, and restricted range of motion [22]. Non-pharmacological management, such as physiotherapy and body weight reduction, should be advised before pharmacological or surgical treatment [22]. Pharmacological treatment will vary between hip and knee OA, but includes NSAIDs, joint injections, and duloxetine [22].

3.1.5. Adhesive capsulitis (AC)

AC, also known as frozen shoulder, is characterized by inflammation and fibrosis of the glenohumeral joint, resulting in pain and restriction of active and passive movement [24,25]. While primary AC is idiopathic, secondary AC is associated with diabetes, with an estimated prevalence from 10,8% to 30% [25]. Potential pathophysiology paths in DM include joint AGEs accumulation, chronic release of inflammatory markers such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), and increased secretion of interleukin-13 (IL-13) with the following collagen and matrix accumulation, collagen linking, and synovial and connective tissue fibrosis [25]. Recently, clinical investigation and ultrasonography of the suspected joint are essential tools in the diagnosis [26]. Early management with NSAIDs, physiotherapy, and joint injections may inhibit the development of AC [11]. The potential therapeutic options for AC are presented in Figure 1.

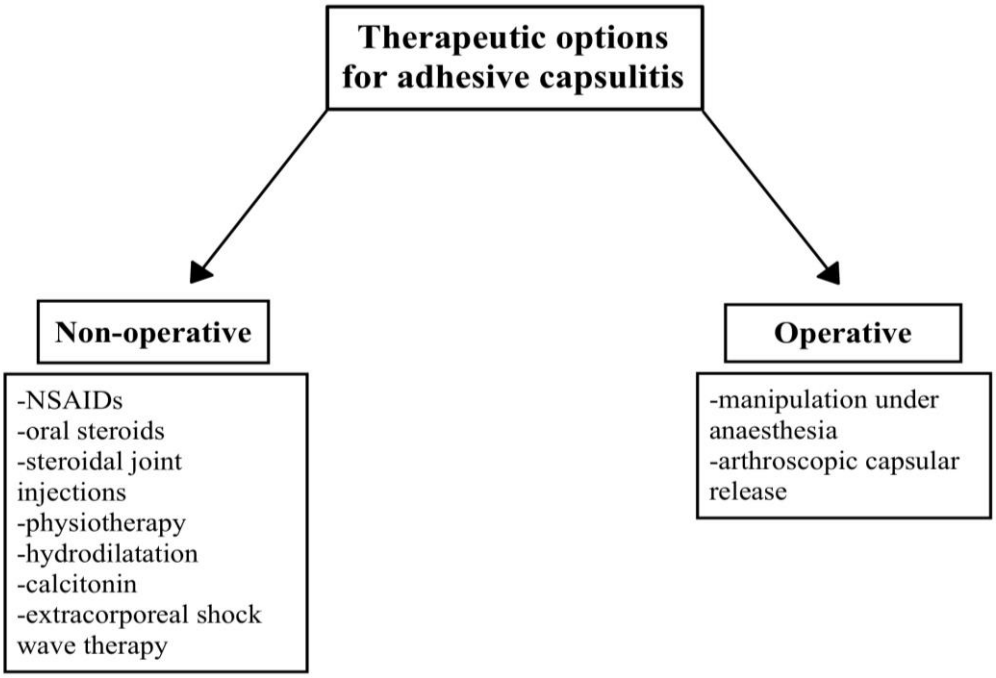

Fig. 1. *Therapeutic options for adhesive capsulitis [26]*

3.2. Muscular complications

3.2.1. Diabetic amyothrophy (DA)

DA, also known as diabetic sarcopenia, is a progressive condition characterized by the decrease of muscle mass (MM) following the loss of its function and increased risk of falling and physical disability [10,27]. Diabetes is an independent factor for sarcopenia, with prevalence reaching to even 29,3 % in patients with type 2 diabetes [27,28]. Various pathophysiological pathways are responsible for MM in DM. Insulin is an anabolic hormone, so its impaired function and increased insulin-resistance may contribute to a reduction in protein synthesis and escalated protein degradation [10,28]. Additionally, the accumulation of AGEs in muscle leads to a decrease in strength and power [28]. Macrovascular and microvascular complications of DM, like kidney disease, neuropathy, and vascular changes, also contribute to the loss of muscle function through increased MM loss, muscle ischemia, and reduced physical activity [28]. Treatment of DS is multi-directional, including controlled glucose levels, an appropriate and patient-adjusted diet with adequate energy intake, supplementation of vitamin D, and muscle-building exercises [27].

3.2.2. Diabetic muscle infarction (DMI)

DMI, otherwise termed as diabetic myonecrosis, is an uncommon complication of long-standing diabetes, characterized by spontaneous muscle necrosis that occurs without arterial thrombosis or blockage of a large artery [10,29]. Thighs and calf muscles are generally affected, manifesting clinically as muscle pain and localized swelling, without signs of infection with possible accompanying erythema above the changed muscle [10,29,30]. Patients report no history of trauma or fever [30]. Laboratory studies can reveal elevated levels of creatine kinase, as well as leukocytes, aspartate transaminase, and lactic dehydrogenase, C-reactive

protein, and increased erythrocyte sedimentation rate [10,29,31]. Radiological studies may aid in diagnosis, but in some cases, a muscle biopsy may be required [10]. Analgesics, optimized glycemic control, and low-dose aspirin are possible treatments, sometimes combined with temporary immobilization [29].

4. Discussion

This review highlights commonly under-recognized musculoskeletal complications of diabetes mellitus, drawing attention to the broad variety of muscular and skeletal pathologies that can significantly decrease patients' mobility. Microvascular and macrovascular complications are the primary clinical focus, while the increasing prevalence of musculoskeletal disorders should raise awareness of the significance of integrated management of diabetes.

Diabetes affects bone metabolism, leading to increased bone fragility in both type 1 and type 2 diabetes. Pathophysiology is complex; in T1D, osteoporosis is primarily caused by impaired osteoblast activity through insulin deficiency, whereas in T2D, advanced glycation end-products induce elevated expression of osteoclast receptors.

Additionally, in T2D, normal bone mass may mask the increased risk of fractures. Fall-prevention strategies and tailored pharmacological treatment, including diabetes medications with neutral/beneficial skeletal effects, should be applied.

Charcot neuroarthropathy is another destructive condition that may affect patients with diabetes, especially those with peripheral neuropathy. The Eichenholtz staging remains a valuable tool in clinical assessment and treatment planning. CN's subtle onset, possible painless course, and potential for irreversible joint deformity should highlight the importance of early diagnosis and intervention.

Diffuse idiopathic skeletal hyperostosis (DISH) and osteoarthritis (OA) also have a higher prevalence in the diabetic population. DISH is characterized by spinal ossifications that may lead to limited mobility, while OA pathogenesis involves cartilage damage through AGEs accumulation and oxidative stress, which manifests asjoint pain and limited range of motion. Both conditions' treatment includes physiotherapy and pharmacological treatment.

Adhesive capsulitis (AC) is particularly common in diabetes. AGEs accumulation, accompanying by chronic inflammation, leads to joint fibrosis. Early diagnosis using imaging studies and early intervention with physiotherapy and pharmacologic treatment are essential to preserve joint mobility.

Muscular complications of diabetes, such as amyotrophy and diabetic muscle infraction, reflect the nature of diabetes-related tissue damage. The pathogenesis of diabetic sarcopenia is multifaceted, involving insulin resistance, microvascular changes, and AGEs accumulation. These findings emphasize the need for a multi-directional management plan including good glycemic control, proper diet, and muscle-building exercises. Diabetic muscle infarction, while rare, presents with swelling and acute pain, requiring increased clinical awareness for accurate diagnosis and appropriate treatment.

Overall, the musculoskeletal complications of diabetes represent a significant clinical burden that is often insufficiently addressed in diabetes management. Increased clinical awareness, prompt diagnosis, and adjusted treatment could significantly mitigate these complications and increase patients' quality of life.

5. Conclusions

Musculoskeletal complications in diabetes mellitus are prevalent, various, and frequently underdiagnosed, even though they may remarkably decrease long-term outcomes for patients. This review aims to highlight the necessity for clinicians to treat diabetes holistically, diagnose and address musculoskeletal pathologies along with micro- and macro-vascular complications. Proper patient' education accompanied by prompt, multidisciplinary interventions is significant in maintaining physical mobility.

Diabetes is a significant clinical burden globally, so further research is needed to establish clinical guidelines for the diagnosis and treatment of musculoskeletal complications in this population.

Disclosures Author's contribution
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