Sarcouncil Journal of Medicine and Surgery

ISSN(Online): 2945-3534

Volume- 03| Issue- 02| 2024

Research Article **Received:** 02-01-2024 | **Accepted:** 19-01-2024 | **Published:** 04-02-2024

Diabetic Spectrum Patients in Orthopaedic Unit

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Abstract: Aims of the study: to investigate the prevalence of musculoskeletal disorders in patient with diabetes mellitus with the aid of the (Spectrum of diabetic patients in orthopaedic unit) questionnaire, and to compare the results with results previously published from studies performed in other populations. **Methods:** This is a clinical descriptive study which hade been carried on 200 diabetic patients, 79 male , and 121 female with different musculoskeletal manifestations due to long term poorly controlled diabetes mellitus. I divide the patients according to the age range, 8 patients (20-29 years), 15 patients(30-39years), 34 patients(40-49 years), 65 patients(50-59), 54 patients(60-69 years) and 24 patients from 70 years and above. Those randomized patients have different types of diabetes mellitus, 21 patients have IDDM, 178 patients have NIDDM, one patient have MODY and 0 patient have GD.26 patients have good control of diabetes and 174 have bad control. 59 patients have oral hypoglycemic agents as treatment of diabetes mellitus, 75 have insulin therapy, 64 patients have both insulin and oral hypoglycemic agents and 2 patients have just diet control. Those randomized patients comes from Al basrah general hospital, Alsader teaching hospital in Basrah, Almoane hospital in Basrah and Alhusain teaching hospital in karbela from both medical and orthopaedic department, 157 outpatients and 43 inpatients. **Result:** I found different orthopaedic complaints in different age groups both male and female. All this associated with long term and poorly control diabetes mellitus.In the upper limb 32 patients have frozen shoulder, 3 patients Reflex sympathetic dystrophy, 23 patients have carpal tunnel syndrom, 19 patients have trigger finger, 4 patients have olecranon bursitis, 16 patients have cheiroarthropathy, 24 patients have De-quervens disease, 11 patients have tennis elebow and 26 patients have dupuytrens contracture. In the lower limb 11 patients have trochantric bursitis, 23 patients have charcots joint, 64 patients have diabetic foot, 15 patients have plantar fasciitis, 3 patients have tarsal tunnel syndrom and 6 patients have Foot deformities. IN the spine 26 patients have Diffuse idiopathic skeletal hyperosteosis.I found also others orhopaedic complains, 24 patients have muscle cramp, 39 patients have Osteoporosis, 32 patients have Osteomylitis, 5 patients have Septic arthritis, 5 patients have Gout and psudogout, 85 patients have peripheral neuropathy, 53 patients have vascular complication and 5 patients have myopathy.

Keywords: Diabetic, Spectrum, Orthopaedic unit.

INTRODUCTION

Diabetes mellitus is a multisystem disease characterized by persistent hyperglycemia that has both acute and chronic biochemical and anatomical sequelae, with type-2 DM representing the most common form of the disease.(1) It is thought to affect 17 million Americans, only 11 million of whom have been diagnosed according to the American Diabetes Association.(1) In type 1 diabetes, a lack of insulin results in poor carbohydrate, fat, and protein metabolism. Insulin is functionally absent, typically due to immune-mediated destruction of the beta cells of the pancreas, though other etiologies of beta cell destruction have also been implicated, including drugs, chemicals, viruses, mitochondrial gene defects, pancreatectomy and ionizing radiation.(1) Type 1 DM occurs most commonly in juveniles. It can occur in adults, especially in those in their late 30s and early 40s. Unlike people with type 2 DM those with Type 1 DM are usually not obese and they may initially present to the clinician in physiologic crises with diabetic ketoacidosis.(1) Symptoms typically do not become apparent until 80-85% of the beta cells have been lost. Although diabetic concordance among first degree relatives is relatively low (6-10%), there does appear to be a genetic disposition toward diabetes mellitus type II, mainly determined by genes in the major histocompatibility complex (i.e. human leukocyte antigen (HLA) region located on the short arm of chromosome 6.(2) Type II DM represents approximately 90% of all cases of diabetes. It usually occurs in older overweight individuals. While the primary defect may be insulin resistance, many of these patients also have poor insulin production, particularly for their level of glycemia. There is a suggested genetic predisposition as well, and the prevalence varies widely by ethnicity, from a high of 18 % among Native Americans and Alaska natives to a low of approximately 7% among non- Hispanic Caucasians.(3) Many patients with DM II will ultimately require insulin treatment for good glycemic control.(3) Presumably, the defects of type II diabetes mellitus occur in patients who live adiabetogenic lifestyle. Excessive caloric intake, inadequate caloric expenditure, and obesity are suspected to be superimposed upon a susceptible genotype.

Types of DM:

There are four basic categories within the American Diabetes Associations classification system for DM.(3) These are: Type I DM, Type II DM, Gestational DM(GDM) and other specific types. It should be noted that the terms (insuline-

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DOI- https://doi.org/10.5281/zenodo.10615772

dependent DM) and (non-insuline dependent DM) have been eliminated because of confusion created by these terms. These terms focus on the treatment of DM rather than the etiology of the disease. Pre-diabetes, a condition between normoglycemia and diabetes is also recognized. These patients typically have normal or near normal glucose levels, but with high levels of circulating insulin and relative insulin-resistance.(4)

Type I (DM): (B-cell destruction, usually leading to absolute insulin deficiency) Immune-mediated diabetes. This form of diabetes, which accounts for only 5–10% of those with diabetes, previously encompassed by the terms insulindependent diabetes, type 1 diabetes, or juvenile-onset diabetes, results from a cellular- mediated autoimmune destruction of the cells of the pancreas. Markers of the immune destruction of the cell include islet cell autoantibodies, autoantibodies to insulin, autoantibodies to GAD (GAD65), and autoantibodies to the tyrosine phosphatases IA-2 and IA-2. One and usually more of these autoantibodies are present in 85–90% of individuals.These patients are also prone to other autoimmune disorders such as Graves' disease, Hashimoto's thyroiditis, Addison's disease, vitiligo, celiac sprue autoimmune hepatitis, myasthenia gravis, and pernicious anemia. Idiopathic diabetes, Some forms of type1 diabetes have no known etiologies. Some of these patients have permanent insulinopenia and are prone to ketoacidosis but have no evidence of autoimmunity.

Type II (DM): Type 2 diabetes (ranging from predominantly insulin resistance with relative insulin deficiency to predominantly an insulin secretory defect with insulin resistance) This form of diabetes, which accounts for 90–95% of those with diabetes, previously referred to as non–insulindependent diabetes, type 2 diabetes, or adult-onsetdiabetes, encompasses individuals who have insulin resistance and usually have relative (rather than absolute) insulin deficiency At least initially, and often throughout their lifetime, these individuals do not need insulin treatment to survive.

Most patients with this form of diabetes are obese, and obesity itself causes some degree of insulin resistance. Patients who are not obese by traditional weight criteria may have an increased percentage of body fat distributed predominantly in the abdominal region. Ketoacidosis seldom occurs spontaneously in this type of diabetes; when seen, it usually arises in association with the stress of another illness such as infection. This form of diabetes frequently goes undiagnosed for many years because the hyperglycemiadevelops gradually and at earlier stages is often not severe enough for the patient to notice any of the classic symptoms of diabetes.Other specific types of diabetesGenetic defects of thecell. Several forms of diabetes are associated with monogenetic defects in cell function. These forms of diabetes are frequently characterized by onset of hyperglycemiaat an early age (generally before age 25 years). They are referred to as maturityonset diabetes of the young (MODY) and are characterized by impaired insulin secretion with minimal or no defects in insulin action. They are inherited in an autosomal dominant pattern. Abnormalities at six genetic loci on different chromosomes have been identified to date. The most common form is associated with mutations on chromosome 12 in a hepatic transcription factor referred to as hepatocyte nuclear factor (HNF)-1. A second form is associated with mutations in the glucokinase gene on chromosome7p and results in a defective glucokinase molecule.(3,4)

DIAGNOSTIC CRITERIA FOR DIABETES MELLITUS:

Diagnosis of diabetes has been based on glucose criteria, either the FPG or the 75-g OGTT. Additionally patients with severe hyperglycemia such as those who present with severe classic hyperglycemic symptoms or hyperglycemic crisis can continue to be diagnosed when a random (or casual) plasma glucose of 200 mg/dl (11.1 mmol/l) is found.(4) It is likely that in such cases the health care professional would also measure an HbA1C test as part of the initial assessment of the severity of the diabetes and that it would (in most cases) be above the diagnostic cut point for diabetes. Further research is needed to better characterize those patients whose glycemic status might be categorized differently by two different tests (e.g., FPG and HbA1C), obtained in close temporal approximation Such discordance may arise from measurement variability, change over time, or because HbA1C, FPG, and postchallenge glucose each measure different physiological processes. In the setting of an elevated HbA1C but "nondiabetic" FPG, the likelihood of greater postprandial glucose levels or increased glycation rates for a given degree of hyperglycemia may be present. In the opposite scenario (high FPG yet HbA1C below the diabetes cut point), augmented hepatic glucose production or reduced glycation

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rates may be present. It is preferable that the same test be repeated for confirmation, since there will be a greater likelihood of concurrence in this case. For example, if the HbA1C is 7.0% and a repeat result is 6.8%, the diagnosis of diabetes is confirmed. However, there are scenarios in which results of two different tests (e.g., FPG and HbA1C) are available for the same patient. In this situation, if the two different tests are both above the diagnostic thresholds, the diagnosis of diabetes is confirmed. On the other hand, when two different tests are available in an individual and the results are discordant, the test whose result is above the diagnostic cut point should be repeated, and the diagnosis is made on the basis of the confirmed test. That is, if a patient meets the diabetes criterion of the HbA1C (two results 6.5%) but not the FPG (126 mg/dl or 7.0 mmol/l), or vice versa, that person should be considered to have diabetes. The healthcare professional might opt to follow the patient closely and repeat the testing in 3– 6 months. The current diagnostic criteria for diabetes are: (4)

1.HbA1C 6.5%.*
2. FPG 126 mg/dl (7.0 mmol/l). Fasting is defined as no caloric intake for at least 8 h.*
3. 2-h plasma glucose 200 mg/dl (11.1 mmol/l) during an OGTT. The test should be performed as described by the World Health Organization, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.*
4. In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose 200 mg/dl (11.1 mmol/l).

*In the absence of unequivocal hyperglycemia, criteria 1–3 should be confirmed by repeat testing

Treatment of Diabetes Mellitus
In the United States, 57.9% of diabetic patients have 1 or more diabetes-related complication, and 14.3% have 3 or more.(5) Strict glycemic control reduces the development and progression of microvascular complications, such as retinopathy, nephropathy, and neuropathy. Aggressive treatment of dyslipidemia and hypertension decreases macrovascular complications.(6,7,10)

Glycemic Control:
The primary techniques available to assess the quality of a patient's glycemic control are self-monitoring of blood glucose (SMBG) and interval measurement of hemoglobin A_{1c} (HbA_{1c}).

Self-Monitoring of Blood Glucose:
SMBG is an effective way to evaluate short-term glycemic control. It helps patients and physicians assess the effect of food, medications, stress, and activity on blood glucose levels. The frequency of checking depends on the type of medical therapy, risk for hypoglycemia, and need for short-term adjustment of therapy.For patients with type 1 diabetes mellitus (T1DM) and insulin-dependent type 2 diabetes (T2DM) patients, clinical trials have demonstrated that SMBG plays a role in effective glycemic control because it helps to refine and adjust insulin doses by monitoring for and preventing asymptomatic hypoglycemia and preprandial and postprandial hyperglycemia.(6,8,9,11)

Hemoglobin A_{1c}:
HbA_{1c} measures nonreversible glycosylation of the hemoglobin molecule, which is directly related to blood glucose concentrations. It reflects a mean of the patient's blood glucose values over a 2- to 3-month period and can be used as a predictor of a patient's risk of microvascular complications.(14)

Pharmacologic Treatment:
When considering appropriate pharmacologic therapy, a major factor to consider is whether the patient is insulin deficient, insulin resistant, or both.

Insulin sensitizer: Biguanides (metformin), Thiazolidinediones
Insulin secretagogues: Sulfonylureas, Glinides, Alphaglucosidase inhibitors

Insulin:
Insulin is the oldest medical therapy available for diabetes. It was discovered in 1921 and clinical testing in humans began in 1922. Today it remains the most direct method of reducing hyperglycemia. There is no upper limit in dosing for therapeutic effect, so it can be used to bring any HbA_{1c} down to near normal levels. Other benefits of insulin include its effects on reducing triglycerides levels and increasing high density lipoprotein(HDL). Hypoglycemia is a concern, but the actual risk of severe episodes is relatively small. Studies have shown that episodes where the patient required assistance due to hypoglycemia occurred in 1 to 3 patients per 100,000 patient-years. Weight gain can occur after initiation and is typically about 2 kg to 4 kg. (8,12,13,15,16,17,18)

Orthopaedic manifestation of diabetes mellitus:
Patients with diabetes mellitus have several rheumatic syndromes, many of which are associated with the severity of the disease. These

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include diabetic neuropathy and its associated arthropathy. The pathogenesis is neurovascularinstead of the generally thought neurotraumatic (resulting from decreased sensitivity of nerve endings) or reduced flow secondary to arterial sclerosis of small vessels.(19) Patients with diabetes have increase blood flow (secondary to neuropathy involving the sympathetic nervous system) to subchondral bone, resulting in increased osteoclastic activity and bone resorption. This occurs even in the absence of peripheral vascular disease, resulting in bone fatigue and disorganization. It appears radiographically as progression from localized osteopenia to osteolysis of subchondral bone, fragmentation of bone and cartilage (part of which may be come embedded in synovial tissue) and sclerosis.(20) Joints involved in order of frequency include ankle, metatarsophalangeal and tarsometatarsal. This distribution differentiates diabetic arthropathy from tabesdosalis in which the knee is more commonly involved. The pathogenic mechanism which may be active in diabetes mellitus causing tissue damage is unknown; however, advanced glycation end products bind to RAGE receptors (increased in diabetes and thought to be responsible for inflammation and increased atherosclerosis) on chondrocytes upregulating matrix metalloproteinase which are involved in inflammation.(21,22) There are relationships between gout, hyperuricaemia and the metabolic syndrome. The metabolic syndrome increases the risk for atherosclerotic cardiovascular disease (CVD) and type 2 diabetes. A recent study looked at the relationship between gout and the development of type 2 diabetes by prospectively studying a cohort of 11 351 male participants from the Multiple Risk Factor Intervention Trial (MRFIT). After adjusting for age, body mass index (BMI), smoking, family history of type 2 diabetes, alcoholic intake, dietary factors, and presence of individual components of the metabolic syndrome the multivariate risk for type 2 diabetes among men with gout at baseline compared to men without gout was 1.34 [95% confidence interval (CI) 1.09–1.64]. These findings from men with a high cardiovascular risk suggests that men with gout are at a higher future risk of developing type 2 diabetes independent of the other known risk factors.(23)The evidence for co-occurrence of two autoimmune diseases RA and diabetes mellitus type 1 has been reported. RA has been linked with the premature development of cardiovascular disease which is related to the inflammatory burden and predisposes to the development of atherosclerosis in these patients. Systemic inflammation has also been implicated in predisposition to developing diabetes mellitus type 2 as well as insulin resistance. Since systemic inflammation is increased in RA it is possible that the prevalence of diabetes is increased in RA. Simard and Mittleman,(24) however, reported a population-based study utilizing data collected from the National Health and Nutrition Examination Study (NHANES) III (included 5302 patients > 60 years of age) in which there was no association between RA and diabetes mellitus. There were several problems with this study including too few patients with RA and diabetes mellitus, a mix of mild and severe RA (also failure to look at anti-CCP positive and negative RA patients), and lastly a concentration of patients with type 2 diabetes mellitus. One established genetic risk factor, the 620W allele of the protein tyrosine phosphate N22gene (PTPN22), is shared by both RA and type 1 diabetes mellitus. In a recent study the presence of this association was again investigated utilizing the Swedish data base. Patients from the RA registry (1419) were compared with matched controls (1674) from the Swedish national population registry during the period 1996–2003. All patients had blood samples that were tested for antibodies to cyclic citrullinated peptide (anti-CCP), rheumatoid factor, and the 620W PTPN22 risk allele. Type 1 diabetes mellitus was associated with an increased risk of RA [odds ratio (OR) 4.9, 95% CI 1.8–13.1]. This association was specific for anti-CCP + RA (OR 7.3, 95% CI 2.7–20.0) and attenuated to an OR of 5.3 when looking further for the presence of the 620W PTPN22 allele (OR 5.3, 95% CI 1.5–18.7), concluding that the association of RA and type 1 diabetes mellitus was limited and specific to one subset (anti-CCP + RA) and the risk in patients with type 1 diabetes mellitus of developing RA in later life was attributed partly to the presence of the 620WPTPN22 allele (possibly representing a common pathway for both auto immune diseases).(25) Further investigations looking for a genetic locus for susceptibility to multiple autoimmune diseases focused on the discovery that a common single-nucleotide polymorphism (SNP) rs6822844p was found in linkage-disequilibrium on chromosome 4q27 with genes KIAA1109, Tenr, IL2, and IL21. IL21 (MIM695384), IL2, KIAA and Tern have been reported to be a strong genetic factor in celiac disease in humans and type 1 diabetes mellitus in mice. Zhernakova, *et al*., (26) investigating common genes for autoimmune disease tested SNP

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rs6822844 from this region for association with disease in 350 type 1 diabetic patients, 1047 patients with RA and 929 controls in a Dutch population. Her results replicated the association found in the KIAA1109/Tenr/IL2/IL21 gene region with type 1 diabetes mellitus and found a similar association with RA implying that this locus may be a general risk factor for multiple auto immune diseases.(27)Diffuse idiopathic skeletal hyperostosis (hyperostosis or DISH) originally described as confined to the axial skeleton involves calcification of tendon and ligament attachments in both spinal and extraspinal locations as well as hyperostosis at bony prominences. Forty to fifty percent of patients with this condition also have diabetes mellitus. Elderly patients with diabetes mellitus have an increased prevalence of DISH over age-matched controls; however, the severity of the diabetes is not related to the extent of DISH present.(28,29) Periarthritis and adhesive capsulitis (frozen shoulder) presenting with shoulder pain (often awaking the patient at night) progressing sometimes to progressive limitation of motion over months is associated with several medical conditions including diabetes mellitus.(30) Abnormality of the involved joint demonstrates thickening and fibrosis of the glenohumeral capsule with the exact mechanism unknown except that proliferating fibroblasts and fibrosing syndromes are seen in diabetes mellitus.When evaluating musculoskeletal complaints, physicians should consider the presence of diabetes because it may influence the management of these conditions. There are several musculoskeletal disorders that exclusively or predominantly occur in people with diabetes . Diabetes mellitus may influence the presentation, severity, andcourse of various musculoskeletal syndromes. Common therapies for the rheumatic diseases may differ in their effects in patients with diabetes compared to others.

Musculoskeletal Disorders Found Predominantly in People with Diabetes: (31,32,33,34)

1. Cheiroarthropathy(diabetic stiff hand syndrome)2. Frozen shoulder3. Flexor tenosynovitis
4. Dupuytren's contracture5. Charcot's arthropathy6. Reflex sympathetic dystrophy(complex regional pain syndrome)7. Diffuse idiopathic skeletalhyperostosis.

Pathophysiology of orthopaedic manifestation in diabetes mellitus:

A-Syndromes Related to Increased Collagen Deposition:

The abnormal accumulation of collagen in skin and tendons leading to impairment of movement underlies several musculoskeletal complications of diabetes. Nonenzymatic glycosylation of proteins (in this case collagen) and excessive deposition of these proteins in tissue has been proposed as an explanation for the development of these musculoskeletal syndromes.(31) The amino groups of lysine residues often are irreversibly glycosylated, leading to increased cross-linking of collagen molecules and reduced breakdown of the protein. In addition, glycosylated collagen could possibly entrap other proteins and thus contribute to further skin thickening.(32) The association with microvascular damage has led to speculation that ischemia may lead to a fibrotic reaction and contribute to the excessive thickening of tissue.(33) It remains unclear why certain patients develop these clinical manifestations. This group include:

1. Cheiroarthropathy 2.Frozen shoulder 3.Flexor tenosynovitis 4.Dupuytrens contracture 5.Carpal tunnel syndrome

B-Syndromes Related to Neuropathy:

Long-standing diabetes is frequently complicated by peripheral neuropathy, particularly involving the sensory nerves. This may predispose to several musculoskeletal syndromes.This group include:1.Charcots arthropathy 2.Reflex sympathetic dystrophy. (complex regional pain syndrome)

1-**cheiroarthropathy:** (Limited joint mobility)

Limited joint mobility (LJM) is a common complication of diabetes mellitus (DM). LJM often is characterized by hand stiffness, but other joints may be involved. The prayer and tabletop signs may be used to detect limitation of joint mobility in the hands. Range of motion should be checked in the large joints as well as in the hand and finger joints. LJM should be distinguished from other musculoskeletal conditions that also are seen frequently in the hands of patients with DM. LJM may be associated with the duration of DM. Treatment is controversial. Strict control of blood glucose usually is advocated. Physical therapy may improve function. No medications have been approved for clinical use. Limited joint mobility (LJM), or diabetic cheiroarthropathy, is a condition characterized by hand stiffness resulting from flexion contractures of the fingers and by thickened, tight, waxy skin.(35) "LJM" is the newer, preferred term used in describing the

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condition because joints other than those in the hands (eg, in the wrists and elbows, feet, and spine) also may be involved.(36))Lundbaek(37) first reported LJM in 5 patients with diabetes mellitus (DM) in 1957, but the syndrome did not receive more attention until 1974, when Rosenbloom and Frias described it again in children with DM. The existence of this clinical entity was confirmed by larger studies of children with insulin-dependent (type 1) DM(38,39) and, subsequently, was demonstrated in adult and geriatric patients with non–insulindependent (type 2) DM.(40,41) LJM is a common complication of DM, occurring in 8% to 58% of patients; most studies suggest that the prevalence is about 30% to 40%.(34,38,42,43) Although early investigators did not find sex differences, one study reported that adolescents who have DM with LJM are predominantly male.(44) No racial differences have been found.

Feature	Description
Symptoms	Initially asymptomatic; decrease in grip strength; difficulty in performing fine hand movements; occasionally painful, especially in adults
Joint involvement	PIP, DIP, and MCP joints in hands; large joints (wrists, elbows, knees, ankles, spine) in later stages
Diagnostic tests	Prayer sign, tabletop sign
Risk factors	Increased duration of diabetes mellitus, prepubescent onset of diabetes mellitus, poor glycemic control
Associations with complications of diabetes mellitus	Increased risk of macroangiopathy (CAD and CVD), increased risk of microangiopathy (retinopathy, nephropathy, and neuropathy), and association with elevated plantar foot pressure and increased risk of foot ulcers

PIP, proximal interphalangeal; DIP, distal interphalangeal; MCP, metacarpophalangeal; CAD, coronary artery disease; CVD, cerebrovascular disease.

Features of limited Joint mobility
(40): Abate M,SchiavoneC,PelottiP,Salini V.

The onset of LJM is insidious and may predate the recognition of overt DM (table 1). Although there is moderate limitation of finger joint mobility, LJM usually is neither painful nor disabling. In making the diagnosis, it is important to differentiate LJM from other DM related hand conditions. LJM has been linked to poor glycemic control and other complications of DM in retrospective studies, but whether it predates the appearance of renal or ophthalmic disease and whether careful blood glucose control with insulin therapy can reduce the rate of its development has not been conclusively determined. However, quick and easy office assessment for LJM should be a part of the routine assessment of patients with DM, and its presence should alert the physician to the likely presence of microvascular or macrovascular disease or both. In this article, we describe the diagnosis and differential diagnosis of LJM, the physical examination and testing, and the association of LJM with DM and various musculoskeletal conditions. We also discuss the controversies involved in treatment of patients with this condition.

Pathogenesis:
The development of LJM is complex and multifactorial. Factors that may lead to impaired mobility include alterations of the structures in the hand (eg, the intrinsic muscles, joint capsule, subcutaneous tissue, and skin). These changes may result from the interaction of vascular ischemia and alterations in the structure or composition ofcollagen(Drug information on collagen). One consequence of prolonged hyperglycemia is nonenzymatic glycosylation of collagen analogous to glycosylated hemoglobin (HbA1c).(45) This glycosylation results in abnormally cross-linked collagens, which are unusually resistant to mechanical and enzymatic degradation,(46) and collagen accumulation in the connective tissue of patients with DM.(42,47)This glycosylated collagen also may entrap potentially harmful nonglycosylated proteins (eg, albumin, immunoglobulins, and coagulation proteins)and

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contribute to increased extracellular matrix accumulation.(45) Glycosylated collagen has been shown to be antigenic in mice and may induce an antibody response.(48) Nonenzymatic glycosylation of collagen also disturbs the cellular and structural components of the microvasculature, resulting in thickening of the capillary basement membrane. This is the fundamental morphological change in diabetic microangiopathy,(49,50) which may then directly contribute to fibrosis by inducing low-grade ischemia and chronic tissue injury.(51) Injury to connective tissue also may be mediated by excessive flux through the aldose reductase pathway, resulting in depletion of essential osmolytes, cytotoxic edema, and membrane injury.(52) Early glycosylation of skin collagen may be decreased with improved glycemic control. However, the long-term cumulative damage that may result from the binding of advanced glycosylation end products to collagen probably is irreversible.(52,53)

Clinical course: In patients with LJM, asymptomatic contractures first develop in the distal interphalangeal and proximal interphalangeal (PIP) joints and, eventually, spread to the metacarpophalangeal (MCP) joints. In the early stages, most patients are completely asymptomatic. Gradually, the joint contractures may extend beyond the hands to the larger joints, including the elbows, shoulders, knees, and axial skeleton. Typically, LJM is painless, especially in juvenile patients with DM. However, adult patients with DM may have coexisting neuropathy and may report pain.

Diagnosis:

Figure 1: (42): Kapoor A,Sibbitt WL jr.

Figure 2: (38) Rosenbloom AL, Silverstein JH,Lezotte DC, *et al*.,

A diagnosis of LJM is made with careful physical examination and exclusion of other conditions in the differential diagnosis. The index of suspicion should be high in all patients with DM, especially those who have had the disease for a long duration. Results of laboratory and radiographic evaluation usually are nonspecific and unremarkable. The erythrocyte sedimentation rate is normal; antinuclear antibodies and rheumatoid factor typically are absent.(54) Two simple clinical tests may be used to detect limitation of joint mobility in the hands. In the prayer sign, the patient is asked to put his or her hands together in a praying position with the fingers fanned and to press together the palmar surfaces of the interphalangeal joints and the palms (figure 2).(38)The tabletop test is conducted by asking the patient to place his hands palms-down on a tabletop with the fingers spread (figure 2).(38) In both tests, the entire palmar surface of the fingers making contact constitutes a normal result.

Skin changes: Skin thickness in the patient's hands is assessed by attempting to tent the skin on the dorsum of the fingers between the examiner's thumb and index finger. When skin changes are severe, loss of the transverse digital skin ridges on the dorsum of the fingers is obvious. However, these skin changes also have been found in patients with DM who did not have LJM (and have been referred to as "diabetic sclerodactyly"). (59,39,60)These skin changes have been likened to those seen in patients with scleroderma. Both DM and scleroderma manifest increased production of collagen by dermal fibroblasts in culture and can be strongly linked to abnormalities of the microvascularcirculation. However, the pathogenesis in LJM seems to be more related to metabolic than to immunological causes.

Association withDM complications: In multiple studies, LJM has been found to correlate with the

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duration of DM.(37,61,62) Campbell and colleagues(35)found a general but asymptomatic reduction in joint mobility as early as 2 years after the diagnosis of DM, with continuing gradual deterioration as the duration of DM increased. Joint contractures were prevalent only in patients with long-standing DM (disease duration of 9 years or longer). The researchers also found that limitation of joint mobility was more marked in patients with a prepubertal onset of DM than in those in whom the diagnosis was made after puberty. They explained this finding with the hypothesis that hyperglycemia results in the laying down of large amounts of highly glycosylated collagens during the pubertal growth spurt. (63) The finding was confirmed by a longitudinal cohort study that reported an increased risk of LJM with longer duration of DM and with puberty.(64)

Musculoskeletal andother associations: Additional rheumatologic problems, such as shoulder bursitis, tendinitis, epicondylitis, osteoarthritis, and Dupuytren contracture, have been found to occur more frequently in patients with DM who have LJM than in those who do not.(65)Fisher and coworkers(66) reported an increased prevalence of frozen shoulder in patients with type 1 DM who had LJM and suggested that both conditions may be related to underlying abnormalities in glycosylation of collagen.

Ultrasonography: Patients with LJM had a tendon sheath thickness of more than 1 mm, compared with a thickness of less than 1 mm in unaffected patients with DM and controls. Collier and associates (67, 68,69) used ultrasonography to study skin thickness in 92 patients with DM and 40 without. They found that thickness increases with the duration of DM and is closely related to the presence of LJM.

Treatment:

Treatment	Considerations
Strict glycemic control	May help prevent development of limited joint mobility; whether it can halt or reverse disease progression is unclear
Physical and occupational therapy	Improvement in mobility seen during therapy may not be sustained with discontinuation of therapy
Medications (penicillamine, aminoguanidine, sorbinil [aldose reductase inhibitor])	Have been used experimentally but are unavailable or not recommended
Local corticosteroid injections or surgery	May help in management of coexisting flexor tenosynovitis/trigger finger or Dupuytren contracture

Treatment of Patients With Limited Joint Mobility
Rosenbloom AL, Buithieu M, Jelliffe KA, *et al*.,(70)

PATIENTS AND METHODS

The research began since august 2012, and many cases calculated from outpatients and inpatients which have both diabetes mellitus with orthopaedic problems.Inpatients include both orhopaedics and medical wards in Alsader educational hospital, Albasrah general hospital. Outpatients include both orthopaedics and medical wards in Alsader educational hospital, Albasrah general hospital,almoane hospital, Alhusaini educational hospital in Karbela. found 200 cases under period august 2012- september 2013(i.e one year).157 outpatients and 43 inpatients, 121 patients are female and 79 patients are male.8 patients from age of (20-29) years, 15 patients from age of (30-39) years, 34 patients from age of (40-49) years, 65 patients from age of (50-59) years, 54 patients from age of (60-69) years, and 24 patients from age of (70 and more) years. 21 patients was (IDDM), 178 patients was (NIDDM) and 1 patient was (MODM).

RESULT

According to my research I found 26 patients of them have good diabetic control and 174 of them have bad control. 59 of them on oral hypoglycemic agent , 75 of them on insulin therapy, 64 of them on both oral hypoglycemic and insulin therapy and just 2 patients was on diet control. 127 of them are smoking, 13 of them are alcoholic and 134 of them have positive family history of diabetes. I found several patients they have either single or multiple

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orthopaedic complain but mostly in the same patient there is multiple orthopaedic complain as acomplication of long term uncontrolled diabetes mellitus . I depend mostly on fasting bood sugar and glycosylated haemoglobin (HbA1c) as indicators of good or bad control of diabetes mellitus. I found many orthopaedic complain in the upper limb, lower limb, spine, myopathy, vasculopathy and peripheral neuropathy.In the upper limb i found 32 patients have frozen shoulder, 3 patients have reflex sympathetic dystrophy, 23 patients have carpal tunnel syndrom, 19 patients have trigger finger, 4 patients have olecranon bursitis, 16 patient have limited joint mobility(cheiroarthropathy), 24 patients have De-quervien, 11 patients have tennis elbow and 26 patients have dupuytrens contracture.In the lower limb I found 11 patients have trochantric bursitis, 23 patients have charcots joint, 64 patients have complicated diabetic foot in many stages of wagner classifications, 15 patients have plantar fasciitis, 3 patients have tarsal tunnel syndrom, 6 patients have foot deformities and no patients have tendinosis. In the spine I found 26 patients which have clinical and radiological manifestations of diffuse idiopathic skeletal hyperostosis (DISH) some of them which is till know not diagnosed by neither physician or orthopaedic doctors. I found also 24 patients have muscle cramp, 39 patients have osteoporosis, 32 patients have osteomylitis, 5 patients have septic arthritis, 5 patients have gout or psudogout, 85 patients have peripheral neuropathy, 53 patients have vasculopathy, 5 patients have myopathy and 0 patient have muscle infarction.I depend in my evaluation of the patients according to laboratory investigations on patients journal to those which is inpatients and i sent investigations to those which is outpatients. In addition to that I depend on previous imaging and I sent patients for plain x-ray and dual energy x-ray absorptiometry (DEXA) especially for osteoporotic patients.168 patients have medical treatment and just 32 patients have surgical treatment. Finally i have good contact with many cooperative patients especially elderly both male and female to follow up for diabetic control and orthopaedic treatment of various complaint. This follow up not included in my study but just I do it for the patients which ask help from me. Summary of my results in the following tables:

Table (1): 200 patients with diabetes mellitus

Outpatients	Inpatients	Male	Female
157	43	79	121
78.5%	21.5%	39.5%	60.5%

Table (2): Number of patients according to age and percentage

Patients age	Patients number	percentage
20-29	8	4%
30-39	15	7.5%
40-49	34	17%
50-59	65	32.5%
60-69	54	27%
70 and above	24	12%

Table (3): Type of treatment and weather good or bad control of diabetes mellitus

Oral hypoglycemic	Insulin	Both	Diet	Good control	Bad control
59	75	64	2	26	174
29.5%	37.5%	32%	1%	13%	87%

Table (4): Orthopaedic manifestations of the upper limb in patients with diabetes mellitus

Orthopedic manifestations	Patients number	Percentage
Frozen shoulder	32	16%
Reflex sympathetic dystrophy	3	1.5%
Carpal tunnel syndrom	23	11.5%
Trigger finger	19	9.5%
Olecranon bursitis	4	2%
Cheiroarthropathy	16	8%
De-quervien	24	12%
Tennis elbow	11	5.5%
Dupuytrens contracture	26	13%

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Table (5): Orthopaedic manifestations of the lower limb in patients with diabetes mellitus

Orthopaedic manifestations	Patients number	Percentage
Trochantric bursitis	11	5.5%
Tendinosis	0	0%
Charcots joint	23	11.5%
Diabetic foot	64	32%
Plantar fasciitis	15	7.5%
Tarsal tunnel syndrom	3	1.5%
Foot deformities	6	3%

Table (6): Orthopaedic manifestations of the spine in patients with diabetes mellitus

Neck and back	Patients number	Percentage
Diffuse idiopathic skeletal hyperostosis	26	13%

Table (7): Other orthopaedic manifestations in patients with diabetes mellitus

Other orthopaedic manifestations	Patients number	Percentage
Muscle cramp	24	12%
Muscle infarction	0	0%
Osteoporosis	39	19.5%
Osteomyelitis	32	16%
Septic arthritis	5	2.5%
Gout and psudogout	5	2.5%
Peripheral neuropathy	85	42.5%
Vascular complications	53	26.5%
Myopathy	5	2.5%

Table (8): Social history in patients with diabetes mellitus

Social history	Patients number	Percentage
Smoking	127	63.5%
Alcoholic	13	6.5%
Positive family history	134	67%

Table (9): Type of treatment in patients with diabetes mellitus

Type of treatment	Patients number	Percentage
Medical	168	84%
Surgical	32	16%

DISCUSSION

Musculoskeletal disorders are common in type 1 and 2 diabetic subjects, and examination of periarticular regions of the hands, the joints, shoulders and feet, as well as the skeleton, should be included in the evaluation of patients with DM. Musculoskeletal disorders in these patients are probably related to the long-term poor glycaemic control of the diabetes and duration of diabetes. The pathophysiology of these disorders in diabetic patients is not obvious. It could be associated with connective tissue disorders, such as the formation of abnormally glycosylated end products or the impaired degradation of byproducts, it could be indirectly related to the vasculopathy and neuropathy commonly complicating the primary disease, or finally, it could be attributed to a combination of factors.I don't find significant difference in musculoskeletal disorders between type 1 and type 2 diabetic patients, despite the substantial difference in the mean age of the patients in the two groups. Hand abnormalities are common in diabetic patients, reflecting pathologic changes in the microvasculature, connective tissue, and peripheral nerves. In my study evaluated 111 patients 55.5% have either single or multiple hand complications in both type I and type 2 diabetes (i.e Reflex sympathetic dystrophy 3 patients, carpal tunnel syndrom 23 patients, trigger finger 19 patients, cheiroarthropathy 16 patients, de-quervien 24 patients and dupuytrens contracture 26 patients). One study, for example, evaluated 100 diabetic patients selected randomly in an outpatient clinic. Hand abnormalities were observed in 50 patients, and more than one abnormality was found in

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26.(71) Carpal tunnel syndrome, Dupuytren's contracture, flexor tenosynovitis, and limited joint mobility were each present in approximately 20 percent.(71) In my study the incidence of carpal tunnel syndrom 11.5% and cheiroarthropathy 8%. In other study carpal tunnel syndrom has been reported in up to 20% of diabetic patients, but the incidence rises to 75% in those with limited joint mobility. (71, 72) Dupuytrens contracture evaluated 26 patients 13% in my study, in others it affects 16%-32% of the patients,(73, 74, 75, 76) being more common among the elderly and those with longer diabetes mellitus duration(73, 76) which is so also in my study. Trigger finger in my study evaluated 19 patients 9.5% but in other study the prevalence ranges from 5%-36% in those with type 1 or type 2 diabetes mellitus.(77, 78) The prevalence of carpal tunnel syndrom in other study range from 11%-25%, being more common in women.(76, 79) The prevalence of adhesive capsulitis of the shoulder being identified in 10%-29% in other study (73,80,81) but in my study 16%. I don't find any patient with muscle infarction in my study because of relatively rare complication. there is also no data present in other study. Diffuse idiopathic skeletal hyperostosis has prevalence of 13%-40% (73,82,83) but in my study has prevalence of 13%. Charcot joint has prevalence estimated among diabetic patients in one study varies from 0,08%-13%(84) but in my study 11,5%. Diabetic neuropathy affects approximately 131 million people as of 2010(1.9% of the population). It is estimated that the prevalence of neuropathy in diabetes patients is approximately 20%. (85) but in my study 42.5%.

CONCLUSION

Diabetes quite commonly affects the musculoskeletal system, resulting in significant morbidity. These manifestations may go unrecognized or simply be overlooked in daily clinical practice. The complications of diabetes mellitus are numerous and include involvement of the musculoskeletal system. Several rheumatic conditions are more prevalent or caused by the long term metabolic consequences of diabetes mellitus. When the control of diabetes is poor, higher levels of diabetic complications result. Poor glycaemic control can lead to worsening of certain rheumatic conditions. Pharmacotherapy, diet, and a regular, sensible physiotherapy programme should be the cornerstone of diabetes management. However, many of these rheumatological complications are treatable (to varying degrees), with resultant improvements in quality of life and more independence in activities of daily living. Thus, clinicians should be aware of the possible musculoskeletal complications of diabetes in order to intervene and provide the best care for affected patients. Asking patients about their symptoms and monitoring for signs of musculoskeletal complications can be an invaluable part of overall diabetes care.

AIM OF THE STUDY

The aim of my study was to investigate the prevalence of musculoskeletal disorders in patient with diabetes mellitus with the aid of the (Spectrum of diabetic patients in orthopaedic unit) questionnaire, and to compare the results with results previously published from studies performed in other populations.

RECOMMENDATION

It is our recommendation that all patients with diabetes have an appropriate exercise programme, overseen by their medical practitioner, as an integral part of their diabetes management in order to reduce the frequency and severity of complications.

ABBREVIATIONS:

1.DM: diabetes mellitus, 2.HLA: human leucocyte antigen, 3.GDM: gestational diabetes mellitus, 4.GAD: glutamic acid decarboxylase, 5.MODY: maturity onset diabetes of the young, 6.HNF: hepatocyte nuclear factor, 7.FPS: fasting plasma glucose, 8.OGTT: oral glucose tolerance test, 9.HbA1c: glycosylated hemoglobin test, 10.SMBG: self monitoring of blood glucose, 11. T1DM: type 1 diabetes mellitus, 12. T2DM: type 2 diabetes mellitus, 13. HDL: high density lipoprotein, 14. RAGE: receptor for advanced glycationendproducts, 15. CVD: cardiovascular disease, 16. MRFIT: multiple risk factor intervention trial, 17. BMI: body mass index, 18.RA: rheumatoid arthritis, 19.PTPN 22: protein tyrosine phosphate N22 gene, 20. Anti-CCP: antibodies to cyclic cirullinated peptide, 21. SNP: single nucleotide polymorphism, 22. DISH: diffuse idiopathic skeletal hyperostosis, 23. LJM: limited joint mobility, 24. CTS: carpal tunnel syndrome, 25.DD: dupuytrens contracture, 26. CN: charcot neuropathy, 27. RANKL: polypeptide receptor activator of nuclear factor-kB ligand, 28.OPG: glycopeptidesosteoprotegerin, 29. CGRP: calcitonin gene-related peptide, 30. ROM: range of motion, 31. SI joint: sacroiliac joint, 32. DXA: dual energy X-ray absorptiometry.

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Source of support: Nil; **Conflict of interest:** Nil.

Cite this article as:
Hameed, A.H. and Majeed, H.R. "Diabetic Spectrum Patients in Orthopaedic Unit." *Sarcouncil Journal of Medicine and Surgery* 3.2 (2024): pp 1-15.

Publisher: SARC Publisher