
MUSCULOSKELETAL MANIFESTATIONS OF
DIABETES MELLITES-A ONE YEAR CROSS
SECTIONAL STUDY

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ENDORSEMENT

This is to certify that the dissertation entitled **MUSCULOSKELETAL MANIFESTATIONS OF DIABETES MELLITES-A ONE YEAR CROSS SECTIONAL STUDY** is a bonafide research work done by **REG NO. BG0114008**.

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LIST OF ABBRIVATIONS

AGEs	:	Advanced Glycosylation End Products
CTS	:	Carpal Tunnel Syndrome
DC	:	Dupuytren's Contracture
DISH	:	Diffuse Idiopathic Skeletal Hyperostosis
DM	:	Diabetes Mellitus
LJMS	:	Limited joint mobility syndrome
OA	:	Osteoarthritis
MSC	:	MusculoSkeletal Complaints
NSAIDS	:	Non-Steroidal Anti-Inflammatory Drugs
RAGEs	:	Receptor for AGEs
WHO	:	World Health Organization

ABSTRACT

Purpose: Diabetes is one of the most common non communicable diseases globally. The complications of diabetes mellitus are numerous and include involvement of the musculoskeletal system. The incidence of diabetes mellitus and the life expectancy of the diabetic patient have both increased in view of wide variety of treatment options, resulting in increased prevalence and clinical importance of musculoskeletal disorders in diabetic subjects. This study represents the common musculoskeletal disorder among the diabetic patients.

Objectives: To study musculoskeletal manifestations of diabetes mellitus and to study correlation of severity with duration of diabetes mellitus

Methodology: A cross sectional study was conducted in patients presenting to Department of Internal medicine at KLES Dr Prabhakar Kore Hospital & MRC, Belgaum fulfilling inclusion criteria. All the patients with type 2 diabetes with duration of 5 year underwent a complete rheumatic examination to assess the musculo skeletal disorders. X ray of involved joint was taken if clinical signs and symptoms are present. Descriptive statistics was used for data analysis.

Results: The study results show that more than half of the participants 69% were male and 31% were female. The study also reveals that (n=100) 96% diabetic patient were suffering from musculoskeletal pain and 4% diabetic patient were free from musculoskeletal pain. . Our study showed prevalence of Charcots joint (35%), followed by Dupuytren contracture(33%), Frozen shoulder and Limited Joint Mobility Syndrome (21%), Trigger Finger(17%), Carpel Tunnel Syndrome (11%) and Diffuse Idiopathic Skeletal Hyperostosis (DISH) in 8%.

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INTRODUCTION

1.1 Background

Diabetes is one of the most common non communicable diseases globally. It is the fourth or fifth leading cause of death in most developed countries and there is substantial evidence that it is epidemic in many developing countries. India being Diabetic capital of world , Diabetes is said to be silent killer of our country. In developing countries like India, rising diabetes rates present enormous challenges to poverty eradication and economic development.

Generally Type-2 Diabetes affects populations between 40 and 70 years of age and it is the most common type of the disease (Ozdirenc et al., 2004). There is a higher incidence of Type-2 Diabetes in urban than in rural areas (Kinget al.,1998) as well as incidence is associated with population whose lifestyle has changed from traditional patterns to a modern “Westernized” model (Bloomgarden, 1996).

An increasing trend in diabetes prevalence has been reported; comparatively it was more in urban areas than rural.

Country	Rural	Urban
India	6.4%	12.1%

According to the WHO approximately 180 million people worldwide currently have Type-2 Diabetes mellitus; over 95 percent of people with diabetes have this form (WHO, 2008). Based on current tendencies, more than 360 million individuals will have the disease by 2030⁵

The prevalence of both type of Diabetes varies considerably across the globe in relation to difference in genetic and environmental factors. The prevalence of known diabetes in Britain around 2-3%, but is the higher in the middle and Far East . A pronounced rise in the prevalence of type 2 diabetes occurs in migrant population to industrialized countries, as in Asian and Afro-Caribbean immigrants to the U.K (Rahim et al., 2011).

Prevalence of diabetes has increased according to recent epidemiological reports in India (8.2%), Turkey (7.2%), Pakistan (11.1%), and Hawaii (20.4%). In European population, age standardized prevalence varied from 3-10%. Some Arab, migrant Asian Indian, Chinese and Hispanic American population were at higher risk with prevalence of 14-20%. The higher prevalence's was found in the Nauruan 41% and the pima/papago Indians 50% (Rahim et al., 2011).

Diabetes has a major and deleterious impact on both individual and national productivity. The socio economic consequences of diabetes and its complications could have a seriously negative impact on the economics of developed and developing nations (WHO, 2011).

Complications from diabetes such as coronary artery and peripheral vascular disease, stroke, diabetic neuropathy, amputations, renal failure, and blindness are resulting in increasing disability, reduced life expectancy and enormous health costs for virtually every society. Diabetes is certain to be one of the most challenging health problems in the 21st century (Richard et al., 2010).

According to 2007 death certificates from U.S.A, Diabetes was the seventh leading cause of death. Diabetes is likely to be under reported as a cause of death.

Studies have found that about 35 to 40 percent of decedents with diabetes had it listed anywhere on the death certificate and about 10 to 15 percent had it listed as the underlying cause of death. Overall, the risk for death among people with diabetes is about twice that of people of similar age but without diabetes (Death among people with diabetes, 2007).

The complications of diabetes mellitus are numerous and include involvement of the musculoskeletal system (Wild et al., 2004). Every diabetic patient has a risk of various bone and joint disorder in future life. The incidence of diabetes mellitus and the life expectancy of the diabetic patient have both increased in view of wide variety of treatment options, resulting in increased prevalence and clinical importance of musculoskeletal disorders in diabetic subjects.

The cause for development of musculoskeletal disorders is often unclear, but several theory predict that it is dependent on age and the duration of diabetes mellitus .Most of these disorders appear in diabetic patients of younger age than their counter parts in the general population. Musculoskeletal disorders in these patients are probably related to the long term glycemic control of the diabetes. However no direct association could be proven with the metabolic control of the disease (Doulaumpakas et al., 2007).

Factors such as nerve damage (diabetic neuropathy), arterial disease and obesity may contribute to these problems, but often the cause is not clear. Most of these disorders can be diagnosed clinically but some radiological examination may help, especially in differential diagnosis (Arkkila et al., 1996). Musculoskeletal disorders are common in type 2 diabetic subjects and examination particular regions

of the hands, the joints, shoulders and feet, as well as the skeleton should be included in the evaluation of patients with diabetes mellitus.

Some musculoskeletal disorders are more prevalent in the diabetic population than in the general population such as rotator cuff tendonitis, frozen shoulder, osteoarthritis etc (Gilek & stahl, 2012).

1.2 Rationale

Diabetes mellitus poses serious health problems both in developed and developing countries. The prevention and control of diabetes in developing countries deserve urgent attention since the disease is expected to double in these countries in the next 20 to 25 years. The problem of diabetes mellitus in India is also increased day by day as like as whole world. Musculoskeletal conditions are prevalent and their impact is pervasive. They are the most common cause of severe long term pain and physical disability, and they affect hundreds of millions of people around the world .¹

Musculoskeletal conditions are a diverse group with regard to patho-physiology but are linked anatomically and by their association with pain and impaired physical function. They encompass a spectrum of conditions, from those of acute onset and short duration to lifelong disorders, including osteoarthritis, rheumatoid arthritis, osteoporosis, and low back pain. The prevalence of many of these conditions increases markedly with age, and many are affected by lifestyle factors, such as obesity and lack of physical activity.¹

The burden of musculoskeletal disorders can be measured in terms of the problems associated with them, that is the pain or impaired functioning (disability) related to the musculoskeletal system, or in relation to the cause, such as joint disease

or trauma. The burden should also be considered in terms of who is at risk. But usually people are not aware about these problems.

Rheumatologic disorder described in the patients with diabetes can be divided into three categories⁴:

1. Disorder which represents intrinsic complications of diabetes which include
 - a. Diabetic muscle infarction (common in type 1 diabetes mellitus).
 - b. Neuropathic arthropathy which include charcot joint, claw toe, roker bottom sole.
2. Rheumatologic disorders under this category can be further divided into two groups:
 - a. Disorder related to metabolic derangement
 - i. DISH (diffuse idiopathic skeletal hyperostosis)
 - ii. Osteopenia
 - iii. Osteoporosis
 - b. Disorders which share similar aetiological mechanism as with microvascular disease and change of collagen
 - i. Limited joint mobility (Cherioarthropathy)
 - ii. Dupuytren's contracture
 - iii. Palmar flexor tenosynovitis (trigger finger)
 - iv. Adhesive capsulitis of the shoulder
 - v. Reflux sympathetic dystrophy (Algodystrophy)

3. Other rheumatological disorders which are common in general population have increased prevalence in diabetic population
 - a. Carpal tunnel syndrome
 - b. Gout
 - c. Osteoarthritis
 - d. Osteolysis of fore foot
 - e. Migratory osteolysis of hip and knee

Despite the multifactorial nature of musculoskeletal disease, obesity consistently emerges as a risk factor in the onset of progression of musculoskeletal conditions of hip, knee, ankle, foot and shoulder². Emerging evidence indicates that DM associated with obesity has profound effect on soft-tissue structures, such as tendon, fascia and cartilage. Although the mechanism remains unclear, the functional and structural limitations imposed by additional loading of the locomotor system in obesity is almost universally accepted to produce aberrant mechanics during locomotor tasks, thereby unduly raising stress within connective tissue structures and the potential for musculoskeletal injury.³

In India this problem is more severe. This study aims to address these problems and design physiotherapy intervention for these diabetic patient with musculoskeletal disorders.

Hence, this study will document the Prevalence of Musculoskeletal disorders in T2DM in Indian population.

1.3 Research Question

What are the common musculoskeletal disorders among the diabetic patients?

1.4 Objectives

1.4.1 General objective

To explore the common musculoskeletal disorders among the diabetic patient.

1.4.2 Specific objectives

To identify the number of diabetic patient affected by musculoskeletal disorder.

To find out the common musculoskeletal disorders in different body region of diabetic patient.

To explore the socio demographic characteristics of diabetic patient.

To study the corelation of severity with duration of diabetes mellitus.

1.5 Operational definition

Diabetes mellitus

Diabetes mellitus is a group of chronic metabolic conditions, all of which are characterized by elevated blood glucose levels resulting from the body's inability to produce insulin or resistance to insulin action, or both.

Musculoskeletal disorder

A musculoskeletal disorder is a condition where a part of musculoskeletal system is injured over time. The term musculoskeletal disorder identifies a large group of conditions that result from traumatizing the body in either a minute or major way over a period of time.

OBJECTIVES

1. To study musculoskeletal manifestations of diabetes mellitus
2. Correlation of severity with duration of diabetes mellitus

REVIEW OF LITERATURE

Musculoskeletal complaints (MSCs) are the major health problems worldwide and the most frequent cause of long-term sickness⁷. Hence this further emphasizes that this group of patients may constitute an important public health problem.

They affect all age groups, cultures, and ethnic. They cause chronic disability, impairments, and handicaps. They consist of a variety of different diseases that cause pain or discomfort in the bones, joints, muscles, or surrounding structures, and they can be acute or chronic, focal, or diffuse. Approximately 33 percent of U.S adults are affected by musculoskeletal signs or symptoms, including limitation of motion or pain in a joint or extremity.

In one study of Detroit residents who kept track of daily health symptoms in a diary, musculoskeletal symptoms constituted the most frequent category of health symptoms.

The prevalence of musculoskeletal disorders generally increases with age, with the majority of persons aged seventy-five and over having some form of musculoskeletal disorder, especially arthritis (Felson, 2000).

These disorders are the leading cause of disability and loss of function, as well as limitation and impairment of activities for people over the age of 18. These conditions affect nearly one in two adults and, among medical conditions, lead to the greatest number of lost workdays and medical bed days in the United States (Steve et al., 2012).

Arthritis and musculoskeletal conditions constitute a major public health problem, as large contributors to illness, pain and disability. They occur frequently,

placing a high economic and personal burden on the community. This burden includes the use of hospital and primary care services, disruptions to daily life, and lost productivity through functional limitations and activity restriction (Rahman et al., 2005).

A large proportion of the musculoskeletal problems for which patients seek medical attention are related to periarticular structures and do not represent a true articular process or a more generalized systemic illness (Wise, 2003).

Diabetes is a multi-system disorder affecting 3-7% of the adult population in different geographical areas. Diabetes mellitus accounts for a number of vascular complications, which impair patients' survival⁵. Musculoskeletal complications are also found, and, although less valued than the vascular ones, they significantly compromise the patients' quality of life⁶. The incidence of DM and the life expectancy of diabetic patients have both increased, resulting in an elevation in the prevalence and clinical importance of those osteomuscular changes.

A survey was done which showed that DM was associated with higher prevalence of chronic MSCs, in particular chronic widespread MSCs⁸. Some of the complications have a known direct association with diabetes, whereas others have a suggested but unproven association (Kim et al., 2001).

DM acts as a base for many musculoskeletal disorders and complications, causing pain, disease or even disability which later on affects and disturbs the quality of life of an individual. MSC are major cause of crippling deformities and other disabilities for many diabetic patients. But if it is correctly diagnosed it is usually controllable by the particular handling and management given by a multidisciplinary team work⁹.

Musculoskeletal disorders are significant health and safety issues for which challenges and opportunities exist to better understand objective causes and effects, economic impacts and effective strategies to prevent and treat this complicated disorders (Seaman, 2013).

Several musculoskeletal disorders have been described in these patients which can be divided into three categories (table1) (Douloumpakas et al., 2007)

Table 1
Musculoskeletal disorders in diabetes mellitus⁵

Intrinsic complications of DM	Increased incidence of DM	Likely association
Limited joint mobility syndrome	Dupuytren's disease	Osteoarthritis
Stiff hand syndrome	Adhesive capsulitis	Carpal tunnel syndrome
Muscular infarctions	Neuropathic arthropathy	
	Flexor tenosynovitis	
	Septic arthritis	
	DISH	
	Diabetic neuropathies	

DISH: diffuse idiopathic skeletal hyperostosis

1. Intrinsic complications of diabetes:

I. LIMITED JOINT MOBILITY SYNDROME:

Limited joint mobility syndrome (LJMS) also known as diabetic cheiroarthopathy (after the Greek word “cheiros” for hand) or "stiff hand syndrome" is a painless noninflammatory limitation of the mobility of hands, feet, and large joints¹¹. Upper limb is more commonly involved than lower limb.

PATHOGENESIS: The LJMS is believed to be influenced by a poor glycemic control, although the association between that musculoskeletal complication

and glycemic control, or even HbA1C levels, is controversial as they have also been reported in children with recent-onset DM^{24 10,15,11}.

The following biochemical abnormalities seem to be related to the appearance of LJMS:

An increase in the non-enzymatic glycosylation of collagen fibers, an increase in collagen crosslinking and its consequent resistance to enzymatic digestion; an increase in the hydration mediated by aldolase reductase pathway; and an increase in the formation of advanced glycosylation end products (AGEs)^{11,12}. The increase in the formation of AGEs might associate the occurrence of LJMS with micro- and macrovascular complications of DM^{10,11}. The AGEs result from the rearrangement of Amadori products or early glycosylation products. They accumulate in tissues, depending on time and glucose concentrations, and damage extra- and intracellular proteins. On cell surface, there is a receptor for AGEs (RAGEs), which is a transmembrane receptor of the family of the immunoglobulins, signaling events that lead to cell dysfunction. Experimental studies have shown that there is a reduction in the vasodilating response to nitric oxide, and that AGEs decrease vascular elasticity^{11,13}.

The influence of a genetic component on the development of the syndrome is controversial^{14,15}. Some authors¹⁰ have reported that diabetic children with LJMS had more relatives with the same findings than children without that syndrome. However, Rosebloom et al¹⁵ have not been able to confirm those findings when assessing 204 individuals with type 1 DM and their 336 first-degree relatives.

The histological examination shows dermal thickening, accumulation of connective tissue in the reticular dermis with increased collagen cross-linking, and small amounts of mucin²³ deposition in periarticular rather than articular region¹⁷.

CLINICAL FEATURES: Diabetic cheiroarthropathy, or "stiff-hand syndrome" is characterized by painless limitation of mobility of the small joints of the hands. The prevalence ranges from 8% to 50% among patients with diabetes, compared with only 4% to 20% among individuals without DM (Serban et al., 2012). This condition is most commonly seen in type 1 diabetics, with a prevalence of 8–50%¹⁶, compared with 0–26% in controls¹⁸, with differences in prevalence estimates possibly related to differences in the definitions used and perhaps differences in glycaemic control¹⁹. The prevalence of proteinuria and retinopathy was of 11% in diabetic patients without diabetic cheiroarthropathy versus 50% in diabetic patients with diabetic cheiroarthropathy (Rosenbloom et al., 1981)^{10,21,22}.

In the early stages, Patients can be asymptomatic or complain from pain, which increases with the use of the extremity, or from paresthesia¹⁰. The symptoms increase very slowly and greater pain, aggravated by movement of the hands, may supervene. Limited joint mobility and Dupuytren's contracture are commonly found in the same patient^{16,21,22}.

It is characterised by thick, tight, waxy skin mainly on the dorsal aspect of the hands, with flexion deformities of the metacarpophalangeal and interphalangeal joints of the fifth finger, progressing to involve all fingers. (increased resistance to passive extension of the joints). The changes in the hands and forearms, with no changes in joint mobility, can also be found^{10,23}.



FIGURE 1: PRAYER SIGN

The cutaneous changes should be differentiated from those of scleroderma, where the lack of Raynaud's phenomenon; dermal atrophy; telangiectasia; and autoantibodies²³ help in differentiation.

The stiff hand syndrome is diagnosed based on its characteristic findings and physical examination. The patients' inability to press their palms together completely without a gap remaining between opposed palms and fingers is known as the "prayer sign" (Figure 1)¹⁰. One alternative manner to test reduced joint mobility is with the so-called "table top test", in which the patient places an open hand on a table top with fingers spread apart. When positive, the fingers and palm cannot lie completely flat on the table top¹⁰. Passive mobility reduction is confirmed by lack of extension of proximal interphalangeal and metacarpophalangeal joints (lower than 180° and 60°, respectively)¹⁰.

The recommended treatment consists of optimal glycemic control, hand therapy programme and non-steroidal anti-inflammatory drugs^{10,11}.

However, NSIADS are usually avoided in view of association of LJMS with diabetic nephropathy. In the presence of cutaneous involvement, the only treatment proposed is glycemic control.

2.MUSCULAR INFRACTION:

That is a relatively rare complication, mainly found in patients with type 1 DM and disease duration over 15 years¹⁰. Clinically, the condition presents as muscle edema and pain of sudden onset¹⁰. A palpable mass can be detected in 34%–44% of the cases^{10,28}. Thigh muscles are involved in approximately 80% of the cases, but more than one infarction point can appear Simultaneously²⁹. The diagnosis is established based on clinical history and imaging, especially magnetic resonance imaging. Muscle enzymes, such as CPK, are slightly increased¹⁰. CT scan is nonspecific. On magnetic resonance imaging, isointense edema on T1 and hyperintense edema on T2 are found in muscle areas, with subcutaneous and subfascial edema. Usually, gadolinium is not required, but, when used a non-enhanced area surrounded by another hyper-enhanced is observed³⁰. Biopsy shows necrosis of muscle fibers, edema, phagocytosis of necrotic fibers, granulation tissue and collagen deposits. Older lesions might show regeneration of muscle fibers, replacement by fibrous tissue and mononuclear infiltration²⁸.

Because most patients with muscular infarction have diabetic retinopathy, neuropathy and nephropathy, those diagnoses are believed to be associated with local ischemia. Hypercoagulable states with changes in the coagulation fibrinolysis system and endothelial dysfunction have also been proposed as potential pathogenic mechanisms³¹. Another hypothesis would be the contribution of antiphospholipid antibodies, but that has not been proven³². Muscular infarction resolves spontaneously

in weeks or months, but half of the patients have recurring episodes. Treatment consists of rest and analgesia¹¹.

3.NEUROPATHIC (CHARCOT'S) JOINTS:

Charcot's disease, or joints, is a result of diabetic peripheral neuropathy. A reduction in the normal afferent protective neural impulses, and therefore loss of protection from trauma to the joint leads to progressive, painless joint destruction. Charcot's joints are typically seen in patients over the age of 50 who have had diabetes for many years and have existing neuropathic complications. The estimated prevalence of Charcot joint involving foot and knees is 3.2% diabetic patients compared to 0.6% in the non-diabetic group involving one or both knee joints.

The joints most commonly affected are weight-bearing joints such Tarsal and tarsometatarsal joints, followed by metatarso-phalangeal joints and ankles⁹³. Joints such as the hand and wrist are rarely affected⁸⁸.



FIGURE 2: CHARCOTS JOINT

PATHOGENESIS: Charcot's arthropathy, or diabetic neuropathic arthropathy, results from a likely combination of mechanical and vascular factors secondary to diabetic neuropathy.⁸⁹ Lack of proprioception has been postulated to cause ligament looseness, joint instability and joint lesion from small traumas. Another possibility is

that autonomic neuropathy causes vasomotor alterations with the formation of arteriovenous shunts and a reduction in the effective blood flow to the skin and bones, despite the good amplitude of peripheral pulses⁹⁰. The third hypothesis is an excessive inflammatory response to traumas, mediated by pro-inflammatory cytokines⁹¹. Regardless of the cause of the problem, there is an initial phase, which is resorptive, followed by a repair or hypertrophic phase⁹².

CLINICAL FEATURES: The patient can present with sudden-onset erythema and unilateral edema of the foot or ankle. Recurring attacks might follow, and chronic arthropathy, characterized by plantar arch collapse and bony prominences, develops⁸⁹. Complications such as infected ulcers might develop. In 20% of the patients, Charcot's arthropathy is bilateral⁸⁹. The arthropathy is either painless or the pain is disproportionately milder than expected. Initial warmth and erythema mimic osteomyelitis or septic arthritis, but the absence of fever, elevated white cell count, and elevated erythrocyte sedimentation rate helps to differentiate the latter two conditions.

Diagnosis is established via imaging, which shows, at an initial stage, only osteopenia, a reduction in joint space, and soft tissue edema. With progression, areas of osteolysis develop, with phalangeal and metatarsal head resorption. Luxations, bone fragmentation, sclerosis and neoformation can be seen at the final stages^{89,92}. Contrast-enhanced magnetic resonance imaging might be required to discard associated osteomyelitis⁹⁴.

TREATMENT: Management consists of optimising glycaemic control and regular foot care and review, particularly in those with grossly impaired sensation. Prevention

of weight bearing and use of orthotics and crutches can relieve pressure on the affected joints during ambulation⁸⁸.

The use of bisphosphonates (alendronate and pamidronate) might be useful^{94,96}. Calcitonin has been used in patients with renal failure who cannot receive bisphosphonates, but its benefits are yet to be proven⁹⁷. Occasionally surgery may be required if complicated fractures develop.

2.INCREASED INCIDENCE WITH DIABETES :

2.DUPUYTREN'S CONTRACTURE

The prevalence of Dupuytren's contracture in diabetic patients ranges from 20 to 63%^{10,40,33,11,20,36,37} compared with 13% in the general population. Among patients with Dupuytren's contracture, 13–39% have diabetes^{36,38}. The contractures are generally milder in diabetics than in patients with Dupuytren's contracture who do not have diabetes, and the prevalence increases with advancing age^{10,34,39,40} but may also be seen early in the course of disease.

PATHOGENESIS: Pathogenesis is thought to be the same as that for cheiroarthropathy. DC results from local hypoxia followed by the release of free radicals, which affect the function of fibroblasts that produce altered collagen fibers.

The histological examination shows a dense collagenous matrix containing fibroblasts longitudinally aligned along the lines of stress. The nodules contain myofibroblasts and collagen bands, and local blood vessels are narrowed⁴¹. There is an increased amount of glycosaminoglycans, and the local collagen has a higher proportion of type 3 fibers as compared with type 1 fibers⁴¹.

CLINICAL FEATURES: Dupuytren's contracture is the palmar or digital thickening, tethering, palmar and digital nodules, skin thickening and adherence, pretendinous band formation, and digital flexor contracture of ring and middle finger^{10,11,33} but sometimes involving any of the second through fifth digits. In patients with diabetes, the ring and middle finger are more commonly affected, compared with the fifth finger in patients without diabetes⁴⁰.



FIGURE 3: DUPUYTREN'S CONTRACTURE

The DC of diabetic patients have some peculiarities. It usually affects the third and fourth fingers first, rather than the fourth and fifth fingers, as typically occurring in cases associated with other etiologies^{40,36}. The second is that, females are more commonly involved in diabetic DC compared to non diabetic DC where males are more commonly involved, although the manifestation are still more severe among men^{10,40,36}. Thirdly Diabetic cheiroarthropathy and Dupuytren contracture may coexist in the same patient (Smith et al., 2003).

TREATMENT: Treatment consists of optimizing glycaemic control, physiotherapy, and hand exercises. The contractures are usually mild and require surgery only if function is severely affected^{10,35}. Varied success has been reported with local corticosteroid injections.

Recently, the injection of collagenase from *Clostridium histolyticum* has been claimed to be an alternative non-surgical treatment. A study with 308 patients, 6.5% of whom were diabetic, has reported an improvement in the flexion contracture and range of motion of finger joints with three or more collagenase injections. In that study, two patients had tendon rupture and one developed reflex sympathetic dystrophy.

3. ADHESIVE CAPSULITIS:

Diabetes can affect the shoulder in several ways. First, adhesive capsulitis, or frozen shoulder. The most disabling of the common musculoskeletal problems is adhesive capsulitis, which is also known as frozen shoulder, shoulder periartthritis, or obliterative bursitis. The prevalence of adhesive capsulitis of the shoulder is five-fold higher in the diabetic population than in the general population, being identified in 10%–29% of the former^{10,52,53}. A higher prevalence of frozen shoulder (20–29%) has been reported in diabetes mellitus (DM) patients (Jung et al., 2010). The estimated prevalence is 11–30% in diabetic patients and 2–10% in nondiabetics⁴⁶⁻⁴⁹. Adhesive capsulitis is associated with the duration of diabetes and age^{48,49}. Diabetes of long duration treated with insulin was associated with a larger percentage of shoulder calcification (Mavrikakies et al., 1989). It appears in both type 1 and type 2 DM, is more common in the elderly, and can be bilateral¹⁰ Up to 50% of patients have bilateral involvements..

This term refers to a stiffened glenohumeral joint usually caused by a reversible contraction of the joint capsule. Adhesive capsulitis appears at a younger age in patients with diabetes and is usually less painful⁴⁴, although it responds less well to treatment and lasts longer⁴⁵. The typical patient is female with NIDDM of long duration who present with diffuse soreness and global loss of motion of shoulder.

PATHOGENESIS: The exact origins of adhesive capsulitis are not known, although it has been associated with several other conditions, including shoulder trauma, cerebral conditions, cardiac conditions, and respiratory conditions. Some researchers, studying patients with frozen shoulder, have reported a higher prevalence of myocardial infarction in those with type 1 DM, and of autonomic neuropathy in those with types 1 and 2 DM¹⁰. The histological exam of the capsule shows proliferation of fibroblasts and their transformation into myofibroblasts, which produce an excessive amount of type 1 and type 3 collagen. Those findings are similar to those of DC^{10,51}

CLINICAL FEATURES: Patients report shoulder stiffness, along with decreased range of motion. It is characterised by progressive, painful restriction of shoulder movement, especially external rotation and abduction¹¹. The thickened joint capsule is closely applied and adherent to the humeral head, resulting in considerable reduction in the volume of the glenohumeral joint. Adhesive capsulitis of the shoulder (also known as frozen shoulder) presents as an almost complete limitation of passive and active mobility of the shoulder, mainly on adduction and external rotation¹⁰. That condition occurs in a progressive and painful manner, leading to contracture of the joint capsule, which adheres to the humeral head, reducing the joint volume¹⁰. The pain appears at night initially and installs gradually¹⁰. The natural history of adhesive capsulitis of the shoulder can be divided into the following three phases: (a) pain; (b) stiffness; and (c) recovery.^{10,43} Diabetic patients with frozen shoulder are more likely to have other diabetic complications such as limited joint mobility than diabetics without a frozen shoulder, although this may be explained by age^{48,49}. Pall et al⁵² have proposed criteria for diagnosing adhesive capsulitis of the shoulder that include shoulder pain for at least one month, impossibility of lying on

one's shoulder, and limited active and passive mobility in at least three planes.



FIGURE 4:FROZEN SHOULDER

TREATMENT: Therapy is largely conservative and involves minimizing overimmobilization (gentle stretching/range of motion exercises) and the use of analgesics and/or intra-articular injections.

Most cases of adhesive capsulitis will resolve over time, but, in the interim, management consists of adequate analgesia and intra-articular corticosteroid injections in the painful early stages if required. Corticosteroid injections may increase blood sugar levels in diabetics over the 24–48 hour period after the injection, and therefore blood sugar monitoring and contingency plans for elevated blood sugar levels should be considered. Distension or manipulation under anaesthesia are occasionally considered. An appropriately graded, regular physiotherapy programme should be maintained, after the painful phase, throughout the course of the condition.

Calcific peri-arthritis of the shoulder is also seen in diabetes, where it is roughly three times more common than in people without diabetes. Reflex sympathetic dystrophy, also known as “shoulder-hand syndrome,” is seen in diabetic

patients, although whether it occurs with increased frequency is controversial (Kim et al., 2001). In DM, impairment of the shoulder has been described as the most disabling musculoskeletal manifestation⁵⁰.

The treatment of adhesive capsulitis of the shoulder consists of analgesics, corticosteroid infiltrations, and physical therapy. Most patients recover normal function^{10,11}. In the adhesive phase, capsule release can be performed via manipulation under anesthesia or surgery^{10,11}. Surgical release is preferably performed via arthroscopy rather than open surgery, because the former reduces the post-operative recovery period^{10,54}.

4.FLEXOR TENOSYNOVITIS OR TRIGGER FINGER:

The prevalence of trigger finger in patients with DM ranges from 5% to 36% in those with type 1 or type 2 DM as compared with 2% in the general population^{55,56,58}. There is also an increased incidence in people with impaired glucose tolerance⁵⁹. Its development being associated with longer disease duration but not age.^{59,10,11} When compared with non-diabetic patients, those with DM have a tendency to the simultaneous involvement of multiple fingers^{40,57}. According to Koh et al⁵⁸ the involvement of three or more fingers is highly suggestive of the association with DM, which should be investigated if not yet diagnosed.

PATHOGENESIS: The pathogenesis is said to be similar to as diabetic cheiroarthropathy. It results from fibrosis, with tendon thickening as it passes through the pulley or one bone prominence, limiting its motion inside the sheath. A volume increase distal to the constriction point causes pain and difficulty in flexing and extending the corresponding finger, which might become locked¹¹.

CLINICAL FEATURES: Stenosing flexor tenosynovitis presents typically as fingers locked in flexion, extension or both, more commonly involving the thumb, the third finger and/or the fourth finger^{10,11}. Patients complain of a catching sensation or locking phenomenon that may be associated with pain in the affected fingers. Examination shows a palpable nodule, usually in the area overlying the metacarpophalangeal joint, and thickening along the affected flexor tendon sheath on the palmar aspect of the finger and hand. Also, the locking phenomenon may be reproduced with either active or passive finger flexion.



FIGURE 5 TRIGGER FINGER

TREATMENT: The treatment of stenosing flexor tenosynovitis comprises a change in activities, use of NSAIDS, splinting, infiltrations. A corticosteroid injection into the symptomatic flexor tendon sheath is often curative. If this is unsuccessful, patients will most likely need a minor surgery that can provide permanent relief. A small transverse incision just distal to the flexion crease over the metacarpal head exposes the flexor tendons and sheath. A complete longitudinal incision along the thickened fibrous tendon sheath relieves the constriction and allows the finger to move freely.^{10,11}

5.REFLEX SYMPATHETIC DYSTROPHY

Reflex sympathetic dystrophy is also known as algodystrophy, Sudeck's atrophy, and chronic regional pain syndrome type 1. It is characterised by localised or diffuse pain, associated with swelling, trophic changes, and vasomotor disturbances,⁹⁸ with impaired mobility of the affected region. Pathogenesis is usually unclear. The condition may occur spontaneously, or after minimal trauma—following surgery or a fracture. Concurrent medical conditions may predispose to reflex sympathetic dystrophy, including diabetes mellitus, hyperthyroidism, hyperparathyroidism, and type IV hyperlipidaemia.⁹⁹ A variety of treatments have been used with including analgesics, physiotherapy, intravenous bisphosphonates, calcitonin, oral corticosteroids, and sympathetic ganglion blocks.⁹⁹ The outcome is usually good, although some patients develop chronic pain and contractures.

6.DIFFUSE IDIOPATHIC SKELETAL HYPEROSTOSIS (DISH)

(FORESTIER' DISEASE):

Diffuse idiopathic skeletal hyperostosis, also known as ankylosing hyperostosis or Forestier's disease, defined as the involvement of four contiguous vertebral segments with preservation of intervertebral disc spaces and lack of degenerative apophyseal involvement and of sacroiliac inflammatory changes⁶¹. Later, it was modified to include symmetrical peripheral enthesopathy⁶². DISH affects mainly the thoracic spinal column, but the lumbar and cervical segments might also be involved⁶⁰. It is characterised by new bone formation, particularly in the thoracolumbar spine sometimes leading to bony ankylosis. New bone appears to “flow” from one vertebra to the next, and is more prominent on the right side of the thoracic vertebra⁶⁹.

Its estimated prevalence is 13–49% in diabetic patients and 1.6–13% in non-diabetics^{10,37,63,64}. Among patients with diffuse idiopathic skeletal hyperostosis, 12–80% have diabetes or impaired glucose tolerance. The high prevalence of abnormal glucose tolerance tests in patients with diffuse idiopathic skeletal hyperostosis is partly a result of an association with obesity, with 83% of patients being male and 30% obese⁶⁸. Obesity and diabetes seem to have independent contributions to the development of the condition^{10,68}. Rates of hyperostosis increase with age in both the normal and diabetic populations, although the age related increase in incidence begins earlier in diabetics.

Ossification of ligaments and tendons elsewhere may occur, such as the skull, pelvis, heels, or elbows⁷⁰.

PATHOGENESIS: Insulin has been proposed as a factor that promotes growth in DISH. In one study, patients with DISH and those with osteoarthritis had elevated levels of insulin and growth hormone, however, the level of IGF-1 was higher in patients with DISH than in those with osteoarthritis (Denko et al., 1994). A proposed mechanism of causation is the prolonged and high levels of insulin or insulin-like growth factors occurring in diabetic patients, stimulating new bone growth^{10,60}, and may explain the higher prevalence in type 1 compared with type 2 diabetes (ratio 3:1)³⁷. One study showed serum levels of matrix Gla protein, which inhibits bone formation to be paradoxically higher than in controls⁶⁶.

CLINICAL FEATURES: Patients can be asymptomatic or have pain in the affected sites, column stiffness on arising in the morning, dysphagia, and odynophagia, in the presence of large cervical osteophytes^{60,62}. There may be associated pain in one third of patients who have hyperostosis of the heels or elbows. Patients with hyperostosis of

the spine may have associated mild, and 16% of affected persons may develop dysphagia⁶⁸. Neurological complaints might result from spinal cord compression due to ossification of the posterior longitudinal ligament¹⁰. Peripheral pain results from peripheral enthesal involvement⁶⁰.

Management consists of education, diabetic control, and physiotherapy¹⁰.

3.LIKELY TO BE ASSOCIATED WITH DIABETES:

CARPAL TUNNEL SYNDROME (CTS) :

Carpal tunnel syndrome (CTS) is a painful disorder caused by the compression of the median nerve between the carpal ligament and other structures within the carpal tunnel, diabetic neuropathy, or a combination of both^{71,72}. CTS is common in patients with diabetes, with an estimated prevalence of 11–16%^{85,86} compared with an incidence of about 125 per 100000 population over a five year period⁸⁷. The incidence rises to 75 percent in those with limited joint mobility (Botek et al., 2010). About 5–8% of patients with CTS have diabetes^{76,78,73}. CTS is more common in women^{40,76} and in patients with polyneuropathy⁷⁷. Associations between carpal tunnel syndrome and age and the duration of diabetes have also been suggested⁷².

However, some authors believe that the real predisposing factor to CTS is obesity, common in patients with type 2 DM⁷⁹. A study of 791 patients with CTS referred for electrophysiological study⁸⁰ has shown that a diagnosis of DM, female sex, obesity and age between 41 and 60 years were risk factors for CTS; however, when data were stratified according to the patients body mass index, the association with DM disappeared.

CLINICAL FEATURES: Carpal tunnel syndrome (CTS) is characterized by paraesthesia over the median nerve cutaneous distribution i.e thumb, index, middle, and lateral half of the ring fingers, can radiate to the forearm and arm, which is often worse at night^{11,74}. In advanced cases, atrophy of the thenar musculature and loss of grip strength can occur⁷⁴.

Clinical diagnosis is established by use of the Phalen's maneuver and Tinel's test⁷⁴. A positive Tinel's sign is described as a tingling sensation in a specific anatomic distribution, which occurs as a result of light percussion over a nerve. Phalen's maneuver, or the wrist flexion test, is described as positive when full flexion of the wrist for 60 seconds causes paresthesia in the territory of the median nerve. In dubious cases, electrophysiological study might help⁷⁵.

TREATMENT: Treatment of CTS consists of the use of simple analgesics, splints, and possibly local steroid injections for the milder cases of compressive CTS. Effect of steroids is temporary and the response of patients with DM is poorer^{81,83}.

Release surgery might be required, at a 4–14-times greater frequency in diabetic patients than in the general population^{73,83}. The post-operative recovery of these patients is worse as DM impairs peripheral nerve regeneration due to microangiopathy, macrophagic and Schwann cell dysfunction, and reduced expression of neurotrophic factors and their receptors.^{58,84}

Thus the prevention of diabetes related musculoskeletal problems requires preventive approach. Diabetic management in an optimal way with musculoskeletal care, education for patients and their family, implementing screening and risk assessment tools by health providers will have a critical role in prevention of diabetic related musculoskeletal problems.

METHODOLOGY

3.1 Study design:

This study aimed to find out common musculoskeletal disorders among the diabetic patient in KLES DR PRABHAKAR KORE HOSPITAL. A cross sectional study design was used to conduct the study.

3.2 Study site and area:

Data was collected from the patients presenting to Department of Internal medicine at KLES Dr Prabhakar Kore Hospital & MRC, Belgaum fulfilling inclusion criteria.

Duration of study: One year

Period of study: January 2015 to Dec 2015

3.3 Study population and sampling:

1. Sample size :100

As per data provided by MRD , KLE's hospital, Belgaum

According to formula: $n=4 \cdot pq / d^2$

Where n= sample number , P=prevalence , Q= 100-p , D= 10

Hence $n= 4 \cdot 50 \cdot 50 / 100$,therefore $n=100$.

3.5 Sample selection criteria:

3.5.1 Inclusion criteria:

Patients with Type 2 Diabetes mellitus with duration of 5 years.

3.5.2 Exclusion criteria:

Diabetes mellitus with acute complications like DKA, shock, CRF.

3.6 Data collection instrument:

A structured questionnaire and demographic information chart used as a data collection instrument. In that time some other necessary materials are used like pen, pencil, and white paper and clip board. The English questionnaires were converted into kannada, hindi, Marathi to ask the participants during interviews. Informed written consent was taken from each volunteer participant.

3.7 Procedure of data collection:

At very beginning it was clarified that the participant had the right to refuse to answer of any question during completing questionnaire. They could withdraw from the study at any time. It was also clarified to all participants about the aim of the study. Participants were ensured that any personal information would not be published anywhere. After taking consent form the participants, standard questionnaire was used to identify the musculoskeletal complain and collect demographic information. Questions were asked according to the performa.

For conducting the interview, Face to face interview and asked questions. Physical environment was considered strictly. Stimuli that can distract interviewee were removed to ensure adequate attention of interview. Interviewee were asked questions alone as much as possible with consent as sometimes close relatives can guide answer for them. Then built rapport and clarified questions during the interview. According to the participants' understanding level, sometimes the questions were described in the native language so that the patients can understand the

questions perfectly and answer accurately. All the data were collected by the researcher own to avoid the errors.

3.8 Field test:

Prior to collect data a field test was performed with 10 participants in the KLES Dr Prabhakar Kore Hospital. To make a feasible questionnaire was translated into kannada, Marathi and hindi. This test was performed to determine any difficulties that are exist in the questionnaires as well as the procedure of data collection. This test also helped the researcher to check the appropriateness of wording as well as ease of understanding of the questions.

3.9 Data analysis:

Descriptive statistics was used to analyze data. Descriptive statistics refers methods of describing a set of results in terms of their most interesting characteristics . Data were analyzed with the software named Statistical Package for the Social Science (SPSS) version 16.0. The variables were labeled in a list and established a computer based data definition record file that consist of a list of variables in order, put the name of the variables in the variable view of SPSS and defined the types, values, decimal, label alignment and measurement level of data. The next step was cleaning new data files to check the inputted data set to ensure that all data had been accurately transcribed from the questionnaire sheet to the SPSS data view. Then the raw data was ready for analysis in SPSS. Data was analyzed by descriptive statistics and calculated as percentages and presented by using table, bar graph etc. Microsoft office Excel 2007 is used to decorating the bar graph.

3.10 Ethical consideration:

Ethical committee clearance was taken before starting the study. During the course of this study, interested subjects were given consent forms and the purpose of the research and the consent form were explained to them verbally in Kannada, Marathi or Hindi. The participants were informed that their participation would be fully voluntary and they had the right to withdraw or discontinue from the research at any time without any hesitation or risk. They were also informed that confidentiality would be maintained. Information might be published in any presentations or writing, but their personal identity such as their name and address will not be mention in the study. The participants were informed that the data was collected by written questionnaire. The supervisor also checked the consent form and questionnaire. For this study took permission during interview from every single participant with signature or thumb impression on a written consent form of the participants who were interested. The participants were informed about their role in the research process. The participants were informed about the aim of the research and procedures involved in the study. They were also informed that if they wish they were free to withdraw from the study any time. The study information only discusses with supervisor but this would not share with any other person. These materials were disposed off after completion of the research project. The study results might not have any direct effects on them. Participants were also informed that they would not get any harmful things from the study.

3.11 Limitations

Despite best efforts with research, the present study was not completely free from all limitation and impediments. Limitations are:

- Sample size was small to generalize the study result.
- This study was done in a short period, so all factors in relation to diabetes patient's musculoskeletal problem may not be highlighted.
- Study was conducted only in BIRDEM General Hospital. So this study result would not be generalized for whole Bangladesh.
- To identify musculoskeletal problem laboratory diagnosis was not available to all participants. This can be limitation of this study.
- Time and resources are limited have a great deal of impact on the study.

RESULTS

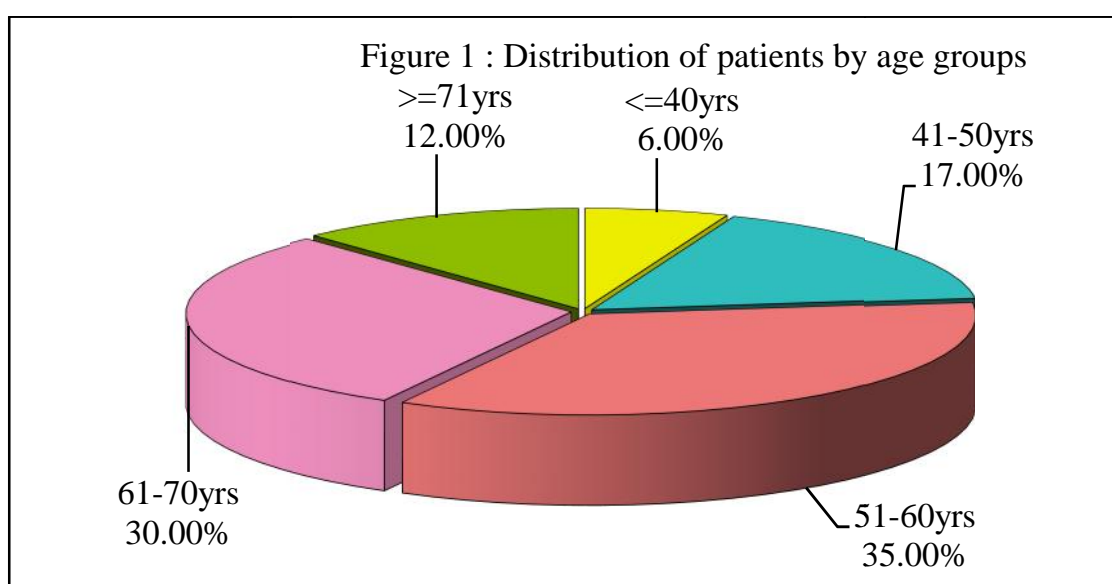
This study was conducted in KLES DR PRABHAKAR KORE HOSPITAL between January 2015 to December 2015.

4.1 AGE GROUP

The distribution of the subjects into the age band was as follows:

Table2: Distribution of patients by age groups

Age groups	No of patients	% of patients
<=40yrs	6	6.00
41-50yrs	17	17.00
51-60yrs	35	35.00
61-70yrs	30	30.00
>=71yrs	12	12.00
Total	100	100.00
Mean age	58.37	
SD age	10.77	

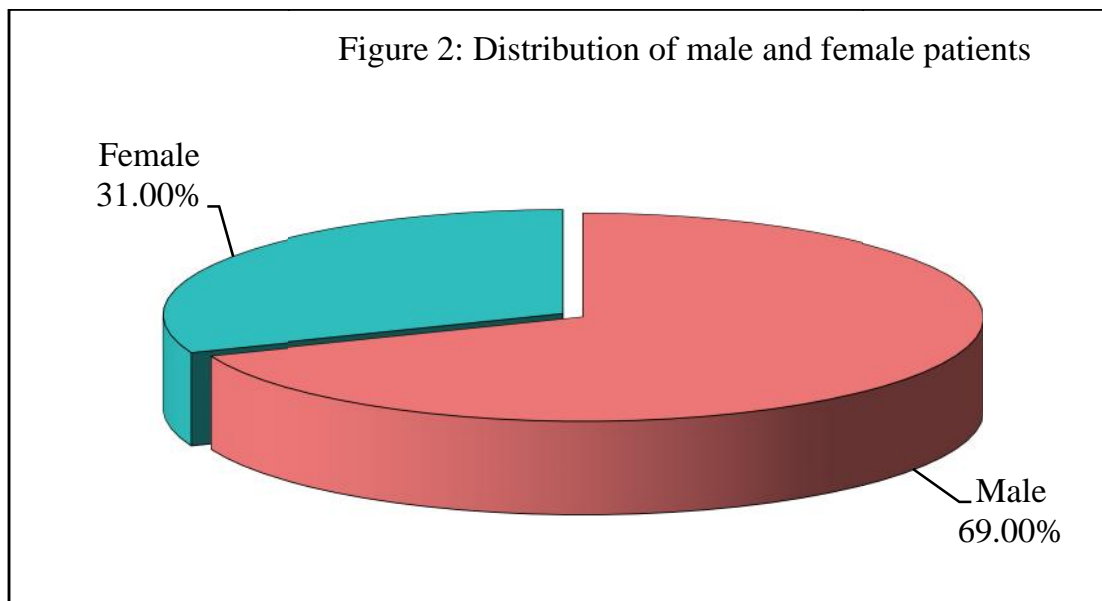


A total of 100 diabetic patients were participant. There were maximum number of cases between age group of 51-60 (35%) followed by 61-70 years (30%), 41-50 years (17%), more then 71 years(12%), less then 40 years (6%).

4.2 GENDER :

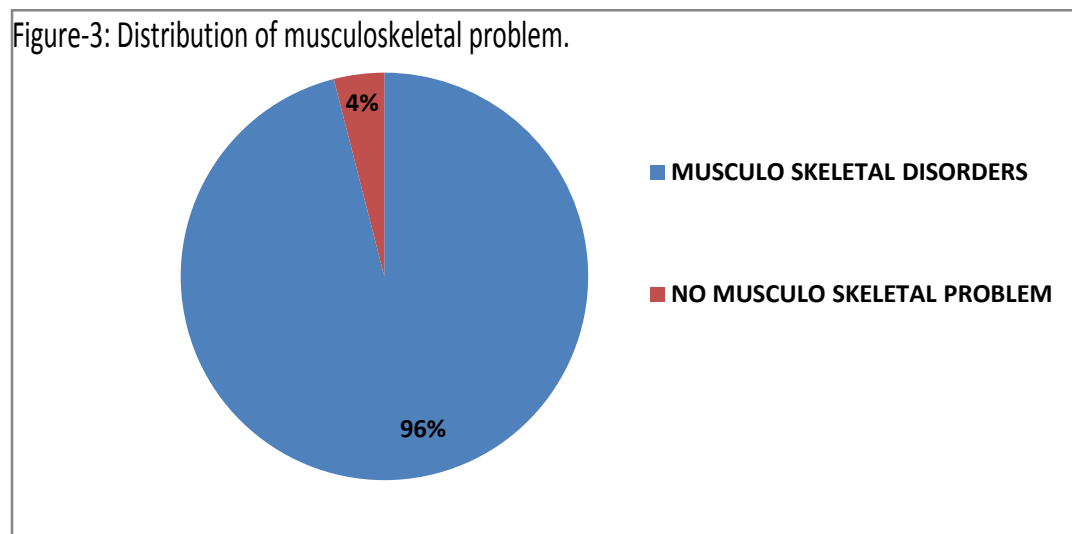
Table 3: Distribution of patients by gender

Gender	No of patients	% of patients
Male	69	69.00
Female	31	31.00
Total	100	100.00



The bar chart showed that among the 100 participants it was found that 31% were female and 69% were male. (Figure-2)

4.3 DISTRIBUTION OF DIABETIC PATIENT HAVING MUSCULOSKELETAL PROBLEM:



Among the participants (N=100) about 96% of diabetic patient suffering from musculoskeletal problems and 4% not having musculoskeletal problems. The Distribution of diabetic patient having musculoskeletal problem was showed in (Figure-3).

4.4 PREVALENCE OF MUSCULO SKELETAL DISORDERS:

Table 4: Prevalence of musculo skeletal disorders in the study

Musculo skeletal disorders	Yes	%	No	%
Limited joint mobility syndrome	21	21.00	79	79.00
Dupuytren contracture	33	33.00	67	67.00
Trigger finger	17	17.00	83	83.00
Carpel tunnel syndrome	11	11.00	89	89.00
Frozen shoulder	21	21.00	79	79.00
Diffuse idiopathic skeletal hyperostosis	8	8.00	92	92.00
Charcot joint	35	35.00	65	65.00

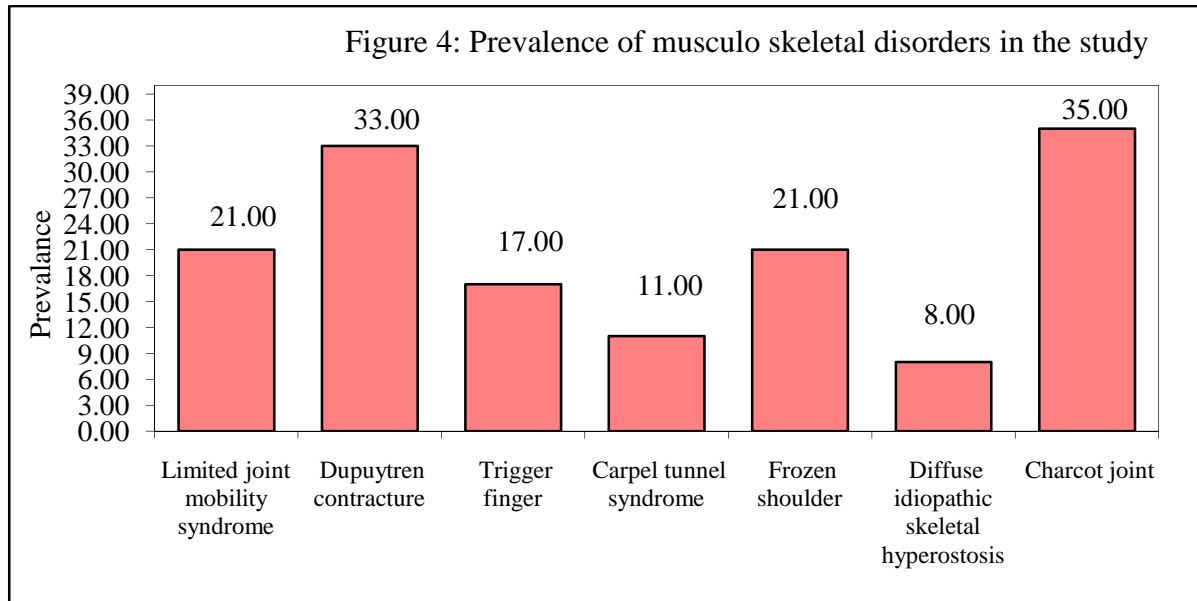


Figure 4 shows the distribution of Musculoskeletal Disorders among patients who are known Diabetic for more than 5 years. Among 100 participants we found highest prevalence of Charcot's joint being (35%), followed by Dupuytren contracture (33%), Frozen shoulder and Limited Joint Mobility Syndrome (21%), Trigger Finger (17%), Carpal Tunnel Syndrome (11%) and Diffuse Idiopathic Skeletal Hyperostosis (DISH) in 8%.

4.5 CORRELATION BETWEEN HBA1C WITH MUSCULO SKELETAL DISORDERS:**TABLE 5: CORELATION BETWEEN HBA1C WITH MUSCULO SKELETAL DISORDERS IN THE STUDY**

Disorders	N	Spearman R	t-value	p-level
Limited joint mobility syndrome	100	0.0532	0.5278	0.5988
Dupuytren contracture	100	-0.0393	-0.3896	0.6977
Trigger finger	100	0.0809	0.8036	0.4236
Carpel tunnel syndrome	100	0.1071	1.0659	0.2891
Frozen shoulder	100	-0.0441	-0.4368	0.6632
Diffuse idiopathic skeletal hyperostosis	100	0.1228	1.2251	0.2235
Charcot joint	100	0.0241	0.2383	0.8122

Correlation between HbA1C with musculo skeletal disorders was done by Spearman's rank correlation method. In our study we found that there was no co-relation between HbA1c levels and prevelance of musculo skeletal disorders.

4.6 CORRELATION BETWEEN DURATION OF DIABETES WITH MUSCULO SKELETAL DISORDERS:

TABLE 6: CORRELATION BETWEEN DURATION OF DIABETES WITH MUSCULO SKELETAL DISORDERS IN THE STUDY

Disorders	Correlation between duration of diabetes with			
	N	Spearman R	t-value	p-level
Limited joint mobility syndrome	100	0.2589	2.6536	0.0093*
Dupuytren contracture	100	0.1865	1.8797	0.0631
Trigger finger	100	0.2338	2.3800	0.0192*
Carpel tunnel syndrome	100	-0.1614	-1.6186	0.1087
Frozen shoulder	100	0.1960	1.9783	0.0500*
Diffuse idiopathic skeletal hyperostosis	100	-0.0908	-0.9031	0.3687
Charcot joint	100	0.3106	3.2343	0.0017*

*p<0.05

Correlation between duration of diabetes with musculo skeletal disorders was done by Spearman's rank correlation method. In study we found that as duration of diabetes increases prevalence of Limited Joint Mobility Disorder, Trigger finger, Frozen shoulder and Charcots joint was increased. P value was highly significant for Charcot's joint and limited joint mobility syndrome.

4.8 COMPARISON OF AGE GROUPS OF PATIENTS WITH DURATION OF DIABETES:

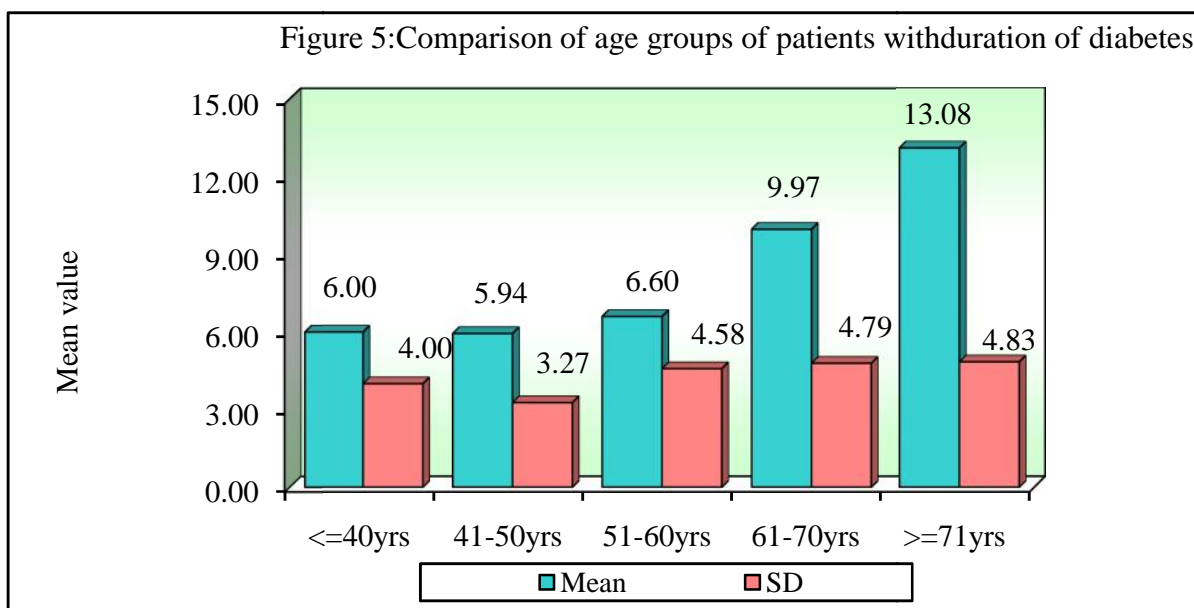
TABLE 7: COMPARISON OF AGE GROUPS OF PATIENTS WITH DURATION OF DIABETES IN THE STUDY

Summary	<=40yrs	41-50yrs	51-60yrs	61-70yrs	>=71yrs	Total	H-value	P-value
Mean	6.00	5.94	6.60	9.97	13.08	8.24	27.3180	0.0001*
SD	4.00	3.27	4.58	4.79	4.83	5.00		

*p<0.05

Comparison of age groups of patients with duration of diabetes was done by Kruskal Wallis ANOVA test.

Mean of patients aged more than 70 years was 13.08 with SD 5, followed by 61-70 years with mean of 9.97 and SD 4.79, 51-60 years with mean of 6.60 and SD 4.79, less than 40 years with mean of 6 and SD 4, 41-50 years mean of 5.94 and SD 4.58.



4.9 COMPARISON OF MALE AND FEMALE PATIENTS WITH MUSCULO SKELETAL DISORDERS

TABLE 8: COMPARISON OF MALE AND FEMALE PATIENTS WITH MUSCULO SKELETAL DISORDERS IN THE STUDY

Variables	Summary	Male	Female	Total	Z-value	P-value
Limited joint mobility syndrome	Mean	3.06	1.16	2.47	-2.2347	0.0254*
	SD	6.00	4.56	5.64		
Dupuytren contracture	Mean	5.48	5.90	5.61	-0.0134	0.9893
	SD	9.61	10.16	9.73		
Trigger finger	Mean	0.59	3.61	1.53	-4.3449	0.0001*
	SD	2.81	5.22	3.96		
Carpel tunnel syndrome	Mean	0.33	1.39	0.66	-1.8729	0.0611
	SD	1.45	3.14	2.16		
Frozen shoulder	Mean	2.74	1.35	2.31	-1.7428	0.0814
	SD	5.59	4.51	5.30		
Diffuse idiopathic skeletal hyperostosis	Mean	0.42	0.23	0.36	-1.1327	0.2573
	SD	1.45	1.26	1.39		
Charcot joint	Mean	6.59	5.84	6.36	-0.0919	0.9268
	SD	11.02	10.16	10.71		

*p<0.05

Comparison of male and female patients with musculo skeletal disorders values was done by Mann-Whitney U test. In study we found that females are more prone for Trigger Finger and limited joint mobility syndrome then males with p value of 0.0001 and 0.0254 respectively.

DISCUSSION

This study was conducted in KLES DR.PRABHAKAR KORE HOSPITAL, BELGAUM. Among 100 participants there were maximum number of cases between age group of 51-60 (35%) followed by 61-70 years (30%), 41-50 years (17%), more than 71 years(12%), less than 40 years (6%).

Among the 100 participants it was found that 31% were female and 69% were male.

In study conducted by Smith LL, Burnet SP, McNeil JD. Musculoskeletal manifestations of diabetes mellitus. Br J Sports Med 2003; 37(1): 30–5, the prevalence of musculoskeletal disorders was 60%.Among the participants (n=100) about 96% were suffering from musculoskeletal disorders and 4% had no problems.

In study by Lebiez-Odrobina D, Kay J. Rheumatic manifestation of diabetes mellitus. Rheum Dis Clin N Am 2010; 36(4):681–99 showed that prevalence of musculoskeletal disorders among diabetic patients as follows: 8% frozen shoulder,4% are carpal tunnel syndrome, 9% are neck pain, 27% are low back pain, 8% are other musculoskeletal problem, 24% are no problem. Our study showed prevalence of Charcots joint (35%), followed by Dupuytren contracture(33%), Frozen shoulder and Limited Joint Mobility Syndrome (21%), Trigger Finger(17%), Carpel Tunnel Syndrome(11%) and Diffuse Idiopathic Skeletal Hyperostosis (DISH) in 8%.

In study by Jennings AM, Milner PC, Ward JD. Hand abnormalities are associated with the complications of diabetes in type 2 diabetes. Diabet Med 1989;6:43–7, there was no co-relation found between HbA1C and prevalence of

musculo skeletal disorders. In our study we found that there was no co-relation between HbA1c levels and prevalence of musculo skeletal disorders.

In study conducted by R N Sarkar et al J Indian Rheumatol Assoc 2003 : 11 : 25 – 29 duration of diabetes was associated with increased prevalence of Limited Joint Mobility Disorder, Trigger finger, Frozen shoulder and Charcot's joint. In our study duration of diabetes was associated with increased prevalence of Limited Joint Mobility Disorder, Trigger finger, Frozen shoulder and Charcot's joint. p value was highly significant for Charcot's joint and limited joint mobility syndrome.

In study done by Lebedz - Odrobina D, Kay J. Rheumatic manifestation of diabetes mellitus. Rheum Dis Clin N Am 2010; 36(4):681–99 found that prevalence of Dupuytren's contracture, Trigger finger, CTS is increased in females compared to males. In our study we found that females are more prone for Trigger Finger and limited joint mobility syndrome than males with p value of 0.0001 and 0.0254 respectively.

SUMMARY & CONCLUSION

Musculoskeletal disorders have a high prevalence rate in diabetics. Many of these disorders are treatable especially if diagnosed early and can improve quality of life by reducing morbidity associated with these disorders. Diabetes must be considered as one of differential diagnosis for any patient with musculo skeletal disorder. Locomotor system examination yields lot of information regarding musculo skeletal manifestations in diabetes mellitus. Thus, having an awareness of the potential musculoskeletal complications of diabetes can be an invaluable part of diabetes care.

BIBLIOGRAPHY

1. Special Theme – Bone and Joint Decade 2000- 2010 ;Bulletin of the World Health Organisation 2003;81(9)646-56.
2. World Health Organisation- Media Centre : Overweight and Obesity 2011.
3. Wearing SC, Hennig EM, ByrneScott NM: Musculoskeletal disorder associated with diabetes: A biomechanical perspective 2006;239-50.
4. Badal Pal. Rheumatic disorders and bone problems in diabetes mellitus. Text Book of Diabetes 2. Edited by John C. Pickup and Garath Williams. Third edition. Blackwell Publishing.
5. Alvin C Power. Diabetes mellitus. In: Kasper DL, Braunwald E, Fauci A, Hauser S, Longo D Jameson JL (eds.). *Harrison's Principle of Internal Medicine*. 16.ed. McGraw-Hill, 2004; pp. 3779–829.
6. Savas S, Köro lu BK, Koyuncuo lu HR, Uzar E, Celik H, Tamer NM. The effects of the diabetes related soft tissue hand lesions and the reduced hand strength on functional disability of hand in type 2 diabetic patients. *Diabetes Res Clin Pract* 2007; 77(1):77–83.
7. Report of a WHO Scientific Group. The burden of musculoskeletal conditiona at the start of the new millinium, WHO Technical Report Series 919.2003. Available at URL: http://whqlibdoc.who.int/trs/WHO_TRS_919.pdf
8. Hoff, Ole M et al. The association between diabetes mellitus, glucose, and chronic musculoskeletal complaints. Results from the Nord-Trøndelag Health Study. *BMC musculoskeletal disorders* 2008;9(1):160.
9. Ahmad I, Nadeem D, Aziz A. Musculoskeletal disorder in long-standing DM Cases. *JPOA* 2008 FEB;20(1):38

10. Lebiedz-Odrobina D, Kay J. Rheumatic manifestation of diabetes mellitus. *Rheum Dis Clin N Am* 2010; 36(4):681–99
11. Arkkila PE, Gautier JF. Musculoskeletal disorders in diabetes mellitus: an update. *Best Pract Res Clin Rheumatol* 2003; 17(6):945–70.
12. Kapoor A, Sibbitt WL Jr. Contractures in diabetes mellitus: the syndrome of limited joint mobility. *Semin Arthritis Rheum* 1989; 18(3):168–80.
13. Bucala R, Tracey KJ, Cerami A. Advanced glycosylation products quench nitric oxide and mediate defective endothelium-dependent vasodilatation in experimental diabetes. *J Clin Invest* 1991; 87(2):432–8.
14. Traisman HS, Traisman ES, Marr TJ, Wise J. Joint contractures in patients with juvenile diabetes and their siblings. *Diabetes Care* 1978; 1(6):360–1.
15. Rosebloom AL, Silverstein JH, Riley WJ, Maclaren NK. Limited joint mobility in childhood diabetes: family studies. *Diabetes Care* 1983; 6(4):370–3.
16. Gamstedt A. Hand abnormalities in patients with NIDDM. *Prog Diabetes* 1993;4:1–6.
17. Buckingham BA, Uitto J, Sandborg C, et al. Scleroderma-like changes in insulin-dependent diabetes mellitus: clinical and biochemical studies. *Diabetes Care* 1984;7:163–9.
18. Rosenbloom A. Connective tissue disorders in diabetes. *International textbook of diabetes mellitus*. 2nd ed. Chichester: John Wiley, 1997:1517–31.
19. Crisp AJ, Heathcote JG. Connective tissue abnormalities in diabetes mellitus. *J R Coll Physicians Lond* 1984;18:132–41.
20. Jennings AM, Milner PC, Ward JD. Hand abnormalities are associated with the complications of diabetes in type 2 diabetes. *Diabet Med* 1989;6:43–7.

21. Starkman HS, Gleason RE, Rand LI, et al. Limited joint mobility (LJM) of the hand in patients with diabetes mellitus: relation to chronic complications. *Ann Rheum Dis* 1986;45:130–5.
22. Eadington DW, Patrick AW, Frier BM. Association between connective tissue changes and smoking habit in type 2 diabetes and in non-diabetic humans. *Diabetes Res Clin Pract* 1991;11:121–5.
23. Yosipovitch G, Loh KC, Hock OB. Medical pearl: Scleroderma-like skin changes in patients with diabetes mellitus. *J Am Acad Dermatol* 2003; 49(1):109–11.
24. Seibold JR. Digital sclerosis in children with insulin-dependent diabetes mellitus. *Arthritis Rheum* 1982; 25(11):1357–61.
25. Kuryliszyn-Moskal A, Dubicki A, Zarzycki W, Zonnenberg A, Górska M. Microvascular abnormalities in capillaroscopy correlate with higher serum IL-18 and sE-selectin levels in patients with type 1 diabetes complicated by microangiopathy. *Folia Hystoch Cytobiol* 2011; 49(1):104–10.
26. Fitzcharles MA, DUBY S, Wadell RW, Banks E, Karsh J. Limitation of joint mobility (cheiroarthropathy) in adult with noninsulin-dependent diabetic patients. *Ann Rheum Dis* 1984; 43(2):251–4.
27. Arkilla PE, Kantola IM, Vikari JS, Rönnemaa T, Vähätalo MA. Limited joint mobility is associated with the presence but does not predict the development of microvascular complication in type 1 diabetes. *Diabet Med* 1996; 13(9):828–33.
28. Trujillo-Santos AJ. Diabetic muscle infarction: an underdiagnosed complication of long-standing diabetes. *Diabetes Care* 2003; 26(1):211–5.

29. Kapur S, Brunet JA, McKendry RJ. Diabetic muscle infarction: case report and review. *J Rheumatol* 2004; 31(1):190–4.
30. Kattapuram TM, Suri R, Rosol MS, Rosenberg AE, Kattapuram SV. Idiopathic and diabetic skeletal muscle necrosis evaluation by magnetic resonance imaging. *Skeletal Radiol* 2005; 34(4):203–9.
31. Bjornskov EK, Carry MR, Katz FH, Lefkowitz J, Ringel SP. Diabetic muscle infarction: a new perspective on pathogenesis and management. *Neuromuscul Disord* 1995; 5(1):39–45.
32. Palmer GW, Greco TP. Diabetic thigh muscle infarction in association with antiphospholipid antibodies. *Semin Arthritis Rheum* 2001; 30(4):272–80.
33. Ardic F. , Soyupek F. , Kahraman Y. , *et al* . The musculoskeletal complication seen in type II diabetics : predominance of hand involvement .*Clinical Rheumatology* ,Springer London 2003;22(3);229-233.
34. Gudmundsson KG, Arngrimsson R, Sigfusson N, *et al*. Epidemiology of Dupuytren's disease: clinical, serological, and social assessment. The Reykjavik Study. *J Clin Epidemiol* 2000;53:291–6.
35. Kozin F. Reflex sympathetic dystrophy syndrome: a review. *Clin Exp Rheumatol* 1992;10:401–9.
36. Noble J, Heathcote JG, Cohen H. Diabetes mellitus in the aetiology of Dupuytren's disease. *J Bone Joint Surg [Br]* 1984;66:322–5.
37. Forgacs SS. Diabetes mellitus and rheumatic disease. *Clin Rheum Dis* 1986;12:729–53.
38. Lennox IA, Murali SR, Porter R. A study of the repeatability of the diagnosis of Dupuytren's contracture and its prevalence in the Grampian region. *J Hand Surg [Br]* 1993;18:258–61.

39. Gudmundsson KG, Arngrimsson R, Sigfusson N, et al. Epidemiology of Dupuytren's disease: clinical, serological, and social assessment. The Reykjavik Study. *J Clin Epidemiol* 2000;53:291–6.
40. Chammass M, Bousquet P, Renard E, Poirier JL, Jaffiol C, Allieu Y. Dupuytren's disease, carpal tunnel syndrome, trigger finger, and diabetes mellitus. *J Hand Surg Am* 1995; 20(1):109–14
41. Hart MG, Hooper G. Clinical associations of Dupuytren's disease. *Postgrad Med J* 2005; 81(957):425–8.
42. Hurst LC, Badalamente MA, Hentz VR, Hotchkiss RN, Kaplan FT, Meals RA *et al.* Injectable collagenase clostridium histolyticum for Dupuytren's contracture. *N Engl J Med* 2009; 361(10):968–79.
43. Reeves B. The natural history of the frozen shoulder syndrome. *Scand J Rheumatol* 1975;4:193–6.
44. Forgács SS. Endocrine and hemoglobin arthropathies: diabetes mellitus. *Rheumatology*. London: Mosby-Year Book Europe Ltd, 1994.
45. Griggs SM, Ahn A, Green A. Idiopathic adhesive capsulitis. A prospective functional outcome study of nonoperative treatment. *J Bone Joint Surg [Am]* 2000;82:1398–407.
46. Bridgman JF. Periarthritis of the shoulder and diabetes mellitus. *Ann Rheum Dis* 1972;31:69–71.
47. Lequesne M, Dang N, Bensasson M, et al. Increased association of diabetes mellitus with capsulitis of the shoulder and shoulder-hand syndrome. *Scand J Rheumatol* 1977;6:53–6.

48. Balci N, Balci MK, Tuzuner S. Shoulder adhesive capsulitis and shoulder range of motion in type II diabetes mellitus: association with diabetic complications. *J Diabetes Complications* 1999;13:135–40.
49. Arkkila PE, Kantola IM, Viikari JS, et al. Shoulder capsulitis in type I and II diabetic patients: association with diabetic complications and related diseases. *Ann Rheum Dis* 1996;55:907–14.
50. Tighe CB, Oakley WS Jr. The prevalence of a diabetic condition and adhesive capsulitis of the shoulder. *South Med J* 2008; 101(6):591–5.
51. Bunker TD, Anthony PP. The pathology of frozen shoulder. A Dupuytren-like disease. *J Bone Joint Surg Br* 1995; 77(5):677–83.
52. Pal B, Anderson J, Dick WC, Griffiths ID. Limitation of joint mobility and shoulder capsulitis in insulin- and non-insulindependent diabetes mellitus. *Br J Rheumatol* 1986; 25(2):147–51.
53. Balci N, Balci MK, Tüzüner S. Shoulder adhesive capsulitis and shoulder range of motion in type II diabetes mellitus: association with diabetes complications. *J Diabetes Complications* 1999; 13(3):135–40.
54. Sheridan MA, Hannafin JA. Upper extremity: emphasis on frozen shoulder. *Orthop Clin North Am* 2006; 37(4):531–9.
55. Cagliero E, Apruzzese W, Perlmutter GS, Nathan DM. Musculoskeletal disorders of the hand and shoulder in patients with diabetes mellitus. *Am J Med* 2002; 112(6):487–90.
56. Yosipovitch G, Yosipovitch Z, Karp M, Mukamel M. Trigger finger in young patients with insulin dependent diabetes. *J Rheumatol* 1990; 17(7): 951–2.
57. Kameyama M, Meguro S, Funae O, Atsumi Y, Ikegami H. The presence of limited joint mobility is significantly associated with multiple digit

- involvement by stenosing flexor tenosynovitis in diabetics. *J Rheumatol* 2009; 36(8):1686–90.
58. Koh S, Nakamura S, Hattori T, Hirata H. Trigger digits in diabetes: their incidence and characteristics. *J Hand Surg Eur Vol* 2010; 35(4):302–5.
59. Leden I, Schersten B, Svensson B, et al. Locomotor system disorders in diabetes mellitus. Increased prevalence of palmar flexor tenosynovitis. *Scand J Rheumatol* 1983;12:260–2.
60. Mader R, Sarzi-Puttini P, Atzeni F, Olivieri I, Pappone N, Verlaan JJ *et al.* Extraspinal manifestations of diffuse idiopathic skeletal hyperostosis. *Rheumatology (Oxford)* 2009; 48(12):1478–81.
61. Resnick D, Niwayama G. Dish. In: Resnick D, Niwayama G (eds.). *Diagnosis of bone and joint disorders*. 2.ed. Philadelphia: WB Saunders, 1983; p.2436.
62. Utsinger PD. Diffuse idiopathic skeletal hyperostosis. *Clin Rheum Dis* 1985; 11(2):325–51.
63. Mader R, Dubenski N, Lavi I. Morbidity and mortality of hospitalized patients with diffuse idiopathic skeletal hyperostosis. *Rheumatol Int* 2005; 26(2):132–6.
64. Kiss C, Szilágyi M, Paksy A, Poór G. Risk factors for diffuse idiopathic skeletal hyperostosis: a case-control study. *Rheumatology (Oxford)* 2002; 41(1):27–30.
65. Sencan D, Elden H, Nacitarhan V, Sencan M, Kaptanoglu E. The prevalence of diffuse idiopathic skeletal hyperostosis in patients with diabetes mellitus. *Rheum Int* 2005; 25(7):518–21.

66. Sarzi-Puttini P, Atzeni F. New developments in our understanding of DISH (diffuse idiopathic skeletal hyperostosis). *Clin Opin Rheumatol* 2004; 16(3):287–92.
67. Crisp AJ, Heathcote JG. Connective tissue abnormalities in diabetes mellitus. *J R Coll Physicians Lond* 1984;18:132–41.
68. Rosenbloom A. Connective tissue disorders in diabetes. *International textbook of diabetes mellitus*. 2nd ed. Chichester: John Wiley, 1997:1517–31.
69. Lipson S.J. Low back pain. *Textbook of rheumatology*. 5th ed. Philadelphia: Saunders WB, 1997.
70. Bland JH, Frymoyer JW, Newberg AH, et al. Rheumatic syndromes in endocrine disease. *Semin Arthritis Rheum* 1979;9:23–65.
71. Nicholas A., Nicki R. , Brain R. , John A. Musculoskeletal disorders ,Regional periarticular pain in Davidson s principles & practice of medicine 20th edn. , CHURCHILL livingstone 2006;1079-80.
72. Jung Y, Hohmann TC, Gerneth JA, *et al.* Diabetic hand syndrome. *Metabolism* 1971;20:1008–15.
73. Deal C. The endocrine system. *Oxford textbook of rheumatology*. 2nd ed. Oxford: Oxford University a Press, 1998:282–5.
74. Nashel DJ. Entrapment neuropathies. In: Klippel JH, Dieppe PA (eds.). *Rheumatology*. 2.ed. vol.1, S-4. London: Mosby, 1998; pp.16.1–16.12.
75. Jillapalli D, Shefner JM. Electrodiagnosis in common mononeuropathies and plexopathies. *Semin Neurol* 2005; 25(2):196–203.
76. Papanas N, Maltezos E. The diabetic hand: a forgotten complication? *J Diabetes Complications* 2010; 24(3):154–62.

77. Perkins BA, Olaleye D, Bril V. Carpal tunnel syndrome in patients with diabetic polyneuropathy. *Diabetes Care* 2002; 25(3):565–9.
78. Comi G, Lozza L, Galardi G, Ghilardi MF, Medaglini S, Canal N. Presence of carpal tunnel syndrome in diabetics: effects of age, sex, diabetes duration and polyneuropathy. *Acta Diabetol Lat* 1985;22(3):259–62.
79. Geoghegan JM, Clark DI, Bainbridge LC, Smith S, Hubbard R. Risk factors in carpal tunnel syndrome. *J Hand Surg Br* 2004; 29(4):315–20.
80. Becker J, Nora DB, Gomes I, Stringari FF, Seitensus R, Panosso J *et al.* An evaluation of gender, obesity, age and diabetes mellitus as risk factors for carpal tunnel syndrome. *Clin Neurophysiol* 2002; 113(9):1429–34.
81. McClure P. Evidence-based practice: an example related to the use of splinting in a patient with carpal tunnel syndrome. *J Hand Ther* 2003; 16(3):256–63.
82. Girlanda P, Dattola R, Venuto C, Mangiapane R, Nicolosi C, Messina C. Local steroid treatment in idiopathic carpal tunnel syndrome: short- and long-term efficacy. *J Neurol* 1993; 240(3):187–90.
83. Yasuda H, Terada M, Maeda K, Kogawa S, Sanada M, Haneda M *et al.* Diabetic neuropathy and nerve regeneration. *Prog Neurobiol* 2003; 69(4):229–85.
84. Kennedy JM, Zochodne DW. Impaired peripheral nerve regeneration in diabetes mellitus. *J Peripher Nerv Syst* 2005; 10(2):144–57.
85. Phalen GS. Reflections on 21 years' experience with the carpal-tunnel syndrome. *JAMA* 1970;212:1365–7.
86. Comi G, Lozza L, Galardi G, *et al.* Presence of carpal tunnel syndrome in diabetics: effect of age, sex, diabetes duration and polyneuropathy. *Acta Diabetol Lat* 1985;22:259–62.

87. Stevens JC, Sun S, Beard CM, et al. Carpal tunnel syndrome in Rochester, Minnesota, 1961 to 1980. *Neurology* 1988;38:134–8.
88. Bayne O, Lu EJ. Diabetic Charcot's arthropathy of the wrist. Case report and literature review. *Clin Orthop* 1998;357:122–6.
89. Giurini JM, Chrzan JS, Gibbons GW, Habershaw GM. Charcot's disease in diabetic patients. Correct diagnosis can prevent deformity. *Postgrad Med* 1991; 89(4):163–9.
90. Brower AC, Allman RM. Pathogenesis of the neurotropic joint: neurotraumatic vs. neurovascular. *Radiology* 1981; 139(2):349–54.
91. Jeffcoate WJ, Game F, Cavanagh PR. The role of proinflammatory cytokines in the neuropathic osteoarthropathy: (acute Charcot foot) in diabetes. *Lancet* 2005; 366(9502):2058–61.
92. Sequeira W. The neuropathic joint. *Clin Exp Rheumatol* 1994; 12(3):325–37.
93. Förgacs SS. Diabetes mellitus and rheumatic disease. *Clin Rheum Dis* 1986; 12(3):729–53.
94. Ahmadi ME, Morrison WB, Carrino JA, Schweitzer ME, Raikin SM, Ledermann HP. Neuropathic arthropathy of the foot with and without superimposed osteomyelitis: MR imaging characteristics. *Radiology* 2006; 238(2):622–31.
95. Selby PL, Young MJ, Boulton AJ. Bisphosphonates: a new treatment for diabetic Charcot neuroarthropathy? *Diabet Med* 1994; 11(1):28–31.
96. Pitocco D, Ruotolo V, Caputo S, Mancini L, Collina CM, Manto A *et al.* Six-month treatment with alendronate in acute Charcot neuroarthropathy: a randomized controlled trial. *Diabetes Care* 2005; 28(5):1214–5.

97. Bem R, Jirkovská A, Fejfarová V, Skibová J, Jude EB. Intranasal calcitonin in the treatment of acute Charcot neuroosteoarthropathy: a randomized controlled trial. *Diabetes Care* 2006; 29(6):1392–4.
98. Kozin F. Reflex sympathetic dystrophy syndrome: a review. *Clin Exp Rheumatol* 1992;10:401–9.
99. Marshall AT, Crisp AJ. Reflex sympathetic dystrophy. *Rheumatology (Oxford)* 2000;39:692–5.

ANNEXURE – I - INFORMED CONSENT FORM

CLINICAL AND RADIOLOGICAL STUDY OF MUSCULOSKELETAL MANIFESTATIONS OF DIABETES MELLITES – ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY

Objective and purpose of the study: This research is intended to study Musculoskeletal manifestations of Diabetes Mellites. The principal investigator of the study is Dr. _____ under the guidance of Dr. _____. This study is intended for early diagnosis of musculoskeletal disorders in diabetes and hence to reduce morbidity associated with it.

Procedure: If you agree to be part of the research study you will be asked the relevant history in relation to musculoskeletal disorders and will be subjected for clinical examination. If necessary radiological investigation will be done.

Risk and Benefits: There is no risk associated with study. Early diagnosis of musculoskeletal disorders will help to improve the quality of life of patient.

Alternatives: Taking part in this study is voluntary. You may choose not to take part in this study, or if you decide to take part you can later change my mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsorer may stop your participation in this study any time. If you choose not to take part in the study you will receive the standard treatment for patients with your condition

Privacy and Confidentiality: All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy: Does not apply to this research

VOLUNTARY PARTICIPATION/ WITHDRAWAL:Your participation in this study is entirely voluntary and you may withdraw from the study at any time.

Financial incentives for participation

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results

The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MD degree, review and publishing.

CONSENT FORM

I voluntarily agree to take part in this study by signing on the line below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicated that I have read this entire consent form or it has been read to me, and has been explained to me in my vernacular language and had all my questions answered. I will be given a copy of this consent form.

Signature /Left Thumb print of the Participant or legally authorized representative.

Participant's Name/ :

Signature/ Left Thumb

Impression of the participant's :

Signature/ Left Thumb Impression. :

Witness's Name :

Signature/ Left Thumb Impression. :

Investigators name and Signature :

Date and Place :

Date:

Place :

ANNEXURE – II - PROFORMA

1. SL.NO
- 2 NAME:
3. AGE:
4. SEX
5. OCCUPATION:
6. RELIGION:
7. I.P. NO./O.P. NO.:
8. ADDRESS:
9. DATE OF ADMISSION:
- 10.DATE OF DISCHARGE:
11. OUTCOME :

HISTORY:

PRESENTING COMPLAINTS

HISTORY OF PRESENTING ILLNESS :

PAST HISTORY:

Significant personal history:

Significant family history

TREATMENT HISTORY:

Received any treatment for similar complaints in the past

GENERAL PHYSICAL EXAMINATION

Pallor: Yes/No

Icterus: Yes/No

Lymphadenopathy: Yes/No

Cyanosis: Yes/No

Clubbing: Yes/No

Edema: Yes/No

Vital signs :

Pulse

Blood Pressure

Respiratory rate

Temperature

PaO₂

Any significant findings

SYSTEMIC EXAMINATION:

R. S.:

C.V.S.:

P.A.:

C.N.S.:

Sugars:

FBS:

PL:

PD:

HbA1C:

MUSCULOSKELETAL DISORDERS IN DIABETES:

LIMITED JOINT MOBILITY SYNDROME: prayer sign, thick waxy skin on dorsal surface, paraesthesia or pain on passive extension of fingers	
DUPUYTREN CONTRACTURE: palmar or digital thickening, contractures in ring or middle fingers	
TRIGGER FINGER: fingers locked in flexion or extension	
CARPEL TUNNEL SYNDROME: paraesthesia over thumb, index, middle finger, lateral half of ring finger.	
FROZEN SHOULDER	
DIFFUSE IDIOPATHIC SKELETAL HYPEROSTOSIS: early morning stiffness in back.	
CHARCOT JOINT: joint destruction	
REFLEX SYMPATHETIC DYSTROPHY: local or diffuse pain with swelling, tropic changes.	
DIABETIC AMYOTROPHY	