Rheumatology and Diabetology are two branches of medicine that often go together in various features. Diabetics suffer from specific rheumatological syndromes and most rheumatological disorders (and indeed their treatment) are associated with a higher prevalence of glucose intolerance or diabetes than the general population. This article however shall review only the important rheumatological problems in diabetes mellitus. Connective tissue defects in diabetes mellitus may be classified as syndromes affecting the bones, joints, skin, periarticular tissues etc. and these are shown in Table 1.

Table 1. Connective tissue disorders in diabetes mellitus

<table>
<thead>
<tr>
<th>Bone disorders</th>
<th>Joint disorders</th>
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<tbody>
<tr>
<td>Diabetic Osteopenia</td>
<td>Osteoarthritis</td>
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<tr>
<td>Hyperostosis</td>
<td>Neurogenic arthritis</td>
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<tr>
<td>Osteolysis</td>
<td>Infective arthritis</td>
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<tr>
<td>Osteomyelitis</td>
<td>Limited Joint Mobility</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Skin and periarticular disorders</th>
<th>Muscle disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scleroderma</td>
<td>Myalgia</td>
</tr>
<tr>
<td>Diabetic cirrhosis</td>
<td>Myositis</td>
</tr>
<tr>
<td>Dupuytren’s Disease</td>
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<tr>
<td>Carpal Tunnel Syndrome</td>
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<tr>
<td>Flexor Tenosynovitis</td>
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<tr>
<td>Adhesive capsulitis of the shoulder</td>
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</table>

Bone diseases in diabetes mellitus

Diabetic Osteopenia is known to occur in both type-1 (IDDM) (Lopez-Ibarra et al, 1992) and type 2 (NIDDM) diabetic patients (Patsan and Cohen, 1978). Although the etiopathogenesis of osteopenia remains obscure, various hypotheses have been put forward. Abnormal mineral and Vitamin D metabolism, inhibition of cartilage synthesis, its calcification and ossification have been proposed as possible causes for the osteopenia (Goodman and Hori, 1984). Diabetic patients lack the trophic effect of insulin on their tissues especially bones (Frank et al, 1994). In addition, hypoalbuminemia associated with diabetic nephropathy predisposes to diminished bone density (Auwerx et al, 1988). However the widely acclaimed etiology is the concept of osteocalcin (Ishide and Seino, 1988), a gamma - carboxy glutamate protein. The levels of the anabolic bone hormone has been found to be lower in diabetic patients in compared to controls.

Children with type 1 diabetes (IDDM) have been shown to have diminished vertebral density, when compared with age and gender matched population (McNair et al, 1978). This decrease in bone mass density, specifically affects the trabecular bone. Attributes to the cause are intermittent hyperglycaemia, microangiopathy and abnormal Vitamin D metabolism (Forgacs, 1997; Health and Melton, 1980). Associated contributing factors are age and diminished physical activity. Subjects with type 2 diabetes (NIDDM) have differential involvement of bones. Most show diabetic osteopenia but some type 2 diabetic patients demonstrate an increase in bone density termed as hyperostosis, especially those on oral hypoglycaemic agents (Van Dale et al, 1994).

Hyperostosis

This is a fairly characteristic manifestation of NIDDM and may involve the spine (hyperostotic spondylosis) (Resnick and Niwayama, 1976), skull bones (Hyperostosis Frontalis Interna), pelvis (osteoitis condensans iliiasis) heel or elbow (calcaneal and olecranon spurs). It is more common in older and obese males. Spinal hyperostosis resembles Ankylosing spondylitis but mainly involves
the thoracic region and does not affect the inter-vertebral disc spaces. Apart from obesity as an aetiological factor, the possibility of insulin like growth factor (IGF) stimulating bone growth has been postulated. The growth hormone levels are comparatively less in these patients.

**Osteolysis**
Diabetics can manifest with osteolysis of the forefoot with either localized or generalized osteoporotic changes in distal metatarsals and proximal phalanges. Juxta articular erosions, local erythema and pain are characteristic features (Rosenbloom et al, 1977).

**Osteomyelitis**
Infection of the bony tissues especially the narrow cavity is more common in diabetic patients due to the immuno suppressed state (Bassmann and Sapir, 1992). Common organisms include staphylococcus, pseudomonas etc. Malignant otitis externa (Hernandez, 1994) is a fairly specific lesion occurring in diabetics involving the bony external canal by pseudomonas species.

**Ionic homeostasis in diabetes**
The major disturbance in diabetes is hypercalcaemia, the etiology of which is obscure. However the serum levels of calcium are well within the normal range (Verhaege et al, 1990). During diabetic ketoacidosis, there is a variable loss of total body magnesium and phosphate necessitating replacement.

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**Articular manifestations of diabetes mellitus**

**Osteoarthritis**: is the commonest joint problem in diabetes mellitus (Figure 1). Destruction of articular cartilage, bone-overgrowth with lipping and spur formation are characteristic lesions. Studies have confirmed that in age and gender matched diabetic patients, the incidence of osteoarthritis was higher, earlier in onset and greater in severity (Weiss et al, 1981). Diminished synthesis of polysaccharides necessary for the normal function of articular cartilage may also lead to degenerative changes.

**Neurogenic arthritis**: is fairly common complication of diabetes and occurs due to persistent damage to articular surfaces secondary to loss of proprioception (due to Diabetic peripheral and autonomic neuropathy) (Rossi and Fossaluzza, 1985). Typically, this involves the joints of the forefoot, metatarsophalangeal and interphalangeal joints.

**Infective arthritis**: occur in diabetes mellitus more frequently than in non-diabetics owing to immuno-compromised state. For example, E. Coli arthritis of shoulder, pyoarthritis of hip caused by campylobacter fetus and streptococcal septic arthritis of the small joints of hands (Ho-G, 1992).

**Syndrome of limited joint mobility (LJM)**
This is a fairly specific and unique complication of both type 1 and type 2 diabetes (Fitzcharles et al, 1984). LJM consists of painless, marked restriction of the flexion and extension of joints especially the interphalangeal, metacarpophalangeal (MCP) and wrist joints. It is associated with thick, tight, waxy skin. This syndrome was first described in children with IDDM and comprised of short stature, delayed sexual maturation and early microvascular complications in addition to limited joint mobility (Kennedy et al, 1982).

Although a genetic basis has been suggested, no sexual or racial prevalence exists in the occurrence of this syndrome. Changes commence in the metacarpophalangeal and proximal interphalangeal joints of little finger and extend more medially. The distal interphalangeal may also be involved.

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![Figure 1. Osteoarthritis of the knee.](image-url)
Rheumatological manifestations of diabetes mellitus

less commonly, as also the wrist, elbow and ankle joints and the cervical, and thoracolumbar spine.

A simple screening method is to find out if the patient is able to keep his hands folded in a “praying” position (Figure 2). The severity of LSM may be classified as shown in Table 2.

<table>
<thead>
<tr>
<th>Grade</th>
<th>No limitation</th>
<th>Equivocal/Unilateral findings</th>
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<tbody>
<tr>
<td>1</td>
<td>Mild limitation</td>
<td>One or two proximal interphalangeal joints or large joint or metacarpophalangeal joints bilaterally</td>
</tr>
<tr>
<td>2</td>
<td>Moderate limitation</td>
<td>Three or more proximal interphalangeal joints or one finger joints and large joint bilaterally</td>
</tr>
<tr>
<td>3</td>
<td>Severe limitation</td>
<td>Hand deformity at rest, or associated cervical spine involvement</td>
</tr>
</tbody>
</table>

Although LJM and glycaemic control (Buithieu et al, 1988) has no relationship, considerable importance has been given to its association with other microvascular complications like retinopathy and nephropathy (Lawson et al, 1983). Autonomic dysfunction has been noted in upto 50% of type 1 diabetic patients with LJM. Similarly pulmonary changes in form of a restrictive defect may be present in approximately 80% of patients with LJM. Neuropathy is also four times more common in patients with LJM.

LJM has been associated with skin abnormalities like thickened waxy tight bound skin on the hand and forearm. The underlying pathology is an increase in large fibre collagen of subcutaneous tissue.

Skin and periarticular abnormalities in diabetes mellitus

Scleroderma diabeticorum is seen in patients with both type 1 and type 2 diabetes (Buckingham et al, 1984). Middle aged obese males are more likely to develop this lesion and it is associated with ischaemic heart disease, hypertension and retinopathy. It involves the skin of posterolateral aspect of the neck, upper back face and upper trunk making it difficult to pinch up the skin. There may be hard pitting edema. Histologically this lesion shows hyperplastic collagen (Collier et al, 1986).

Dupuytren’s contracture
This refers to subcutaneous fibrosis of the palmar aponeurosis of the hands (Bergaouiri and Dibej, 1991). Incidence in diabetes is around 40%. It commonly affects the third and fourth digits. Surgical excision may be required in severe cases with deformity. Microscopically, it consists of large quantities of Type III collagen with higher contents of collagen - water - chondroitin sulfate.

Carpal tunnel syndrome
Compression of median nerve within the carpal tunnel at the wrist is the commonest entrapment neuropathy, resulting in paraesthesia of thumb, index finger and little finger with pain that is worse at night (Florack and Miller, 1992).

Flexor tenosynovitis: also termed “trigger
The table below lists the differential diagnosis of diabetic hand syndromes:

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Structure Involved</th>
<th>Special Features</th>
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<tbody>
<tr>
<td>Painless</td>
<td>5th finger radially</td>
<td>No disability; Associated with thick/waxy skin of closure of hand</td>
</tr>
<tr>
<td>Dupuytren's Contracture</td>
<td>3rd and 4th finger</td>
<td>Female predominance; Often precedes diabetes</td>
</tr>
<tr>
<td>Painful</td>
<td>All finger</td>
<td>Ulnar and median nerve involvement</td>
</tr>
<tr>
<td>Carpal Tunnel Syndrome</td>
<td>1st, 3rd and 4th finger</td>
<td>Disabling, Female predominance</td>
</tr>
<tr>
<td>Flexor Tenoynovitis</td>
<td>All fingers</td>
<td>Disabling, hard, thick, palmar skin with soft dorsum</td>
</tr>
<tr>
<td>Stiff hand Syndrome</td>
<td>All fingers</td>
<td>Usually bilateral</td>
</tr>
<tr>
<td>Shoulder hand Syndrome</td>
<td>All fingers</td>
<td></td>
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</table>

Adhesive capsulitis of shoulder and shoulder hand.
Thickening of the fibrous capsule and its adherence to the humeral head of the shoulder joint results in marked reduction in movement joints. This syndrome precedes by swelling, erythema and pain of the shoulder joint associated with hyperhidrosis of hands (Mavrikakis et al, 1989). After weeks to months, the local changes disappear with residual dystrophic changes, finger contracts and osteoporosis of the bones of the upper limb.

Table 3 shows the differential diagnosis of various diabetic hand syndromes.

Muscle disorders in diabetes mellitus
Myalgia is a common complaint in middle-aged elderly people and investigations often reveal diabetes mellitus.

Pyomyositis
Infective pyomyositis by anaerobic organisms, although not very common, is a recognised feature of diabetes mellitus.

Rheumatological manifestations of diabetes mellitus


