Chapter 2
Various Kinds of Bone Disease in Diabetes: Rheumatic Conditions

Hitoshi Goto

Abstract Diabetes mellitus (DM) is a chronic systemic disease with a wide range of complications in the musculoskeletal system. Diabetic patients suffer from diverse rheumatic conditions, which are common and, while not life threatening, are an important cause of morbidity, pain, and disability that affect their quality of life. Joints affected by diabetes include peripheral joints and the axial skeleton. This article reviews the rheumatic conditions that are associated with DM and the pathophysiologic relationships that might link these conditions (Table 2.1). A number of fibrosing conditions of the hands and shoulder are recognized, including limited joint mobility, Dupuytren's contracture, flexor tenosynovitis, carpal tunnel syndrome, and adhesive capsulitis. Charcot osteoarthropathy is an important cause of deformity and amputation associated with peripheral neuropathy. Diabetes patients are more prone to diffuse idiopathic skeletal hyperostosis, osteoarthritis, diabetic muscle infarction, crystal-induced arthritis, osteoporosis, sarcopenia, and complex regional pain syndrome (CRPS) type 1. Management of these conditions requires early recognition and close liaison between diabetes and rheumatology specialists.

Keywords Rheumatic conditions • Diabetes mellitus • Joint • Hand • Shoulder • Muscle • Bone

2.1 Limited Joint Mobility (Diabetic Cheiroarthropathy)

Limited joint mobility (LJM), or diabetic cheiroarthropathy, is characterized by thick, tight, waxy skin, mainly on the dorsal aspect of the hands, with flexion contracture of the metacarpophalangeal and interphalangeal joints (Fig. 2.1). It is a common complication of type 1 and type 2 diabetes mellitus (DM). The prevalence of LJM ranges between 30 and 58 % among patients with type 1 DM and between 45 and 76 % among those with type 2 DM, as compared between 4 and 20 % among individuals without DM [1–5]. LJM is usually painless; however, in

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Table 2.1 Rheumatic complications of DM

Conditions unique to DM
Diabetic muscle infarction
Conditions occurring more frequently in DM
Limited joint mobility
Dupuytren's contracture
Stenosing flexor tenosynovitis (trigger finger)
Adhesive capsulitis of shoulder
Carpal tunnel syndrome
Neuropathic arthropathy
Conditions sharing risk factors of DM and metabolic syndrome
Diffuse idiopathic skeletal hyperostosis
Osteoarthritis
Gout
Pseudogout
Calcific periarthritis of shoulder
Osteoporosis
Sarcopenia

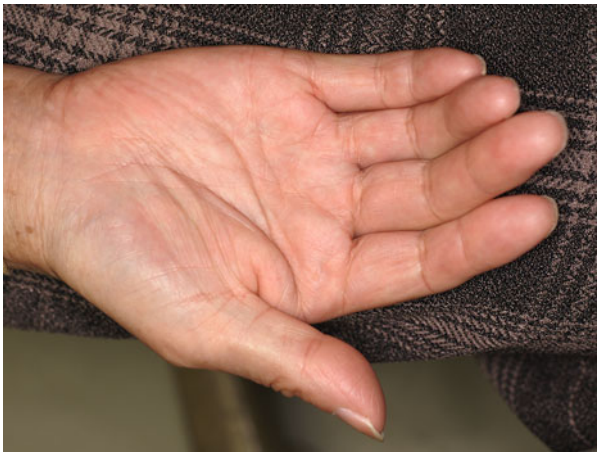

Fig. 2.1 Limited joint mobility.
Flexion contracture of the metacarpophalangeal and proximal interphalangeal joints in fingers

the early stage, slight pain and paresthesia may develop. LJM is diagnosed based on the presence of the characteristic clinical signs on physical examination. The prayer sign is defined as an inability to oppose the palmar surfaces of the hands and fingers with the wrists dorsiflexed. The tabletop sign is defined as the entire surface of the palm and fingers cannot make contact with a flat surface when patients lay their palms on a tabletop with the fingers spread apart. The long duration of DM and poor glycemic control possibly predispose patients to developing LJM and influencing its progression as LJM is associated with the presence of retinopathy and nephropathy [6]. The pathophysiology is thought about as follows. Increased glycosylation and degradation of collagen in the skin and periarticular tissues, diabetic

microangiopathy, and diabetic neuropathy have been implicated as contributing factors [7]. The treatment of LJM is mainly physical therapy to increase the range of motion. Improved glycemic control may not reverse the conditions but may help to prevent future progression.

2.2 Dupuytren's Contracture

Dupuytren's contracture is characterized by palmar or digital thickening, tethering, pretendinous bands, and flexion contractures of the fingers (Fig. 2.2). In patients with diabetes, both genders are equally affected, and the ring and middle finger are more commonly involved, while in nondiabetic patients, men are more likely affected, and the small finger is more involved. The prevalence of Dupuytren's contracture in diabetic patients ranges between 16 and 42 % [8, 9] compared with 13 % in the general population [10]. The prevalence is higher in patients who are older and with a longer duration of DM. Patients with Dupuytren's contracture should be evaluated for DM, as 13–39 % of them are found to have DM [11]. The pathophysiology of Dupuytren's contracture is likely to be multifactorial. Genetic predisposition explains the higher prevalence in some population over others. Trauma, long-term hyperglycemia, microangiopathy, and ischemia are also important factors. Microvascular disease and ischemia will result in increased production of oxygen free radicals. Ischemia also stimulates platelets and macrophages to produce different cytokines such as interleukin 1, tumor necrosis factor, and growth factors, such as platelet-derived growth factor, epidermal growth factor, connective tissue growth factor, vascular endothelial growth factor, and basic fibroblast growth factor, resulting in local collagen overproduction and fibrosis [12].

Treatment consists of optimizing glycemic control and physiotherapy. Recently, injections of collagenase clostridium histolyticum have been developed for the treatment of Dupuytren's contracture. Hurst et al. in 2009 [13] did a prospective,

Fig. 2.2 Dupuytren's contracture in the small finger characterized by palmar thickening, tethering, pretendinous bands, and flexion contractures

randomized, and placebo-controlled trial of 308 patients with Dupuytren's contracture, 6.5 % of whom had DM; up to three collagenase injections significantly reduced fixed flexion contractures and improved the joint range of motion. There were no recurrences after follow-up of up to 90 days. Only three serious adverse events were reported in this study, including two tendon ruptures and one case of complex regional pain syndrome. Surgery is required if the hand function is severely compromised.

2.3 Stenosing Flexor Tenosynovitis (Trigger Finger)

Flexor tenosynovitis is caused by fibrous tissue proliferation in the tendon sheath (Fig. 2.3) leading to limitation and restriction of the movement of the tendon, also known as trigger finger. The prevalence of flexor tenosynovitis is estimated at 11 % in diabetic patients, compared with less than 1 % in nondiabetic individuals [14]. Patients with DM are more likely to have multiple digit involvement. It most commonly involves the ring, middle fingers, and thumb. The occurrence of flexor tenosynovitis correlates significantly with the duration of DM, but not with glycemic control [15]. Treatment of flexor tenosynovitis includes modification of activities, splinting, NSAIDs, corticosteroid injection into the tendon sheath, and surgical release [16].

2.4 Carpal Tunnel Syndrome

Carpal tunnel syndrome (CTS) is an entrapment neuropathy caused by compression of the median nerve within the carpal tunnel. It is estimated to occur in 3.8 % of the general population [17]. The prevalence of CTS in diabetic patients is higher and

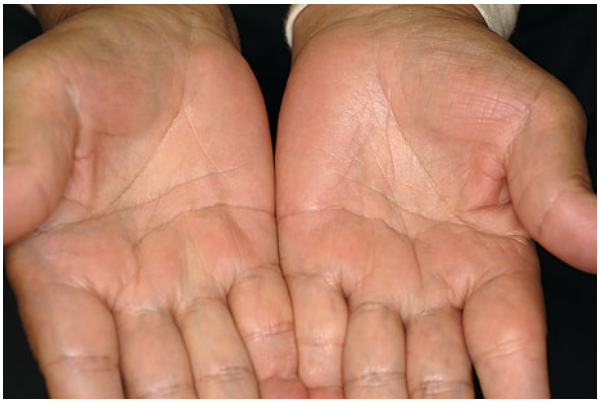

Fig. 2.3 Stenosing flexor tenosynovitis in the right small finger and the left ring finger characterized by fibrous tissue proliferation in the palmar tendon sheaths

estimated to occur in 14 % of patients without diabetic polyneuropathies and up to 30 % in those with diabetic polyneuropathies [18]. CTS prevalence increases as the DM duration extends. Increased prevalence of CTS in diabetes suggests the presence of intrinsic nerve pathology in addition to mechanical compression. The intrinsic nerve factors include loss of normal regenerative ability in the peripheral nerve because of microangiopathy, macrophage dysfunction, abnormalities in the retrograde cell body reaction, Schwann cell dysfunction, or decreased expression of neurotrophic factors and their receptors [19]. CTS manifests as pain, tingling, and paresthesia of the thumb, index, middle fingers, and the radial aspect of the ring finger. The symptoms may be improved by shaking or flicking the wrists known as "flick sign." A reduction of the pinch strength caused by the atrophy of the thumb ball and function of the affected hand may occur (Fig. 2.4). Symptoms tend to worsen at night. Bilateral CTS is common, but the symptoms may not occur simultaneously in both hands. It is usually diagnosed by history and clinical examinations, by percussion of the median nerve at the wrist (Tinel's test), by asking the patient to do wrist dorsiflexion (Phalen's test), or by employing the hand elevation test which is conducted by asking the patient to raise the affected hand and holding it in that position for 1 min. The test is considered positive if the patient experiences tingling and numbness in the median nerve distribution area [20, 21]. The diagnosis of CTS is confirmed by a nerve conduction study. Imaging studies, including magnetic resonance imaging (MRI) and ultrasonography (US), can be useful for this purpose. US is a simple, easy-to-perform, and noninvasive procedure. It has been implicated in the diagnosis of CTS as it can demonstrate the thickening of the median nerve, the flattening of the nerve within the tunnel, and the bowing of the flexor retinaculum, which are all features that indicate the presence of CTS. Several studies have concluded that the cross-sectional area is the most predictive measurement, but there is debate regarding the level within the tunnel

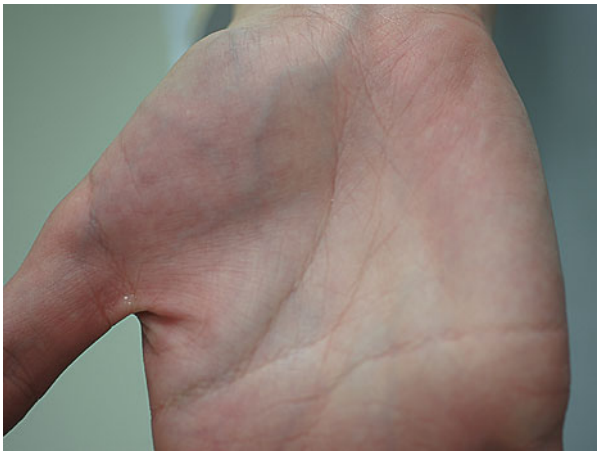

Fig. 2.4 The atrophy of the thumb ball due to median nerve palsy caused by carpal tunnel syndrome

that this measurement should be taken and what constitutes abnormal values. The sensitivity of US is 64.7 % [22]. An MRI will demonstrate swelling of the median nerve and increased signal intensity on T2-weighted images, indicating accumulation of the axonal transportation, myelin sheath degeneration, or edema, which are the signs to look out for when diagnosing CTS. An MRI shows the severity of the nerve compression and has a sensitivity of 96 %. However, its specificity is extremely low at 33–38 % [22]. The treatment options for CTS include splinting and local injection of corticosteroids or NSAIDs. Although corticosteroid treatment is effective in reducing inflammation and edema, it limits the tenocyte function by reducing collagen and proteoglycan synthesis, thus reducing the mechanical strength of the tendon and ultimately leading to further degeneration [23]. When conservative treatment fails, surgery is indicated. Surgery is performed more frequently among patients with DM and is estimated to be 4–14 times higher than the general population [24].

2.5 Adhesive Capsulitis of the Shoulder (Frozen Shoulder)

Adhesive capsulitis, or frozen shoulder, or shoulder periarthritis is characterized by progressive painful restriction of the shoulder movements, especially the external rotation and abduction. The prevalence in diabetic patients ranges 10–29 % [5, 16], as compared with 3–5 % of the age-matched controls [25]. The presence of shoulder adhesive capsulitis increases the incidence of DM and may be a presenting symptom. Connie et al. in 2008 studied the prevalence of a diabetic condition (DM and prediabetes) and adhesive capsulitis of the shoulder which revealed that a patient presenting with adhesive capsulitis had a 71.5 % chance of having a diabetic condition (38.6 % chance of being diabetic and a 32.95 % chance of being prediabetic) [26]. The natural history of the condition is characterized by three phases: pain, adhesion or stiffness, and recovery phases. Among diabetics, it occurs at a younger age, is less painful, lasts longer, and responds less well to treatment [27]. Bilateral involvement is more frequent in patients with diabetes than in nondiabetic subjects (33–42 % vs. 5–20 %) [28]. It occurs more in older patients with longer disease duration. Shoulder adhesive capsulitis is found to be associated with other diabetic complications such as limited joint mobility, autonomic neuropathy with either type of DM, and with myocardial infarction in patients with type 1 DM [5]. The exact mechanism is unknown, but it is thought that excessive glucose concentration in diabetic patients can lead to a faster rate of collagen glycosylation and cross-linking in the shoulder capsule, restricting shoulder range of motion [29]. Management of shoulder adhesive capsulitis in the painful early phase consists of adequate analgesia, intra-articular corticosteroid injections, and an appropriately graded exercise program. During the adhesive phase, physical therapy and operative treatments (arthroscopic capsular release or open surgical release) are used.

2.6 Diffuse Idiopathic Skeletal Hyperostosis

Diffuse idiopathic skeletal hyperostosis (DISH) is a condition that is characterized by diffuse calcification and ossification of the ligaments and enthesis (Figs. 2.5 and 2.6). The prevalence of DISH in type 2 DM was reported at 13–40 %, while in general population, 2.2–3.5 %. The prevalence of metabolic syndrome is higher among patients with DISH [16]. It most commonly affects the spine, particularly the thoracic spine. Patients with DISH are rarely symptomatic. However, it can cause spinal rigidity and impingement of nearby structures and nerves, resulting in hoarseness, stridor, sleep apnea, and dysphagia. It also can occur in extraspinal sites with prominent bony reactions at ligamentous and tendinous insertions, particularly in the pelvis, greater trochanters, patellae, and calcaneus. The diagnosis of DISH is based on radiologic features that are usually based on Resnick and Niwayama's 1976 criteria: (1) flowing ligamentous calcifications involving at least four contiguous vertebral bodies, (2) preservation of intervertebral disk space, and (3) absence of changes of degenerative spondylosis or spondyloarthropathy. The exact mechanism of DISH is not known. However, insulin, growth hormone, and growth factor (IGF-1) are proposed as factors that promote bone growth in DISH. Also, atherosclerosis, which is common in metabolic syndrome, will lead to damage in the endothelium and aggregation of platelets that result in increased levels of IGF-1 and then more osteoblast proliferation and bone formation. The treatment of DISH is symptom based and generally limited to analgesia as needed. Rarely, surgical

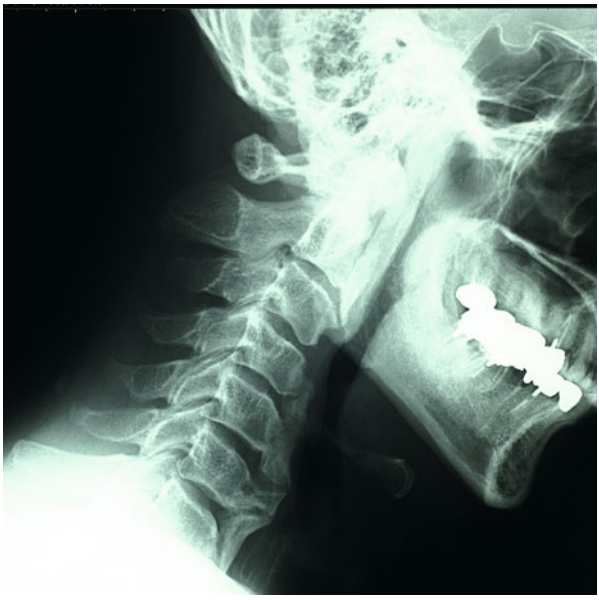

Fig. 2.5 The anterior longitudinal ligamentous calcifications of cervical vertebrae in diffuse idiopathic skeletal hyperostosis

removal of impinging bone bridges is undertaken when critical functions, such as swallowing, are compromised.

2.7 Osteoarthritis

Osteoarthritis (OA) is a very common form of arthritis in adults. Several risk factors are described for OA, including obesity, which is part of metabolic syndrome and not uncommon in DM. Dahaghin et al. [30] described an association between diabetes and hand osteoarthritis that was noted in people aged 55–62 years and was absent in other age groups. Peripheral neuropathy may increase the risk of advanced, aggressive forms of osteoarthritis. Recently, an association between knee osteoarthritis and diminished lower extremity vibratory perception was identified [31]. However, there is no clear evidence that supports DM or metabolic syndrome as risk factors for developing early or severe hip or knee OA [32].

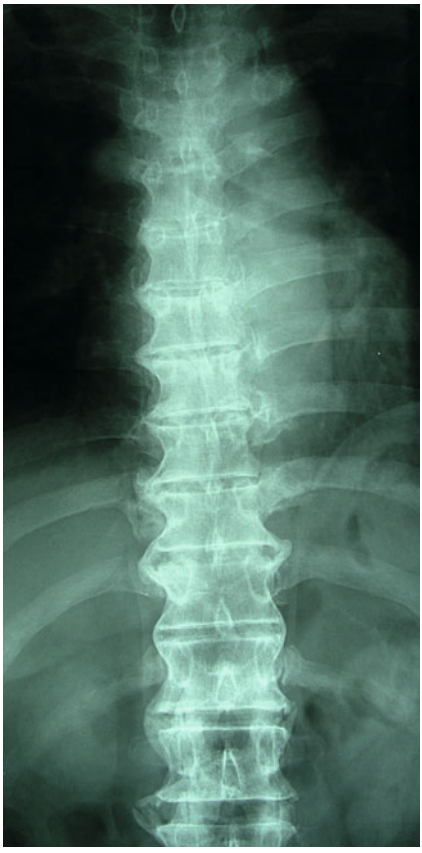

Fig. 2.6 The ligamentous calcifications of thoracolumbar vertebrae in diffuse idiopathic skeletal hyperostosis characterized by no lesion in heart side of thoracic vertebrae

2.8 Neuropathic Osteoarthropathy (Charcot Osteoarthropathy)

Neuropathic osteoarthropathy, also known as Charcot osteoarthropathy, is a progressive destructive process affecting the bone and joint structure associated with various diseases in which neuropathy occurs. However, DM is by far the most common etiology. It mainly affects the foot and ankle in diabetic patients. It is a devastating, limb-threatening condition resulting in dramatic deformities and recurrent ulceration that may ultimately lead to amputation. The pathogenesis of neuropathic osteoarthropathy remains debatable. It may result from repeated trauma, often minor, in the setting of decreased sensation due to a sensory neuropathy which results in increased damage with microfractures. An alternative possibility is that the neuropathy triggers an increased blood flow or distal hyperemia that results in stimulation of osteoclasts with increased bone resorption, osteoporosis, fractures, and joint damage. Recently, inflammation has been identified as another factor in the development of neuropathic osteoarthropathy. The release of proinflammatory cytokines leads to increased expression of the polypeptide receptor activator of nuclear factor-κB ligand (RANKL). RANKL triggers the synthesis of the nuclear transcription factor nuclear factor-κβ, and this in turn stimulates the maturation of osteoclasts from osteoclast precursor cells. At the same time, NF-κβ stimulates the production of the glycopeptide osteoprotegerin from osteoblasts [33]. The proinflammatory cytokines, RANKL, NF-κβ, and osteoclasts will result in increased osteolysis. The neuropathic osteoarthropathy is characterized by acute and chronic phases. In the acute phase, the foot is warm, edematous, and erythematous, mimicking cellulitis. Pain may or may not be present, depending on the degree of neuropathy. The chronic phase is characterized by deformity of the foot with abnormal pressure on the plantar surface due to the collapse of the plantar arch and the development of a rocker bottom deformity. Calluses may form which are liable to ulcerations, especially in the midfoot. The plain radiographs may not be useful in an acute phase; however, they may demonstrate excessive destruction in the chronic phase (Fig. 2.7). An MRI may show bone marrow edema, bone bruising,

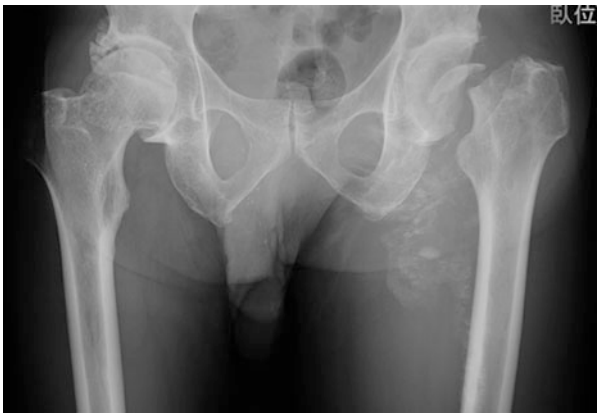

Fig. 2.7 The severe destructive lesion in the left hip joint due to Charcot osteoarthropathy

or microfractures. If osteomyelitis is a clinical possibility, radionuclide bone scanning, such as indium-111-leukocyte (111In-WBC) scanning and technetium-99m-methylene diphosphonate (99mTc-MDP) bone scanning, virtually excludes the osteomyelitis if both studies have negative results. Treatment involves weight-bearing limitations for at least 3 months for healing [34]. NSAIDs, calcitonin, and bisphosphonates may be used. Bisphosphonates inhibit the osteoclastic resorption, may have direct anti-inflammatory properties, and might slow or even stop the bony destruction. However, the data are weak to support their use as a routine treatment for acute neuropathic osteoarthropathy [35]. Surgical treatment may be required when this conservative treatment fails. Moreover, surgery in the presence of DM and diabetic complications carries higher risk for complications.

2.9 Osteoporosis

Patients with diabetes mellitus (DM) are at higher risk of bone fracture relative to their bone mineral density (BMD). A number of reports indicate a higher prevalence of vertebral fracture (VF) in those with type 2 DM (T2DM) than in those without, irrespective of the insignificant difference in BMD between patients with or without T2DM. Meta-analysis studies reported that T2DM patients exhibited a higher fracture rate, particularly in appendicular bones, despite their comparable BMD, suggesting the possible involvement of impaired bone quality, but not BMD, in the development of bone fragility. It is recognized that the higher fracture rate in T2DM patients is explained by increased cortical porosity, as shown by high-resolution peripheral quantitative computed tomography (HR-pQCT) or quantitative ultrasound (QUS) device [36].

It has been recently recognized that serum parathyroid hormone (PTH) plays an important role in the development of cortical porosity, which starts to increase in chronic kidney disease (CKD) patients as they progress to stage 3, for whom evidence shows a higher fracture rate at the femur neck. Since T2DM patients are complicated with stage 3 CKD more often than non-DM patients, it is important to examine whether DM by itself or in association with CKD might be a more important contributing factor to the development of cortical porosity [36].

2.10 Frailty, Sarcopenia

Frailty is a pre-disability condition that can be defined clinically. The major factors leading to frailty are sarcopenia and a decline in executive function. Stressors precipitate frail individuals into a state of disability. Diabetics develop the conditions necessary for frailty earlier than other aging individuals. Appropriate treatment of diabetes mellitus and frailty precursors can result in a slowing of the aging process [37].

2.11 Diabetic Muscle Infarction

Diabetic muscle infarction is a rare complication of DM. It is usually reported in association with long-standing, poorly controlled DM, particularly patients with type 1 DM, in the presence of microangiopathic complications such as retinopathy, nephropathy, or neuropathy. Patients with diabetic muscle infarction usually present with acute pain with swelling (and a palpable mass in 34–44 % of the patients) in an extremity that persists at rest and worsens with exercise and expands during a period of days to weeks without any prior history of trauma. The thigh muscles are commonly involved. However, the calf muscles, upper extremity, and abdominal wall muscles have also been reported. The diagnosis is established based on a clinical presentation and radiological finding. Laboratory studies generally demonstrate an elevated ESR and normal or mildly elevated WBC counts. Measurements of creatine kinase (CK) may be normal or elevated. Therefore, CK is not a reliable marker. The MRI is the diagnostic test of choice for diabetic muscle infarction. MRI findings demonstrate diffuse edema and swelling of multiple thigh or calf muscles, often in more than one compartment. Muscle biopsy should be reserved for patients with an atypical clinical presentation. The biopsy will demonstrate muscle necrosis and edema, phagocytosis of the necrotic muscle fibers, granulation tissue, and collagen deposition. Findings at advanced stages include replacement of the necrotic muscle fibers by the fibrous tissue, myofiber regeneration, and mononuclear cell infiltration [38]. The underlying pathophysiology remains incompletely understood. The most likely hypothesis is that muscle infarction is caused by vascular disease such as arteriosclerosis and diabetic microangiopathy. Hypercoagulability resulting from alteration of the coagulation–fibrinolytic system and endothelial dysfunction in DM has been proposed as a potential pathogenic mechanism [38]. The treatment involves bed rest, analgesics, and aggressive control of DM. Other medical therapies that have been suggested as being beneficial include antiplatelet agents such as low-dose aspirin, dipyridamole, NSAIDs, and nifedipine; however, there are no randomized control trials to support the use of these agents. Vigorous physical therapy should be avoided since it may lead to an exacerbation. Patients usually recover spontaneously over a period of weeks to months of bed rest, although the recurrence rate in the same or the contralateral extremity is approximately 40 % in all treatment groups [39].

2.12 Crystal-Induced Arthritis

2.12.1 Gout

Gout is a condition that is characterized by hyperuricemia, which is defined as urate levels >6.8 mg/dl (≥360 mmol/L), and monosodium urate crystal deposition in the joints. It has two stages: an acute stage characterized by recurrent attacks of arthritis

and a chronic stage that manifests as chronic tophaceous gout. The relationships between hyperuricemia, gout, and metabolic syndrome have been evaluated in several studies. It has been demonstrated that the prevalence of the metabolic syndrome among patients with hyperuricemia or gout is higher than the others. Insulin resistance and type 2 DM have also been noted to be associated with gout. It was found that the incidence of insulin resistance in gout patients increased by as much as 35 % over individuals without gout [40]. Recently, it was shown that both gout and type 2 DM share most of the common genetic risk factors and that there exists a mutually interdependent effect with regard to higher incidences between these two diseases [41]. Indeed, hypertension, chronic kidney disease, and hyperlipidemia are not uncommon in patients with DM, which are known risk factors for gout. Hyperuricemia was found to be a risk factor for the development of DM. Vidula et al. [42] evaluated the impact of serum uric acid levels on the future risk of developing type 2 diabetes independent of other factors, and they reported that as the level of uric acid increases, the incidence of DM also increases. These associations persisted in both genders and were independent of other known risk factors including age, BMI, alcohol consumption, smoking, physical activity level, hypertension, and levels of glucose, cholesterol, creatinine, and triglycerides [38]. Hyperuricemia may lead to endothelial dysfunction and nitric oxide inhibition, which in turn contribute to insulin resistance and thus diabetes. An alternative possibility is that the higher insulin levels associated with prediabetes can reduce renal excretion of uric acid.

2.12.2 Calcium Pyrophosphate Dihydrate Crystal Deposition Disease (Pseudogout)

Calcium pyrophosphate dihydrate (CPPD) crystal deposition in the hyaline or fibrous cartilage may be asymptomatic and identified by plain radiography which detects the calcification characteristic of CPPD. It may manifest as acute or chronic inflammatory arthritis. DM is considered as a possible risk factor for CPPD deposition disease. This is based on a small collection of cases [43]. Nevertheless, the association of CPPD disease with DM has not been proven.

2.12.3 Basic Calcium Phosphate Crystal Deposition Disease

Basic calcium phosphate (BCP) crystal depositions can occur in the intra-articular and periarticular components known as calcific tendonitis or calcific periarthritis. It most commonly affects the shoulder in which BCP crystals deposit predominantly in periarticular areas resulting in tendonitis or bursitis. The incidence of calcific shoulder periarthritis is increased in DM. Shoulder calcifications were detected in 31.8 % of patients with DM, while only 10 % in controls without DM [44]. The

diagnosis is made by a proof of the crystal in synovial fluid but it may not be easy, as BCP crystals are not detected even by polarized light microscopy. Therefore, the diagnosis depends upon excluding of the other causes. There is no specific treatment. Analgesics, NSAIDs, and joint aspirations with or without glucocorticoid injections are used. Calcific tendonitis may coexist with adhesive capsulitis in the shoulder.

2.12.4 Complex Regional Pain Syndrome Type 1 (Reflex Sympathetic Dystrophy)

Complex regional pain syndrome type 1 (CRPS 1), or reflex sympathetic dystrophy (RSD), is characterized by localized or diffuse pain in the upper or lower extremity usually associated with swelling, vasomotor disturbances, and trophic skin changes including loss of hair, skin color changes, skin temperature changes, and skin thickening. The condition may occur after minimal trauma or even spontaneously. DM may predispose one to CRPS 1. Walling [45] conducted a review on 387 patients with CRPS 1 and found that 28 patients have secondary CRPS 1 and DM was the commonest associated disease (in 11 out of 28). Treatments have been used with variable results, including analgesics, physiotherapy, intravenous bisphosphonates, calcitonin, oral corticosteroids, and sympathetic ganglion blocks.

2.13 Conclusions

DM is associated with various rheumatic conditions. Recognition of these conditions is important, as they affect the patient's quality of life. Several rheumatic conditions are more prevalent or caused by the long-term metabolic consequences of DM. Some of the rheumatic conditions associated with DM may be presented before the diagnosis of DM is established. Therefore, management of these conditions requires early recognition and close liaison between diabetes and rheumatology specialists.

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