Musculoskeletal Features of Diabetes Mellitus

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A great variety of musculoskeletal conditions are found associated with diabetes. The conditions may have a variable degree of association with diabetes and include musculoskeletal and neuromuscular disorders and gout. The present review is intended to cover the information available regarding musculoskeletal disorders. The discovery of one of these disorders may provide a clue to underlying diabetes and other complications of diabetes to which these patients are prone.

Neuropathy(Charcot joint)

Diabetes is definitely associated with neuropathic joint disease and is the commonest cause of Charcot joint affecting approximately 1 in 900 diabetics.[1.2] One earlier study showed a figure of 1 in 680 diabetics[2].
Charcot joint develops in the setting of long standing poorly controlled diabetes complicated with diabetic peripheral neuropathy[1]. Blood supply of the foot is well preserved. The exact mechanism of damage to the joint is far from clear. Increased susceptibility to fractures due to diabetic osteopenia is an important pathogenic factor in diabetic neuroarthopathy. It is important to realize that repeated trauma is at least one prerequisite for progression of joint destruction. It has been confirmed in experimental animal models of Charcot joint that repititive trauma is harmful and reduction of trauma leads to improvement[2]. Loss of proprioception allows hypermobility of the joints. This coupled with impaired pain perception makes the foot highly vulnerable to joint injury from mechanical trauma. Additionally, it has been suggested that autonomic diabetic neuropathy leads to defective temperature regulation and hypotonia of vessels which may play a role in pathogenesis.

The joints that are affected include tarsometatarsal joints, metatarsophalangeal joint and the ankle and subtalar joints in that order of frequency[3]. This is in distinct contrast to the Charcot knee of syphilis and the upper limb neuroarthopathy of syrinomyelia which are the other types of Charcot joints[2]. Ocassionally however, the knee, upper extremity, or spine may be involved in diabetes[2]. Some authorities have classified diabetic neuroarthopathy as a type of either peripheral (somatic) or autonomic (visceral) diabetic neuropathy.

The highest prevalence of Charcot joints is said to be in patients who have had diabetes for 12 to 18 years. The onset of symptoms occurs typically in the sixth decade, but ocassionally earlier. The sex ratio appears equal.

Signs and symptoms depend upon the joint involved. Tarsometatarsal involvement is usually preceded by a minor injury such as tripping. Initially there is no deformity. The patient presents with a hot swollen foot which is painful in one third of the cases and is often misdiagnosed as cellutitis or gout. However, disproportionate signs as compared to symptoms, lack of fever and leucocytosis, normal sedimentation rate and non-specific fluid analysis will differentiate this disease from gout or infection. In severe cases deformities can occur: 'rocker' sole deformity and medical convexity of great toe[4]. Metatarsophalangeal affection is not preceded by trauma; instead, there is an ulcer overlying a callus. In contrast, ankle joint involvement is frequently preceded by severe trauma of lower leg. The joint is typically painless, hypermobile and deformed. The feet are warm to touch and blood supply is intact. Signs of autonomic neuropathy and hyperhydrosis or anhydrosis may be present.

X-ray findings are not specific. There is joint destruction and disorganization with irregular narrowing, fragmentation and juxta-articular sclerosis. Sometimes there is new bone formation and remodeling. Radiological features are late and the diagnosis should be made before the development of radiological abnormalities.

Tarsometatarsal joint involvement carries an excellent prognosis[3]. and therefore, early diagnosis is essential. One should be careful when faced with unilateral warmth and swelling in a neuropathic joint after an episode of minor trauma[4].

Diabetic neuroarthopathy should also be differentiated from diabetic osteopathy or osteolysis, which does not affect joint space, is generally accomplished by plantar ulcer, and often is reversible. As for osteomyelitis, the destructive joint changes seen in that condition are more severe and are accompanied by signs of inflammation such as erythema, heat and tenderness.

Management of diabetic Charcot joint has not been satisfactory, although one group stated that the disease process can be halted in most cases. The

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main components of treatment are rest and education. Rest is achieved by bed rest or by non-weight bearing crutches or by a well moulded, non-walking plaster cast. Education is equally important and should concentrate on proper foot care. Special shoes to redistribute the weight can also be helpful. Partial non weight- bearing has been induced by patellar tendon-bearing joint fusion[5]. Lumbar sympathectomy has been used as therapy, but is no longer recommended because of lack of effectiveness. surgical procedures like arthodesis may require further assessment. These operative procedures in diabetic patients with vascular compromise may be complicated by wound infection, poor healing, and non-union of bone[6].

Diabetic Osteolysis

Osteolysis detected on X-ray, in the absence of symptoms and without evidence of infection, is definitely associated with diabetes[2]. It has also been called diabetic osteopathy[7]. The bones involved are those of the distal foot, usually the distal metatarsals and proximal phalanges. The process starts at the metaphysis as an ill-defined loss of bone cortex and spreads throughout the bone expect the central part of the diaphysis. The joint space remains intact inspite of complete destruction of metatarsal heads and phalanges.

The pathogenesis of diabetic osteopathy is not clear. Increased blood flow , diminished pain sensation and trauma are all involved. It is interesting to note that the end bone destruction closely resembles that seen in anaesthetic form of leprosy.

The patient presents with a warm foot and joint deformity. Arterial pulses area good and there is no evidence of neuropathy. There is no infection or inflammation. The end result of this destructive process is complete absence of the tips of phalanges. After fragmentation of the bone, the soft tissues may become infected. The X-ray shows bone end destruction as already described[4]. Diabetic osteopathy leads to more destruction than ostemylitis but can be deferentiated by the absence of signs of inflammation.

The most striking thing about diabetic osteolysis is its complete reversibility at least in some cases. The decision to amputate must not be rushed indiscriminately. Complete reversibility has not been described by a number of authors[7]. The reversibility is not possible in osteomyelitis. Some workers, however, believe that the presence of diabetic osteopathy implies previous osteomyelitis[8].

Diabetic Hand Syndrome [Limited joint mobility (LJM), cheiroarthropathy, joint contractures and waxy skin]

There are many reports of contractures of the finger joints in patients. In 1971 Jung and co-workers described finger contractures in 23 patients with diabetes[9]. Mean age and duration of diabetes were 43 and 17 years respectively. Evidence of diabetic peripheral neuropathy was present in most. In 1974 Rosenbloom and Friast[10] described 3 adolescents with joint contractures, thick, tight, waxy skin, impaired growth and menstrual delay after 8 to 14 years of developing diabetes. Subsequently the same group[11] reported 28.4% prevalence in campers with diabetes. Traisman[12] reported that 8.4% of 310 diabetic children were affected but he also found some non-diabetics with similar problem. Rosenbloom[13] subsequently reported that the presence of joint contractures could not be related with race, sex, dose of insulin, or estimated control of diabetes. Studies at Joslin Clinic confirmed these observations.

Thus diabetic hand syndrome is a common, although often overlooked, complication of diabetes[14]. It is present in 30 t0 40 % of insulin-dependent diabetes[16]. The joints affected are mainly of hand but the foot can also be affected. Skin may be thick, tight and waxy, sometimes referred to as pseudoscleroderma.

The pathogenesis is not clear but, there is some evidence of an association with an abnormality of collagen metabolism[17,18].Increased cross-linking of collagen by metabolites of diabetic patients has been suggested by some. Increased cross-linkage with accumulation of inflexible collagen, decreased response to collagenase[19], decreased elasticity of skin have been suggested as the possible abnormalities. Rosenbloom [13] found an extraordinary association of joint contractures with microangiopathy. Joint lessions preceded the findings of microangiopathy. The overall risk of microangiopathy by the 16 th year of diabetes for all of Rosenbloom's patients[13] was 42% and is comparable to data on retinopathy by others[15]. It is important to realize that the finding of joint contractures may indicate a high risk of subsequent microangiopathy. Patients with LJM have an increased incidence of diseases like frozen shoulder, Dupuytren contracture, flexor tenosynovitis and increased left ventricular, lung and arterial stiffness.
Diabetic hand syndrome can be demonstrated by the following simple tests:

- **Prayer Sign** Inability to completed approximate the palmer surfaces of hands indicate finger contractures[14,20]. To confirm it further the

examiner should passively extend the patient's fingers. The degree of possible extension in a normal person should be 180° or more at the proximal interphalangeal joints, and 60° at the metacarpophalangeal joints.

- **Table Test**[21] Patient is asked to approximate palms of both hands on a table. Any contractures will not allow perfect approximation.
- **Hand-Print Test** The degree of contract of hand with the surface is accurately assessed by obtaining an impression of a printed hand on a flat surface.

Osteopenia : There are a number of reports indicating that patients with IDDM and NIDDM have reduced bone mass [22,23]. The significance of this reduction in bone mass is controversial. Hath et al believed that it is of no significance. However there are others who believe this predisposes the diabetic patient to pathological fractures particularly metatarsal fractures. The mechanism responsible for osteopenia may be increased blood flow secondary sympathetic denervation[1] or poor diabetic control for many years.

Dupuytren Contracture Dupuytren classically described fibrous palmar nodules causing finger contratures in a coachman and the deformity is since then called Dupuytren'c contractures. This deformity is more common in diabetics and its frequency has been variously estimated as 12 to 32% in diabetics and only 6% in non-diabetes[20,24]. One group has even reported that diabetes was definitely present in 179 of 185 patients with Dupuytren's contracture (96.7%) [25].

Probable Association

Frozen shoulder(Periarthritis of shoulder) There are many studies to suggest that bursitis and periarthritis, particularly involving shoulder, occur more frequently in diabetics than non-diabetics

In one retrospective study of 800 patients periarthritis was found in 10.8% diabetics compared to 2.3% in a control group of 600 normal persons. Bilateral involvement was more common in diabetics. In another study[26] X-ray pictures of diabetic patients were retrospectively studied and it was that diabetics showed periarthritis with calcification in 22% cases while non- diabetics were similarly affected in 22% cases while non-diabetics were similarly affected in only 8% of cases. In a third study of cases of ' frozen shoulder' syndrome, 30 to 40 patients had blood sugar in the range of 134 to 150mg%

There may be three types of conditions i.e. capsulitis, tendinitis and true frozen shoulder. In capabilities there is restriction of movement but abduction range is somewhat more than 90°[27]. In contrast, the true frozen shoulder is associated with more severe restriction of movement. The pathogenesis may be an abnormal collage metabolism\ affecting the capsular tissue of joint[15]. Sometimes a triad of periarthritis, hand syndrome (including LJM, stiff hand flexor tenosynovitis, carpal tunnel syndrome and Dupuytren's contracture), and restricted hip mobility may occur[15,28]. The presence of this triad is associated with the duration of diabetes and retinopathy.

The treatment of frozen shoulder include physiotherapy, analgesis and in occasional cases submacromial injections of steriods

Ankylosing Spondylosis(Ankylosing hyperostosis) Ankylosis hyperostosis is a common disease of spine characterized by thick calcific bony bridges between the anterior and the lateral surfaces of the vertebral bodies. It commonly involves the thoraic vertebrae and has a predilection for the right side[29]. Though there can be pain and limitation of motion the condition is often asymptomatic and is incidentally discovered on X-ray. It occurs more frequently in obese individuals and the incidence is more in males than females.

Diabetes has been found in a high propotion of cases with ankylosing hyperostosis[30]. In one study 13% of 510 diabetics had ankylosing spondylosis on lateral X-ray[31]. Abnormal glucose tolerance was found in 23% of 164 patients with ankylosing hyperostosis and in 9% of 164 controls[31].

Ankylosing hyperostosis was found in 6 of 21 patients with acromegaly. There was a suggestion that increased growth hormone may be responsible for both, diabetes and bone proliferation. Though an increased level of growth hormone has not been confirmed in patients of ankylosing spondylosis without acromegaly, an increased sensitivity to growth hormone can not be ruled out.

Possible Associations

Carpal tunnel syndrome : Carpal tunnel syndrome in diabetics can not be differentiated from non-diabetics and is caused by median nerve entrapment. Diabetics however are also predisposed to peripheral neuropathy. If both the median and ulnar nerves show poor conduction, the mechanism is more likely to be neuropathy than carpal tunnel syndrome and surgery will be of no benefit [32, 33].

Phalen[34] reported that diabetes was present in 63 of 379 patients with carpal tunnel syndrome and family history of diabetes was present in another 40

patients. Other workers have also confirmed a frequency of 5-6% diabetes in patients with carpal tunnel syndrome. It is possible that underlying diabetic neuropathy renders the median nerve more susceptible to compression effects.

Flexor tenosynovitis Flexor tenosynovitis (trigger finger) is a condition characterized by painful snapping of the fingers and sometimes locking of the fingers in a fixed position and is thought to be more common in diabetics. The mechanism may be microangiopathy or abnormality in collagen metabolism. Females are affected more commonly and the right hand is more prone than the left. In one study 11 of 63 patients presenting with flexor tenosynovitis had diabetes[35].

Osteoarthritis It has been suggested by postmortem studies and the study of radiographs that diabetics have higher frequency, greater severity and earlier onset of osteoarthritis [36]. Since phosphorylated glucose intermediates in diabetics may be precursors of glycosaminoglycans, diminished formation of polysaccharides may explain this correlation.

Gout and pseudogout Despite two centuries of discussion on the possible association of gout and diabetes, the subject remains controversial due to a lack of controlled studies. Weight of evidence indicates that clinical gout is uncommon in patients with definite diabetes. Diabetes is also not particularly common in gout patients. However, exception to this general rule occur depending on a number of variables including criteria used for the diagnosis of diabetes and whether clinical gout or simple hyperuricaemia are used for the diagnosis of 'gout'. There are some reports in favour of such an association while other failed to confirm any link [2]. Similarly inspite of some early reports of higher frequency of pseudogout in diabetes there is no confirmation in subsequent studies.

Sudeck's osteodystrophy This is a 'reflex sympathetic dystrophy' after a relatively minor injury. Although some diabetologists feel that this condition is seen more frequently in diabetics, it is dificult to find a confirmation.

Fibrous dysplasia of bone Diabetes has occurred as a part of polyendocrinopathy associated with fibrous dyplasia [2]. Other abnormalities are also found like abnormalities of thyroid and growth hormone.

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