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Clinical Representation of the Different Rheumatological Manifestations of Diabetes

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Abstract: The various rheumatological clinical manifestations related to diabetes constitute a group of pathologies secondary to diabetes, regardless of the type of diabetes (type 1 or type 2), in most cases the manifestations are discovered at an advanced stage of diabetes, on average after 10 years of evolution. The different conditions of this group are frequent and diverse because they affect the majority of patients followed for diabetes. They are most often unrecognized or under-diagnosed in current practice, due to their clinical presentation which is characterized by a great polymorphism. These different manifestations can affect several structures of the locomotor system (bones, tendons, muscles, nerves or even joints and their synovial membranes) and constitute a real problem of differential diagnosis with the most commonly encountered rheumatological conditions. These clinical manifestations are either directly related to diabetes (chronic sustained hyperglycemia) or indirectly related to the various complications of diabetes; their prevalence is therefore correlated with poor glycemic control and/or the occurrence of other complications of diabetes. In this article, we describe the main rheumatological manifestations, their clinical and paraclinical diagnostic methods, their evolutionary profiles and their prevention as well as the different management in practice, and this will be a reminder in order to improve the practitioner's attitude towards the different clinical presentations of these affections.

Keywords: Diabetes, Bone, Joint, Entheses, Muscles, Tendon, Capsule, Hyperostosis

1. Introduction

Osteoarticular and abarticular complications of diabetes are represented by a clinical polymorphism. Although frequent, they are often ignored. They can constitute a major handicap in daily life. Their physiopathological mechanisms remain poorly understood. Nevertheless, some manifestations are the direct consequence of diabetes, others are simply associated with it and most of these complications are correlated with the age of diabetes. We report the main ones.

2. Stiffening Syndrome

Is painless, non-inflammatory limitation of hands and feet joints mobility. Corresponding clinical lesions include:

2.1. Diabetic Cheiroarthropathy (DC)

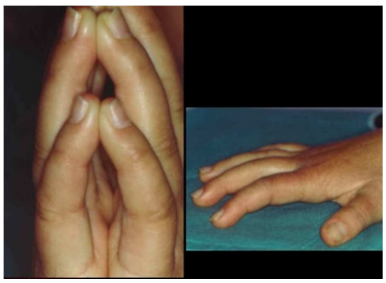

Figure 1. *Diabetic cheiroarthropathy called the prayer sign"with extension deficit of the fingers [3].*

This is the most common complaint expressed by diabetics. Its frequency varies between 8 and 34% compared to 2% in non-diabetics. DC is a painless limitation of flexion and especially extension of the fingers, mainly in metacarpophalangeal (MCP) and proximal interphalangeal

(PPI) joints [1], PPI of 5th finger being most affected [2]. The diagnosis of DC is clinical, it is responsible for a flessum attitude of the fingers, giving the classic prayer sign "palm joined as if to pray" (figure 1) and table sign "inability to flatten the palm and fingers against a flat surface" with an inability to close the fist completely.

The evolution [1, 4] is in three stages which only take into account the restriction of joint mobility:

Stage1: involvement of one finger, usually PPI, unilateral.

Stage 2: involvement of two or more fingers, mainly the 4th and 5th.

Stage3: Involvement of the MCP and PPI of all fingers with limitation of at least one large joint, most often the 4th and 5th. At least one large joint, most often the wrist and elbow.

X-rays may show vascular calcifications and exceptionally juxta articular bone erosions. Ultrasound demonstrates thickening of the dermis and tendon sheaths [1, 5, 6]. Treatment of DC is mainly based on rehabilitation, and night orthoses may be proposed.

2.2. Dupuytren's Disease (DD)

15-20% of diabetics have isolated DD or it is integrated into a picture of diabetic complications [7]. DD is found in 46% of diabetic patients with DC, compared to 21% of diabetics without DC [8]. Characterized by thickening and retraction of middle aponeurosis, due to fibroblastic proliferation responsible for progressive and irreducible flexion of the fingers. Initially, nodules (figure 2) and flanges are formed in the palm and on the palmar surface of the fingers (the 3rd and 4th++), generally painless and more or less bilateral and stable over several years, or they evolve towards the formation of aponeurotic bends, leading to irreducible flexion of the fingers [6]. The diagnosis of DD is clinical with the presence of a persistent palmar nodule that does not follow finger movements. This is pathognomonic of the condition.

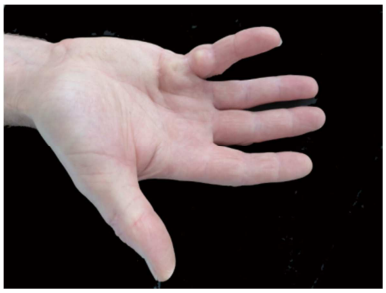

Figure 2. *Dupuytren's nodule on the 5th finger [9].*

The decision of a medical and/or surgical management is guided by the importance of the functional discomfort. Medical treatment is based on needle fasciotomy [1].

2.3. Tenosynovitis of the Flexors (Spring Finger)

Spring finger is a stenosing tenosynovitis in which constriction of the tendon sheath is associated with the presence of a nodule on the flexor tendon. The mobility of digital flexion or extension may be limited. The phenomenon of protrusion may occur in flexion or extension movement of finger. Manifestation frequency is 10% in diabetics. Flexor tenosynovitis may occur not only in patients with NIDDM but also in those with latent diabetes. It is probably a risk factor for impaired glucose tolerance [10, 11]. Treatment involves local infiltration of cortisone derivatives and, if this fails, surgery [1].

2.4. Retractile Capsulitis

Retractile capsulitis of the shoulder is characterized anatomically by capsular, ligament (acromiohumeral ligament) and sometimes bursal (subacromial-deltoid bursa) fibrosis, with possible primary damage to the collagen and its structures [12]. In a diabetic population, the prevalence of capsulitis is estimated at 13.4%. The risk of capsulitis in this population is multiplied by 5 regardless of the type of diabetes. Conversely, in a capsulitis population, diabetes is noted in 30% of cases [13]. The shoulder is initially painful for a few months. The pain then gradually diminishes, while stiffness sets in, just as gradually. Some patients are totally impotent in their upper limb, being unable to groom or dress themselves. The whole process can last from 6-9 months to 3 years. Classically, retractile capsulitis of the shoulder evolves in three phases: the painful phase or "congestion" phase, the phase of progressive stiffening with a decrease in pain (the shoulder is said to be "frozen") and, finally, the "thawing" phase during which movements gradually regain their amplitudes. The diagnosis is confirmed by arthrography (figure 3).

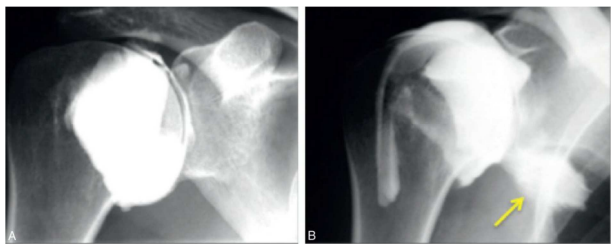

Figure 3. *Arthrography of retractile capsulitis. (A) Disappearance of the subscapular and axillary cul-de-sacs; (B) Same patient after distension of the joint and mobilization to achieve capsular rupture (arrow) [14].*

The treatment of retractile capsulitis is based on rehabilitation with active physiotherapy assisted, supervised and completed by daily home exercises, which does better than simple passive mobilization [15]. After obtaining indolence through the use of analgesics.

2.5. Carpal Tunnel Syndrome (CTS)

Carpal tunnel syndrome (CTS) is the nerve median compression in carpal tunnel, constituted with carpus anterior groove and closed front by anterior annular ligament. It results in paresthesias, predominantly nocturnal, affecting the first three fingers of the hand. According to Chammas [7], CTS is observed in 7% diabetics patients, vs 4% controls. CTS treatment is based on balancing the diabetes, wearing a

night splint to immobilize the wrist in a neutral position. Physiotherapy with ionizations aimed at anti-edema and anti-inflammatory could give an improvement of more than 40%. Local infiltrations of a glucocorticoid drift are proscribed because risk of infection and imbalance of diabetes. Surgical treatment is proposed in case of failure of medical treatment, and in forms with objective disorders of thenar eminence amyotrophy or motor disorders [16, 1].

3. De Quervain's Tenosynovitis

It is an inflammation of the synovial sheath of the abductor pollicis brevis and the extensor pollicis brevis of the dorsal compartment of the wrist. The predominance of women is classic [17]. De Quervain's tenosynovitis is present in 3.6% of diabetics versus 0.7% of non-diabetics [18]. The diagnosis is clinical, with the patient usually reporting wrist pain and swelling. On clinical examination, palpation over the radial styloid is painful. The Finkelstein maneuver is used, which consists of flexing the thumb in the palm of the hand and causing an ulnar deviation of the wrist; the pain is then reproduced. However, this test can be misleading in radial styloiditis and ligament injuries. Medical treatment is always proposed as a first line of treatment, and combines resting the thumb and stopping the activities that contribute to the injury, immobilization with a palm-area splint, which is recommended at the beginning of the injury, and oral and/or local anti-inflammatory medication. For resistant cases, one or two local corticosteroid infiltrations may relieve some patients [19, 20]. Finally, surgical treatment will be proposed in intractable forms.

4. Osteoporosis (Diabetoporosis)

Osteoporosis is a bone disorder defined by low bone strength with an increased risk of fracture [21]. The densitometric definition of osteoporosis is a T-score less than or equal to -2.5. Diabetic osteoporosis" is a form of osteoporosis resulting from diabetes and whose main risk factors are related to the imbalance of diabetes, its age and the micro and macrovascular and architectural complications. Patients with long-standing, severe or complicated diabetes should therefore be monitored to effectively prevent and treat the risk of fractures [22].

5. Nerveous Osteo-arthropathy (Charcot's Foot)

Is progressive and chronic disease of bones and joints, characterized by destruction of one or more joints on a neuropathic ground and is paradoxically little or even painless. Foot problems affect 15% of diabetic subjects [23, 24]. The prevalence of nerve osteoarthropathy reported in the literature seems to be between 0.1 and 0.4%, and up to 3% when minimal forms are taken into account [25, 26]. However, higher prevalences have been reported at 7.5 and 28% [27, 28]. For Charcot's foot, a distinction is made between:

5.1. An Acute Phase

It presents as swelling of only one of the two feet that has a very inflammatory appearance on inspection. The patient is not in pain because of the neuropathy. The inflammation will lead to bone lysis (osteolysis) which results in dislocation of the foot bones. The diagnosis is made by eliminating the causes of inflammatory edema of the foot (gout, microcrystalline or septic arthritis...). X-rays may initially be unremarkable. The patient will thus continue his activities and in particular walking, which will lead to the destruction of the architecture of the foot and to the constitution of irreversible deformations of the Charcot foot which then becomes chronic.

5.2. A Chronic Phase

It is characterized by an anarchic reconstruction of the foot bones responsible for important deformations. It is often at this time that foot sores appear, linked to the friction of shoes not adapted to the deformation of the foot (figure 4).

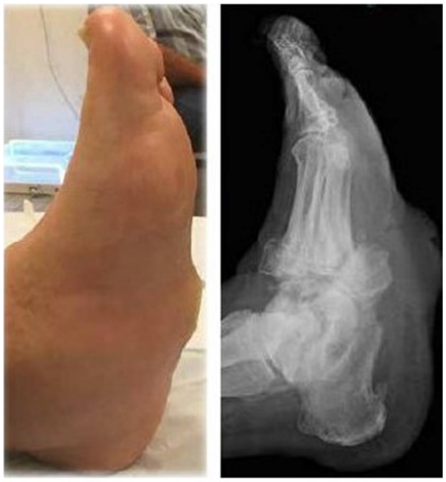

Figure 4. *On the left, chronic Charcot foot with plantar perforating injury. On the right, radiograph of the foot showing tarsal collapse with dorsal dislocation of the cuneiforms and plantar dislocation of the cuboid [29].*

MRI remains the gold standard for the diagnosis of a Charcot foot. The goal of treatment is to limit the deformity by immobilization and unloading [30].

6. Ossifient Enthesopathy (Diffuse Idiopathic Skeletal Hyperostosis (DISH))

DISH is a rheumatologic disease of unknown etiology [31]. Characterized by ossification of the paravertebral ligaments (figure 5) and peripheral entheses [32]. Type 2 diabetes appears to be an important risk factor, as 25-50% of patients with vertebral hyperostosis are diabetic and vertebral hyperostosis is found in 30% of type 2 diabetics. It can take a

long time for vertebral hyperostosis to become overt. People with spinal hyperostosis are usually asymptomatic and in this case the discovery is incidental on an X-ray, especially at the beginning of the disease. However, they may complain of pain and stiffness in the back or joints, making movement difficult.

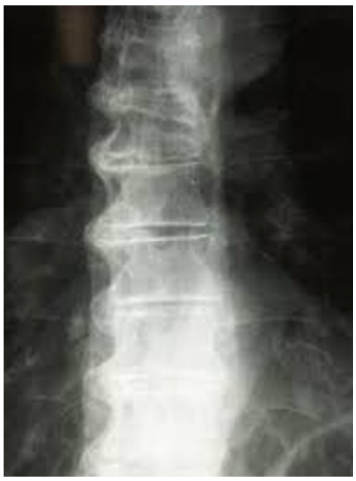

Figure 5. *Vertebral hyperostosis, ossification of the vertebral ligaments [33].*

In order to help the patient to control the pain and reduce the stiffness, he/she can use symptomatic treatment based on analgesics, non-steroidal anti-inflammatory drugs or corticoids. Physiotherapy or chiropractic treatment can limit stiffness and improve mobility. Physical activity and moderate stretching are also an important aspect of management. They can reduce fatigue, relieve joint pain and stiffness, and help protect the joints by strengthening the muscles around them [34].

7. Other Manifestations

7.1. Tendinitis

Tendinitis of the diabetic hand, as in the general population, is explained by:

7.1.1. Anatomical Factors

Multiplicity of joints, long spindly and therefore fragile tendons, the importance of sheaths and inextensible fibrous tunnels.

7.1.2. Mechanical Factors

Very mobile region, complexity and fineness of gestures, frequency of micro-trauma.

In addition to these factors, diabetics have an accumulation of inflexible collagen, an increase in its arrangement and its resistance to collagenases. Similarly, the association with diabetes of an increase in triglycerides, cholesterol and uric acid contributes to the formation of periarthritis [11]. The treatment consists of relieving the patient with analgesics or non-steroidal anti-inflammatory drugs. Heat, cold, shortwave, ionization and orthotics may be used. Cortisone infiltrations may be of help provided that diabetes is well controlled and asepsis is rigorous.

7.2. Diabetic Muscle Infarcus (DMI)

Diabetic muscle infarction (DMI) is a rare complication of diabetes, with less than 200 cases reported since its initial description [35]. Its exact prevalence remains unclear and its pathophysiology is debated, but atherosclerosis and microangiopathy seem leading to muscle ischemia. The diagnosis must be suspected in case of acute painful muscle induration without any notion of trauma. IMD is most often found in the thigh, particularly in the vastus medialis and vastus lateralis muscles, and then in the calf. Bilateral involvement is possible. MRI is the examination of choice, showing a T2 hypersignal and a T1 hypo or isosignal [36]. Treatment is based on NSAIDs, restriction of physical activity and physical therapy until symptoms resolution. Low-dose aspirin may shorten recovery time [37].

7.3. Arthrose and Diabetic Hands

Concerning this association, the authors' opinions are divergent. For some, diabetic patients are at greater risk of developing osteoarthritis and this predisposition could be explained by the fact that insulin stimulates the synthesis of protogyns and the growth of collagen; insulinopenia at the cellular level and diabetic vascular disease attenuate the chondrogenesis and osteogenesis required for osteophyte formation. Other studies [1] demonstrate the absence of increased osteoarthritis in diabetics, and no significant association was found between diabetes and radiological signs of osteoarthritis.

7.4. Infection

Although infection of the diabetic foot, a cause of morbidity and mortality, has been the subject of several scientific studies, few studies have focused on the incidence and prevalence of osteoarticular infections [38, 39].

8. Conclusion

Osteoarticular complications of diabetes are frequent and diverse. Their pathophysiological mechanisms remain imperfectly elucidated. Some are the direct consequence of chronic hyperglycemia and its effect on collagen, others are simply associated with diabetes without its role being directly incriminated. Diabetes control is the general rule for their management.

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