Hypothyroidism presenting as destructive arthropathy of the fingers

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Summary: A patient presenting with destructive arthropathy of the proximal interphalangeal (PIP) joints of the hands is described. She was initially believed to have rheumatoid arthritis but non-steroidal anti-inflammatory drugs were of no help. The patient was subsequently found to have hypothyroidism and erosive osteoarthritis of the fingers. Joint swelling, pain and stiffness responded dramatically to thyroid hormone substitution. The PIP joint spaces reappeared on the radiographs within 9 months.

This case suggest that hypothyroidism may induce destructive arthropathy of the finger joints. As thyroxine replacement may reverse the rheumatic complaints, hypothyroidism should be considered in the differential diagnosis of a destructive arthropathy of unclear aetiology.

Introduction

Patients with hypothyroidism may present rheumatic complaints due to chronic joint effusions (Bland & Frymoyer, 1970), muscular pains (Wilke *et al.*, 1981) or proximal muscle weakness (Cabili *et al.*, 1982). We report a patient who suffered from a painful destructive arthropathy affecting the proximal interphalangeal (PIP) joints of the fingers. Pain and swelling improved after hormonal replacement. To our knowledge, such an observation has not been made previously. It raises the question of a possible aetiological role of hypothyroidism in some cases of destructive arthropathy of the fingers.

Case report

A woman born in 1921 was in good health until 1976, when she suffered from progressive pain and stiffness in the PIP of both hands. There had been no previous local trauma. The menopause had occurred in 1970. Simultaneously with the rheumatic symptoms she complained of cold sensitivity, fatigue and dry skin. In view of symmetrical involvement of the PIP joints, she was thought to be suffering from seronegative rheumatoid arthritis. However, there was no response to trials with non-steroidal anti-inflammatory drugs and homeopathy also did not help. From 1980, she was unable to work as a secretary.

On admission in October 1981, physical examination revealed a hoarse voice, a puffy face with periorbital oedema, delayed relaxation time in the reflexes, regular pulse rate at 56 beats/min, no goitre, normal deep and superficial sensation and no skin or nail lesions. There was swelling of the PIP joints of both hands with local tenderness, and lateral subluxation of the medial phalanges. The wrists, metacarpophalangeal (MCP) and distal interphalangeal (DIP) joints were normal, as were the other peripheral joints and the spine. Grip strength of the hands was diminished to 80 mm Hg for each side. Schirmer's test was normal. Laboratory examination on admission showed an erythrocyte sedimentation rate at 16 mm/h, haemoglobin of 11.2 g/dl, normal leukocytes and thrombocytes, cholesterol at 7.8 mmol/l. The following values were normal or negative: serum calcium, phosphorus, glucose, creatinine, urate, alkaline phosphatase, bilirubin, transaminases, creatine kinase, latex fixation test, antinuclear antibodies, antisalivary duct antibodies. Serum thyroxine (T_4) titre was 17 nmol/l (normal: 60–150), TSH was 65.9 μ U/ml (0.5–7). The titre of circulating thyroglobulin antibodies was 1/20480 and that of thyroid microsomal antibodies 1/5400.

A drop of synovial fluid from the right index finger PIP joint showed low leukocyte count (about 100/mm³) and no bi-refringent crystals.

Electrophysiological tests including motor distal latencies of the median nerves were normal. Salivary gland scintigraphy with sodium Tc-99 m was normal.

Radiographs of both hands (July 1979) showed soft tissue swelling and joint space narrowing of all PIP joints, but no subluxation of the phalanges; erosions

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were observed in the left 2nd, 3rd and 5th PIP, and in the 4 last fingers of the right hand. Large cystic changes were observed on both sides of the PIP of the right 3rd finger. The wrists, the MCP and DIP joints were normal, apart from the left 3rd DIP with discrete erosive changes. On radiographs taken in January 1981 (Figure 1) there was a severe erosive arthropathy involving all the PIP joints with subluxation of the medial phalanges particularly evident in the right 2nd and 3rd fingers. The thumbs were normal as were the DIP, MCP and wrist joints. Periarticular calcific deposits were seen around the PIP joint of the right middle finger. In October 1981, the same changes were observed in the hands; radiographs of the knee joints, feet, pelvis, thoracic and lumbar spine were normal. A well defined cyst was observed in the superior part of the right femoral neck.

A surgical biopsy of the PIP of the right index finger was performed in October 1981. The synovium was thickened with scar remodelling. Histological signs of chondrocalcinosis were not seen. There were scarce infiltrates with lymphocytes; much bone debris and calcium deposits considered as apatite were noted in the synovial tissue. There were 1 to 2 lining border cells. These histological findings were highly suggestive of erosive osteoarthritis of the fingers.

Progress

From the beginning of November 1981, the patient was treated with l-thyroxine in gradually increasing doses to 100 µg/d. Thyroxine replacement was followed within 3 months by the disappearance of the symptoms of hypothyroidism. In March 1982, the swelling of the joints had disappeared and the grip strength had increased to 140 mm Hg. During the following months, the patient remained well, without stiffness or pain except on maximum flexion of the fingers. She remains well at follow-up examination 2 years later.

Radiographs of the hands in July 1982 showed that the soft tissue swelling had disappeared and there was marked subchondral sclerosis with well defined articular margins. The articular spaces had become visible. No progression of the erosions could be seen; osteophytes were prominent around the PIP joints of both index fingers and the right middle (Figure 2). MCP, DIP and wrist joints remained normal.

Discussion

It is probable that the first manifestations of hypothyroidism began in 1976 in view of the cold sensitivity and fatigue. Rheumatoid arthritis, Sjögren's syndrome and systemic lupus erythematosus may be associated with autoimmune thyroiditis (Camus *et al.*,

Figure 1 Right hand, January 1981. Joint space narrowing of the PIP joints. Erosions of the articular surfaces. Soft tissue swelling of all PIP joints; periarticular calcific deposits of the PIP joint of the middle finger. The MCP and DIP joints are normal.

Figure 2 Right hand, July 1982. Nine months after thyroxine replacement the joint spaces of the PIP have become visible. There is no longer soft tissue swelling. The articular surfaces are well defined with marked subchondral sclerosis. Osteophytes in the vicinity of the PIP joint of the middle finger.

1968) but none of these rheumatic diseases were present in this patient.

Arthralgia, stiffness, joint swelling of the knees and small joints of the hands and feet have been reported in hypothyroidism (Bland & Frymoyer, 1970). Synovial fluid has been reported as having a normal cell count and a high viscosity; calcium pyrophosphate dihydrate (CPPD) crystals have been frequently found (Dorwart & Schumacher, 1975). Interestingly, erosion-like radiolucencies in small bones of hands and feet and destruction of the tibial plateaus have also been substantiated in some cases of hypothyroidism (Bland & Frymoyer, 1970).

In the present case, articular complaints were the presenting features of hypothyroidism. The articular involvement was strictly confined to the PIP joints. The clinical and radiological picture was very much like that described in erosive osteoarthritis (OA) of the fingers (Peter *et al.*, 1966). However it should be noted that the DIP joints were not involved, apart from that of the left middle finger, a difference from classical erosive OA of the hands in which erosions are observed initially in the DIP joints and only later in the PIP joints.

Rheumatic complaints started simultaneously with the first symptoms of hypothyroidism, and joint pain and swelling disappeared with thyroxine substitution. During therapy, the radiographs of the hands changed with reappearance of the joint spaces which were better defined by subchondral sclerosis of the articular surfaces. These clinical and radiological data suggest that hypothyroidism was responsible for the arthropathy. The absence of any evident underlying rheumatic disease was confirmed at the 2 y follow-up examination.

Destructive arthropathy has been reported in patients with pyrophosphate arthropathy but the

incidence of erosive OA of the fingers has not been found increased in these cases (Gerster *et al.*, 1975). In the present case, there were no radiological or histological signs of pyrophosphate arthropathy. Erosive OA of the fingers may be associated with periarticular apatite deposition, which could be the initiating event of the joint destruction (Schumacher *et al.*, 1981). In this case calcified deposits were seen on X-ray in 1981. It can be presumed that these calcifications were the result of calcific material release from exposed bone; this is suggested by the absence of periarticular calcification in 1979 when the erosions started and by the finding of bone debris in the synovial tissue in 1981.

Whether hypothyroidism, which is frequent in elderly women, could be a causative or precipitating factor of erosive OA of the fingers in elderly women remains uncertain; prospective studies are warranted. The possible role of hypothyroidism in erosive OA has been outlined by Singleton *et al.* (1982). *In vitro*, TSH was shown to have a strong mitogenic action on chondrocytes (Corvol *et al.*, 1972), but there is no information on the effects of this hormone on articular cartilage *in vivo*.

Hypothyroidism appears to be a cause of joint complaints. As the symptoms generally regress, as illustrated in the present case and in others (Bland & Frymoyer, 1970), after thyroxine replacement, hypothyroidism should always be suspected in patients with rheumatic complaints of unclear aetiology; early diagnosis could prevent severe articular sequelae.

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