Rheumatol Int (1997) 17: 79–82

CASE REPORT

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Coexisting hyperparathyroidism, renal osteodystrophy and psoriatic arthritis

Received: 13 April 1996 / Accepted: 16 May 1997

Abstract We report the case of a 60-year-old woman with hyperparathyroidism, renal osteodystrophy and psoriatic arthritis. The coexistence of findings of hyperparathyroidism and renal osteodystrophy has been described and there are also reports of patients suffering from renal arthropathy mimicking hyperparathyroidism. To our knowledge, there is no description to date of a case displaying findings of the co-occurrence of these conditions in a patient. We would like to emphasize that attention should be paid to the possible diagnosis of a coexisting inflammatory rheumatic disease when rheumatological symptoms of recent onset occur in patients with long-standing renal osteodystrophy and/or symptoms mimicking hyperparathyroidism occur in these patients.

Key words Hyperparathyroidism · Renal osteodystrophy · Psoriatic arthritis

Introduction

Hyperparathyroidism and renal osteodystrophy due to haemodialyis treatment for renal failure are relatively common diseases that may typically coexist in the same patient [1]. Some of these patients might suffer from further bone involvement of the underlying disease due to elevated levels of parathormone (PTH), vitamin D deficiency and hypocalcaemia, i.e. osteomalacia, osteopenia or osteoporosis, finally leading to complications resulting from an increased risk of bone fractures [2]. We report here a case with the co-occurrence of clear radiological findings of hyperparathyroidism, renal osteodystrophy and psoriatic arthritis.

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Case report

A 60-year-old woman was referred to our rheumatology clinic in December 1994 for physical and occupational therapy following hip replacement for a femoral neck fracture after a sudden fall. She had been on haemodialysis treatment (RHT) three times a week for approximately 10 years for renal failure due to analgesic nephropathy. Five years before presenting to us, she developed secondary hyperparathyroidism and was suffering from musculosceletal and vertebral spine symptoms that were ascribed to osteoarthritis. From 1987 it was suspected that she was suffering from renal osteodystrophy. The lady was known to have concomitant diseases such as hypertension and asymptomatic cholecystolithiasis. Psoriasis vulgaris was diagnosed in 1986 and, shortly before referral to us, she had developed clinical symptoms of carpal tunnel syndrome of both hands. During the months prior to referral she had noticed an increasing restriction of movement, as well as intermittent swelling of peripheral joints and numbness in the first three fingers of boths hands. Finally, she had a severe sudden fall leading to surgical hip replacement due to femoral neck fracture of he right thigh.

Physical examination showed a woman in a reduced physical condition with a dirty brownish complexion. Her weight was 40 kg and her height was 1.58 m. Psoriatic skin lessions were found on the scalp, trunk, the extension sides of the upper and lower limbs and the internatal cleft. She had multiple scars on the right forearm and a Cimino-Brescia fistula on the left for RHT. Her blood pressure and heart rate were normal, as were physical signs relating to her lungs and heart. Long bones were normal except for restricted movement of the left and right hips. The peripheral joints of the fingers were asymmetrically thickened and the third metacarpophalangeal joint (MCP III) and the third and fifth distal interphalangeal joints (DIP III and V) of the left hand displayed active synovitis. Her muscles were generally weak and the spine was slightly kyphotic and was painful on dorso-lumbar percussion. Other physical findings were within normal limits of the patient's age. Laboratory tests, radiographs of the chest, pelvis, dorso-lumbar column and peripheral

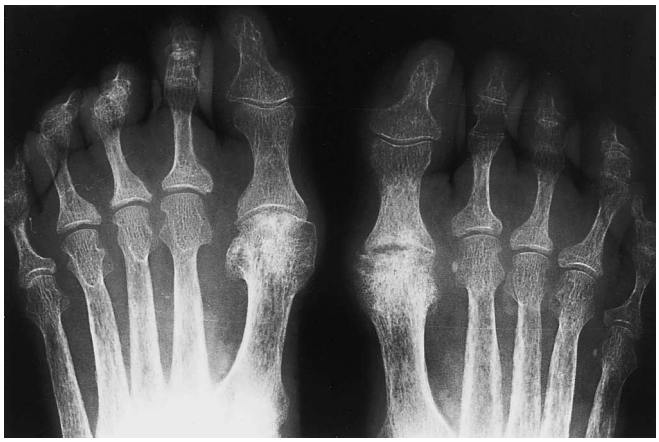


Fig. 1 Radiographic findings of renal osteodystrophy: renal osteodystrophy is considered to be a mixture of osteopenia, osteomalacia and osteosclerosis caused by chronic renal failure. This figure highlights blurred osteopenia of the spongy bone as the main feature of osteomalacia

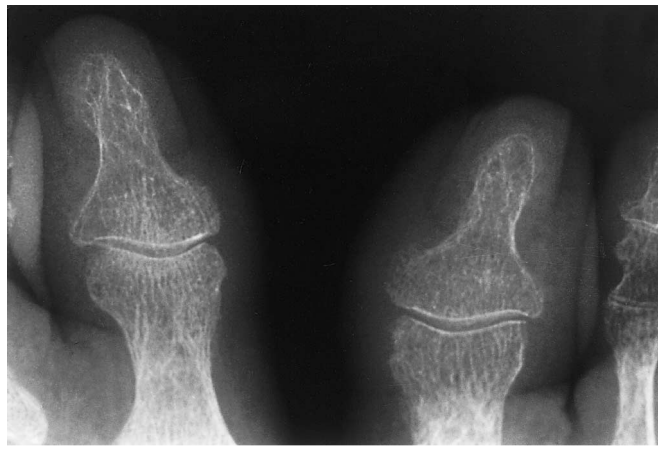
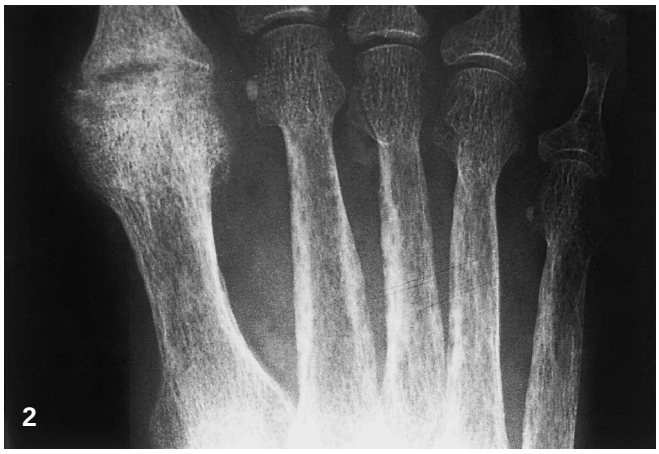


Fig. 4 Depicts the radiographic characteristics of psoriatic arthritis: joint narrowing, tapering and early destruction of the interphalangeal joint, proliferation both of the intraosseous and cortical bone, focal discontinuity and irregularity of cortical tuft with erosions in the bare area of the joints



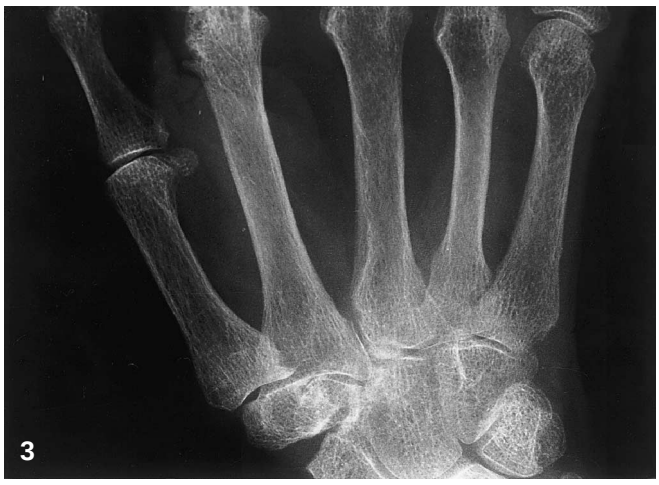
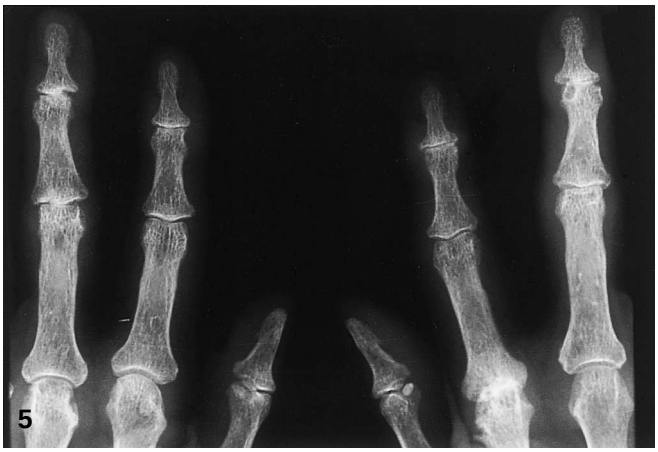


Fig. 2, 3 Show the radiographic findings of secondary hyperparathyroidism: speckled osteopenia and reduction of trabecular and cortical bone mass; subperiostal resorption and bone cysts or "brown tumours" in the lunate bone

skeleton, including hands and feet, and densitometry were performed. Relevant findings included an erythrocyte sedimentation rate (ESR) of 50 mm/h according to Westergren's method, haemoglobin of 10.2 g/dl, erythrocyte count of 3.46×10^{12} l, haematocrit of 29.8 and a normal white blood cell (WBC) and platelet count. Findings for BUN, creatinine, cholesterol and triglycerides were elevated, as is usually the case in chronic renal failure and RHT. Total protein was 6.0 g/l; electrophoresis revealed hypogammaglobulinaemia and an elevation of the alpha-2 globulin fraction, with the percentages of gamma and of alpha-2 being 8.5% and 11.5%, respectively. Readings for alkaline phosphatase (ALP; 301 U/l) the bone isoenzyme fraction (BALP; 255 U/l) and inorganic phosphorus (1.8 mmol/l) were elevated; osteocalcin (RIA) was 5.9 ng/ml (normal range 3.5–8.0 ng/ml). Ferritin (736 µg/ml, normal range 25–150 µg/ml) and PTH levels were markedly elevated (PTH intact molecule 364 pg/ml, normal range 9–55 pg/ml; PTH mid molecule >1000 pmol/l, normal <80 pmol/l) whereas serum iron, transferrin, acid-base balance and all other investigated parameters (iontogram, uric acid, liver function tests, serum thyroid hormones, 1.25 OH vitamin D3, 25-OH vitamin D3) were within physiological limits. Screenings for hepatitis B and C, the LFT=latex fraction test, as well as for antinuclear antibodies (ANAs; IF) were negative. Ultrasonography showed cholecystolithiasis, splenomegaly (13×5×10 cm), nephrocalcinosis and multiple renal cysts; the liver and pancreas were found to be normal.

Chest radiography revealed a reticular-type thickening of the pulmonary parenchyma and signs of an organized pleural effusion of the left phrenico-costal sinus; the thoracic aorta displayed signs of extended sclerosis. Radiographs of the pelvis and lumbar column showed generalized osteopenia, but no signs of vertebral fractures; there were signs of osteoarthritis of the left hip and, of course, the hip replacement on the right side. Radiographs of the hands and feet revealed a general blurred demineralization of the spongy bone, a speckled osteopenia due to renal osteodystrophy and hyperparathyroidism (i.e. subperiostal resorption), multiple bone cysts or "brown tumours" partly masking the radiological features of psoriatic arthritis (such as focal discontinuity and irregularity of the cortical tuft with destruction and erosion of the interphalangeal joints, widening of joint spaces, both intraosseous and periostal bone proliferation and subluxation of joints). The exact radiological details are depicted in Figs. 1–6. Bone densitometry by DEXA [Lunar DPX-L (TM)] of the left femoral neck and lumbar column revealed mineral densities of 0.591 g/cm^2 and 0.971 g/cm^2, respectively, with Z values of –0.94 (femoral neck) and 0.86 (lumbar spine) compared to normal age-matched controls.



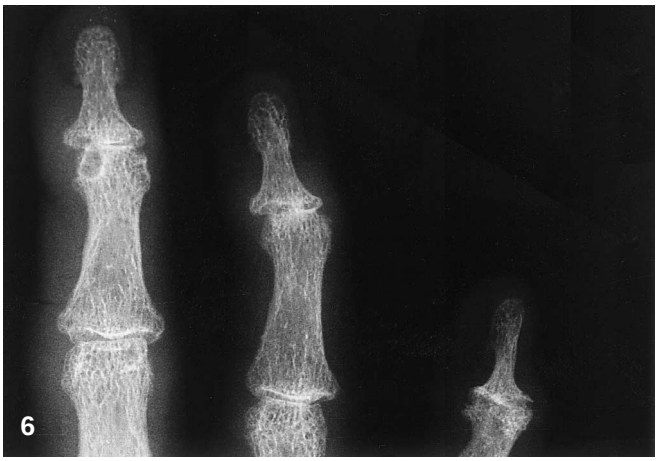


Fig. 5, 6 Also depict the radiographic characteristics of psoriatic arthritis, displaying the involvement of the metacarpophalangeal, and the proximal and distal interphalangeal joints of the third finger, destruction of the distal interphalangeal joints of the third and fifth fingers, severe erosions, osteolysis and proliferation resulting in joint deformities, e.g. subluxation

Discussion

During long-term RHT a variety of musculoskeletal complications and features of bone involvement have been reported [1–3]. Common findings usually comprise calcific periarthritis [4], recurrent acute (gouty?) arthritic episodes [5], carpal tunnel syndrome [6], synovial amyloidosis, renal osteodystrophy, dialysis arthropathy and secondary hyperparathyroidism [7, 8]. The additional occurrence of destructive spondylarthropathy in haemodialized patients has been discussed in the past as a possible new syndrome [9–11]. Ongoing destructive arthritis or spondylarthritis has been reported following long-term haemodialysis treatment, usually lasting more than 10 years. In these cases larger joints (e.g. shoulders or hips) and the vertebral spine are commonly involved, and beta-2 microglobulin has been found in the cysts of bones of affected joints or in the synovial membrane. Furthermore, beta-2 microglobulin has been found to play an important role in the development of beta-2-amyloid-containing amyloidosis in haemodialyzed patients, thus contributing to carpal tunnel syndrome and to the pathogenesis of spine involvement resembling spondylitis or spondylodiscitis [8, 11–13]. In subsequent studies, the aetiopathological role of several crystals in the development of arthritic complications in haemodialyzed patients has been established and increasing evidence of apatite-induced arthropathies due to secondary hyperparathyroidism has been found [10]. Ellis et al. have described the role of aluminium contributing to impaired bone mineralization and have detected aluminium compounds in the joint tissue of patients with chronic renal failure undergoing RHT [14]. The increasing knowledge of factors involved in the pathogenesis of complications due to long-term haemodialysis treatment has led to therapeutic considerations comprising the lowering of serum phosphate levels and supplementation of vitamin D3 thus preventing secondary hyperparathyroidism. Furthermore, it has been suggested that deferoxamine is helpful in the treatment of bone impairment due to aluminium compounds, and early renal transplantation could prevent dialysis arthropathy, beta-2-microglobulin-associated complications and amyloidosis [9, 14, 15]. There is ample evidence of rheumatic and musculoskeletal complications due to long-term RHT, but only in sufficient data of the co-occurrence of other rheumatic conditions exist. Even if there were no histological data of amyloidosis, a condition which has to be assumed in our patient, this case unquestionably displayed the co-occurrence of clinical and radiological signs of hyperparathyroidism, renal osteodystrophy and psoriatic arthritis in the same person. The clinical findings of active asymmetrical arthritis, markedly enhanced inflammatory disease activity, as well as elevated values of ALP, BALP, and the presence of hyperparathyroidism suggest enhanced osteoblastic activity, thus profoundly impairing bone metabolism leading or contributing to generalized osteopenia via enhanced bone turnover. Psoriatic skin lesions, asymmetrical arthritis, the radiological findings of joint destruction and osteoproliferation of the peripheral joints confirmed the diagnosis of psoriatic arthritis and suggested the coexistence of these different rheumatic conditions. To our knowledge, this is the first description of a case displaying the radiological findings of hyperparathyroidism, renal osteodystrophy and psoriatic arthritis in a patient. Findings of coexistent hyperparathyroidism and renal osteodystrophy, and even patients suffering from renal osteodystrophy mimicking hyperparathyroidism have been reported [9, 10]. Our present report highlights once again the attention should be paid to diagnosing ongoing inflammatory rheumatic disease, e.g. psoriatic arthritis, co-occurring in a patient suffering from hyperparathyroidism and renal osteodystrophy due to long-term haemodialysis treatment.

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