and joint involvement has become an important limitation of long term prognosis.

Severe secondary hyperparathyroidism can cause osteoarticular pain and an erosive enthesopathy which carries a risk of tendinous rupture.

Urate gout is a classical feature of terminal renal failure which usually does not persist after the onset of haemodialysis, as efficient uric acid clearance is achieved during dialysis sessions.

Periarticular calcifications are usually composed of calcium phosphate crystals and are favoured by hyperphosphataemia and other poorly known factors. Deposits are frequently asymptomatic but can grow into tumoral masses or cause acute articular or periarticular flares. Bone erosions, in the vicinity of deposits can be observed. In dialysis patients, secondary oxalosis usually follows vitamin C supplementation of the diet and may be responsible for soft tissue calcification, articular effusion, chondrocalcinosis and a disabling finger flexor contracture.

The incidence of bone and joint infection in haemodialysis patients is 10 to 100 fold the one in the population at large, and this must remain a permanent concern as prompt diagnosis and treatment are required to improve a gloomy prognosis. The leading causative organisms are *Staphylococcus* species, the usual portal of entry being the arteriovenous fistula. In kidney transplant patients, very unusual pathogens can be identified.

Beta 2-microglobulin amyloidosis has been documented in a few long-standing terminal renal failure patients before the start of dialysis, but most usually develops in long term dialysis patients. Up to 65 p.cent of individuals who have received ten or more years of maintenance haemodialysis using standard cuprophane membranes are affected. Ageing is also an important risk factor. Carpal tunnel syndrome is a prominent feature, although oedema, uremic neuropathy and crystal deposition may play a role in some carpal tunnel syndromes of dialysis patients. A chronic amyloid arthropathy is also frequently observed. Features include chronic arthralgias, particularly of the shoulders, chronic joint swelling, finger tendon synovitis, large subchondral bone erosions which may lead to femoral neck fracture, and destructive arthropathy. Synovial fluid is noninflammatory and may contain numerous blood cells. Destructive spondylarthropathy predominantly affects the cervical spine and can lead to spinal cord and/or nerve root compression, usually through spine instability. Shoulder pain may be related to amyloid synovium involvement and/or amyloid thickening of the cuff tendons and subacromial bursa. Systemic deposits are rare. They can be responsible for intestinal haemorrhage or cardiac dysfunction. Lack of β 2-microglobulin catabolism by the deficient kidney and unsatisfactory elimination through the dialysis membrane are important in the pathogenesis of the disorders. The use of permeable and biocompatible membranes appears to delay the onset of the disease, but has been thus far unable to totally prevent its development.

SP0012 MALIGNANCIES IN RHEUMATIC DISEASES

A Ekblom. Unit of Clinical Epidemiology, Department of Medicine, Karolinska Hospital, Stockholm, Sweden

10.1136/annrheumdis-2001.54

Patients with rheumatic diseases have an increased risk overall for cancer compared to the normal population. This is most pronounced for patients with SLE, but patients with rheumatoid arthritis have also a significantly increased risk. Unpublished data

from Sweden also indicate that such an association exist for patients with Morbus Bechterew as well as patients with psoriatic arthritis.

However, the increased cancer risks are not general phenomenon for cancer as such. There are distinct differences in the patterns of cancer occurrence compared to the general population. Smoking related cancers, especially lung cancer, occur in excess for both rheumatoid arthritis and SLE patients as opposed to cancers of the gastrointestinal tract (especially colorectal cancer) which are inversely associated with all four disease entities mentioned above, possibly due to the pharmacotherapy these patients are exposed to.

Hematopoietic cancers (lymphomas and leukemias) also occur more than that expected in patients with rheumatic diseases. In the case of SLE and rheumatoid arthritis these excess risks are especially prominent for lymphomas, but there is also a somewhat less pronounced increased risk for leukemias. This increased risk can probably, to some extent, be explained by immunosuppressive therapy. However, the increased risk for lymphomas in a Swedish setting was already present in these patient groups before the introduction of such therapies during the 1970's. Moreover, the risk estimates for lymphomas have not been altered after the introduction of such therapies. The absence of an impact of immunosuppressives can, perhaps, to some extent be due to a substantially increased absolute risk of lymphomas in the general population, which means that the cumulative incidence of lymphomas among patients with rheumatic disease is presently higher than it was 20 years ago.

High disease activity in patients with rheumatic arthritis seems to be associated with an even more pronounced risk for lymphomas. There are indications that anti-inflammatory therapy have a protective effect, although better data is needed before such an association can be firmly established.

In conclusion, rheumatic diseases are associated with increased risk for cancer. Hematopoietic cancers are, however, the only malignancy where a causal association has been established.

SP0013 STRESS SYSTEM RESPONSE IN RHEUMATIC DISEASES

M Cutolo. Division Rheumatology Department Internal Medicine, University of Genova, Genova, Italy

10.1136/annrheumdis-2001.55

The role of stress is emerging among the factors involved in the pathophysiology of rheumatic diseases including genetic predisposition, infections and gender. A stressor is any stimulus that disturbs or threatens to disturb homeostasis. Stressors can be exogenous or endogenous and metabolic, physiologic, infectious, inflammatory, or emotional in nature. The stress system response (SSR) is the array of physiological and behavioural adaptation to restore homeostasis and is mediated through activation of the hypothalamic-pituitary-adrenal axis (HPA) and the sympathetic nervous system (SNS). Activation of HPA axis induces synthesis and release of corticotropin-releasing hormone (CRH), adrenocorticotrophic hormone (ACTH and finally glucocorticoids (GC) such as cortisol. GC act in a permissive way to increase readily available energy resources to brain and heart during stress and terminate defense mechanisms to stress to prevent these responses to become destructive themselves. SNS activation leads to release of norepinephrine (NE) and the sympathetic division