also recorded as palpable. In two of these patients splenic enlargement was confused with a massively enlarged left lobe of the liver and in another a large intra-abdominal tumour was subsequently shown to have caused the mass. Nevertheless, palpation gave false-positive results in 10.5% of cases and percussion in 16.6%.

Comment

Our findings show that the combination of splenic percussion and palpation will detect up to 88% of spleens considered to be enlarged on hepatic scintiscans.

In other studies the correlation between clinical detection of splenomegaly and radiography or radioisotope scanning has been poor. Although as little as a 40% increase in spleen size may be palpable, even massively enlarged spleens have occasionally gone clinically undetected. Perhaps the error is in the standard used for comparison. The borders of the spleen may not be identifiable on a radiograph and hepatic scans in which the peak count rate is set over the liver will often underestimate the actual size of the spleen. Some enlarged spleens may be undetected by both clinical and radiological techniques. Likewise, some spleens that appear enlarged by palpation or percussion, although not by radiography or scanning, may be enlarged. Nevertheless, not every palpable spleen is abnormal or enlarged, and up to 2.9% of healthy young people⁴ and up to 5.6% of a general patient population⁵ may have palpable spleens. In only slightly over half an adequate explanation for the splenic palpability be found.

We can confirm that apparently normal spleens may be palpable but, in contrast to other studies, we have also shown that, with false-positives often due to hepatomegaly or abdominal masses, careful percussion and palpation will detect most enlarged spleens. Careful delineation of the left lobe of the liver may improve accuracy even further.

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recorded in the respiratory function tests. No tubercle bacilli were found in sputum or gastric washings. The results of skin testing to tuberculin, streptokinase/streptodornase, *Candida albicans*, and mumps antigens were negative (the patient had received BCG some years previously). Surgical biopsies of tendon sheath synovium, a skin plaque lesion, and the Kveim test site all showed multiple non-caseating epithelioid granulomata consistent with sarcoidosis. Cultures of these tissues were sterile.

The initial diagnosis of seronegative rheumatoid arthritis was changed to chronic sarcoidosis with joint, skin, and lung disease. Prednisone 20 mg/day was started. There was an immediate improvement in joint pain, and a gradual reduction in joint swelling over one month. Six months later, while taking prednisone 7.5 mg/day, he had minimal joint symptoms or swelling. Joint and chest x-ray films were unchanged.

Discussion

Two main types of joint disease occur in sarcoidosis.¹⁻³ The more common is an acute transient arthritis, predominantly affecting the ankles and knees, occurring with or sometimes before erythema nodosum, and bearing the same relation to the course of the disease. Less frequently a more persistent chronic form occurs, as in this patient, reflecting granulomatous disease of the synovium, which may lead to joint destruction. Active systemic disease in other organs is generally associated. Negroes appear to be more susceptible to this type.⁴ Corticosteroids were successful in controlling troublesome symptoms of synovitis in this patient. We hope that active granulomatosis will be suppressed with this treatment, and further joint damage averted.¹

It is reported that Nigerian patients with polyarthritis satisfying the American Rheumatism Association criteria for a diagnosis of rheumatoid arthritis differ from Caucasian patients with the disease in several important respects. Though the initial features of their joint disease are typical, nodules and vascular lesions are rarely seen; radiological erosions are generally mild; and most are seronegative. A chest x-ray film and careful search for skin lesions may help in distinguishing chronic sarcoid arthritis in this group.

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Chronic sarcoid arthritis

We report here a patient with chronic sarcoid arthritis whose initial presentation resembled rheumatoid arthritis. Subsequent recognition of pulmonary and skin disease led to the correct diagnosis.

Case report

A 34-year-old Nigerian man was referred with a two-year history of chronic symmetrical polyarthritis, thought to be rheumatoid arthritis, which had not responded to salicylates or indomethacin. He had appreciable synovial thickening and chronic effusions in his knees, ankles, interphalangeal joints, and in the extensor tendon sheaths of the wrists. Four months previously he had first noted several small, well-demarcated plaques in the skin of his back, and small indurated nodules in the sites of old scars on his forehead and limbs. Mild superficial lymphadenopathy and splenomegaly were also noted on examination. His eyes were normal. There had been minimal systemic disturbance. He had no respiratory symptoms.

Erythrocyte sedimentation rate was 40 mm/h; haemoglobin 12.1 g/dl; IgG 34 g/l, other immunoglobulins normal; serum urate 0.44 mmol/l, calcium 2.4 mmol/l. Serum rheumatoid factor and antinuclear antibodies were absent. Joint radiographs were normal. On the chest x-ray film there was considerable diffuse shadowing throughout both lung fields. The hila were normal. A slight diminution of predicted lung volume and transfer factor was

Nitrofurantoin crystalluria

Nitrofurantoin is widely prescribed in the treatment and prophylaxis of urinary tract infections. It is well absorbed, rapidly concentrated in urine, and its toxic effects are rare. One substantial advantage over sulphonamides is that crystalluria—with its dangers of renal colic, haematuria, and oliguria—is believed not to occur. Goodman and Gilman state that "even supersaturated solutions of nitrofurantoin do not cause crystalluria."¹ We report the first three described cases of nitrofurantoin crystalluria.

Case 1

An 84-year-old man sustained a traumatic quadriplegia from a "whiplash" injury during a car crash in March 1975. His resulting urinary incontinence required long-term catheterisation. During the next year he developed recurrent *Escherichia coli* urinary tract infections. Prophylactic nitrofurantoin