PELVIC INFECTION AS A CAUSE OF BILATERAL SACRO-ILIAC ARTHRITIS AND ANKYLOSING SPONDYLITIS*

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About 30 years ago it became evident that bilateral sacro-iliac arthritis was a very frequent feature of ankylosing spondylitis. Since then, this finding has been repeatedly confirmed. It is now widely believed by rheumatologists and radiologists that the diagnosis of ankylosing spondylitis cannot be substantiated unless there is radiological evidence of sacro-iliac arthritis. Particularly significant is the fact that this sacro-iliac joint involvement is almost invariably the first radiological manifestation of the disease. Moreover, after the initial years of the development of ankylosing spondylitis, both sacro-iliac joints are always involved to an approximately equal degree.

So constant has been the occurrence of bilateral sacro-iliac arthritis, that there has now developed a general belief among clinicians and radiologists that bilateral non-pyogenic sacro-iliitis is synonymous with the diagnosis of ankylosing spondylitis. There are however several other pathological conditions in which these joints undergo slow destruction, and a review of these reveals that a feature common to them is infection situated within the pelvic cavity.

Considerable evidence has now accumulated that patients with ankylosing spondylitis also frequently have a focus of chronic pelvic sepsis. It is thought, therefore, that this infection may be a provocative aetiological factor in the production of bilateral sacro-iliac arthritis. It is suggested that, in patients with the necessary genetic and constitutional make-up, this sacro-iliac arthritis may herald the onset of the full syndrome of ankylosing spondylitis with spinal and peripheral involvement.

Diagnosis

Reiter's Syndrome. — The radiological changes found in this condition have recently been reviewed by Reynolds and Csonka (1958) and by Murray, Oates, and Young (1958). Personal experience is in full agreement with their findings. The radiological changes in Reiter's syndrome are seen most commonly in the feet and in the sacro-iliac joints. In the feet, tendinitis, affecting the Achilles tendon, and plantar fasciitis occur frequently, often accompanied by bursitis. The cortex of the os calcis at the attachment of these tendons becomes destroyed, and the immediately subcortical bone is eroded. Periosteal osteoblastic metaplasia is stimulated and extends into the tendinous attachment (Fig. 1, opposite). These changes are particularly well marked on the under surface of the os calcis, and the periostitis frequently extends to the adjacent lower surface of the cuboid and base of the fifth metatarsal. It tends to be considerably more extensive and florid than in the similar changes seen in ankylosing spondylitis. The end-result is the formation of calcaneal spurs, sometimes of considerable length, and a contraction of the plantar fascia to cause a painful claw foot. The metatarsal-phalangeal and interphalangeal joints may develop superficial erosions similar to the changes of rheumatoid arthritis, but they are more frequently accompanied by a neighbouring periostitis. This periostitis is more frequent and more marked in Reiter's syndrome than in classical ankylosing spondylitis or in rheumatoid arthritis.

Frequently all the metatarso-phalangeal joints are affected by this erosive arthritis, and lateral subluxation of the phalanges commonly results (Fig. 2, overleaf).

This pattern is frequently seen in patients with Reiter's syndrome or with non-specific urethritis. When these changes in the feet are much more marked than changes in the hands, urethritis with subsequent arthritis should be suspected, as such a dominance of changes in the feet is rare in rheumatoid arthritis.
Sacro-iliac arthritis occurs with particular frequency in those patients who develop recurrent attacks of Reiter's syndrome. The incidence increases with the duration of the disease, being variously reported as 33 per cent. (Reynolds and Csonka, 1958), 40 per cent. (Murray, Oates, and Young, 1958), and 49 per cent. (Oates, 1958) of patients with recurrent attacks of the disease for whom pelvic radiographs were available. Sacro-iliac arthritis occurs much more frequently in those patients who have developed anterior uveitis. This association of uveitis and bilateral sacro-iliitis also occurs in about 10 to 20 per cent. of patients with classical ankylosing spondylitis.

The radiological features of the sacro-iliac arthritis in Reiter's syndrome are very similar to those found in ankylosing spondylitis. The earliest changes are a patchy destruction of articular cortex, small superficial erosions and an irregular joint space—at first increased by the bone destruction, but soon decreased by the continuing destruction of the articular cartilage (Fig. 3, overleaf).

There is frequently a para-articular reactive bone sclerosis, more marked in the ilium. The diminishing joint space becomes ankylosed by the osteoblastic metaplasia occurring between the destroyed articular surfaces (Fig. 4, overleaf).

From cases personally observed, the impression has been gained that the main differences in the radiological changes of the sacro-iliac arthritis of Reiter's syndrome and those of ankylosing spondylitis are differences of symmetry and degree.

Patients with the genital syndrome more frequently develop unilateral sacro-iliac arthritis and, if the arthritis later becomes bilateral, there is generally less symmetry in degree than is commonly seen in ankylosing spondylitis. Complete joint destruction and complete bony ankylosis were not found to be common features in those patients examined. These differences are mainly ones of degree, and probably do not indicate two completely distinct radiological patterns.

The main radiological differences between patients with Reiter's syndrome and those with ankylosing spondylitis are seen in the spine. Pathological and radiological changes affecting the vertebral bodies, the periphery of the intervertebral disks, the synovial apophyseal joints, and the spinal ligaments are very prominent features of ankylosing spondylitis—and, indeed, are responsible for its name. Although occasional cases of Reiter's syndrome showing similar, but less marked, changes have been reported by Marche (1950) and Ford (1953), they must be of considerable rarity, and none has been seen by the author or has been reported by Reynolds and Csonka (1958) or Murray and others (1958).
FIG. 2.—Same patient as in Fig. 1, showing multiple erosive arthritis of metatarso-phalangeal joints with subluxation.

FIG. 3.—Non-gonococcal urethritis in a male aged 44 yrs. Previous gonorrhoea, and persistent non-gonococcal urethritis, bilateral plantar fasciitis, and arthralgia of ankles. The x-ray shows bilateral sacro-iliac arthritis, much more marked on the left where ankylosis is beginning.
If the radiological changes of ankylosing spondylitis are confined to the sacro-iliac joints (as is found in the first few years of the disease), it may be impossible to give an exact diagnostic label to the patient with bilateral sacro-iliac arthritis, calcaneal erosions, anterior uveitis, and an unobtrusive urethral discharge.

Polyarthritis may follow a non-specific (non-gonococcal) urethritis without the cutaneous, conjunctival, and oral lesions seen in the complete clinical picture of Reiter's syndrome. The sacro-iliac joints may be affected in these patients, and the radiological appearances produced do not differ in any way from those seen in the complete Reiter's syndrome. It is now generally believed that "non-specific urethritis with arthritis" is merely an incomplete variant of Reiter's syndrome. Patients with the complete clinical picture of this syndrome frequently develop recurrences in which only the urethritis and the arthritis are evident.

Patients with the complete or incomplete form of Reiter's syndrome should routinely be referred for radiographs of the sacro-iliac joints, soft-tissue true lateral films of the os calcis, and films of the feet, and of any other joint which may be affected. All of these patients have had a chronic or recurrent urethritis, and it has been shown by Mason, Murray, Oates, and Young (1958) that at least 95 per cent. have evidence of chronic prostatovesiculitis as demonstrated by the cytological examination of expressed secretion.

Paraplegia.—Destructive arthritis of the sacro-iliac and apophyseal joints of the lumbar spine have been reported in patients with total paraplegia from the level of L.1 or above (Abramson and Kamberg, 1949; Abel, 1950). These changes proceed to complete ankylosis and, together with similar lesions in the symphysis pubis and ischial tuberosities, may produce a radiological picture of the pelvis very
similar to that frequently seen in ankylosing spondylitis (Fig. 5). Male paraplegics invariably develop a chronic urinary infection, which involves the prostate. This infection is a possible cause of the bone and joint lesions seen in the pelvis.

**Ulcerative Colitis.**—In the past 3 years, seven patients with ulcerative colitis have been observed by one of us (R.G.G.) to develop a bilateral sacro-iliac arthritis within 2 or 3 years of the onset of colitic symptoms. This arthritis produced a radiological picture identical with that seen in early ankylosing spondylitis (Fig. 6, opposite). In two of these patients changes have developed in the lumbar vertebral bodies and ligaments typical of those of ankylosing spondylitis. Similar cases have been reported by Steinberg and Storey (1957) and by Wilkinson and Bywaters (1958). It is suggested that, in these patients, chronic infection of the colon and rectum has stimulated the development of bilateral sacro-iliitis. In several patients this has proved to be the onset of classical ankylosing spondylitis.

**Ankylosing Spondylitis.**—The radiological features of the arthritis of the sacro-iliac joints has been previously described by Knutsson (1950) and Grainger (1957). There appears to be no essential difference between these changes and those seen in Reiter's syndrome, non-specific urethritis, paraplegia, and ulcerative procto-colitis. The minor differences that have been observed are probably only ones of degree, intensity of reaction, and symmetry.

The complete pelvic radiological picture of ankylosing spondylitis is shown in Fig. 7 (overleaf).

**Discussion**

Chronic pelvic infection is present in all the other clinical varieties of sacro-iliac arthritis mentioned above. If this can be shown to be also present in a statistically significant proportion of patients with ankylosing spondylitis, it may well be that this pelvic infection is of aetiological significance in the development of this condition. Romanus (1953) found that
PELVIC INFECTION AS A CAUSE OF BILATERAL SACRO-ILIAC ARTHRITIS

95 per cent. of his patients with ankylosing spondylitis had a chronic prostatovesiculitis. Recently Mason and others (1958) have confirmed this surprisingly high incidence, by finding that 83 per cent. of 54 patients with ankylosing spondylitis had a chronic prostatovesiculitis, as opposed to 33 per cent. of a control group.

Conclusions

Clinical and radiological evidence of bilateral, non-pyogenic sacro-iliitis must not be regarded as an exclusive feature of ankylosing spondylitis. The essential similarity between the radiological changes in the sacro-iliac joints found in diseases with a focus of urogenital pelvic infection, and those found in ankylosing spondylitis, suggests that there may be some common aetiological factor. The development of apparently typical, classical ankylosing spondylitis a few years after the onset of ulcerative proctocolitis has been observed personally in seven patients. Similar case reports have recently been published from other clinics, suggesting that chronic rectal or colonic sepsis may also be an aetiological factor.

It has recently been confirmed that 80 to 90 per cent. of patients with ankylosing spondylitis have a chronic prostatovesiculitis. It is therefore suggested, following the lead of Romanus (1953) and other authors, that chronic pelvic infection may be an exciting or provocative factor in the development of sacro-iliac arthritis. In some patients, probably genetically determined, this heralds the onset of classical ankylosing spondylitis.

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A more detailed presentation of this thesis by R. G. G. is to be published in the Journal of the Faculty of Radiologists. I am grateful to the Editors of both Journals for permitting this arrangement.

REFERENCES
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