Our patient had been receiving the drug for ~3 yr and her sicca symptoms became rather profound over recent months, associated with severe swelling of the parotids. It is difficult to be certain about how long she had had positive serology for the Ro antibody in her blood as it was tested soon after the development of the sicca symptoms. Although anti-Ro is commonly associated with various connective tissue diseases, none of these were present on clinical examination.

The clinical presentation of our patient had raised several possibilities, including metastatic carcinoma, but the lymph node biopsy finding was reassuring as it had no evidence of secondaries or primary lymphoreticular malignancy. The lymph node biopsy showed no evidence of focal necrosis, or eosinophilic or histiocytic cell infiltrate which is a characteristic feature of PLS [2-5].

It was most interesting to note the transient serological abnormality, which took ~3 months to become normal together with complete resolution of her symptoms. The dramatic effect of withdrawal of the drug on the size of her parotid glands was also remarkable.

Our case highlights problems in many areas of clinical management, including multiple unproven diagnoses, polypharmacy and perhaps indiscreet duration of therapy. An aggressive but cautious approach to the reduction of steroid had met with no untoward effect and she remains well at a considerably lower dose of steroid. Her blood glucose control is very much improved and it is possible that she may not require any anti-hypertensive therapy in future.

To our knowledge, this is the first case of pseudo-Sjögren's syndrome associated with phenytoin therapy and rheumatologists should be aware of this reversible clinical entity.

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Cervical Spondylodiscitis in a Patient with Ankylosing Spondylitis

Sr—First described in 1937, spondylodiscitis is an uncommon but well-recognized complication in patients with ankylosing spondylitis (AS) [1]. The literature suggests that such lesions are invariably confined to the thoracic and lumbar spines [2]. We report here the case of a 58-yr-old man with a 22 yr...
Having had stable disease for >10 yr, Mr AB was seen urgently in December 1992 complaining of increasing pain in his spine with paraesthesiae in his right hand following a fall 6 months earlier, whereby he fell backwards and landed on his shoulder blades. Physical examination revealed total spinal movement restricted to 35° (normal range >60°), chest expansion diminished to 3 cm, a lateral flexion deformity in his cervical spine with reduction in total movement and a wall to tragus distance of 23.5 cm. No localized tenderness in the spine was found and, in particular, there were no objective neurological signs. Investigations revealed haemoglobin of 11.6 g/dl, total white cell count of 10.3 x 10⁹/l, ESR of 70 mm/h and CRP of 33 mg/l. X-rays showed bony ankylosis of the lower cervical spine mainly at the level of C4/5/6 and some degree of spondylosis of C6/7.

Despite initial improvement with physiotherapy/hydrotherapy, a year later he was noted to have increasing pain in his cervical spine radiating into both arms. In addition, neurological examination revealed weakness of his right hand and sensory loss in the C7/8 dermatome on the same side. Reflexes were generally brisk, but symmetrical, plantar responses flexor. Repeat X-rays of his cervical spine now showed a 'pseudoarthrosis' at the level of C6/7. Urgent MRI revealed marrow replacement involving the body and posterior neural arch of C7 with some tissue swelling causing cord compression at this level (Fig. 1), suggesting an inflammatory or, possibly, a neoplastic process. The patient subsequently underwent an open biopsy of the lesion via the posterior approach after a failed CT-guided needle biopsy. Multiple specimens showed only inflammatory infiltration with no evidence for neoplasm (Fig. 2), thought to be consistent with spondylodiscitis. The patient was treated conservatively with a cervical collar, analgesia and further physiotherapy with subsequent improvement in his neurological symptoms. He remained stable 18 months later on follow-up.

Pain and tenderness localized to the affected disc are the most common presenting features of spondylodiscitis, although it can be asymptomatic and only detected on routine radiographic examination many years later. Based on the proposed classification by Cawley et al. [3], our patient had a Type III lesion which is typically associated with disease of 15 yr or more and is uncommon in non-ankylosed spines. The

**Fig. 1.**—Plain lateral radiograph of Mr AB's cervical spine (left panel) showing extensive bony fusion of the cervical vertebrae at several levels with a 'pseudoarthrosis' at C6/7. The corresponding magnetic resonance image is shown in the right panel.
The aetiology of spondylodiscitis in AS is unclear. Our patient gave a history of preceding trauma, but the role of trauma in the pathogenesis of these lesions remains contentious. Interestingly, there appears to be a strong association between trauma and/or heavy manual work and Type III lesions [4]. Currently, MRI is the most sensitive and reliable method of diagnosing these lesions [5]. However, it is not specific and histological diagnosis is still desirable, especially if the lesion is in an unusual site, as in our patient. The histology of these lesions, however, is variable depending on the different phases of the disease process. During the initial inflammatory phase, there is marked lymphocytic infiltration while reactive new bone formation with scanty inflammatory foci is seen during the healing phase [6]. The histology of Type III lesions is consistent with a pseudoarthrosis with haemorrhage, fibrous tissue, a small amount of callus, sclerosis of the adjacent vertebral bone and only mild inflammatory cell infiltrate. The literature suggests that patients with spondylodiscitis ought to be managed conservatively with spinal support during the acute phase, physiotherapy and subsequent modification of occupation [3]. The majority of patients with Type III spondylodiscitis, however, require spinal fusion as these lesions tend to heal with incomplete ankylosis and thus are unstable. It remains to be seen whether our patient will progress to cervical vertebral fusion in the future.

In conclusion, the presence of cervical spinal pain in late-stage AS should alert the clinician to the possibility of spondylodiscitis, particularly if the disease has previously been quiescent and there is a history of preceding trauma.

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