arthroscopy (7), a pubic osteitis after bladder surgery (8) and a dorsal spondylodiscitis in a patient treated with steroids have been reported (9). Trauma or immunosuppression appear to be important promoting factors.
Spondylodiscitis after discectomy is rare, but an increasing number of reports have emerged during this last decade (10). Hematoma or tissue remaining after an operation have been presented as favourable growth media. Our case shows that absence of fever, normal WBC counts and a moderate increase of ESR and CRP do not exclude the possibility of an acute spondylodiscitis, especially with low virulent bacteria. Moreover, SL spondylodiscitis can develop in non-immunosuppressed patients. SL disclosed in blood samples should not be considered as a contamination, especially if more than one sample reveals the pathogen.

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References

1. HERCHLINE TE, AYERS LW: Occurrence of *Staphylococcus lugdunensis* in consecutive clinical cultures and relationship of isolation to infection. *J Clin Microbiol* 1991;29:419-21.
2. VANDENESCH F, ETIENNE J, REVERDY ME, EYKYN SJ: Endocarditis due to *Staphylococcus lugdunensis*: Report of 11 cases and review. *Clin Infect Dis* 1993; 17: 871-6.
3. LUDLAM H, PHILLIPS I: *Staphylococcus lugdunensis* peritonitis [Letter]. *Lancet* 1989; 2: 1394.
4. FLEURETTE J, BÈS M, BRUN Y *et al*.: Clinical isolates of *Staphylococcus lugdunensis* and *S. schleiferi*: Bacteriological characteristics and susceptibility to antimicrobial agents. *Res Microbiol* 1989; 140: 107-18.
5. ORTIZ DE LA TABLA V, GUTIERREZ-RODERO F, MARTIN C, ZORRAQUINO A, BELINCHON I: *Staphylococcus lugdunensis* as a cause of abscesses in the perineal area. *Eur J Clin Microbiol Infect Dis* 1996; 15: 405-7.
6. WAGHORN DJ: *Staphylococcus lugdunensis* as a cause of breast abscess [Letter]. *Clin Infect Dis* 1994; 19: 814-15.
7. PALAZZO E, PIERRE J, BESBES N: *Staphylococcus lugdunensis* arthritis: A complication of arthroscopy. *J Rheumatol* 1992; 19: 327-8.
8. RUIZ L, CORBELLA X, AGULLO JL, VERDAGUER R, CABO X: Pubic osteitis caused by *Staphylococcus lugdunensis* [Letter]. *Enferm Infec Microbiol Clin* 1998; 16: 152-3.
9. MURDOCH DR, EVERTS RJ, CHAMBERS ST, COWAN IA: Vertebral osteomyelitis due to *Staphylococcus lugdunensis. J Clin Microbiol* 1996; 34: 993-4.
10. RHODE V, MEYER B, SCHALLER C, HASSLER WE: Spondylodiscitis after lumbar discectomy. *Spine* 1998; 23: 615-20.

Peculiar myelopathy in a patient with overlap syndrome with lupus- and rheumatoid-like symptoms

Sir,
Spinal cord lesions are rare in patients with systemic lupus erythematosus (SLE) or rheumatoid arthritis (RA) except for transverse myelitis (TM) in SLE or compression myelopathy induced by atlantoaxial subluxation in RA (1-4). Small vessel vasculitis may lead to peripheral neuropathy in RA patients with persistent elevation of rheumatoid factor (RF) but seldom cause myelopathy (2). We describe a patient with overlap syndrome characterized by features of SLE and RA who developed widespread plaques in the thoracic spinal cord mimicking multiple sclerosis (MS), but who presented with neurological signs much more like those found in TM (3, 4).
A 32-year-old woman had bilateral arthritis for 9 years, involving the proximal interphalangeal (PIP), metacarpophalangeal (MCP), metatarsophalangeal, wrist, knee, elbow, and ankle joints. At onset these presentations were recognized as RA because the patient had high and fluctuating serum RF, and juxtaarticular osteoporosis of the PIP and MCP joints, as demonstrated by radiography. Earlier treatments included non-steroidal antiinflammatory drugs, corticosteroids, gold, hydroxychloroquine, and D-penicillamine, but were administered irregularly. Deformities including hallux valgus and subluxation of the interphalangeal joints of thumbs occurred despite treatment. Weight loss (10 kg), splenomegaly, and leukopenia developed later. However, there was no hematuria, proteinuria, rash, hair loss, sicca complex, Raynaud's phenomenon, hypocomplementemia, or serositis.
Subsequently, the patient's polyarthritis and myalgia deteriorated and methotrexate (7.5 mg/week) was started. This drug was only used for 2 months because abnormal liver function appeared and the patient suffered from upper gastrointestinal bleeding. Numbness of the lower limbs occurred soon afterwards, but was not associated with motor deficits. A nerve conduction study, electromyography (EMG), and magnetic resonance imaging (MRI) of the spine failed to show abnormalities. Four months prior to admission to our hospital, a sudden and complete paralysis of the lower extremities bilaterally and urinary incontinence occurred, which were complicated by the rapid development of a large decubitus ulcer in the sacrum.
Neurological examination on admission revealed slightly diminished strength in the quadriceps, and hip flexion and extension. Ankle extension and flexion were poor. Knee jerks were normal, but ankle hyperreflexia and bilateral Babinski signs were present. Pin prick and light touch sensation was impaired below T7, vibration sensation was impaired below the knees, and proprioceptive sensation was impaired below the ankles. There was flaccid paresis over both legs. The upper extremities were entirely normal. Another EMG, motor and sensory nerve conduction studies, and MRI were unrevealing. A tibial nerve sensory evoked potential (EP) study disclosed marked diminution of the cortical potentials, suggesting thoracic myelopathy. A urodynamic study showed poor compliance of the urinary bladder. Visual EP, computerized tomography (CT) myelogram, and MRI of the brain were negative. However, a follow-up MRI disclosed multiple discrete hyperintense plaques in the thoracic spinal cord (Fig. 1). A follow-up radiograph showed flexion deformities and ulnar deviation of the MCP joint with minimal bone and cartilage erosions.
Laboratory studies showed WBC 2,600/mm^3 without shift, hemoglobin 10.0 g/dL, mean corpuscular volume 88.5 fL, mean corpuscular hemoglobin 30.9 pg, mean corpuscular hemoglobin concentration 34.9 g/dL, RF

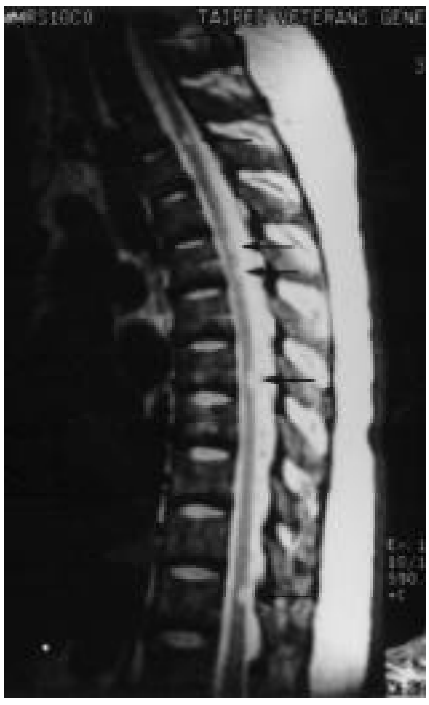

Fig. 1. MRI of the thoracic spine 2 months after paralysis shows 5 discrete high signal plaques, 2 x 5 mm in size, on a long repetitive time (T2-weighted) sagittal image at the level of T3-4, T6, T8 and T12 (**arrows**), which are very similar to the lesions of multiple sclerosis.

1,590 IU/ml, IgG 4,490 mg/dL, IgA 574 mg/dL, IgM 245 mg/dL, C3c 58 mg/dL, C4 20.2 mg/dL, and C-reactive protein (CRP) 2.0 mg/dL. The partial thromboplastin time and prothrombin time were normal. Direct and indirect Coombs' tests were negative. Antinuclear antibodies (ANA) were 1:640 (homogenous) and anti-dsDNA antibodies were 40 IU/ml. Anti-histone and anti-U1 ribonucleoprotein (RNP) antibodies, as well as circulating immune complexes (CIC, 90 mg/ml heat aggregated human globulin) were present, but anti-Smith, anti-SS-A/Ro, IgM and IgG anticardiolipin antibodies (aCL), and cryoglobulin were absent. Urinalyses were negative. A cerobrospinal fluid (CSF) study revealed the presence of oligoclonal bands. Antibodies against HIV and Type I human T cell leukemia virus were negative.

Intravenous (IV) methylprednisolone 125 mg q 6 hr was given initially. A course of IV pulse therapy with cyclophosphamide (600 mg/month) (1) was given soon after the diagnosis was confirmed. There was no discernible recovery of neurological deficits at the 4th month. However, muscle power, sensation in the lower extremities and voiding strength showed partial improvement at the 6th month and another MRI revealed the complete disappearance of the plaques. Approximately two years later, however, the patient died of a febrile episode in another hospital, which was associated with bacteremia originated from *E. coli* and *P. aeruginosa*. There was no newly developed neurological insult up to her death.

The patient's overall presentation seemed to fulfill the diagnosis of Felty's syndrome because she had clinical and laboratory evidence of RA, anti-histone antibody, massive splenomegaly, weight loss, and granulocytopenia (5, 6). However, the diagnosis of SLE or mixed connective tissue disease (MCTD) cannot be definitely excluded because oral ulcer, leukopenia, high ANA titers, low anti-dsDNA antibody titers, and CIC were present, despite the absence of hair loss, photosensitivity, renal disease, hemolytic anemia, hypocomplementemia, or Raynaud's phenomenon. Besides, the joint lesions in the hands became much more like the Jaccoud's arthropathy found in patients with SLE as time lapsed. Although most of the laboratory findings could also be present in a severe form of RA such as Felty's syndrome (5, 6), they would not appear simultaneously or in the same patient. The significance of anti-U1 RNP antibodies is unclear. Whether they can be present in Felty's syndrome is unknown. Interestingly, the association of this autoantibody with Jaccoud's arthropathy has been reported (7). Because the patient did not develop Raynaud's phenomenon throughout the course of her disease, no sufficient evidence suggested the definite presence of MCTD.

The most confusing but interesting point was the remarkable disagreement between the neurological manifestations and the anatomical distribution of the myelopathy. As revealed by MRI, the lesions spread from T3 to T12, much more like the discrete plaques of MS than the cord widening seen in TM (3, 8). The brain MRI and visual EP were normal, but CSF contained oligoclonal bands (9-11). The clinical presentation resembled TM at the T7 level. Thus, it could not be determined whether she had TM or MS. Some TM cases may evolve to MS (12). On the other hand, plaques in MS may be totally asymptomatic (13). Hence, it was possible that only one plaque accounted for her TM-like symptoms.

Numerous factors may be implicated in the pathogenesis of these neurological manifestations (4). IgM and IgG aCL were absent and there was no strong evidence to suggest the lupus anticoagulant. Sjögren's syndrome was less likely because the patient did not present with sicca complex or anti-SS-A/Ro antibodies. HIV-associated myelopathy is radiologically similar to TM (14), but enzyme immunoassays had excluded this possibility. Whether it was a presentation of "pseudo-Felty syndrome with large granular lymphocytes" or non-Hodgkin's lymphoma was unknown because we did not study the bone marrow or lymphocyte subpopulation. On the other hand, there was no trauma, radiation injury, vaccination, or evidence indicating a viral or bacterial infection prior to paraparesis. The presumable cause may be immune complex-induced vasculitis affecting vertebral vessels, which could lead to cord infarction. Supportive evidence included raised CIC, persistently high RF, and oligoclonal bands in the CSF, which were parallel to paraparesis. Regardless of the underlying pathogenesis, the development of this bizarre thoracic myelopathy in a patient with overlapping symptoms mimicking RA and SLE was very unusual.

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References

1. MOK CC, LAU CS, CHAN YET, WONG RWS: Acute transverse myelopathy in systemic lupus erythematosus: Clinical presentation, treatment, and outcome. *J Rheumatol* 1998; 25: 467-73.
2. BROWN MM, SWASH M: Polyarteritis nodosa and other systemic vasculitides: Vasculitis associated with connective tissue diseases: Rheumatoid arthritis. *In:* VINKEN PJ, BRUYN GW and KLAWANS HL (Eds.) *Handbook of Clinical Neurology*, Revised Series 11, Vascular Diseases, Part III, vol. 55. Amsterdam, Elsevier Science 1989: 353-68.
3. PROVENZALE JM, BARBORIAK DP, GAENSLER EHL, ROBERTSON RL, MERCER B: Lupus-related myelitis: Serial MR findings. *AJNR Am J Neuroradiol* 1994; 15: 1911-7.
4. WALLACE DJ, METZGER AL: Systemic lupus erythematosus and the nervous system. *In:* WALLACE DJ and HAHN BH (Eds.) *Dubois' Lupus Erythematosus*, 5th ed. Baltimore, Williams and Wilkins 1997: 723-54.
5. CAMPION G, MADDISON PJ, GOULDING N *et al.*: The Felty syndrome: A case-matched study of clinical manifestations and outcome, serologic features, and immunogenetic associations. *Medicine* 1990; 69: 69-80.
6. PINALS RS: Felty's syndrome. *In:* KELLEY WN, HARRIS ED JR, RUDDY S and SLEDGE CB (Eds.) *Textbook of Rheumatology*, 5th ed. Philadelphia, W.B. Saunders 1997: 951-4.
7. REILLY PA, EVISON G, MCHUGH NJ, MADDISON PJ: Arthropathy of hands and feet in systemic lupus erythematosus. *J Rheumatol* 1990; 17: 777-84.
8. NILSSON O, LARSSON E-M, HOLTÅS S:. Myelopathy patients studied with magnetic resonance for multiple sclerosis plaques. *Acta Neurol Scand* 1987; 76: 272-7.
9. POSER CM, PATY DW, SCHEINBERG L *et al.*: New diagnostic criteria for multiple sclerosis: Guidelines for research protocols. *Ann Neurol* 1983; 13: 227-31.
10. PATY DW, OGER JJF, KASTRUKOFF LF *et al.*: MRI in the diagnosis of MS: A prospective study with comparison of clinical evaluation, evoked potentials, oligoclonal banding, and CT. *Neurology* 1988; 38: 180-5.
11. OFFENBACHER H, FAZEKAS F, SCHMIDT R *et al.*: Assessment of MRI criteria for a diagnosis of MS. *Neurology* 1993; 43: 905-9.
12. TIPPETT DS, FISHMAN PS, PANITCH HS: Relapsing transverse myelitis. *Neurology* 1991; 41: 703-6.
13. HARRIS JO, FRANK JA, PATRONAS N, MCFARLIN DE, MCFARLAND HF: Serial gadolinium-enhanced magnetic resonance imaging scans in patients with early, relapsing-remitting multiple sclerosis: Implications for clinical trials and natural history. *Ann Neurol* 1991; 29: 548-55.
14. BARAKOS JA, MARK AS, DILLON WP, NORMAN D: MR imaging of acute transverse myelitis and AIDS myelopathy. *J Comput Assist Tomogr* 1990; 14: 45-50.