Table I. Clinical features of 2 female patients with joint hypermobility syndrome.

	J.S. (b. 1980)	B.P. (b. 1969)
Family history for JHS	+	?
Marfanoid habitus	+	+
Pectus carinatum	+	+
Kyphoscoliosis	+	+
Arachnodaktyly	0	+
Beighton score	9/9	8/9
BMD (T-score)	-1.8	-2.3
Arthralgia/arthritis	+	++
Back pain / headache	++	++
Flat feet	+	0
Skin striae	0	++
Easy bruising	+	+
Varicose veins	0	++
Mitral valve prolapse	+	+
Anxiety, panic	++	0
SRP due to total bilateral occlusion of the ulnar arteries	+	+

BMD = bone mineral density; 0 = absent, +/++ = present, ? = unknown.

the cause of the bilateral occlusions of the ulnar arteries remained unclear.

Both patients were successfully treated with iloprost infusions and calcium channel blockers.

Only a few rare cases of SRP/hand-arm vibration syndrome due to bilateral occlusion of the forearm arteries have been published (9, 10). SRP caused by bilateral occlusion of the ulnar arteries may represent a new extra-articular feature in benign JHS.

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Spondylodiscitis due to *Staphylococcus lugdunensis*

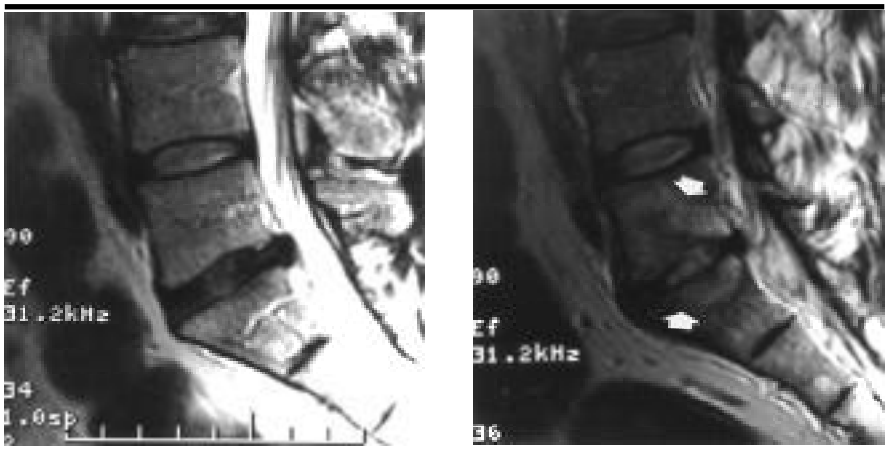
Sir,

Staphylococcus lugdunensis (SL) is a recently described coagulase-negative staphylococcus and a normal constituent of the human skin flora (1) which can occasionally induce an opportunistic infection. We describe a case of spondylodiscitis due to this species which occurred 2 months after disc surgery.

In December 1998, a 39-year-old man with no relevant medical history was operated on for a L5-S1 disc herniation. One month later a clinical control by the neurosurgeon revealed only a slight persistence of impaired lumbar mobility. Two months after surgery, however, he presented with acute low back pain irradiating into the left leg. MRI showed a hypointensity of the disc and of the adjacent parts of the vertebral bodies on T1-weighted images; these structures appeared hyperintense on T2-weighted sequences (Fig. 1b), markedly different from the pre-operative images (Fig. 1a). The patient was then referred to our department.

He had no signs of fever and the general examination was normal. Tenderness was elicited by palpation of the lower lumbar vertebrae and spinal movements were reduced. A neurological examination was normal. Laboratory tests were normal at admission, but 3 days later the erythrocyte sedimentation rate (35 mm) and C reactive protein (29 mg/l) were increased. The white blood counts remained normal. Two sets of blood culture taken at one-day intervals revealed gram-positive cocci that were identified as SL. Antibiotic profiles by a disc susceptibility test were identical on both isolates, which were sensitive to penicillin. There was no portal entry disclosed. Transthoracic and transoesophagic echocardiographies were normal. Penicillin G (1 g IV every 6 hrs) was started 48 hours after admission and continued for 4 weeks, followed by oral administration of rifampicin (600 mg/d) and ciprofloxacin (3 x 750 mg/d) for another 4-week period. Pain and lumbar stiffness diminished gradually. Blood cultures became negative and both the ESR and CRP decreased to within normal ranges after 30 days.

SL is differentiated from other coagulase-negative staphylococci by a positive ornithine decarboxylase reaction (1). They are preferentially located at the perineum (2) and are associated with skin infections (1), endocarditis (2), peritonitis in patients undergoing peritoneal dialysis (3), vascular prosthesis infection (4), and brain, perineal and breast abscesses (2, 5, 6). A knee infection after



(a) **(b)**

Fig. 1. MRI T2-weighted images of the lumbar spine (sagittal view): (**a**) disc herniation of the 5th lumbar interspace (2 months before the operation); (**b**) high-signal intensity of the intervertebral disc L5-S1 and oedema (**arrows**) of the adjacent vertebral bodies (at admission).

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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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

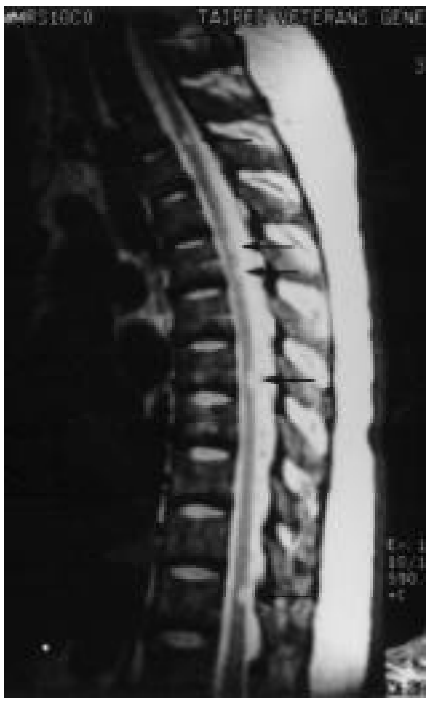

Fig. 1. MRI of the thoracic spine 2 months after paralysis shows 5 discrete high signal plaques, 2 x 5 mm in size, on a long repetitive time (T2-weighted) sagittal image at the level of T3-4, T6, T8 and T12 (**arrows**), which are very similar to the lesions of multiple sclerosis.