serum VZ CFT titre was 1/64; serological screening for other viruses was negative; syphilitic serology, Paul-Bunnell and sickle tests were negative; a second lumbar puncture performed one week after admission revealed 432 WBC/mm³ (95% lymphocytes), CSF protein 0·38 g/l, CSF glucose 2·7 mmol/l (blood glucose 5·6 mmol/l). The immunoglobulin and complement levels were normal and skin tests for cell-mediated immunity showed normal responses. He was well at follow-up six months later.

During chickenpox infection, virus in the skin vesicles probably ascends intra-axonally along nerves to the sensory ganglia where it may reside latently for many years. Reactivation of zoster causes inflammatory necrosis of the ganglion with degeneration of related nerve roots accompanied by severe neuritis, a unilateral segmental inflammatory myelitis and localised leptomeningitis.[2] As many as 40% of patients with uncomplicated zoster have a mild pleocytosis or a slightly elevated CSF protein or both.[1] Nevertheless, despite this "localised meningitis" clinical meningitis is extremely rare. Where it occurs it is usually in association with encephalitis.[3 4] The most common presentation is meningitis with a disturbed sensorium but ataxia, convulsions and coma may supervene and in addition myelitis and cranial nerve palsies may further complicate the clinical picture.[1 2 5 6] Hypoglycorrhachia is a recognised feature of infection with herpes zoster where meningitis,[7] meningoencephalitis[4 8] and encephalomyelitis[5] occur. The CSF glucose of 2·7 mmol/l (blood glucose 5·6 mmol/l) in our patient is consistent with, but less marked than in the previous reports cited. The diagnosis of herpes zoster meningitis may be rapidly established by immunofluorescent staining of varicella-zoster antigen on CSF cells[9–11] or measurement of varicella-zoster antibody in CSF by indirect immunofluorescence.[12] Varicella-zoster antibody measured by complement fixation tests must be interpreted in relation to the serum antibody level. Dissemination of herpes zoster occurs in less than 2% of the general population and usually among older age groups.[1] In immunosuppressed patients particularly those with lymphoproliferative disease or taking corticosteroids, this rises to 15% of cases of zoster.[13] Although the prognosis is good[1] complications are both unpleasant and serious; effective therapy may now be available in the form of acyclovir[14–16] and possibly vidarabine.[17 18]

I thank Dr CM Oakley for permission to report this case, Dr D Carrington for the virological studies, Dr PC Gautier-Smith for helpful comments and advice and Mrs J Salter for kindly typing the manuscript.

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Accepted 1 February 1983

Neurological lupus erythematosus with tonic seizures simulating multiple sclerosis

Sir: Neurological manifestations occur in 20–60% of all patients with systemic lupus erythematosus (SLE).[1–4] Included among previous clinical reports of neurological lupus are references to features resembling multiple sclerosis.[5–9] Although well recognised in multiple sclerosis,[10 11] tonic seizures have not been previously described in patients with SLE. A patient with serologically proven SLE is described who presented with clinical features consistent with multiple sclerosis including tonic seizures.

A 51-year-old single woman presented in February 1980 with a seven week history of pain, numbness and weakness of the right arm. Numbness gradually progressed to the right leg, left leg and then left arm associated with a band of numbness over the epigastrium. There was a transient Lhermitte's phenomenon and for three weeks horizontal diplopia. Over seven weeks she had become increasingly unsteady with leg weakness and was confined to bed. Apart from pneumonia one year previously there was no significant past history. On examination she was alert but bed bound with a moderately severe spastic quadriparesis. There was pseudoathetosis and moderate ataxia of the arms. The deep reflexes were normal with bilaterally extensor plantar responses. Superficial sensation was intact but there was marked loss of proprioception and vibration sense in hands and feet. Apart from a first degree horizontal nystagmus on right and left lateral gaze, cranial nerve examination was normal. The general examination was normal. It was felt that the patient had evidence of two lesions, one in the brain stem and the other, major, lesion in the posterolateral columns of the high cervical cord. Investigation revealed an ESR of 94 mm in the first hour with a white cell count of 3,800/mm³ and normal differential white cell count. Anti-nuclear anti-

bodies (ANA) were positive at a titre of 1:400 in an homogenous pattern. Antibodies to double stranded DNA were demonstrated by positive DNA – binding of 57% (Farr assay)[12] and by positive immunofluorescence using Crithidia luciliae.[13] VDRL and TPHA were negative. The cerebrospinal fluid (CSF) contained 24 lymphocytes/μl and protein 0·45 g/l. Radiology of the chest, skull and cervical spine, computed tomography of the brain and visually evoked potentials (VEPs) were normal.

The patient was treated with a reducing course of corticosteroids and almost immediately improvement occurred. Two weeks after steroid therapy had been commenced the patient developed tonic seizures. Each episode was precipitated by extending the left arm, lasted approximately 30 seconds, consisted of sudden extension of all four limbs with flexion of the trunk and was associated with a sensation of electric shocks down both arms. The patient had up to 20 attacks each day which were stopped by carbamazepine 300 mg daily. The patient was discharged walking slowly with one stick two months after admission and at the time of discharge the ESR was 21 mm/hr. Two weeks later the carbamazepine had to be increased to 600 mg daily because of recurrence of tonic seizures. The patient's clinical course is illustrated schematically in the figure.

She remained well without corticosteroids, with mild disability, for three months but in July 1980, over two weeks, she developed increasing right sided weakness with numbness across the abdomen and urgency of micturition. On examination there had been recurrence of the severe quadriparesis with weakness more marked on the right. Superficial sensation was reduced up to the C8 dermatome bilaterally. She was unable to stand. Investigation revealed ESR 72 mm/hr, ANA titre 1:400, DNA-binding 64% and the CSF contained 40 lymphocytes /μl. VEPs were now abnormal with a prolongation in the latency of the P2 component in the right eye to 151 ms (normal mean + 3 SD = 120 ms). With prednisolone 60 mg daily a rapid improvement occurred and she was discharged walking with one stick six weeks after admission. ESR at this time was 13 mm/hr. She again remained only mildly disabled for three months when, over one week, there was a deterioration in strength of the right arm and leg so that she needed the support of one person to walk. Neurological signs were similar to those of the previous admission but with only mild weakness of the right arm and leg. On this occasion the ESR was 50 mm/hr, ANA titre 1:1000 and DNA binding 57%. CSF examination showed an IgG/albumin ratio of 0·46 with a prominent slow gamma globulin region on polyacrylamide gel electrophoresis but no evidence of oligoclonal IgG. A skin biopsy for immunofluorescent examination showed no evidence of immunoglobulin deposition. Azathioprine 100 mg and prednisolone 30 mg daily were commenced and slow improvement occurred. Over the following two years no further relapses have occurred and the patient remains minimally disabled with a mild ataxia due to mild persistent posterior column sensory loss. Laboratory investigations have shown a normal ESR, DNA binding result and a reduced ANA titre of 1:100. No further tonic seizures have occurred on carbamazeprine 600 mg daily, steroids have been discontinued and the azathioprine dosage has been maintained. Throughout the course of her illness the patient has not developed any clinical evidence of cutaneous, articular, cardiopulmonary or renal disease.

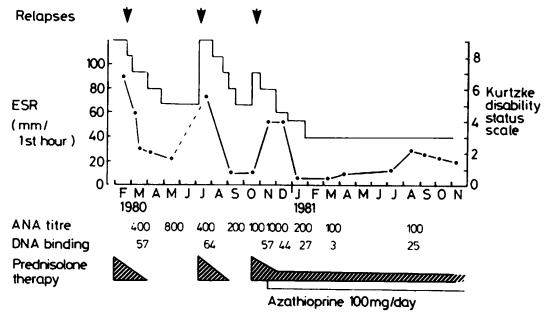

Fig *The course of the patient's illness is illustrated by reference to disability measured by Kurtzke's Disability Status Scale[14] (histogram). The three relapses are noted by the arrows. The ESR (closed circles) was elevated during relapses and fell during recovery following treatment with prednisone and azathioprine. The antinuclear antibody (ANA) titre tended to rise with relapse and fall during remission.*

All of the neurological features of this patient's illness, the remitting and relapsing pattern, evidence of multiple site involvement and tonic seizures are consistent with a diagnosis of multiple sclerosis. The clinical features indicated involvement of the cervical cord, brain stem and right optic pathway. Moreover, examination of the CSF demonstrated a raised IgG/albumin ratio. However the elevated ESR, leucopenia, presence of antinuclear antibodies in high titre, and positive tests for anti-DNA antibody suggested a diagnosis of SLE.[15,16] Neurological manifestations of SLE include convulsions, focal vascular lesions, chorea, cerebellar ataxia, cranial and peripheral neuropathies and psychiatric disturbances.[17] There are reports of patients with SLE resembling multiple sclerosis.[5–9] Fulford *et al* described six patients with a clinical picture resembling multiple sclerosis with laboratory findings which included positive LE cells and antinuclear antibodies. Five of the six patients had a slowly progressive spastic paraparesis and the term lupoid sclerosis was suggested. Involvement of other systems was minimal and included mild arthralgia and rash. Allen and her colleagues[9] described a patient with SLE having a relapsing remitting course characterised by diplopia, a spinal cord lesion, vertigo, deafness and later central retinal artery occlusion. Post-mortem examination showed involvement of the central nervous system only with vasculitis and necrosis of the mid-thoracic cord. One further patient with an illness resembling multiple sclerosis presented with a spinal cord lesion and optic neuritis due to SLE[8] but this patient had other systemic features including malar rash, a history of transient arthritis and later developed neurological features not consistent with multiple sclerosis. Tonic seizures have not been reported in SLE but are a characteristic feature of multiple sclerosis.[10,11] The patient described in this

report is therefore quite unusual in the way the clinical course mimicked that of multiple sclerosis but, in contrast to previously reported patients, responded well to steroids and azathioprine. This case further emphasises the wide spectrum of neurological manifestations of SLE and highlights the need for serological tests of antinuclear antibodies in patients presenting as multiple sclerosis.

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Accepted 1 February 1983

Ethmoid sinus carcinoma metastasing to the cauda equina: a case report.

Sir: A wide variety of neoplasms metastasise to the spine. The bony elements of the spine are most commonly affected but spread into the extradural space with subsequent compression of the spinal cord or cauda equina may occur. Intradural secondaries are rare. Extradural metastases usually present in subjects known to have malignant disease with constant unremitting low back pain which is particularly troublesome at night.[1] A period of up to several months may elapse before neurological symptoms and signs develop in the lower limbs. Later in the course of the disease sphincter problems develop. We report here a patient who developed both extra and intradural metastases in the lumbosacral region from a primary carcinoma of the ethmoid sinus and whose presentation was with low back pain and a neurogenic bladder. Cauda equina compression by metastases from a paranasal sinus carcinoma has not been reported previously and this mode of presentation is seen only infrequently.

A 48-year-old man presented with left proptosis and nasal discharge in 1979. Investigations showed a very extensive tumour arising from the left ethmoid sinus with local extension into the nasal cavity, the orbit, sphenoid sinus and left petrous bone. The histology showed a solid undifferentiated neoplasm. He was treated with radiotherapy and had an excellent response with marked clinical regression of the tumour. In July 1981 he developed pain over the ischial tuberosities which subsequently spread to the posterior thighs. He was first seen in this department in February 1982 when he described the pain as increasingly severe, constant and particularly troublesome at night. On specific questioning he also admitted to urinary frequency and poor flow. Examination revealed reduction in straight leg raising to 45° on both sides. The ankle jerks were depressed but the other reflexes were normal. There was no sensory loss. In the abdomen the bladder was palpable almost up to the umbilicus. There was no clinical evidence of local recurrence of his carcinoma. Investigations showed normal routine haematology. Radiographs of the spine and pelvis and a bone scan revealed no evidence of metastases. On the excretion urogram the bladder was greatly enlarged and did not empty after micturition. Lumbar myelography showed multiple irregular indentations at the level of L5 indicative of tumour. At surgery he was found to have extradural tumour extending from the upper border of the L5 vertebrae to below the S2 level and invading sacral bone. In addition there was tumour within the dural sac involving the nerve roots of the canda equina. Biopsy of intradural and extradural tumour showed undifferentiated carcinoma. Decompressive laminectomy produced marked improvement in his symptoms. In particular micturition returned almost to normal and the bladder could no longer be palpated on abdominal examination.

Carcinoma of the paranasal sinuses almost always presents with local disease, although this is frequently extensive at the time of the diagnosis. Distant metastases are only rarely evident at this stage, but are frequent in the later course of the disease, although again usually overshadowed by the problems of local extension of the original tumour.[2] Metastases to the vertebrae have been rarely described with these tumours[3] but neither extradural nor intradural secondaries have been reported. In addition the mode of presentation was unusual in that sphincter involvement occurred early as the major neurological problem, the only other neurological abnormality being symmetrically depressed ankle reflexes. Presentation of extradural tumour in this way is uncommon[4] but has been described as a feature of primary intradural tumours.[5] It therefore seems likely in this case that the intradural metastases were responsible for the bladder symptoms. We would like to draw attention to this hitherto undescribed complication of paranasal sinus carcinoma and also