Transverse Myelitis as the First Manifestation of Systemic Lupus Erythematosus or Lupus-like Disease: Good Functional Outcome and Relevance of Antiphospholipid Antibodies

DAVID P. D'CRUZ, SUSANA MELLOR-PITA, BEATRIZ JOVEN, GIOVANNI SANNA, JUDITH ALLANSON, JAMES TAYLOR, MUNTHER A. KHAMASHTA, and GRAHAM R.V. HUGHES

ABSTRACT. ***Objective.*** Transverse myelitis (TM) is a rare complication of systemic lupus erythematosus (SLE). Although usually a late manifestation of SLE, it can occur at presentation. We investigated the clinical presentation, treatment and outcome of 15 patients with TM as the presenting manifestation of SLE or lupus-like disease.

Methods. All patients received corticosteroids, while 13 also received immunosuppressive therapy. Five patients were fully anticoagulated with warfarin.

Results. A sensory level with spastic lower limb weakness and sphincter disturbance was the most common presentation: 14/15 patients had a thoracic or cervical sensory level. Cerebrospinal fluid examination showed high protein concentrations in 3 patients and oligoclonal bands in 8. Eleven of the 15 (73%) had antiphospholipid antibodies (aPL). Of the 15 patients, 3 had complete resolution of the symptoms, 6 had good functional improvements, 5 had good to fair outcome with some functional deficit, and one patient who received corticosteroids alone later died from pneumonia.

Conclusion. We describe 15 patients with TM as the presenting manifestation of SLE or lupus-like disease with a high prevalence of aPL. Our data support the view that early diagnosis and immunosuppressive therapy may be superior to corticosteroids alone in improving functional outcome. In those patients with aPL, antiplatelet agents and/or warfarin should also be considered. (J Rheumatol 2004;31:280–5)

Key Indexing Terms:
TRANSVERSE MYELITIS SYSTEMIC LUPUS ERYTHEMATOSUS
LUPUS-LIKE DISEASE ANTIPHOSPHOLIPID ANTIBODIES CYCLOPHOSPHAMIDE

Central nervous system (CNS) involvement may occur in 24–51% of patients with systemic lupus erythematosus (SLE)[1-3]. Transverse myelitis (TM), however, is a rare complication of SLE that occurs in 1–2% of patients. Although usually a late complication of SLE, several investigators have observed that it can occur at presentation. The clinical presentation of TM is usually characteristic and may have a rapid onset. Magnetic resonance imaging (MRI) commonly shows high signal lesions and cord edema on T2 weighted imaging, occasionally with enhancement implying blood–brain barrier leakage on T1 weighted sequences after gadolinium administration. There is no consensus on optimum management given the small number of patients who present with TM. However, early diagnosis and the use of immunosuppressive therapy appear to be associated with an improved outcome. It is increasingly clear that antiphospholipid antibodies (aPL) may have a role in the development of TM. In aPL-positive patients there may be a role for antiplatelet therapy or full anticoagulation in addition to immunosuppression. We review the clinical and serological features, treatment, and outcome of 15 patients with TM as the first manifestation of SLE.

From The Louise Coote Lupus Unit, St. Thomas' Hospital, London; Rivermead Rehabilitation Centre, Oxford; and Northampton General Hospital, Northampton, United Kingdom.

D.P. D'Cruz, MD, FRCP; S. Mellor-Pita, MD, PhD; B. Joven, MD, PhD; G. Sanna, MD, PhD; M.A. Khamashta, MD, PhD, FRCP; G.R.V. Hughes, MD, FRCP, Louise Coote Lupus Unit, St. Thomas' Hospital; J. Allanson, PhD, MRCP, Rivermead Rehabilitation Centre; J. Taylor, MRCP, Northampton General Hospital.

Address reprint requests to Dr. D.P. D'Cruz, The Lupus Research Unit, The Rayne Institute, St. Thomas' Hospital, London SE1 7EH, UK. E-mail: david.d'cruz@kcl.ac.uk

Submitted July 23, 2002; revision accepted April 15, 2003.

MATERIALS AND METHODS

We describe 15 patients who attended our lupus clinics over the past 10 years and who presented with TM as their initial manifestation of SLE. Only 4 fulfilled the updated American College of Rheumatology (ACR) criteria for the classification of SLE[4] at presentation. Subsequently 12 patients were classified as having SLE on the cumulative clinical features as well as fulfilling the ACR nomenclature for neuropsychiatric lupus[5], and 3 had lupus-like disease (Table 1). No patient fulfilled the accepted classification criteria for Sjögren's syndrome or multiple sclerosis.

TM was diagnosed by the characteristic clinical features of a spinal cord lesion, with associated sensory or motor deficits and sphincter disturbance documented clinically and on imaging.

Table 1. Summary of the clinical features, treatment, and outcome of TM as the presenting manifestation of SLE. Column 5 indicates the cumulative clinical features during the disease course. 1. LLD: lupus-like disease, APS: antiphospholipid syndrome, aCL: anticardiolipin antibodies (+ low, ++ moderate, +++ strong positive), DVT: deep venous thrombosis, PE: pulmonary emboli, AHAI: autoimmune hemolytic anemia, CSF: cerebrospinal fluid, OCB: oligoclonal bands, TB: tuberculosis, HSV: herpes simplex virus, MP: methylprednisolone, CYC: cyclophosphamide, AZA: azathioprine, MTX: methotrexate, ASA: acetylsalicyclic acid, LMWH: low molecular weight heparin, LP: lumbar puncture, LAC: lupus anticoagulant. [Continued next page]

Patient Diagnosis	Age	Sex	Clinical Neurology	Cumulative Features	CSF	MRI Spine	Treatment	Outcome
1 SLE	29	F	Spastic quadriparesis Asymmetric sensory level R C2; L T8	ANA 1/640 Proteinuria 0.6 g/day Leukopenia Anti-RNP+	WBC 5, RBC 5 Prot. 0.65 g/l OCB negative IgG > 140 mg/l	C2-T1 swollen with multiple enhancing areas within cord and pons	MP 1g (4 pulses) CYC (3 pulses) →AZA	Complete resolution MRI: improved
2 SLE	43	F	R leg monoparesis Sensory level C8-T1 Neurogenic bladder	Photosensitivity Malar rash ANA+ aCL IgM+, LA+ uveitis	WBC 5 Prot 0.57 g/l OCB positive IgG = 100 mg/l	Posterolateral disc C4-C5	ASA, warfarin Pred. CYC (6 pulses) AZA	Recovery to normal MRI: improved
3 LLD	68	F	Spastic paraparesis Sensory level T8 Neurogenic bladder	ANA 1/640 aDNA +	WBC 1 OCB positive	C2-6 patchy high signal within cord	MP (3 pulses) CYC (3 pulses) → AZA	Able to walk with stick. Sphincter control
4 LLD	26	F	Spastic paraparesis Sensory Level C4	ANA 1/640 aCL IgM + Raynaud's Dry eyes	WBC 0 Prot 0.24 g/l OCB positive IgG = 40 mg/l	C3 fusiform swollen lesion	ASA Pred high dose	Imbalance Sphincter control
5 SLE APS	28	F	Quadriparesis Sensory level C7 Bilateral Horner	ANA 1/1280 Anti-Sm, Ro + aCL IgG ++ DVT, PE Discoid rash	WBC 130 (90%L) Prot 1.3 g/l Gluc 1.8 TB, HSV cultures negative	C2-T1 swollen with patchy intrinsic contrast enhancement	Anti-TB, acyclovir Warfarin Pred	Walking with sticks Died from pneumonia MRI: myelomalacia
6 SLE	37	F	Spastic paraparesis Sensory level T4-5	ANA 1/1280 aCL IgG ++ Anti-Ro + Leukopenia	WBC 4 (98%L) Prot 0.33 g/l OCB positive IgG 10.9 mg/l	T1-T4 swollen with high signal changes	MP 1g (3 pulses) CYC (6 pulses) AZA	Able to walk with stick MRI: improved
7 SLE	41	F	Spastic paraparesis Sensory level T4 Loss of sphincter control	ANA 1/640, aDNA, Ro, La + aCL IgG ++, IgA + Thrombocytopenia AHAI, proteinuria Mouth ulcers	LP not done (warfarin)	Paraspinal lipomatosus without cord compression	MP (3 pulses) CYC (6 pulses), MTX LMWH	Improved to walking with difficulty. No sphincter control MRI: no change
8 SLE	45	F	Spastic paraparesis Neurogenic bladder Sensory loss S1 only	Thrombocytopenia Leukopenia ANA 1/640 aDNA, Ro + aCL IgG ++, Low C4	WBC 1 OCB negative Prot 1.3 g/l	High signal lesions T10-L2 Arterio-venous malformation Lumbar conus	CYC (6 pulses) Warfarin Spinal artery embolization	Normal gait MRI: improved
9 SLE	46	F	Spastic paraparesis Neurogenic bladder Ataxic gait Sensory loss T5	Raynaud's ANA 1/320 aDNA + aCL IgG +++, LA + low C4	Prot 0.41 g/l WBC 0 Gluc 3.3 OCB positive	High signal lesions: Brain stem, cerebellum C2-T6 cord L3/4 disc bulge	CYC (4 pulses) AZA Warfarin	Normal gait with minimal ataxia MRI: improved
10 SLE	43	F	Spastic paraparesis Loss of sphincter control Sensory level T6 L'hermitte's sign	Malar rash polyarthralgia ANA 1/320, LAC +	Prot normal OCB negative WBC 0	High signal lesions: L frontal lobe Cervico-medullary junction and T2-T6	MP (3 pulses) CYC (6 pulses) and warfarin	Excellent response Walking unaided with minimal ataxia L'hermitte's resolved
11 SLE	21	F	L arm weakness and Paresthesiae Sensory level C6	Oral ulcers Polyarthralgia Thrombocytopenia ANA 1/640 LAC+	Prot 0.33 g/l OCB positive WBC < 3	R periventricular lesion High signal lesions: C2-C4	MP 1 pulse Pred 7.5 mg ASA 75 mg	Mild sensory loss L arm only MRI: improved
12 LLD	49	F	Weakness/paresthesiae L arm. Upgoing plantars Normal sphincters	Arthralgia Livedo reticularis ANA + DNA +	Prot 0.3 g/l OCB positive WCC < 1	High signal lesions: Periventricular areas C4-C6, R C6/7 disc bulge	CYC (7 pulses) AZA, cyclosporine Now: MTX	Stable L arm weakness MRI: no change

Table 1. Continued.

Patient Diagnosis	Age	Sex	Clinical Neurology	Cumulative Features	CSF	MRI Spine	Treatment	Outcome
13 SLE	48	F	T10 sensory level, sphincter disturbance, spastic paraparesis	Butterfly rash, photosensitivity, oral ulcers, arthritis. DNA +, aCL IgG ++ LAC+	Prot 0.3 g/l OCB positive WCC < 1 Gluc 3.6	High signal lesions: Periventricular areas and pons	MP (3 pulses) CYC (6 pulses) Pred 7.5 mg ASA 75 mg	Marked improvement walking normally, MRI pons and cord lesions resolved
14 SLE	24	F	Spastic lower limb weakness, urinary incontinence. Brisk reflexes. Upgoing plantar bilaterally. Sensory loss T10	Leukopenia, lymphocytopenia, psychosis, malar rash, arthritis, ANA 1/320, anti-RNP +, Sm +, Ro +, low C4, LAC +, aCL IgG +++	Prot 2.1 g/l OCB not done Lymphocytosis Gluc 3.0	1994: normal brain MRI; unable to tolerate spine MRI 1998: normal spine MRI 2001: normal spine MRI	CYC (5 pulses) → AZA; now: pred 10 mg, aspirin	Improvement; residual L leg weakness; able to walk with stick
15 SLE	32	F	Increased tone, paresthesiae lower limbs. Sensory level T7 Normal sphincters	Malar rash, Raynaud's, livedo reticularis, arthritis, mouth ulcers, peripheral neuropathy, lymphopenia, teleangectasia, ANA +, RNP +, Sm +	Not done	Not done	Pred 20 mg, AZA Now: MTX alone	Complete recovery, residual sensory loss below knees from neuropathy

RESULTS

The clinical features are summarized in Table 1. All patients were women and the mean age at onset of TM was 38.7 years (range 21–68).

The most common motor deficit was a spastic paraparesis (11 patients), and 2 patients each had quadriparesis and monoparesis. Fourteen had a sensory level: 8 thoracic, 4 cervical, one sacral, and one patient had asymmetric cervical and thoracic sensory levels. Sphincter disturbance including a neurogenic bladder was present in 11 patients. Cerebrospinal fluid (CSF) analysis for cell count was normal in all but 2, who showed lymphocytosis. A high protein concentration was seen in 3 patients. Oligoclonal bands were detected in 8 of the 11 patients where it was performed. CSF cultures and bacterial serologies were negative.

Fourteen patients underwent spinal MRI scans at presentation and 13 were abnormal, although in 2 there were no cord lesions that were related to the neurological deficit (posterolateral C4/5 disc bulge and paraspinal lipomatosis without cord compression). One patient had 2 spinal MRI scans 4 and 7 years after presentation and these were normal. The other 11 patients all had the typical findings of high signal lesions within the cord, with cord edema in 4 patients and pontine lesions in 2 on T2 weighted imaging. At followup 8 patients had improvements in the MRI lesions (Figure 1); one progressed to severe myelomalacia, and in 2 the MRI appearances remained unchanged.

Characteristic features of SLE or lupus-like disease were detected in all patients, although at presentation with TM, only 4 fulfilled the ACR classification criteria. At followup, 12 patients could be classified as having SLE and 3 had a lupus-like disease. Lupus nephritis developed in one patient shortly after TM was diagnosed. Eleven patients (73%) had aPL, although in 2 patients (Patients 9 and 14) these were negative at presentation and became persistently strongly positive one and 7 years later, respectively.

Drug therapy was commenced according to the best judgment of the treating physician. All patients received corticosteroids [5 patients high dose prednisolone, 10 moderate dose (20–40 mg daily)]. Thirteen also received immunosuppressive therapy: 11 patients received low dose (500 mg) intravenous cyclophosphamide, followed by azathioprine in 7 and methotrexate in one, and azathioprine was started in 3 patients as the initial immunosuppressive treatment and methotrexate in one. In one case, antituberculous therapy and acyclovir were given because SLE was not initially suspected. This patient (Patient 5) later developed deep venous thrombosis and pulmonary embolism requiring warfarin and was anticardiolipin antibody (aCL)-positive — she thus had the antiphospholipid syndrome. Six of the 11 aPL-positive patients were treated with aspirin and 5 received warfarin.

Of the 15 patients, 3 had complete resolution of the symptoms, 6 had good functional improvements, 5 had a good to fair outcome with some functional deficit, and one

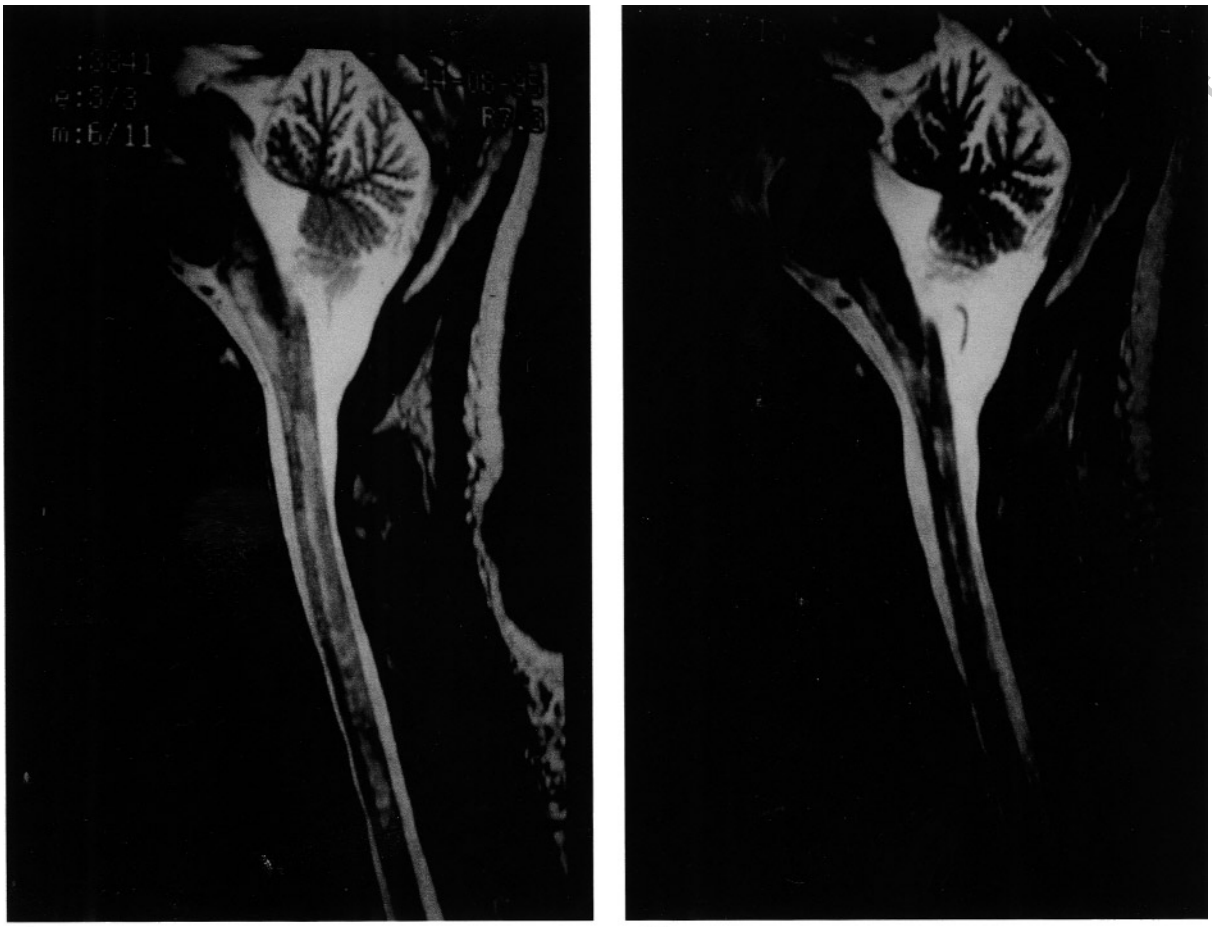

Figure 1. A. T2 weighted MRI of Patient 1 showing extensive high signal lesions with marked edema of the cervical cord. B. T2 weighted MRI 4 months later shows resolution of edema and improvement in high signal lesions after intravenous methylprednisolone and cyclophosphamide followed by azathioprine.

died one year later from pneumonia. This was after recurrent episodes of TM led to severe myelomalacia of the high cervical cord and severe quadriparesis requiring longterm ventilation. She had received only oral prednisolone with no immunosuppressive therapy (Table 1). The only other patient to receive prednisolone alone has remained stable with a mild sensory disturbance of the left arm. In the 11 patients who received intravenous low dose cyclophosphamide (Patients 1, 2, 3, 6, 7, 8, 9, 10, 12, 13, 14), there was a rapid response in neurological symptoms within 3 months of commencing cyclophosphamide. In comparison, of the other 3 patients, one died and the other 2 remained stable or had a slow incomplete recovery over the subsequent one year (Patients 4 and 11). In addition, 5 of the 11 patients with aPL received warfarin (Patients 2, 5, 8, 9, 10) and of these, 4 who had received both warfarin and intravenous cyclophosphamide had an excellent functional outcome, and Patient 5, who had warfarin but no immunosuppressive therapy, had a poor outcome (death from pneumonia). These 4 patients with aPL suggest that the addition of warfarin provided additional benefit to intravenous cyclophosphamide. It should be noted that 3 patients (Patients 11, 13, and 14) were aPL-positive and received aspirin with corticosteroids (Patient 11) and intravenous cyclophosphamide (Patients 13 and 14) and also had good outcomes.

DISCUSSION

TM is an uncommon manifestation of lupus that occurs in 1–2% of patients[3]. Although usually considered a late complication of SLE, some studies suggest that this can occur as the presenting manifestation in up to 39% of SLE patients who develop TM. Of the small number of patients who develop TM, 42% do so within 5 years of diagnosis with SLE[4]. An important observation from our clinical series is that SLE was not an obvious diagnosis in the majority of these patients when they presented. Indeed, only 4 patients had the necessary classification criteria for SLE at presentation, although typical features did develop later in most of the other patients. An ad hoc committee of the ACR

has suggested that a patient with 3 of the ACR classification criteria and one of the 19 neuropsychiatric syndromes defined by the committee could be classified as having SLE[5]. Thus all our patients had a demyelinating syndrome according to this nomenclature and 12 fulfilled the ACR nomenclature for neuropsychiatric lupus[5]. At followup and using these classification criteria[4,5], 12 patients had SLE and 3 were classified with a lupus-like disease (Table 1). All were positive for antinuclear antibodies and/or anti-DNA antibody, emphasizing the usefulness of autoantibodies as a screening tool in patients with nonspecific symptoms and long tract neurological signs.

A discrete sensory level with lower limb weakness was the most common presenting feature. A thoracic sensory level was seen in the majority, similar to previous studies[4,6], followed by a cervical level. Several studies[7,8] have reported CSF pleocytosis, high protein concentrations, and low glucose levels, but in 6 of our patients CSF cell counts were normal. Only 2 patients had pleocytosis with high glucose and protein concentrations. Interestingly, oligoclonal bands were detected in all but 2 of the patients in whom investigation was performed.

MRI is the diagnostic tool of choice as it is noninvasive and sensitive in detecting spinal cord pathology compared to other imaging methods[9,10]. In addition it is useful in excluding other causes of myelopathy and assessing followup[11]. However, MRI may not be able to visualize abnormalities in all cases. Mok, *et al*[8] reported abnormal MRI signals in 56% of cases at the time of active myelitis, while Kovacs, *et al*[3] reported abnormalities in 70%. Kovacs, *et al* suggested that patients with abnormal MRI had more unfavorable outcomes than patients with normal MRI. All their patients who underwent an MRI scan at presentation had abnormal high signal lesions within the spinal cord, but this did not seem to be related to their outcome[4]. Indeed, one of our patients (Patient 7) had a severe paraparesis, but the MRI failed to show any intrathecal abnormality.

Lavalle, *et al* found a strong association between TM in SLE and the presence of aPL[12]. Indeed, as early as 1910, spinal cord thrombosis was observed in a patient with "acute myelitis"[13]. Recently, Sherer, *et al* described 4 patients out of a cohort of 100 aPL-positive patients who had TM[14]. We found a similar strong association with aPL, with 11 of 15 patients positive (73%). In 2 patients, however, these autoantibodies did not become detectable until long after presentation. Although other studies have differed on the relevance of these antibodies, considered with previous data our case series provides good supporting evidence for a role for aPL in the development of TM. It is difficult to be precise about whether the pathology within the cord is inflammatory, thrombotic, or a combination of the 2.

There is no consensus on the best treatment for this manifestation of SLE because of the rarity of TM. Although the true pathology of TM remains unclear, the rationale is that this is an autoimmune inflammatory manifestation of SLE that would benefit from corticosteroids and/or immunosuppression. Corticosteroids have been used alone and have led to significant recovery in some cases[15]. A recent study of 6 patients, however, suggested a rather poorer outcome, with improvement in only one patient despite intravenous methylprednisolone and cyclophosphamide[16]. The choice of immunosuppressive agent varies in the literature and depends upon the approach of the individual treating physician. Recent reviews have recommended the use of combined treatment with intravenous methylprednisolone and cyclophosphamide, and this appears to be more effective than methylprednisolone alone[6,17,18]. Our experience confirms this: all our patients who received immunosuppressive therapy had a good to fair functional outcome; the patient who died had recurrent episodes of TM leading to myelomalacia and had received prednisolone alone. In addition, 2 of our patients recovered taking azathioprine without having used cyclophosphamide, although Mok, *et al*[8] observed that cyclophosphamide appears to be better than azathioprine in terms of functional outcome. Another possible explanation for the relatively good outcomes in our patients is that TM was the presenting feature of their disease, which was diagnosed very early, and treatment commenced immediately — a point stressed by other studies[14,18].

The role of anticoagulation remains controversial. In those patients who have aPL, it would seem reasonable to consider anticoagulation in addition to immunosuppressive therapy on the basis that these autoantibodies are highly predictive of thrombosis. Certainly in our patients the combination of anticoagulation with immunosuppression was associated with good functional outcomes in the aPL-positive patients. One of our patients (Patient 8) continues taking warfarin alone, having had an excellent outcome. Whether anticoagulation should include aspirin as well as warfarin or indeed could be with aspirin alone remains unclear. Indeed, we noted 4 aPL-positive patients who received aspirin rather than warfarin in combination with other drugs who did well. An alternative approach might even be to consider low molecular weight heparin in the acute phase, followed by longterm aspirin, although only one of our patients was treated in this way — warfarin was avoided due to severe thrombocytopenia.

We describe a series of 15 patients presenting with TM as the initial manifestation of SLE or lupus-like disease. To our knowledge this is the largest such series to date. Early diagnosis and rapid commencement of immunosuppressive therapy may be superior to corticosteroids alone in improving functional outcome. The high prevalence of aPL in our patients, with good functional outcomes in those patients who were anticoagulated, suggests a potentially

pathogenic role for these antibodies in the development of TM. We would advocate considering longterm anticoagulation in addition to immunosuppressive therapy in these aPL-positive patients, although the role of aspirin remains to be clarified.

REFERENCES

1. West SG. Systemic lupus erythematosus and the nervous system. In: Wallace DJ, Hahn BH, editors. Dubois' lupus erythematosus. 6th ed. Philadelphia: Lippincott, Williams and Wilkins; 2002:693-738.
2. West SG. Neuropsychiatric lupus. Rheum Dis Clin North Am 1994;20:29-58.
3. Kovacs B, Lafferty TL, Brent LH, DeHoratius RJ. Transverse myelopathy in systemic lupus erythematosus: an analysis of 14 cases and review of the literature. Ann Rheum Dis 2000;59:120-4.
4. Hochberg MC. Updating the American College of Rheumatology revised criteria for the classification of systemic lupus erythematosus [letter]. Arthritis Rheum 1997;40:1725.
5. The American College of Rheumatology nomenclature and case definitions for neuropsychiatric lupus syndromes. Arthritis Rheum 1999;42:599-608.
6. Barile L, Lavalle C. Transverse myelitis in systemic lupus erythematosus. The effect of IV pulse methylprednisolone and cyclophosphamide. J Rheumatol 1992;19:370-2.
7. Andrianakos AA, Duffy J, Suzuki M, Sharp JJ. Transverse myelopathy in systemic lupus erythematosus: report of three cases and review of the literature. Ann Intern Med 1975;83:616-24.
8. Mok CC, Lau CS, Chan EYT, Wong RWS. Acute transverse myelopathy in systemic lupus erythematosus: clinical presentation, treatment, and outcome. J Rheumatol 1998;25:467-73.
9. Larsson EM, Holtas S, Cronqvist S. Emergency magnetic resonance examination of patients with spinal cord symptoms. Acta Radiol 1988;29:69-75.
10. Merine D, Wang H, Kumar AJ, Zinreich SJ, Rosenbaum AE. CT myelopathy and MRI imaging of acute transverse myelitis. J Comput Tomogr 1987;11:606-8.
11. Boumpas DT, Patronas NJ, Dalakas MC, Hakim CA, Klippel JH, Balow JE. Acute transverse myelitis in systemic lupus erythematosus: magnetic resonance imaging and review of the literature. J Rheumatol 1990;17:89-92.
12. Lavalle C, Pizarro S, Drenkard C, Sanchez-Guerrero J, Alarcon-Segovia D. Transverse myelitis: a manifestation of systemic lupus erythematosus strongly associated with antiphospholipid antibodies. J Rheumatol 1990;17:34-7.
13. Bastian HC. Thrombotic softening of the spinal cord. A case of so-called "acute myelitis." Lancet 1910;ii:1531-4.
14. Sherer Y, Hassin S, Shoenfeld Y, et al. Transverse myelitis in patients with antiphospholipid antibodies — the importance of early diagnosis and treatment. Clin Rheumatol 2002;21:207-10.
15. Propper DJ, Bucknall RC. Acute transverse myelopathy complicating systemic lupus erythematosus. Ann Rheum Dis 1989;48:512-5.
16. Tellez-Zenteno JF, Remes-Troche JM, Negrete-Pulido RO, Davila-Maldonado L. Longitudinal myelitis associated with systemic lupus erythematosus: clinical features and magnetic resonance imaging of six cases. Lupus 2001;10:851-6.
17. Lopez Dupla M, Khamashta MA, Sanchez AD, Ingles FP, Uriol PL, Aguado AG. Tranverse myelitis as a first manifestation of systemic lupus erythematosus: a case report. Lupus 1995;4:239-42.
18. Chan KF, Boey ML. Transverse myelopathy in SLE: clinical features and functional outcomes. Lupus 1996;5:294-9.