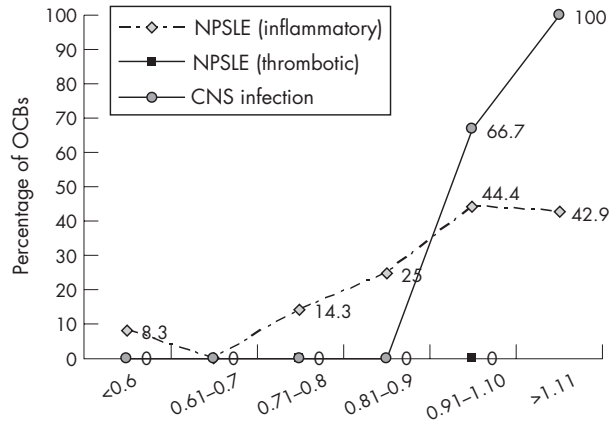

Figure 1 Detection rate of cerebrospinal fluid oligoclonal bands (OCBs) in different ranges of IgG index in inflammatory and non-inflammatory neurological conditions in patients with systemic lupus erythemaosus. CNS, central nervous system; NPSLE, neuropsychiatric systemic lupus erythematosus.

SLE (NPSLE) according to the American College of Rheumatology criteria[5] and other non-NPSLE conditions. A total of 57 results from 54 patients were eligible for the analysis.

The 63 neurological presentations among the 57 neurological episodes were classified as inflammatory (n = 49) and non-inflammatory (n = 13) according to the underlying pathogenesis determined by clinical features, MRI findings and response to immunosuppressive treatment. Table 1 shows the distribution of CSF OCBs, positive IgG index (>0.6) and the discrepancy between the two methods among these conditions. CSF OCBs were more frequently detected in inflammatory (26.5%) than in non-inflammatory conditions, including focal NPSLE and non-SLE-related conditions (0%; p = 0.05), but not for positive IgG index (p = 1.0). The different rates of detection of CSF OCBs in NPSLE reflected heterogeneity in the underlying pathogenesis where microthrombi or vasculitis may cause damage to the blood–brain barrier.[6–8] Although serum autoantibodies that cross react with antigens present in brain tissue have been shown to cause neuronal death through a damaged blood–brain barrier in the mouse model,[9] our study suggested intrathecal synthesis of autoantibodies as another possible pathogenetic mechanism.

There was agreement between IEF and IgG index on the presence (17.5%, n = 10) and absence (31.6%, n = 18) of intrathecal IgG synthesis, giving a discrepancy rate of 50.9% (29/56). This discrepancy was found to correlate with the presence of serum hypogammaglobulinaemia or hypergammaglobulinaemia (r = 0.86, p = 0.004), which was present in 23 (40.3%) samples. This suggested a non-linear relationship between the IgG ratio and the albumin ratio in the formula for IgG in these ranges of IgG.[10] IEF is thus superior to the IgG index for detection in patients with SLE.

CSF OCBs were found to correlate with the IgG index in inflammatory neurological conditions with higher sensitivity using a higher cut-off of the IgG index (≥0.81; fig 1). The presence of CSF OCBs irrespective of the IgG index may suggest a specific immune response, whereas quantitatively increased IgG without OCBs might indicate a non-specific polyclonal response. Studies on the specificities of CSF OCBs may provide clues to the underlying pathogenesis.

In conclusion, our study showed that intrathecal production of IgG was found in inflammatory NPSLE. IEF is the preferred method of detection. Other investigation results should be considered for diagnosis because of its lack of discriminative power for NPSLE and CNS infections.

Authors' affiliations

Mo Yin Mok, Woon Sing Wong, Chak Sing Lau, Department of Medicine, Queen Mary Hospital, Hong Kong
Eric Y T Chan, Department of Pathology, Queen Mary Hospital, Hong Kong

Competing interests: None.

Correspondence to: Dr M Y Mok, Department of Medicine, Queen Mary Hospital, Pokfulam Road, Hong Kong; mymok@netvigator.com

Accepted 9 October 2006

REFERENCES

1 **West SG**. Neuropsychiatric lupus. *Rheum Dis Clin North Am* 1994;**20**:129–58.
2 **Link H**, Tibbling G. Principles of albumin and IgG analysis in neurological disorders. The evaluation of IgG synthesis within CNS in multiple sclerosis. *Scand J Clin Lab Invest* 1977;**37**:385–401.
3 **Correale J**. Oligoclonal bands and antibody responses in multiple sclerosis. *J Neurol* 2002;**249**:375–89.
4 **Tan EM**, Cohen AS, Fries JF, Masi A T, McShane D J, Rothfield N F, *et al*. The 1982 revised criteria for the classification of systemic lupus erythematosus. *Arthritis Rheum* 1982;**25**:1271–7.
5 **ACR Ad hoc Committee on Neuropsychiatric Lupus Nomenclature**. The American College of Rheumatology Nomenclature and case definitions for neuropsychiatric lupus syndromes. *Arthritis Rheum* 1999;**42**:599–608.
6 **Kerr DA**, Ayetey H. Immunopathogenesis of acute transverse myelitis. *Curr Opin Neurol* 2002;**15**:339–47.
7 **Lampropoulos CE**, Hughes GR. The antiphospholipid (Hughes') syndrome: changing the face of neurology. *Eur J Intern Med* 2004;**15**:147–50.
8 **Abbott NJ**, Mendonca LL, Dolman DE. The blood-brain barrier in systemic lupus erythematosus. *Lupus* 2003;**12**:908–15.
9 **Kowal C**, DeGiorgio LA, Nakaoka T, Hetherington H, Herta PT, Diamond B, *et al*. Cognition and immunity; antibody impairs memory. *Immunity* 2004;**21**:179–88.
10 **Mclean BN**, Luxton RW, Thompson EJ. A study of immunoglobulin G in the cerebrospinal fluid of 1007 patients with suspected neurological disease using isoelectric focusing and the log IgG-index. *Brain* 1990;**113**:1269–89.

FORTHCOMING EVENTS

Future EULAR congresses

13–16 June 2007; EULAR 2007; Barcelona, Spain
11–14 June 2008; EULAR 2008; Paris, France

VI Meeting of the European Forum on Antiphospholipid Antibodies

12–13 October 2007, Ljubljana, Slovenia
Informal meeting of the clinicians, scientists that are active in the field of antiphospholipid antibodies and antiphospholipid syndrome
Vodnikova 62, 1000 Ljubljana, Slovenia
Tel: +386 1 5225533
Fax: +386 1 5225598
Email: info.apl2007@revma.net
Web: http://www.revma.net

CORRECTION

doi: 10.1136/ard.2006.058776.corr1

An error occurred in the February 2007 issue of the journal (Iannone F, Trotta F, Monteccuco C, Giacomelli R, Galeazzi M, Matucci-Cerinic M, *et al*. Etanercept maintains the clinical benefit achieved by infliximab in patients with rheumatoid arthritis who discontinued infliximab because of side effects. *Ann Rheum Dis* 2007;66:249–52.) The correct spelling of the third author's name is Montecucco C.