Acute abdominal pain in systemic lupus erythematosus: factors contributing to recurrence of lupus enteritis

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Lupus enteritis is a term applied to the alimentary tract lesions in systemic lupus erythematosus (SLE) and may affect any part of the gastrointestinal tract. Previously, we reported clinical findings in patients with SLE who had acute abdominal pain, focusing on lupus enteritis.¹ During follow-up, several patients with lupus enteritis experienced recurrence after complete remission with high-dose corticosteroids. To find the factors contributing to recurrence of lupus enteritis, we investigated 16 patients who were diagnosed for lupus enteritis between January 1993 and January 2003.

At first admission, we retrospectively reviewed patients' demographic data, laboratory data, including autoantibody profiles (anti-dsDNA, anti-Sm antibody, anti-ribonucleoprotein antibody, anti-Ro antibody, anti-La antibody and antiphospholipid antibody), abdominal computed tomography scans and SLE disease activity index (SLEDAI; table 1). Lupus enteritis was diagnosed clinically and from abdominal

computed tomography findings: bowel thickening ≥ 3 mm, target sign, dilatation of intestine, engorgement of mesenteric vessels and increased attenuation of mesenteric fat (fig 1).^{2,3} Patients were treated with intravenous methylprednisolone (1 mg/kg/day) and switched to oral prednisolone (median 5 days), which was tapered slowly over a period of 10 months. Bowel wall thickness was measured by an experienced radiologist who had no information about the clinical outcome.

Of the 16 patients followed up for >30 months (median 67 months), 7 patients showed no evidence of recurrence whereas 9 had recurrent lupus enteritis. When we compared clinical characteristics in patients with non-recurrent and recurrent lupus enteritis there were no differences in demographic and laboratory indices, including autoantibody profiles and SLEDAI score, between the two groups. However, in patients with non-recurrent lupus enteritis, the cumulative dose of prednisolone ($p = 0.02$) and duration of

Table 1 Comparison of groups with non-recurrent and recurrent lupus enteritis at initial admission

	Non-recurrent group (n = 7)	Recurrent group (n = 9)	p Value
Age (years)*	32.1 (9.4)	30.0 (6.3)	0.75
Sex			
Male	1	1	0.70
Female	6	8	0.70
White cell count ($\times 10^9$)/l*	8.6 (4.7)	5.4 (0.9)	0.12
Haemoglobin (g/l)*	10.9 (2.6)	12.1 (1.4)	0.05
Platelets ($\times 10^9$)/l*	259.0 (88.7)	257.6 (103.3)	0.71
ESR (mm/1/h)*	52.1 (18.1)	41.3 (19.2)	0.34
CRP (mg/l)*	4.2 (4.2)	1.6 (3.1)	0.15
C3 (mg/l)*	56.2 (13.2)	48.0 (9.0)	0.24
C4 (mg/l)*	13.4 (5.1)	13.0 (6.5)	0.23
Anti-dsDNA (IU/ml)*	69.8 (65.2)	128.7 (156.2)	0.87
SLEDAI score*	7.6 (6.0)	7.9 (4.7)	0.75
PD cumulative dose (g)*	14.7 (6.0)	7.6 (2.4)	0.02
Duration of PD treatment (month)*	50.0 (32.8)	16.9 (7.4)	0.04
Thickness of bowel wall† (mm)*	6.1 (2.6)	9.0 (3.7)	0.08

C, complement; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; PD, prednisolone; SLEDAI, systemic lupus erythematosus disease activity index.

*Values are given as mean (SD).

†Measured at the most thickened site in the affected bowel on abdominal computed tomography scan.

Figure 1 Computed tomography scans of patients with non-recurrent (A) and recurrent (B) lupus enteritis. Both scans show ascites, mesenteric congestion and diffuse oedematous change in nearly whole halo viscous with engorged mesenteric vessels. The arrow indicates the thickest wall segment of the affected bowel.

treatment with prednisolone ($p=0.04$) was significantly higher than in patients with recurrent lupus enteritis.

The wall thickness of the most thickened bowel in patients with recurrent lupus enteritis (mean (SD); 9.0 (3.7) mm) was greater than in patients with non-recurrent lupus enteritis (6.1 (2.6) mm), although the difference was not significant ($p=0.08$). All the five patients with bowel wall thickness ≥ 9 mm experienced recurrence. This finding may be considered a high-risk factor for recurrent lupus enteritis.

Cumulative dose and duration of treatment may be influenced by other complications of SLE, including lupus nephritis. In our study, class II lupus nephritis was newly diagnosed during prednisolone tapering in three patients (2/7 in non-recurrent, 1/9 in recurrent group), prolonging the duration of treatment with prednisolone. Despite this confounding variable, the dose and duration of corticosteroids was related more closely to the recurrence of lupus enteritis than laboratory indices, including autoantibody profiles and SLEDAI score reflecting lupus activity.

Successful treatment with high-dose prednisolone and intravenous pulse cyclophosphamide has been reported in many patients with intestinal vasculitis, and immunosuppressants such as cyclophosphamide may be recommended for patients with severe or steroid-resistant lupus enteritis.^{4,5} In conclusion, longer treatment with corticosteroids or treatment with additional immunosuppressants may be considered in patients with lupus enteritis and with initial bowel wall thickening >9 mm.

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Lack of association between signaling lymphocytic activation molecule family member 1 gene and rheumatoid arthritis in the French and Tunisian populations

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Signaling lymphocytic activation molecule family member 1 (*SLAMF1*) is a member of the CD2 immunoglobulin superfamily, which is expressed in T cells, B cells, macrophages and dendritic cells.¹ *SLAMF1* is associated with B cell proliferation and immunoglobulin synthesis.² The expression of *SLAMF1* in peripheral blood B cells from patients with rheumatoid arthritis was raised compared with healthy controls,³ and high expression of this molecule was observed in synovial fluid and tissue of patients with rheumatoid arthritis. Moreover, mice deficient in the *SH2D1A* molecule, which binds to the *SLAMF1* receptor, were susceptible to experimental autoimmune diseases.^{4,5} Considering these biological data and the potential role of the *SLAMF1* gene in regulating the immune system, we investigated the candidate gene *SLAMF1* polymorphism, located on chromosome 1 (1q21.3–32.3), in patients with rheumatoid arthritis.

Three *SLAMF1* single-nucleotide polymorphisms were studied: C>A (rs 2295612) located in exon 1, G>C (rs 1809963) located in intron 4 and A>C (rs 3796504) located in exon 7. Firstly, 100 French patients with rheumatoid arthritis (87 women and 13 men; mean age: 32 years) in both parents were studied. Secondly, 103 Tunisian patients (85 women and 18 men; mean age: 50.7 years) and 111 healthy controls (64 women and 47 men; mean age: 54.8 years) were analysed in a case–control study. All patients selected fulfilled the 1987

American College of Rheumatology criteria for rheumatoid arthritis.⁶

The polymerase chain reaction-restriction fragment length polymorphism technique was used for genotyping all samples (*DdeI* for rs2295612; *BslI* for rs1809963; *Hpy188II* for rs3796504; primer sequences and amplification programmes given on request).

Statistical analysis was examined by three different methods: transmission disequilibrium test, genotype relative risk and affected family-based controls. Results from Tunisian control subjects and patients with rheumatoid arthritis were compared using the χ^2 test and Fisher's exact test. *SLAMF1* haplotypes in the French Caucasian trio and Tunisian population were inferred using Genhunter and Phase 2.1 softwares, respectively.

Allele frequencies for the three single-nucleotide polymorphisms were in accordance with Hardy–Weinberg equilibrium in both Tunisian controls and French family-based controls. Statistical analysis did not show any linkage disequilibrium between these markers in the Tunisian as well as in the French population ($p>0.05$). Transmission disequilibrium test analysis showed that rs2295612, rs1809963 and rs3796504 are not preferentially transmitted from heterozygotic parents to affected offsprings in the French trios with rheumatoid arthritis. Similar results were