

A case of systemic lupus erythematosus (SLE) and myasthenia gravis (MG)

THE COEXISTENCE of systemic lupus erythematosus (SLE) and myasthenia gravis (MG) is well recognized (1). Both must be considered immune diseases, although rarely presenting in acute form at the same time (2). MG usually reveals itself before SLE, whose onset, on the contrary, is rather late and often follows thymectomy. In fact, the clinical features of MG usually appear during the periods of quiescent SLE obtained after steroidal treatment. We report the case of a 53-year-old

Are SLE and MG characterized by different antibody populations?

housewife admitted to our hospital for rhinolalia, dyspnea, dysphagia, bilateral ptosis, diplopia, and weakness of the lower limbs. At the age of 35 years, she had suffered from arthritis of the right ankle, and acute articular rheumatism was diagnosed and controlled by penicillin treatment. When she was 44 years old, an arthritic symptomatology involving wrists and fingers appeared, and a diagnosis of rheumatoid arthritis was made; consequently, gold salts, NSAIDs, antimalarials, and steroids were used with moderate benefit.

At admission to our department, pertinent laboratory data were as follows. ESR = 74 mm/1st h; RCP +++; antinuclear (ANA), anti-dsDNA, AMA, SMA, and PCA antibodies were negative; serum antiacetylcholine receptor antibodies (AChRab) = 0.8 pmol/ml (n.r. = <0.8 pmol/ml); and C₃ was increased. The Tensilon test was positive. Mediastinal tomography revealed no thymic enlargement. Visual evoked potentials (VEP) and electromyographic patterns manifested marked signs of myasthenic compromise of the neuromuscular transmission.

In July 1992, the patient was discharged with the diagnosis of MG (group II B according to Osserman), under treatment with pyridostigmine bromide (60 mg five times PO daily) and prednisone (50 mg PO daily), and myasthenic symptomatology improved immediately. Five months after discharge, intense pain involving the left shoulder and the spine appeared, requiring a new hospitalization. On this occasion, at the level of some ribs and vertebral bodies, signs of benign fractures, related to marked

osteoporosis, appeared, and we reduced the steroidal therapy progressively until its withdrawal. Twenty days after steroidal suspension, tachycardia and an evident myasthenic symptomatology suddenly occurred. Simultaneously, and for the first time, butterfly erythema (dorsum of the nose and cheeks bilaterally) was evident. Antibody tests performed some days later were positive for ANA and anti-dsDNA. Treatment with prednisone (75 mg PO daily) was immediately reinstituted with notable improvement.

The case reported is particularly interesting for both its clinical and etiopathogenetic features. It was that of a 53-year-old woman with a disease that had appeared many years before, characterized by arthritic poussées first diagnosed as acute articular rheumatism and later as rheumatoid arthritis since the manifestations of the underlying disease were absent. Subsequently, the sudden onset of the myasthenic symptomatology led to the diagnosis of MG. However, much later and only after suspension of steroidal therapy were clinical signs and laboratory data analyzed and the diagnosis of SLE possible. In the presence of a clearly myasthenic pathology, we first point out how difficult and sometimes prolonged is the process of diagnosing SLE. The lack of evidence of symptoms and signs does not often allow one to identify lupoid disease. At other times, patients may present typical osteoarticular and muscular symptoms and are believed to be affected by osteoarthropolyalgias of a different nature. In fact, in our case, only the sudden manifestation of tachycardia, usually considered the onset symptom of SLE, associated with butterfly erythema, and the presence of antinuclear and anti-dsDNA antibodies allowed us to clarify the picture of the disease, which progressively improved as soon as the high-dosage steroidal treatment was resumed. Furthermore, we would like to emphasize that older patients often present with a long history of clearly polyarthromyalgic symptoms for which they undergo steroidal treatment. Therefore, such features create the situation which may contribute to slowing the onset and disguising the symptoms and signs of SLE.

Moreover, the coexistence of these two immunologic entities has convinced various authors to continue attempts to demonstrate the presence of a possible cross-reaction between anti-DNA antibodies and antibodies against acetylcholine receptors (3). Although this possibility has been demonstrated in the serum of SLE patients for phospholipid antigens and other components (4), it has not yet been demonstrated for AChR (1). In conclusion and in agreement with other authors (5), we believe that the coexistence of SLE and MG supports the hypothesis of two different antibody populations.

Within the spectrum of autoimmune disease, such populations should be considered the opposite extremes, modulated by the thymus, as far as immune response is concerned, on the basis of a prevalence of one or the other pathologic form, both strictly related to the intake or withdrawal of steroids.

Key words: myasthenia gravis; systemic lupus erythematosus.

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Accepted for publication 28 March 1996

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Contact urticaria from stock, a Cruciferae plant

Stock (*Matthiola incana*) is an ornamental plant of the Cruciferae family, which also includes cabbage, cauliflower, Brussels sprouts, turnips, radishes, red cabbage, caper, rape, and mustard.

■ A 28-year-old male student suffered from seasonal rhinoconjunctivitis and oral allergy syndrome with facial edema after eating the seeds of broad beans. He had a 5-year history of contact urticaria (CU) 15 min after handling dry sheath with seeds of stock for sowing while gardening. The urticaria resolved completely 30 min after contact ceased. He ate vegetables of the Cruciferae family and legumes without problems. He had not eaten mustard in the last few years but had previously tolerated it.

Skin prick tests (SPT) were performed with the

usual inhalant antigens, commercial extracts of legumes (Aristegui), mustard 10% w/v (Bencard), our own extracts of fresh legumes, fresh Cruciferae vegetables, and dry stock sheaths (SS). The extracts of fresh vegetables were obtained by liquefaction with a domestic blender. After centrifugation, the fluid was put through a Millipore filter. The dry SS was triturated with a coffee grinder, and 2 g of it was homogenized with 20 ml of PBS. After filtration and centrifugation, the fluid was filtered through a Millipore filter (1/10 w/v concentration). Histamine and saline serum were used as controls. The SPT were considered positive if the largest diameter of the wheal was equal to or larger than 3 mm. The results of SPT are shown in Table 1. SPT with the previous extracts were negative in 20 atopic controls, 10 of them with positive SPT to pollens.

The rub test was carried out by delicately rubbing the forearm for 30 s with dry SS, and it showed

Although he was RAST-negative to stock, an IgE-mediated mechanism is likely for the urticaria in this atopic patient.

Table 1 Results of skin prick tests and specific IgE

Allergens	Prick (mm)	Specific IgE in kU/l (class)
Histamine	5	
Inhalants		
<i>Lolium perenne</i>	5.5	26.50 (4)
<i>Olea europaea</i>	9	7.15 (3)
<i>Artemisia vulgaris</i>	6.5	2.61 (2)
<i>Chenopodium album</i>	5.5	2.15 (2)
<i>Salsola kali</i>	4	1.47 (2)
Mites, molds, and danders	0	Not done
Legumes		
French bean	4.5	4.7 (3)
Broad bean sheath	4	Not done
Broad bean seed	4.5	Not done
Hanicot bean	0	< 0.35 (0)
Chickpea	0	< 0.35 (0)
Lentil	0	0.55 (1)
Peanut	0	4.52 (3)
Cruciferae vegetables		
Stock sheath	6	0 (0)
Mustard	8.5	0.95 (2)
Cabbage	7.5	1.19 (2)
Cauliflower	6.5	2.80 (2)
Brussels sprout	4	0.90 (2)
Turnip	4	Not done
Red cabbage	6	Not done
Radish	0	Not done

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