mal limit. HCV antibody was positive by a third generation ELISA. Muscle biopsy demonstrated cellular necrosis, regenerating fibers and inflammatory infiltrate. A lumbar puncture was normal. The patient was treated with antibiotics and corticosteroid. Because of her respiratory distress intubation and mechanical ventilation were required. A dialysis procedure was performed. She died on the fourth day of hospitalization.

Since its discovery in 1989, HCV is more than just a liver disease (7). Chronic HCV infection is associated with an array of autoimmune laboratory and clinical manifestations (2, 8). HCVmay represent a chronic stimulus for the immune system and/or direct invasion and replication of the virus in extrahepatic tissues (1).

The most useful enzymes in diagnosis and prognosis of PM/DM are CK and aldolase. The AST, ALT, and LDH enzymes may appear in increased amounts as well. These 3 enzymes share a site of origin in both muscle and liver. Our patient had HCV infection for years prior to the emergence of PM. She presented with clinical and histologic features of PM, no rash involvement, and raised CK levels which were related with acute renal failure. Further controlled studies are justified to determine an association between HCV infection and PM, thus eventually leading to a better understanding of mutual relationship between virus and myositis.

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A new case of dermatomyositis following the rupture of a silicone gel breast implant

Sirs,

We read the interesting report of two cases of dermatomyositis which occurred in patients who had undergone silicone gel filled breast implant for cosmetic reason (1); in the first case the disease was diagnosed 6 months after breast augmentation and in the second one after 8 years; in the latter one chest HRCT demonstrated retraction of the prosthesis. HLA typing showed identity in one of two aplotypes of class II antigens: HLA-DRB1* [0301], HLA-DQB1* [0201]. The first patient strongly ameliorated after prosthesis removal, but required chronic immunosuppressive therapy to control the disease; the second patient refused the explantation of breast prosthesis.

Recently we have observed a case of dermatomyositis in a 57-year-old woman who first underwent silicone gel augmentation mammoplasty for cosmetic reason in 1970; in November 2003 the mammary prosthesis were substituted for silicone spreading following capsular rupture. Six months later she complained of painful proximal muscle weakness and periorbitary edema. CPK level was 8026 U/L; antinuclear antibodies were positive at dilution 1:1280 with speckled pattern; anti-ENA antibodies tested by ELISAwere negative. The extensive search of an occult tumour resulted fruitless. Muscle biopsy showed some atrophic cells, necrosis and inflammatory infiltrate. HLA typing showed HLA-A [24, 34], HLA-B [8(Bw6), 18(Bw6)], HLA-Cw [07, 12]; HLA-DRB1*[0301], HLA-DQB1* [0201]. The patient was treated with immunoglobulin (0.4 g/Kg/day for 5 consecutive days), prednisone at the initial dosage of 50 mg/day and azathioprine 100 mg/day with good result; the dosage of prednisone was then reduced to 25 mg/day.

During the last years the relationship between silicone prosthesis implant and induction of connective tissue diseases has been largely investigated with an initial enthusiasm; obviously the discovery of an etiologic factor or of a trigger involved could allow to increase our knowledge in this complex and intriguing context and could favour the comprehension of the pathogenetic mechanisms which lead to the damage in some rheumatic diseases whose etiopathogenesis remains still obscure. Nevertheless epidemiological studies have so far not shown an increased incidence of connective tissue diseases in women with silicone breast implants (2-7), even in case of prosthesis rupture (8); instead conflicting data have been collected about a possible relationship between silicone gel breast implant rupture and fibromyalgia (9, 10) as well as silicone breast implant and symptoms evocative of rheumatic disorders (11). Moreover, an increased frequency of low titre positive ANA, but not of high titre ANA in patients with silicone breast implants compared to controls has been observed (6).

On the grounds of the results of the abovementioned epidemiological studies silicone is still largely used in cosmetic surgery; nevertheless these reports do not allow to exclude the possibility that in susceptible individuals the stimulation of the immune system by silicone may induce the appearance of a connective tissue disease; silicone gel may escape from ruptured implants, became extracapsular thus representing an adjuvant for the development of an autoimmune disease. Our patient presented homozygosis for the same aplotypes of class II HLA antigens (DR and DQ) as the two patients previously described (1), supporting the hypothesis of a predisposing genetic background. In 1997 a report about two HLA-identical sisters bearing rheumatoid arthritis susceptibility genes that developed polyarthritis after silicone breast implants has been published; after removal of the mammary prosthesis one patient achieved a complete remission and the other one a significant improvement (12). The genetic background might condition the type of autoimmune disease that starts after silicone exposure.

Few other cases of dermatomyositis after insertion of silicone breast implants have been described (5, 13); among these we cite a case of dermatomyositis-like disease in which silicone particles within phlogistic areas have been observed in a specimen of the inflamed upper eyelid (14).

We have hypothesized that in our case the dermatomyositis was more probably provoked by the old ruptured breast implants than by the second insertion. At the moment however we are not able to propose its explantation.

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Reply

Sirs

The debate over the potential harmful effects of silicone from breast implants has persisted for more than a decade (1). The case reported by Caramashi *et al*. (2) provides further evidence regarding a potential relationship between silicone spread from breast implants and dermatomyositis. Interestingly, one of two class II haplotypes in the case reported by the authors was identical to the two patients we have reported earlier (3). In our opinion, these findings support the hypothesis of a predisposing genetic background. It is well recognized that several HLA alleles are related to autoimmune diseases. Dermatomyositis and anti-synthetase antibodies have been associated with HLA-DRB1 and DQA1 alleles (4), as was the case in our patients. Class II HLAis involved in the process of antigen presentation to CD4 T cells in the humoral immune response and silicone may act as a hapten-like substance and combine with other molecules to form an antigenic complex. It would be of interest to know if the patient reported by Caramashi *et al.* was positive for anti-synthetase antibodies.

Although epidemiological studies have failed to demonstrate a clear association between connective tissue disorders and silicone breast implants, clinicians have sometimes felt that in specific cases, silicone may act as a factor for human adjuvant disease factor in patients with a predisposing genetic background. The case reported by Caramashi *et al*. lends support to this hypothesis. The scarce number of patients with dermatomyositis (prevalence of 3-4 cases per million inhabitants) could explain in part the lack of association in epidemiologic studies. Low- or medium-molecular-weight polymers have been known to migrate from the intact silicone elastomer shells into surrounding tissues in a process known as a "gel bleeding" (5). The assay of low- molecular-weight silicones in plasma and blood using chromatographic techniques is probably the best way to demonstrate the relationship between silicone and disease (6). Work is currently underway to determine these parameters in our patients.

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Coexistence of non-specific and usual interstitial pneumonia in a patient with severe cystic scleroderma lung involvement and parvovirus B19 infection

Sirs,

Scleroderma lung disease (SLD) is one of the most frequent clinical manifestations of systemic sclerosis (SSc) (1-3). Recently, we observed a very severe SLD characterised by the coexistence of nonspecific interstitial pneumonia (NSIP) and usual interstitial pneumonia (UIP), and diffuse parenchymal cyst formation.

A 25-year-old woman was referred to our Rheumatology Unit in March 2003 because of 2-year history of Raynaud's phenomenon and recent onset ulceration of the second finger of the left hand. The diagnosis of SSc was made on the basis of the physical examination (acrocyanosis, swollen hands, with digital pitting scars and mild sclerodactyly, melanoderma, and microstomia), 'active' scleroderma pattern on capillaroscopy, presence of anti-centromere antibodies (titre 1: 160), and mild bibasilar interstitial involvement on standard chest x-ray (Fig. 1a). Surprisingly, high resolution computed tomography (HRCT) showed the presence of multiple, disseminated cysts in lung parenchyma (Fig. 1c), reminiscent of lymphoangioleiomyomatosis (LAM) (4). Spirometry revealed a restrictive pattern (forced vital capacity 54% of predicted) with marked reduction of lung diffusion capacity (23% of predicted).

One month later the patient developed exertion dispnoea. Chest examination revealed crackles (velcro rales) over the bases in both lungs. Bronchoalveolar lavage (BAL) showed an active alveolitis (eosinophil 29%, n.v. 0%; lymphocyte 14%, n.v. 6.8 ± 1.3), while desmin and HMB45 immunoreactive cells were absent.

In order to exclude LAM, two lung biopsies (lingula and inferior lobe) were carried out