

RETRACTION

Stricker RB, Abrams DI, Corash L, Shuman MA. Target platelet antigen in homosexual men with immune thrombocytopenia. N Engl J Med 1985; 313:1375-80.

To the Editor: Data suggesting that people with AIDS or AIDS-related conditions may have an antibody against a 25-kd platelet protein that is not present in those without this diagnosis were reported by our laboratories (Stricker RB, Abrams DI, Corash L, Shuman MA. Target platelet antigen in homosexual men with immune thrombocytopenia. N Engl J Med 1985;313:1375-80). During the course of an investigation conducted by UCSF into the data for this paper, after questions were raised about other work by R.B. Stricker, it was discovered that some of the Western blots performed by Dr. Stricker with "control" serum (i.e., serum from subjects without AIDS or AIDS-related conditions) had also been positive for a band at approximately 25 kd. Dr. Stricker did not provide this information to his colleagues, and thus the cited paper suggested that the 25-kd platelet band was specific for HIV-infected patients.

The fact that the band could also be found in the serum of patients without AIDS or AIDS-related conditions shows that it is not specific for HIV infection, and the hypothesis that the antibody may partially

account for immune thrombocytopenia in homosexual men is invalid. This interpretation is further supported by our finding that with the same technique of Western immunoblotting as that reported in the paper, normal serum samples from three volunteers also gave a band at 25 kd. In addition, reports by other groups have failed to find a specific antibody against the 25-kd protein in patients with HIV infection and immune thrombocytopenia.¹⁻³ We therefore wish to retract the paper.

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REVIEW ARTICLE

MEDICAL PROGRESS

POLYMYOSITIS, DERMATOMYOSITIS, AND INCLUSION-BODY MYOSITIS

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THE polymyositis and dermatomyositis complex encompasses a heterogeneous group of acquired muscle diseases called inflammatory myopathies because muscle weakness and inflammatory infiltrates within the skeletal muscles are the principal clinical and histologic findings.¹⁻¹² Traditionally, polymyositis and dermatomyositis have been viewed as pathogenetically similar and part of the spectrum of "idiopathic" inflammatory myopathies,^{9,10,13,14} despite the variability in their clinical and laboratory characteristics, prognosis, and response to therapy. With the evolu-

tion over the past 10 years of rather well defined clinical, demographic, histologic, and immunopathological criteria and the identification of inclusion-body myositis as a distinct type of polymyositis,¹⁵ the inflammatory myopathies now can be viewed as comprising three major and discrete groups: polymyositis, dermatomyositis, and inclusion-body myositis.^{6,8,11,12} Each group retains its characteristic clinical, immunopathologic, and morphologic features regardless of whether it occurs separately or in connection with other systemic diseases.

This review, the first in the *Journal* since the one appearing 16 years ago,¹⁴ reflects current knowledge of the clinical manifestations, immunopathogenesis, diagnosis, and treatment of the inflammatory myopathies, emphasizing the views and developments of the past decade.

CLINICAL MANIFESTATIONS

The incidence of polymyositis, dermatomyositis, and inclusion-body myositis is approximately 1 in 100,000.^{3,16} Dermatomyositis affects both children and adults, and females more often than males, whereas polymyositis is seen after the second decade of life and very rarely in childhood. Inclusion-body myositis is

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