

1. Nikkari S, Mertsola J, Korvenranta H, Vainionpää R, Toivanen P: Wegener's granulomatosis and parvovirus B19 infection. *Arthritis Rheum* 37:1707-1708, 1994
2. Corman LC, Dolson DJ: Parvovirus B19 and necrotizing vasculitis (abstract). *Arthritis Rheum* 35 (suppl 9):S165, 1992
3. Schwarz TF, Roggendorf M, Deinhardt F: Human parvovirus B19: ELISA and immunoblot assays. *J Virol Methods* 20:155-168, 1988
4. Anderson LJ, Tsou C, Parker RA, Chorba TL, Wulff H, Tattersall P, Mortimer PP: Detection of antibodies and antigens of human parvovirus B19 by enzyme-linked immunosorbent assay. *J Clin Microbiol* 24:522-526, 1986

Reply

To the Editor:

Drs. Corman and Staud describe 2 patients, in addition to the 2 described previously (1,2), whose cases suggest an association between parvovirus B19 infection and WG. Since our initial case (2), we have studied sera from 42 adult patients with recent-onset WG (23 male, 19 female; ages 18-79 years). Primers specific for DNA sequences from 2 different segments of the B19 genome were used (2); primers that amplify positions 2060-2369 were used in the sera of all 42 patients and those that amplify positions 3365-3564 were used in the sera of 36 patients. The polymerase chain reaction (PCR) products were further studied by Southern hybridization using B19-specific oligonucleotide probes. Strict measures were taken to avoid false-positive results in the PCR assay, usually caused by amplicon contamination of the PCR reagents. Consecutive serum samples were available from 13 patients; from these samples, B19-specific circulating IgM and IgG antibodies were determined using an enzyme-linked immunosorbent assay with a recombinant antigen (Progen, Heidelberg, Germany).

Molecular or serologic evidence of recent B19 infection was not observed in any of these adult patients. An explanation for our negative findings might be the age distribution of the patients; the previously described WG patients who were found to be B19 positive by PCR were 12 and 16 years old (1,2). Yet, Drs. Corman and Staud's finding of 2 adult patients with serologic evidence of B19 infection and WG further complicates the final evaluation. However,

on the basis of our present findings, we prefer to conclude that no clear evidence exists for a B19 etiology of WG. It should also be mentioned that suggestions about the association of B19 infection with Kawasaki disease (3) or polyarteritis nodosa (1,4) have appeared inconclusive (5,6).

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1. Finkel TH, Török TJ, Ferguson PJ, Durigon EL, Zaki SR, Leung DYM, Harbeck RJ, Gelfand EW, Saulsbury FT, Hollister JR, Anderson LJ: Chronic parvovirus B19 infection and systemic necrotizing vasculitis: opportunistic infection or aetiological agent? *Lancet* 343:1255-1260, 1994
2. Nikkari S, Mertsola J, Korvenranta H, Vainionpää R, Toivanen P: Wegener's granulomatosis and parvovirus B19 infection. *Arthritis Rheum* 37:1707-1708, 1994
3. Nigro G, Zerbini M, Krzysztowiak GG, Porcaro MA, Mango T, Musiani M: Active or recent parvovirus B19 infection in children with Kawasaki disease. *Lancet* 343:1260-1261, 1994
4. Corman LC, Dolson DJ: Polyarteritis nodosa and parvovirus B19 infection. *Lancet* 339:491, 1992
5. Yoto Y, Kudoh T, Haseyama K, Suzuki N, Chiba S, Matsunaga Y: Human parvovirus B19 infection in Kawasaki disease. *Lancet* 344:58-59, 1994
6. Leruez-Ville M, Laugé A, Morinet F, Guillevin L, Dény P: Polyarteritis nodosa and parvovirus B19. *Lancet* 344:263-264, 1994

BOOK REVIEW

Reactive Amyloidosis and the Acute Phase Response. Baillière's Clinical Rheumatology. Vol. 8, No. 3. Edited by Gunnar Husby. London, Baillière Tindall, 1994. 200 pp. Illustrated. Indexed. \$40.00.

The acute-phase response and its role in the production of the reactive or secondary (AA) type of amyloidosis is an exceedingly complex topic. The association was first recognized in the early 1970s, and the mechanism appeared

simple: arthritis and other forms of inflammation produced interleukin-1, which caused an acute-phase response and an elevated level of serum amyloid A (SAA) protein, which in turn led to AA amyloidosis. Now the acute-phase response is known to be regulated by many cytokines, each with separately regulated gene expression, and SAA is known to be a whole family of proteins.

Research on the acute-phase response has become a fast-paced field, with the discovery of numerous cytokines

leading to a multitude of physiologic changes, one of which is AA amyloidosis. This 11-chapter multiauthored monograph brings together a rich assortment of topics related to reactive amyloidosis, presented by outstanding investigators in the US, Canada, and Europe. The first chapter, by the editor, defines the classification of amyloidosis, a topic for which he is internationally recognized. The book then proceeds to address, in an orderly manner, the acute-phase response, SAA protein, other constituents of connective tissue which may influence amyloid deposition, and clinical aspects of amyloidosis.

The first half of the book is outstanding! The chapter on the general aspects of the acute-phase response is a comprehensive review of inflammation as a complex, highly orchestrated process involving many cell types and molecules. It describes the acute-phase response and the role of cytokines as an integrated sum of multiple, separately regulated changes in gene expression. It contains an excellent list of up-to-date references, as is also found in most of the other chapters. The chapter on SAA as an apolipoprotein and precursor of reactive amyloidosis discusses its regulation and presents a complete review of SAA in all species. Another first-rate chapter is one that reviews the components that are universally present in amyloid substance but are not the integral elements of the fibrils: namely, P component, apolipoprotein E, basement membrane, proteoglycans, and glycosaminoglycans. Although some overlap in content is found in these chapters, they are presented from different perspectives and are generally complementary.

The last 4 chapters address clinical aspects of amyloidosis. A chapter on epidemiology has the best information available on the incidence of the AL (primary) type of amyloidosis and reveals how little information there is on this topic. Two other chapters provide incidence figures for AA amyloidosis in different countries and discuss the variations found due to disparate underlying diseases. They point out large differences, with a rather high prevalence of AA amyloidosis in western Europe and a lower prevalence in the US. A chapter on the diagnosis of amyloidosis is a disappointingly biased review which highlights the SAP scintigraphy test and undermines the "gold standard" of tissue biopsy. The value of SAP scintigraphy is as yet unconfirmed since its use is restricted to the investigators' laboratory.

This 200-page issue of *Baillière's Clinical Rheumatology* will be most valuable for its detailed review of the acute-phase response. Investigators in fields of inflammation will appreciate the comprehensive and well-referenced chapters on this topic. The chapters on the clinical aspects of reactive amyloidosis will be of great interest and value to clinicians who study amyloid disease, but of limited value to the practicing rheumatologist.

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