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LRA by ELISA/ACT®

CLINICAL PEARLS UPDATE#33

Arteritis

July 9, 2004

Dear Colleague,

Arteritis is due to immune complexes (type III hypersensitivity; IgM anti-IgG antigen complexes) invading the wall of large blood vessels. This may be a marker of cumulative repair deficit in distressed arteries. LRA by ELISA/ACT® tests and plans can identify an individual's immune reactive burden. When the items identified as reactive are substituted to the extent possible, improved outcomes are routinely observed. Inflammatory markers such as sed rate and CRP reduce to healthy levels concomitant with intensive repair. With your inspiration and encouragement to comply along with our nutrition and program counseling staff, consistently outstanding results can be achieved. The studies by Rizzi and colleagues as well as Gillot and colleagues support this hypothesis, as are our work combining comprehensive, *ex vivo* functional delayed allergy detection by lymphocyte response with specific Alkaline Way diet, targeted supplementation, and healing actions to reset the body to healthy tolerance, homeostasis, and sustained remission.

We encourage you to share this valuable clinical update newsletter with your colleagues and staff so they can learn more about how our comprehensive approach can be applied to their practice with beneficial results. Please also let us know if any of your colleagues or staff would like to be added to our email distribution list.

We are grateful for the opportunities to be of service to you and your patients.

Sincerely,

Russ Jaffe, MD, Ph.D., CCN, NACB
Lab Director

Rizzi R, Bruno S, Stellacci C, Dammacco R. Takayasu's arteritis: a cell-mediated large-vessel vasculitis. *Int J Clin Lab Res* 1999;29(1):8-13.

Department of Biomedical Sciences and Human Oncology, University of Bari Medical School, Italy.

Takayasu's arteritis is an idiopathic, systemic inflammatory disease, typically involving the aorta and its main branches. Cell-mediated autoimmunity has been strongly implicated in its pathogenesis. Early or active-stage pathology consists of continuous or patchy granulomatous inflammation, which progresses to intimal and adventitial fibrosis and scarring of the media. Multiple focal or segmental stenoses result and aneurysms may occasionally occur. Clinical presentation is heterogeneous, ranging from asymptomatic to catastrophic. In some patients, constitutional signs and symptoms indicating a systemic inflammatory response are observed, usually in the early stages. Specific features reflect arterial involvement, and result from end-organ or limb ischemia; they include vascular, neurological, cardiac, and pulmonary manifestations. The course of Takayasu's arteritis usually extends for many years with varying degrees of activity. Takayasu's arteritis has a worldwide distribution, with the greatest prevalence in eastern countries. Women of reproductive age are preferentially affected, but the illness is being recognized with increasing frequency in males. Variable phenotypes are recently emerging in different ethnic groups. Diagnosis is based on clinical features and vascular imaging studies that document typical patterns of stenoses or aneurysms of the aorta and its primary branches. Assessment of the activity of Takayasu's arteritis is imprecise, in that clinical features and acute-phase reactants do not accurately reflect active blood vessel inflammation. High-dose corticosteroids alone or a cytotoxic agent in addition to a corticosteroid may be effective in treating active disease. Critical lesions may require correction by surgery or interventional radiology.

Gillot JM, Masy E, Davril M, Hachulla E, Hatron PY, Devulder B, Dessaint JP. Elastase derived elastin peptides: putative autoimmune targets in giant cell arteritis. *J Rheumatol* 1997;24(4):677-682.

Service de Medecine Interne A, Hopital Claude Huriez, CHU Lille, France.

OBJECTIVE: Histological analysis of giant cell arteritis (GCA) reveals a granulomatous reaction around the internal elastic lamina. Elastolysis by multinucleated giant cells has also been reported. We investigated elastin derived peptides as putative recall antigens for peripheral blood mononuclear cells (PBMC) from patients with GCA. **METHODS:** PBMC were collected from 17 patients with GCA (Group 1), 17 patients with vascular diseases, connective tissue diseases, or polymyalgia rheumatica without GCA (Group 2), and 17 healthy controls (Group 3). Cultures of PBMC with different elastin derived peptides or elastase were analyzed. **RESULTS:** A proliferative response was obtained only with elastate derived elastin peptides in 12/13 untreated patients with GCA. Steroid treatment was believed to abolish this proliferative response in 4 patients with GCA. PBMC from only 3/34 non-GCA subjects responded to these antigens. No proliferative response was obtained for other elastin derived peptides or elastase in any subject. **CONCLUSION:** Degradation of native elastin by leukocyte elastase can provide elastin derived peptides that act as autoimmune targets for T cells in GCA.

Weyand CM. The Dunlop-Dottridge Lecture: The pathogenesis of giant cell arteritis. *J Rheumatol* 2000;27(2):517-522.

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