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LRA by ELISA/ACT®

CLINICAL PEARLS UPDATE#35

Vasculitis

July 23, 2004

Dear Colleague,

Autoimmune conditions often occur together. This is particularly true in the immune aspect of the human surveillance system where **vasculitis, Wegeners Granulomatosis, accelerated atherosclerosis, stroke, and thrombotic syndromes** often occur together. Repair deficits in distressed individuals may promote the autoimmunity. The cumulative repair deficit is clinically often described as an inflammatory syndrome. Inflammatory markers increase. Examples are **hsCRP, ferritin, fibrinogen, insulin and related growth factors, free cortisol, sed rate, PR3-ANCA tH1 cytokine profile, and prealbumin**. **LRA by ELISA/ACT® tests and plans** determine each individual's delayed allergic reactions or mystery hypersensitivities. Our LRA tests are **functional, ex vivo, and comprehensive assays**. **Substitution for reactive items is blended into an alkaline way repair diet**. **Targeted supplementation aims to correct antioxidant deficits and enhance detoxification ability**. **Healing actions engage the mind and body in an integrated direction**. **Go by results**. **Several retest cycles will be routinely needed in complex cases**. **The sequential improvement in health quotient speaks clearly about how effective are LRA by ELISA/ACT interpretation plans in integrating health fundamentals**.

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

Finley JC Jr, Bloom DC, Thiringer JK. Wegener granulomatosis presenting as an infiltrative retropharyngeal mass with syncope and hypoglossal paresis. *Arch Otolaryngol Head Neck Surg* 2004;130(3):361-365.

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We describe a woman who presented with syncopal episodes and unilateral hypoglossal paresis in association with a highly infiltrative retropharyngeal mass. After an extensive malignancy workup, the patient was found to have Wegener granulomatosis (WG), an autoimmune necrotizing vasculitis that presents with inflammatory lesions anywhere in the respiratory tract and variable renal involvement. The archetypal presentation in the head and neck is erosive sinonasal crusting, though otologic, pharyngeal, and laryngeal findings are common. Highly uncharacteristic lesions are occasionally encountered and may contribute to significant diagnostic dilemmas. Neurologic involvement is not uncommon, but few reports of hypoglossal paresis and no reports of syncope as a result of WG are found in a review of the literature. Given the variability of presentation of WG in the head and neck, the **otolaryngologist** must maintain a high degree of suspicion for this disease in the **evaluation of airway lesions**.

7: Lamprecht P, Gross WL. *Wegener's granulomatosis*. *Herz*. 2004;29(1):47-56.

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Wegener's granulomatosis is an organ- and/or life-threatening autoimmune disease of as yet unknown etiology. The classic clinical triad consists of necrotizing granulomatous inflammation of the upper and/or lower respiratory tract, necrotizing glomerulonephritis, and an autoimmune necrotizing systemic vasculitis affecting predominantly small vessels. The detection of antineutrophil cytoplasmic antibodies directed against proteinase 3 (PR3-ANCA) is highly specific for Wegener's granulomatosis. ANCA positivity is found only in about 50% of the patients with localized Wegener's granulomatosis (which is restricted to the respiratory tract and affects < or = 5% of the patients), whereas PR3-ANCA positivity is seen in 95% of the patients with generalized Wegener's granulomatosis. Studies showing an expansion of circulating tumor necrosis factor-(TNF-)alpha-producing Th1-type CD4(+)CD28(-) T-cell effector memory T-cells and their presence as Th1-type cytokine profile- driving cell population within granulomatous lesions provide the rationale for using TNF-alpha-blocking agents in Wegener's granulomatosis refractory to standard induction therapy with cyclophosphamide and corticosteroids ("Fauci's scheme"). **Vasculitis is an independent risk factor for diffuse endothelial dysfunction and may be a consequence of TNF-alpha action on endothelial cells.** Recently, another study has shown intima-media thickening of the wall of the common carotid artery and bulb, as well as a significantly increased incidence of stroke, myocardial infarction and occlusive artery

disease in Wegener's granulomatosis. This study suggests that systemic inflammation and vasculitis contribute to accelerated arteriosclerosis in Wegener's granulomatosis.