Rheumatology 2012, 2:3
http://dx.doi.org/10.4172/2161-1149.S1.010

OMICS Group Conferences
Accelerating Scientific Discovery

International Conference and Exhibition on
Orthopedics & Rheumatology

August 13-15, 2012 Hilton Chicago/Northbrook, USA

Implication of circulating natural antibodies against angiotensin-converting enzyme in the peripheral blood sera of patients with knee osteoarthritis: a marker of disease activity or regulator of inflammation and pain? Inflammation and Immunity

Yulia Savitskaya
National Rehabilitation Institute, Mexico

Purpose: Knee osteoarthritis (kOA) results, at least in part, from overloading and inflammation leading to cartilage degradation. Inflammatory mediators such as bradykinin, histamine, prostaglandins, lactic acid, substance P, and calcitonin generelated peptide are released into the joint. Innate immune system activation, best documented in responses to pathogens, likely plays a role in induction of inflammatory mediators in kOA. Angiotensin-converting enzyme (ACE) plays an important role in a number of inflammatory and immune related disorders. The aim of our work was to study the expression of natural antibodies against ACE (ACE-NA) in the peripheral blood sera of kOA patients and found a correlation between serum ACE-NA level and other markers.

Methods: Sera were obtained from 57 patients with primary kOA fulfilling the American College of Rheumatology criteria and 57 ethnically matched healthy controls. All kOA patients had involvement of the knee joint with typical radiographic changes graded Kellgren & Lawrence classification. The presence of ACE-NA was examined by a novel ELISA. Affinity chromatography yielded ACE-NA (revealed upon Ion-exchange Chromatography on QAE Sephadex) from both kOA patients and healthy individuals. Expression of cytokines was measured by Bio-Plex Human Cytokine Assay (Bio-Rad Inc, Hercules, CA, USA).

Results: ACE antibodies (IgM, IgG, IgA), reacting with ACE tested, were present in the sera of kOA patients as well as in the sera of normal individuals. Affinity chromatography yielded three (IgM, IgG, IgA) isotypes of ACE-specific NA from the both kOA patients and healthy individuals. Purified ACE-NA displayed the expected characteristics and was functionally fully active. No statistically significant differences were found between ACE-IgG and ACE-IgA for kOA patients and healthy individuals. The level of ACE-IgM in the sera from the kOA patients was significantly higher than those from the control group ($P<0.005$). ACE-IgM was expressed at higher levels in kOA than in other OA. No correlation was found between serum ACE-IgM level and patient's age and body mass index. There was a positive correlation between serum ACE-IgM level and expression of pain-associated molecules such as inducible nitric oxide synthase ($r=0.652$; $P<0.01$), IL-6 ($r=0.815$; $P<0.05$) and proinflammatory cytokine as such IL-1 ($r=0.789$; $P<0.01$).

Conclusions: We first identified ACE-NA in the sera of kOA patients. The ACE-IgM test gives significant information about kOA patients. Serum ACE-IgM is a good discriminator between kOA patients versus patients with other OA and healthy people. Serum ACE-IgM level may help to classify OA patients. We shown that their could be used a specific marker for diagnosis and prognosis of primary kOA. Renewed interest in ACE antibodies opens up a new area for kOA diagnostics and therapeutics.

yulia.savitskaya2012@gmail.com

Vascular complications in patients with systemic sclerosis (Scleroderma)

Julius Bankole
University of Northern British Columbia, Canada

Vascular disease is a common symptom in scleroderma patients, and it often leads to morbidity and mortality. Some patients develop severe and sometimes life-threatening vascular dysfunction. Vascular dysfunction represents a fundamental role in the pathogenesis of systemic sclerosis. Vascular abnormalities are characterized by injury to vascular wall and extensive damage of the microvessels. Though scleroderma pathogenesis is still unknown, there are suggestions that endothelial cell layer of the microvascular is activated/injured early in the disease process leading to endothelial dysfunction, over-expression of adhesion molecules, enhanced leukocytes, proliferation of pericytes, adhesion and activation of platelets and influx of a perivascular infiltrate. The possibility of identifying microvascular activities at the early stage will improve the treatment of vascular complications with time. There are also studies on pathophysiology of macrovascular dysfunction in patients with SSc, a presentation of both vascular involvements their processes will be highlighted. This paper will present review of potential biomarkers that serve as surrogates for the vascular disease process in patients with scleroderma, and also present biomarkers that could be used as a predictor of pulmonary hypertension. This presentation will provide key reviews of causes and clinical consequences of vascular disease in scleroderma.

bankole@unbc.ca