Researchers' Work from Kumamoto University Focuses on Inclusion Body Myositis (Pathomechanisms of Anti-Cytosolic 5 '-Nucleotidase 1A Autoantibodies in Sporadic Inclusion Body Myositis)

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Full Text:

2017 MAY 26 (NewsRx) -- By a News Reporter-Staff News Editor at Health & Medicine Week -- Data detailed on Musculoskeletal Diseases and Conditions - Inclusion Body Myositis have been presented. According to news reporting from Kumamoto, Japan, by NewsRx journalists, research stated, "Sporadic inclusion body myositis (sIBM), an intractable progressive muscle disease, frequently occurs in older persons. sIBM pathogenesis may involve protein degradation dysfunction and immune abnormalities. Autoantibodies recognizing cytosolic 5'-nucleotidase 1A (cN1A) were found in plasma and serum from sIBM patients."

The news correspondents obtained a quote from the research from Kumamoto University, "However, whether anti-cN1A autoantibodies play a pathogenic role in sIBM is controversial. This study investigated the pathogenic properties of anti-cN1A autoantibodies in sIBM pathogenesis. We developed a cell-based assay to detect anti-cN1A autoantibodies, which we found in serum from patients with neuromuscular diseases including sIBM. We also investigated the clinicopathological differences between sIBM patients with and without the autoantibodies. We used passive in vitro and in vivo immunization models to evaluate the pathogenic role of the autoantibodies. Of 67 patients with sIBM, 24 (35.8%) possessed anti-cN1A autoantibodies as determined via our cell-based assay. In the anti-cN1A-positive group, the percentage of patients with hepatitis C virus antibodies was significantly lower and the mean area of type 2 myofibers was significantly smaller compared with the autoantibody-negative group. In the in vitro passive immunization model, p62/SQSTM1 significantly increased in anti-cN1A-positive sIBM immunoglobulin G (IgG)-supplemented cells. In the in vivo passive immunization model, anti-cN1A-positive sIBM IgG-injected mice demonstrated p62/SQSTM1-positive sarcoplasmic aggregates in myofibers, associated with macrophage infiltration. Our cell-based assay is useful for anti-cN1A autoantibodies detection. Patients with anti-cN1A autoantibodies demonstrated unique clinicopathological features."

According to the news reporters, the research concluded: "In vitro and in vivo passive immunization model results suggest that anti-cN1A autoantibodies may affect protein degradation in myofibers."

For more information on this research see: Pathomechanisms of Anti-Cytosolic 5 '-Nucleotidase 1A Autoantibodies in Sporadic Inclusion Body Myositis. Annals of Neurology, 2017;81(4):512-525. Annals of Neurology can be contacted at: Wiley, 111 River St, Hoboken 07030-5774, NJ, USA. (Wiley-Blackwell - www.wiley.com/; Annals of Neurology - onlinelibrary.wiley.com/journal/10.1002/(ISSN)1531-8249)

Our news journalists report that additional information may be obtained by contacting S. Yamashita, Kumamoto University, Grad Sch Med Sci, Dept. of Neurol, Kumamoto, Japan. Additional authors for this research include N. Tawara, X. Zhang, M. Korogi, Z.W. Zhang, T. Doki, Y. Matsuo, S. Nakane, Y. Maeda, K. Sugie, N. Suzuki, M. Aoki and Y. Ando.

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