NEW YORK (Reuters Health) Nov 18 - Brains obtained at autopsy from autism patients show widespread neuroglial activation and inflammation, according to a report in the November 15th online edition of Annals of Neurology.

Despite suggestions that immune dysfunction plays a role in the pathogenesis of autism, the authors explain, neuropathological studies have given little attention to immune and neuroglial activity in autism. Dr. Carlos A. Pardo from Johns Hopkins University School of Medicine, Baltimore, and colleagues studied brain tissues obtained at autopsy from 11 autistic patients and cerebrospinal fluid from 6 living autistic patients. Neuropathological examination of autistic brains revealed extensive neuroglial responses, along with patchy loss of neurons in the Purkinje cell layer and granular cell layer of the cerebellum. "The marked neuroglial activity in the cerebellum is consistent with previous observations that the cerebellum is one focus of pathological abnormalities in morphological and neuroimaging studies of patients with autism," Dr. Pardo commented. There was, however, no evidence of adaptive immune reactions in autistic brains, the authors report.

Brain tissues from autistic patients showed increased levels of proinflammatory cytokines, the results indicate, particularly in the region of the anterior cingulate gyrus. Cytokines originated principally from reactive astrocytes.

Cerebrospinal fluid from living autistic patients showed significant increases in MCP-1, IL-6, IFN-gamma, IL-8, MIP-1beta, and other proinflammatory cytokines and modulatory cytokines.

"These cytokines play important roles in immune mediated processes, and their presence in the CSF in autistic patients may reflect an ongoing stage of inflammatory reactions likely associated with neuroglial activation and/or neuronal injury," Dr. Pardo explained. "At present, there is no indication for using anti-inflammatory medications in patients with autism," Dr. Pardo cautioned. "There are ongoing experimental studies to examine the effect of drugs that limit the activation of microglia and astrocytes, but their use in humans must await further evidence of their efficacy and safety."

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