

REPORT

Serum Test for Neuronal and Glial Autoantibodies

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John Hoyte

Sera obtained from John Hoyte and from healthy adults (control) were assayed for the presence of autoantibodies against proteins associated with neurogenesis, i.e., high molecular weight neurofilament protein (NFP-200, NFH), microtubule associated protein-2 (MAP-2), and Tau proteins, myelinogenesis, i.e., myelin basic protein (MBP), and gliogenesis, i.e., glial fibrillary acidic protein (GFAP), that have been used as markers for injury to the central nervous system. Autoantibodies against tubulin, a protein present in all tissues, including the nervous system, have been determined as markers for global tissue damage. Finally, autoantibodies against the glial calcium-binding protein S-100 were determined as markers for acute traumatic brain injury.

RESULTS

Levels of autoantibodies from neuronal and glial proteins, normalized to albumin are listed in Table 1. The results that are expressed as percentage of healthy controls represent the mean values of duplicate assays of optical density arbitrary units at 1:50 serum dilution.

Table 1: Serum autoantibodies expressed as percentage of healthy controls

<u>NFH</u>	<u>MAP-2</u>	<u>TAU</u>	<u>MBP</u>	<u>GFAP</u>	<u>Tubulin</u>	<u>S100</u>
140	257	358	227	167	185	80

Serum autoantibodies against neuronal proteins, NFH, MAP-2, Tau proteins, and MBP were significantly higher than that of controls. Autoantibodies against the protein associated with gliogenesis, the glial protein, GFAP were significantly increased. Autoantibodies against the neuronal and global protein, tubulin were significantly more than controls.

Autoantibodies against S-100 protein are used as an internal standard to determine the precision of the assay. The results show that the level of these autoantibodies was low in the patient and controls. The results indicate the high precision of the results. They also suggest the absence of acute traumatic brain injury in the cases and controls.

DISCUSSION

Alterations of the cytoskeletal structure are prominent features in some neurological diseases and chemically induced neurological disorders. Neurofilament and Tau proteins are major constituents of the axon and MAP-2 is mostly present in the dendrites. Increased autoantibodies of these proteins is indicative of axonal degeneration. Also, increased autoantibodies against MBP is consistent with demyelination. The increase of GFAP autoantibodies is an indication of neuronal injury.

Many neurotoxicants, such as organophosphorus insecticides, as well as other insecticides, solvents and heavy chemicals cause neuronal cell death and axonal degeneration and over-expression of GFAP, with subsequent release of neuronal, myelin, and glial proteins into circulation, followed by the formation of autoantibodies against these proteins. While not diagnostic for specific disease, the presence of circulating autoantibodies against neuronal and glial proteins, at higher levels in patients who had been exposed to neurotoxic chemicals and developed neurological deficits, over that of controls, can be used as further confirmation for chemical-induced nervous system injury. The low level of autoantibodies of S100 protein in the serum indicates that the neuronal condition is not related to an acute injury, but is rather a chronic condition.

CONCLUSIONS

The serum profile of increased autoantibodies against nervous system proteins, is consistent neurological deficits, and in the absence of other neurological diseases, it is concluded that this profile is consistent with chemical-induced nervous system injury.

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