**WORGU VICTORY IZEOMA**

**16/MHS03/032**

**HISTOCHEMISTRY ASSIGNMENT**

1. **If nissil stain is used to demonstrate RNA/DNA in the neurons of CNS, what staining technique is used for identifying the same in peripheral neurons?**

De Olmos stain is a new amino-cupric silver protocol, and is described for detection of neuronal degeneration. It describes its selectivity in visualizing both early and semi acute degeneration after intracerebral or systemic administration of a variety of neurotoxicants in rats, and after transient ischemic episodes in gerbils. As early as 5 min after physical trauma, or 15 min following either intrastriatal injections of glutamate analogs or exposure to ischemic episodes, neuronal silver staining was evident at primary sites of trauma (i.g. injection sites) and at hodologically related secondary sites. With intoxication by peripheral injections of trimethyltin (IP) or intracerebral injections of Doxorubicin, reproducible patterns of degeneration are demonstrable after 24 h or after 9-13 days, respectively. The amino-cupric silver method permits simultaneous detection of all neuronal compartments against a clear background. Degeneration in the neuronal cell bodies, dendrites, axons and terminals, as well as the recruitment of new structures in a progressive pathologic process, could be accurately followed. The amino-cupric silver method is an especially useful tool for surveying neuronal damage in basic neuroscience investigations and in neuropathologic and neurotoxic assessment.

1. **Is luxol fast blue stain also used to detect demyelination in the PNS? explain the procedure involved in the demonstration of demyelination in the PNS.**

Luxol fast blue is commonly used to detect demyelination in the central nervous system (CNS),

But cannot discern myelination in the peripheral nervous system.

**Procedure for Demyelination in The PNS**

Insults to the PNS by various infectious agents, genetic predisposition and immune-related mechanisms jeopardize Schwann cell functions and cause demyelination. To date, there are no effective and reliable biomarkers for PNS-related diseases.

Peripheral demyelinating diseases (PDD) refer to a spectrum of disorders that involves substantial damage to axons and glial cells, particularly Schwann cells (SC) in the peripheral nervous system (PNS). The incidence of these diseases is variable. Disease states are manifestations of damage against the myelin sheath caused by various inciting factors, such as infectious agents, auto-immune processes or genetic mutations. Oxidative stress, the primary risk factor in many diseases, has also been implicated in demyelination disorders. Schwann cells are principal glial cells in peripheral nerves that originate from the neural crest, which is a multipotent embryonic structure that also differentiates into other main glial subtypes of the PNS. SC development occurs through a series of embryonic and postnatal phases, which are tightly regulated by a number of cellular signaling pathways. During the early embryonic phase, neural crest cells differentiate into SC precursors that represent the first transitional stage in the SC lineage, that subsequently further differentiate into immature SC. At time of birth, these immature SC differentiate into either myelinating or non- myelinating SC that populates the mature nerve trunks and wrap around axons through a process known as myelination. Myelination is a process whereby SC develops a multi-layered membrane called the myelin sheath around the axonal membrane. Mostly, larger axons (>1 um) are selected specifically by SC to form multiple internodes of the myelin sheath. Myelination begins with the establishment of a 1:1 relationship with the axon. At this level, the production of myelin structural proteins such as myelin protein zero, peripheral myelin protein, myelin basic protein is increased along with lipid biosynthesis

PNS Demyelinating Diseases

Demyelination describes the loss of the myelin sheath, where SC are being destroyed or unwrapped from axons. Demyelination causes neurological disability due to conduction block and axonal degeneration.

* Acquired Demyelinating Disease

Guillain-Barre Syndrome Guillain-Barre Syndrome (GBS) is an acute idiopathic autoimmune demyelinating disease of the PNS that is characterized by acute flaccid ascending neuromuscular paralysis. Most cases of GBS are preceded by antecedent infections of several microbes of the gastrointestinal and upper respiratory tracts. Apart from infections, some GBS cases are results of trauma, surgical interventions, treatment with monoclonal antibodies and vaccination (rare).

* Chronic Inflammatory Demyelinating Polyradiculoneuropathy Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is an acquired immune mediated demyelinating disease of the PNS characterized by progressive loss of motor and sensory functions. CIDP sometimes is quite similar to GBS, with the distinction that its clinical course is chronic with relapses. The onset is insidious and occurs more commonly in older age individuals.

The immune system primarily attacks and damages the myelin sheath of the PNS followed by segmental demyelination and axonal degeneration.

Reference

Lorenzo S. Use of an amino-cupric-silver technique for the detection of early and semi acute neuronal degeneration caused by neurotoxicants, hypoxia, and physical trauma. Neurotoxicol Teratol. 1994;16(6):545–561.