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IS4. METAL NEUROTOXICITY

Kürşat Bora ÇARMAN
Department of Pediatric Neurology, Osmangazi University, Turkey

Metal neurotoxicity has address the molecular, pathological, and functional responses of central and peripheral nervous systems. Lead, mercury, arsenic, manganese and aluminum are the most common neurotoxic metals.

Lead: Lead occurs naturally in trace amounts in soil, rocks and water. Acute lead encephalopathy is most commonly seen in occupationally exposed adults or in children following ingestion of lead-containing items. Children tend to present with lethargy, confusion, ataxia and impaired motor functions, and irritability. Hallucinations, seizures, and coma can occur in patients. Brain edema occurs with higher levels of exposure and can mimic a mass lesion with papilledema, positive Babinski sign, and even focal or lateralizing deficits. Neurological sequelae are more persistent in children, with the most profound effect on intelligence quotient levels.

Mercury: Mercury has three forms, elemental, organic, and inorganic forms. Neurological manifestations of methylmercury toxicity range from mild paresthesias and tremor to severe ataxia, spasticity, seizure, memory loss, insomnia, hallucination and visual and hearing loss. Encephalopathy may be a prominent feature and in severe cases may progress to coma and death. The rate of encephalopathy depends on the rate of peripheral metabolism and their ability to cross the blood brain barrier.

Arsenic: Arsenic is used in different industries. Neurotoxicity can result in a profound leukoencephalopathy following either acute or chronic exposures. Acute toxicity primarily manifests as confusion, with headache initially. In the hours to days following, delirium, hallucinations, and seizures may occur. Diffuse encephalopathy at may be profound as well. Chronic encephalopathy is more commonly caused by exposure to organic than inorganic arsenic. Chronic arsenic encephalopathy generally manifests with confusion and irritability. Paranoid delusions and auditory or visual hallucinations can occur. Brain imaging and EEG is often normal.

Manganese: Manganese (Mn) is essential and act as cofactor for several enzymatic reactions in human body.

The classic and most prominent manifestation of Mn toxicity is parkinsonism, but encephalopathy also occurs

with both acute and chronic exposures. Acute toxicity can cause frank psychosis, with visual and auditory hallucinations, euphoria, and compulsive behaviors. Headache, irritability, and memory disturbance can be seen with acute or chronic Mn encephalopathy. With continued exposure, behavioral changes progress. Emotional lability, compulsive laughter, and hallucinations may all present before the appearance of the typical motor features. Tremor, dysarthria, increased tone, and gait disturbance occur relatively late in the process. Brain magnetic imaging studies reveals increased signal on T1-weighted images within basal ganglia.

Aluminum: Encephalopathy is a primary feature of acute or chronic aluminum toxicity. Motor incoordination, poor memory, impaired cognition, and depression are the hallmark symptoms.

Dialysis-induced encephalopathy is due to the toxic effects of aluminum in dialysis fluid and in the phosphate binders used in dialysis patients. This syndrome occurs in patients after 2 to 7 years of dialysis. Often presenting initially with isolated speech abnormalities, neurological symptoms progress at varying rates and include episodic confusion, behavioral changes, myoclonus, seizures, and frank dementia. Blood levels can be used to evaluate patients with potential aluminum toxicity.

* kbcarman@gmail.com