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Inorganic mercury exposure following Indian indigenous (Siddha) medicine intake - A rare cause of anti-VGKC antibodies-associated acquired neuromyotonia.

Dr Amith Balachandran[1], Mohan Jambugulam[1], Krupa George[1], Annie F Stephy[1], A Praveen Kumar[1], Ravikar Ralph[1]

[1]Poison Control Center, Department of Medicine, Christian Medical College, Vellore, Tamil Nadu, India

Aim and objectives:

The aim is to discuss the challenging diagnosis and treatment of Inorganic Mercury exposure induced neuromyotonia in a series of five patients treated at the Clinical Toxicology Unit of a Poison Control Center in South India. The specific objectives are:

1) To describe the clinical characteristics, laboratory investigations, treatment and outcome of patients with inorganic mercury exposure related acquired neuromyotonia
2) Discuss the autoimmune basis of the syndrome and pathophysiology based on available published literature

Methodology:
A retrospective search of electronic medical records (EMR) was conducted and patients with inorganic mercury exposure related acquired neuromyotonia admitted to the Clinical Toxicology Unit from January-2020 to July-2023 were identified. The clinical and laboratory details were obtained from the in- patient records.

Results:

Five patients with inorganic mercury toxicity following indigenous (Siddha) medicine intake with acquired neuromyotonia, based on their clinical and autoantibody profile were identified. Four were adults and one was a child. All had anti-VGKC antibodies positivity. Some had elevated catecholamines and proteinuria. We discuss pathogenic aspects of mercury-induced autoimmunity. All were treated with chelation therapy and short course glucocorticoids in addition to source control. All recovered with near complete resolution of symptoms. We also highlight a rare subset of patients in our series who were positive for dual anti-VGKC-autoantibodies (LGI1 and CASPR2). While several reports of anti- CASPR2 antibody positive acquired

neuromyotonia exist, there is only one other report of dual-antibody positive neuromyotonia following chronic mercury exposure in the literature.

Conclusions:

Inorganic mercury exposure from indigenous medicines is a rare cause of acquired neuromyotonia and can be difficult to diagnose and treat. Pathogenic mechanisms need to be better studied; autoimmunity plays an important role. Treatment includes source control, immunomodulation and chelation therapy, and recovery is near complete following appropriate therapy.