Parsonage-Turner syndrome diagnosis

Diagnosing Parsonage-Turner syndrome can be challenging due to its variable presentation and the absence of specific diagnostic tests. However, healthcare professionals typically use a combination of clinical features, medical history, and sometimes imaging studies to make a diagnosis.

Clinical Evaluation:

Medical History: A thorough history of the symptoms, including the onset and progression of pain, weakness, and any preceding illness or trauma, is important. Physical Examination: A neurological examination is crucial to assess muscle strength, reflexes, and sensory function in the affected area. Electrodiagnostic Studies:

Electromyography (EMG): This test involves the insertion of small needles into muscles to measure the electrical activity. In Parsonage-Turner syndrome, EMG may reveal denervation changes in affected muscles. Nerve Conduction Studies (NCS): NCS assesses the ability of nerves to transmit electrical signals. Abnormalities in nerve conduction may be observed. Imaging Studies:

MRI (Magnetic Resonance Imaging): While there is no specific imaging finding that confirms Parsonage-Turner syndrome, an MRI of the affected area may be performed to rule out other causes of symptoms, such as nerve compression or structural abnormalities.

Blood Tests:

Blood tests: These may be conducted to rule out other conditions, including infections or autoimmune disorders. It's important to note that there is no definitive diagnostic test for Parsonage-Turner syndrome, and the diagnosis is often one of exclusion.

The postsurgical patient who develops PTS poses a specific diagnostic dilemma. In these individuals, the patient has undergone a rather traumatic experience to the body considering perioperative administration of antibiotics, intraoperative anesthesia, operative manipulation of tissue, and various positioning techniques used to facilitate surgical techniques. It is no mystery that, when this cohort of patients develops PTS, it is often attributed to intraoperative positioning or even part of the rehabilitative process manifested as shoulder strain with therapy.

Parsonage-Turner syndrome and hereditary brachial plexus neuropathy (HBPN) present with indistinguishable attacks of rapid-onset severe shoulder and arm pain, disabling weakness, and early muscle atrophy. Their combined incidence ranges from 3 to 100 in 100,000 persons per year. Dominant mutations of SEPT9 are the only known mutations responsible for HBPN. Parsonage and Turner termed the disorder “brachial neuralgic amyotrophy,” highlighting neuropathic pain and muscle atrophy. Modern electrodiagnostic and imaging testing assists the diagnosis in distinction from mimicking disorders. Shoulder and upper limb nerves outside the brachial plexus are commonly affected including the phrenic nerve where diaphragm ultrasound improves diagnosis. Magnetic resonance imaging can show multifocal T2 nerve and muscle hyperintensities with nerve hourglass

swellings and constrictions identifiable also by ultrasound. An inflammatory immune component is suggested by nerve biopsies and associated infectious, immunization, trauma, surgery, and childbirth triggers. High-dose pulsed steroids assist initial pain control; however, weakness and subsequent pain are not clearly responsive to steroids and instead benefit from time, physical therapy, and non-narcotic pain medications. Recurrent attacks in HBPN are common and prophylactic steroids or intravenous immunoglobulin may reduce surgical- or childbirth-induced attacks. Rehabilitation focusing on restoring functional scapular mechanics, energy conservation, contracture prevention, and pain management are critical. Lifetime residual pain and weakness are rare with most making dramatic functional recovery. Tendon transfers can be used when recovery does not occur after 18 months. Early neurolysis and nerve grafts are controversial [1)]

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Meiling JB, Boon AJ, Niu Z, Howe BM, Hoskote SS, Spinner RJ, Klein CJ. Parsonage-Turner Syndrome and Hereditary Brachial Plexus Neuropathy. Mayo Clin Proc. 2024 Jan;99(1):124-140. doi: 10.1016/j.mayocp.2023.06.011. PMID: 38176820.