CLINICAL VIGNETTE

Vaccine-Related Parsonage-Turner Syndrome

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Case Presentation

A 40-year-old female presented in a tele-health visit for left shoulder pain 3 days after her first COVID-19 vaccination. Her vaccination site was on the left deltoid and there was no evidence of local erythema or swelling. Range of motion in her left shoulder was limited in all directions due to pain. A presumptive diagnosis of shoulder injury after vaccine administration (SIRVA) was made and anti-inflammatory medications were recommended. Five days later she was seen for an in-person evaluation for worsening shoulder pain, spreading to left breast and axilla, as well as new finger numbness. She was afebrile and exam revealed reduced abduction, flexion and internal rotation of the left shoulder with 4/5-muscle strength proximally and left-hand dysesthesia. Breast exam was normal but there was lymphadenopathy in the left axilla. Labs included complete blood count, comprehensive metabolic panel, erythrocyte sedimentation rate, and C-reactive protein all returned within normal limits. MR brachial plexus showed left axillary and supraclavicular lymphadenopathy and edema surrounding the deltoid muscle and subtle asymmetric STIR hyperintensity within the region of the left brachial plexus. This established the diagnosis of vaccine-related Parsonage-Turner syndrome.

Discussion

Parsonage-Turner syndrome (PTS), which is also known as neuralgic amyotrophy, is characterized by acute onset shoulder and arm pain followed by weakness. Pain is typically severe and hyperesthesia and paresthesias may also be present. Muscle weakness and wasting typically follows the painful episode, which may last days or weeks. There are two main types of Parsonage-Turner syndrome: hereditary, which accounts for the minority of cases, and sporadic. In most cases, there is an antecedent trigger such as infection, vaccination, trauma, surgery, childbirth, or physical activity.[1] About one-third of patients will present with involvement of other peripheral nerves, including the phrenic nerve, which can manifest as dyspnea.[2]

While the etiology of Parsonage-Turner syndrome is not fully known, evidence suggests an autoimmune mediated process in addition to hereditary and biomechanical factors. For example, nerve biopsies in acute PTS patients revealed lymphocytic inflammatory infiltrates in the affected nerves.[1] Mononuclear inflammatory infiltrates, without features of necrotizing vasculitis were found surrounding epineurial and endoneurial vessels.[1,2] Because many patients with PTS report strenuous upper body exercise prior to the onset of symptoms, biomechanical factors are also thought to play a role. One theory is that repeat microtraumas to the brachial plexus lead to increased permeability of the blood-nerve barrier, allowing immune factors access to the endoneural space.[2,3] Genetic factors are also at play, with 1 in 10 patients with PTS reporting a family history of PTS.[3]

Diagnosis of PTS is made on clinical grounds with the classic symptoms of rapid onset severe shoulder girdle pain, followed by weakness, usually with a history of inciting event such as infection, vaccination, or strenuous exercise of the upper extremities. Laboratory findings are generally useful only to evaluate for infectious etiologies and rule out elevation in liver function tests that can be seen with Hepatitis E infection, which is an emerging cause for PTS.[2,4] Nerve conduction studies (NCS) and electromyography (EMG) can be useful later in the disease process. Because it may take up to four weeks for the denervation to be apparent, EMG,[3] may be normal if done in the acute setting. MRI of the brachial plexus with and without contrast may show edema, focal thickening, and T2 and short-tau inversion recovery (STIR) hyperintensity or enhancement with gadolinium.[2]

Treatment for PTS is predominantly supportive with analgesics. In the acute phase, case reports suggest oral prednisolone can shorten duration of symptoms and severity.[5] Physical and occupational therapy are prescribed to help maintain range of motion and assist with the recovery phase. Recurrences within the first years after initial attack can be as high as 75% in patients with hereditary PTS and 25% for sporadic PTS.[2]

Parsonage Turner syndrome has occurred with COVID-19 infection[1,6] as well as following COVID vaccination.[6-8] This patient developed classic symptoms of PTS following vaccination against COVID-19. It is important for primary care, emergency, as well as sports and orthopedic doctors familiarize themselves with the symptoms of PTS and consider it in the differential for patients presenting with rapid onset severe shoulder pain followed by weakness.

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