CLINICAL VIGNETTE

Mononucleosis and Proximal Arm Weakness: Parsonage Turner Syndrome

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A 20-year-old female, with a prior history of long-COVID syndrome following her first covid infection in 2021 and family history of multiple sclerosis, presented to the emergency room for abdominal pain. Six weeks prior to this presentation, the patient experienced her second COVID-19 infection, which was mild and treated with Paxlovid by an outside physician due to her long-COVID history. One week prior to her presentation, she had sinusitis with some lingering sinus pain and drainage, and took a course of azithromycin and prednisone. The night before her presentation, she was awakened with abdominal pain. She also reported fevers, chills, nausea, and diarrhea. At the emergency room, her labs included AST of 109, ALT of 180, and Alkaline phosphatase of 144. CT of abdomen and pelvis showed mild hepatosplenomegaly but no other findings. She was diagnosed with a viral illness and discharged to follow up with her primary care physician. On follow up, mononucleosis was thought to be the most likely etiology to her symptoms and findings, and was confirmed with a positive EBV-VCA IgM antibody. The patient was counseled on safety precautions for someone with splenomegaly and agreed to follow up for repeat exams and labs. However, one day after her outpatient visit and 5 days following her emergency room visit, she reported to the primary care office sudden onset left shoulder and neck pain awakened her at night. Movement exacerbated her pain and she held her arm at her side to minimize discomfort. The following morning, the patient reported pure left arm weakness. Even after the pain resolved, she continued to have difficulty raising her arm overhead. She noted brief numbness and tingling in her middle three fingers, which resolved. During a video visit to assess this new complaint, she reported the pain had improved over several hours but weakness in her arm persisted. Although the tele video visit was limited, she appeared to have 4/5 weakness with abduction and flexion at the left shoulder.

A cervical MRI was performed showing a small focus of abnormal T2 hyperintensity in the spinal cord at C6-C7 with a differential that could include transverse myelitis. This finding, led to hospitalization with neurology consulting to complete evaluation for autoimmune processes secondary to her recent viral illness. MRI of her spine revealed no cord lesions and the initial finding on cervical spine was likely a syrinx. Brachioplexus MRI disclosed no abnormalities. Lumbar puncture showed normal findings. Given no findings on diagnostic testing, she was discharged with close outpatient follow up.

Initial follow up after hospitalization, demonstrated continued 4/5 weakness with shoulder flexion. Her neurologic exam was otherwise intact. At neurology follow up, repeat imaging and EMG revealed abnormalities consistent with brachial plexus neuritis diagnosis. She was offered a trial of steroids, with uncertain efficacy. She worked with physical therapy her weakness slowly improved months after onset.

Discussion

This case raises a number of neurologic conditions that her symptoms could have been attributed, including transverse myelitis, acute viral myelitis, and an acute brachioplexus neuropathy. Because of the potential transverse myelitis etiology, hospitalization was needed to expedite evaluation and treatment. However, the most likely diagnosis based on history and physical examination was an acute brachioplexus neuropathy, an uncommon cause of shoulder and neck pain. Often confused with cervical radiculopathy and shoulder pathologies, brachial neuritis should be considered when patients present with reduced shoulder motion and pain. Due to the low incidence of this condition, literature on this topic predominately includes case reports.

Clinical features of this case that suggest brachial plexopathy include the predominant symptom of severe pain preceding the neurologic symptoms. The pain, which is sometimes described as “deep” and “burning,” can be severe enough to mask perception of any sensory changes. The pain due to brachial neuritis is always followed by profound weakness. What makes the diagnosis difficult is that involvement of the brachial plexus can be patchy. It may not follow one trunk but involve different trunks, roots, and peripheral nerves leading to a varied pattern of neurologic changes.[1] In this case, the upper trunk (C5-C6) involvement was suspected due to the significant proximal arm involvement, specifically shoulder girdle weakness. Consistent with possible patchy involvement, the patient reported sensory changes involving digits 2-4 on the left hand which would fit a middle trunk pathology (C7-C8). Interestingly, the patient was not certain if she experienced numbness and tingling at the initial onset because her pain was quite severe (classic brachial plexus pathology).

Notably, the patient’s initial MRI results were normal. Magnetic Resonance Neurography of the brachial plexus is the ideal radiologic evaluation as it provides the best view of the brachial plexus and muscles.[2] An MRI helps confirm the diagnosis, which is typically made using clinical data and EMG, and also rules out other causes that can mimic this condition like a rotator cuff tear or cervical pathology. When there is a brachial

neuritis, the plexus appears thickened and has a hyperintense signal on T2W and STIR images. Surprisingly, this was not seen on initial imaging, and only observed weeks after the hospitalization when MRI was repeated. This lag supports findings that the plexus can appear normal with mild neuritis or because imaging has been performed too early.[2] Affected muscles, may take two weeks to see changes on MRI scans.[2] A study on MRI imaging timelines in the context of brachioplexus neuropathy reported 24 out of 27 patients had initially normal MRI plexus findings.[3]

There are many potential causes of brachial plexus injury: trauma, neoplastic infiltration, injury from radiation, and neurogenic thoracic outlet syndrome. In this patient, an inflammatory brachial plexopathy known as Parsonage-Turner syndrome was the culprit. Other names for this disorder include idiopathic brachial plexopathy, acute brachial plexitis, and brachial plexus neuropathy. Though deemed idiopathic, half of patients have histories of recent immunizations, operations, or viral illnesses within 3 weeks of symptoms. This timeline suggests such events are triggers of a T-lymphocytic inflammatory reaction, which has been confirmed on biopsies.[1] The immune-mediated inflammatory reaction involves complements, antibodies directed towards nerve myelin, and T cells leading to Wallerian degeneration.[4] Parsonage-Turner Syndrome fits this case, given the recent diagnosis of mononucleosis prior to her presentation.

Steroid treatment was offered to this patient but not guaranteed to work as no treatments have been proven effective in reducing neurologic impairment and improving prognosis.[1] There are no controlled studies on steroid and immunosuppressive treatments. Despite this being an immune mediated process, existing studies suggest early steroid use can reduce the pain phase of the disease but will not alter prognosis.[5] Despite lack of available effective treatments, prognosis is good. After several weeks, pain usually resolves, and by 2-3 years, weakness typically improves, consistent with a reinnervation process.[1] A case series published in the UK reported complete recovery in 89% of patients by 3 years.[6]

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