CLINICAL VIGNETTE

A Case of Zoster Associated Brachial Plexopathy

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Case Report

The patient is a 65-year-old female with type II diabetes and a remote history of left breast cancer. She presented with several days of tingling and pain in the left arm. One day prior to her visit, she developed a vesicular rash along the left arm. Initial evaluation was consistent with herpes zoster of the left arm in the cervical-thoracic C8-T1 dermatomal distribution.

The patient was seen within twenty-four hours of onset of the eruption and given high-dose acyclovir and analgesics (acetaminophen with codeine). The patient presented five days later with two days of progressive left arm weakness. The patient noted inability to move her arm to any degree but was able to move her hand. She noted increased numbness and tingling along the left side of the neck and radiating to the lateral aspect of her arm. The patient denied any headaches, fevers, chills, sweats, or trauma.

On physical exam, the patient appeared visibly distressed with pain. She had a heart rate of 125 and was normotensive at 120/65 and afebrile. Head and neck exam was normocephalic and atraumatic. No cervical, clavicular, or axillary adenopathy was present. Cervical motion was within normal limits with no cervical spine tenderness. Lung exam was normal, and cardiovascular exam was notable only for tachycardia. On inspection, the left arm was of normal bulk and tone with one and two mm vesicles consistent with herpes zoster along the deltoid and extending distally to the mid-forearm along the cervical-thoracic distribution of C8-T1. Additionally, the patient had vesicles along the left upper chest. There was generalized erythema without exudate or edema. Vascular examination was normal.

Neurologic examination revealed normal cranial nerve**s** II-XII. Motor examination of the left arm revealed abduction strength of 2/5, biceps of 1/5, triceps of 2/5, and distal strength of 4/5 with normal fine motor function of the hand. She had normal motor strength of bilateral lower extremities. Sensory exam was normal to light touch and pinprick. Deep tendon reflexes were 2/4 in all extremities. Babinski, cerebellar maneuvers, and Romberg exam were negative. The patient had a normal casual gait.

Discussion

The patient's history of acute left arm weakness was initially concerning for both a cervical spine and a peripheral nerve process. The neurologic exam was consistent with a peripheral lesion. The patient underwent MRI of the cervical spine, left shoulder, and brachial neurography. The MRI of the cervical spine revealed age-related changes, and the left shoulder revealed a normal rotator cuff. Brachial neurography demonstrated enhancement of the left brachial plexus. One month later, the patient underwent electromyography and nerve conduction velocity studies, which demonstrated evidence of demyelination in the brachial plexus distribution.

Trauma is the most common cause of brachial plexopathy. Compression, transection, ischemia, inflammation, neoplasia, autoimmune disorders, and radiation therapy are additional common causes of brachial plexopathy. Parsonage-Turner syndrome, also known as neuralgic amyotrophy or acute brachial plexus neuritis, was also considered. It is characterized by the sudden appearance of severe shoulder pain followed by progressive motor weakness.

This patient's history of breast cancer was also concerning for possible compressive plexopathy. The MRI and chest x-ray were without obvious compressive pathology. The preceding herpes zoster eruption in the motor distribution of the weakness was concerning for an acute brachial plexopathy related to zoster.

Paresis due to herpes zoster is a less common and less well-recognized complication than post-herpetic neuralgia. The reported incidence of segmental limb paresis with cutaneous zoster is 1–5%.[1] The data on zoster associated brachial plexopathy are limited. The cause of the neuritis has not been conclusively established. Studies indicate that the virus spreads proximally as well as distally, causing local neuritis in the spinal nerve, anterior and posterior horn cells, and anterior and dorsal roots.[2] It is unclear if the neuritis is a direct viral associated neuritis versus an inflammatory process. The basic pathological neural reaction to herpes zoster is axonal degeneration with a degree of secondary segmental demyelination.[3] Evidence of demyelination has been shown on EMG.[4]

Segmental zoster paresis does not always involve the same distribution as the sensory dermatomal distribution, which may cause diagnostic confusion.[5] Weakness usually occurs within 2–3 weeks of rash onset but can precede the skin eruption or occur long after the skin eruption in rare cases.

Diagnosis can be supported based on history, MRI, and nerve conduction testing. MRI of the brachial plexus can demonstrate inflammatory changes in the brachial plexus with swelling and thickening of divisions or cords of the brachial plexus. MR neurography can detect denervation edema of affected muscle. MRI findings concur in only 60% of patients with symptoms suggestive of radiculopathy and in 76% of cases when weakness is present.[6,7]

Nerve conduction findings such as positive sharp waves and fibrillations on diagnostic electrophysiological studies usually occur two weeks after the onset of symptoms.[4] Therefore, electromyography is limited for the early diagnosis of brachial plexus neuritis. MR imaging may be a more useful study for early diagnosis of herpetic brachial plexopathy.

Treatment of the plexopathy is largely directed towards rehabilitation, control of the neuropathic pain, and maintaining muscle strength.[3] This patient underwent six months of aggressive physical therapy aimed at maintaining muscle tone and distal strength. Her strength improved as the neuritis diminished. Steroids or anti-inflammatories have generally been discouraged**,** although no randomized studies have been conducted to date. Case studies have demonstrated that about 50-60% patients recover most if not all function within 6 months. Roughly 50% will have some residual deficit,[4] and there are high rates of post herpetic neuralgia.[4] This patient had near full recovery by 6 months.

Conclusion

Herpes Zoster is an unusual cause of paresis and of brachial plexopathy. Most patients will recover within one year. The treatment is largely supportive with physical therapy.

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