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A Case of Acute Brachial Neuritis: Parsonage-Turner Syndrome and Literature Review

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Abstract

The global push to eradicate SARS-CoV-2 has resulted in an unprecedented number of intramuscular vaccines given to the world population, as such we are seeing an increase in possible side effects. One such side effect may be Parsonage-Turner Syndrome (PTS). Parsonage-Turner Syndrome (PTS) presents post-Subcutaneous (SQ) or Intramuscular (IM) injection of a vaccine and is a particular brachial plexopathy. When assessing the degree of plexus injury, relevant diagnostic testing, including Electromyography (EMG) and nerve conduction study (NCS), is of particular importance to avoid delays in treatment. We hereby present a case of a 56-year-old male with acute brachial neuritis following IM flu vaccination.

Keywords: Turner syndrome; Parsonage-Turner syndrome; Viral infection; Vaccination

Introduction

The widespread use of the ever-evolving influenza vaccines has led to previously unknown and undiagnosed side effects becoming more and more prevalent. One such side effect is Parsonage-Turner Syndrome (PTS). Parsonage-Turner Syndrome (PTS) is a brachial neuritis that presents as an inflammatory brachial plexopathy that can occur following physiological stress such as systemic viral infection, surgery, or vaccination. Parsonage-Turner Syndrome (PTS) is characterized by an acute often asymmetric severe pain localized to the shoulder (in the case of IM vaccination) and radiating distally to the arm, neck, and back. The pain usually appears within several days following a bacterial or viral illness, surgery, or immunization. The syndrome is usually self-resolving within several days and most patients spontaneously recover without intervention. However, corticosteroids, analgesics, and physical therapy have shown to be key components in the treatment and supportive management of PTS when deemed necessary.

Case Report

A 56-year-old male presented to the neurology clinic with a chief complaint of pain in his left shoulder. The patient had received an intramuscular influenza vaccine, after which he noted severe arm pain for which he ultimately went to the emergency room, where he was given an injection of anti-inflammatory medication with no improvement. His symptoms worsened over the week when he noticed increased pain and swelling along with the development of weakness in the affected arm. The pain began to radiate from his shoulder, down his arm, and to his neck. The pain was continuous and described as "achy" with 10/10 in severity and exacerbated with movement. On physical examination, there was tenderness to palpation over the left cervical paraspinal muscle and trapezius muscle. There was mild

ecchymosis over the left trapezius muscle. Empty can and drop arm tests were positive. Strength was noted to be 3/5 in deltoids, 4/5 in biceps and triceps with 5/5 distal strength including wrists and fingers flexion and extension. Diagnostic tests included a CT of the shoulder showed calcific tendinitis and no obvious rotator cuff tear. He was not an MRI candidate in the setting of having a pacemaker not compatible with MRI. A Nerve conduction study and electromyography were done. The nerve conduction study (Figure 1) revealed median nerve entrapment at the level of the wrist which is consistent with mild carpal tunnel syndrome. EMG (Table 1) showed active denervation potentials in multiple muscles concerning plexopathy supporting the diagnosis of brachial plexitis.

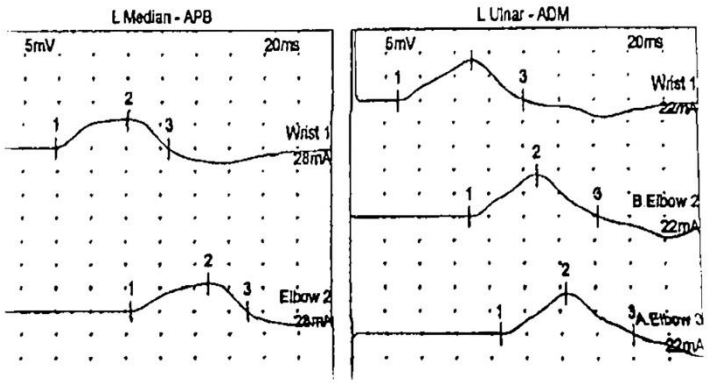

Figure 1: *NCS.*

Table 1: *EMG.*

EMG Summary Table	Spontaneous					MUAP			Recruitment
	IA	Fib	PSW	Fasc	H.F.	AMP	Dur.	PPP	Pattern
L. Abductor pollicis brevis	N	None	None	None	None	N	N	N	N
L. First dorsal interosseous	N	None	None	None	None	N	N	N	N
L. Pronator teres	N	None	1+	None	None	N	N	N	N
L. Biceps brachii	N	None	3+	None	None	N	N	N	N
L. Triceps brachii	N	None	None	None	None	N	N	N	N
L. Deltoid	N	4+	4+	None	None	N	N	N	Reduced
L. Cervical paraspinals (up)	N	None	None	None	None	N	N	N	N
L. Cervical paraspinals (mid)	N	None	None	None	None	N	N	N	N
L. Cervical paraspinals (low)	N	None	None	None	None	N	N	N	N
L. Supraspinatus	N	4+	4+	None	None	N	N	N	N
L. Infraspinatus	N	4+	4+	None	None	N	N	N	N

Discussion

P Parsonage-Turner syndrome (PTS) also known as acute brachial neuritis, idiopathic brachial plexus neuropathy, brachial plexus neuritis or neuralgic amyotrophy following an influenza vaccination is an uncommon but clinically important presentation [1]. The incidence of this syndrome is about two to three individuals per every 100,000 people, most commonly between the 3rd and 7th decades of life. This syndrome has also shown a greater predilection for males over females [2].

Its pathophysiology remains obscure, and the majority of the cases have been ascribed to surgical procedures, trauma, recent viral infections (varicella virus, herpes simplex, HIV, Coxsackie B virus, Hepatitis B virus, Hepatitis C virus, Epstein-Barr virus, cytomegalovirus, SARS-CoV2), autoimmune disorders such as systemic lupus erythematosus, polyarteritis nodosa, temporal arteritis, and vaccinations [3-6]. Most occurred after polio, chickenpox, hepatitis B, influenza, and HPV immunizations. However, post-vaccination PTS is an infrequent entity, with only 4.3-15% of all cases being attributed to vaccines [7]. Possible immune-mediated mechanisms include molecular mimicry and bystander activation, both of which may ensue following either infection (e.g., hepatitis E and SARS-CoV-2 [6]), or vaccination.

PTS commonly presents with acute, diffuse shoulder girdle and upper arm pain followed by proximal upper extremity weakness, commonly in the muscles innervated by the upper plexus (supraspinatus, infraspinatus, serratus anterior, deltoid, and biceps) [8]. This syndrome most commonly affects the upper trunk of the brachial plexus, suprascapular nerve, long thoracic nerve, axillary nerve, and anterior interosseous nerve [4]. Progressive neurological deficits, including weakness, atrophy, and occasionally sensory abnormalities usually appear between 2 and 6 weeks [9]. Discomfort can last for several weeks, with one study reporting more than 10% of patients having initial pain lasting more than 60 days and more than 75% of patients who experienced two additional phases of position-dependent neuropathic pain lasting several months. This same study documented pain primarily at night in 60% of patients [10].

Differential diagnosis of PTS includes focal extremity pathologies including subacromial bursitis, facioscapulohumeral dystrophy, adhesive capsulitis, or other nervous pathologies including radiculopathy, entrapment neuropathies, multifocal motor neuropathy, hereditary neuropathy, and mononeuritis multiplex [11,12]. Intrinsic hourglass-like constrictions of affected nerves or nerve fascicles have been recognized in the acute (≤4 weeks) phase of PTS [13] to date, they have not been observed in other spontaneous neuropathies [13,14].

Diagnosis is further complicated by the heterogeneity of symptoms among patients, which vary according to the nerves injured and the speed at which the disease progresses [12]. Therefore, a comprehensive approach involving accurate history taking, physical examination, and specific tests (e.g., electromyography and brachial plexus magnetic resonance imaging [MRI]) is required to ensure a proper diagnosis, while a delay of diagnosis and treatment may result in lasting functional damage [15].

There is no consensus on the treatment of PTS, but treatment generally involves conservative measures such as analgesia, corticosteroids, and physical therapy [4]. A proposed protocol for

corticosteroid treatment is oral prednisone at 1 mg/kg/day for 1–2 weeks followed by a taper-off over an additional 1–2 weeks [4,16]. Recently, there has been some degree of evidence suggesting benefits from the administration of immunoglobulins, with some patients responding positively, but there is little data, and more rigorous research is required to determine their ultimate efficacy [17]. PTS is typically a self-resolving process, with 80-90% recovery of muscle strength within 2-3 years, but residual paresis and exercise intolerance can happen in up to 70% of patients [18,12].

Presently, a causal relationship between PTS and the influenza vaccine has been suggested by only a few case reports, hence our case is significant as it adds weight to the existing literature. Our patient presented with PTS after receiving the influenza vaccine, presenting symptoms being severe upper extremity pain and weakness. Other etiologies of the shoulder pain were ruled out by doing a CT scan that showed calcific tendinitis and no frank rotator cuff tear. EMG/NCS was also indicative of brachial plexopathy.

Conclusion

Parsonage-Turner syndrome can present after influenza vaccination and clinicians should keep high suspicion for this diagnosis. Prompt investigation with imaging including EMG/NCS and timely management with corticosteroids, and analgesics followed by rehabilitation may reduce morbidity in this patient population. Also, in this era of increased drive for vaccines especially analgesics in immunosuppressed populations, clinicians need to be aware of this rare potential neurologic complication of the influenza vaccine**.**

Funding and Conflict of Interest

None

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