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Case Report

Parsonage-Turner syndrome following COVID-19 vaccination and review of the literature

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ABSTRACT

Background: Parsonage-Turner syndrome (PTS) is a rare brachial plexopathy characterized by self-limiting shoulder girdle and upper arm pain followed by the upper extremity weakness and sensory changes. While the etiology is not well-understood, the most common cause of PTS is thought to be postviral. There are at least nine reports, to the best of our knowledge, of PTS associated with COVID-19 infection and nine reports associated with COVID-19 vaccination.

Case Description: Here, we present a case of PTS after COVID-19 vaccination in a 64-year-old male and a review of the current literature.

Conclusion: PTS can occur post-COVID-19 vaccination and should be on the differential diagnosis when patient continues to experience shoulder pain and develops weakness or sensory changes in the extremity.

Keywords: COVID-19, Idiopathic brachial neuritis, Parsonage-Turner syndrome, SARS-CoV-2, Vaccine

INTRODUCTION

Parsonage-Turner syndrome (PTS) is a rare upper extremity disorder with an incidence of 1.64/100,000 people. Furthermore, it is known as idiopathic acute brachial neuritis or neuralgic amyotrophy, the syndrome typically involves the upper and middle brachial plexus trunks.[15,43] It is predominantly unilateral and manifests with initial shoulder girdle pain and progression to the upper extremity patchy sensory changes and proximal weakness.[15,43] The syndrome is usually self-limiting, resolving over weeks to months, with full resolution by 3 years.[15,41] The pathophysiology and etiology of PTS are not well-understood. Several published etiologies include hereditary, infection, vaccination, surgery, trauma, or autoimmune.[1,4,6,16,18,22,32,35,36,39-41,46] The most common etiology is postviral infection. To date, there are only nine published case reports of PTS associated with the novel SARS-CoV-2 (COVID-19) infection and nine case reports associated with COVID-19 vaccination.[2,3,7-12,17,21,24,26,28,30,33,37,44,45]

CASE DESCRIPTION

A 64-year-old previously healthy right-handed senior neurosurgeon with a medical history of hypertension and hyperlipidemia received his first dose of COVID-19 vaccine (mRNA-1273; ModernaTX, Inc.; Cambridge, Massachusetts) without any clinically significant side effects other

than transient malaise and fatigue. After his scheduled second COVID-19 vaccine dose administered again into the left deltoid, he experienced mild shoulder soreness and transient rigors in the first 12–24 h. However, 2 weeks postvaccination, he developed intense, unremitting left shoulder girdle pain with associated muscle tenderness that was not relieved with conservative measures such as stretching, heat, or anti-inflammatories (ibuprofen 800 mg Q.I.D.). A week thereafter, he noticed decreased strength and dexterity in the left hand with difficulty opening bottle caps and manipulating small instruments. Within the next few days, he also noted sensory loss with paresthesia and hypoesthesia in the left fourth and fifth digits, as well as on the ulnar aspect of the forearm. His neurologic examination was notable for decreased strength in the left finger extensors (4+/5), particularly in the left fourth and fifth digits, and thenar eminence (4+/5). Reflexes were normal. Sensation was impaired to light touch distally in the left fourth and fifth digits and to a lesser extent in the third digit. Brachial plexitis was suspected and he was started on a steroid taper treatment with prednisone (80 mg/day for 3 days followed by a rapid taper of 20 mg decrease per day to off).

Three weeks after his initial symptom onset, he underwent an electromyogram (EMG) and nerve conduction study (NCS). The right median and right radial sensory nerve action potentials (SNAPs) (unaffected side) had low amplitude, but these were felt to be incidental and related to known prior trauma to that hand. The left median SNAPs were normal, including the lateral and medial antebrachial cutaneous sensory nerves. The left median abductor pollicis brevis compound motor action potentials (CMAPs) were normal as well. The left ulnar SNAP and left ulnar-abductor digiti minimi CMAPs had borderline low amplitude. The left median and ulnar F waves were unremarkable. Needle EMG demonstrated a reduced recruitment pattern in the left extensor indices and left flexor digitorum profundus to digit IV, with increased spontaneous activity in the left first dorsal interosseus. These findings were felt to reflect a mild, patchy, and acute-to-subacute lower trunk brachial plexopathy [Table 1]. Magnetic resonance imaging (MRI) of the left brachial plexus revealed increased short-TI inversion recovery (STIR) signal and increased T2-weighted signal, with mild T1 postcontrast enhancement of the medial left scalene muscles along the inferior brachial plexus consistent with inflammatory changes and intramuscular edema [Figure 1].

Over the course of the next month, there was gradual improvement in finger sensation and strength with near complete resolution by 4 months.

Table 1: EMG/NCS results.

Nerve conduction studies	
Left median digit II	Normal SNAP
Right median digit II	Reduced SNAP amplitude compared to contralateral side (thought to be incidental from known prior trauma).
Left ulnar digit V	Normal SNAP
Right ulnar digit V	Normal SNAP
Left radial snuffbox	Normal SNAP
Right radial snuffbox	Reduced SNAP amplitude compared to contralateral side (thought to be incidental from known prior trauma).
Left lateral antebrachial cutaneous	Normal SNAP
Right lateral antebrachial cutaneous	Normal SNAP
Left medial antebrachial cutaneous	Normal SNAP
Right medial antebrachial cutaneous	Normal SNAP
Left median abductor pollicis brevis	Normal CMAP
Right median abductor pollicis brevis	Reduced CMAP amplitude compared to contralateral side (thought to be incidental from known prior trauma).
Left ulnar abductor digiti minimi	Reduced CMAP amplitude compared to right side. Normal distal latency and conduction velocity.
Right ulnar abductor digiti minimi	Normal CMAP
EMG studies	
Left first dorsal interosseous	Increased insertional activity. Normal recruitment, normal motor unit action potentials.
Left extensor indicis proprius	Mildly reduced recruitment. Normal motor unit action potentials.
Left flexor digitorum profundus 4	Severely reduced recruitment. Normal motor unit action potentials.
Left C8 paraspinal muscles	Normal
Left infraspinatus	Normal
Left deltoid	Normal
Left pronator teres	Normal
Left extensor digitorum communis	Normal

SNAP: Sensory nerve action potential, CMAP: Compound muscle action potential, EMG: Electromyogram

DISCUSSION

PTS is a rare brachial plexopathy famously described by Parsonage and Turner in 1948, but was first reported by Dreschfeld in 1886.[13,31] It commonly affects males more than females and has been reported in ages ranging from 3 months to 75 years, with peak incidence between the third and seventh decades.[23,27,29,41] 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,

Table 2: Summary of Parsonage–Turner syndrome cases associated with COVID-19 vaccination in the current literature.

Publication	Age	Sex	Vaccine	Vaccination site	Affected extremity	MRI	EMG/NCS findings	Treatment
Diaz-Segarra *et al.*, 2021[12]	35	Female	Pfizer-BioNtech	Right deltoid	Left upper	NA	Reduced SNAP of lateral antebrachial cutaneous nerve. Active demyelination of peripheral nerves with C5–6 root contributions.	High-dose prednisone
Mahajan *et al.*, 2021[28]	50	Male	Pfizer-BioNtech	Left deltoid	Left upper	Reported as normal	Decreased motor unit recruitment in the left first dorsal interosseus, extensor digitorum, extensor indicis, abductor digiti minimi, and flexor carpi ulnaris.	High-dose prednisone
Queler *et al.*, 2021[33]	49	Male	Pfizer-BioNtech	Right deltoid	Left upper	Abnormal	Normal at 9 days post symptom onset. Repeat at 8 weeks with severe denervation and no motor unit recruitment in pronator teres and flexor carpi radialis.	Prednisone taper
Queler *et al.*, 2021[33]	44	Male	ModernaTX	Left deltoid	Left upper	Normal at 24 days post symptom onset. Abnormal at 5 weeks.	Decreased motor unit recruitment in infraspinatus.	Gabapentin + physical therapy
Coffman *et al.*, 2021[9]	66	Female	Pfizer-BioNtech	Right deltoid	Right upper	NA	Mild chronic neuropathic changes in serratus anterior.	Physical therapy
Burillo *et al.*, 2021[7]	38	Male	AstraZeneca	Not reported	Left upper	Mild left subacromial tendinopathy, otherwise reported as normal.	Decreased action potential amplitude in the left axillary, musculocutaneous, median, and radial nerves. Fibrillations and positive waves in extensor digitorum communis, abductor digiti minimi, first dorsal interosseous, and abductor pollicis brevis.	IV methylprednisolone followed by high-dose prednisone
Kim *et al.*, 2021[24]	45	Female	AstraZeneca	Left deltoid	Left lower	Initial with mild L4–5 disc protrusion, otherwise reported as normal. One month post onset abnormal.	Initial NCS normal. Initial EMG with decreased motor unit recruitment. One month post onset with denervation in the left vastus medialis and iliopsoas, decreased motor unit recruitment in the left leg, axonal and demyelinating lumbosacral plexopathy.	High-dose prednisolone
Koh *et al.*, 2021[26]	50	Male	Pfizer-BioNtech	Right deltoid	Right upper	Abnormal	Reported as normal at 10 and 31 days post onset.	Corticosteroids

(Contd...)

Table 2: *(Continued).*

Publication	Age	Sex	Vaccine	Vaccination site	Affected extremity	MRI	EMG/NCS findings	Treatment
Koh *et al.*, 2021[26]	44	Male	Pfizer-BioNtech	Left deltoid	Right upper	Abnormal	Brachial plexus lower trunk involvement.	Not reported, did not receive steroids.
Koh *et al.*, 2021[26]	58	Male	ModernaTX	Left deltoid	Left upper	Abnormal	Brachial plexus lower trunk involvement.	Corticosteroids
Vitturi *et al.*, 2021[45]	51	Male	AstraZeneca	Left deltoid	Left upper	NA	Reinnervation of deltoid, biceps brachii, triceps brachii, infraspinatus, extensor pollicis longus, extensor pollicis brevis, first interosseous. Decreased action potential amplitude of the left axillary nerve.	NSAID, pregabalin + physical therapy
Flikkema and Brossy, 2021[17]	43	Male	Pfizer-BioNtech	Right deltoid	Right upper	Abnormal	NA	NA

EMG: Electromyogram, NCS: Nerve conduction study, SNAP: Sensory nerve action potential

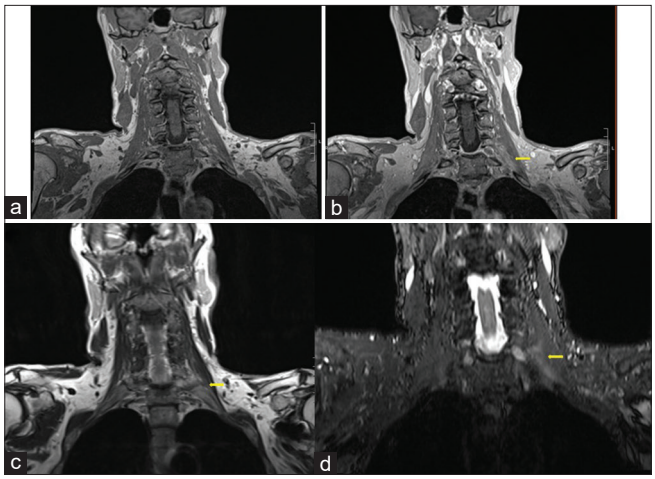

Figure 1: (a-d) Magnetic resonance imaging of the brachial plexus. T1 precontrast (a), T1 postcontrast (b), T2 (c), and STIR (d) demonstrating increased STIR signal, T2-weighted signal, and mild T1 postcontrast enhancement of the medial left scalene muscles along the inferior brachial plexus consistent with inflammatory changes and intramuscular edema (yellow arrows).

infraspinatus, serratus anterior, deltoid, and biceps).[19] Many patients will go on to develop sensory deficits including paresthesias and hypoesthesia and can also have muscle atrophy. Symptoms are usually self-limited and resolve slowly over several months, with complete resolution in majority of patients at 2–3 years.[19,41,43] 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.[25,43]

EMG, NCS, and MRI can help confirm the diagnosis of PTS.[14,34,38] Routine NCS can be normal but may have low amplitudes in affected segments; rarely, proximal conduction block can be demonstrated. EMG typically shows signs of denervation and reduced recruitment, at times even in clinically unaffected muscles, MRI is helpful and is evaluating for PTS as it can show diffuse T2-weighted signal changes secondary to denervation and edema of the muscles in the distribution of the affected brachial plexus nerves.[5,20,34] Similarly, in our case, EMG showed evidence of the left lower trunk denervation, supported by MRI findings of edema and inflammation of muscles along the inferior brachial plexus.

There is no consensus on the treatment of PTS, but generally involves conservative measures including analgesia, corticosteroid treatment, and physical therapy.[15] A proposed protocol for corticosteroid treatment is oral prednisone at 1 mg/kg/day for 1–2 weeks followed by a taper to off over an additional 1–2 weeks.[15,42]

At the time of writing, there are only nine other published case reports of PTS post-COVID vaccination [Table 2]. Our case is similar in that we find NCS/EMG changes in the affected extremity as well as similar MRI findings. A postulated mechanism of the development of PTS after COVID vaccination could be related to a reactive inflammatory response. Further studies investigating post-COVID vaccination PTS would be helpful in better understanding its pathogenesis. A limitation of a case report such as this includes lack of generalizability and inability to directly

determine a cause-and-effect relationship. However, as more case reports arise, we can establish a likely association. Given the limited nature of the course of PTS, the authors still feel that the benefit of the COVID-19 vaccination continues to highly outweigh any associated risks.

CONCLUSION

We present a case of post-COVID-19 vaccination PTS which improved following a corticosteroid taper and conservative measures. This is important as short-term shoulder soreness is a common post-COVID-19 vaccination, however, PTS should be in the differential if a patient continues to experience shoulder pain and develops weakness or sensory changes in the extremity.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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