ORIGINAL RESEARCH PAPER
Volume - 11 | Issue - 01 | January - 2022 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

MYELITIS ASSOCIATED WITH SARS-COV-2 VACCINATION

Orthopaedics
Dr Sapna Dhiman
Dr Sidhant Rana* *Corresponding Author

ABSTRACT

To review and report post COVID-19 vaccination associated transverse myelitis on MRI. With mass immunization against coronavirus disease , the safety concerns and adverse events requiring prompt evaluation are also emerging. Post vaccination is associated with several neurological manifestations such as stroke, Guillain-Barré syndrome, meningoencephalitis amongst others Other possible etiologies of her symptoms were ruled out, and she was treated successfully with steroids and plasma exchange. Here, we identify, reviewed and studied 3 cases of post COVID-19 vaccination patient diagnosed with acute transverse myelitis. A retrospective study of 3 cases diagnosed with post vaccination related

KEYWORDS

COVID-19, SARS-CoV-2, Transverse myelitis, MRI spine, Post vaccination myelitis.

INTRODUCTION

Coronavirus disease (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was proclaimed as a pandemic on March 11, 2020 by the World Health Organization (WHO). The predominant post-vaccination side effects are the nonspecific systemic symptoms, among which the neurological symptoms include dizziness, headache, pain, muscle spasms, myalgia and paresthesia. On some rare occasions, tremor, dysphonia, diplopia, tinnitus, seizures and reactivation of herpes zoster have been reported [2].. To combat the pandemic, vaccines for COVID-19 started developing at an exceptional rate. Many of them were approved for emergency use by different regulatory authorities like the Food and Drug Administration in the United States, the Medicines and Healthcare Products Regulatory Agency in the United Kingdom, and the European Medicines Agency after reviewing clinical efficacy data from phase 3 results, which subsequently led to mass vaccination worldwide. While it is an excellent achievement amid the pandemic, the vaccines still have to undergo post-marketing surveillance to monitor common and rare adverse events that need to be reported.

In our study we reported two cases of myelitis from India associated with a different, viral-vectored, recombinant ChAdOX1 nCoV-19 (Oxford/AstraZeneca, COVISHIELD™) vaccine.

CASE I

A 54-year-old previously healthy man presented to the medicine department with a three-day history of worsening back pain associated with numbness and weakness in the lower extremities for the last 5 days along with low-grade fever. Patient received the COVIDSHIELD COVID-19 immunization 10 days before presentation. Her family history was unremarkable for muscular disorders, multiple sclerosis, stroke, and any rheumatological disorders.

On physical examination, blood pressure was 124/80 mmHg, heart rate of 78 beats per minute, temperature of 100.1 Fahrenheit, respiratory rate of 15 breaths per minute, and oxygen saturation of 99% on room air. She was awake, alert, attentive, and oriented to time and person and did not have any nuchal rigidity, meningismus, or Lhermitte's sign. Her cranial nerves II-XII examination was grossly intact. She had normal muscle tone and strength in both upper and lower extremities with normal coordination and gait. Exaggerated (3+) deep tendon reflexes were noted in bilateral upper and lower extremities and positive Babinski signs were present bilaterally. Sensory examination was abnormal with decreased vibration in bilateral toes, and mild paresthesia in the neck and abdomen. The rest of the physical examination was unremarkable.

The patient underwent magnetic resonance imaging (MRI) of the cervical, thoracic spine, and lumbar spine with and without contrast, showing increased signal throughout the spinal cord extending from the C2-3 segment into dorsal spinal cord.

The patient's symptoms of numbness, urinary retention, and Bell's palsy resolved over 14 days of treatment with a pulse dose of steroids and plasmapheresis.(Figure 1).

CASE II

A 35-year-old male, with no prior comorbidities, received the first dose of COVISHIELD™ around 2 weeks back. On the 15th post-vaccination day, he presented to the primary referral unit with complaints of abnormal sensations in both lower limbs. Magnetic resonance imaging (MRI) of the spine, done on 13th post-vaccination day, showed an ovoid T2-hyperintense lesion in the dorsal aspect of spinal cord at D6 and D10 vertebral levels. A possibility of vaccine-associated demyelination was kept, and the patient was initiated on oral methylprednisolone (16 mg; 12 hourly). The patient took treatment for a week and was referred to tertiary care facility for further evaluation.

On examination, no motor deficit was found. Deep tendon jerks were exaggerated in the lower limbs with an extensor plantar response on the left side. Amongst sensations, sense of vibration was found to be impaired till manubrium sterni. MRI of the spine confirmed the presence of an ovoid T2-hyperintense lesion that showed mild to moderate peripheral enhancement on T1-gadolinium contrast administration. (Figure 2) MRI of the brain was normal. Cerebrospinal fluid examination (CSF) showed an increased protein (54 mg%; normal limit: 15–45 mg%) level; other parameters, including a panel to screen for infection, were normal.

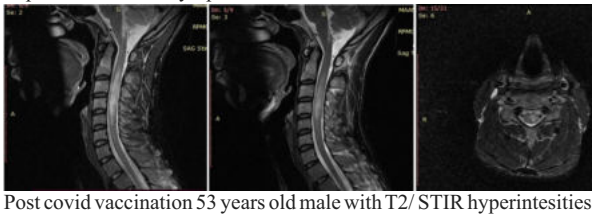
DISCUSSION

TM is a rare, acquired focal inflammatory disorder of the spinal cord in the absence of a compressive lesion. The most prevalent causes are demyelinating illnesses such as multiple sclerosis, neuromyelitis optica, infections, and vaccines. The cause of TM is unknown in up to 30% of instances, making it essential to distinguish idiopathic from disease-associated TM [2]. TM is clinically defined by the onset of acute or sub-acute motor, sensory, and autonomic dysfunction [3]. A recent literature review demonstrated cervical T2 signal anomaly in 44%, thoracic T2 signal abnormalities in 37%, and multifocal lesions in 5% of patients [4]. In our case, the MRI study demonstrated a long segment of increased signal throughout the spinal cord extending at least from C2-3 up to the thoracic spine and D6-D9 in second patient, suggestive of TM. The role played by the SARS-CoV-2 vaccine, in this case, was found to be significant after ruling out other causes such as connective tissue disorders, vasculitis, infectious etiology, and multiple sclerosis.

The idea of autoimmunity, where antibodies and T cells respond cross-reactively to central and peripheral nervous system neural epitopes, is emphasized in the hypothesis for vaccine-induced neuroinflammatory disease.The "Molecular Mimicry" concept emphasizes that vaccination might cause autoimmune disease by microbial pathogen proteins similar to human proteins . Only with the oral poliovirus vaccination, a pathogenic causal link for TM was identified. A common denominator among vaccines such as an adjuvant may play a role in the pathogenesis of TM. According to Vaccine Adverse Event Reporting System (VAERS), 254 (2.69%) of the 9442 adverse events following immunization recorded in association with Pfizer-BioNTech, Moderna, and Johnson & Johnson's COVID-19 vaccines were neurological, with nine cases of TM reported in VAERS .

Furthermore, two ATM serious adverse events were reported with the

ChAdOx1 nCoV-19 (recombinant) vaccine trials. The SARS-CoV-2 structural surface vector glycoprotein antigen (spike protein; nCoV-19) gene is included in a replication-deficient chimpanzee adenoviral ChAdOx1 vaccine (AZD1222). The antigen may also be present in the COVID-19 vaccination AZD1222, or its chimpanzee adenovirus adjuvant could be a possible trigger leading to ATM . Johnson & Johnson's COVID-19 vaccine incorporates the adenovirus, a prevalent cause of respiratory illnesses. The adenovirus's DNA is altered to form a critical component of the SARS-CoV-2 virus particle, to which the body responds with an immunological response . This could be a possible immunological trigger for ATM. To our knowledge, this is the first reported longitudinally extensive TM following the SARS-CoV-2 vaccine with the lesion of TM extending for more than three vertebral segments in length . Currently, there are no standard guidelines to treat TM secondary to the COVID-19 vaccine. Our patient was treated with intravenous steroids and plasma exchange and showed significant improvement in her symptoms .



Post covid vaccination 53 years old male with T2/ STIR hyperintesities in cervicodorsal cord with increased bulk of cords s/o post vaccination induced myelitis

CONCLUSIONS

The COVID-19 vaccines were approved for emergency use based on phase 3 clinical efficacy data and they have to go through post-marketing surveillance. As mass immunization continues across the world, adverse events are expected to be increasingly reported. Numerous COVID-19 vaccine-related adverse events involving the nervous system were described in the available literature; however, TM and Bell's palsy have not been specifically reported. The physicians should be aware of this adverse effect after Johnson and Johnson's COVID-19 vaccination and maintain a high index of suspicion in patients coming with typical symptoms of TM after receiving the vaccine, report it, and treat it immediately.

REFERENCES

1. Lim S, Park SM, Choi HS, Kim DK, Kim HB, Yang BG, Lee JK. Transverse myelitis after measles and rubella vaccination. *J Paediatr Child Health*. 2004;40(9–10):583–584. doi: 10.1111/j.1440-1754.2004.00470.
2. Kozic D, Turkulov V, Bjelan M, Petrovic K, Popovic-Petrovic S, Vanhoenacker FM. Extensive myelitis after oral polio vaccination: MRI features. *JBR-BTR*. 2014;97(6):358–360. doi: 10.5334/jbr-btr.134.
3. Matsui M, Kawano H, Matsukura M, Otani Y, Miike T. Acute transverse myelitis after Japanese B encephalitis vaccination in a 4-year-old girl. *Brain Dev*. 2002;24(3):187–189. doi: 10.1016/s0387-7604(02)00020-7.
4. Fonseca LF, Noce TR, Teixeira ML, Teixeira AL, Jr, Lana-Peixoto MA. Early-onset acute transverse myelitis following hepatitis B vaccination and respiratory infection: case report. *Arq Neuropsiquiatr*. 2003;61(2A):265–268. doi: 10.1590/s0004-282x2003000200020.