Case Series

Quadriparesis with different diagnoses after COVID-19 vaccination: Case series and literature review

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Abstract

Following vaccination with adenoviral vector-based ChAdOx1 nCoV-19, serious neurological adverse events have been reported. Here we report two cases who presented with quadriparesis following the adenoviral vector-based ChAdOx1 nCoV-19 vaccine. A 55-year-old male patient presented with quadriparesis after 8 days of the second dose of ChAdOx1 nCoV-19 vaccination. Imaging showed features of stroke with right basilar artery thrombosis; he was started on anticoagulation following which the patient's neurological status improved and he was discharged during the 7th week of hospital stay. A 19-year-old male patient presented with quadriparesis after 16 days of the first dose of ChAdOx1 nCoV-19 vaccination. Cerebral spinal fluid and nerve conduction study was suggestive of Guillain–Barre syndrome (GBS). Two doses of intravenous immunoglobulin were given, following which the patient's neurological status improved and he was discharged in the 11th week of his hospital stay. Awareness of neurological adverse effects and emphasis on the underlying mechanism of vaccine-induced thrombotic thrombocytopenia (VITT) and molecular mimicry in patients presenting with quadriparesis following ChAdOx1 nCoV-19 vaccination is important.

Keywords: ChAdOx1 nCoV-19 vaccine, Guillain–Barre syndrome, molecular mimicry, quadriparesis, stroke, vaccine-induced thrombotic thrombocytopenia

Introduction

Since the outbreak of coronavirus 2 (SARS-CoV-2), it had become challenging for healthcare services worldwide and it has been considered a global public threat. Due to the high mortality and global burden of COVID-19 infection across the world, the regulatory authority granted emergency approval of vaccines during the clinical trial.[1] Four major vaccine types have been explored for COVID-19 adenoviral vector based vaccines, mRNA-based vaccines, protein-based vaccines, and inactivated viruses. Following the use of vaccines, there has been a reduction in infection, transmissions, hospitalizations, and deaths related to COVID-19.[1,2] However various adverse events following immunization (AEFI) have been reported, AEFI is any untoward occurrence that follows immunization and does not necessarily have a causal relationship with the use of the vaccine.[3] With the use of the adenoviral vector-based ChAdOx1 nCoV-19 vaccine, neurological conditions such as transverse myelitis, cerebral venous sinus thrombosis, Guillain–Barre syndrome (GBS), Bell's palsy, acute demyelinating encephalopathy myelitis, and stroke have been reported.[3,4]

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Received: 21-11-2022 **Revised:** 29-03-2023
Accepted: 31-03-2023 **Published:** 29-08-2023

Access this article online

Quick Response Code:

Website: http://journals.lww.com/JFMPC

DOI: 10.4103/jfmpc.jfmpc_2274_22

How to cite this article: Wali S, Gutte S, Yadav S, Gurjar M, Paliwal VK, Singh V, *et al*. Quadriparesis with different diagnoses after COVID-19 vaccination: Case series and literature review. J Family Med Prim Care 2023;12:1724-9.

Here we present two cases who presented with quadriparesis following ChAdOx1 nCoV-19 vaccination.

Case Details

Case 1

A 55-year-old male patient was hospitalized with complaints of giddiness for 4 days and the onset of weakness in all four limbs with altered sensorium for 1 day. He had no history of any comorbidity, was a non-smoker, and was non-alcoholic with no previous episodes of transient ischemic attacks. He was vaccinated with a second dose of adenoviral vector-based ChAdOx1 nCov-19 vaccine (8 days before the onset of first symptoms). After hospitalization (day of illness-1, DOI-1), immediate intubation was performed for airway protection. His neurological examination revealed blinking of his eyes spontaneously, normal cranial nerves, and a motor power of 0/5. Modified medical research council scale (MRC scale) in all four limbs, deep tendon reflex of +2, and mute plantar. Magnetic resonance imaging (MRI) brain (DOI-4) revealed acute infarct in the pons and bilateral cerebellar hemisphere. Antiplatelets (aspirin 150 mg once a day) were started. Because of no improvement in neurological status, a repeat MRI brain was performed on DOI-16 to rule out a hemorrhagic transformation of infarcts. Findings of the imaging were subacute infarcts in the pons, bilateral middle cerebellar peduncles, and left cerebral hemisphere with a thrombosed basilar artery [Figure 1]. Laboratory parameters showed elevated D-dimer levels of 4500 ng/mL (reference value <500 ng/mL normal) with a normal platelet count of 2×10^9/L (reference value 1.5–4 $\times 10^9$) and serum fibrinogen levels of 572 mg/dL (reference value <200 mg/dL). Further workup for stroke was performed. Lipid profile, homocysteine levels, and auto-immune work-up (anti-nuclear antibody, complement levels) were normal. Echocardiography showed normal left ventricle function with no evidence of left atrial clot, and carotid Doppler showed normal carotid vessels. Because of ischemic stroke and basilar artery thrombosis, the anti-platelet agent continued and therapeutic anticoagulation with dalteparin 5000 IU subcutaneously twice a day was started. Over the next 3 weeks, he showed gradual improvement in motor power (3/5 in upper limbs and 2/5 in lower limbs–MRC scale) and was weaned off from mechanical ventilation and shifted to the neurology ward in the 5th week of the ICU stay. Further course of in ward anticoagulants and supportive treatment continued, and in the 7th week, the patient was discharged from the hospital. The patient was conscious and oriented with a power of 4/5 (MRC scale) in both upper limbs, and the tracheostomy tube was removed. He was taking an oral diet and doing minimal bedside mobilization.

Case 2

A 19-year-old male patient was hospitalized for complaints of acute-onset paraesthesia and progressive weakness in both lower limbs for 4 days and difficulty in speech and swallowing for 1 day. He had no history of comorbidities or preceding viral/bacterial infection, he had received the first dose of the adenoviral vector-based ChAdOx1 nCov-19 vaccine (16 days before the onset of first symptoms). After hospitalization (DOL-1), he required intubation because of the pooling of oral secretions and respiratory distress. Clinical examination revealed lower motor neuron involvement presenting as bifacial weakness-House–Brackmann grade VI, severe neck muscle weakness, and flaccid areflexic quadriparesis with prominent proximal upper and lower limb weakness 0/5 (MRC scale), pin-prick sensation was distally reduced in both lower limbs with associated autonomic instability in the form of tachycardia and hypertension. MRI of the brain and spine (DOI-5) showed normal brain parenchyma with thickened nerve roots [Figure 2]. With clinical and radiological suspicion of GBS, a cerebrospinal fluid study was performed (DOI-6) that showed albumin–cytologic dissociation (protein 1.14 g/L and nil cell), and a nerve conduction study (DOI-7) revealed bilateral motor nerve axonal neuropathy with the demyelinating pattern. Immunoglobulin (IVIG) therapy was started (DOI-7) at a dose of 2 g/kg over 5 days. With no significant improvement in neurological status, an IVIG dose of 2 g/kg over 5 days was repeated in the 5th week of illness. The patient showed gradual improvement over the next 4 weeks and was weaned from mechanical ventilation during the 9th week of illness. At discharge in the 11th week of illness, the patient had a residual facial weakness-House–Brackmann grade II, motor power of 4/5 (MRC scale) in upper and lower limbs, and was taking an oral diet.

Discussion

The ChAdOx1 nCoV-19 vaccine is a recombinant adenoviral vector-based vaccine that encodes SARS-CoV2 spike protein. Adenovirus vector enters the cells and translocates the spike

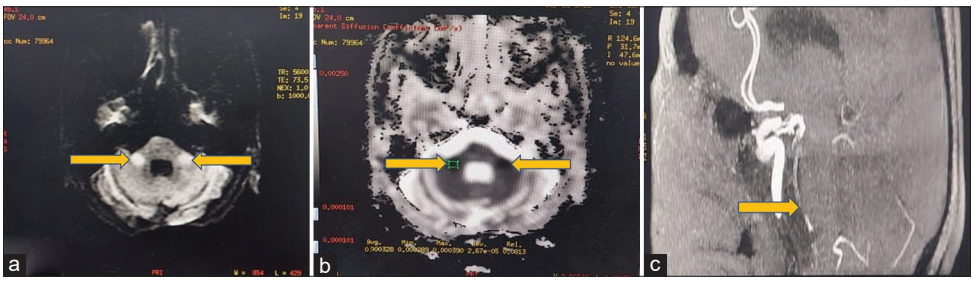

Figure 1: Magnetic resonance images brain. (a) Diffusion-weighted image–Arrow head showing hyperintensity in the bilateral cerebral peduncle, (b) Arrowhead showing apparent diffusion coefficient–Hyper-intensity in the bilateral cerebral peduncle, (c) Time of flight-filling defect in basilar artery

protein, which is recognized by the host immune system to produce antibodies against SARS-CoV2.[3] To date, various serious neurological AEFI have been reported including stroke and GBS following vaccination with the ChAdOx1 vaccine.[4] Both patients were male with no co-morbidities and presented with quadriparesis within 3 weeks of immunization with the ChAdOx1 vaccine. However, we were unable to trace the vaccine lot number due to the ongoing mass vaccination drive during the pandemic crisis.

Cerebrovascular adverse events such as cerebral venous thrombosis, arterial stroke, and hemorrhage have been reported following the use of the adenoviral-based vaccine [Table 1].[5,6] The mechanism proposed is vaccine-induced thrombotic thrombocytopenia (VITT), which is similar to heparin-induced thrombocytopenia, where cationic PF-4 and anionic-free DNA present in recombinant adenoviral vaccine react, resulting in the formation of antibodies that act on platelets leading to destruction. Hence, a prothrombotic state is formed.[6,7] Criteria of VITT are the onset of symptoms 4–24 days after vaccination, thrombocytopenia, and markedly elevated D-dimer levels.[8] In our patient, the constellation of symptoms within 3 weeks post-vaccination, and evident thrombosis in the basilar artery with raised D-dimer levels prompted consideration of VITT as a diagnosis. Concerning coagulation studies, patients with VITT have significantly elevated D-dimer with low to normal fibrinogen levels; however, in patients with a platelet count of 1–2.0 × 10^9, VITT cannot be excluded and further coagulation studies are required to rule out. The presence of thrombocytopenia elevated D-dimer and low fibrinogen should guide further evaluation for confirmation of VITT by enzyme linked immunosorbent assay (ELISA) and platelet activation tests such as serotonin release assay (SRA) or classical heparin-induced platelet activation test (HIPA).[5,9] Treatment of suspected cases of VIIT is high-dose IVIG as it inhibits the action of platelet-activating anti-PF4 antibodies by blocking platelet Fc receptors, hence increasing the platelet count and decreasing hypercoagulability. In our patient, low molecular weight heparin was used; however, the use of non-heparin anticoagulants such as direct oral Xa inhibitors, direct thrombin inhibitors, and fondaparinux are preferred where similarities in the mechanism of HIT and VITT have led to this recommendation. However, it is shown that the use of low molecular weight heparin has shown improvement in certain patients; hence, further studies are required as the use of non-heparin anticoagulants is associated with bleeding risk.[5] Though detection of PF-4/heparin antibodies by ELISA or positive platelet activation essay is required to confirm the diagnosis, treatment should not be delayed. However, the cause-and-effect relationship of VITT is not clearly established as 5–7% of blood donors have anti-PF4/heparin antibodies, and also thrombosis might be the result of SARS-CoV-2 infection concomitant to vaccination.[5,6,8] Also, all reported patients are to be tested for immune and systemic conditions leading to complement pathway activation, inflammation, and coagulation.[8,9]

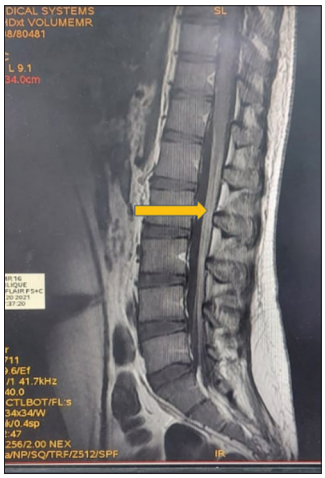

Figure 2: Magnetic resonance image of the lumbosacral spine. Arrowhead showing post-contrast sagittal T1 weighted image–Enhancement around cauda equina nerve roots

GBS is a post-infectious disorder of peripheral nerves, manifesting as lower motor neuron-type sensor motor quadriparesis, most

Table 1: Clinical, imaging, and treatment details of patients who developed stroke after COVID-19 vaccination

Author	Age/ sex	Type of vaccine	Clinical presentation (onset after vaccination)	Neuroimaging	Treatment
Bayas *et al.*[6]	55/f	ChAdOx 1nCoV-19	Flu-like illness, diplopia, vision loss, right-sided hemiparesis, aphasia, focal seizures (D-10)	MRI: ischemic stroke-left middle cerebral artery (MCA) territory	IV steroids and anticoagulants
Al-Mayhani *et al.*[7]	35/f *	ChAdOx 1nCoV-19	Left face, arm, and leg weakness (D-11)	CT angiography (CT-A): occlusion of the right middle cerebral artery (MCA) with extensive ischaemia	IVIG, plasmapheresis, anticoagulants
	37/f		Diffuse headache, left visual field loss, confusion, and left arm weakness (D-12)	CT A: occlusion of both internal carotid arteries	IVIG, IV steroids, and anticoagulants
	43/f		Dysphasia (D-21)	MRI brain: acute left frontal and insular infarct	IVIG and anticoagulants
Blauenfeldt *et al.*[8]	60/m*	mRNA	Intractable abdominal pain (D-7), CT revealed bilateral adrenal hemorrhages. On the following day, developed a right-sided ischemic stroke	Angiography showed occlusion of the right internal carotid artery	Palliative care
Walter *et al.*[9]	31/m	ChAdOx1 nCoV-19	Acute headache, aphasia, and hemiparesis (D-8)	MRI brain: ischemia of left MCA territory due to left MCA occlusion	Thrombolysis

(*)-Patient expired, IVIG–intravenous immunoglobulin

Table 2: Clinical, imaging, and treatment details of patients who developed GBS following COVID-19 vaccination

Author	Age/ sex	Comorbidities	Type of vaccine	Clinical presentation (onset after vaccination)	Neuroimaging	Treatment
GBS with respiratory failure						
Maramattom *et al.*[3]	43/f	Not mentioned	ChAdOx	Quadriparesis with facial diplegia (D-10)	-	IVIG
	67/f		1nCoV-19	Distal limb paraesthesia, facial diplegia, and dysphagia (D-14)	MRI brain: Normal	IVIG
	53/f			Bilateral distal upper and lower limb weakness with facial diplegia (D-11)	MRI brain: Normal	IVIG
	70/f			Bilateral distal upper and lower limb weakness with facial diplegia (D-11)	-	-
	69/f			Upper and lower limb weakness with facial diplegia (D-13)	-	-
Wai *et al.*[10]	51/m	NSTEMI	ChAdOx	Bifacial weakness, dysphagia with respiratory failure (D-14)		IVIG
	65/f	None	1nCoV-19	Dysphagia, diplopia, respiratory failure (D-7)		IVIG
Hasan *et al.*[11]	62/f		ChAdOx 1nCoV-19	Bilateral lower limb weakness (D-11)	MRI spine-normal	IVIG
GBS without respiratory failure						
Allen *et al.*[12]	54/m	Nil	ChAdOx	Dysthesia over hands and feet with bifacial weakness (D-16)	Contrast-enhanced (CE) MRI brain: enhanced facial nerves	Oral steroids
	20/m	Ulcerative colitis	1nCoV-19			Oral steroids
	57/m	Asthma, Osteoarthritis Hypertension (HTN)		Dysthesia over distal lower limbs with bifacial weakness (D-26)	MRI brain: normal	IVIG
	55/m			Dysarthria and facial weakness, distal dysthesia in feet (D-21)	MRI brain: normal	-
				Bilateral thigh paraesthesia, facial diplegia (D-29)	CE MRI brain: Enhanced facial nerves	
Finsterer[13]	32/m	AIDP 14 years back-recovered	Vector based vaccine	Paraesthesia in both foot soles and hands followed by dysphagia (D-8)	MRI brain: white matter hyperintensities	IVIG and plasmapheresis
Ogbebor *et al.*[14]	86/f	HTN, Rheumatoid arthritis, Ductal carcinoma *in situ* of the breast	mRNA	Weakness in bilateral lower limbs (D-1)	MRI spine: no cord signals	IVIG
Waheed *et al.*[15]	82/f	Nil	mRNA	Difficulty in walking (D-14)	MRI spine: enhanced cauda equina nerve roots	IVIG
Maramattom *et al.*[3]	68/f	Not mentioned	ChAdOx	Bilateral upper and lower limb weakness (D-14)	MRI brain: normal	Not mentioned
	60/f	Not mentioned	1nCoV-19	Bilateral upper and lower distal limb weakness with facial diplegia (D-12)	Not mentioned	Not mentioned
Patel *et al.*[16]	37/m	Not mentioned		Back pain, new onset distal paraesthesia, progressive ascending muscle weakness (3rd week)	MRI spine: thickened cauda equina nerve roots	IVIG
Theuriet *et al.*[17]	72/m			Areflexic quadriparesis with facial diplegia (3rd week)	NM	IVIG
Nasuelli *et al.*[18]	59/m			Distal paraesthesia and postural instability (D-15)	MRI brain and MRI spine: unremarkable	IVIG
Mckean *et al.*[19]	48/m	Dyslipidaemia		Bilateral lower limb weakness with facial diplegia (D-10)	MRI brain: unremarkable	IVIG and oral prednisolone
Bonifacio *et al.*[20]	43/m	-		Bilateral facial weakness with paraesthesia, a variant of Guillain–Barre syndrome (1 week, 11 days, 7 days, 12 days, 8 days)	MRI brain: contrast enhancement along a facial nerve in three patients	IVIG was given to two patients
	51/m	-				
	53/m	-				
	66/m	-				
	71/f	-				

(*)-Patient expired, AIDP-Acute inflammatory demyelinating polyneuropathy, GBS-Guillain–Barre syndrome HTN-Hypertension, IVIG-Intravenous immunoglobulin, NSTEMI-Non–ST-elevation myocardial infarction

commonly following a preceding bacterial or viral infection.[10] Post-infectious GBS onset led to a theory of molecular mimicry where certain infections such as *Campylobacter jejuni* caused the formation of antibodies that cross-react with gangliosides, which are part of peripheral nerves.[11-13] The occurrence of GBS in the general population is rare with incidence ranging from 0.8 to 1.9 per lac worldwide. Historically, following influenza, polio, meningococcal, and rabies vaccination, there was a surge in GBS cases but the association was only defined with the influenza vaccine where an additional one to two GBS cases per 1 million

influenza vaccines were administered occurred.[14] Following the COVID-19 pandemic and mass vaccination, a few case reports suggested a causal association between GBS and COVID-19 vaccination [Table 2]. Also, a large population-based study of 32 million people found that there was an increased risk of admission for GBS following adenoviral vector-based vaccines compared to mRNA vaccines.[13,15-17] Our patient presented with GBS on the 16th day following the adenoviral vector-based vaccine and there was no preceding respiratory or gastrointestinal infection, a usual trigger factor. A possible mechanism of GBS following the vaccination is related to the molecular mimicry theory. The ChAdOx1 vaccine produces antibodies and T-cells, which provide immunity against COVID-19 infection. It is postulated that this response leads to the action of antibodies or T cells against myelin sheath or axon proteins of peripheral nerves, resulting in immune-mediated damage and presentation of GBS.[18,19] However, the presence of asymptomatic infection as a trigger cannot be ruled out but our patient had received vaccination before developing symptoms of GBS, and various cases are reported in literature where GBS occurred weeks after vaccination.[16,17,20] Hence, we attribute this association to vaccine-induced GBS. However, this association is temporal; hence, further studies are required. These two cases highlight that following the ChAdOx1 nCoV-19 vaccination, various serious neurological adverse events such as stroke and GBS can occur. In patients presenting with stroke, the concept of vaccine-induced thrombotic thrombocytopenia is the most likely cause; hence, it mandates early workup for thrombosis and access to treatment such as IVIG and anticoagulants. The theory of molecular mimicry explains the occurrence of GBS following vaccination but further studies are required to confirm the causal association.

Conclusion

This manuscript highlights that the history of recent COVID-19 vaccination (within 3 weeks) is of paramount importance for primary care physicians to identify associated complications, which will further guide early diagnostic workup, management, and hence outcome.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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