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Guillain-Barré Syndrome After COVID19 Vaccination: A Single -Center Study in Tunisia and 1 Year Follow-Up

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Abstract

Introduction: After the beginning of the COVID-19 pandemic, several research teams looked for developing vaccines against SARS-CoV-2 and a mass vaccination campaign started worldwide. Since then, few neurological side effects of COVID-19 vaccination were reported mainly demyelinating diseases. We aimed to describe the clinical features of Guillain-Barré Syndrome (GBS) following COVID-19 vaccination and follow-up in order to identify the impact of the COVID-19 vaccine side effects on peripheral nerves.

Material and Methods: This is a single-center retrospective study including patients who developed GBS after a COVID-19 vaccination (≤30 days). The clinical, laboratory, electroneuromyogram (ENMG) characteristics and follow-up data were collected and analyzed.

Results: We collected 8 patients (2 males and 6 females) with no history of COVID-19 infection. Neurological symptoms suggestive of GBS occurred at a mean time of 10 days following the COVID-19 vaccination. All patients developed a mild phenotype with a progressively ascending weakness and paresthesia, 3 patients had autonomic disturbances but none needed ventilatory assistance. Regarding Cerebrospinal fluid (CSF) findings, classical albumin-cytological dissociation was detected in 6 patients. On ENMG, 7 patients fulfilled the electrophysiological criteria of a typical acute inflammatory demyelinating neuropathy (AIDP). Our cases were given intravenous immunoglobulin with a good outcome within a few days. After 1 year of follow-up, 6 patients showed full recovery.

Conclusions: Although the causal relation between COVID-19 vaccination and GBS is yet not confirmed, the GBS following the COVID-19 vaccination appears to be quite gentle and recovery seems to be rapid and complete.

Keywords: *Guillain-Barré syndrome, COVID19 vaccine, SARS-CoV-2 infection, Adverse events, Follow-up*

1. Introduction

In 2020, The world faced the COVID-19 pandemic [1]. Within less than 12 months after the beginning of the pandemic, several research teams rose to the challenge and developed vaccines that protected from this disease. Several vaccines were approved by the Food and Drug Administration (FDA) and a mass vaccination campaign was processed worldwide [2]. Since then, many side effects related to vaccines were reported. Among them, guillain-barré syndrome (GBS), is increasingly described following COVID-19 vaccinations.

Herein, we report cases of GBS following the use of COVID-19 vaccination. Our aim is to investigate the clinical features, clinical course and long-term outcome of post-vaccine GBS, in order to identify the clinical impact of COVID-19 vaccines side effects on peripheral nerves.

2. Material and Methods

We retrospectively reviewed the medical records of patients with GBS, who attended the Mongi Ben Hmida National Institute of Neurology in Tunis between March and November 2021. Among them, we selected those who had a vaccination against COVID19 during the month prior to the occurrence of GBS. We excluded patients who had a history of COVID19 infection or any other infectious episode closely before this neurological event. All patients had cerebrospinal fluid (CSF) analysis and electroneuromyography (ENMG) examination. The diagnosis of GBS was confirmed according to the National Institute of Neurological Disorders and Stroke (NINDS) criteria [3]. The clinical, CSF and ENMG characteristics, clinical course and outcome were assessed. Patients were examined by neurologists at baseline, at 3 months and 1-year follow-up.

3. Results

Eight patients were included (2 males and 6 females) with a mean age of 54 years old (between 36 and 69). Comorbid history was limited to high blood pressure and dyslipidemia, all patients denied any history of nervous system disease or any other systemic disease.

3.1 COVID19 vaccines

The time between the last COVID-19 vaccination and the onset of neurological symptoms ranged between 3 and 24 days (mean: 10 days). Among them, 4 received their first injection of Oxford/AstraZeneca (ChAdOx1-S recombinant vaccine), 3 had their second Pfizer-BioNTech injection (BNT162b2) and one patient received his sole injection of Janssen (Ad26.COV2.S).

3.2 Clinical features of GBS spectrum

At the onset, all patients experienced weakness in distal lower limbs associated with distal paresthesia. Cranial nerve involvement was not described initially. During the disease course, an ascending evolution of clinical symptoms was detected in 5 patients. Common clinical manifestations included motor deficits with variable severity. In fact, 5 patients had a flaccid tetraparesis (MRC score ≤ 42), 2 had a flaccid paraparesis (MRC score= 48), and one patient had a slight motor deficit in distal limbs (MRC score =52). Facial nerve paralysis was detected in 3 patients and no one showed oculomotor nerve impairment. Generalized areflexia was found in 5 patients and only 3 patients had gait ataxia. Two patients showed bulbar involvement with dysphagia and dysphonia, while no patient did experience any respiratory distress and none needed ventilatory assistance. Autonomic disturbances were reported in 3 patients (sinusal tachycardia in 2 patients and urinary disturbances in 1 patient) and 2 patients had hyponatremia.

3.3 Results of CSF, electrophysiological and neuroimaging investigations

Regarding CSF analysis, the classical albumin-cytological dissociation (cell count <5/ul with elevated CSF proteins) was detected in 6 patients with a mean CSF protein of 2,29 g/l (between 0,65 and 6,53g/l) and all patients had CSF cell count <5/ul (Table 1).

Detailed ENMG results were reviewed (Table 2). A pattern compatible with demyelinating polyradiculoneuropathy was observed in 3 patients, while axonal damage associated with a demyelinating pattern was found in 4 other patients and nerve conduction studies were normal in one patient.

Routine blood tests revealed mild leukocytosis in 4 patients. All patients had normal hemoglobin rate, normal d-dimer rate and no lymphopenia. C-reactive protein was elevated in 3 patients (>10mg/dL). Blood SARS-Cov-2 RNA was undetectable in all tested patients (5 patients). Extensive infectious workup including Cytomegalovirus (CMV), Epstein Bar Virus (EBV), Human Immunodeficiency Virus (HIV), Hepatitis B and C and *Campylobacter Jejuni* serology, was negative in all patients.

Cranial and spinal MRI scans were performed in 4 cases, they were normal in 3 cases and showed spinal nerve root enhancement in one case.

Table 1. summary of Guillain Barré syndrome characteristics in our patients post COVID19 vaccination.

Patients	P1	P2	P3	P4	P5	P6	P7	P8
Sex/age (years)	M/53	F/42	F/43	M/67	F/55	F/67	F/69	F/36
Medical -history	None	None	None	Dyslipidemia	None	High blood pressure	none	none
History of COVID	no	No	no	no	no	no	no	no
History of infection other than COVID (≤4 weeks)	no	No	no	no	no	no	no	no
Interval vaccine-GBS symptoms (days)	7	5	3	24	4	10	14	13
Type of vaccine/ dose number	Astrazeneca/1st	Astrazeneca/1st	Astrazeneca/1st	Astrazeneca/1st	Pfizer-BioNTech /2nd	Pfizer-BioN-Tech /2nd	Janssen/1	Pfizer-BioN-Tech /2nd
GBS symptoms	Flaccid tetraparesis, gait ataxia, paresthesia and generalized areflexia	distal weakness and paresthesia, facial diplegia, loss of Achillean tendon reflexes	Flaccid paraparesis, gait ataxia, paresthesia over 4 limbs and loss of deep tendon reflexes in lower limbs	Flaccid tetraparesis, paresthesia of hands and feet, facial diplegia and generalized areflexia	Flaccid tetraparesis, paresthesia in lower limbs, Gait ataxia, facial diplegia, dysphonia, dysphagia, generalized areflexia	Flaccid paraparesis, paresthesia of lower limbs and loss of deep tendon reflexes in lower limbs	Flaccid tetraparesis, paresthesia over 4 limbs, dysphagia and weak deep tendon reflexes in 4 limbs	Flaccid tetraparesis, paresthesia of hands and feet, generalized areflexia
MRC score at onset	36	52	48	40	38	48	36	42
Ascending course	yes	no	yes	yes	yes	no	yes	no
Progressive phase duration	20	20	30	20	20	5	9	17
Autonomic and electrolyte disturbance	no	no	no	Sinusal tachycardia, hyponatremia	no	Sinusal tachycardia, dysuria	hyponatremia	no
CSF findings (WBC/protein in g/l)	1/2.08	1/0.41	2/0.65	1/6.53	2/2.93	4/0.33	1/0.85	1/0.71
EMG findings	sensorimotor demyelinating PRN with secondary axonal damage,	sensorimotor demyelinating PRN	sensorimotor demyelinating PRN with secondary axonal damage,	sensorimotor demyelinating PRN	sensorimotor demyelinating PRN	sensorimotor demyelinating PRN	sensorimotor demyelinating PRN with secondary axonal damage	Normal nerve conduction studies
Electrophysiological variant	AIDP	AIDP	AIDP	AIDP	AIDP	AIDP	AIDP	-
BCC	normal	normal	normal	Leukocytosis (13800/mm3) thrombopenia (135000)	Mild leukocytosis (11500/ mm3)	Mild leukocytosis (10360/ mm3)	Mild leukocytosis (11700/ mm3)	normal
CRP	12	8	3	27	1	163	28	1
d-dimer rate	<500	<500	<500	<500	<500	<500	569	<500
Blood SARS-CoV-2 RNA	negative	negative	NA	negative	NP	negative	negative	NP
Brain + spine MRI	Spinal nerve roots enhancement	NA	NA	NA	normal	NA	normal	normal
Treatment	IVIg	IVIg	IVIg	IVIg	IVIg + pregabaline	IVIg	IVIg + pregabaline	IVIg + pregabaline
Invasive mechanical ventilation	no	no	no	no	no	No	no	no
GBS disability score at discharge	2	2	3	2	2	3	3	4
GBS disability score After 3 month follow-up	1	0	2	1	0	1	2	3
GBS disability score After 1 year follow-up	0	0	1	0	0	0	0	1

AIDP : auto-immune demyelinating polyradiculoneuropathy; BCC : blood cell count; CRP : C-reactive protein; CSF : cerebrospinal fluid ; F : female; GBS : guillain-barré syndrome; IVIg : intravenous immunoglobulin; M : male; MRC : medical research counsil muscle strength grading system; MRI : magnetic resonance imaging; NA : not available ; P : patient; PRN : polyradiculoneuropathy ;SARS-CoV-2 RNA : severe acute respiratory syndrome coronavirus 2 ribonucleic acid ; WBC : white blood cell ;

Table 2. Summary of electromyography and nerve conduction studies.

Patient	Normal value	P1		P2		P3		P4		P5		P6		P7		P8	
		L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R
Motor nerve conduction																	
DML (ms)																	
Median	≤3,8	11	11,9	12,9	15,9	17	19	9,5	13,6	11	12,9	2	2,8	8,9	11	7,25	7,46
Ulnar	≤3,2	9	9,1	2,4	2,6	9	2,8	4,5	4,6	7,8	8,9	3	4,3	6,9	7,3	1,83	2,00
Peroneal	≤5	11,5	10,3	4,9	3,1	N	N	12,5	11,7	5,7	N	4,7	3,7	A	21,8	5,02	4,25
Tibial	≤4,5	11,6	17,2	8,3	7,2	N	N	7,3	8,8	13	N	2,8	3,4	12,5	A	8,63	2,06
F wave (median)	25-30	A	A	30	18,6	12	N	55,6	53,1	47,6	31,6	30	27,1	37,4	N	N	27
F wave (ulnar)	25-30	A	A	25,2	25,2	N	N	45,5	36,5	N	29,7	30,2	29	N	N	N	N
F wave (tibial)	45-50	53,7	31,9	40,2	43,8	N	N	63	65	66	N	48,9	48,5	N	N	56	49
CMAP (mV)																	
Median wrist	≥6	2,3	3,2	1,5	0,5	0,5	2	1,5	1,1	0,6	0,8	9	6,2	1,5	1,8	7,3	10,6
Ulnar wrist	≥6	3,8	3,7	3,4	2,5	3	3,5	3,7	2	0,7	0,3	1	12,7	0,9	1,1	6,8	4,1
Ulnar below elbow		2,2	2,5	3,3	2,1	1,2	1,5	3,2	3,4	N	N	0,7	8,9	0,3	0,2	7	6,5
Ulnar above elbow		1,6	1,7	N	N	N	N	N	N	N	N	N	9,3	0,2	A	6,8	N
Peroneal	≥3	1	1,2	1,4	0,8	N	N	0,3	0,1	0,4	N	6,4	4,3	A	0,3	3,6	4,3
Tibial	≥6	1,3	0,4	1,6	1,7	N	N	0,5	0,3	0,6	N	3,4	3,4	0,3	A	12	6,5
MCV (m/s)																	
Median	≥45	16	12	52,8	50,1	35,3	34,2	23	34	60	N	60,1	48,8	63,7	37,1	56	50,6
Ulnar	≥45	18	17	86,6	85,7	55	89,2	49	39	N	N	71	93,4	37,8	15,2	56,2	61,9
Peroneal	≥42	N	N	N	N	N	N	19	20	N	N	N	N	0	N	N	N
Tibial	≥42	20	17	39,3	63,1	N	N	22	19	N	N	47,6	47,7	43	0	52,1	43,7
Sensory nerve conduction																	
SNAP (uV)																	
Ulnar	≥5	A	A	9,6	5,8	17	11,5	A	A	5,7	N	7,6	6,9	4,4	3,1	10,1	8,4
Median	≥15	A	A	3,2	2,4	20	20	A	A	1,2	N	3,4	25,2	6,6	3,9	24,9	15,7
Sural	≥10	A	A	N	N	N	N	9,4	15,3	N	N	10,2	5,9	N	N	10,8	5,5
musculocutaneous	≥6	A	A	2,3	2,8	N	N	N	N	N	N	4,6	7,9	2,2	2,8	10,9	11
SCV (m/s)																	
Ulnar	≥45	A	A	96,8	93,8	56	57	A	A	50	54	120	65,2	63,3	43,9	60,8	60,2
Median	≥45	A	A	22,1	23,6	43	45	A	A	89,7	80	70	72,9	31,7	31,2	61	59,8
Sural	≥40	A	A	N	N	N	N	156	236	N	N	62,5	59,9	N	N	46,2	40,3
Musculocutaneous	≥40	A	A	64	61,2	N	N	N	N	N	N	111	100	A	68,7	46,2	46,2

A : absent ; CMAP : compound muscle action potential ; DML : distal motor latency ; L : left ; MCV : motor nerve conduction velocity ; N : not performed ; P : patient ; R: right ; SCV : sensory nerve conduction velocity ; SNAP : sensory nerve action potential

3.4 Variants and diagnosis of GBS

According to the clinical presentation, all patients showed the classical sensorimotor variant of GBS. None of them had acute motor axonal neuropathy (AMAN), Miller-Fisher syndrome (MFS), or other subtypes. From electrophysiological point of view, 7 patients fulfilled the electrophysiological criteria of a typical acute inflammatory demyelinating neuropathy (AIDP) and one patient had normal nerve conduction studies.

3.5 Management of GBS and patients outcome

All patients presented to our clinic during the progressive phase of the disease. The mean duration of this phase was 17 days (between 5 and 30 days). After treatment with intravenous immunoglobulin, all patients showed significant clinical improvement of motor weakness within 5 days on average. Due to the persistent uncomfortable sensory symptoms, 3 patients needed treatment with pregabalin.

At 3 months follow-up, all patients showed significant improvement of their motor symptoms (8 patients) and their facial nerve paralysis (3 patients). Seven patients were able to walk without assistance within 2 months. However, 2 patients were still experiencing disturbing sensory symptoms in their hands and feet.

At 1 year follow-up, six patients fully recovered and their neurological examination revealed no motor nor sensory deficit in four limbs. While 2 patients were still having minor signs of neuropathy mainly in the lower limbs but both were capable of manual work and walking long distances unaided.

4. Discussion

GBS is an acute, immune-mediated polyradiculoneuropathy widely described in the world. It occurs in 0,4-4 cases per 100000 population per year [4,5]. This disease is preceded by infection in approximately 60% of patients. Rarely, GBS can occur following vaccination [5,6]. Here, we reported 8 cases of GBS following different types of COVID-19 vaccination with different mechanisms of action. Interestingly, none of our patients had experienced a recent infection prior GBS and the infectious investigation was negative. Eventually, we supposed that vaccines could possibly be responsible for GBS.

The GBS following COVID-19 vaccination was initially described in February 2021 by Waheed et al [7], in 82 years-old women 14 days after the first dose of Pfizer-BioNTech. Since then, several cases were reported in the literature mostly after the first dose of COVID-19 vaccination [8]. In our cohort, we described patients with GBS after the first dose of AstraZeneca vaccination and others after the second dose of Pfizer vaccination. It suggests that probably there is no difference between the transient immune changes caused by the first dose of the vaccine or the booster. Besides, our data suggest that the occurrence of post-COVID-19 vaccine GBS is more common in the middle-aged population, generally within 10 days after the vaccine administration. Interestingly, most patients were healthy before the GBS episode and had no history of COVID-19 infection, similar to cases described in the literature [9].

Compared with other types of GBS, patients from our cohort had a non-severe form of GBS, as they developed a classical demyelinating neuropathy with a mild phenotype, no need for ventilatory assistance was described and no deaths were reported. At short-term follow-up, patients showed a rapid recovery trend, they retrieved normal functioning within 1-year follow-up and no recurrence was found in the long run, in accordance with the review of Fernandez et al [10].

Regarding COVID-19 vaccination, the putative role of these vaccines in generating GBS is still debated and several hypotheses were proposed. In fact, GBS could result from the generation of host antibodies against SARS-CoV-2 spike protein which cross-reacts against peripheral nerve components. Less likely, a cross-reactivity to components of the adenovirus vector was also discussed [9]. Moreover, the SARS-CoV-2 spike protein can bind to sialic acid-containing glycoprotein and gangliosides on cell surfaces, increasing its viral transmissibility. Antibody cross-reactivity between the SARS-CoV-2 spike protein and peripheral nerve glycolipids may also be involved [11].

Even if these hypotheses plead for the relationship between COVID-19 vaccines and GBS, the actual causal link is yet to be proved. Indeed, it is inevitable that many sporadic cases of GBS caused by other non-evident factors can be temporally associated with COVID-19 vaccination. Therefore, although our patients had neurological symptoms suggestive of GBS that are temporally associated with COVID-19 vaccination, causality cannot be confirmed.

5. Conclusion

In conclusion, Although the causal relationship between COVID-19 vaccination and GBS is yet uncertain, post COVID-19 vaccine GBS seems to be non-serious, non-life-threatening and potentially has a mild phenotype and a rapid recuperation.

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Competing Interest

None declared under financial, general and institutional competing interests.

Ethical Approval Information

The authors attest that the ethical consent for publication of this article was obtained from all the patients.

Consent to Participate

The authors give consent to participate in the preparation and the submission of this manuscript

Consent for Publication

The authors declare that they take fully responsibility over decisions to submit the manuscript for publication.

Availability of Data and Material

The authors attest that they had full access to all case data, take fully responsibility for the accuracy of the data analysis, have authority over manuscript preparation and that all data used in this case are available.

Code Availability

Not applicable

Authors' Contributions

Dr Rania Zouari : was responsible for writing the manuscript, analysis and interpretation of data.

Dr Dina Ben Mohamed : provided the conception of the manuscript, analysis and interpretation of data

Dr Mohamed Zakaria Saeid: supplied the acquisition of data and revised the manuscript critically for better grammar and spelling content

Dr Fatma Nabli: revised the manuscript critically for important intellectual content.

Prof Samia Ben Sassi : revised the manuscript critically for important intellectual content and gave final approval of the version to be submitted.

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