International Journal of Research in Medical Sciences
Zafar S et al. Int J Res Med Sci. 2022 Jun;10(6):1364-1367
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20221495

Case Report

Guillian-Barre syndrome after COVID-19 infection: a case report

Sharjeel Zafar[1], Muhammad Waseem Abbas[2]*, Rukhsar Javaid[1]

[1]Department of Family Medicine, Tehsil Headquarter Hospital Sabzazar, Lahore, Pakistan
[2]Tehsil Headquarter Hospital, Jahanian, Khanewal, Pakistan

Received: 01 April 2022
Revised: 29 April 2022
Accepted: 06 May 2022

***Correspondence:**
Dr. Muhammad Waseem Abbas,
E-mail: wiz46221@outlook.com

ABSTRACT

Guillain-Barre syndrome (GBS) is an auto-immune disorder characterized by ascending motor weakness with hypo-reflexia or areflexia. In GBS molecular mimicry leads to the targeting of peripheral nerves. The treatment of GBS is mainly supportive, however, the definitive management of GBS includes IVIGs (intravenous immunoglobulins) and plasma exchange (plasmapheresis). We reported a case of 42 year old man who presented with weakness of both lower limbs associated with pain and numbness after recovering from COVID-19 infection. Clinical examination, radiological findings and lab results confirmed the diagnosis of GBS. He was admitted in the hospital and treated with intravenous immunoglobulins along with periodic cardiac, respiratory and neurological assessment. The patient showed a substantial response to intravenous immunoglobulins and resulted in a remarkable recovery with no residual motor or sensory deficit.

Keywords: Auto-immune, Guillain-Barre syndrome, COVID-19, Immunoglobulins, Reflexes

INTRODUCTION

GBS is an auto-immune disorder characterized by polyrediculoneuropathy with typical presentation of ascending motor weakness and hypo-reflexia or areflexia.[1] Almost 94% or patients suffering from GBS exhibit decreased motor strength. Raised protein levels in cerebrospinal fluid (CSF) and normal glucose level with no pleocytosis is diagnostic of GBS.[2]

In GBS molecular mimicry leads to the targeting of peripheral nerves. This aberrant molecular mimicry and auto-immune response is triggered by any viral or bacterial infection. The typical features of GBS include ascending motor weakness associated with decreased or absent deep tendon reflexes (DTRs). However, some patients may exhibit sensory or autonomic symptoms. The management of GBS is mainly supportive, which is comprised of rest, patient education, physiotherapy, nutritional support, avoidance of heavy physical activities and regular follow ups. The definitive management of GBS includes IVIGs and plasma exchange (plasmapheresis).[3]

CASE REPORT

We reported a case of 42 year old man, who presented in the outpatient department of medicine, THQ hospital, Jahanian, Khanewal, Pakistan. He complained of weakness of both lower limbs associated with pain and numbness. Weakness of both legs started 4 days ago, gradually worsened leading to inability of bearing weight. He stated no history of trauma, however, he was recently recovered from COVID-19 infection. The chest X-ray of the patient during COVID-19 infection is shown in Figure 1. Examinations revealed motor weakness in all muscle groups (gastrocnemius soleus, hamstrings and quadriceps) of both lower extremities with sensory loss in both feet extending up to knees. Deep tendon reflexes were also sluggish in both lower limbs. General physical

examination concluded no other neurological, autonomic or orthopedic findings. The other systemic examination (cardiovascular, respiratory, gastrointestinal and genitourinary) were completely normal. Furthermore, he was a healthy person with no comorbidities.

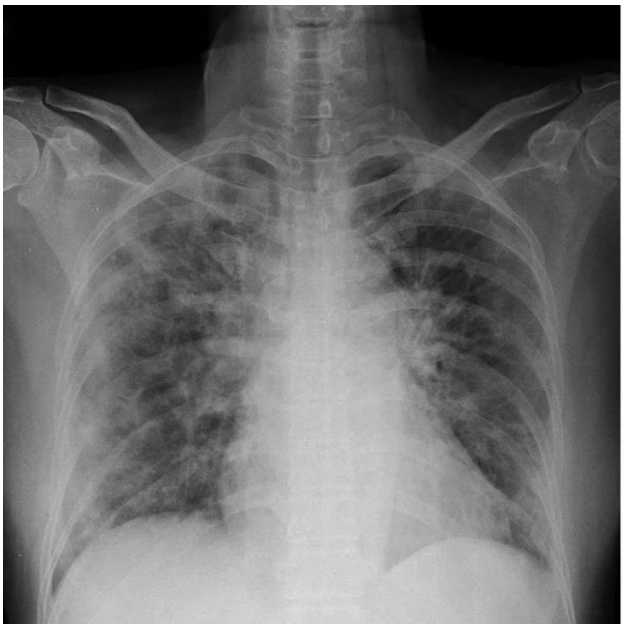

Figure 1: Chest X-ray showing bilateral lungs infiltrates suggestive of viral (COVID-19) infection.

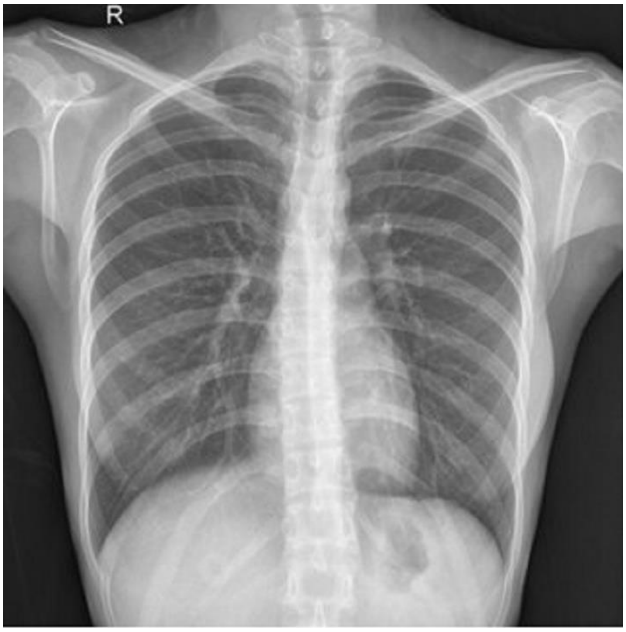

Figure 2: Chest X-ray showing no abnormal findings.

The patient was advised complete blood count along with peripheral film and chest X-ray. The complete blood profile showed the increased white cell count. This is explained in Table 1. This increased white cell count correlated with his recent COVID-19 infection. Furthermore, peripheral film and chest X-ray revealed no pathology. This is exhibited in Figure 2. Keeping in view of his recent respiratory tract infection, the provisional diagnosis of GBS was made and he was referred to tertiary care hospital (Nishtar hospital, Multan, Pakistan) for further evaluation and management.

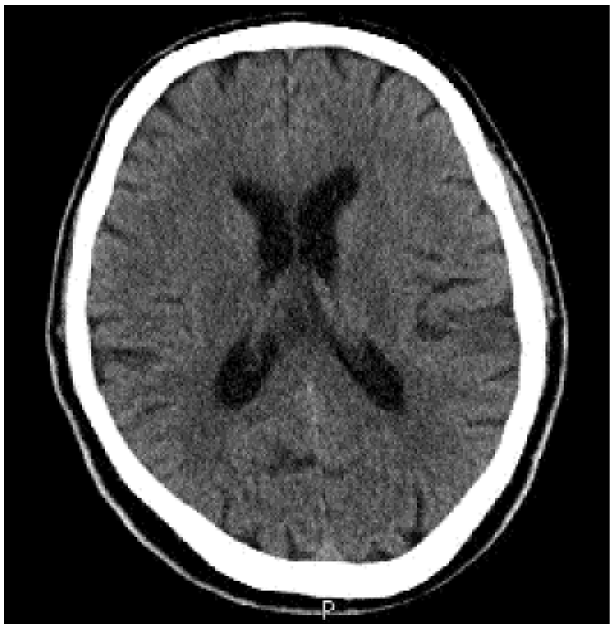

Figure 3: CT scan brain showing no abnormal findings.

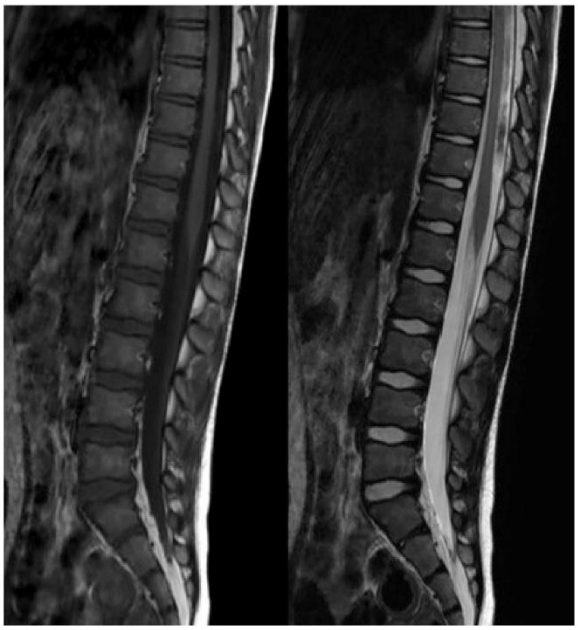

Figure 4: MRI spine revealing thickening of cauda equina.

The physician in tertiary care hospital advised CT scan brain, MRI spine, CSF analysis and serum electrolytes. CT scan of brain reported no abnormal finding, however, the MRI of spine revealed mild thickening of cauda equina. This is illustrated in Figure 3 and Figure 4

respectively. CSF analysis suggested normal cell count but raised protein level. Normal electrolytes ruled out hypokalemic periodic paralysis. The lab reports of serum electrolytes and CSF analysis are demonstrated in Table 1. Hence, the diagnosis of GBS was confirmed and physician admitted the patient and advised intravenous immunoglobulins along with periodic cardiac, respiratory and neurological assessment. After 4 days, the patient slowly improved and he was discharged after 15 days. The physician advised him supportive management which included rest, patient education, physiotherapy, nutritional support, avoidance of heavy physical activities and regular follow-ups with GP at THQ hospital Jahanian, Khanewal, Pakistan. After 2 months, the patient gained full motor strength with no sensory deficit.

Table 1: Lab reports of patient, complete blood profile, serum electrolytes and CSF analysis.

Investigations	Tests	Results	Reference value	Units
Complete blood profile	Hb	14	13-18	g/dl
	Total RBC	5.3	4.4-6.5	×1012/l
	HCT	41	35-52	%
	MCV	81	75-95	fl
	MCH	29	26-32	pg
	MCHC	32	30-35	g/dl
	Platelet count	344	150-400	×109/l
	WBC count (TLC)	13	4-11	×109/l
	Neutrophils	40.1	40.0-75.0	%
	Lymphocytes	51.3	20.0-50.0	%
	Monocytes	6.4	2.0-10.0	%
	Eosinophils	2.0	1.0-6.0	%
	Basophils	0.2	Less than 1.0	%
Serum electrolytes	Sodium	141	136-146	mmol/l
	Potassium	3.9	3.5-4.5	mmol/l
	Chloride	102	96-106	mmol/l
	Calcium	2.5	2.2-2.7	mmol/l
CSF analysis	Appearance	Clear		
	Color	Colorless		
	Glucose	70	45-80	mg/dl
	Leukocytes	2	0-5	/mm^3
	Total proteins	57	20-40	mg/dl
	Oligoclonal bands	Absent		

DISCUSSION

GBS is an acute autoimmune disorder characterized by the demyelination of peripheral nerves. The presenting features of the disease involved growing weakness in motor strength associated with hypo-reflexia or areflexia. Almost 50% cases of GBS followed the viral infection mainly upper respiratory and gastrointestinal infection. The clinical data suggested that 1/3rd of the patients suffering from GBS required respiratory support during the course of management. Furthermore, new management techniques like plasmapheresis and IVIGs had played a successful and significant role in the management of GBS.[4,5] In this case, the patient showed a substantial response to intravenous immunoglobulins and resulted in a remarkable recovery with no residual motor or sensory deficit. A similar case of GBS following COVID-19 infection was reported in Kuwait, who was treated with intravenous immunoglobulins and made a successful recovery.[6]

Researchers had reported other cases of GBS following COVID-19 infection. A study conducted in Italy, reported 4 patients of COVID-19 infection, who were diagnosed with GBS and developed progressive motor weakness and diminished deep tendon reflexes.[7] In this case, the patient had a positive history of COVID-19 infection 1 week prior to the onset of neurological symptoms. The exact mechanism of GBS following COVID-19 infection was not clear, however, scientists have suggested that various mechanisms were responsible for the development of neurological symptoms after COVID-19 infection. The COVID-19 virus can cause molecular mimicry with nerve antigens and can lead to neural damage. The virus can also bind to ACE2 receptors and make their way into glial and nerve cells leading to damage.[8-10] However, further researches should be carried out to find out the exact course of mechanism of disease.

CONCLUSION

We can conclude from this case report that any patient who has been suffering from COVID-19 or has recovered from COVID-19 infection, presenting with sensory or motor weakness must be evaluated for GBS. If confirmed

by investigations, then in addition to supportive treatment, intravenous immunoglobulins and plasmapheresis should be considered as management options in early stages of disease since they have shown remarkable results with complete recovery. However, further researches should be carried out to assess pathophysiology of GBS post COVID infection.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Dutta AK. Introduction to skull. In: Dutta AK, eds. Essentials of Human Anatomy Head and neck Part II. 3rd ed. Calcutta: Current Books International; 1999: 1-69.
2. Mwachaka PM, Hassanali J, Odula P. Sutural morphology of the pterion and asterion among adult Kenyans. Braz J Morphol Sci. 2009;26(1):4-7.
3. Singh R. Incidence of sutural bones at asterion in adult Indian skulls. Int J Morphol. 2012;30(3):1182-6.
4. Day JD, Kellog JX, Tschabitscher M, Fukushima T. Surface and superficial surgical anatomy of the posterolateral cranial base: significance for surgical planning and approach. Neurosurgery. 1996;38(6):79-84.
5. Uz A, Ugur HC, Tekdemir I. Is the asterion a reliable landmark for the lateral approach to posterior fossa? J Clin Neurosci. 2001;8(2):146-7.
6. Martinez F, Laxague A, Vida L, Prinzo H, Sgarbi N, Soria VR, et al. Topographic anatomy of the asterion. Neurocirgia. 2005;16(5):441-6.
7. Leon SG, Rodriguez AN, Avalos RM, Giron M, Omana REE, Lopez SG. Morphometric characteristics of the asterion and the posterolateral surface of the skull: relationship with dural venous sinuses and neurosurgical importance. Cir Cir. 2013;81(4):251-5.
8. Berry AC, Berry AJ. Epigenetic variation in the human cranium. J Anat. 1967;101(2):361-79.
9. Kellock WL, Parson PA. A comparison of the incidence of minor nonmetrical cranial variants in Australian aborigines with those of Melanesia and Polynesia. Am J Phys Anthropol. 1970;33(2):235-40.
10. Gumusburun E, Sevim A, Katkici U, Adiguzel E, Gulec E. A study of sutural bones in Anatolian Ottoman skulls. Int J Anthropol. 1997;12(2):43-8.
11. Saheb HS, Mavishettar GF, Thomas ST, Prasanna LC, Muralidhar LC, Muralidhar P. A study of sutural morphology of the pterion and asterion among human adult Indian skulls. Biomed Res. 2011;22(10):73-5.
12. Sudha R, Sridevi C, Ezhilarasi M. Anatomical variations in the formation of pterion and asterion in south Indian population. Int J Cur Res Rev. 2013;5(9):92-100.
13. Ucerler H, Govsa F. Asterion as asurgical landmark for the lateral cranial base approaches. J Craniomaxillofac Surg. 2006;34(7):415-20.
14. Xia Y, Li X, Han D, Zheng J, Long H, Shi J. Anatomic structural study of cerebellopontine angle via endoscope. Chin Med J. 2007;120(20):1836-9.
15. Suazo GIC, Zavando MDA, Smith RI. Sex determination using mastoid process measurements in Brazilian skulls. Int J Morphol. 2008;26(4):941-4.
16. Sudha R, Sridevi C, Ezhilarasi M. Anatomical variations in the formation of pterion and asterion in south Indian population. Int J Cur Res Rev. 2013;5(9):92-100.

Cite this article as: Zafar S, Abbas MW, Javaid R. Guillian-Barre syndrome after COVID-19 infection: a case report. Int J Res Med Sci 2022;10:1364-7.