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Post COVID-19 Gillian Barre syndrome in Basra city: A Case series.

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Abstract

Background: The pandemic of COVID-19 has been causing millions of cases of severe pneumonia and respiratory distress in more than 200 countries and territories of the world. The causative agent, SARS-CoV-2, is a novel coronavirus, with well recognized lung complications. However, the evidences are mounting about both central and peripheral nervous system complications.
Objectives: This is a case series study aimed to show the incidence of GBS among recently cured post COVID-19 infection cases and to describe their clinical and investigational characteristics.
Methods: This study is a descriptive study, in which we have included fifteen patients diagnosed as COVID-19 cases in the last 2-4 weeks before the onset of their weakness. All of them have been admitted to Basrah hospitals in the period between 15/7/2020 and 1/9/2020 complained from rapidly progressive ascending weakness fulfills the clinical criteria of GBS.
Results: The incidence rate of GBS in post COVID-19 cases during the period of study was about (0.375%). This result is much higher than the commonly known incidence rate of GBS in the community (0.017).

Keywords: Covid-19, GBS, Peripheral neuropathy, SARS-COV infection.

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Introduction

An unanticipated infection caused by a corona virus, SARS-CoV-2, has shocked the health of the world population and, consequently, affect people lives in many aspects and exerting a negative impact on global economy [1]. Since its first discovery in China in the city of Wuhan, December 2019, the primary manifestation of COVID-19 infection was mainly respiratory including pneumonia, cough and dyspnea.

Nevertheless, with further spread of the pandemic globally there was an increasing evidence suggests another effects of the virus on key organs other than the respiratory system, among them the central and peripheral nervous system [2].

Newly described case reports add to growing evidence that COVID-19 infections can result in severe, long-lasting neurological complications including brain inflammation, psychosis, delirium, nerve damage, and strokes; even among patients experiencing mild cases of the virus with few other symptoms.

These case reports and case series start to appear since May 2020 and increasing the evidence that the virus affect nervous system centrally and peripherally [3].

A growing body of evidence shows that neurotropism is one common feature of coronaviruses. The involvement of the nervous system can be due to a direct action of these viruses on the nervous tissue and/or to an indirect action through the activation of immune-mediated mechanisms.

While the first action can be verified during the acute phase of the disease, the second can be apparent only after days or weeks following the acute phase [4]. Many viral infections can damage the structure and function of the nervous system, manifesting as encephalitis, toxic encephalopathy, and post-infectious demyelinating disease.

Coronaviruses can invade the nervous tissues involving immune-functioning macrophages, microglia, or astrocytes and cause nerve damage through direct infection pathways (circulatory and neuronal), hypoxia, immune injury, attack to ACE2 enzymes, and other mechanisms [5]. Although the family of coronaviruses have caused deadly outbreaks in the past.

The first one caused by SARS-COV, occurred in China in 2003 and affected approximately 8,000 people, with a 10% mortality rate. The Middle-East Respiratory Syndrome (MERS) outbreak began in Saudi Arabia in 2012, and affected 2,500 individuals with a 35% mortality rate [6].

SARS-COV-2 has approximately 80% structure homology with SARS-CoV, but 96% homology with a bat coronavirus and 92% with a pangolin coronavirus, suggesting it arouse in animals and then spread between species to humans.

The spike protein of SARS-CoV-2 binds to its cellular receptor, the Angiotensin Converting Enzyme-2 (ACE2), which also acts as receptor for SARS-COV [7]. Viral entry occurs after proteolytic cleavage of the spike protein by the trans-membrane protease TMPRSS2.

ACE2 is expressed abundantly in lung alveolar cells, but also in many cell types and organs in the body, including the cerebral cortex, digestive tract, kidney, gallbladder, testis, and adrenal gland [6]. In both MERS and SARS, significant neurological complications were fortunately extremely rare.

Reported cases of neurological disease suggests a minimum incidence of ~1:200 cases (MERS) -1:1,000 cases (SARS) [6].

However, it is important to recognize that the total number of confirmed cases of MERS and SARS together is only ~10,500 cases. It is likely that the sheer numeracy of COVID-19 compared to MERS and SARS, with nearly 20 million cases reported worldwide to date, will bring out a broader spectrum of neurological manifestations [8].

In MERS and SARS neurological disease could be considered in three major categories: The neurological consequences of the associated pulmonary and systemic diseases, including encephalopathy and stroke, direct Central Nervous System (CNS) invasion by virus, including encephalitis, and post infectious and potentially immune-mediated complications, including Guillain-Barre syndrome (GBS) and its variants and Acute Disseminated Encephalomyelitis (ADEM) [9-11,6,7].

Although, different neurologic complications have been reported with its ancestors, SARS-CoV was occasionally associated with the development of different neurologic manifestations including axonopathic polyneuropathy, myopathy, rhabdomyolysis, and large artery ischemic stroke, among others.

During or after MERS-CoV treatment, Bickerstaff encephalitis overlapping with GuillainBarr´esyndrome, intensivecareunit–acquired weakness, or other toxic or infectious neuropathies have been reported [4,5]. Current knowledge points to the possibility of SARS-COV 2 to achieve its neuroinvasion to CNS by several mechanisms.

These include the transfer of the virus across thesynapses of infected cells, entry into the brain through the olfactory nerve, infection of the vascular endothelium, and the migration of infected white blood cells across the Blood Brain Barrier (BBB) [7].

The identification of post-infectious complications of SARS-COV-2 would be expected to temporally lag behind those resulting from acute infection. Occasional cases of GBS and its variants and of ADEM were reported after MERS and SARS. Reports are now emerging of similar associations with COVID-19 and GBS, and with GBS variants, including the miller-fisher syndrome.

The largest series to date, describes five patients, in this series, all patients developed GBS 5 to 10 days following COVID-19 symptom onset. The clinical presentation included bilateral multi-limb flaccid weakness with areflexia, hypotonia, and facial palsy in one case [2].

The current study aims to contribute the knowledge about the post infectious neurologic complications of SARS-COV-2 and the possibility for acute flaccid paralysis to be as a sole presenting symptom in otherwise healthy individuals except being PCR positive for SARA-COV-2.

We tried to record the flare up of GBS among group of Iraqi population following COVID-19 infection and document the peripheral nervous system complications related to this pandemic spread. We also thoroughly investigate the history of our patients to clarify any possible link with other disease that they might have in the past, associated illness and other complications if exist [12].

Methodology

This study is a descriptive study, in which we included fifteen patients diagnosed asdeveloping GBS after 14-30 days from their onset of COVID-19 infection. All of them havebeen admitted to Basra hospitals in the period between 15/7/2020 and 1/9/2020 complained from rapidly progressive ascending weakness fulfill the clinical criteria of GBS (1)and we performed the following steps

- We have included patient's in the following conditions:
- They had been diagnosed as cases of COVID-19 according to the WHO interim guideline and confirmed by a positive result to real-time Reverse-Transcriptase Polymerase-Chain-Reaction (RT-PCR) assay from throat swab specimens [11].
- Weakness started between 14 and 30 days after their proved diagnosis of COVID-19 infection.
- Second PCR tests for COVID-19 infection from throat swab of the patients 14 days after the first PCR test were negative.
- The diagnosis of the GBS cases and their follow up in the ward had been done by a consultant neurologist.
- Blood investigations that included the following tests: complete blood count, renal function test, liver function test, blood sugar test, thyroid function test, electrolytes, ESR, C-reactive protein, Rose Bengal test, viral and bacterial antibodies screen (hepatitis B and C, CMV, EBV, HIV, campylobacter jejune), immunological screen (rheumatoid factor, antinuclear antibodies, ant double strand DNA antibodies, c-ANCA and p-ANCA), toxicology for heavy metal poisoning including lead, mercury and arsenic.
- CSF study for sugar, protein, cells count, VDRL, Rose Bengal test, gram stain and culture and sensitivity. (NB unfortunately only four (26.6%) of our patients accepted this test).
- EMG and NCS had been done for all patients by an expert neurophysiologist and in five patients of them (33.3%), we repeated the test after few weeks as a follow up test.
- All patients received IVIG.
- Plasmapheresis has been done for one patient only.
- We have excluded all cases that have features of GBS but have one or more of the following points:
- Negative first PCR test for COVID-19 infection.
- NCS not done to the patient or was normal and patient refused exposed to CSF study.
- Have positive anti hepatitisantibodies, antiCMV antibodies or anti campylobacter jujini antibodies.
- Abnormal metabolic or thyroid function.
- Positive immunological tests for vasculitis.
- Did not complete the above investigations.
- Progression of weakness exceededone-month duration.

- Fever at presentation.
- Cases with clearly asymmetrical presentation and refused CSF study.
- Cases with previous or present neurological diseases like multiple sclerosis or transverse myelitis.
- Patients that refused admission.

This study was conducted in accordance with a protocol approved by the committee on clinical investigations at Basra college of medicine and Basra health directorate. All patients were informed about the aim of study and their acceptance obtained. I summarized the information of all cases in a three tables.

Aim of the study

This is a case series study aimed to show the incidence of GBS among recently cured post COVID- 19 patients and to describe their clinical and investigational findings.

Results

This study included fifteen patients developed rapidly progressive ascending weakness started from legs then arms and then involved, over many hours to few days, facial, bulbar and/or eyes muscles after between 17-30 days from their proved diagnosis as COVID-19 cases; all of them admitted to neurological ward in Basra hospitals in the period between 15/7/2020 and 1/9/020. The lowest period of starting weakness after first documented positive PCR test for COVID-19 infection is 17 days and the longest one was 30 days. Age of patients lay between 38 and 58 years except one case, which was child aged 6 years and had only mild cough and fever, proved to be COVID-19 case, 3 weeks prior to the onset of weakness.

Eleven of our cases were males and four were females that is uncommon gender distribution of GBS cases but the small number of cases and the short period of collection of these cases in addition to the high selectivity of cases in our study weakened the significance of this finding [13]. In spite of that, this finding and also some other findings in this study can open the door for further follow up and study to determine the difference between post COVID-19 GBS cases and other GBS cases.

Case no	Age	Gender	Onset after the date of 1st positive PCR test in days	Symptoms	Neurological examination
1	42	F	17	Ascending paralysis, moderate dysphagia, sever pelvic, upper back and shoulder pain, palpitation, normal sphincters	Areflexia
2	38	F	17	Ascending paralysis, mild dysphagia, normal sphincters	Areflexia
3	38	M	20	Ascending paralysis	Areflexia
4	40	M	18	Ascending paralysis, mild dysphagia, normal sphincters	Areflexia
5	43	M	23	Ascending paralysis ,normal sphincters	Areflexia
6	44	M	24	Ascending paralysis, mild dysphagia, pelvic pain, normal sphincter s,	Areflexia
7	39	M	18	Ascending paralysis, normal sphincters	Areflexia
8	47	F	27	Ascending paralysis, mild dysphagia, pelvic pain, normal sphincters	Areflexia
9	47	M	29	Ascending paralysis, normal sphincters	Areflexia
10	49	M	30	Ascending paralysis, pelvic pain, normal sphincters	Areflexia
11	51	M	24	Ascending paralysis, normal sphincters	Areflexia

12	53	M	25	Ascending paralysis, normal sphincters	Areflexia
13	56	M	27	Ascending paralysis, normal sphincters	Areflexia
14	58	M	25	Ascending paralysis, mild dysphagia, pelvic pain, normal sphincters	Areflexia
15	6	F	21	Ascending paralysis, normal sphincters	Areflexia

Table 1. Shows the detailed history for each patient included in the study (total number of patients is fifteen).

Case no	Duration of progression on in days	Symmetry	Treatment		Fate	Associated disease	Previous or present neurological disease	Chronic drug use
1	4	Asymmetric al	IVI	G	Improve	Nil	Nil	Nil
2	4	Asymmetric al	IVI	G plus Plas map here sis	Minimal Improvement	Nil	Nil	Nil
3	6	Symmetrical	IVI	G	Improvement	DM	Nil	Insulin
4	5	Asymmetric al	IVI	G	Improvement	Nil	Nil	Nil
5	7	Symmetrical	IVIG		Improvement	Nil	Nil	Nil
6	3	Symmetrical	IVI	G	Improvement	Nil	Nil	Nil
7	4	Symmetrical	IVI	G	Improvement	HT	Nil	B-blocker
8	5	Symmetrical	I	VIG	Improvement	DM	Nil	Glucophage, glibinclamide
9	3	Symmetrical	IVI	G	Improvement	Nil	Nil	Nil
10	7	Symmetrical	IVI	G	Improvement	Nil	Nil	Nil
11	5	Symmetrical	IVI	G	Improvement	Nil	Nil	Nil
12	6	Symmetrical	IVI	G	Improvement	HT	Nil	Lisnopril
13	7	Symmetrical	IVI	G	Improvement	Nil	Nil	Nil
14	3	Symmetrical	IVI	G	Improvement	HT	Nil	Amlodipine
15	4	Asymmetric al	IVI	G	Improvement	Nil	Nil	Nil

Table 2. Shows the detailed examinations for each patient included in the study (total number of patients is fifteen).

Case no	NCS findings	Blood invt.: CBP, RFT, LFT, TFT, blood sugar and electrolytes, ESR, ANA,RF,VDRL,VIROLOGY SCREEN (hepatitis B and C, CMV)	CSF	2nd PCR result
1	MCS: prolonged DML, slowing of CV, C B& T D: (L L>UL). Late responses: prolonged latencies at UL and absent from LL. FN M L, T D Sensory normal. And slowing of C V: prolonged D	Elevated ESR	Mildly Increased CSF protein, normal cells and sugar normal other findings	Negative
2	MCS: Prolonged DML, Slowing of CV, CB &TD: (LL>UL) Late responses: Prolonged latencies	N	Mildly Increased CSF, protein, normal cells and sugar	Negative

	at UL and absent from LL. FN: Prolonged DM L, TD and slowing of CV. type) Sensory: normal			
3	MC S: Prolonged DM L, slowing of CV, CB &T D: (LL>UL). Late responses: prolonged latencies at UL and absent from LL. FN: Prolonged DM L and slowing of CV. Sensory: mild imp airment in the LL (axonalt)	Blood sugar variable result	Not done	Negative
4	MC S: Prolonged DM L, Slowing of CV&TD: (LL>UL) late responses: Prolonged latencies at UL and absent from LL FN: Prolonged DM L, TD and Slowing of CV. Sensory: Normal.	N	Normal CSF, protein, cells and sugar, normal other finding	Negative
5	MCS: Prolonged DML, Slowing of CV, CB& TD: (UL& LL equally affected) Late responses: absent from UL &LL FN: Prolonged DML, TD and slowing of CV. Sensory: Normal.	N	Not done	Negative
6	MC S: Prolonged DM L, mild slowing of CV &T D: (LL=UL) late responses: Prolonged latencies at L and LL FN: Prolonged DM L& TD. Sensory: Nor mal.	N	Not done	Negative
7	MCS: Prolonged DM L, slowing of CV&TD: (LL>UL) late responses: Prolonged latencies at UL and absent from LL FN: Prolonged DM L& TD and mild slowing of CV. LLFN: prolonged DML, TD Sensory: nor mal.	N	Not done	Negative
8	MCS: Prolonged DML, slowing of C V, C B & T D: (LL>UL) late responses: Prolonged latencies at U L and absent from	Blood sugar variable result	Not done	Negative
9	MCS: Prolonged DML, Slowing of CV&TD: (LL>UL), late responses: Prolonged latencies from UL and LL, FN: and slowing of CV, Sensory: Normal prolonged DML, Sensory: Normal	N	Not done	Negative
10	MCS: Prolonged DML, Slowing of CV&TD: (LL>UL), late response s: Prolonged latencies from UL and LL, FN: Prolonged DML, Sensory: Normal.	N	Not done	Negative
11	MCS: Prolonged DML and slowing of CVDML & TD, CB&TD: (UL>LL) Late response s: Prolonged latencies at LL and absent from UL FN: Prolonged	N	Not done	Negative
12	MCS: Prolonged DML, Slowing of CV & TD: (L L>UL) late responses: Prolonged latencies from UL and LL, FN: Prolonged DML, Sensory: Normal	N	Not done	Negative
13	MCS: Prolonged DM L, Slowing of CV & TD: (UL=LL) late	N	Not done	Negative

	responses: Prolonged latencies at UL and LL FN of CV, Sensory: Normal: Prolonged DML an d slowing			
14	MCS: Prolonged DML, Slowing of C V & T D: (L L>UL) late responses: Prolonged latencies from UL and LL FN: Prolonged DML, Sensory: Normal	N	Not done	Negative
15	MCS: Prolonged DML, Mild slowing of CV&TD: (UL=LL) late responses: Prolonged latencies from UL and LL FN: Prolonged DML, Sensory: Normal.	N	Mildly Increased CSF, protein, normal cells and sugar, normal other findings	Negative

Table 3. Shows the detailed investigations for each patient included in the study (total number of patients is fifteen).

The age distribution in our cases reflects the age distribution of COVID-19 cases in Basrah city in which we have much lowest incidence rate of COVID-19 children cases in compares to adult cases. However, it is still coincide with the usual age distribution in GBS cases [14]. One of the noticeable finding in our cases is the bilateral facial weakness of variable severity in all of them regardless of the severity of the case. In three patients (20%) (cases number 3,8 and 10) there were moderate to severe weakness of both legs and facial nerves with only mild weakness of arms and neck in a picture as if weakness ascended from lower limbs to the face skipping in its way the upper limbs and neck and this finding is generally uncommon in GBS cases and both two diabetic cases in our study are with in this group [15].

In the only one child case, there was mild weakness of legs, questionable weakness of arms and neck (precise assessment of power at this age is difficult), moderate weakness of both facial nerves, ophthalmoplegia more severely involved both abducent nerves and only mild ataxia with no papilledema ,unfortunately; anti GQB1 antibodies did not done for this patient. In all cases the reflexes were absent at the 1st day of examination (the examination had been done for all cases after 2-4 days from onset of the weakness) [16]. And in all of them the sensory system examination were normal even in those four patients that had severe pain specially at lower back except for our two diabetic cases that already have mild sensory peripheral neuropathy in form of stocks distribution for superficial pain sensation.

In case number one, the patients also complained from sever upper back pain and both shoulders pain. In addition- out of all cases- there was only one case developed intermittent palpitation for two days proved sinus tachycardia on ECG study. Other noticeable finding in our cases is that four patients (26.6%) of them; are clearly showed asymmetrical distribution of weakness. And incase number one there was severe weakness of right leg and left arm, moderate weakness of left leg, mild weakness of right arm, and symmetrical weakness of both facial nerves and moderate dysphagia with mild neck weakness. This mean that about one quarter of our cases showed clearly asymmetrical distribution of weakness, which is uncommon in GBS cases [10].

All cases that have asymmetrical distribution of weakness exposed to CSF study, and those cases with asymmetrical distribution that refused CSF test had been excluded. About pharyngeal muscles involvement in our cases, there was dysphagia in six patients (40%) all of them were mild except case number one who had dysphagia of moderate severity especially to fluid with only mildly suppressed gag reflex on examination. Although five cases (33.3%), were admitted to the ICU, only two (13.3%) of them required O2 therapy for few days; (2 and 3 days); and no case required mechanical ventilation. Neurophysiological study had been done for all patients at the same day of the clinical examination. All patients have prolonged (DML) Distal Motor Latency, temporal dispersion of Compound Muscle Action Potential (CMAP), conduction block and reduction of conduction velocity of most tested nerves as appear in the Tables 1 to 3, which suggests an acquired demyelinating peripheral neuropathy. No case showed axonal form of neuropathy in the 1st neurophysiological test and only case number 2 which exhibited minimal improvement after treatment- showed a decrease in the amplitude of CMAP (40% less than normal limits) in the 2nd neurophysiological test which had been done three weeks after the 1st test and this might suggests an associated axonal degeneration.

Sensory nerve conduction study was normal for all patients except for one diabetic case and it had been attributed to long standing diabetic peripheral neuropathy. CSF study had been done for four (26.6%) cases only; protein was mildly elevated in three (20%) cases with no pleocytosis in all cases. All cases showed normal investigation screen and this might be attributed to the fact that all patients already cured from COVID-19, except for case number one who has mildly elevated (ESR=60) on two occasions one week apart which remained unexplained. All cases showed good improvement on IVIG except case number 2, which needed additional plasmapheresis with only minimal improvement.

Discussion

In regard that our hospital is a referral hospital for neurological cases for whole Basrah city, and in regard that we had the opportunity to collect GBS cases from all main hospitals in Basrah, our results can reflect to a certain limit the incidence of GBS in post COVID-19 cases in this city during the period

between 15/6/2020 and 15/8/2020 which is the period that we considered it in the collection of our GBS cases (NB we included only cases that proved to have COVID-19 infection 14 to 30 days before the onset of their weakness and our first case that we reported in this study was at 15/7/2020). The estimated number of COVID-19 new cases in Basrah city in period between 15/6/2020 and 15/8/2020 was about 4000 cases, so the incidence rate of GBS in post COVID-19 cases during this period is 0.375% (which is the percentage of 15 divided by 4000).

This result is much higher than the commonly known incidence rate of GB Sin the community (0.017%) especially if we take in our consideration the high selectivity of GBS cases that we considered in this study [10]. Another point to refer to is that, from beginning of COVID-19, cases in Basra city at march-2020 until June-2020 no proved GBS cases have been admitted to our hospital with past or present history of COVID-19, and this point is difficult to interpret. Nevertheless, the incidence rate of COVID-19 infections during that time in Basrah city was much less than after beginning of June. We have to note also the relatively short period of collection of cases in our study that might lessen the assumed possible association between COVID-19 and these cases whether causal or coincidental. Thus, a follow up study of long period is recommended to reinforce these data [17].

In this study, we included only the straightforward cases of GBS depending on clinical and neuro-physiological study. Notably, all of the patients that we suggested clinically as GBS cases they also appear to have neuro-physiological findings support this diagnosis although we performed neuro-physiological test for all cases in the day of their clinical examination (2-4 days from onset of weakness) and this finding may indicates early diffuse involvement of the peripheral nerves in post COVID-19 GBS cases [18-26].

Conclusion

Hence, CSF study was not possible to perform to the patients during the time when they suffer from active COVID-19 infection and in isolation; we included only those patients who were cured from COVID-19 infection in order to exclude the possibility of direct invading of the nervous tissue by COVID-19 virus. Also we took in consideration all other differential diagnosis specially toxic PN, metabolic PN, other related viral infection with GBS, myasthenia gravis, spinal cord disease and brain stem lesion. Although most of uncomplicated cases of COVID-19 infection improved within 1-2 weeks of infection, some of our cases left with some manifestation of post viral infection at day of presentation like fatigue, headache, depression, fear or cough but no fever.

Abbreviations:

PCR: Polymerase Chain Reaction; NCS: Nerve Conduction Study; CBP: Complete Blood Picture; RFT: Renal Function Test; LFT: Liver Function Test; TFT: Thyroid Function Test; CSF: Cerebrospinal Fluid; LMN: Lower Motor Neuron; IVIG: Intravenous Immunoglobulin; UL: Upper Limbs; LL: Lower Limbs; FN: Facial Nerve; MCS: Motor Conduction Study; CV: Conduction Velocity; TD: Temporal Dispersion; CB: Conduction Block.

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