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Case Report

Guillain-Barre Syndrome Following Covid-19 in Patient with Breast Cancer-A Case Presentation and Review of the Literature

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Abstract

Background: The emergence of the coronavirus disease pandemic in 2019, caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-Co-V-2), has resulted in a significant public health threat. A significant percentage of patients present with neurological complications in addition to respiratory and cardiac symptoms. Furthermore, a major issue resulting from the COVID-19 pandemic concerns the health of cancer patients.

Objective: We discuss a case of a female patient with history of breast cancer, later complicated with COVID-19 induced Guillain-Barre Syndrome (GBS) and depression.

Method: We performed a literature review, searching databases for cases of GBS manifestations during other pandemics.

Results: COVID-19 cases are commonly characterized by presentations of respiratory, cardiac, and neuropsychiatric manifestations. The main neurological presentations of COVID-19 have been hypogeusia (5.6%), anosmia (5.1%), and myalgia (19.3%). However, evidence suggests involvement of both the Central Nervous System (CNS) and Peripheral Nervous System (PNS) following COVID-19 infection. The most important presentation of PNS involvement in COVID-19 is GBS. There is limited data available regarding specific diagnoses or treatment for COVID-19 induced GBS.

Conclusions: The case presented with history of breast cancer, later complicated with COVID-19 induced GBS and depression. Upon review of various cases, there may be a significant association between COVID-19 infection and GBS, similar to other viral infections. Further research is necessary regarding the specific diagnoses or treatment for COVID-19 induced GBS.

Keywords: Breast carcinoma; Coronavirus; COVID-19; GBS; Neurology

Introduction

Clinical cases of patients with SARS-CoV (SARS) and Middle-East Respiratory Syndrome (MERS) provided valuable information for clinicians that similar neurological symptoms can occur following SARS-CoV-2 (COVID-19) infection [1]. Approximately 25% of patients with COVID-19 had CNS dysfunction including dizziness (17%), headache (13%), altered mental status (7.5%), acute cerebrovascular disease (3%), seizures (0.5%) and ataxia (0.5%) [1]. The incidence of CNS complications was higher (31%) in patients with severe presentation of COVID-19, in patients with Adult Respiratory Distress Syndrome (ARDS), in older patients, and those with comorbidities such as hypertension, diabetes, obesity, malignancy, cardiac or kidney disease [1].

Post infectious complications of SARS-CoV-2 are similar to those seen with SARS and MERS. Occasional cases of Guillain-Barre Syndrome (GBS), GBS variants, and Acute Disseminated Encephalomyelitis (ADEM) were reported following SARS and MERS [1,2]. In this paper we will present a patient with a history of breast cancer who was infected with COVID-19, further complicated with COVID-19 induced GBS and depression.

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Case Report

A 59-year-old female presented to the Emergency Department (ED) with 4 months history of progressive bilateral lower extremity weakness, numbness and burning pain.

The pain and numbness began in the toes and ankles and progressed in an ascending manner toward the waist and back, leading to an inability to ambulate. The patient also complained of weakness and numbness of her hands and fingers. The patient reported that her symptoms began about 2 weeks after she contracted COVID-19. She stated that she developed dry cough and due to her medical history of cancer she was tested for COVID-19 which yielded a positive result.

The patient had a past history of breast carcinoma (T2N1- a invasive ductal carcinoma) diagnosed in 2018. She was treated with lumpectomy, radiation and chemotherapy which consisted of 6 cycles of AC-T regimen including doxorubicin hydrochloride (Adriamycin) and cyclophosphamide followed by 4 treatments with paclitaxel (Taxol) over 12 weeks. She developed mild neuropathy following chemotherapy treatment which was transient and resolved within several weeks. The patient was asymptomatic during the period prior to contacting COVID-19.

The patient had also history of hypertension, Gastro Esophageal Reflux Disease (GERD), gastric bypass surgery and L4 hemilaminectomy many years prior to this presentation (Figure1).

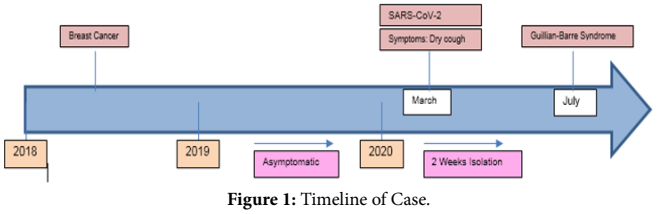

Figure 1: Timeline of Case.

In the ED the patient's vital signs showed a temperature of 98.1F, a heart rate of 96 beats/min, a respiratory rate of 18 breaths per minute, and a blood pressure of 135/86 mmHg. Oxygen saturation was 96% on room air. Neurological examination was notable for proximal and distal weakness in the upper extremities bilaterally and bilateral weakness involving the lower extremities proximally. The patient had diffuse areflexia; plantar responses were mute. There was decreased sensation around the waist, in the arms, and in the legs.

Laboratory specimen data is as follows a White Blood Cell count (WBC) of 4.7 k/ul; hemoglobin 10.9 g/dL; hematocrit 31%; platelets 271 k/uL; mean corpuscular volume (MCV) 104.8 f/L; sodium 127mmol/L; potassium 3.8mmol/L; chloride100 mmo/L; Bicarbonate 25 mmol/L; Urea Nitrogen (BUN) 6 mg/dL; creatinine 64 mg/dL; glucose 64 mg/ml; SED rate 49 mm/hr; reticulocyte count 1.6%; albumin 2.2 g/L; bilirubin 0.3 ml/dL; alkaline phosphatase 48; aspartate aminotransferase 23 units/L; alanine aminotransferase 19 unites/L; lactic acid dehydrogenase 116 U/L. Additional iron-measuring laboratory specimens include: iron 43 mcg/dL; total iron binding capacity 157 mcg/dL; ferritin 638 mg/L; transferrin saturation 50%; B12 1500 pg/mL; and total protein 5.9 g/L. Urinary analysis shows trace blood amounts, moderate leukocytes, WBC> 100 /hpf, RBC 30-50/ hpf, and various bacteria was present. Methicillin-Resistant Staphylococcus Aureus (MRSA) was negative, COVID-19 was negative, and Human Immunodeficiency Virus (HIV) was negative. The patient underwent lumbar puncture, which was normal, except for the Cerebral Spinal Fluid (CSF) protein of 80 mg/dL. The CSF was negative for SARS-coV-2.

Chest CT scan showed focal sub pleural scarring presumably due to left breast radiation, no pleural effusion, no nodules or masses. Head CT scan showed mild patchy white matter hypodensities compatible with sequelae of non-chronic small vessels ischemic disease, no evidence of acute infarction, intracranial hemorrhage, or mass lesion. MRI of the spine showed left L4 hemilaminectomy medial facetectomy and resection of the left flavum, and multilevel degenerative changes in the spine causing mild neuroforaminal stenosis on the left at L4-L5 and bilateral at L5-S1. Mild diffuse edema in the posterior para-spinal musculature, possibly consistent with myositis.

The patient was diagnosed with acute inflammatory demyelinating poly radiculoneuropathy, or GBS. Nerve conduction studies were performed to confirm the diagnosis and were consistent with mild demyelinating polyneuropathy. The patient was treated with a 5 day course of intravenous immunoglogulin (IVIG) as well as with gabapentin, oxycodone, ciprofloxacin, and amlodipine.

Psychiatry was involved on day 3 of admission for depression and anxiety. The patient reported depressed mood which she attributed to pain, inability to work, sense of loss, and a decrease in bodily function. She endorsed frequent crying spells, difficulty speaking, poor appetite, and a 40 pound weight loss since she contracted COVID-19. The patient was treated with antidepressants and was later transferred to rehabilitation.

Review of the Literature

The novel coronavirus-19 (COVID-19) was first reported during December of 2019 in Wuhan, China, and has since developed into a global pandemic. As of January 19th, 2021, nearly a year into the COVID-19 pandemic, there have been 2,053,834 deaths worldwide. As COVID-19 evolved into a widespread pandemic, concern was raised regarding the impact of COVID-19 on the health of cancer patients [3-5].

Reports suggest that patients with various malignancies are at risk of contracting COVID-19 infection [3,6,7]. Cancer patients are at increased risk of contracting COVID-19 due to various factors including an age of 60 years or greater, immunosuppression resulting from antineoplastic medications and steroid use, immunosuppressive properties of the cancer itself as well as other medical comorbidities [8]. These patients may also have an increased immune response to COVID-19 infection prompted by immunomodulatory drugs, such as programmed cell death 1 and programmed cell death ligand 1 inhibitors [9].

Patients with malignancies appear to be at increased risk for severe illness and mortality due to COVID-19 regardless of whether they have active cancer, undergoing antineoplastic therapy, or both [8]. Patients with cancer who contract COVID-19 tend to exhibit higher rates of complications. For example, reports from various healthcare systems in the United States (US) show that the death rate from COVID-19 in cancer patients was between 11-28%, and 11% of patients required intubation [3,10]. According to Miyashita H. et al., patients with cancer were intubated significantly more frequently, but the rate of death was not significantly different [3]. This group also reports that the cancer patients who were younger had higher rates of mortality [3].

Patients with active cancer are at higher risk of developing worse outcomes related to COVID-19, especially in those patients who have progressive disease. Studies have shown that patients with progressive cancer have a higher mortality rate without ICU admission, compared to those who were admitted to the ICU [8]. The opposite association was observed in patients with remission, meaning that patients who were admitted into the ICU did worse than the patients who were not. Patients aged 75 years and older, as well as those who were receiving palliative care, had a higher risk of mortality. Reports have shown that curative surgical resections, adjunct chemotherapy, and maintenance therapy, should continue during the COVID-19 pandemic with extreme caution [8].

The majority of cases of COVID-19 are characterized by presentations of respiratory, cardiac, and neuropsychiatric manifestations [11]. As the COVID -19 pandemic continues to escalate, numerous reports have emerged suggesting significant evidence of neurological manifestations induced by COVID-19 infection. The main neurological presentations of COVID-19 have been hypogeusia (5.6%), anosmia (5.1%), and myalgia (19.3%) [11]. However, there is evidence suggesting involvement of both the Central Nervous System (CNS) and Peripheral Nervous System (PNS) following COVID-19 [12]. Other patients with COVID-19 developed neuromuscular disease, motor neuropathy, myopathy, or both [12]. The neurological manifestations could be a direct effect of the virus on the nervous system, para-infectious or post-infectious immune mediated disease, and neurological complications of the COVID-19 infection [12]. The COVID-19 virus was detected in the Cerebral Spinal Fluid (CSF) by Reverse Transcription Polymerase Chain Reaction (RT-PCR) in a few cases of encephalopathy with seizures. The virus was later cultured from brain tissue during autopsy [12].

Central Nervous System Diseases Associated with COVID-19

Encephalitis is an inflammation of the brain parenchyma, usually caused by various infectious organisms or immune response [12]. Diagnosis was usually confirmed by CNS pleocytosis, imaging changes, or focal abnormalities on electroencephalogram (EEG)

[12]. Evidence of brain inflammation must be present and detection of the virus in the CSF does not provide diagnosis [12]. Studies have shown that patient's age 24-78 who presented with fatigue, headache, fever, sore throat, generalized seizures, reduced consciousness, and meningeal signs were subsequently diagnosed with encephalitis associated with COVID-19 by nasal or nasopharyngeal swab [12]. Many patients developed confusion following a few days of cough and fever. Other manifestations such as irritability, confusion, and reduction of consciousness associated with or without seizures were also seen [12]. One report from Wuhan, China which investigated 214 patients with COVID-19 showed that 25% of the patients present with CNS symptoms including 17% dizziness, 13% with headache, and impaired consciousness with 7%. More than half of patients with CNS symptoms had severe respiratory disease [12].

Acute Disseminated Encephalitis (ADEM), a syndrome of multifocal demyelination, presenting with focal neurological symptoms occurring weeks after infection, is another manifestation of COVID-19 [12]. Few patients developed dysphagia, dysarthria and encephalopathy 9 days after the onset of headache and myalgia [12]. Other patients developed seizures, a decrease of consciousness, and respiratory failure requiring intubation [12]. These patients had normal CSF and high signal intensities on MRI which is common in acute disseminated encephalitis. These patients' medical status had improved with intravenous immunoglobulin and steroids [12].

Myelitis, which is inflammation of the spinal cord, associated with COVID-19 is uncommon [12]. This presented in cases in China with fever, fatigue, and acute flaccid paralysis with incontinence. Myelitis and acute disseminated encephalomyelitis are considered post infectious diseases and both are treated with intravenous immunoglobulin and dexamethasone [12]. Those cases were diagnosed by nasopharyngeal swab, RT-PCR. CSF in these patients often showed normal cell count, protein, and glucose. Brain MRI was positive for extensive areas of high signal in the bilateral frontoparietal white matter, anterior temporal lobes, basal ganglia, external capsules, and thalami. EEG was normal with no evidence of epileptic activity [12].

Cerebral vascular accidents (stroke) affect 62% of patients who presented with neurological manifestations of COVID-19. Of those, 46% with ischemic stroke, 7% with intracerebral hemorrhage, less than 1% with CNS vasculitis, and 8% of patients presented with other cerebrovascular events [12]. Most patients who sustained CVA were older than 60 years, had risk factors such as diabetes, hyperlipidemia, hypertension and obesity. Cerebrovascular symptoms often began 10 days following the onset of respiratory symptoms. Deep venous thrombosis or pulmonary embolisms were found in some stroke patients [12]. Patients with COVID-19 can also present with neuropsychiatric features including 8% presented with psychosis, 5% with neurocognitive dementia-like syndrome, and 3% with affective disorder (depression) [12].

Peripheral Nervous Disease: Guillain-Barre syndrome (GBS)

The most important presentation of PNS involvement in COVID-19 is GBS. It comprises a continuum of various polyneuropathies characterized by acute onset (within 1-4 weeks) of ascending motor weakness, sensory abnormalities which could be mild or moderate, cranial nerve involvement, and muscular and/or radicular pain [11]. The weakness is often proximal rather than distal, but in many patients weakness begins distally and spreads proximally [13].

GBS was first described in 1859 by Landry as ascending post infectious polyneuropathy with all the symptoms of GBS, except for areflexia [13]. By 1916, during the First World War, Guillain, Barre and Strohl described the syndrome in 2 soldiers with similar clinical presentations [13] GBS is the most common cause of acute or sub acute flaccid weakness worldwide, following the eradication of the polio infection [14]. The incidence of GBS is 0.5-2 per 100,000. Males are affected more frequently than females. Lifetime risk is 1:1000 [15].

The syndrome usually evolves over a few days and begins with numbness and weakness in the lower extremities. The disease progression can be rapid, however 50% of patients present with full weakness within 2 weeks, 80% by 3 weeks and 90% by 4 weeks [13]. When progression is beyond 4 weeks, one may think about Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP) [13].

GBS is a polyradiculoneuropathy, where weakness is often proximal rather than distal, but in many patients, weakness begins distally and spreads proximally. There are cases where the weakness is localized to the legs only, giving a clinical picture of paraplegia, however reduced or absent reflexes point to GBS [13]. 66% of patients develop neuropathic pain localized to the lower back and thighs. All patients will show areflexia at some point in the disease process. 50% of patients may develop facial weakness and other cranial nerves abnormalities including ocular dysmotility, pupillary change and ptosis [13]. Respiratory failure due to phrenic nerve weakness develops in 30% of patients. Those patients require intubation ventilation and often progress to tracheostomy placement. Autonomic symptoms are often seen with GBS including hypertension, tachycardia, bradycardia, urinary retention, and a decrease in gastric motility. Minor deficiencies in vibration and in proprioception may be apparent on sensory examination [13].

Based on the motor or sensory nerve involvement, GBS is categorized into a few subtypes. The most common subtypes are Acute Inflammatory Demyelinating Polyneuropathy (AIDP), Acute Motor Axonal Neuropathy (AMAN), Miller-Fisher Syndrome (MFS), and the pharyngeal- cervical-brachial- form. Other rare and poorly defined forms of GBS are acute autonomic neuropathy and acute sensory neuropathy [13]. AIDP presentation of GBS is the most common form of GBS.

The AMAN form is common in children, occurs usually during the summer. It has motor involvement only without sensory manifestations. This form is prevalent in northern China and is less common in the US, Europe, and other parts of Asia. Nerve conduction studies show a decrease in Compound Muscle Action Potential amplitudes (CMAP), normal latencies and conduction velocities. Sensory studies are normal [13].

The AMSAN form of GBS is identical to the AMAN form, except of the additional sensory manifestations. This can be seen clinically, during physical exams and on nerve conduction tests [13]. Symptoms may include progressive weakness in the upper and lower extremities, areflexia or hyporeflexia, relative symmetry of weakness and sensory loss. Weakness tends to be more dominant than sensory loss and pain is common and is often located in the back and legs. Patients are often afebrile and autonomic dysfunction is common. The CSF shows albuminocytological dissociation by the 3rd week of illness. This form presents in various parts of the globe, affects older individuals, has prolonged course and improvement is incomplete [13].

Miller Fisher syndrome is different clinically from GBS. It is an acute or sub acute demyelinating polyradiculoneuropathy which presents with the triad of ophthalmoparesis or ophthalmoplegia, areflexia and ataxia. Lower brainstem involvement such as facial and laryngeal weakness can be seen. Patients with this condition often have GQ1 b antibodies in their serum [13]. Patients with this syndrome usually improve by 1-2 months and completely recover within 6 months without any treatment. Miller Fisher syndrome is often grouped with Bickerstaff brainstem encephalitis due to their similar presentation [13].

GBS is considered to be one of the prototypical auto-immune neurological diseases triggered by viral infection. 70% of patients with GBS present with flu-like symptoms, 10-14 days after contacting an upper or lower respiratory illness or gastroenteritis [11]. Viral agents associated with an onset of GBS are cytomegalovirus, Epstein–Barr virus, Influenza A, enterovirus, herpes simplex virus, hepatitis virus, human immunodeficiency virus (HIV), Zika virus, and most recently, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2 or COVID-19). Bacterial organisms that can trigger GBS are Mycoplasma pneumonia, Haemophilus influenza, and Campylobacter jejuni [13]. Less common causes for GBS are surgery, pregnancy, cancer and vaccinations such as A/H1N1 antigen [11]. GBS outbreaks have been associated with H1N1, swine fluA/New Jersey influenza strain, arthropod-borne flaviviruses, such as the West Nile virus, chikungunya, or Zika virus, and with coronaviruses, including the Middle East respiratory syndrome (MERS)-CoV and SARS-CoV [11].

Studies have shown that GBS triggered by COVID-19 has been reported in Wuhan, Italy, Spain, France, and the United States [11]. The first case described a woman from Wuhan who presented from acute lower extremity weakness and areflexia that progressed over 3 days to the arms without any systemic symptoms. GBS was suspected and confirmed by elevated CSF protein and electromyography signs of demyelinating neuropathy [11]. This initial case from Wuhan demonstrated normal laboratory tests results except of lym phocytopenia and thrombocytopenia. She began treatment with intravenous immunoglobulin (IVIG). 4 days following treatment with IVIG, the patient developed symptoms such as fever, cough, pneumonia, and tested positive for COVID-19 [11]. Another case was of a male from Spain, who presented with Miller Fisher Syndrome (MFS) or polyneuritis cranialis. This patient with MFS presented with oculomotor nerve palsies, diplopia, perioral paresthesias, areflexia, ataxia and anosmia. His laboratory tests results showed elevated CSF proteins concentration, GD1b ganglioside antibodies and positive COVID-19 testing [11].

Recent reports show patients who present with GBS may not have any classical symptoms of COVID-19 at presentation, i.e., dry cough, shortness of breath, pneumonia. These patients tested positive for SARS-COV2 by nasopharyngeal swab, and some had evidence of lymphocytopenia, and thrombocytopenia characteristic of COVID-19. Few patients presented with diarrhea prior to the onset of neurological symptoms [12]. Other patients had anosmia, ageusia, and the presentation of lymphocytopenia and/or thrombocytopenia at presentation prior to the onset of GBS. Thus, it is evident that positive laboratory tests were a red flag pointing towards suspected COVID-19 in otherwise asymptomatic patients with acute neurologic events [11]. In our case, the patient presented with incidental finding of COVID-19, due to her past history of breast cancer. She was asymptomatic at presentation, however, she developed symptoms consisting of GBS 3-4 months after COVID-19 infection.

Studies have shown that lumbar puncture conducted in patients with COVID-19-GBS, did not detect the virus in the CSF implying no direct route of infection or intrathecal viral replication [11]. The presence of GD1b ganglioside antibodies and improvement in clinical picture with IVIG suggests that post-viral triggered immune response similar to other post-viral GBS cases or other post-viral autoimmune neurologic disorders. Analysis of other viral pandemics showed that COVID-19 can trigger neurological autoimmunity [11].

The autoimmune mechanisms involved in COVID-19 - GBS is thought to be related to various gangliosides [11]. The most common ganglioside that can lead to neuropathies are gangliosides containing disialosyl moieties (GD1b, GQ1b, and GT1b), gangliosides that share structures with GM2, and ganglioside that involve in a combination of GM2 and GM1, and of GM1 and GD1b. When IGN recognizes the Gal (pl-3) GaINAc moiety of GM1 found on the surface of motor neurons, a motor neuropathy ensues. When IGM recognizes the epitopes containing disialosyl groups of GD1b which are present on the dorsal root ganglion neurons, ataxic neuropathy emerges [11]. Patients with sensory ataxic neuropathy and GD1b antibodies also present with MFS. It is currently apparent that there is a cross reactivity between COVID-19 spike bearing gangliosides and surface peripheral nerve glycolipids (GalNAc residue of GM1) [11]. This cross-reactivity leading to GBS was also observed between peripheral nerve glycolipids and Campylobacter jejuni or Zika virus. COVID-19 can cause all subtypes including AIDP, AMAN, and MFS, therefore it is prudent to screen for gangliosides antibodies to determine antibodies between COVID 19 and GBS [11]. Several authors have compiled and described cases of patients affected by COVID-19 and GBS syndrome.

Toscano et al. reviewed a case series of 5 cases of COVID-19 complicated with GBS. In this study, most patients developed GBS symptoms consisting of bilateral weakness with paresthesia and areflexia, 5 days following COVID-19 infection. Of the 5 patients described in the study, 3 patients developed respiratory failure and 2 patients had facial weakness [1,2]. The time interval between the onset of COVID-19 symptoms and the neurological manifestations was about 5-10 days. The CSF showed normal protein count in 2 of the patients and all the patients had white cell count of less than 5 per cubic millimeter. Two of the three patients tested positive for anti-ganglioside antibodies. Polymerase chain-reaction assay of the CSF was negative for SARS-Cov-2 in all the patients. Nerve conduction and electromyography were abnormal, consistent with demyelinating disease. Magnetic Resonance Imaging (MRI) using gadolinium showed enhancement of the caudal nerve root in two patients and enhancement of facial nerve was seen in one patient. One patient showed no signal abnormality. All of the five patients received IVIG. Two patients received additional IVIG treatment and one patient underwent plasma exchange. After four weeks of treatment, two patients were intubated and mechanically ventilated in the intensive care unit, two were undergoing physical therapy due to flaccid paralysis, and one patient was able to ambulate and was discharged.

Rodrigos M. Carillo-Lorca et al. conducted a systematic review of case reports of COVID-19 and GBS syndrome. They searched for case reports in MEDLINE, Embase, Global Health, Scopus, Web of Science and MedXriv [16]. These case reports identify 12 patients with COVID-19 induced GBS. One case of Miller-Fisher, a variation of GBS, was recorded. The age range of the patients was between 23-77 years. These cases demonstrated male predominance (9/12). Cases of GBS in COVID-19 patients showed heterogeneous presentations

both clinically (e.g., ascending or cranial nerve paralysis) and electro physiologically (e.g., axonal or demyelinating). In the cases identified during this systematic review, the symptoms of GBS often begin following COVID-19 infection between 5-24 days. Protein levels in cerebrospinal fluid samples ranged between 40 and 193 mg/dl. None of these samples tested positive for COVID-19. 6 of the 12 patients in this review exhibited ascendant weakness and 2 exhibited facial weakness. 5 of these patients demonstrated a mild course of illness, while 4 required critical care due to severe symptoms. However, none of these patients died [16].

In this systematic review, the similarities between COVID-19 and Zika was explored. The authors found that male predominance, age, and clinical presentation of Zika induced GBS is similar to that of COVID-19 with regard to both Zika and COVID-19 - GBS variants with bilateral facial paralysis. Compared to Zika virus, in COVID-19, cerebrospinal fluid protein levels were recorded as being higher [16]. Unfortunately the number of cases explored in this case-report is limited and further studies are required to build upon existing evidence. In conclusion of this systematic review, clinicians should be aware of the suggested association between GBS and the COVID-19 is similar to that of Zika virus [17-19].

Conclusion

The case presented of breast cancer complicated with COVID-19 induced GBS and depression. Similar to other cases reviewed, there is evidence suggesting a significant correlation between COVID-19 infection and GBS, an association that has been described in other viral infections such as Zika. The pathophysiological mechanism that is most likely responsible for triggering GBS, as a result of COVID-19 infection, is that a prior infection evokes an immune response. This immune response then cross-reacts with the peripheral nerve and involves both the cellular and humoral components. Subsequently, there is an activation of T-cells, followed by macrophages, leading to a complement activation and cell damage. The immune response is directed against epitopes in the myelin, ultimately leading to Acute Inflammatory Demyelinating neuropathy (AIDP).

The clinical presentation described in the case presented should alert oncology clinicians to readily identify symptomatic and asymptomatic cases of COVID-19 infections by routine testing. Health care workers should recognize and remedy the risk of further complications due to COVID-19, even beyond the classical presentations of cardiac, respiratory, and neuropsychiatric distress. It has become increasingly evident that neurological complications, affecting both the CNS and PNS, are prevalent manifestations of COVID-19 infection. GBS, along with variants of GBS, deserve further study in order to better understand and potentially decrease the impact of COVID-19 infection on patients with malignancies. As COVID-19 continues to challenge the delivery of medical care worldwide, clinicians will be required to address the neurological conditions afflicting the cancer patient population.

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