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REVIEW ARTICLE

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A Case of Fulminant Guillain- Barre Syndrome in Association with Acute Cytomegalovirus Infection

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ABSTRACT

Guillain-Barre syndrome is typically an ascending predominantly motor polyradiculoneuropathy, rarely presenting in fulminant form with extreme neurological deterioration, and need for mechanical ventilation, prolonged complex ICU management. Principally it can be viral , or autoimmune in origin, with diverse mechanisms of usually reversible nerve injury. Our case report describes a form, AIDP in association with acute cytomegalovirus infection with high blood viral and respiratory DNA CMV load, requiring antiviral therapy, highly activated and dominant CD3+CD8+ T cell response, negative autoantibody panel, suggesting a cytopathic etiology of nerve damage. We also report severe sympathetic dysautonomia presenting with tachycardia and hypertension. We emphasize the need to further enhance technologies enabling dissecting the many faces of GBS clinically.

ARTICLE HISTORY

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Abbreviations

AIDP: Acute Inflammatory Demyelinating Polyneuropathy

ALT: Alanine Aminotransferase

CT: Computed Tomography

CTL: Cytotoxic Lymphocytes

CRP: C Reactive Protein

CMV: Cytomegalovirus

EBV: Epstein-Barr Virus

EMG: Electromyography

EEG: Electroencephalography

EGFR: Epidermal Growth Factor Receptor

GBS: Guillain-Barre Syndrome

HFNO: High Frequency Nasal Oxygenation

HAP: Hospital Associated Pneumonia

ICU: Intensive Care Unit

IL-10: Interleukin 10

MHC: Major Histocompatibility Complex

MFS: Miller Fisher Syndrome

MRI: Magnetic Resonance Imaging

PCR: Polymerase Chain Reaction

PRR: Pattern Recognition Receptors

PDGFR: Platelet- Derived Growth Factor Receptor

PSA: Pseudomonas Aeruginosa

SOB: Shortness of Breath

TLR2: Toll Like Receptor 2

SLE: Systemic Lupus Erythematosus

PEG: Percutaneous Endoscopic Gastrostomy

PLEX: Therapeutic Plasma Exchange

TEE: Transesophageal Echocardiography

M: Meropenem

V: Vancomycin

VALD: Valganciclovir

Introduction

GBS is a multi-etiological polyradiculoneuropathy, with reported incidence of 1-6/100000, geographical variations, male predominance 3:1, fluctuating frequency based on epidemic viral burden [1]. The pathogenesis is referred to as a consequence of immune reaction to self, with details that are perhaps multifactorial and remain to be fully understood. Regardless of etiology, the effector immunological mechanism may be a complement or cell mediated cytopathic effect on peripheral nerve myelin sheets [2]. The etiopathogenic versatility is perhaps difficult to grasp statistically, but for an individual patient it is his or her own individual phenotype that matters.

The most prevalent type is the AIDP form (acute inflammatory demyelinating polyneuropathy) but there are several more-less typical phenotypes with mostly motor, less frequently variable sensory involvement. GBS emergence is reported upon respiratory and other infections and in association with vaccinations with two to three weeks delay [4].

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Anti - ganglioside antibodies were reported after Campylobacter jejunii infection and in association with Sars-COV-2 [3-6]. There are several phenotypes of GBS besides typical motor dominant ascending form, for example Miller-Fisher syndrome, associated with anti GQ1b antibodies [7]. Cerebrospinal fluid detected HSV-1 infection had been reported as aGBS/MF syndrome trigger, likewise influenza association had been found, yet in 60% of cases obvious pathogenic triggers are not detected [8,9]. HIV associated refractory GBS, improving upon retroviral treatment [10]. The majority of case studies is devoid of immune profiling. The condition is potentially reversible, but about 20% of patients remain with partial disability for months to years, comprising usually the most severe and most fulminant forms, placing an extreme burden for the patient, and the society [11].

Case Presentation

A 38 years old gentleman, smoker, with food allergies, BMI of 27.8, was admitted to our hospital via the emergency department. For several days he had been complaining of sore throat, and fever and was prescribed oral antibiotics by the GP for purulent pharyngitis. Due to increasing weakness and dyspnea he visited the emergency department. Upon initial examination inspiratory SOB with relatively small chest movement without stridor and upon examination by direct laryngoscopy without obstruction had been present, with extreme sympathetic and hypertensive reaction, body covered by cold sweat. Initial steroid and antihypertensive approach have led to temporary relief of his symptoms. After CT staging with overall negative results, he was admitted to the intensive care department and commenced HFNO. Initial neurology revealed motor deficit with inability to lift arms and legs. Twelve hours later he had to be intubated, light sedation and mechanical ventilation initiated for impelling respiratory failure and rapidly progressing ascending motor deficit, muscle weakness, the patient denied pain,loss of touch and touch sensation. Complex laboratory diagnostic sample material had been collected, immunoglobulin, antibacterial and antiviral therapy initiated in cooperation with neurologists. Two days after admission the neurologic symptomatology had deteriorated to the level of complete loss of reflexes, including that of pupillary light reflex.

Extensive laboratory investigation revealed high positive PCR load of CMV virus in the blood and in bronchoalveolar lavage fluid day 5, and in blood day 13, the presence of active viral infection was indicated by increased lymphocyte numbers in blood. Concomitantly oxygenation and lung compliance worsened, X ray revealing interstitial edema, the (Figure 1a) patient was proned to secure oxygenation and improve ventilation-perfusion mismatch. Oxygenation was impaired with oxygenation index as low as 80, and initially high oxygen fractions were required using protective mechanical ventilation (figure 1b). Beyond that a CMV related transaminitis emerged, with elevated ALT, total and conjugated bilirubin levels [2]. Ganciclovir had been initiated upon clinical pharmacology advice. EMG revealed extensive conduction delay with greatly reduced amplitudes of predominantly motoric nerves, with enhanced denervation changes distally. Comprehensive autoantibody screening of serum was negative (PANCA, CANCA, CIK, Hu, Ri, Yo, Titin, Recoverin, SOX1, amphiphilic, CV2, antiganglioside IgM and IgG abs-GQ1b, GT1b, GD1a,GD1b, GM1,2,3). Immune profiling revealed CD3 T (CD3+DR+) cell activation with cytopathic CD3+CD8+T cell predominance, reminiscent of viral, typically CMV induced immune response. At the same time low levels of CD4+ T cells, CD19+ B cells and CD16+CD56+ NK cells were depicted (Figure 3). Here we show the overall dynamics of inflammatory parameters, CRP levels maintained at mid high, and during bacterial flare ups in high levels (Figure 5) lymphocyte and neutrophil fluctuations (Figure 4) reflecting the initial viral course, later however neutrophils predominated. Severe autonomic dysautonomia was present through the disease course, signalizing a hyperdynamic, hyperinflammatory phenotype with tendencies towards elevated heart rate, hypertension, overt sweating and hyperpyrexia (Figure 7) requiring beta-blocker and antihypertensive administration in high doses (Figure 8), despite deep sedation initially. Repeatedly increased body temperature could be maintained at levels below 38C using arctic sun whole body cooling technology repeatedly (Figure 9). All the above signalize a hyperinflammatory, hyperdynamic phenotype. IVIG therapy had been repeated as per guidelines. PLEX had not been used and would likely be less efficient due to a cell mediated infective event. Later Pseudomonas HAP emerged requiring cefepime administration and, one short course of septicemia requiring meropenem, vancomycin administration (Figure 6 and Table 1), using TEE infective endocarditis, and other cardiac pathology was excluded. To further enhance recovery, a short course of creatine phosphate was administered. Cerebrospinal fluid examination revealed early on presence of monocytes, later (day 6) albumin cytologic dissociation. MRI imaging supported the diagnosis by targeting the lumbosacral region and encephalitic brain changes had been excluded, ethmoidal sinusitis was depicted Figure 10, lit) [12]. Repeat bronchoscopy airway toilette was provided, percutaneous dilation tracheostomy and PEG (percutaneous endoscopic gastrostomy) inserted as part of complex management. Thorough physiotherapy twice daily including weekends implemented. The patient demonstrated a slow sustained recovery, ultimately having spent 57 days at ICU, and upon expedition his motor functions, particularly those of upper limbs significantly improved. The patient's tracheostomy was extracted on day 58, he was without dysphagic symptoms, could be orally fed, was able to talk, read, and was verticalized using assistance. The dynamics of motoric improvement are summarized in table 2, here we show the body weight loss of more than 17 kg, despite a functional gastrointestinal system, but for a short period and continuous enteral feeding, such loss can be attributed to hyperinflammatory, hyperdynamic phenotype.

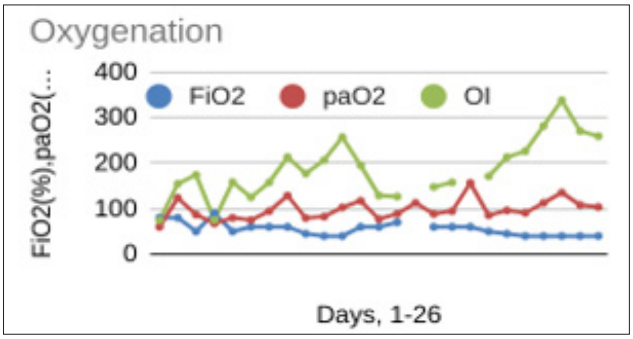


Figure 1a: Legend: Oxygenation Parameters Depicted During the First 26 days

Blue: Oxygen Fraction in %s- Acceptable Below 50%,

Red: Partial Arterial Pressure of Oxygen Achieved Using Protective Mechanical Ventilation - mmHg,

Green: Oxygenation / Horowitz Index, Calculated: OI= paO2 mmHg/oxygen fraction. Normal Values are above 200

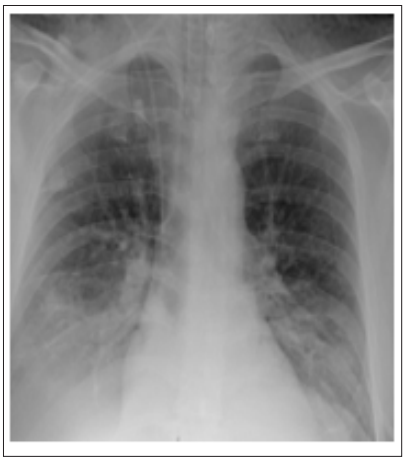


Figure 1b: X Ray Picture of Interstitial Pneumonitis on Day 3 of Hospitalization

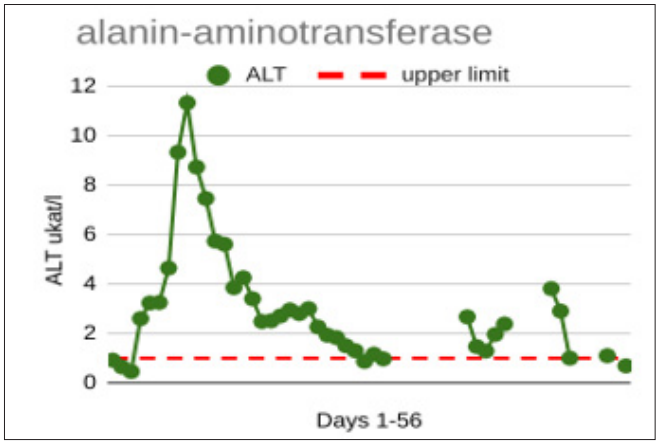


Figure 2: Serum Alanine- Aminotransferase Levels Measured in Ukat/Ml

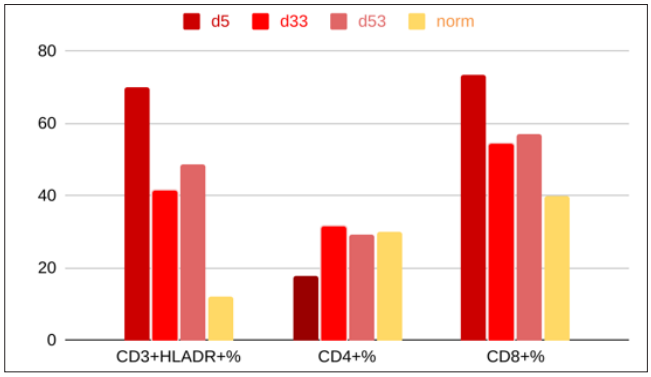


Figure 3: Relative Proportion of CD3+CD8+ and CD3+ CD4+ T Cells on days 5, 33, 53

In Comparison to Upper, resp. lower norm (yellow), Proportion of CD3+DR+ Activated T Cells in Comparison to the Upper Norm

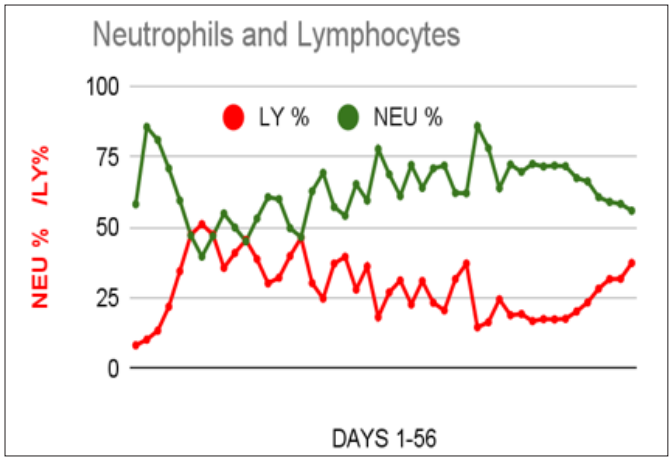


Figure 4: Relative Proportions of Neutrophils and Lymphocytes in Blood, Days 1-56

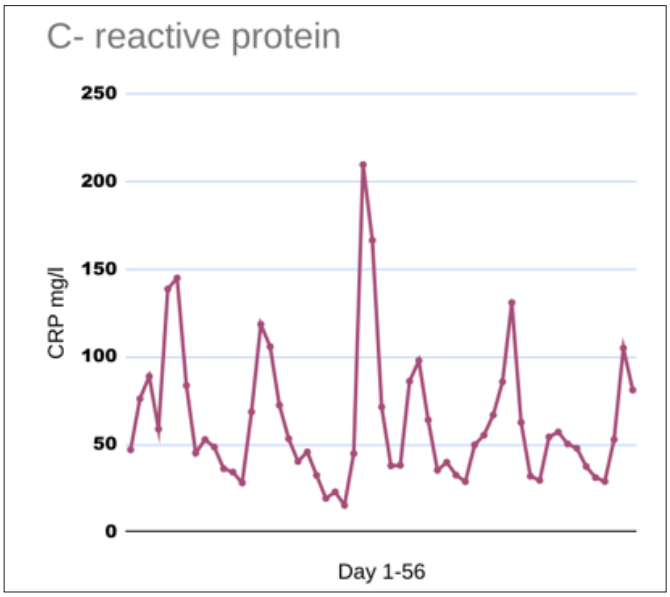


Figure 5: CRP Dynamics During Hospitalization, in mg/l, the Acceptable Upper Norm is 20mg/l

days	atb/antiviral	bug
1-7	A+M+V	CMV BAL
8-12	AZI	Mycoplasma BAL
11-25	GAN	CMV BLOOD
25-32	VALD	
26-32	VALD+CEF	PSA BAL
36-40	M+V	Staph BLOOD
36-44	M	PSA BAL

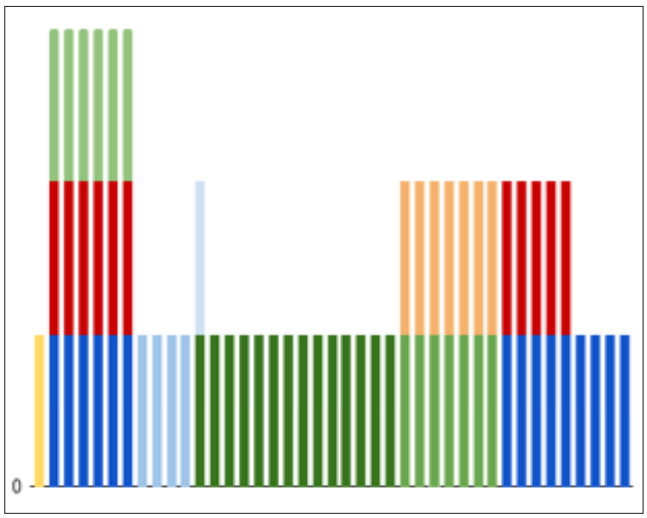


Figure 6 and Table 1: The Dynamics of Antibiotic and Virostatic Administration, and Target Pathogens

Blue: Meropenem,

Red: Vancomycin,

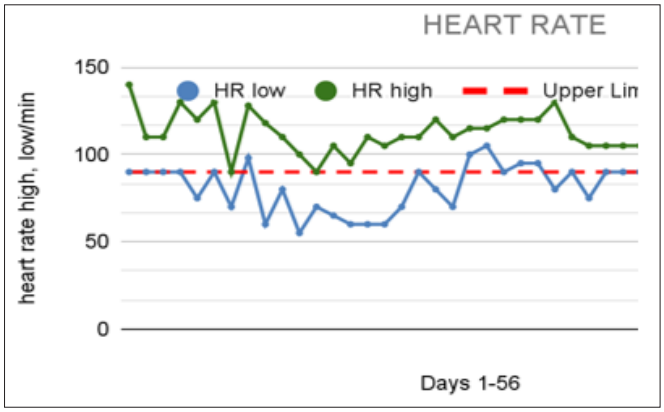
Light Green: Acyclovir,

Light Blue: Azithromycin,

Dark Green: Ganciclovir,

Mid Green: Valganciclovir,

Yellow: Cefepime



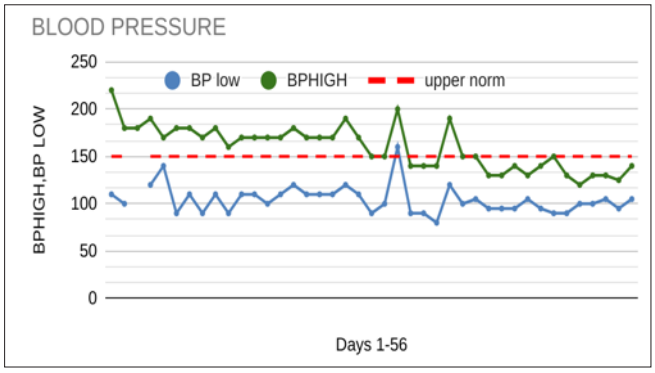
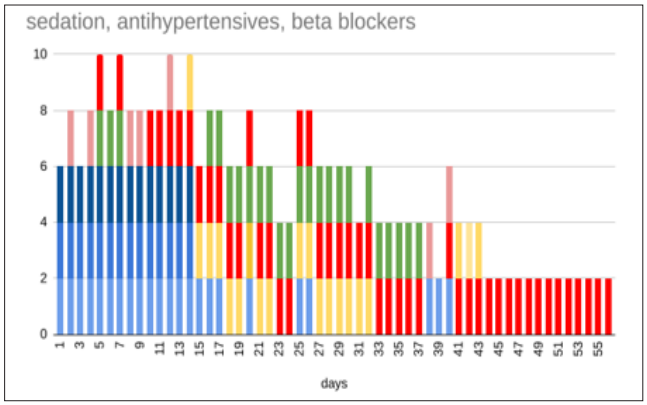


Figure 7: Heart Rate and Blood Pressure, the Highest and Lowest Values/Min are Depicted Daily, During 56 Days Based on Continuous Monitoring and Hourly Registration. Normal Values: HR:50-90/min, sBP:90-159mmHg



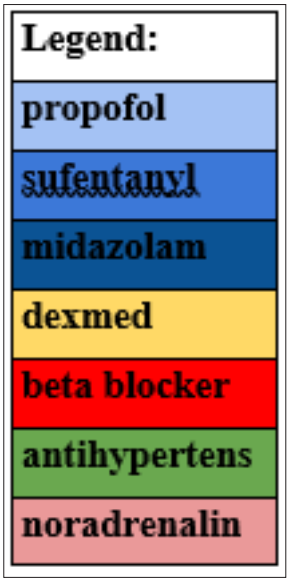
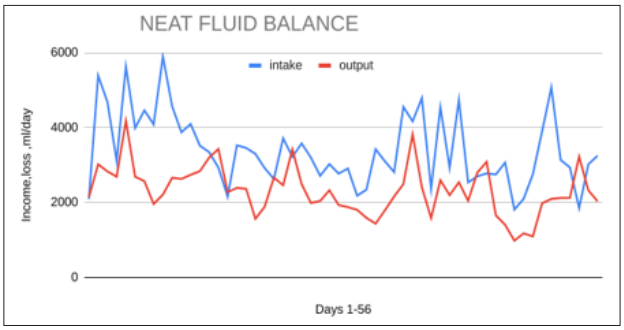


Figure 8: Use of Sedatives, Antihypertensives and Beta- Blockers to Maintain Acceptable Heart Rate and Blood Pressure Values



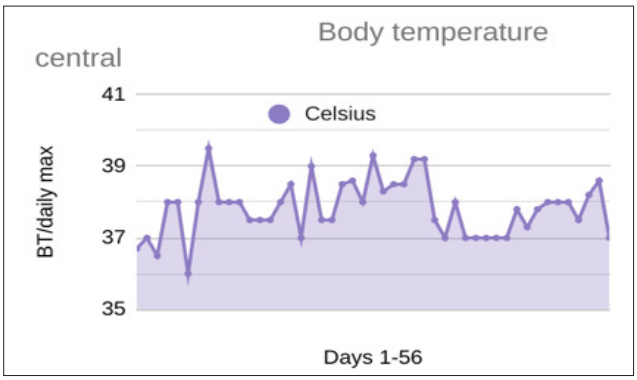


Figure 9: Neat Fluid Balance in ml/24 Hours, During 56 days of Hospitalization, and Temperature Dynamics, Using Central Body Temperature Measurement in the Urinary Bladder

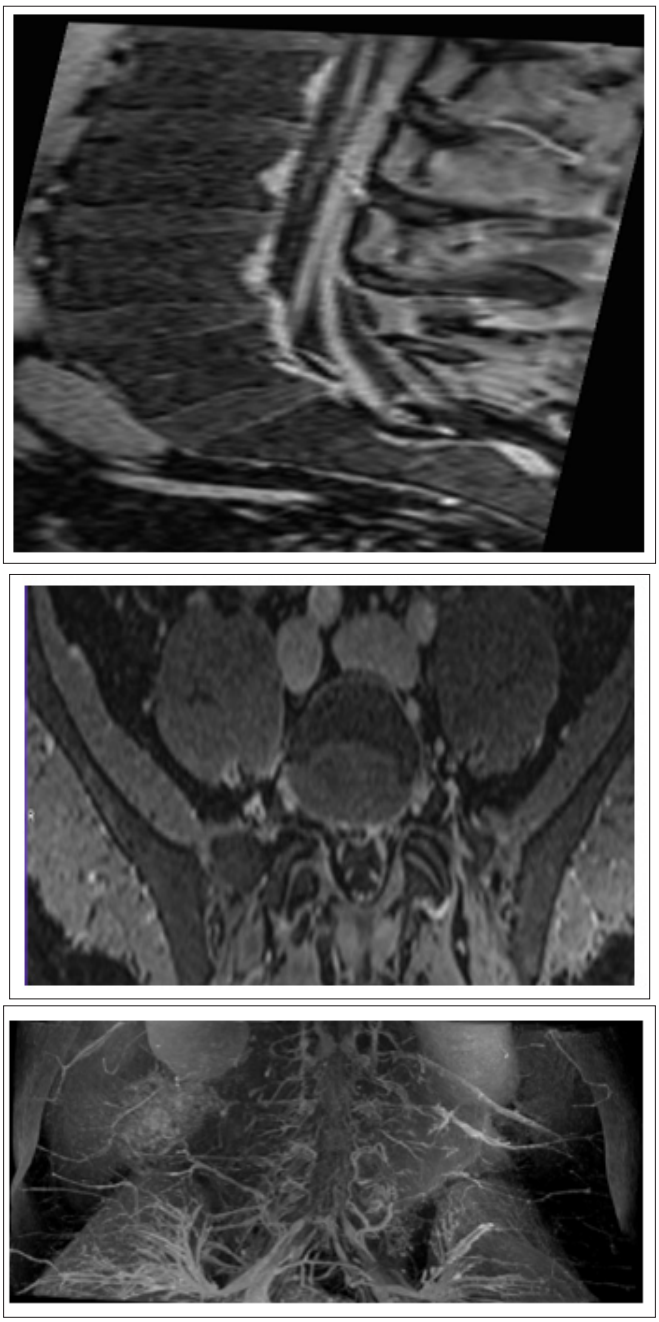


Figure 10: Magnetic Resonance Imaging on day 6, Abnormal Enhancement of Lumbosacral Nerve Roots, in Sagittal and Axial Projections after Gadolinium Contrast, 3D

Table 2: The Dynamics of Neurological Picture and Weight Changes During 56 days of Hospitalization

Neurological condition	Days	Weight
Mechanical ventilation	2	
No reflexes	3	95kg
No improvement	12	
Triggers ventilator	16	
Pupillary reaction	17	
Slight nodding	23	
Moves tongue	30	
Moves shoulders	35	82kg
Moves hands, slight elbow flexion, tones thighs	45	
Moves fingers of hands	47	79kg
Spontaneous breathing	48	
Holds arms above bed	55	78kg
Tracheostomy extracted	57	
Flexion in knees	58	

Discussion

While the vast majority of GBS presentations are transient in nature, intensivists encounter the most severe, fulminant and debilitating forms, with potential for complications such as sepsis, thromboembolism, muscle wasting, joint stiffness, that require multidisciplinary and long term complex health and physiotherapy care, extensive social and psychological support for the patient and the family. Hundreds of dedicated healthcare workers and thousands of hours are invested in these patients, who are typically young or middle aged without severe comorbidities. Moreover, these mentally challenged cases trigger our conscience to respect our limits and reach to depth to enhance our potencies. Our patient has had an extremely severe form of ascending motor weakness with egregious autonomous dysfunction , requiring prolonged sedation and mechanical ventilation, multiple efforts at blunting the sympathetic dysautonomia and complex management of organ functions. We have captured early, using quantitative PCR technology the presence of replicating CMV virus in the BAL and in blood, clinically leading to severe interstitial pneumonia and acute hepatitis / transaminitis, with highly activated CD3+CD8+T cell engagement, impaired oxygenation requiring high oxygen fractions and proning, and increased levels of ALT, indicating liver injury. Beyond the conventional, high dose immunoglobulin therapy we employed targeted virostatic and antibacterial therapy. While conventional clinical and laboratory monitoring was applied in abundance , immune profiling had been applied to a weaker extent due to the lack of funding and technical availability. Comprehensive autoantibody screening had been employed with overall negative results. Autoantibodies signaling an autoimmune condition may appear early, even years prior to clinical disease manifestation and may be dominant driving forces or contributory in autoimmune conditions with predominant T cell phenotype, such as those with male predominance [13]. Autoantibodies, for example Anti GD1 activates complement and calpain and endure axonal injury at motor nodes of Ranvier [14]. GBs may be a clinical presentation of variable phenotypes based on etiology and host phenotype. Alternatively, it is reasonable to hypothesize that direct CD3+CD8+ T cell cytotoxicity could have had the potential to elicit axonal damage via conventional means of perforin/granzyme B/caspase 3 pathways. We have examined complement activation, without dominant abnormalities in blood, C4 levels were increased with concomitantly normal C3 levels, but C50, indicating classical pathway activation was decreased. CMV is a DNA virus, with population prevalence of colonisation by embedding in a latent intracellular form in about 90% of adults and acute infection is likely followed by recall immune response. CMV typically induces a very strong CD3+CD8+ cytotoxic T cell response that is MHCI mediated hence in effort to eliminate the intracellular virus by means of perforin mediated granzyme B

and proapoptotic caspase production, inevitably the host cells harbouring the virus become damaged. Conventionally, CMV is becoming reactivated upon stress in immunocompromised individuals, like transplant recipients, occasionally relatively healthy adults become ill. CMV uses several receptors to enter host cells, influencing tissue tropism. It is PDFGR (platelet-derived growth factor receptor), EGFR(epidermal growth factor receptor), neuropilin2 , TLR2integrins as co- receptors, for example CD90 and CD147 [15]. The virus has tropism towards epithelial , endothelial cells, stem cells, neurons, monocytes, macrophages and dendritic cells, while EBV occupies chiefly immature B cells [16]. Upon stimulation the episomal DNA may become activated. HCMV spreads primarily via cell to cell contact , resisting neutralizing antibody effect, but cell free virion transfer is possible, too.

In our scenario a bacterial pharyngitis could have led to enhanced sensitivity towards viral reactivation via monocyte to macrophage maturation induced by proinflammatory cytokines. While monocytes are permissive for viral replication, neutrophils behave as carriers for the virus.

The typical CMV related immune response is further characterised by persistent T cell activation, with viral IL-10 production leading to NK cell inhibition , and impaired MHCII maturation [17].

We also found another intracellular pathogen DNA in BAL, Mycoplasma pneumoniae. The presence of MP can be a coincidental finding as it may be present in up to 58% of healthy individuals, but associations with GBS based on case studies are reported in the literature [18-20].

We used no statistical analysis due to the nature of the study.GBS pathogenesis is perhaps multifaceted, for example complement MAC mediated axonal injury has been reported [21,22].

We believe that further dissection of the mechanism of axonal injury and immunological behavior of the virus could potentiate our efforts for future therapies and prevention. As opposed to seasonally mutating viruses, CMV has a relatively more stable generic material, suggesting opportunities for vaccination. While CMV inducedGBS is not a frequent event, there are multitude of disease associations reported with the virus or viral particles, such as glioblastoma, Alzheimer disease,and atherosclerosis [23].

In case of EBV Sjogren, SLE, nasopharyngeal, gastric cancer, Hodgkin Disease or Burkitt lymphoma had been reported [24,25]. Moreover CMV and EBV, in active or latent form have a profound potential on modulating the immune response in general, rendering the immune response deviant in the individual and explaining individual susceptibility features without need for viral reactivation. Further the high potential of viral IL-10, that may behave as active cytokine or decoy, raises the issue of frequently encountered low vitamin D levels in these patients, triggering further thoughts to decipher causations and associations. As per immune modulation, CMV may induce chronic inflammation induced immune senescence, loss of T cell repertoire and telomere shortening , rendering the host susceptible for tumor development in an exhausted T cell milieu . There are several antiviral drugs available to tackle CMV, albeit not without adverse effects.

EBV, another sneaky DNA herpesvirus that is similar in its capability of evading and modulating immunity, is currently without viable treatment options. It is our observation that viral replication of CMV, EBV and SarsCovid2 creates a hypermetabolic state in the host, contributing to autonomic dysfunction, to muscle wasting and ultimately energy exhaustion. These patients typically require high doses of sedatives to achieve a state of calmness and vigorous efforts at managing heart rate, and or blood pressure. PRR engagement, particularly via TLR4 engagement contributes to the arrhythmogenic natures of above pathogens, beta antagonists are used habitually. The minimalist constitution of pathogens, particularly viruses and atypical pathogens requires host resources for survival and may tune the host for a hyperinflammatory energy consuming phenotype. Energy supplementation of these patients, even in the case of a functioning gastrointestinal system is a challenge , intermediaries and immediate energy resources seem to be reasonable to implement pathogen clearance. In case of CMV, endothelial tropism of the virus may play a contributory role in sympathetic reaction.

There are several case reports associating CMV and GBS [26]. Our study is unique in that the temporospatial occurrence of replicating CMV virus was demonstrated with an ongoing activated CD3+CD8+T Cell response, suggesting a possible pathogenetic mechanism. A similar mechanism is known for hepatitis B infection, where liver injury is attributed to CD8 T cell cytopathic effect rather than the virus itself [27]. Some studies examined and reported CMV presence in Schwann cells using electron microscopy. Further tissue immunostaining could enhance the association. We further demonstrate that active CMV infection may occur in seemingly immunocompetent hosts. We emphasize that more detailed immune profiling would be desirable in GBS patients, to establish phenotypes. Beyond conventional CMV phenotypes, occurrences and less frequent associations, the immunomodulatory role of persistent viruses is highlighted. As per CMV a role in immune consumption, senescence and exhaustion as well as dominating the T cell epitope repertoire, with potential negative impact on tumor genesis as a delayed indirect consequence of persistent viral presence is hypothesized.

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