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Acute Facial Dyplegia and Rhabdomyolisis: Case Report and Review of Literature

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Abstract: In 1916, Guillain, Barré and Strohl reported on two cases of acute flaccid paralysis with high cerebrospinal fluid protein levels and normal cell counts-novel findings that identified the disease we now know as Guillain–Barré syndrome (GBS). 100 years on, we have made great progress with the clinical and pathological characterization of GBS. GBS is an acute/subacute-onset polyradiculoneuropathy typically presenting with sensory symptoms and weakness over several days, often leading to quadriparesis. Approximately 70% of patients report a recent preceding upper or lower respiratory tract infection or gastrointestinal illness. The interplay between the microbial and host factors that dictate whether and how the immune response shifts towards autoreactivity is still unclear, and nothing is known about the genetic and environmental factors that affect an individual's susceptibility to the disease. Facial Diplegia with Paresthesias is a rare localized variant of GBS in which patient presents with simultaneous facial diplegia, distal limb paresthesias and minimal or no motor weakness. Treatment with intravenous immunoglobulin or plasma exchange is the optimal management approach, alongside supportive care. A common misconception is that the Guillain–Barré syndrome has a good prognosis-but up to 20% of patients remain severely disabled and approximately 5% die, despite immunotherapy. We report the case of a woman with acute facial dyplegia and rhabdomyolisis improved after immunoglobulin treatment.

Keywords: Facial Dyplegia, Hyperckemia, Guillain Barrè Syndrome

1. Introduction

We report the case of a 63 years-old young woman who was admitted to the hospital due to persistent diffuse myalgias with myoglobinuria from about three days. About seven days before she come back from a travel to Cambodia, lasted two weeks. Once at home she suffered for about three days from nausea and vomit without fever, for which she taken antiemetics with benefits.

Her medical history was unremarkable, except for previous excision of basocellular carcinoma, and untreated nodular goiter.

Neurological examination was normal, she only referred diffuse painful myalgias. Cranial nerves, reflexes, strength and sensitivity resulted all normal. She performed blood exams which showed raised value of creatine kinase (2387 UI/L normal value 30-135). Renal function resulted normal. Electromyography resulted normal.

The following day she suddenly developed facial dyplegia, with hypophonia, which was evaluated as grade 6 with House and Brackmann Scale. She also started to complain, after three hours, of worsening dysartria and severe dysphagia.

Lumbar puncture wasn't performed, due to the presence of Arnold Chiari malformation at brain computer tomography and magnetic resonance imaging. She was promptly moved to Intensive care Unit (ICU) due to worsening of dyspnea. Intravenous immunoglobulins 0.4 g/Kg were promptly administered for five days.

While in ICU her Creatine kinase values promptly improved and after two days resulted normal.

Electromyography and electroneurography, showed demyelinating type of facial palsy and involvement of upper arms nerves. Chest X-Ray, abdomen and ultrasound resulted negative.

Other blood exams, particularly antinuclear antibodies, extractable nuclear antigens antibodies, anti-double strand

DNA antibody, serum complement fractions, liver function tests, blood cells count, electrophoresis, hepatitis B, C, human immunodeficiency virus, Syphilis, Coxackievirus, Herpesvirus pattern, Borrelia, resulted all negative, except for mild reduction of thyroid-stimulating hormone (0.181 mcUI/ml normal value 0.350-4.940). A diagnosis of subclinical hyperthyroidism was performed by Endocrinologists and thiamazole 5 mg 1/day was started.

Otorinolaryngologists and logopedists reported oral phase dysphagia and transcutaneous neuromuscular electrical stimulation was performed.

She was moved to Rehabilitation Unit after 15 days from inhospitalization. Right facial weakness slowly improved in about one month, while at left she developed hemifacial spasm for which she was treated with botulinum toxin at lip elevator and zigumaticus maior muscles.

2. Review of Literature

2.1. Guillain Barre' Syndrome (GBS) Definition

Guillain-Barré syndrome is the most common cause of acute flaccid paralysis in the developed world [1]. GBS and its related variants comprise a group of acute radiculoneuropathies, with an inflammatory or autoimmune pathophysiology [2]. The classical form of GBS is also known as acute inflammatory demyelinating polyradiculoneuropathy (AIDP) and accounts for 90% of cases seen in the United States and Europe. Acute inflammatory demyelinating polyradiculoneuropathy typically presents with progressive flaccid paralysis that is often ascending and reaches its nadir within 4 weeks. After a plateau phase of variable duration, recovery begins and often results in a significant return of function [1-2].

2.2. Epidemiology and History

The earliest description of GBS dates to 19th century regarding an afebrile generalized paralysis by Wardrop and Ollivier in 1834. Other important landmarks are Landry's report in 1859 about an acute, ascending, predominantly motor paralysis with respiratory failure, leading to death and Osler's in 1892 description of a febrile polyneuritis Guillain, Barré, and Strohl in 1916 described a benign polyneuritis with albumino-cytological dissociation in the cerebrospinal fluid (CSF) and the first report regarding pathology of GBS was by Haymaker and Kernohan in 1949 who reported that edema of the nerve roots was an important change in the early stages of the disease [3-6]. Asbury, Arnason and Adams in 1969 established that the essential lesion is due to perivascular mononuclear inflammatory infiltration of the roots and nerves [7].

With the near-eradication of polio, GBS has become the most common cause of acute flaccid paralysis in developed countries. The incidence of GBS is best known for the United States and Europe, with a consistent annual incidence of 0.84 to 1.9 cases/100 000 population [8]. Despite improved recognition and treatment, GBS continues to be a severe disease. One-quarter of patients will require mechanical ventilation for respiratory failure or airway protection and 3-11% will die of GBS-related complications [9-11]. Although most patients make substantial recoveries, 20% to 38% experience residual disability and more than one-third are forced to change their work and social lives [9-12].

In a systematic literature review incidence resulted lower in children at 0.34 to 1.34/100,000 [8, 12]. In comparison to younger cases, the incidence of GBS increases after age 50 years from 1.7/100,000 to 3.3/100,000 [8, 12].

Half of GBS patients have an antecedent infection, usually less than 4 weeks prior to symptom onset [13]. The most common infections in adults are respiratory (22–53%) and gastrointestinal (6–26%). Preceding infections are more common in children (67–85%) with a greater percentage of respiratory (50–70%) than gastrointestinal (7–14%) [8, 13]. The most commonly recognized pathogens include Campylobacter Jejuni, Cytomegalovirus, Epstein–Barr virus, and Mycoplasma pneumoniae [14]. Most cases of GBS are sporadic, although rare clusters have been reported after bacterial enteritis [15].

The association of GBS with influenza vaccination was first reported in 1976, when the seasonal vaccination campaign was stopped in the United States due to an excess of GBS cases (relative risk [RR]: 7-8) [16]. However, few studies addressing the potential relationship of GBS to influenza vaccination were published between 1976 and 2009. Since the pandemic outbreak of influenza A in 2009, the vaccine A/H1N1/2009 were rapidly developed, manufactured and commercialized, and surveillance systems were reinforced, adapted or set up with the aim of identifying as early as possible any incidence excess of GBS, notably in the United States, wherein an increased risk of GBS associated to influenza vaccine was found [17, 18]. A recent research performed in 2019 concluded that GBS should be considered an infrequent adverse effect of influenza vaccination, which should not negatively influence the vaccination acceptance [19].

It is now clear that GBS is a true syndrome that encompasses several specific disorders, including the demyelinating form, acute inflammatory demyelinating polyneuropathy (AIDP) and axonal forms, acute motor axonal neuropathy (AMAN) and acute motor and sensory axonal neuropathy (AMSAN) [13, 19]. Other clinical presentations include the Miller Fisher syndrome (a triad of ophthalmoplegia, ataxia, and areflexia), pure sensory neuropathy/neuronopathy, pandysautonomia, oropharyngeal variant, and overlap syndromes [13, 19].

2.3. Variants

2.3.1. Acute Inflammatory Demyelinating Polyneuropathy (AIDP)

Acute inflammatory demyelinating polyneuropathy accounts for the majority (probably 80%) of patients [13, 19]. It is the most common form of GBS in the United States and Western Europe [13, 19]. AIDP typically presents with progressive flaccid paralysis that is often ascending and

reaches its nadir within 4 weeks [1, 19]. After a plateau phase of variable duration, recovery begins and often results in a significant return of function [1, 19]. Associated clinical features include areflexia, variable sensory loss, albumin-cytological dissociation in the cerebrospinal fluid, and variable degrees of demyelination on electrophysiologic testing [1, 19].

In AIDP, pathological studies reveal patchy multifocal mononuclear cell infiltrates throughout the peripheral nervous system, with the distribution of inflammation often corresponding to the pattern of clinical deficit [20]. Activated macrophages invade intact myelin sheaths resulting in myelin damage and demyelination [20].

Alternative hypotheses focus on the importance of activated helper (CD4) T cells, which should react against specific antigens on the surface of Schwann cells or the myelin sheath thereby directing activated macrophages to this region, or on humoral immunity, expecially at early stages of disease [20].

2.3.2. Acute Inflammatory Demyelinating Polyneuropathy (AMAN)

Acute motor axonal neuropathy (AMAN) is the most common GBS variant, accounting for as many as 5–10% of cases in the Western world, with much higher incidence in Asia, particularly for childhood onset GBS, where rates approach 50% [21].

AMAN is generally characterized by rapidly progressive symmetrical weakness and ensuing respiratory failure [22]. Patients typically have high titers of antibodies to gangliosides (ie, GM1, GD1a, GD1b). Inflammation of the spinal anterior roots may lead to disruption of the blood-CNS barrier [23]. Biopsies show Wallerian-like degeneration without significant lymphocytic inflammation. Prognosis is often quite favorable. Although recovery for many is rapid, severely disabled patients with AMAN may show improvement over a period of years [22, 23].

Nearly 70-75% of patients with AMAN are seropositive for Campylobacter, with the majority of cases of AMAN being associated with preceding C Jejuni diarrhea. Studies of AMAN post C. Jejuni infection have provided strong support for the concept of molecular mimicry in the pathophysiology of immune neuropathy [21, 23]: epidemiological association between GBS and C. jejuni infection, identification of antibodies directed against host antigens (gangliosides) in affected patients, identification of microbial mimics of the target antigen (lipo-oligosaccharides extracted from C. jejuni were found to be identical to GM1 ganglioside), and experimental development of neuropathy in an animal model [24, 25].

2.3.3. Acute Motor-Sensory Axonal Neuropathy (AMSAN)

Acute motor–sensory axonal neuropathy is similar to AMAN, but with concurrent involvement of the sensory axons, and has a pathomechanism similar to that of AMAN, including frequent antibodies against GM1 and GD1a gangliosides following C. jejuni infection. It often carries a much worse prognosis but the reason of different prognosis is

unknown [13, 24].

2.3.4. Miller-Fisher Syndrome

The second most common GBS variant is the Miller-Fisher syndrome (MFS), characterized by a triad of ophthalmoplegia, areflexia and ataxia. MFS accounts for up to 5% of GBS cases in the Western world, with rates as high as 25% in Japan and other Asian countries [25]. This syndrome has been strongly linked to serum anti-GQ1b antibodies in up to 85% of cases, and less frequently to the closely related ganglioside GT1a, though there is strong cross-reactivity between these two antibodies [26, 27]. There is even greater specificity for the presence of anti-GQ1b antibodies in CSF [28]. This antibody reacts with paranodal myelin epitopes strongly expressed in oculomotor nerves, dorsal root ganglia and muscle spindles, and cerebellar molecular layer, a distribution that fits well with cardinal clinical manifestations [29]. Incomplete forms with acute ophthalmoparesis without ataxia or acute ataxic neuropathy without ophthalmoplegia have also been described [21, 29]. There may also be overlap between subtypes. Patients with MFS or Bickerstaff's brainstem encephalitis who develop limb weakness can be diagnosed as having overlap with GBS. Patients with prominent ophthalmoplegia or ataxia must have overlap with MFS [30].

2.3.5. Pharyngeal-Cervical-Brachial (PCB)

Pharyngeal-cervical-brachial variant of Guillain–Barré syndrome is defined by rapidly progressive oropharyngeal and cervicobrachial weakness associated with areflexia in the upper limbs [31]. Serial nerve conduction studies suggest that PCB represents a localised subtype of Guillain–Barré syndrome characterised by axonal rather than demyelinating neuropathy [31]. Power in the lower limbs is usually preserved or only mildly affected, indicating that PCB represents a localised subtype of GBS [32]. It presents with neck, arm, and oropharyngeal weakness, and upper extremity areflexia. Leg strength and reflexes are usually preserved. Findings may be asymmetric [32]. Electrophysiologic features may include mild conduction velocity slowing, low upper extremity CMAP amplitudes, or can be normal [33]. Up to 40% of patients have anti-GQ1b IgG antibodies [31, 33]. Very often patients presenting with PCB are initially misdiagnosed as having brainstem stroke, myasthenia gravis or botulism, which can often be excluded on clinical history and examination alone [31, 33].

Clinical, immunological and neurophysiological studies have shown that PCB forms a continuous spectrum with Miller Fisher syndrome and represents a localised form of axonal GBS [31, 33].

2.3.6. Pure Sensory Guillain-Barré Syndrome (GBS)

Sensory Guillain-Barré syndrome (GBS) is an acute demyelinating neuropathy that presents clinically with involvement of the sensory peripheral nerve only. However, the existence of a purely sensory form of GBS remains subject to controversy, since these cases always demonstrate a degree of motor weakness or abnormalities in motor nerve

conduction studies (NCSs) and are difficult to distinguish from acute sensory neuronopathy [34]. To date, only a few cases of pure sensory GBS have been reported, with the majority of cases being anecdotal and few studies describing a peripheral nerve pathology. Thus, the clinical and pathological features of sensory variant GBS have not been well characterized, and reduced awareness of these features has resulted in delays in the diagnosis and treatment [35].

2.3.7. Facial Diplegia with Paresthesias (FDP)

Facial Diplegia with Paresthesias is a rare localized variant of GBS in which patient presents with simultaneous facial diplegia, distal limb paresthesias and minimal or no motor weakness [36, 37]. Deep tendon reflexes are generally absent in FDP variant but rarely can be present or even exaggerated [38]. Diagnosis of GBS is based upon good clinical examination, Cerebrospinal fluid (CSF) analysis and clinical neurophysiological studies. CSF examination done after first week of disease onset often shows albumin cytological dissociation. Nerve conduction studies and Electromyography are very helpful in establishing diagnosis of GBS. NCV studies in FDP often show demyelinating type of neuropathy but axonal polyneuropathy has been also described [39].

2.3.8. Acute Pandysautonomia

Acute pandysautonomia presents with orthostatic hypotension, gastroparesis, constipation, diarrhea, ileus, micturition problems, sudomotor/pupillary abnormalities, and neuropathic pain, at times severe, reaching its peak within 1 to 3 weeks [40]. Some, but not all, patients experience sensory loss. Symptoms are mostly due to parasympathetic and cholinergic dysfunction. Half of patients experience significant disability [40].

2.4. Pathogenesis

GBS was long considered a homogeneous disorder whose severity was related to the extent of axonal injury arising from demyelinisation; however, it is now known that there are various phenotypes, including acute inflammatory demyelinating polyneuropathy (in which the immune-related injury affects the myelin sheath and related Schwann cells) and acute motor axonal neuropathy, in which the membranes of nerve axons are the primary target [41].

GBS occurs in healthy people, and is not typically associated with an autoimmune or other systemic disorder. It is a mainly humoral mediated rather than T-cell mediated disorder and, in this context, acute motor axonal neuropathy appears as an antibody-mediated attack driven by molecular mimicry between microbial (i.e. glycans) and axolemmal surface molecules (i.e. GM1 and GD1a gangliosides) [42]. On the contrary, the immunological mechanisms involved in acute inflammatory demyelinating polyneuropathy is less clear because of the wide range of immune stimulants that can cause it and the absence of specific antibody biomarkers [43].

Although the distinction between acute motor axonal neuropathy and acute inflammatory demyelinating polyneuropathy seems to be conceptually clear, the margins between the two conditions are not so well defined. Electrophysiological findings are often ambiguous as they indicate acute inflammatory demyelinating polyneuropathy in the early phases and acute motor axonal neuropathy later, or both conditions simultaneously [44].

2.5. Diagnosis

2.5.1. Electrophysiologic Features

When GBS is suspected, electrophysiologic studies are essential to confirm the diagnosis and exclude its mimics. The finding of multifocal demyelination on early electrodiagnostic testing (or repeated a week later) is extremely helpful in confirming the diagnosis of AIDP with a high sensitivity and specificity. Needle electrode examination is non-specific as it demonstrates reduced recruitment initially and fibrillations potentials three to four weeks after onset [45]. The earliest findings in AIDP are prolonged F-wave latencies or poor F-wave repeatability due to demyelination of the nerve roots. This is followed by prolonged distal latencies (due to distal demyelination) and temporal dispersion or conduction block. Slowing of nerve conduction velocities is less helpful as it tends to appear two to three weeks after the onset. However, the sensitivity of nerve conduction studies (NCS) based on reported criteria may be as low as 22% in early AIDP, rising to 87% at five weeks into the illness [46, 47].

2.5.2. Lumbar Puncture

A lumbar puncture is often performed in patients with suspected GBS. Importantly, this procedure especially should be done to exclude other diagnoses rather than to confirm GBS. A combination of elevated protein level and normal cell counts in the CSF (termed albumin cytological dissociation) is considered a hallmark of GBS (48). A frequent misconception, however, is that albumin cytological dissociation must always be present to confirm the diagnosis, as only 64% of patients with GBS have this feature [48]. Elevated CSF protein levels are found in approximately 50% of patients in the first 3 days after onset of weakness, which increases to 80% after the first week [49]. CSF cell counts >50 cells per μl should cast doubt on the diagnosis of GBS, and other differential diagnoses should be considered, such as leptomeningeal malignancy, lymphoma, cytomegalovirus radiculitis, HIV polyneuropathy and poliomyelitis [48, 50]. In the Miller Fisher syndrome, the proportion of patients with raised CSF protein has been reported to range from 25%in the first week to 84% in the third week [50].

2.5.3. Serum Antiganglioside Antibodies

Although antiganglioside antibodies are involved in the pathogenesis of GBS, their role in diagnosis has not been established. In general, the frequency of each specific antibody is low, implying that the negative predictive value of detection tests will also be low, and that the negative test

findings cannot, therefore, exclude GBS as a diagnosis [50, 51]. Possible exceptions to this rule might be anti-GQ1b antibodies, which are present in the serum of at least 90% of patients with MFS [51], and anti-GM1 and anti-GD1a IgG antibodies, which are frequently found in patients with AMAN [52].

2.5.4. Brain and Spinal Magnetic Resonance Patterns

Typical findings in Guillain-Barré syndrome are surface thickening and contrast enhancement on the conus medullaris and the nerve roots of the cauda equine. The most common site of enhancement in Guillain–Barré syndrome is considered to be anterior nerve roots, although enhancement of the posterior nerve roots is also seen [53, 54].

2.6. Therapy

2.6.1. Intravenous Immunoglobulins (IVig)

The postulated mechanisms of action of IVIg in neuromuscular disorders include interference with costimulatory molecules involved in antigen presentation and modulation of autoantibodies, cyotokines and adhesion molecules production as well as macrophage Fc receptor. It also disrupst complement activation and membrane attack complex formation [45, 55]. Sialylated IgG Fc fragments are important for the in vivo activity of intravenous immunoglobulin since they initiate an anti-inflammatory cascade through the lectin receptor SIGN-R1 or DC-SIGN [45, 55]. This leads to upregulated surface expression of the inhibitory Fc receptor, Fc gamma receptor IIb, on inflammatory cells, thereby attenuating autoantibody-initiated inflammation [45, 55]. According to the standard treatment regimen, immune globulin is given at a total dose of 2 g per kilogram of body weight over a period of 5 days [55].

2.6.2. Plasma Exchange

Plasma exchange was the first treatment that was found to be effective in hastening recovery in patients with the Guillain–Barré syndrome and it appeared to be most effective when it was started within the first 2 weeks after disease onset in patients who were unable to walk [56]. An electrophysiological examination is not always required for the initiation of immunotherapy. Plasma exchange nonspecifically removes antibodies and complement and appears to be associated with reduced nerve damage and faster clinical improvement, as compared with supportive therapy alone [57]. The usual empirical regimen is five exchanges over a period of 2 weeks, with a total exchange of 5 plasma volumes [56, 57].

2.7. Aan Practice Parameters

They recommend PE for nonambulant adult patients with GBS who seek treatment within four weeks of the onset of symptoms (level A). PE should also be considered for ambulant patients examined within two weeks of the onset of symptoms (level B). IVIg is recommended for nonambulant adult patients with GBS within two (level A) or possibly four weeks (level B) of the onset of neuropathic symptoms. It also indicates that sequential treatment with PE followed by IVIg, or immunoabsorption followed by IVIg is not recommended for patients with GBS. As stated earlier, corticosteroids are not recommended for the management of GBS. In children with severe GBS, PE and IVIg are treatment options [57].

2.8. Rhabdomyolisis and Guillain Barre'

Rhabdomyolysis is a complex medical condition involving the rapid dissolution of damaged or injured skeletal muscle. This disruption of skeletal muscle integrity leads to the direct release of intracellular muscle components, including myoglobin, creatine kinase (CK), aldolase, and lactate dehydrogenase, as well as electrolytes, into the bloodstream and extracellular space. Rhabdomyolysis ranges from an asymptomatic illness with elevation in the CK level to a life-threatening condition associated with extreme elevations in CK, electrolyte imbalances, acute renal failure (ARF), and disseminated intravascular coagulation [58].

We performed a literature review considering the terms "Hyperckemia and Guilain-Barrè", "Creatinkinase and Guillain-Barrè", "Creatinkinase and AIDP", "Hyperckemia and AIDP", "Rhabdomyolisis and Guillain-Barrè". "Rhabdomyolisis and AIDP", "Hyperckemia and facial diplegia", "Creatinkinase and facial diplegia", "Rhabdomyolisis and facial diplegia".

Table 1. Describes case reports in literature with rhabdomyolisis and Guillain Barrè Syndrome ABBREVIATIONS: F= female, M= male, U=unknown data.

Author	Age	Sex	Neurological symptoms	Creatinkinase	General symptoms
Hargis M 2017	45	M	Progressive weakness of the lower extremities. Facial and left, more than right, proximal upper extremity weakness.	U	none
Van Den Berg M et al 2016	60	F	muscle weakness, sensory disturbances, hyporeflexia in her limbs and facial diplegia	raised	diarrhoea, fever and an unsteady gait after returning from Surinam.
Sanena A et al 2014	24	F	20 days history of weakness of both lower and upper extremities	7002UI/L	none
Gupta R et al 2016	25	F	acute respiratory distress syndrome (ARDS), difficulty in moving all four limbs	11510IU/L	high-grade fever, anorexia, malaise, dry cough and breathlessness
Scott AJ et al 2000	25	M	weakness of his hands and legs which progressed over several hours until he was unable to walk.	10,150IU/l	anterior chest pain radiating to the inner aspect of both arms
Satoh J et al 1999	21	M	Progressive weakness after an episode of diarrhea, worsening to rapid quadriplegia and areflexia	1917UI/L (raised two weeks after plasmapheresis)	diarrhea

Table 1. *Continue.*

Author	Electroneurography	Csf	Aetiology	Treatment
Hargis M 2017	slowed conduction velocities without conduction block, and absent F-waves consistent with acute inflammatory demyelinating polyradiculoneuropathy	U	U	Plasmapheresis
Van Den Berg M et al 2016	Confirmed GCS	Albumin-cytological dissociation protein of 68mg/dL, glucose of 90mg/dL and a CSF cell count of 2 leukocytes/mm	Zika virus	Intravenous immunoglobulins
Sanena A et al 2014	acute motor and sensory axonal neuropathy		U	U
Gupta R et al 2016	reduced amplitude of compound motor action potential in all the nerves tested with relative preservation of the nerve conduction velocities, F waves not recordable. Sensory nerve conduction: reduced amplitude of the sensory nerve action potentials with relative preservation of the nerve conduction velocities. Electromyogram (EMG): fibrillation potentials and positive sharp waves	U	Anti-mycoplasma IgG antibodies were persistently elevated 84 U/ml after one month (>30 U/ml positive)	U
Scott AJ et al 2000	severe demyelinating neuropathy affecting both proximal and distal segments of motor and sensory nerves in all four limbs.	Protein 0.28g/l. No cells	U	plasmapheresis
Satoh J et al 1999	severe axonal degeneration of motor nerve terminals	Protein 235mg/dl without pleocytosis	Campylobacter jejuni enteritis	plasmapheresis

3. Discussion

Our patient was in-hospitalized to rhabdomyolisis quickly followed by acute facial diplegia with dysphagia, hypophonia and dyspnea quickly worsening. Due to brain computer tomography and magnetic resonance imaging of Arnold Chiari malformation, we decided not to perform lumbar puncture but to move the patient to ICU, where we promptly administered IVig on the basis of the electroneurography results (myopathic pattern was excluded by electromyography). Considering the quick improvement of bulbar symptoms diagnosis of AIDP was confirmed. We couldn't find infective source (laboratory tests for Zika Virus, an arthropod-borne virus (arbovirus) in the genus Flavivirus and the family Flaviviridae which had been isolated also in Cambodia at the beginning of 2016, couldn't be available).

Modestly raised levels of CK (up to seven times the upper limit of normal) have been documented in the early stages of GBS, correlating with the occurrence of pain [59], which was a presenting symptom in our patient. The cause of raised levels of CK in early GBS is uncertain. Muscle denervation can result in release of muscle enzymes. The muscle pathology in this patient was typical of acute rhabdomyolysis, which was not found in muscle biopsies from patients with GBS in Ropper's series [59]. Negative history of trauma, illicit drug or alcohol abuse, toxin consumption or recurrent episodes of rhabdomyolysis in the past ruled out the other possible causes of rhabdomyolysis [59].

4. Conclusion

In literature we haven't found cases of rhabdomyolisis associated to facial dyplegia. In such cases is important to promptly diagnose and treat AIDP without forgetting to monitor renal function and creatin kinase value.

Conflicts of Interest

All the authors denies any conflict of interest.

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