Case Report

Pain abdomen and cardiac arrest as peculiar presenting symptoms in AIDP- AMSAN variety

E.A.Ashok Kumar[1*], Muppala Hanvitha[2]

[1]Professor & HOD, Department of General Medicine, Maheshwara Medical College & Hospital, Sangareddy Dist., Telangana, India
[2]Post Graduate, Department of General Medicine, Malla Reddy Institute of Medical Sciences, Hyderabad, Telangana, India
[*]Corresponding author email: **ashokedla@gmail.com**

International Archives of Integrated Medicine, Vol. 9, Issue 10, October, 2022.
Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 5-10-2022 **Accepted on:** 12-10-2022
Source of support: Nil **Conflict of interest:** None declared.

How to cite this article: E.A.Ashok Kumar, Muppala Hanvitha. Pain abdomen and cardiac arrest as peculiar presenting symptoms in AIDP- AMSAN variety. IAIM, 2022; 9(10): 43-51.

Abstract

AIDP (Acute Inflammatory Demyelinating Polyneuropathy) is a heterogenous condition encompassing several variants. It is a post infectious neurological disorder with an autoimmune pathogenesis with molecular mimicry mechanism. They present commonly with symmetrical ascending type paralysis and absent or diminished deep tendon reflexes. Cranial nerve palsies may or may not be present. The diagnosis is based on the clinical signs and symptoms, nerve conduction studies and cerebrospinal fluid analysis. We herewith report a rare case of AIDP, where the patient came walking to the emergency room with pain abdomen as the only complaint and had no neurological deficits at the time of presentation, hours later, the patient went into cardiac arrest, the cause of which was later thought to be dysautonomia and respiratory failure. The next day, patient developed motor weakness and multiple cranial nerve palsies which is an overlap of AMSAN and Acute Ophthalmoplegia. It is extremely uncommon to present with pain abdomen and cardiac arrest as presenting features in AIDP, AMSAN (Acute Motor Sensory Axonal Neuropathy) variety.

Key words

Acute Inflammatory Demyelinating Polyneuropathy, Acute Motor Sensory Axonal Neuropathy, Dysautonomia, Ophthalmoplegia, Cardiac arrest.

Introduction

AIDP is an important cause of acute flaccid paralysis worldwide. Octave *Landry* has reported the first case of AIDP in 1859 [1]. Barre and Strohl described the CSF findings in AIDP, also known as Landry Guillain-Barré Strohl syndrome. It has an autoimmune pathogenesis, triggered by preceding conditions such as infections/ vaccinations/ and surgeries. It occurs worldwide with an annual incidence of 1.3 cases per 100000 population, affecting children and adults of both genders [2].

AIDP is a heterogenous condition encompassing several variants. It most commonly presents as ascending, symmetrical weakness with decreased/ absent deep tendon reflexes. It is one of the most serious emergencies in neurology and timely treatment improves outcome.

Epidemiology

AIDP can occur at any age. There is a higher incidence among young adults, due to increased risk of infections and in the elderly due to decreased immune suppression responses. AIDP has a male preponderance [3]. Two third of AIDP cases have antecedent viral infections.

Variants

AIDP has several variants based on the mode of nerve fiber injury (demyelinating or axonal) and the types of nerve fibers involved (motor, sensory, combined sensory and motor, cranial nerves, or autonomic nervous system).

1) AIDP (Acute Inflammatory Demyelinating Polyneuropathy): It is the most common variant and has a demyelinating pathology.
2) AMAN (Acute Motor Axonal Neuropathy): It is an axonal variant with only motor involvement. It was first described in China in 1993. Therefore, it is also known as Chinese paralytic illness. [4, 5].
3) AMSAN (Acute Motor Sensory Axonal Neuropathy): It is an axonal variant with both motor and sensory involvement.
4) MFS (Miller Fisher Syndrome): It consists of ophthalmoplegia, ataxia, and areflexia without any weakness. The patient's sensorium is normal. They have Anti GQ1b antibodies.

Rare variants

- Bickerstaff brainstem encephalitis (BBE) is a variant of MFS. It consists of altered sensorium, ataxia, and ophthalmoparesis. Paradoxical hyperreflexia is present [6].
- The pharyngeal-cervical-brachial motor variant- it is present in about 3% patients. They present with ptosis, facial, pharyngeal, and neck flexor muscle weakness that spreads to the arms and lower limbs are spared.
- Paraparetic motor variant- It selectively affects the lower limbs. They have areflexia. It is associated with back pain [7].
- Facial Diplegia- It is a rare variant presenting with bilateral facial nerve palsy and paraesthesias in extremities [8].
- Ropper's variant- Bilateral 6^{th} and 7^{th} cranial nerve palsy [8]
- Pure sensory ataxic variant- These patients present with only sensory involvement and ataxia secondary to demyelination of peripheral sensory nerves. They do not have any motor weakness.
- Pan dysautonomic variant- It is a rare variant. It presents predominantly with features of autonomic involvement. Motor involvement is absent or less predominant. Sensory involvement is absent.

Pathogenesis

AIDP is a postinfectious disorder. Two-thirds of patients have symptoms of a respiratory or gastrointestinal tract infection preceding the onset of AIDP. C. jejuni is the most commonly involved organism. Other organisms involved are cytomegalovirus, Epstein–Barr

virus, Mycoplasma pneumonia, Haemophilus influenzae, and influenza A virus [9, 10].

Antibodies that cross react with specific gangliosides, are produced after infection with C. jejuni. Cross reactive antibodies are produced only in susceptible individuals, proving the role of genetics in AIDP. AIDP is because of an immune response against non - self antigens (infectious agents, vaccines) that acts against the host's nerve tissue through molecular mimicry. The cross reactive antibodies are formed against neural gangliosides which are mainly situated at the nodes of Ranvier.

Clinical features

Acute rapidly progressing weakness of limbs, involving lower limbs more than the upper limbs. Proximal muscle weakness is more predominant. Weakness may or may not include tingling and paresthesias of the limbs. Other sensory deficits may be present. Large fiber sensations are more frequently lost. AIDP is divided into subtypes based on the presence or absence of sensory or motor deficits. Deep tendon reflexes are usually diminished or absent.

Cranial nerve involvement may be present. Facial nerve is more frequently involved. 9^{th} and 10^{th} cranial nerves may be involved causing bulbar weakness. Autonomic involvement may be present causing bladder dysfunction, postural hypotension and cardiac dysrhythmias causing cardiac arrest.

Investigations

CSF examination: CSF analysis reveals an elevated CSF protein concentration with normal cell count (albumin - cytological dissociation). This may not be seen during the first week of the disease. Elevated CSF protein concentration occurs in more than 90% of patients at the peak of the disease. When the cell count is elevated (>50 cells/ micro liter), alternative diagnosis should be considered such as HIV, CMV, Lyme's disease, sarcoidosis, carcinomatous, or lymphomatous polyradiculoneuropathy [12].

Nerve conduction studies (NCS): NCS are essential to differentiate between demyelinating and axonal subtypes in AIDP. The distal motor latency is prolonged >150% of the normal upper limit. There is slowing of conduction velocity <70% of the normal lower limit and prolongation of F wave latency >150% of the normal upper limit. There is low CMAP amplitude or proximal CMAP drop suggestive of conduction block, or abnormal temporal dispersion in 2 or more nerves [13].

Antibody testing: Role of antibody testing in diagnosis has not been established. The frequency at which each specific antibody occurs is low, therefore the negative predictive value of detection tests is low. There is limited positive predictive value for these antibody tests, as antiganglioside antibodies (especially those of the IgM class) also occur in other diseases [11].

CT/MRI brain: CT/MRI Brain do not have any role in the diagnosis of AIDP. AIDP is mainly diagnosed through clinical features and CSF findings and electrophysiological studies [14].

Diagnostic criteria

Asbury Criteria and Brighton Criteria are used in the diagnosis of AIDP. Brighton Criteria is used more commonly. [15].

Treatment

AIDP patients who are symptomatic, but are able to walk without support for more than 5 m and who are stable, can be managed conservatively. However, they should be monitored for progression of the disease, especially if they are in the first week of disease and haven't reached the nadir of illness. Signs of respiratory failure, blood pressure, heart rate and vital capacity should be monitored at regular intervals. IVIg, plasma exchange and corticosteroids remain the mainstay of treatment in AIDP.

Intravenous immunoglobulin (IVIg)

IVIg is administered at a dose of 2g/kg body weight, divided over 5 days. In general in

patients with renal dysfunction the rate of infusion should be decreased to half of the normal infusion rate.

Plasma exchange

Plasma exchange is done at a rate of 40-50 ml/kg, 4-5 times over 7-10 days. IVIg and plasmapheresis are equally effective [16]. IVIg and PE have no significant difference with respect to the improvement in disability grade, the duration of mechanical ventilation, mortality, or residual disability [17]. The combination of PE and IVIg was not superior to PE or IVIg alone. The combination of IVIg and intravenous methyl prednisolone was not better than IVIg alone [18, 19].

Role of corticosteroids

Corticosteroids are effective in the treatment of AIDP [20, 21]. Theoretically, corticosteroids would be expected to reduce inflammation and so lessen nerve damage in inflammatory neuropathy. Several retrospective cohort studies have compared patients treated with and without corticosteroids. The baseline characteristics in both groups appeared similar [22]. However, one comparison series of corticosteroid-treated participants showed a beneficial effect from corticosteroids when given in combination with IVIg [23].

Supportive care: Supportive care includes mechanical ventilation, DVT prophylaxis and management of dysautonomia. Care should be taken to prevent infections and to treat them if needed. Hyponatremia is a frequent complication of AIDP and should be managed. Presence of hyponatremia is an indicator of poor prognosis [24].

Prognosis

AIDP patients have a favorable prognosis and recover well within 1 year [25, 26]. Some patients may have residual weakness [27]. Mortality rates in AIDP range from 1-18%. The causes of death include pneumonia, sepsis, respiratory failure, acute respiratory distress syndrome (ARDS) and less frequently autonomic dysfunction or pulmonary embolism [28].

Predictors of poor prognosis in GBS [29, 30]

- Older age (>50–60)
- Rapid onset before presentation (<7 days)
- Ventilator dependency
- Severely reduced distal CMAP amplitudes (<20% of lower normal limits)
- Bedbound or chairbound
- Preceding diarrheal infection
- Higher disability score at presentation
- Axonal type of nerve injury

Differential diagnosis of acute flaccid paralysis

1. Brainstem stroke
2. Brainstem encephalitis
3. Acute anterior poliomyelitis
4. Acute myelopathy (Space-occupying lesions , Acute transverse myelitis)
5. Peripheral neuropathy
    - AIDP
    - Post-rabies vaccine neuropathy
    - Diphtheritic neuropathy
    - Heavy metals, biological toxins or drug intoxication
    - Acute intermittent porphyria
    - Vasculitic neuropathy
    - Critical illness neuropathy
    - Lymphomatous neuropathy
6. Disorders of neuromuscular transmission
    - Myasthenia gravis
    - Biological or industrial toxins
7. Disorders of muscle
    - Hypokalaemia
    - Hypophosphataemia
    - Inflammatory myopathy
    - Acute rhabdomyolysis
    - Trichinosis
    - Periodic paralysis

Case report

A 47 year old male patient presented with severe pain in epigastric region of 4 hours duration. He also had difficulty in swallowing since 4 hours,

both for liquids and solids. There was no history of vomiting, loose stools and fever.

1. At the time of presentation: Day I – 0 hrs

General examination:

Patient was conscious, coherent, answering questions well, moderately built and nourished, no pallor, no icterus, no cyanosis, no clubbing, no generalized lymphadenopathy and no pedal edema, no thyroid swelling, no neurocutaneous markers, no trophic ulcers, head and spine was normal.

Vital data: Temp: 37^0 C, Pulse- 84/min, regular, normal volume, BP- 140/90 mm Hg, RR- 18/ min, Spo2- 98% at room air

Systemic examination: Cardiovascular and respiratory systems were normal. Patient had tenderness at epigastrium. There was no rigidity of abdomen and there was no organomegaly. CNS examination was normal at the time of presentation.

2. After 7 hours of hospital admission:

Patient was conscious, obeying commands and had complaint of severe pain in epigastrium. Patient also had frothing from mouth.

Vital data: Temp- 36.7^0 C, Pulse- 72/min, regular, normal volume, BP- 110/70 mm Hg, RR- 20/min, Spo2- 97% at room air

Systemic examination: Examination of cardiovascular and respiratory systems was normal. Patient had tenderness in epigastrium. There was no rigidity of abdomen and there was no organomegaly. CNS examination was normal.

3. 7 hrs 30 minutes later:

Patient was found drowsy, responding to painful stimuli, Patient had involuntary micturition.

Temp- 36.4^0 C, Pulse- 40/min, feeble, BP- Not recordable, CVS- S1, S2 +, RS- NAD, CNS- Pupils- bilateral 6mm, sluggishly reacting to light, Motor System: Bulk- normal, Tone- normal, Power- not elicitable, Deep Tendon Reflexes- absent, Plantars- bilateral mute.

Patient went into Cardiac Arrest. Cardiac monitor showed Ventricular Tachycardia. CPR was done and DC Cardioversion was given and reverted to normal sinus rhythm. The patient was intubated and put on mechanical ventilation. Patient was started on Noradrenaline and Dobutamine.

4. Day 2 of hospitalization:

Patient drowsy, responding to verbal stimuli.

Pupils- bilateral 6mm, sluggishly reacting to light, Extraoccular movements absent in both eyes, Bilateral eye ptosis+. Motor System: Bulk- normal, Tone- decresed in all the 4 limbs, Power- 1/5 in all the 4 limbs, Deep tendon reflexes were absent in all 4 limbs, Plantars were not elicitable. Sensory system examination- normal, there were no signs of meningeal irritation, skull and spine were normal.

A diagnosis of AIDP was made, Nerve Conduction Studies revealed severe sensorimotor mixed axonal and demyelinating neuropathy affecting both upper and lower limbs and patient was started on Inj. METHYL PREDNISOLONE 1gm/IV/OD x 5 days. Then, Tab. WYSOLONE 1mg/kg body weight/day was given for 1 month and then tapered gradually.

Patient improved and ionotrope support was gradually tapered over 2 days and was extubated 3 days later. The motor weakness and ptosis also improved gradually and patient was discharged after complete recovery.

Investigations:

CBP- Hb- 14.4 g/dl; WBC- 10,100 cells/cumm; DC: N- 85%, L-13%, E-2%, M- 0%, B- 0%; Platelets- 2.0 lakhs/ Cumm; ESR- 14mm/hr; CUE- normal; RBS- 146 mg/dl; B. Urea-11 mg/dl; S.Creatinine: 1.0 mg/dl; Na- 141 mmol/l; K- 3.6 mmol/l; Cl- 103 mmol/l; HIV 1&2- Negative; HbsAg- Negative; HCV- Negative; S Amylase- 183 U/L; S Lipase- 11 U/L; CPK- 1599 U/L; CK- MB- 3.96 ng/ml; ECG- a) initial ECG was normal, b) Scope during cardiac arrest showed Ventricular Tachycardia, c) subsequent ECGs post CPR and DC Cardioversion were normal; Chest X ray- Normal;USG ABDOMEN: Normal; CT BRAIN- Normal; Nerve Conduction

Studies- revealed severe sensorimotor mixed axonal and demyelinating neuropathy affecting both upper and lower limbs.

Diagnosis

- Quadriparesis- LMN type- Peripheral mixed nerve
- 3,4 and 6 cranial nerve palsy
- AIDP – AMSAN+ Ophthalmoparesis Overlap Variety
- Dysautonomia
- Recovered from Cardiac arrest

Discussion

AIDP patients usually present with symmetric ascending type of paralysis. But pain abdomen which is a peculiar symptom [31] was the presenting symptom in our case. Pain precedes weakness in about one third of AIDP patients [32, 33]. The pain may be muscular, radicular, arthralgic or meningitic pain. In the acute phase and during the follow-up period, pain is usually present in the extremities. Low-back or back pain is notably present in the acute phase. Small nerve fibers can also be affected in AIDP. Affected small nerve fibers in AIDP may play a role in pain and autonomic dysfunction [33]. Pain in the acute phase of AIDP might be of nociceptive origin due to inflammation of nerve fibers, whereas later in the course of the disease, non-nociceptive neuropathic pain could result from degeneration and maybe regeneration of sensory nerve fibers [34]. Pain was reported in 38% of AIDP patients at the end of 1 year, and the intensity of pain was highest in patients with sensory disturbances and in those with higher level of weakness and disability [33]. However, pain abdomen is a very rare symptom of AIDP. Pain abdomen in AIDP can be due to dysautonomia causing ileus or urinary retention. However, our patient did not have any gastric or urinary symptoms. Inflammation of nerve roots can also cause pain and tingling in the affected areas.

The other diseases presenting with pain abdomen and neurological deficits include hereditary diseases such as acute intermittent porphyria, Fabry disease, poisoning with heavy metals such as arsenic and lead, toxins like ethylene glycol, drugs like thallium and fluoroquinolones and infections such as Lyme disease (neuroborreliosis), poliovirus, and West Nile virus. Krait bite can also cause pain abdomen and neurological deficits such as quadriparesis and ptosis.

The most critical features of AIDP are involvement of the respiratory and oropharyngeal musculature, and autonomic nervous system. AIDP is associated with autonomic dysfunction in up to two-thirds of patients [35]. Autonomic nervous system complications tend to occur more frequently in those with severe paralysis and ventilatory difficulties. This includes blood pressure fluctuations, arrhythmias, vasomotor dysfunction, gastrointestinal motility dysregulation and urinary retention. The most common cardiac manifestations include sinus tachycardia, sinus bradycardia, sinus arrest and other supraventricular arrhythmias, paroxysmal hypertension, hypotension (especially postural hypotension). Dysautonomia also includes vagal spells that consist of bronchorrhea, bradycardia, and hypotension. This patient had profuse sweating at the time of presentation, which can be attributed to autonomic dysfunction. Also, this patient had involuntary micturition, frothing from mouth (bronchorrhea), bradycardia and hypotension prior to cardiac arrest which is due to the vagal spells seen in AIDP patients. This patient also had ventricular tachycardia. Dysautonomia preceded motor weakness in this patient. Hence, it is very important to screen all AIDP patients for dyautonomia and treat them early in the course of the disease. Weakness of the diaphragm that leads to respiratory failure and a requirement for ventilator support occurs in approximately 20–30 % of patients with AIDP [36, 37]. Both dysautonomia and respiratory failure were causes of cardiac arrest in our patient.

Cranial nerve (CN) palsies are common symptoms of AIDP, but multiple cranial

neuropathies are a rare variant of AIDP and account for only 5% of patients [38]. Approximately one-half of AIDP patients will have cranial nerve involvement at some time during the disease course. Facial nerve is the most commonly involved cranial nerve. But, this patient did not have facial palsy. Weakness of the ocular muscles arises in 10–20 % of patients, the abducens nerve being most commonly affected. The cranial nerve involvement is usually bilateral and symmetrical [39]. Polyneuritis cranialis is a rare variant of AIDP involving multiple cranial nerve palsies without any ataxia or limb weakness. Because of its rarity, multiple cranial neuropathies as variant of AIDP exhibiting normal motor and sensory limb functions have seldom been described in the literature. The GQ1b ganglioside is enriched in the paranodal regions of the extra-medullary portion of the human oculomotor, trochlear, and abducens nerves [40].

GQ1b antibodies are also frequently positive in patients with autonomic involvement. This explains the co existence of dysautonomia and 3,4 and 6 cranial nerve palsies in our patient. There are several variants of Anti GQ1b antibody syndrome including Miller fisher syndrome, Bickerstaff brainstem encephalitis and acute ophthalmoplegia. Our patient had quadriparesis with areflexia and total ophthalmoplegia. This could represent a rare form of overlap between acute ophthalmoplegia and AMSAN variants of AIDP. Nerve conduction studies and Cerebrospinal fluid analysis confirmed the diagnosis of AIDP.

Conclusion

The diagnosis was difficult initially as the patient did not have any neurological deficits at the time of presentation and he was evaluated in terms of causes of pain abdomen. We could make the diagnosis only on the 2nd day after reassessing the patient and could correlate the cause of the various findings noticed on the first day. Pain abdomen is an extremely rare presentation of AIDP. To the best of our knowledge, this is only the 3rd reported case of AIDP as a cause of pain abdomen. Henceforth, AIDP has to be considered as a cause of pain abdomen after ruling out other medical and surgical causes of abdominal pain. The patient also had features of dysautonomia such as excessive sweating and vagal spells, including bronchorrhea, hypotension and bradycardia, leading to cardiac arrest. On the second day, patient developed quadriparesis with areflexia, bilateral ptosis and bilateral total ophthalmoplegia. The second interesting feature regarding this case, where the pattern of neurological deficits is not confined to one particular variant of AIDP but represents an overlap between AMSAN and Acute ophthalmoplegia syndrome. The patient was given supportive care and showed significant improvement and was discharged in a stable condition.

References

1. Wong PS, Fothergill NJ, Touquet R. Abdominal pain as a presenting symptom of the Guillain-Barré syndrome. Arch Emerg Med., 1988; 5: 242–245.
2. Kuwabara S. Guillain-Barré Syndrome. CNS Drugs, 2004; 64: 597–610.
3. Hughes RA, Rees JH. Clinical and epidemiologic features of Guillain-Barre syndrome. J Infect Dis., 1997; 176 Suppl. 2: S92-8.
4. McKhann GM, Cornblath DR, Griffin JW, et al. Acute motor axonal neuropathy: a frequent cause of acute flaccid paralysis in China. Ann Neurol., 1993; 33(4): 333–42.
5. Feasby TE, Gilbert JJ, Brown WF, et al. An acute axonal form of Guillain-Barre´ polyneuropathy. Brain, 1986; 109(Pt 6): 1115–26.
6. Bickerstaff E, Cloake P. Mesencephalitis and rhombencephalitis. Br Med J., 1951; 2: 77–81.

7. Ropper AH. Unusual clinical variants and signs in Guillain-Barre´ syndrome. Arch Neurol., 1986; 43(11): 1150–2.
8. Ropper AH. Further regional variants of acute immune polyneuropathy. Bifacial weakness or sixth nerve paresis with paresthesias lumbar polyradiculopathy and ataxia with pharyngeal-cervical-brachial weakness. Arch Neurol., 1994; 51(7): 671–5.
9. Jacobs B. C., et al. The spectrum of antecedent infections in Guillain–Barré syndrome: a case–control study. Neurology, 1998; 51: 1110–1115.
10. Hadden R. D., et al. Preceding infections, immune factors, and outcome in Guillain–Barré syndrome. Neurology, 2001; 56: 758–765.
11. Van den Berg B., Walgaard C., Drenthen J., et al. Guillain–Barré syndrome: pathogenesis, diagnosis, treatment and prognosis. Nat Rev Neurol., 2014; 10: 469–482.
12. Meena AK, Khadilkar SV, Murthy JM. Treatment guidelines for Guillain-Barré Syndrome. Ann Indian Acad Neurol., 2011; 14(Suppl 1): S73-81.
13. Gordon PH, Wilbourn AJ. Early Electrodiagnostic Findings in Guillain-Barré Syndrome. Arch Neurol., 2001; 58(6): 913–917.
14. O. Alkan, T. Yildirim, N. Tokmak, M. Tan. Spinal MRI Findings of Guillain - Barré Syndrome. Journal of Radiology Case Reports, 2009; 3(3): 25-28.
15. Sejvar JJ, Kohl KS, et al. Brighton Collaboration GBS Working Group. Guillain-Barré syndrome and Fisher syndrome: case definitions and guidelines for collection, analysis, and presentation of immunization safety data. Vaccine, 2011; 29(3): 599-612.
16. Van der Meché FG, Schmitz PI. A randomized trial comparing intravenous immune globulin and plasma exchange in Guillain-Barré syndrome.Dutch Guillain-Barré Study Group. N Engl J Med., 1992; 326: 1123–9.
17. Hughes RA, Raphael JC, Swan AV, van Doorn PA. Intravenous immunoglobulin for Guillain-Barré syndrome. Cochrane Database Syst Rev., 2006; 1: CD002063.
18. Susuki K, Yuki N. Effect of methylprednisolone in patients with Guillain-Barré syndrome. Lancet, 2004; 363: 1236–7.
19. Van Koningsveld R, Schmitz PI, van der Meche FG, Visser LH, Meulstee J, van Doorn PA. Effect of methylprednisolone when added to standard treatment with intravenous immunoglobulin for Guillain-Barré syndrome: Randomised trial. Lancet, 2004; 363: 192–6.
20. V. Shanthan, E.A. Ashok Kumar. A rare combination of acute inflammatory demyelinating polyradiculoneuropathy (AIDP) AMSAN variety in hypothyroidism – A case report. IAIM, 2020; 7(3): 104-110.
21. Darryl C, De Vivo, W. King Engle. Remarkable recovery of a steroid – responsive recurrent polyneuropathy. J. Neurol. Neurossurg. Psychiat., 1970; 33: 62-69.
22. Peter JV, Gnanamuthu C, Cherian AM, Prabhakar S. Outcomes in the Guillain-Barré syndrome: the role of steroids. Journal of the Association of Physicians of India, 1996; 44(3): 172-4.
23. The Dutch Guillain-Barré Study Group. Treatment of Guillain-Barré syndrome with high dose immune globulins combined with methyl prednisolone: a pilot study. Annals of Neurology, 1994; 35(6): 749-52.
24. Saifudheen K, Jose J, Gafoor VA, Musthafa M. Guillain-Barré syndrome and SIADH. Neurology, 2011; 76: 701–4.
25. Van den Berg B, Bunschoten C, van Doorn PA, Jacobs BC. Mortality in Guillain-Barre syndrome. Neurology, 2013; 80: 1650–1654.
26. Soysal A, Aysal F, Caliskan B, Dogan Ak P, Mutluay B, Sakalli N, Baybas S,

Arpaci B. Clinico-electrophysiological findings and prognosis of Guillain-Barré syndrome—10 years' experience. Acta Neurol Scand., 2011; 123: 181–186.

27. Forsberg A, Press R, Holmqvist LW. Residual disability 10 years after falling ill in Guillain-Barré syndrome: a prospective follow-up study. J Neurol Sci., 2012; 317: 74–79.
28. Ng KK, Howard RS, Fish DR, Hirsch NP, Wiles CM, Murray NM, et al. Management and outcome of severe Guillain Barré syndrome. Q J Med., 1995; 88: 243–50.
29. Walgaard C, Lingsma HF, Ruts L, van Doorn PA, Steyerberg EW, Jacobs BC. Early recognition of poor prognosis in Guillain-Barre syndrome. Neurology, 2011 Mar 15; 76(11): 968-75.
30. Shangab M, Al Kaylani M. Clinical Course and Predictors of Poor Functional Outcome in Guillain-Barré Syndrome. A Retrospective Study. Dubai Med J., 2020; 3: 93-98.
31. Michas G, Nikolopoulou A, Varytimiadi E, Xydia N. A rare case of Guillain-Barré syndrome presenting with abdominal pain. Hippokratia, 2015; 19(4): 374-375.
32. Yuki N, Hartung HP. Guillain-Barré Syndrome. N Engl J Med., 2012; 366: 2294–2304.
33. Ruts L, Drenthen J, Jongen JL, Hop WC, Visser GH, Jacobs BC, et al. Dutch GBS Study Group. Pain in Guillain-Barre syndrome: a long-term follow-up study. Neurology, 2010; 75: 1439–1447.
34. Van Doorn PA, Ruts L, Jacobs BC. Clinical features, pathogenesis, and treatment of Guillain Barré syndrome. Lancet Neurol., 2008; 7: 939–950.
35. Ropper AH, Wijdicks EFM, Truax BT. Guillain–Barré syndrome. Contemporary neurology series. Muscle and Nerve. FA Davis, Philadelphia, 1992; 15(2): 259-259.
36. Walgaard C, Lingsma HF, Ruts L, Drenthen J, van Koningsveld R, Garssen MJ, et al. Prediction of respiratory insufficiency in Guillain-Barré syndrome. Ann Neurol., 2010; 67(6): 781–7.
37. McKhann GM, Griffin JW, Cornblath DR, Mellits ED, Fisher RS, Quaskey SA. Plasmapheresis and Guillain-Barré syndrome: analysis of prognostic factors and the effect of plasmapheresis. Ann Neurol., 1988; 23(4): 347–53.
38. Wang Q, Xiang Y, Yu K, Li C, Wang J, Xiao L. Multiple cranial neuropathy variant of Guillaine-Barre syndrome: a case series. Muscle Nerve, 2011; 44: 252–257.
39. Bakshi N, Maselli RA, Gospe Jr SM, Ellis WG, McDonald C, Mandler RN. Fulminant demyelinating neuropathy mimicking cerebral death. Muscle Nerve, 1997; 20(12): 1595–7.
40. Edvardsson B, Persson S. Polyneuritis cranialis presenting with anti-GQ1b IgG antibody. J Neurol Sci., 2009; 281: 125–126.