

NEUROLOGY

ACUTE FLACCID PARALYSIS BEYOND POLIO- A CASE BASED APPROACH

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Abstract: *Acute flaccid paralysis is a complex clinical syndrome that requires immediate and careful evaluation for making a diagnosis. Each case of acute flaccid paralysis is an emergency from both clinical as well as public health perspective. The precise knowledge of the etiology, underlying pathophysiology and concurrent changes have profound implications in the treatment and prognosis. With the eradication of polio, Guillain Barre Syndrome has become the major acute flaccid paralysis. Seasonal occurrence of Guillain Barre Syndrome with spurt of viral fever is also seen. However, the clinical features of polio must be taught to the younger residents since imported or vaccine associated polio can still occur. Better usage of Magnetic resonance imaging scanning will help in establishing the diagnosis. Acute management of such patient with acute flaccid paralysis due to different causes in intensive care unit has become a necessity. Based on severity IVIg for Guillain Barre Syndrome and Methyl prednisolone for transverse myelitis are now accepted protocols. We are still in the process of consolidating the eradication of polio by the endgame strategy from 2019-2023.*

Keywords: *Acute flaccid paralysis, Guillain-Barre Syndrome, Lower motor neuron localization, Transverse myelitis*

Acute flaccid paralysis (AFP) is an acute onset flaccid weakness, less than 4 weeks, reaching its maximum severity in days to weeks. Some of the causes of AFP are Guillain-Barre syndrome (GBS), poliomyelitis, transverse myelitis (TM), traumatic neuritis and post diphtheritic neuropathy. Continued surveillance of AFP is required to completely eradicate poliomyelitis. A case for surveillance of AFP is defined as any case of AFP in children <15 year old, or any paralytic illness at any age when polio is suspected. Many reviews are available on AFP and this article outlines a case based approach, to highlight the varied presentation.

Etiologies of AFP¹

It is easier to analyse the cause based on anatomic location

- Brain stem: GBS with cranial nerve involvement, brain stem encephalitis and stroke
- Spinal cord: Acute transverse myelitis, acute myelopathy due to spinal cord compression (abscess, space occupying lesion), anterior spinal artery syndrome.
- Anterior horn cells: Poliomyelitis, non-polio enteroviruses.
- Nerve root (radiculopathy): Guillain Barre syndrome.
- Peripheral nerve: Guillain Barre syndrome, toxic neuropathies (diphtheria, tick bite paralysis, lead, arsenic poisoning) traumatic neuritis, acute intermittent porphyria, critical illness neuropathy.
- Neuromuscular junction: Myasthenia gravis, botulism, snake bite, organophosphorus poisoning.
- Muscle: Polymyositis, trichinosis, hypokalemia, hypophosphatemia.

Clinical approach

Usually sudden occurrence of flaccid weakness denotes lower motor neuron (LMN) weakness. However, upper motor neuron (UMN) weakness of sudden onset also can have flaccid weakness in the initial stages due to the neuronal shock state. Following a systematic clinical approach based on the distribution and progression of weakness, associated sensory involvement, fever etc. will

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help in differentiating various conditions. Gradual onset of weakness and chronic conditions like spinal muscular atrophy are not included here.

Localization based on clinical signs (Table I)

First step in the diagnosis is localizing the lesion.

LMN: Flaccid weakness with absent reflex is in favor of LMN. After ascertaining that the motor weakness is due to LMN, exact site of lesion like anterior horn cell, root, nerve, myoneural junction or muscle is determined by verifying the presence or absence of cranial nerve lesions, bladder involvement, tendon reflexes and sensory involvement. Associated clinical features may give a clue to the etiological diagnosis (Table II).²

Anterior horn cell disorders

Case history

Seven year old child was seen with history of fever, severe myalgia, back pain and weakness of abduction of right shoulder. Examination revealed meningeal signs, mild bulbar weakness, normal sensory and sparing of bladder and bowel. CSF showed lymphocytic pleocytosis. NCV/EMG suggested anterior horn cell disease. Serology revealed enterovirus (type was not determined) on follow up flail shoulder with severe wasting of deltoid was persisting.

Comment: Child had a poliomyelitis like presentation, without isolation of any type of polio virus, but due to another enterovirus.

Febrile illness, rapidly progressive asymmetric weakness, preserved sensory function, severe myalgia and residual paralysis after 60 days are features of polio like illness.^{3,4} Enterovirus 71, Coxsackie, Echo and West Nile viruses are the non polio viruses reported.⁵ After the

eradication of polio, new viral infections can present as polio like illnesses. As oral polio vaccine is still used for vaccination, there is a very remote risk of vaccine derived poliomyelitis.

Radicles and peripheral nerve

Case history

Ten year old boy was admitted with weakness of lower limb of five days and upper limb of two days duration. It started as knee buckling, difficulty climbing stairs and getting up from sitting position. He had difficulty sipping water from a glass but was able to wear slippers and hold the objects in hand. On 3rd day as he developed difficulty raising arm above shoulder and respiratory muscle weakness. He had pain in the legs but no sensory loss. There was no difficulty in urination. He had an upper respiratory tract infection 2 weeks ago.

Comment: Pure proximal motor limb weakness with bifacial palsy and presence of respiratory muscle weakness is diagnostic of GBS. GBS presenting as painful limping may be confused as synovitis. Absent reflexes points towards GBS. Bifacial palsy can be missed as there is no facial deviation due to the symmetric weakness. Difficulty puckering the lips or sipping, incomplete burying of eye lashes on tight eye closure are the clues.

Guillain Barre syndrome (GBS)

It is a progressive, near symmetrical weakness occurring in more than one limb with areflexia (Modified Asbury's criteria) (Box 1). The common subtypes are acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor sensory axonal neuropathy (AMSAN), Miller Fisher syndrome (MFS) and polyneuritis cranialis. The rare variants of GBS are acute pan-dysautonomia, acute sensory

Table I. Localization based on clinical signs

Anatomical level	Clinical signs
Brain stem	Altered consciousness, cranial nerve paralysis, Upper motor neuron (UMN) lesion signs
Spinal cord	Bladder involvement, UMN lesion signs (below the level of lesion), plantar extensor
Anterior horn cells	Lower motor neuron (LMN) type weakness, areflexia
Nerve root	Lower motor neuron weakness, areflexia. No sensory loss
Peripheral nerve	Sensory loss, glove and stocking sensory involvement, loss of ankle reflex
Neuromuscular junction	Diurnal variation, fluctuating weakness
Muscle	LMN weakness but reflexes preserved

Table II. Clues to diagnosis

Features	Diagnosis
Fever	Polio, Lyme disease
Myalgia or sensory symptoms	GBS
Recent exanthem	Lyme disease
Patch over tonsillar area, bull neck	Diphtheria
Trauma/IM injection	Traumatic neuropathy
Abdominal pain	Porphyria, lead poisoning
Exposure to chemicals	Arsenic, lead poisoning
Tick bite	Tick paralysis
Ptosis, respiratory paralysis	Snake bite, botulism
Dermatological manifestations - Edema, Gottron's papules	Dermatomyositis
Recurrent flaccid paralysis	Periodic paralysis - channelopathies
Acute paralysis occurring in an ICU patient	Critical illness neuropathy

ataxia with or without ophthalmoplegia, pharyngeal-cervical-brachial weakness and facial diplegia with paresthesias. Preceding infections associated with GBS include *Campylobacter jejuni*, cytomegalovirus, Epstein-Barr virus, mycoplasma pneumonia and HIV.⁶

MFS is characterized by ataxia, ophthalmoplegia and areflexia without weakness. Anti-ganglioside GQ1B antibodies are commonly detected in MFS. Brain stem encephalitis also can have a similar presentation with ataxia and ophthalmoplegia. However somnolence will be a distinguishing feature.

Electrophysiological abnormalities of GBS: Nerve conduction studies (NCV) are helpful in confirming the

Box 1. Guillain Barre syndrome - Diagnostic criteria

- Presence of progressive weakness and areflexia
- Symmetrical involvement
- Mild sensory involvement, cranial nerve involvement, at least partial recovery
- Autonomic dysfunction
- Absence of fever
- Cerebrospinal fluid features supporting the diagnosis are an increase in protein beyond the first week, cell count < 10 (albumin-cytological dissociation)

diagnosis. The first changes in AIDP are delayed or absent F and H responses, reflecting proximal demyelination. Prolonged distal latencies, decreased conduction velocities along with evidence of segmental demyelination, (conduction block and temporal dispersion) are other changes present in 50% of patients by 2 weeks and in 85% by 3 weeks.

The features raising doubt on the diagnosis of GBS are persistent asymmetry of weakness, presence of a sensory level, bowel/bladder involvement at onset and a prominent CSF pleocytosis.

Conditions that may have a presentation like GBS include neuropathy associated with HIV, Diphtheria, Lyme disease, porphyria and critical illness; polyneuropathy/myopathy and vasculitis syndromes.

Acute transverse myelitis (TM) (Box 2)

It presents as sudden limb weakness, bowel-bladder and sensory disturbances and commonly occurs in toddlers and adolescents.

Postinfectious TM: Preceded by viral or bacterial infection. Viruses implicated are enteroviruses, coronavirus, coxsackie, cytomegalovirus, Epstein-Barr, herpes-simplex, hepatitis A, HIV, influenza, measles, rubella, varicella, and West Nile virus. Bacteriae include mycoplasma pneumoniae, rickettsia, beta hemolytic *Streptococcus*, borrelia, chlamydia and leptospira.

Box 2. TM - Criteria for diagnosis

- Sensory, motor, or autonomic dysfunction attributable to the spinal cord
- Absence of compressive cord lesion
- Bilateral symptoms and signs
- Definite sensory level
- Spinal T2 MRI- hyperintense signals
- Evidence of inflammation
- CSF pleocytosis, elevated IgG index, or gadolinium enhancement on MRI
- Progression to nadir between 4 hours and 21 days^{6,7}

Post-vaccinal TM: May follow immunization for rabies, hepatitis B, influenza, Japanese encephalitis, diphtheria/pertussis/tetanus, measles, mumps, rubella, pneumococcus, polio, smallpox and varicella.

Acute flaccid paralysis with sensory level is a feature of TM. In children, mild sensory symptom like hyperaesthetic sites may be present which helps for localization. If legs are affected the sensory level is commonly at umbilicus or at the level of nipple. If the arms are weak, look for sensory level at cervical region. Bowel and bladder involvement cause constipation and urinary retention. Respiratory failure (intercostals or diaphragmatic weakness) may occur with higher level lesions.

Variants of TM: Identifying TM variants will help in etiological diagnoses and thus treatment and prognoses

1. Longitudinally extensive TM (LETM) - Lesion in >3 spinal segments associated with bilateral optic neuritis = neuromyelitis optica spectrum disorder.
2. Acute flaccid myelitis (AFM) - Like poliomyelitis they have segmental LMN (AHC) and additional UMN lesion in the setting of other viral infections - e.g. enterovirus D68. These patients may recover with residual wasting and weakness
3. Acute partial TM - Asymmetric extending one to two spinal segments.
4. Acute complete TM - Symmetric, complete or near complete manifestations

An important differential of TM is acute spinal cord infarct due to anterior spinal artery occlusion.⁶ Compressive myelopathy can present as acute syndrome. Both intramedullary and extramedullary compression like neuroblastoma can present acutely.

Case report

13 year old boy presented with episodes of vomiting, retching, hiccup and rapidly progressive weakness of both lower limbs and left upper limb. He had urinary retention and was frequently slipping to sleep. Sensory level was localized to T2 and tendon reflexes were sluggish with extensor plantar.

His MRI showed T2FLAIR hyperintensities involving both grey and white matter of cerebral hemispheres and a large lesion on the floor of 4th ventricle. He had an extensive hyperintensity of spinal cord extending from C5 -T1. His myelin oligodendrocyte glycoprotein (MOG) antibody titer was positive. This case exemplifies transverse myelitis with area postrema syndrome. An aquaporin 4 negative MOG positive neuromyelitis optica spectrum disorders (NMOSD).

Comment: Though bladder symptoms, sensory level localize the lesion to spinal cord, retching, vomiting and hiccup point to area postrema involvement (The area postrema is a highly vascular paired structure in the medulla oblongata in the brainstem, in the caudal fourth ventricular floor. It is a critical homeostatic integration center for humoral and neural signals by means of its function as a chemoreceptor trigger zone for vomiting in response to emetic drugs).

Traumatic neuropathy (TN)

Traumatic injury to peripheral nerves presents as focal AFP. It includes injury to plexus, roots or peripheral nerves. Nerve injuries can result from penetrating trauma (injections, falling on sharp objects), entrapment or from traction injuries. Focal weakness is attributable to a single or multiple nerve distribution.

It is asymmetric. History of trauma is present near a nerve or site of predilection. The common cause is an injection to gluteal or deltoid region. Commonly affected nerves are common peroneal / sciatic (foot drop) or radial (wrist drop). The knowledge of anatomy of nerve will help in identifying the specific nerve. The diagnosis is confirmed by nerve conduction studies. The prognosis depends on severity of injury. Persistent, severe and refractory neuropathic pain may be present along with weakness.

Muscle**Case report**

Eleven year old girl was admitted with 2 weeks history of painful weakness of upper and lower limbs. No sensory signs. Examination showed grade 3 power in proximal and grade 4 in distal muscles. She had neck flexor weakness. Knee jerk and biceps jerk were very sluggish with easily

Table III. Differential diagnosis of AFP

Features	Poliomyelitis	GBS	Transverse myelitis	Traumatic neuritis
Progression to full paralysis	24-48 hrs	Hours to days	Hours to 4 days	Hours to days
Fever onset	High, always present at onset of paralysis	No	Present before paralysis	No
Distributing of weakness	Asymmetrical, patchy	Symmetrical, distal Ascending	Symmetrical lower limbs	Confined to nerve distribution
Muscle tone	Diminished	Diminished	Diminished in lower limbs	Diminished
Deep Tendon reflexes	Decreased or absent	Absent	Absent early, hyperreflexia late	Decreased or absent
Plantar reflex	Absent/Flexor	Absent/Flexor	Extensor	Absent/Flexor
Sensation	Severe myalgia or backache no sensory changes	Cramps, tingling, hypo anesthesia of palms and soles	Anesthesia of lower limbs with sensory level	Pain in the gluteal region
Cranial nerves	Only in the presence of bulbar or bulbo-spinal involvement	Often present, affecting nerves VII, IX, X, XI, XII	Absent	Absent
Respiratory insufficiency	Only in the presence of bulbar and bulbo-spinal involvement	In severe cases	Sometimes	Absent
CSF examination	Cell count raised, protein normal or slightly increased protein	<10 leukocytes, high protein	Cell count: Normal/moderate lymphocytic pleocytosis Protein:Normal/slightly elevated	Cell count: Normal Protein: Normal
Bladder dysfunction	Absent	Transient	Present	Never
EMG at 3 week	Abnormal	Normal	Normal	May be abnormal
NCV at 3 week	Normal	Abnormal demyelination /axonal	Normal	Abnormal
Sequelae at 3 months	Severe, asymmetrical atrophy	Symmetrical atrophy of distal muscle	Diplegia, atrophy after years,	Moderate atrophy of affected limb

elicitable ankle jerk. Skin examination showed typical features of dermatomyositis. CPK was found be raised.

Comment: Pure motor syndrome with proximal involvement (grade 3 power, sluggish knee and biceps jerk) and high CPK indicated muscle disease. Acute onset and neck weakness suggested polymyositis and skin manifestations clinched the diagnosis of dermatomyositis.

Polymyositis-dermatomyositis

AFP can be due to myositis also. Polymyositis-dermatomyositis can be suspected when there is a symmetric proximal muscle weakness without sensory symptoms or signs and with preserved reflexes, neck muscle weakness, muscle pain and raised CPK. Acute myositis following viral infection also are not

uncommon. Temporal relation with viral illness, high CPK and rapid improvement are the features. Some children may require short course of steroid.

Case report

Four year old boy developed weakness of all four limbs and shallow breathing following a diarrheal illness. He required respiratory support for a brief period. Abdominal distension and poor bowel sounds were present. Serum potassium was very low and ECG showed depression of the ST segment, flat T wave with U wave. Consciousness was preserved but tendon reflexes were absent. Weakness rapidly improved with correction of hypokalemia.

Comment: Syndrome of hypokalaemic paralysis represents a heterogenous group of disorders characterised clinically by hypokalaemia and acute pure motor weakness. Sporadic cases are associated with, renal disorders, endocrinopathies and gastrointestinal potassium losses. In adolescents hypokalemic periodic paralysis also can be a possibility.

Neuromuscular junction disorders

Case report

Girl aged nine years was admitted in ICU with rapidly worsening limb weakness and severe respiratory paralysis who required ventilation for 5 days. This happened after a bout of urinary tract infection, which was treated with ciprofloxacin. History of ptosis for 2 weeks occurring in evening hours gave the diagnosis of myasthenic crisis. Rapid deterioration was due to the usage of ciprofloxacin. She improved with IVIg and neostigmine treatment. Acetylcholine receptor (AChR) antibody was positive.

Comment: Usage of certain drugs can worsen treated as well as naïve myasthenia gravis and can present as AFP. Fluctuating weakness, simultaneous ocular and facial muscle involvement and response to neostigmine are the diagnostic pointers. Bite marks and children playing in dark, may suggest snake envenomation. Anti-snake venom and neostigmine will be life saving.

The important features of common causes of AFP are given in Table III.

Investigations

According to AFP surveillance, two stool samples must be collected 24 to 48 hours apart in the first 14 days following the onset of paralysis and to be sent to the accredited lab after following the surveillance protocol for polio or related viral isolation (Fig.1). For diagnosis of other disorders investigations such as blood counts, ESR, peripheral smear, electrolytes (Ca,Mg,K) and CPK. Electrophysiological studies, NCV and EMG are done to evaluate types of GBS, myasthenia, muscle and nerve disorders. MRI of spinal cord and brain for distinguishing various demyelinative disorders.

CSF analysis

Raised CSF cell count is seen in transverse myelitis and infective myelitis viz. polio or enteroviral myelitis, varicella or herpes myelitis and rabies. Albuminocytological dissociation (increased CSF protein with normal cell count) is seen commonly in GBS but rarely in post-diphtheritic polyneuropathy, transverse myelitis and Froin’s syndrome (coexistence of xanthochromia, high protein level and marked coagulation of cerebrospinal fluid due to obstruction of CSF flow may indicate a spinal tumor).

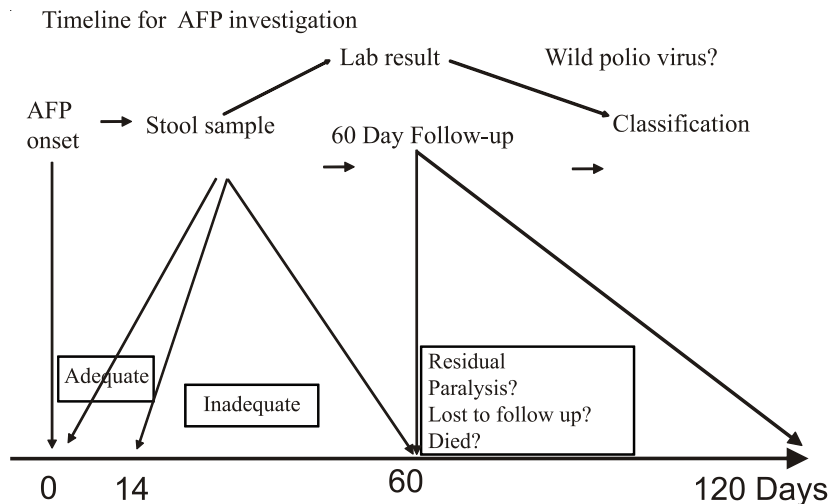


Fig. 1. Time line for AFP evaluation



Fig.2. Longitudinally extensive Transverse Myelitis – T2 hyperintensity extending from C5 - T7. Possibilities are NMO Spectrum disorder or Enterovirus myelitis

MRI^s

TM: Up to 40% of cases have no findings on MRI. The Diagnostic sign is T2 hyperintensity which most commonly extends for 3-4 spinal segments with a variable enhancement pattern.

Spinal cord infarct: It is characterized by an enlarged spinal cord, which is hyperintense on T2 weighted images and DWI. The signal intensity abnormality may be limited to the central gray matter. The signal abnormality typically extends over multiple vertebral body segments (Fig.2). The vertebral body T2 hyperintensity may occasionally be seen due to a concomitant infarction.

GBS: Typical findings in GBS are surface thickening and contrast enhancement on the conus medullaris and the nerve roots (anterior nerve roots) of the cauda equina. Contrast is must as non-contrast sequences are essentially normal (Fig.3a,b,c).

MRI in inflammatory myositis may show increased signal intensity in the quadriceps bilaterally (Fig.4)

Management

General principles

All children with AFP need meticulous supportive care. Anticipation and identification of respiratory and bulbar weakness is important. ICU admission will be required if airway obstruction, respiratory failure or significant autonomic disturbance is observed. Management of shock due to reduced vascular tone, management of autonomic instability and complications

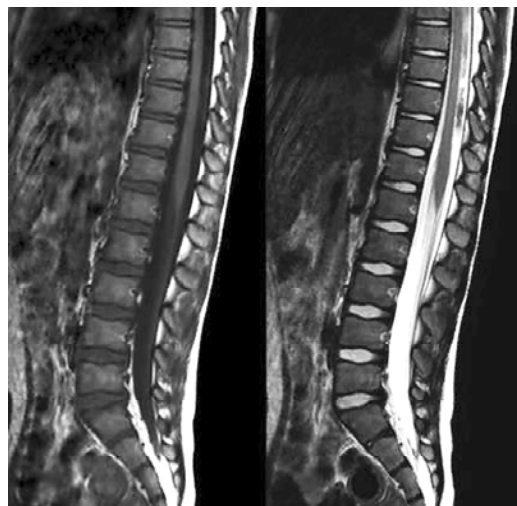


Fig.3a, b. T1 plain and T2 Lumbo-sacral spine MRI in GBS

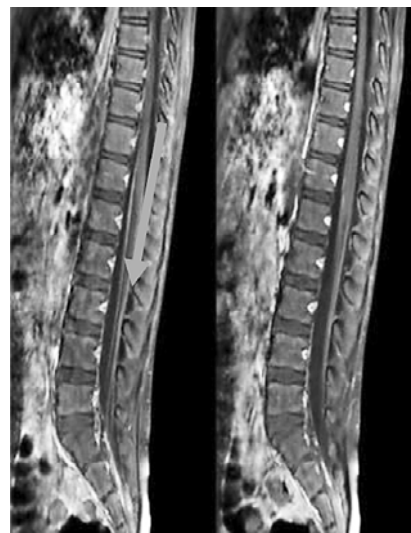


Fig.3c. Radiographic features of GBS contrast T1 MRI lumbo-sacral. Typical findings in GBS - nerve root thickening and enhancement surrounding the conus and the cauda equina

of immobilization and prevention of nosocomial infections are important considerations. The specific therapy depends on the underlying etiology identified.

TM

Intravenous methyl prednisolone (30 mg/kg up to 1000 mg daily) for five days. Plasma exchange or, intravenous cyclophosphamide (800 to 1200 mg/m² administered as a single pulse dose).

GBS

IVIg 400mg/kg/day for 5days in the presence of rapidly progressive weakness (Modified Hughes GBS

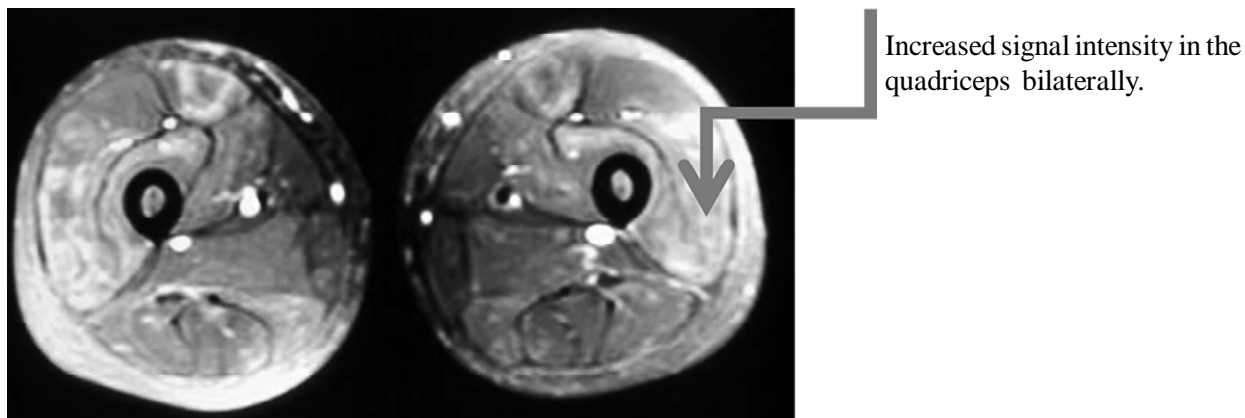


Fig.4. MRI in inflammatory myositis

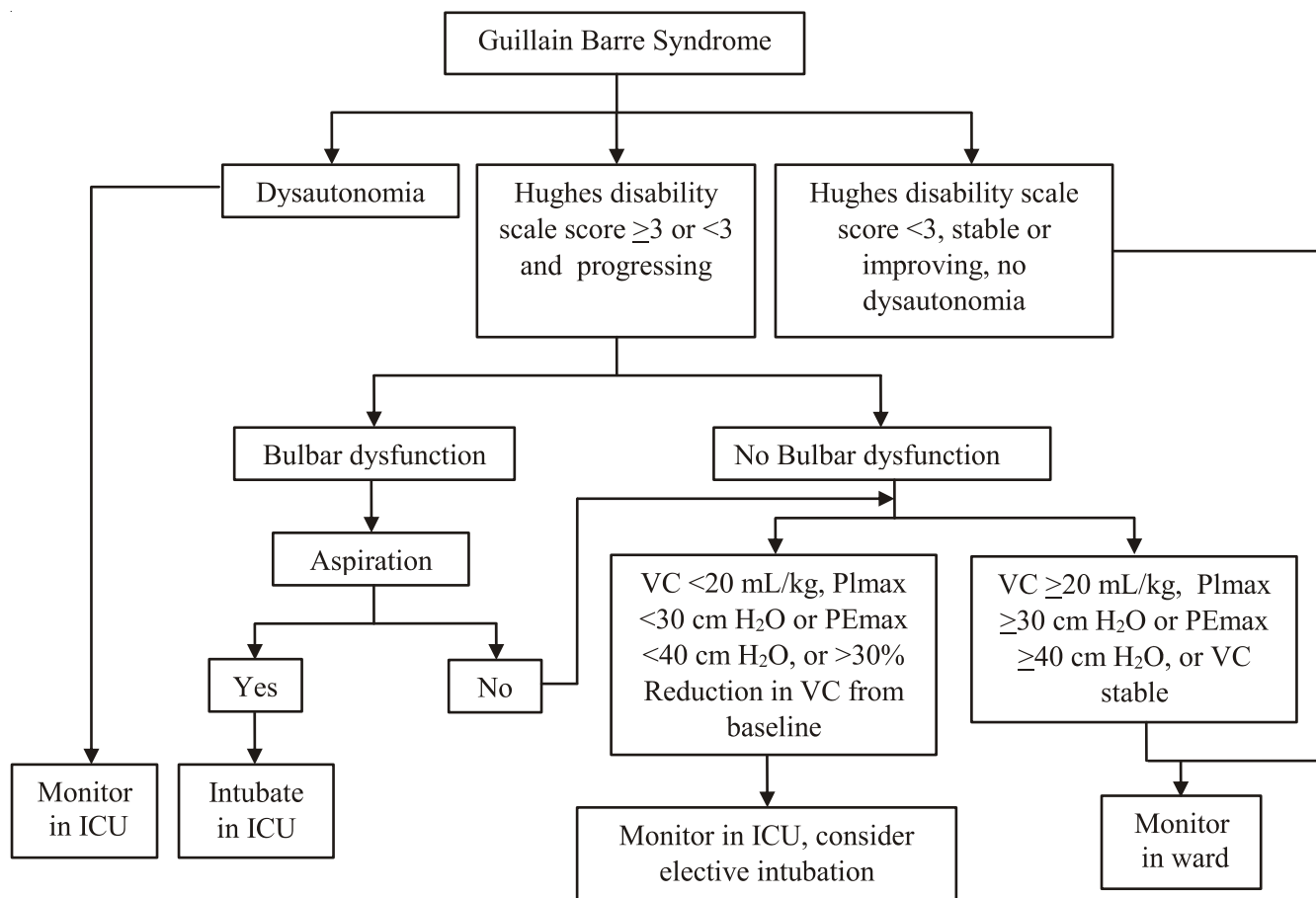


Fig.5. Management of GBS

PE max- Expiratory pressure; PI max- maximum inspiratory pressure VC vital capacity

AFP surveillance - Current status

disability scale), presence of respiratory or bulbar involvement (Fig.5). Ventilatory support may be required when there is respiratory failure. IVIG is not indicated if the degree of weakness is non-progressive and has been present for more than 4weeks.

AFP surveillance - Current status

There has been a sharp fall in the incidence of poliomyelitis across the world from 350,000 cases in 125 countries in 1988 (year of starting Global Polio Eradication Initiative (GPEI)) to 85 cases in 2 countries

(Pakistan-69 and Afghanistan-16) as of 2019. This was achieved by the active surveillance, immunization and improvement in sanitation. India was declared polio-free on 27 March 2014, three polio free years after the last case was reported in January 2011 in West Bengal. As for world, type 2 virus serotype was declared globally eradicated in 2015 and Type 3 on 24th Oct 2019.

Endgame Polio Strategy 2019-23 is the current strategy by the GPEI. The goals have three major components (Table IV).

India has switched over to bivalent OPV in April 2016 excluding the OPV 2 strain which is mainly responsible for vaccine derived polio. Meanwhile, inactivated polio vaccine (IPV) was introduced in 2016 with two fractional intradermal doses at 6 and 14 weeks along with bivalent OPV.

AFP surveillance is the strategy to screen for circulating wild polio virus in the post-polio eradication phase. The patients with AFP within the last 6 months should be reported to the surveillance Medical Officer of WHO. The four steps of AFP surveillance are finding and reporting children with AFP, transport and analysis of stool sample, identify poliovirus in laboratory and determine the virus strain and origin. Within 48 hours of notification, a trained medical officer investigates the case, proceeds with transportation of stool samples, outbreak response immunizations done in the affected community. A 60 day follow up examination of the case is also done.

The non-polio AFP rate is an indicator of surveillance sensitivity and should be equal to or more than 1: 1,00,000 (background rate of AFP) according to National Polio Surveillance Project.⁹

AFP surveillance is for detecting polio virus transmission.

Outbreak response immunization (ORI)

The incubation period of the polio virus is 4-35 days prior to weakness and all children 0-59 months of age in the affected area (around 500 children) where the child resided or visited in the incubation period are given active immunization. The cases that are likely to be polio in the community are also actively investigated by the SIO and in AFP cases with inadequate stool specimen, 60 day follow up is done between 60 and 90 days.

The post certification strategy is also developed to maintain a polio free world. Its goals are

1. Contain poliovirus sources by ensuring that they are controlled and removed
2. Withdraw OPV and immunize with IPV against possible re-emergence of any polio virus
3. Defect and respond promptly to any polio virus reintroduction.

The trivalent OPV which was in use till 2016 had a highest sero conversion rates for type 2 and hence wild polio virus type 2 was eradicated in 1999. Most cases of vaccine derived Polio Myelitis are due to OPV 2 and the trivalent OPV was withdrawn and replaced with bivalent OPV since April 2016.

Conclusion

AFP is a complex clinical syndrome that requires immediate and careful evaluation of the differential diagnoses. Each case of AFP is an emergency from both clinical and public health perspective. The precise knowledge of the etiology, underlying pathophysiologic mechanisms and anatomic changes have profound implications for prognosis and treatment. The role of

Table IV. Endgame Polio Strategy 2019-23 – Goals

Goal one: <i>Eradication</i>	Interrupt transmission of all wild poliovirus (WPV) Stop all circulating vaccine-derived poliovirus (cVDPV) outbreaks within 120 days of detection and eliminate the risk of emergence of future VDPVs
Goal two: <i>Integration</i>	Contribute to strengthening immunization and health systems to help achieve and sustain polio eradication Ensure sensitive poliovirus surveillance through integration with comprehensive vaccine preventable disease (VPD) and communicable disease surveillance systems Prepare for and respond to future outbreaks and emergencies
Goal three: <i>Certification & containment</i>	Certify eradication of WPV Contain all polioviruses

infectious agents and immune processes as significant causes of AFP are complemented by different natural and man made toxins. Epidemiologic and clinical surveillance require detailed knowledge of the potential differential diagnosis of AFP. Clinicians must be aware of the causes of AFP and of the need to continuously investigate AFP cases. The health workers also need to be aware of the need for reporting all AFP cases, collecting stool specimens and testing for the poliovirus.

Points to Remember

- *Clinical features of polio must be taught to the younger residents as imported or vaccine associated polio can still occur*
- *GBS requires prompt diagnosis and management and it is the major AFP now because of the spurt of various viral infections.*
- *Transverse myelitis (TM) and traumatic neuritis are the other common causes of AFP.*
- *TM with long segment involvement may be mistaken for GBS because of lack of sensory level and prolonged spinal shock which may be due to enterovirus related TM, or NMO (neuromyelitis optica).*
- *In TM preservation of dorsal column (joint position sensation) → Anterior cord syndrome → Anterior spinal artery occlusion*
- *Rabies can present with features of GBS.*

References

1. Kasper DL, Fauci AS, Hauser SL, Longo DL, Jameson JL, Loscalzo J (eds). *Harrisons Principles of Internal Medicine*. 19th Ed. New Delhi: Mc Graw Hill; 2015.
2. Campell WW. *DeJong's The Neurologic Examination*. 7th Ed. Philadelphia. Lippincot Williams Wilkins; 2012.
3. Daroff RB, Jankovic J, Mazziotta JC, Pomeroy SL (editors). *Bradley's Neurology in Clinical Practice*. 7th Ed. Amsterdam Elsevier; 2015.
4. World Health Organization. *Acute flaccid paralysis*. [Internet] 2013. Available at [https://www.hpsc.ie/acute flaccid paralysis afp > guidance > File, 14207, en](https://www.hpsc.ie/acute-flaccid-paralysis-afp/guidance/File_14207_en). Last accessed on January, 2020.
5. Suresh S, Forgie S, Robinson J. Non-polio Enterovirus detection with acute flaccid paralysis: A systematic review. *J Med Virol* 2018; 90(1):3-7.
6. Messacar K, Schreiner TL, Van Haren K, Yang M, Glaser CA, Tyler KL, et al. Acute flaccid myelitis: A clinical review of US cases 2012-2015. *Ann Neurol* 2016; 80(3):326-338.
7. Krishnan C, Kaplin AI, Pardo CA, Kerr DA, Keswani SC. Demyelinating disorders: update on transverse myelitis. *Curr Neurol Neurosci Rep* 2006; 6:236-243.
8. Maloney JA, Mirsky DM, Messacar K, Dominguez SR, Schreiner T, Stence NV. MRI findings in children with acute flaccid paralysis and cranial nerve dysfunction occurring during the 2014 enterovirus D68 outbreak. *AJNR Am J Neuroradiol* 2015; 36(2):245-250.
9. Suresh S, Rawlinson WD, Andrews PI, Stelzer-Braid S. Global epidemiology of nonpolio enteroviruses causing severe neurological complications: A systematic review and meta-analysis. *Rev Med Virol* 2019; e2082-2087.

CLIPPINGS

High-flow nasal cannula therapy as apneic oxygenation during endotracheal intubation in critically ill patients in the intensive care unit: a systematic review and meta-analysis.

Hypoxemia, a frequently reported complication of intubation, is considered a predisposing factor for cardiac arrest and death. Therefore, oxygenation during endotracheal intubation plays an important role in prolonging the maintenance of acceptable oxygen saturation levels. Authors conducted asystematic review and meta-analysis to assess the clinical efficacy of high-flow nasal cannula (HFNC) therapy as apneic oxygenation in critically ill patients who require endotracheal intubation in the intensive care unit (ICU). Review included six randomized controlled trials and a prospective study identified in PubMed, Embase, Cochrane Library, and the Web of Science until August 18, 2019 involving 956 participants Risk ratio of severe hypoxemia decreased with increasing baseline partial oxygen pressure (PaO₂) to fraction of inspired oxygen (FiO₂) ratio in the study group. In subgroup analysis, HFNC significantly reduced the incidence of severe hypoxemia during endotracheal intubation in patients with mild hypoxemia (PaO₂/FiO₂> 200mmHg. The authors concluded that HFNC was non inferior to standard of care for oxygen delivery during endotracheal intubation and was associated with a significantly shorter ICU stay. The beneficial effect of HFNC in reducing the incidence of severe hypoxemia was observed in patients with mild hypoxemia.