A Brainstem Death that recovered !!

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Introduction

- 44 Year Old Male Farmer by occupation
- K/C/O Hypertension
- Old Right MCA Territory ischemic stroke

Chief Complaints

- Presented with
- Fever with Chills 6 days back
- Vomiting – 4-5 episodes since 4 days
- Breathlessness since 2 days
- Cough since 2 days
- Tingling and numbness since 2 days
- Bilateral Lower Limb Weakness since 2 days
- Swallowing difficulty since 1 day

Negative History

No c/o bowel and bladder involvement

No c/o blurring of vision

No c/o seizures/ altered sensorium

No c/o loose stools/ vomiting/ URTI

No c/o Diurnal variation of symptoms

No h/o similar complaints in the past

No h/o rash

No h/o drug intake

No h/o sleeping on ground/ fang marks

No h/o pesticide exposure

ON EXAMINATION

- BP – 130/90 mmhg
- Pulse- 84 bpm
- Higher Mental Function – Could not be assesed
- **Single breath count – 10**
- Pupils- Bilateral equally reactive to light
- Fundus- Normal

- Cranial Nerve Examination-
- Bilateral LMN facial palsy
- EOM – Ophthalmoplegia Present
- Gag reflex – weak
- Nasal Annotation of voice present

Motor examination

- Tone Normal in both lower limbs

POWER	PROXIMAL	DISTAL
UPPER LIMB	0/5	0/5
LOWER LIMB	0/5	0/5

Neck flexor weakness present

- Reflexes- Areflexia in all 4 limbs
- Plantars- mute

Provisional Diagnosis

1. **Fulminant GBS**
2. **OP Poisoning**
3. **Neuroparalytic Snake Bite**
4. **Myasthenia Gravis**
5. **Bickerstaff Encephalitis**

Investigations :

- CBC – Normal
- ESR -49
- RFT/ELECTROLYTES/ CPK-NAC – normal
- HIV/HBSAG/ HCV- negative
- T3/T4/TSH – normal
- Coagulation Profile- Normal

- Patient intubated in view of Respiratory Distress
- However obeying commands

Investigations :

- CSF-R/M done in first week of illness

-Proteins – 65.3

-Glucose – 68 mg/dl (corresponding BSl- 110)

- Cells -2 (both lymphocytes)

- **NCS- AMSAN Variant of GBS**

- Serum Cholinesterase- 7624 (7000-19000 U/L)

- Drug Panel- Cannabis, Cocaine, Methadone, Methamphetamine, Morphine, Phencyclidine, Propxyphene, Cotinine, Ketamine- NEGATIVE

- CSF-R/M done in second week of illness

-Proteins – 389

-Glucose – 74 mg/dl (corresponding BSl- 112)

- Cells -2 (Both lymphocytes)

Treatment

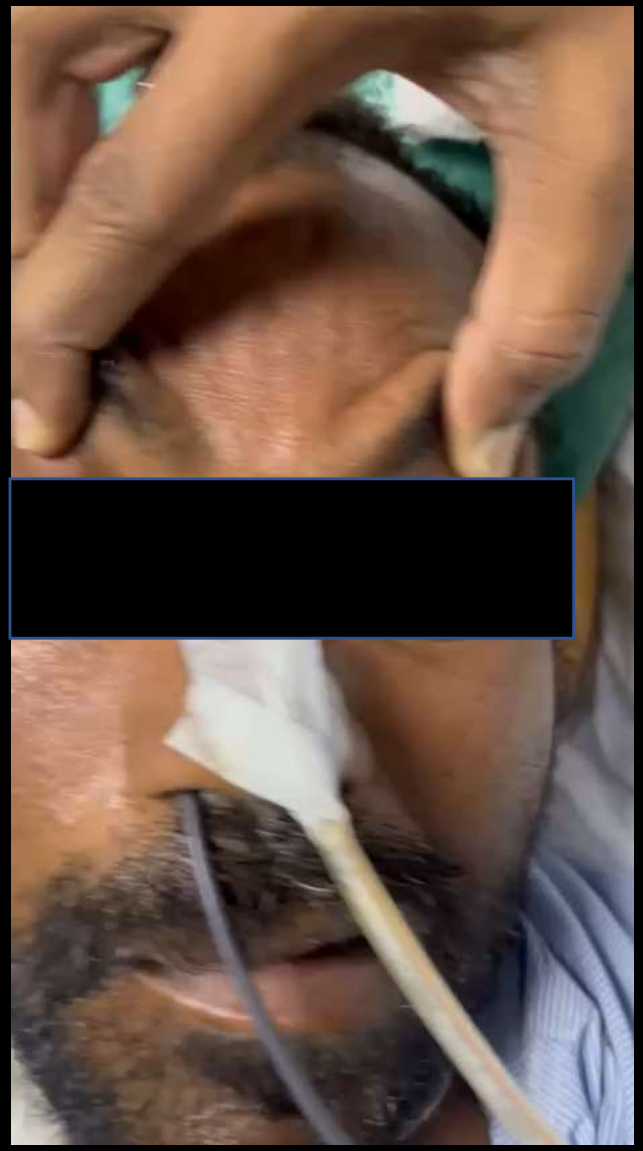
PLASMAPHARESIS

- Patient was clinically deteriorating even on Plasmapharesis,
- After 3rd Cycle of Plasmapharesis, patient became unresponsive overnight
- O/E E1VTM1

Brain stem Reflexes

1. Pupils- normal size, **Non Reactive** to Light
2. Cough Reflex- Absent
3. Corneal Reflex- Absent
4. Conjuctival Reflex- Absent
5. Doll's Eye Reflex- Absent
6. Respiratory Triggers Absent on CPAP Mode

Clinically no Brainstem responses. Short of Apnea test, patient Clinically had Brainstem death



What was the cause of brainstem death?

- Any thoughts ?

- No h/o hypoxic brain injury
- Were we dealing with some other illness ?

We were checking his responsiveness everyday….. Then after 9 cycles of PLEX

- Over the next few days
- Pupils- Bilateral Reactive to Light
- Brain stem reflexes - Present

- Completed 11 Cycles of Plasmapharesis
- Completed 5 days of IVIG

Other Brain Death Mimics

- Snake envenomation
- Locked In Syndrome
- OP Poisoning
- Drugs- Baclofen , Tricyclic antidepressants ,Bupropion, Barbiturates.
- Hypothermia.
- Bickerstaff Encephalitis

- This case highlights the importance of recognizing GBS as a potential cause of severe coma that can mimic brain death and respiratory distress.

- Further diagnostic evaluation, including normal brain activity on EEG and CSF results with albuminocytological dissociation, pointed toward a rare, severe form of GBS.

- Early identification and initiation of Plasmapharesis was a crucial step in the management.

- Thorough history-taking with repeated investigations was crucial in accurately diagnosing GBS.

- This case underscores the significance of conducting a comprehensive neurological exam in patients with atypical presentations to ensure an accurate diagnosis and avoid potentially fatal misdiagnosis.

- When patients appear atypically, the diagnosis of GBS is commonly missed.

- this case to raise awareness of the possibility that GBS may initially emerge with symptoms resembling false brain stem death.

Guillain Barré Syndrome Mimicking Brain Death: A Case Report

Lijo James [1], Aiswarya Mohan [2], Jithu Mol Thankam [3], Jeeva George [3]

Affiliations + expand

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Abstract

Here, we present a case of Guillain-Barré syndrome (GBS) that mimicked brain death. A 66-year-old lady with a medical history of breast cancer (now receiving hormone therapy), hypertension, and hypothyroidism, presented to the emergency department. The patient was admitted to the neuro ICU with absent brainstem and spinal cord responses, concerning for possible brain death. Further evaluation, however, identified the uncommon GBS with respiratory failure. This case emphasizes the need to recognize this severe manifestation of GBS since misinterpreting this as brain death might result in the discontinuation of ventilatory support.

Keywords: false brain death; guillain-barré syndrome (gbs); immunotherapy; quadriparesis; respiratory failure.

Parallel session 6: Clinical Neurology 2

240 A case of fulminant guillain-barré syndrome mimicking brain death

Jananee Sivagnanasundaram , Rebecca Broad , Lucy Tague , Catarina Quaresma , Richard Dittrich , Fadi Alkufri , Sreedharan Harikrishnan

Abstract

Guillain-Barré syndrome (GBS) encompasses a spectrum of acute immune-mediated polyneuropathies. Rarely, in its fulminant form, it can rapidly progress to mimic brain death. We present a case with diagnostic challenges due to a limited history.

A 58-year-old gentleman presented generally unwell and within hours of admission choked on his dinner and suffered a cardiorespiratory arrest. Review on ITU revealed absent brainstem reflexes apart from a weak pupillary light response.

EEG showed slowing with subtle reactivity, MRI Brain was normal, and CSF revealed albuminocytological dissociation.

On day 27 of admission, he was found to follow commands with eye movements, appearing to be in an incomplete locked-in state. Nerve conduction studies confirmed a severe axonal sensorimotor neuropathy with sural sparing. He was treated with plasma exchange and intravenous immunoglobulins and continues to make significant motor improvements each week.

This case highlights the crucial recognition of this extreme presentation of GBS, as a misdiagnosis of brain death may lead to the withdrawal of ventilatory support. In a third of reported similar cases there has been a good recovery and in the majority (as in our case) serum ganglioside antibodies have been negative, raising the possibility of alternative nodal and paranodal target antigens.

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Table 2 Chronology of clinical events

Day 1: diagnosis of GBS, conscious, flaccid quadriplegia, restricted extraocular movements
Day 2: coma, bilaterally fixed and dilated pupils and loss of extraocular movements
Day 7–10: inexcitable nerves on NCV with CSF protein 57.7 mg/dL
Day19: second cycle of IV immunoglobulins
Day 45: plasma exchange initiated
Day 55: eyelid and extraocular muscle movement
Day 134: extraocular movements, word mouthing, self-voiding, shoulder elevation and neck movements
9 months: as described in *Tables 3* and *4*

causes of acute flaccid areflexic quadriparesis with multiple cranial nerve involvement. Five sessions of plasma exchange (50 mL/kg) were carried out between day 45 to 55. He showed gradual recovery after being in "locked in" state for almost 55 days. At 9 months post on-set the patient has regained bulbar function and has MRC grade 3/5 power in all four limbs. Other published cases with a similar admission pattern were reviewed. The prognosis is usually very poor as most of the patients died or remained severely disabled.

THANK YOU