A CASE OF ACUTE ONSET QUADRIPLEGIA

DEPARTMENT OF GENERAL MEDICINE

- A 27-year-old female, interior designer, was admitted with history of

  - Fever with chills since 3 days
  - Breathlessness on exertion since 2 days
  - Vomitings- 3 episodes
  - Altered sensorium since 1 day

- No H/O Diabetes, Hypertension, Thyroid disorder, and other systemic illness

On examination

- Febrile 101 F, Pr – 98/min, regular, all peripheral pulses palpable
- BP – 120/80 mmHg on admission
- There was mild pallor, no icterus, clubbing, cyanosis, edema and lymphadenopathy

Systemic examination

➢ **CNS examination**:-

- Altered sensorium, GCS-13/15
- Tone - normal in all the limbs.
- Moving all 4 limbs spontaneously
- Reflexes were normal

➢ RS ,CVS and Per abdomen WNL

Investigations

Hb	10.2 gm/dl
Total count	7800
Platelet count	2,28,000
Mcv	88
Urea	22
Creatinine	0.54
BSL random	**550 mg/dl**
Procalcitonin	**2.5**

Total Bilirubin	0.46
Direct	0.06
Indirect	0.41
ALT/SGPT	10
AST/SGOT	16
ESR	**44**
lactate	**1.8**

Sodium	138
Potassium	**1.9**
Chloride	99
Calcium	8.58
Magnesium	1.8
Phosphorus	**4.46**
Uric acid	1.4
Urine ketones	**large**
Urine sugars	**3+**

PH	**6.95**
pco2	15
Hco3	6
p02	75

- BLOOD AND URINE CULTURES WERE SENT

TREATMENT

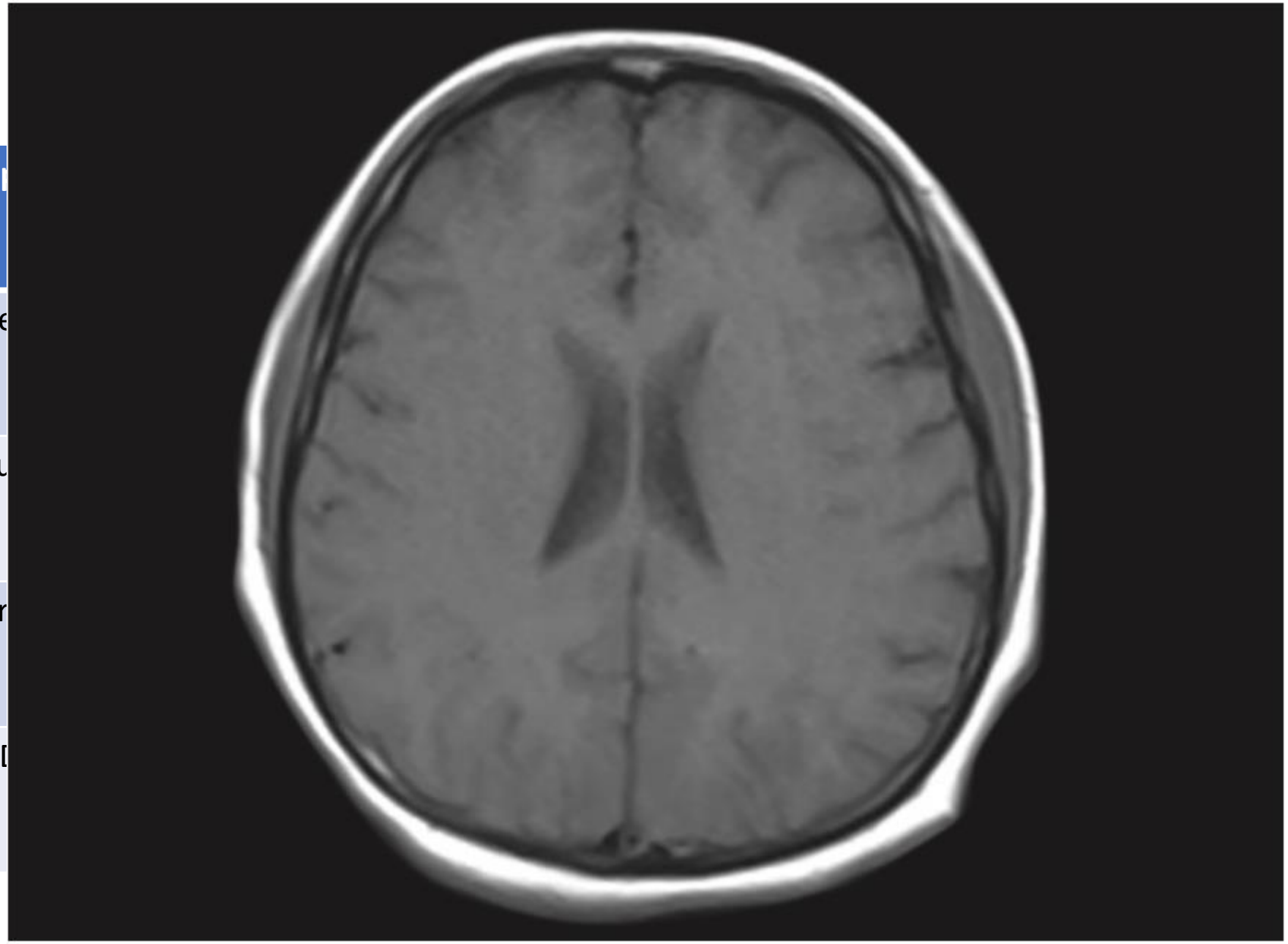
- Inj POTASSIUM CHLORIDE 40 meq iv over 4 hours
  followed by potassium correction according to the serum electrolytes
- Inj CEFTRIAXONE 2gm BD
- Inj HUMAN ACTRAPID INSULIN 6 UNITS iv stat
  Followed by IV infusion and titrated according to BSL
- 2 litres of NORMAL SALINE was given over 3 hours to correct large fluid deficits
  Followed by continuous IV fluids
- Inj SODIUM BICARBONATE 100 meq stat
  (corrected according to the deficit)
- O2 inhalation

- On day 2 , she started developing acute onset flaccid paralysis in all four limbs
- On detailed examination, power was **1/5** in both upper limbs, **0/5** in both lower limbs, all deep tendon reflexes were diminished, and bilateral plantars were mute
- After 4–5 hours, she developed paradoxical breathing, not maintaining saturation in room air, We intubated her immediately and kept her on mechanical ventilation
- Later, She went into cardiac arrest and was revived

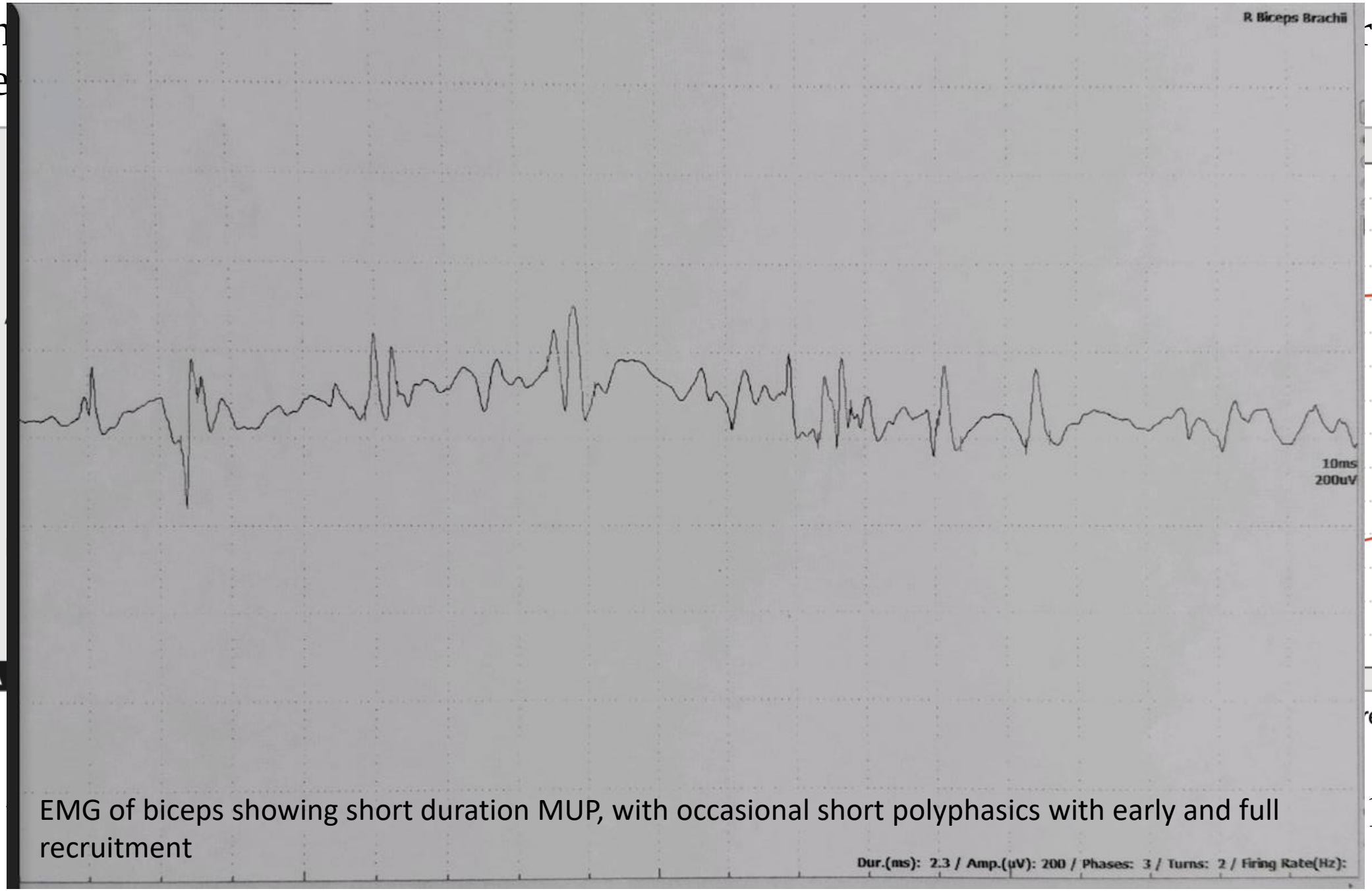
Hb	11.2 gm/dl
Total count	5800
Platelet count	2,28,000
Hba1c	9 %
Urea	25
Creatinine	0.64
BSL random	**220 mg/dl**
Potassium	**2.5**

PH	7.35
Hco3	21
Pco2	36
po2	55

Urine ketones	moderate
glucose	2+
Pus cells	5-6
Rbc	1-2



MRI of brain image showing diffuse cerebral edema



EMG of biceps showing short duration MUP, with occasional short polyphasics with early and full recruitment

DIAGNOSIS

TYPE 1 DIABETES MELLITUS

SEVERE DIABETIC KETOACIDOSIS,SEPSIS

CRITICAL ILLNESS POLYNEUROMYOPATHY

TREATMENT

Mechanical ventilation was continued

Inj MEROPENEM 1gm iv TDS

Inj TEICOPLANIN 200 mg BD

Inj IV IMMUNOGLOBULINS 2gm/kg divided over 5 days

Inj MANNITOL 100ml TDS

Inj HUMAN ACTRAPID INSULIN s/c TDS

- Parenteral nutritional support, antioxidant therapy, and physiotherapy was given accordingly

- Later on, she improved clinically, power was regained, and reflexes were present, We weaned off mechanical ventilation

- Repeat electrophysiological (NCV and EMG) studies suggested the recovery phase of polyneuromyopathy, all other laboratory tests were improved, and she was discharged after 45 days of hospital stay with grade 4/5 power in all the limbs

- She was advised to continue regular physiotherapy and insulin analogues

DISCUSSION

- Critical illnes eferred to as ICU-acquired wea
- The probable ia, severe electrolyte imbalances, r lorgan failure, hyperosmola apy, vasopressors, corticosteroic ycosides
- Clinical susp off from mechanical ventilation, d ness Muscle wasti

Differential diagnoses of failure to wean from mechanical ventilation

Motor neuron	Critical illness polyneuropathy
	Critical illness polyneuropathy/ myopathy
	Amyotrophic lateral sclerosis
	Heavy metal toxicity
	Guillain–Barré syndrome
	Poliomyelitis vasculitis
Neuromuscular junction	Neuromuscular blockade
	Myasthenia gravis
	Lambert–Eaton myasthenic syndrome
	Botulinum toxicity
Muscle	Critical illness myopathy
	Mitochondrial myopathy
	Muscular dystrophy (e.g., myotonic dystrophy)

- The precise mechanism of developing CIPNM is unclear, but circulating factors include cytokines associated with sepsis, and multiorgan failure is thought to play a key role

- It has been reported that 70% of the patients with sepsis syndrome have certain degree of neuropathy and severe respiratory muscle weakness requiring prolonged ventilation resulting in failure to wean off

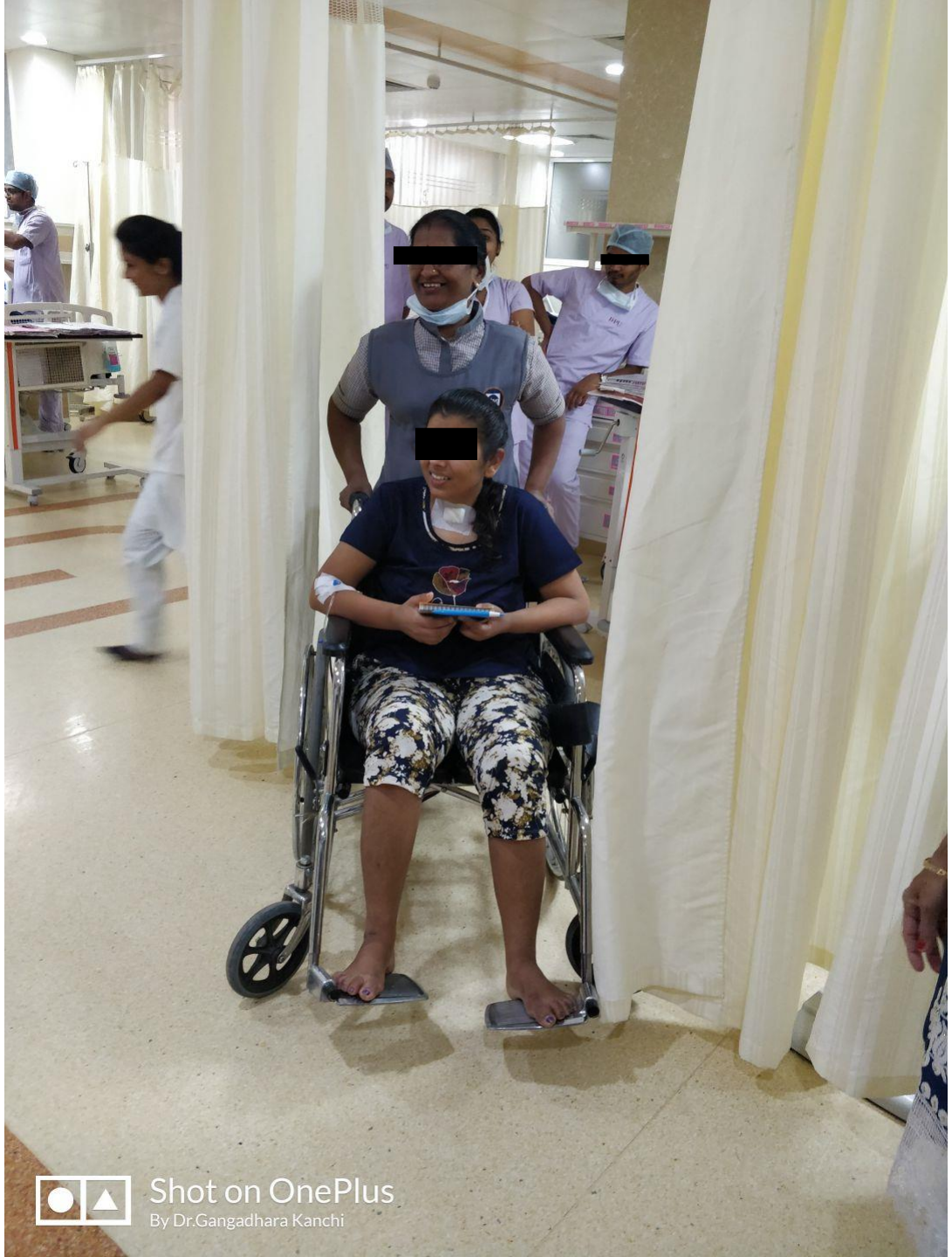
- There might be muscle wasting due to the loss of thick myosin filaments However, the process may take place for many days and needs tracheostomy for prolonged mechanical ventilation

- Some patients have residual long-term weakness with atrophy and muscle fatigue, which was not present in our patient

TAKE HOME MESSAGE

- CIP, CIM, and CIPNM are commonly seen as neuromuscular weakness in patients admitted in ICU
- Once the diagnosis of CIPNM has been established, it is advisable to initiate the management as early as possible in early stages
- Future management strategies should target the proinflammatory cytokines, free radical pathways, controlling the other risk factors that include hyperglycemia, electrolyte imbalances, and sepsis
- According to the analysis in the study by Mohr et al early administration of IVIg might improve and mitigate the CIPNM


Shot on OnePlus
By Dr.Gangadhara Kanchi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Special thanks to the NEUROLOGY department for their effort in prompt diagnosis

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An Unusual Case of Critical Illness Polyneuromyopathy

Madhulika Mahashabde[1], Gaurav Chaudhary[2], Gangadharam Kanchi[3], Shalesh Rohatgi[4], Prajwal Rao[5], Rahul Patil[6], Varun Nallamothu[7]

THANK YOU