CASE REPORT

Diphtheritic Polyneuropathy: A Case Report
Harshavardhan L1, M Ananthakrishna2*
Received: 03 July 2022; Revised: 15 September 2022; Accepted: 29 September 2022

ABSTRACT
Diphtheria is showing a resurgence in recent years. A fall in the immunity of adults to diphtheria due to multiple reasons is showing a rise in diphtheria cases in the adult population. Diphtheritic polyneuropathy shows a prevalence of 20–27% of infections. It affects the axial muscle and the palatine muscle fast.

Here we report a case of diphtheritic polyneuropathy in a 27-year-old COVID-19-infected man.

Introduction
Diphtheritic polyneuropathy can occur 2–50 days after the local diphtheria infection. This is due to exotoxin-mediated inhibition of protein synthesis.

Cases with diphtheritic polyneuropathy show a descending type of muscle paralysis. It shows around 20% of cases with ventilator dependence. It usually shows a slower recovery in comparison to GBS.

Case Description
A 27-year-old male was admitted to KR hospital on 6th May 2021 with moderate COVID-19 infection. The patient had no history of childhood immunization. The patient started treatment according to COVID-19 protocol and was provided with dexam 6 mg OD, Inj remdesivir, and oxygen supplementation. The patient’s oxygen requirement gradually reduced from 8 L/min to 2 L/min over the time of hospital stay. At around 2 weeks of the hospital, stay patient developed difficulty in swallowing both liquid and solid followed by which the patient’s sensorium also started deteriorating, for which the patient was shifted to ICU. Where on examination patient was found to be in a stuporous state with flaccid paralysis of all four limbs with upper limb predominance and the patient was having paradoxical breathing and other signs of diaphragmatic weakness.

CNS Examination

- HMF: Not elicited as the patient was in a stuporous state.
- Cranial nerves: Normal (EOM: Normal) (Pupils: BERL)
- Bulk: Equal
  - ↓↓
  - ↓
- Tone:
  - Absent

- Reflex: Deep
- Sensory examination: Not done
- B/l plantar: Mute

The patient was ruled out of any neuro infections from the MRI brain with contrast and CSF analysis being normal. The patient’s ABG was suggestive of type 2 respiratory failure in CO2 narcosis (PCO2–111 mm Hg). For the same, the patient was intubated to put on mechanical ventilator support, during the process of intubation a greyish-yellow membrane was noted in the patient’s tonsillar, para-tonsillar, and parapharyngeal area. This membrane was removed with great difficulty with no bleeding manifestation from the site and was sent for microscopic examination (Fig. 1). Which suggested clumps of C. diphtheria with Albert staining showing bipolar metachromatic

![Fig. 1: Microscopy of the membrane, Albert stain showing k.l.b](image)

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![Fig. 2: Patient showing improvement in weakness after antitoxin injection](image)

Fig. 2: Patient showing improvement in weakness after antitoxin injection

1Associate Professor, 2 Junior Resident, Department of Medicine, Mysore Medical College and Research Institute, Mysuru, Karnataka, India; 3 Corresponding Author

How to cite this article: L H, Ananthakrishna M. Diphtheritic Polyneuropathy: A Case Report. J Assoc Physicians India 2023;71(3):92–94.

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Diphtheritic Polyneuropathy

The patient was started on procaine penicillin G 6 lakh unit BD and also with 1 lakh unit of diphtheria antitoxin IV stat after consultation with WHO personnel.

After intubation and CO₂ washout, the patient regained sensorium. And then after the antitoxin dose, the patient drastically showed improvement in his weakness to the extent he was able to lift all four limbs (Fig. 2).

A detailed CNS examination was reviewed: he had gained consciousness and revealed quadriplegia with UL power 3/5 and LL power 4/5, with no sensory involvement. Pure motor quadriplegia.

But still, the patient’s diaphragmatic weakness persisted for which the patient required around 2 months of ventilator support and diaphragmatic exercise.

The patient was further evaluated with NCS of both upper limbs and lower limbs with phrenic nerve NCS, which concluded the patient was having demyelinating polyneuropathy suggestive of diphtheritic polyneuropathy (Figs 3 and 4).

Patient was discharged after the patient was no longer ventilator dependent and had no residual weakness (Fig. 5). On follow-up, patients’ diphtheria IgM titers were evaluated which showed an increasing trend in a gap of 2 months. A repeat NCS was not done due to financial constraints (Fig. 6).

Discussion

Acute flaccid paralysis is a common neurological abnormality that occurs in a wide variety of situations like infections and immunological neuronal damage.¹

One of the most common causes of flaccid paralysis in adults is GBS.²

Fig. 3: NCS of patient showing demyelinating polyneuropathy

Fig. 4: Phrenic nerve NCS showing demyelinating with axonopathy

Fig. 5: Patient showing improvement in diaphragmatic weakness, patient off the ventilator
But studies show that diseases like diphtheritic polyneuropathy must be considered above GBS in clinical circumstances suggestive of diphtheria exposure.\(^1,2\)

The exotoxins of diphtheria bind to the HB-EGF receptor found predominantly on Schwann cells and myocardium. This exotoxin causes inhibition of protein synthesis.\(^3,4\)

The first symptom occurred 2–50 days after the onset of local diphtheria infection. The neurological deterioration continued for a median of 49 days and improvement started 73 days after onset. Bulbar dysfunction occurred in 98% of patients with diphtheritic polyneuropathy. Patients were ventilator dependent for longer duration in diphtheritic polyneuropathy with a comparison with GBS.\(^3-5\)

Antitoxin has been tried in diphtheritic polyneuropathy and shows the most effective within 2–3 days of disease onset.\(^1,3,4\)

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