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A STUDY OF ACUTE FLACCID PARALYSIS IN AN ADOLESCENT WORKER: OCCUPATIONAL HEALTH IMPLICATIONS OF VITAMIN D DEFICIENCY–RELATED METABOLIC BONE DISEASE

Dr. Gopinath Gorige* Junior Resident, Department of General Medicine,DY Patil University, Navi Mumbai, Maharashtra, India*Corresponding Author

Dr. Prachi Sankhe Associate Professor, Department of General Medicine,DY Patil University, Navi Mumbai, Maharashtra, India

Dr. Smita Patil Professor and Head of Department, Department of General Medicine, DY Patil University, Navi Mumbai, Maharashtra, India

ABSTRACT

Acute flaccid paralysis (AFP) is a neuromuscular emergency in which Guillain–Barré syndrome, spinal cord pathology and metabolic derangements are key differentials. Vitamin D deficiency is highly prevalent among Indian adolescents, particularly indoor workers with limited sun exposure, and can cause secondary hyperparathyroidism, hypocalcemia and hypokalemia. A 15-year-old indoor factory helper presented with 24-hour ascending paraparesis (power 3/5, areflexia, preserved sensation). MRI brain and spine, cerebrospinal fluid and nerve conduction studies were normal. Biochemistry showed hypocalcemia (7.1 mg/dL), hypokalemia (3.1 mEq/L), severe vitamin D deficiency (3.7 ng/mL), elevated total CPK (1124 U/L), and markedly elevated parathyroid hormone (214.6 pg/mL), consistent with vitamin D deficiency-related metabolic bone disease with secondary hyperparathyroidism. Intravenous calcium, potassium supplementation and high-dose cholecalciferol led to complete neurological recovery within 72 hours. Early metabolic profiling in AFP, especially among adolescent indoor workers, is essential to identify this reversible etiology, avoid unnecessary immunotherapy and enable timely, safe return to work.

INTRODUCTION

Acute flaccid paralysis (AFP) denotes rapid onset limb weakness with hypotonia and areflexia, constituting a neuromuscular emergency due to risk of respiratory failure and autonomic instability. Classical causes include Guillain-Barré syndrome (GBS), poliomyelitis, transverse myelitis, and acute myelopathies; however, metabolic and electrolyte disturbances are increasingly recognized as important reversible differentials. Vitamin D deficiency is a major public health problem in India, with high prevalence among children and adolescents due to indoor lifestyles, limited sunlight exposure, skin pigmentation, and inadequate dietary intake. Indoor and shift workers constitute an occupational risk group because their routine restricts ultraviolet B exposure and often coincides with nutritionally suboptimal diets. Chronic hypovitaminosis D leads to secondary hyperparathyroidism, osteomalacia or rickets, and symptomatic hypocalcemia, which may present as neuromuscular irritability, tetany, seizures, arrhythmias, or acute weakness resembling AFP. This study presents a descriptive case report of AFP in an adolescent indoor worker due to vitamin D deficiency-related metabolic bone disease and discusses diagnostic differentiation from GBS and occupational health implications.

CASE STUDY

A 15-year-old male, employed as an indoor factory helper, presented to the tertiary care centre with acute-onset progressive weakness of both lower limbs over 24 hours. He reported difficulty climbing stairs and rising from squatting, progressing to inability to stand without support on the day of admission. There was no history of preceding fever, respiratory or gastrointestinal illness, vaccination, trauma, new medication, or toxin exposure.

He worked long daytime shifts in an enclosed industrial setting with minimal sun exposure and had no regular outdoor physical activity. His diet was predominantly cereal-based with low intake of dairy products and no vitamin D-fortified foods. There was no history of similar episodes, seizures, back pain, sphincter disturbance, or sensory symptoms.

On examination, vital signs were stable. Higher mental functions and cranial nerves were normal. Motor examination revealed hypotonia in both lower limbs with Medical Research Council grade 3/5 power at hips and knees bilaterally; upper limb tone and power were normal. Deep tendon reflexes were absent in both lower limbs but preserved in upper limbs; plantar responses were flexor bilaterally. Sensory examination was intact for all modalities. No spinal deformity, tenderness, meningeal signs, or systemic illness features were present. Provisional diagnoses considered included GBS, acute myelopathy, and metabolic or electrolyte-mediated paralysis.

INVESTIGATIONS

MRI of the brain and entire spine was normal, without evidence of demyelination, cord compression, or transverse myelitis. Cerebrospinal fluid (CSF) analysis showed normal cell count and protein, without albuminocytologic dissociation, making classic GBS less likely. Nerve conduction study and electromyography did not reveal demyelinating or axonal features of acute inflammatoryRoutine hematolo gical, renal, and liver function tests were within normal limits. Electrocardiogram showed normal sinus rhythm without QT prolongation.

Key biochemical abnormalities were: corrected serum calcium 7.1 mg/dL (low), serum potassium 3.1 mEq/L (low), 25-hydroxyvitamin D 3.7 ng/mL (severely deficient), total CPK 1124 U/L (elevated), and parathyroid hormone (PTH) 214.6 pg/mL (markedly elevated). Serum magnesium, phosphate, fasting blood glucose, and thyroid function were within reference ranges. These findings were consistent with vitamin D deficiency-related metabolic bone disease with secondary hyperparathyroidism and associated metabolic myopathy. A final diagnosis of metabolic myopathy secondary to vitamin D deficiency-related metabolic bone disease with secondary hyperparathyroidism was established.

Table 1: Key Biochemical Abnormalities

Parameter	Value	Reference Range	Interpretation
Serum calcium	7.1 mg/dL	8.5–10.2 mg/dL	Low
Serum potassium	3.1 mEq/L	3.5–5.0 mEq/L	Low

25-OH vitamin D	3.7 ng/mL	30–100 ng/mL	Severely deficient
Total CPK	1124 U/L	Approx. up to 170–308 U/L	Elevated
Parathyroid hormone	214.6 pg/mL	15–65 pg/mL	Elevated

TREATMENT

The patient was initiated on intravenous calcium gluconate with continuous cardiac monitoring, followed by oral calcium supplementation. Intravenous and oral potassium replacement were administered to correct hypokalemia with serial electrolyte monitoring. High-dose cholecalciferol was started for rapid vitamin D repletion, followed by maintenance oral vitamin D and calcium supplementation per adolescent deficiency protocol. Early physiotherapy and graded mobilization were instituted.

Within 12 hours of initiating metabolic correction, the patient reported improvement in lower limb strength. At 36 hours, objective motor power had improved and lower limb deep tendon reflexes began to return. By 72 hours, motor power in both lower limbs was 5/5, reflexes were normal, and the patient walked independently. No respiratory compromise or arrhythmia occurred. He was discharged on oral calcium and vitamin D supplementation with nutritional and sun-exposure counselling and scheduled follow-up.

Table 2: Differentiation of Metabolic Paralysis from Guillain-Barré Syndrome

Feature	This Case (Metabolic)	Guillain-Barré Syndrome
Antecedent infection	Absent	Usually present
CSF protein	Normal	Elevated / albuminocytologic dissociation
NCS/EMG	Normal	Demyelinating / axonal pattern
Biochemical profile	Hypocalcemia, low vitamin D, elevated PTH, high CPK	Usually normal
Recovery timeline	Complete in 72 hours	Weeks to months
Treatment	Metabolic correction	IVIG / plasmapheresis

CONCLUSIONS

Vitamin D deficiency-related metabolic bone disease with secondary hyperparathyroidism must be considered as a reversible cause of AFP in adolescent indoor workers, particularly in the Indian context. Routine metabolic profiling—serum calcium, electrolytes, 25-hydroxyvitamin D, PTH, and CPK—should be integrated early in the AFP workup to identify treatable causes and prevent misdiagnosis as GBS. Timely correction of metabolic derangements enables complete neurological recovery within 72 hours, avoiding unnecessary immunotherapy. Occupational health strategies including scheduled sun-exposure breaks, nutritional counselling, and targeted supplementation programs for adolescent indoor workers are essential to reduce preventable neuromuscular morbidity and support safe return to work.

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