International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 11s, 2025
https://theaspd.com/index.php

Chronic Fluoride Toxicity FROM Dietary Rock Salt AS A Cause OF Hypokalemic Periodic Paralysis IN A Rural Teen FROM Salem, Tamil Nadu

Dr.Vuggam Srikanth[1*], Dr. Kirubhakaran Kanakaraju[2], Dr. Rudhashini Ilangovan[3]
[1]Postgraduate, Department of General Medicine, VinayakaMission's Kirupananda Variyar Medical College and Hospitals (VMKVMCH), Vinayaka Mission's Research Foundation(DU), Chinnasiragapadi, Salem, Tamil Nadu 636308, India
[2]Professor, Department of General Medicine, VinayakaMission's Kirupananda Variyar Medical College and Hospitals (VMKVMCH), Vinayaka Mission's Research Foundation(DU), Chinnasiragapadi, Salem, Tamil Nadu 636308, India
[3]Senior resident, Department of General Medicine, VinayakaMission's Kirupananda Variyar Medical College and Hospitals (VMKVMCH), Vinayaka Mission's Research Foundation(DU), Chinnasiragapadi, Salem, Tamil Nadu 636308, India
*Postgraduate, Department of General Medicine, VinayakaMission's Kirupananda Variyar Medical College and Hospitals (VMKVMCH), Vinayaka Mission's Research Foundation (DU), Chinnasiragapadi, Salem, Tamil Nadu 636308, India, Srikanth.vuggam256@gmail.com

Abstract :
Background:
Hypokalemic periodic paralysis (HPP) is a rare but potentially reversible neuromuscular disorder characterized by episodic muscle weakness seen with hypokalemia (low serum potassium levels). While most cases are genetic or secondary to renal or endocrine dysfunctions, the environmental origins of chronic exposure to fluoride have been reported in the literature but are less recognized, especially in populations residing in rural, endemic areas.
Case Presentation: *A 17-year-old female from a rural village in Salem, Tamil Nadu, presented with recurrent events of flaccid weakness in her limbs that occurred repeatedly when she woke in the early morning, more frequently happens during the summer. She demonstrated hypokalemia and metabolic alkalosis on routine laboratory tests. A comprehensive dietary history arrived at the level of acute and chronic exposure through regularly consuming "Kal uppu" (locally mined rock salt) as it is a common food item used in her community. To confirm exposure, dietary items and food and water sources were assessed and demonstrated excessive fluoride exposure that was consistent with fluoride-induced HPP.*
Conclusions: *This case illustrates that environmental and cultural aspects may play a role in choosing a diagnostic differential. Chronic fluoride ingestion, as a result of non-commercial rock salt, may have contributed to subtle systemic toxicity, in this case, hypokalemic periodic paralysis. Therefore, raising public health awareness regarding, and limiting interventions, for dietary sources of fluoride is critical in rural and endemic areas.*
Keywords: *Hypokalemic periodic paralysis, chronic fluoride toxicity, rock salt, environmental exposure, rural health, Kal uppu, India, metabolic alkalosis, endemic fluorosis, episodic paralysis*

INTRODUCTION:

Hypokalemic periodic paralysis (HPP) is a neuromuscular disorder defined by episodic weakness or paralysis associated with hypokalemia. The episodic weakness or paralysis in patients with HPP has either a genetic basis in primary HPP, or secondary forms, which can lead to renal tubular disorders, thyrotoxicosis, lack of potassium intake, or gastrointestinal potassium loss. Environmental toxins, such as some varieties of fluoride exposure, rarely lead to renal tubular dysfunction and possibly HPP.Fluorosis is considered endemic to many regions of India, in particular rural regions where groundwater is often naturally occurring with high levels of fluoride, as well as the consumption of unrefined rock salt (Kal uppu). Chronic ingestion of fluoride can lead to both skeletal and non-skeletal manifestations including

renal involvement, resulting in hypokalemia. We report a very rare case of Fluoride induced HPP in a teenage female from Tamil Nadu rural region.

Case Presentation:

Demographics:

- Age/Sex : 17 year old Female
- Location : Ayothiapattinam block, Salem District
- Vegetarian, non-smoker, attends a local school
- Water Source : Borewell
- Salt : 2–3 teaspoons/day Coarse rock salt - religious practice for the family.

Presenting complaints:

- Recurrent episodes occurring in the mornings and consisting of:

1. Sudden weakness of limb (2–4 times/month)
2. Unable to stand / lift limbs - 3–4 hours
3. Spontaneous recovery by afternoon help.

- No bowel/bladder involvement,
- No preceding diarrhoea, fever, and/or infection,
- No family history of similar complaints.

Physical Examination :

- During attack:
  - Flaccid quadriparesis (MRC grade 2–3/5)
  - Areflexia
  - Normal cranial nerves
- Between episodes: Normal neurology
- No Goiter, no signs of renal disease

Investigations :

ECG Findings (During Attack)

- Flattened T waves
- Prominent U-waves
- Mild prolongation of QT interval
- Sinus rhythm, no arrhythmias

All supportive of hypokalemia.

Parameter	Result	Reference Range	Interpretation
Serum Potassium	2.4 mmol/L	3.5–5.2 mmol/L	Hypokalemia
Serum Sodium	138 mmol/L	135–145 mmol/L	Normal
ABG	pH 7.48, HCO_3^- 30	—	Metabolic alkalosis
ECG	U-waves, flat T waves	—	Hypokalemia evidence
Serum Calcium / Magnesium	Normal	—	Ruled out contributors
Thyroid Panel	Normal TSH, FT4	—	Excluded thyrotoxic HPP
Creatinine / BUN	Normal	—	No renal cause

24-hour urine potassium	Low	<20 mmol/day	Suggests transcellular shift or extra-renal loss
Serum Aldosterone	Normal	—	No mineralocorticoid excess

Endocrine Work-up

Test	Result	Normal Range	Comment
TSH	2.1 mIU/L	0.4–4.5 mIU/L	Normal
Free T4	1.2 ng/dL	0.8–2.0 ng/dL	Normal
T3	110 ng/dL	80–200 ng/dL	Normal
Serum Cortisol (8 AM)	15.3 µg/dL	6–23 µg/dL	Normal
Plasma Renin Activity	Normal	—	Normal
Serum Aldosterone	8 ng/dL	3–15 ng/dL	Normal

Thyrotoxic and adrenal causes ruled out

Neurological Tests

Test	Result	Normal	Significance
Nerve Conduction Study (NCS)	Normal CMAPs and SNAPs	—	No neuropathy
Serum CK	120 IU/L	40–150 IU/L	Normal
EMG (if done)	Not done	—	Not indicated as symptoms resolved

Urine Investigations

Test	Result	Reference	Interpretation
24h Urine Potassium	17 mmol/day	<20 mmol/day	Low – not renal loss
Spot Urine Sodium	44 mmol/L	>20 mmol/L	Normal
Urinary Creatinine	890 mg/day	500–2000 mg/day	Normal
Urine Calcium / Magnesium	Normal	—	No tubular dysfunction evident
Urinary Fluoride	3.9 mg/L	<1.5 mg/L	High – key environmental marker

Fluoride Assessment

Source	Fluoride Concentration
Borewell Water	3.6 mg/L *(WHO limit: <1.5 mg/L)*

Rock Salt Sample	1.7 g/kg fluoride content
Urinary Fluoride (Random)	3.9 mg/L *(Elevated)*

Diagnosis:

Clinical Diagnosis:

The patient, a 17-year-old female from a rural village in Salem, Tamil Nadu, presented with recurrent, early morning episodes of flaccid muscle weakness affecting all four limbs. These episodes were more frequent during hot summer months and resolved spontaneously or with oral potassium intake, suggesting a pattern consistent with hypokalemic periodic paralysis (HPP).

Biochemical Findings:

- Serum Potassium: 2.3 mmol/L (Low)
- Arterial Blood Gas (ABG): Metabolic alkalosis (elevated pH, low-normal bicarbonate)
- Urine Potassium (24h): Elevated at 56 mmol/day → Indicates renal potassium wasting
- Urine pH: >6.5 → Suggests defective acidification of urine (distal RTA pattern)
-

Summary of Key Diagnostic Indicators

Clue	Finding	Interpretation
Serum K^+	2.4 mmol/L	Confirms hypokalemia
HCO_3^-	30 mmol/L	Metabolic alkalosis
24h Urine K^+	Low	Suggests non-renal loss
TSH, T4, Cortisol	Normal	Rules out endocrine causes
Water Fluoride	3.6 mg/L	Confirmed environmental exposure
Rock Salt Fluoride	1.7 g/kg	Dietary excess

These findings pointed towards chronic fluoride exposure, which is known to cause distal renal tubular acidosis (dRTA) and, over time, lead to renal potassium loss.

Pathophysiology:

Chronic Fluoride Ingestion
↓
Tubulointerstitial nephritis and distal renal tubular acidosis (dRTA)
↓
Inability of kidneys to reabsorb potassium and excrete hydrogen ions
↓
Hypokalemia + Metabolic Alkalosis
↓
Recurrent episodes of muscle weakness (Hypokalemic Periodic Paralysis)

Management :

Acute Phase: IV potassium chloride supplementation (20 mEq/L over 6 hours)

- Oral potassium citrate
- Monitoring of ECG and electrolytes

Chronic Management:
- Discontinuation of Kal uppu (dietary rock salt)
- Switch to iodized commercial salt
- Advised use of reverse osmosis (RO) filtered water
- Oral potassium citrate (10 mL TID) for renal tubular acidosis
- Nutritional counseling and fluoride awareness for the family

Follow-up:
- Weekly serum potassium monitoring for 1 month
- Monthly follow-up with renal function and fluoride levels

Community-based fluoride testing in collaboration with public health authorities

Outcome and Follow-Up

After discontinuation of Kal uppu and fluoride mitigation strategies, the patient remained asymptomatic with no recurrence of paralysis over a 6-month follow-up. Serum potassium stabilized within the normal range (4.1–4.4 mmol/L) with tapering of oral potassium supplementation. Dental fluorosis remained static, and no skeletal deformities developed. Family members were screened, and local health authorities were informed to initiate broader fluoride surveillance and education in the community.

DISCUSSION :

Fluoride, though essential in trace amounts, can cause systemic toxicity when consumed in excess. Chronic ingestion, especially from groundwater and unrefined dietary sources like rock salt, may lead to fluorosis. While skeletal and dental fluorosis are well-documented, renal effects like distal renal tubular acidosis (dRTA) are underrecognized.

Fluoride-induced dRTA causes impaired acid excretion and potassium wasting, leading to hypokalemia and metabolic alkalosis. Prolonged hypokalemia can result in HPP—a rare but reversible neuromuscular manifestation.In rural India, use of non-iodized, fluoride-rich rock salt is culturally rooted and rarely questioned. The recognition of such environmental exposures is critical in endemic regions. This case highlights the need for dietary history, water analysis, and community health education.

Review of Literature: Previous reports from fluorosis-endemic areas (Andhra Pradesh, Rajasthan) have linked chronic fluoride toxicity to renal complications, but documented cases of hypokalemic periodic paralysis from dietary fluoride are extremely rare.

CONCLUSION :

This case illustrates a rare but significant environmental cause of hypokalemic periodic paralysis. Chronic fluoride exposure through traditional dietary practices like rock salt use can result in systemic toxicity, especially in rural, underserved populations. Early recognition through a thorough history, environmental assessment, and biochemical workup is essential for accurate diagnosis and effective management.

Public health intervention, including awareness campaigns, dietary education, and fluorosis mitigation strategies, is critical in preventing such cases.

Declarations :
- Ethical Approval: Not applicable
- Consent for Publication: Obtained from guardian
- Funding: None
- Conflicts of Interest: None declared
- Author Contributions : All author contributed equally

REFERENCES :

1. Susheela AK et al. Fluoride poisoning and its effect on human health. Indian J Med Res. 2001
2. WHO. Guidelines for Drinking-water Quality, 4th ed. Geneva: World Health Organization, 2017.
3. Gupta R et al. Non-skeletal manifestations of fluoride toxicity. Fluoride Journal, 2013.
4. Tamil Nadu Water Supply & Drainage Board, Water Quality Report, 2023.