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AN INTERESTING CASE OF ACUTE FLACCID PARALYSIS: A DIAGNOSTIC CHALLENGE

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INTRODUCTION

Acute Flaccid Paralysis (AFP) is a complex, neurological clinical syndrome characterized by the rapid onset of profound muscle weakness, hypotonia, and diminished or absent deep tendon reflexes. The progression of weakness can be aggressive, occasionally leading to devastating complications such as bulbar palsy, acute respiratory failure, and death if not promptly identified and managed. While it is not a single disease entity, AFP serves as a critical red-flag presentation in emergency medicine and neurology, necessitating an immediate and systematic diagnostic approach to prevent irreversible morbidity. Hypokalemic periodic paralysis is a condition characterized by transient attacks of flaccid muscle weakness. It can be broadly classified into primary (familial) and secondary (acquired) forms. Familial cases are typically due to genetic channelopathies affecting skeletal muscle sodium or calcium channels. Secondary hypokalemic paralysis, on the other hand, arises from a massive intracellular shift of potassium (as seen in thyrotoxic periodic paralysis) or severe depletion of total body potassium due to gastrointestinal or renal losses.

When severe hypokalemia is accompanied by a hyperchloremic, non-anion gap metabolic acidosis, the differential diagnosis narrows significantly toward Renal Tubular Acidosis (RTA). Distal RTA (Type 1), characterized by the failure of the alpha-intercalated cells of the cortical collecting duct to secrete hydrogen ions, leads to a compensatory but catastrophic renal wasting of potassium. This form of acquired RTA is frequently associated with underlying autoimmune disorders, such as Sjögren's syndrome, rheumatoid arthritis, or chronic thyroiditis.

This presentation discusses the compelling case of Ms. Petchiammal, a 23-year-old female who presented with acute, rapidly progressive quadriparesis clinically mimicking Guillain-Barré Syndrome. Her diagnostic workup unmasked a severe underlying metabolic and acid-base disorder, highlighting the critical importance of evaluating electrolyte profiles in all patients presenting with acute flaccid paralysis.

CASE PRESENTATION

Patient Profile and History

Ms. Petchiammal, a 23-year-old female student residing in Ambasamuthiram, presented to the emergency department of Tirunelveli Medical College Hospital (TVMCH) under the VI Medical Unit (under the clinical supervision of Chief Dr. Shavana, M.D.) with a primary complaint of rapidly progressive, acute difficulty in using all four limbs for the past 3 days.

The patient was in her usual state of health until 3 days prior to presentation, when she developed mild-to-moderate, aching muscle cramps localized to the posterior aspect of her calves and thighs. She self-administered Tablet Paracetamol with no relief. The following evening, she experienced a sudden onset of weakness in both lower limbs. This weakness was rapidly progressive and flail-type in nature, starting with an inability to rise from a sitting position, which quickly advanced to an inability to hold her slippers.

She was initially taken to a local hospital where intravenous fluids were administered. However, her condition continued to deteriorate. By the night of the second day, the motor weakness ascended to involve her upper limbs, characterized by difficulty raising her arms above her shoulders and an inability to hold objects. Upon arrival at TVMCH on the third day, the paralysis had progressed to the point where she was completely unable to roll over in bed or lift her head off the mattress.

Review of Systems & Pertinent Negatives

- **Gastrointestinal/Oral:** Positive history of constipation and a persistent burning sensation in her mouth.
- **Respiratory:** No history of shortness of breath, dyspnea, or difficulty breathing.
- **Sensory/Neurological:** No history of numbness, paresthesias, sensory loss, or a band-like sensation around her chest or abdomen. No history of loss of consciousness, speech disturbances, or behavioral/emotional changes. No sleep disturbances, tremors, or cranial nerve symptoms (e.g., normal smell, no blurring of vision, no diplopia, normal facial sensation, no deviation of the mouth, no dysphagia, nasal regurgitation, or hoarseness).
- **Systemic/Infectious:** No history of fever, neck pain, chest pain, cough, diarrhea, or vomiting.
- **Rheumatological:** No history of joint pain, hair loss, oral ulcers, dry eyes, excessive tearing, or dry skin.
- **Other:** No history of recent vaccinations, dog bites, claudication pain, chronic drug intake, or toxin exposure.

Past, Personal, and Menstrual History

- **Tuberculosis History:** Known case of pulmonary tuberculosis at age 18, which was successfully treated with a complete course of Anti-Tubercular Therapy (ATT).
- **Thyroid History:** Recently diagnosed with hypothyroidism 1 month prior; currently managed on Tablet Thyroxine 50mu once daily.
- **Comorbidities:** Not a known case of Type 2 Diabetes Mellitus, Systemic Hypertension, Coronary Artery Disease, seizure disorder, or malignancy. No prior episodes of similar limb weakness or transient ischemic attacks.
- **Menstrual History:** Regular 5/28-day cycles with moderate flow. Last Menstrual Period (LMP) was November 22, 2023.
- **Personal Habits:** Normal bowel/bladder habits, normal sleep and appetite, consumes a non-vegetarian diet, no history of substance abuse or high-risk exposure.

Physical Examination

- **General Status:** Conscious, cooperative, moderately built and nourished. No clinical signs of pallor, icterus, clubbing, cyanosis, generalized lymphadenopathy, or pedal edema. No peripheral nerve thickening or visible muscle fasciculations.
- **Vital Signs.**
  - **Pulse Rate:** 89 beats per minute, regular rhythm, normal volume, no radio-radial or radio-femoral delay.
  - **Blood Pressure:** 110/70mmHg measured in the supine position in the left arm.
  - **Respiratory Rate:** 17 breaths per minute, thoraco-abdominal pattern.
  - **Temperature:** 98.2(afebrile).
  - **Jugular Venous Pressure (JVP):** Not elevated.
  - **Carotid Arteries:** No thrills or bruits.

Detailed Neurological Examination

- **Higher Mental Functions:** Patient was conscious and fully oriented to time, place, and person. Speech, memory (immediate, recent, remote), and cognitive functions were intact. No delusions, hallucinations, or behavioral abnormalities.
- **Cranial Nerves:** All cranial nerves (CN I through CN XII) were tested systematically and found to be completely intact bilateral (with symmetric pupillary responses of 3mm reacting to light, normal extraocular movements, intact facial sensations, normal facial symmetry, intact gag reflex, midline tongue, and normal shoulder shrugging).
- **Motor System**
  - *Bulk:* Symmetric and normal bulk in all muscle groups. No focal or generalized wasting (Arm: 25cm, Forearm: 17cm, Thigh: 45cm, Leg:27cm bilaterally).
  - *Tone:* Marked, symmetric hypotonia (flaccidity) in both upper and lower extremities.
  - *Power:* Severe, symmetric quadriparesis, more pronounced proximally than distally.
    - **Shoulder Joint:** 3/5 (Flexion, Extension, Abduction, Adduction, Rotations)
    - **Elbow Joint:** 3/5 (Flexion, Extension)
    - **Wrist Joint:** 4-/5 (Dorsiflexion, Palmar flexion)
    - **Finger Grip:** 60\% bilaterally
    - **Hip Joint:** 2/5 (Flexion, Extension, Abduction, Adduction, Rotations)
    - **Knee Joint:** 2/5 (Flexion, Extension)
    - **Ankle Joint:** 3/5 (Dorsiflexion, Plantar flexion)
- **Reflexes**
  - *Superficial:* Corneal, conjunctival, palatal, pharyngeal, and abdominal reflexes were all present and symmetric. Plantar response was flexor bilaterally.
  - *Deep Tendon Reflexes (DTRs):* Uniformly sluggish (1+) at the biceps, triceps, supinator, knee, and ankle joints.

- **Sensory System:** Normal and symmetric perception of fine touch, crude touch, pressure, pain, temperature, vibration, and proprioception across all dermatomes. Cortical sensations (tactile localization,

two-point discrimination, stereognosis, and graphesthesia) were entirely normal.

- **Coordination & Meningeal Signs:** Cerebellar signs (finger-to-nose, heel-to-shin) could not be reliably assessed due to severe motor weakness. Romberg's test could not be performed because the patient was unable to stand. There were no involuntary movements or signs of meningeal irritation (Kernig's and Brudzinski's signs were negative). Spine and cranium examination revealed no deformities or focal tenderness.

Other Systemic Examinations

- **Cardiovascular System:** Normal S1 and S2 heart sounds, regular rhythm, no murmurs, rubs, or gallops
- **Respiratory System:** Normal vesicular breath sounds bilaterally, clear lung fields, no added sounds (crepitations or wheezing).
- **Abdomen:** Soft, non-tender, no organomegaly (no hepatosplenomegaly), normal bowel sounds. External genitalia examination was normal.

Laboratory Investigations

Table 1: Hematological Trends (Complete Blood Count).

Parameter	17/12 (Day 1)	18/12 (Day 2)	19/12 (Day 3)	28/12 (Discharge)
Total Count (TC	8,100	17,000	13,700	4,300
Differential Count (%)	75 / 23\	78 / 18	75 / 21	4647
Red Blood Cells (RBC,	3.67	3.18	3.31	3.27
Hemoglobin (Hb,	11.7	10.1	10.2	10.6
PCV (%)	30.1	24.2	26.7	28.0
MCV	82.0	76.1	78.1	85.6
Platelets	310	345	330	316

Peripheral Smear: Normocytic normochromic RBCs, normal WBC count with relative lymphocytosis, adequate platelets, no atypical cells or hemoparasites. ESR was 20.

Table 2: Renal Function & Daily Electrolyte Monitoring.

Parameter	17/12	18/12	19/12
Urea	15.2	30.1	18.0
Creatinine	27.1	0.90	0.80
Sodium	138.0	140.0	137.0
Potassium	2.10	2.50	3.50
RBS	166	79	78

Table 3: Daily Liver Function Monitoring (LFT)

Parameter	17/12	18/12	19/12
Total Bilirubin	0.6	0.6	0.5
Indirect Bilirubin	0.3	0.3	0.3
Direct Bilirubin	0.3	0.3	0.2
SGOT / AST	55	44	56
SGPT / ALT	23	18	32
Alkaline Phosphatase	72	60	51
Total Protein	7.8	5.9	7.1
Albumin	3.8	3.5	3.1
Globulin	4.0	2.4	4.0

Table 4: Acid-Base & Blood Gas Analysis (ABG).

Parameter	17/12 (Day 1)	21/12 (Day 5)
pH	7.29	7.31
pCO_2	14.0	24.0
pO_2	134.0	108.0
Sodium	153.0	145.0
Potassium	1.3	2.8
Bicarbonate	6.7	12.1
Ionic Calcium	0.96	0.59

Interpretation: Severe, hyperchloremic non-anion gap metabolic acidosis with profound, life-threatening hypokalemia.

Specialized Urine and Serum Osmolality Studies

- **Urine Potassium (17/12):** 21.4 **Urine Sodium:** 81.0**Urine Albumin-to-Creatinine Ratio (ACR):** 843.75 (elevated).
- **Urine Osmolality:** 426mOsm/kg.
- **Serum Osmolality:** 289.75mOsm/kg.
- **Serum Calcium:** 9.9 (normal).
- **Thyroid Function Panel:**
  - T3**:** 0.49
  - T4**:** 6.89
  - TSH 1.12 (confirming euthyroidism on Levothyroxine replacement therapy).

Imaging and Electrophysiology

1. **Electrocardiogram (ECG)**
   - *Initial ECG (17/12/2023 at 10:30 AM):* Showed profound electrophysiological consequences of severe hypokalemia. Features included marked ST-segment depressions, flattened T-waves, and prominent, pathologic U-waves.
   - *Follow-up / Monitored Tracings:* Demonstrated PVC trigeminy (premature ventricular complexes occurring in a regular 3-beat cycle) and significant, high-risk prolongation of the corrected QT interval QTc - 577, posing an immediate threat of Torsades de Pointes or ventricular fibrillation.
2. **Chest Radiograph (20/12/2023):** AP view demonstrated clear lung fields, normal cardiothoracic ratio, and no evidence of active pulmonary tuberculosis, consolidation, or effusion.
3. **Abdominal & Pelvis Ultrasonography:** Normal study with no structural abnormalities of the kidneys, liver, or spleen. No nephrocalcinosis was observed.

Differential Diagnosis

Given the clinical triad of acute flaccid paralysis, severe hypokalemia, and metabolic acidosis, the differential diagnosis included.

1. **Secondary Hypokalemic Paralysis due to Distal Renal Tubular Acidosis (d-RTA):** Strongly suggested by the profound non-anion gap metabolic acidosis, renal potassium wasting, and female gender (frequently associated with autoimmune conditions like Sjögren's syndrome).
2. **Guillain-Barré Syndrome (GBS):** A primary consideration for any ascending AFP, but the sensory sparing, profound hypokalemia, and prompt resolution with potassium replacement make this highly unlikely.
3. **Familial Hypokalemic Periodic Paralysis:** Usually presents without severe metabolic acidosis.
4. **Thyrotoxic Periodic Paralysis:** Ruled out by normal TSH levels and lack of hyperthyroid clinical features.

Treatment

The primary focus of treatment was the urgent, cautious correction of the potassium deficit and metabolic acidosis, alongside continuous cardiac monitoring due to the presence of PVC trigeminy and prolonged QTc.

- Intravenous and oral potassium supplementation was initiated.
- Serial monitoring demonstrated a steady improvement in serum K levels from 2.1mEq/L to 3.5 mEq/L on day 3, reaching 4.1 mEq/L by day 4.
- Concurrently, the acid-base status improved, with a follow-up ABG showing a pH of 7.31 and HCO_3 rising to 12.1 mmol/L.
- As the severe electrolyte derangement was corrected, the patient's motor weakness dramatically improved, confirming the diagnosis of hypokalemic paralysis.

DISCUSSION

This presentation clinical course illustrates a classic "diagnostic trap" in emergency neurology and nephrology. Acute flaccid paralysis (AFP) with ascending quadriparesis and hyporeflexia in a young female is highly suggestive of Guillain-Barré Syndrome (GBS) or acute transverse myelitis. However, diagnosing a primary neurological disorder without evaluating basic serum chemistry can lead to catastrophic diagnostic error. In Ms. Petchiammal's case, checking her arterial blood gas (ABG) and serum electrolytes unmasked profound metabolic disturbances that were completely, rapidly reversible—saving her from unnecessary invasive procedures like lumbar punctures or expensive therapies like intravenous immunoglobulin (IVIG) and plasma exchange.

Pathophysiology of Hypokalemic Paralysis

Skeletal muscle contractility is fundamentally dependent on the maintaining of an appropriate concentration gradient of potassium ions across the sarcolemma. The resting membrane potential of skeletal myocytes is calculated via the Nernst equation.

where K e and K i represent extracellular and intracellular potassium concentrations, respectively. Under normal physiologic conditions, the membrane potential is roughly 90mV.

When the extracellular potassium concentration drops severely (falling to a life-threatening 1.3mEq/L on our patient's admission ABG), the ratio decreases dramatically. This hyperpolarizes the resting muscle membrane, driving much further from the activation threshold. Consequently, the muscle membrane becomes highly refractory to generating or propagating action potentials. This electrical inexcitability manifests clinically as acute flaccid paralysis, hypotonia, and a diminution of deep tendon reflexes.

The Acid-Base Crux: Distal Renal Tubular Acidosis (Type 1 d-RTA)

The pivotal diagnostic clue in this case was the coexistence of severe hypokalemia with a hyperchloremic, normal anion gap metabolic acidosis (NAGMA). In patients with severe hypokalemia, a normal physiologic response from the kidneys is to minimize potassium excretion However, Ms. Petchiammal's urinary potassium was inappropriately elevated at 21.4mEq/L at admission, rising further to 51.0mEq/L during active therapy. This established the diagnosis of renal potassium wasting.

When renal potassium wasting is coupled with a systemic NAGMA and an inappropriately alkaline urine (pH > 5.5 typical of d-RTA), the diagnostic algorithm points directly to Type 1 (distal) Renal Tubular Acidosis (d-RTA).

Pathophysiologically, d-RTA is caused by a severe secretory defect in the α-intercalated cells of the cortical collecting duct. These). When this mechanism fails.

1. **Inability to Excrete Acid:** Hydrogen ions accumulate systemically, dropping serum pH (7.29) and exhausting bicarbonate reserves HCO_3 down to 6.7mmol/L).
2. **Failure of Urine Acidification:** The distal nephron cannot acidify the urine, causing urine pH to remain persistently high (> 5.5) despite severe systemic acidemia.
3. **Active Renal Potassium Wasting**

- Systemic metabolic acidosis reduces sodium and water reabsorption in the proximal convoluted tubule, resulting in increased delivery of sodium and water to the distal nephron.
- This distal sodium delivery, combined with mild volume contraction from renal sodium wasting, activates the renin-angiotensin-aldosterone system (RAAS), resulting in secondary hyperaldosteronism.

- Aldosterone stimulates the epithelial sodium channels (ENaC) on the principal cells of the collecting duct. The rapid reabsorption of sodium creates an electrogenic lumen-negative potential.
- Because the neighboring-intercalated cells cannot secrete positive hydrogen ions (to balance this negative charge, the lumen electronegativity is neutralized by massive, passive dumping of potassium ions into the urine through apical ROMK channels, creating profound, hypokalemia-induced paralysis.

The Autoimmune Connection: Sjögren's Syndrome

Acquired d-RTA in young adult females is strongly associated with underlying autoimmune disorders. The most common etiology is primary Sjögren's syndrome (pSS), followed by systemic lupus erythematosus and rheumatoid arthritis.

In this patient, several clinical details point strongly toward Sjögren's syndrome as the underlying cause.

1. **Symptom Linkage:** She described a chronic, burning sensation of her mouth, which is a classic symptom of xerostomia (dry mouth) resulting from autoimmune lymphocytic destruction of the salivary glands.
2. **Comorbid Autoimmunity:** She was diagnosed with primary hypothyroidism just 1 month prior. Autoimmune thyroiditis (Hashimoto's) is known to cluster frequently with primary Sjögren's syndrome under the umbrella of multiple autoimmune syndromes.
3. **Renal Pathology:** In primary Sjögren's syndrome, lymphocytic infiltration of the renal tubulointerstitium occurs (interstitial nephritis). This active lymphocytic invasion of the distal tubules selectively downregulates or completely destroys the apical membrane ATPase pump in the intercalated cells, or autoantibodies directly inhibit carbonic anhydrase II. This autoimmune-mediated destruction selectively disables the kidney's acid-base handling, causing acquired d-RTA.

CONCLUSION

This case brilliantly illustrates the axiom that "not all acute flaccid paralysis is GBS." A basic metabolic panel, including electrolytes and an arterial blood gas, is mandatory in the initial workup of any patient presenting with acute weakness. Early identification of hypokalemic paralysis allows for immediate, life-saving potassium replacement, preventing fatal arrhythmias and respiratory failure, and guides the physician toward diagnosing underlying systemic conditions like Renal Tubular Acidosis.

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