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ACUTE FLACCID PARALYSIS: GUILLAIN-BARRE SYNDROME MIMICS

Neurology

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KEYWORDS

INTRODUCTION

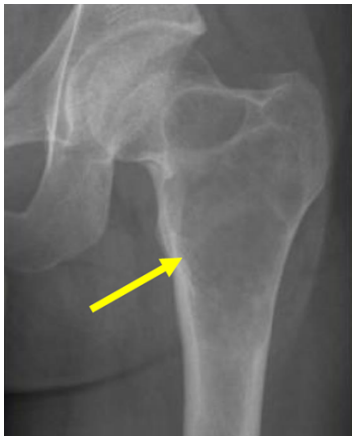
- Guillain Barrésyndrome (GBS) has emerged as one of the commonest cause of acute flaccid paralysis worldwide However, clinicians should not miss the other causes of acute flaccid paralysis which may mimic GBS. Herein, we report 3cases of flaccid are flexic paralysis which presented acute / sub acutely resembling GBS.

Case 1

- A 29- year old lady presented with 2 week history of bilateral lowerlimb weakness with history of left hip pain for the past 3years. On Examination: she was generally are flexic with paraparesis. NCS showed evidence of peripheral neuropathy with mixed sensori motor axonal pattern. CSF analysis revealed evidence of cyto-albuminergic dissociation (protein2.46g/L). Blood investigations as follows:

- **Calcium** **2.6 mmol/L** ↑
- **Phosphate** **0.3 mmol/L** ↓
- **Alkaline phosphatase** **1036 u/L** ↑
- **Vitamin D** **14 nmol/L** ↓
- **Parathyroid hormone** **149 pmol/L** ↑

She had plasma exchange for presumed GBS, but her lower limbs weakness worsened and progressed to involve upper limbs and facial muscles. Ultrasonography revealed right parathyroid adenoma. Her weakness improved after parathyroidectomy and the diagnosis was revised to **hyperparathyroidism- related polyneuropathy.**



Case 2

- A 28- year old lady with under lying epilepsy and intellectual disability presented with progressive weakness of extremities over 3 weeks after prolonged fasting. Examination revealed she was generally are flexic with tetra paresis. NCS show edevidence of neuropathy with mixed sensorimotor axonal pattern. CSF analysis was negative for cyto albuminergic dissociation. Her weakness did not improve after treatment with immunoglobulin for GBS. Blood investigations showed the following results.

- **Urine porphobilinogen** **Positive**
- **Urine delta-aminolaevulinic acid (ALA)** **3.4 µmol/L** ↑

A diagnosis of **acute intermittent porphyria** was considered most likely and her weakness improved after continuous glucose loading.

Case 3

- A 52- year old obese lady who had bariatric surgery presented with bilateral lowerlimb weakness over 4 weeks after recurrent bouts of vomiting and excessive weightloss (35kg). On examination, reflexes were absent with paresis of both lower limbs. NCS showed mixed sensor motor axonal polyneuropathy. CSF analysis was negative for cyto albuminergic dissociation. Blood investigations showed the following results.

- **Vitamin D** **38 nmol/L** ↓
- **Thiamine** **87 nmol/L** ↓ **(low normal)**
- **Albumin** **28 g/L** ↓
- **Vitamin B12** **872 pmol/L** ↔
- **Homocysteine** **17.2 µmol/L** ↑
- **Folate** **Low** ↓

As her weakness continued to progress, a diagnosis of **acute nutritional neuropathy** was considered more likely. Her weakness improved following multivitamins replacement.

DISCUSSION

1. Case 1: Acute flaccid paralysis as a result of hypophosphataemia were secondary to hyperparathyroidism, hyperalimentation, chronic renal failure and chronic alcoholism. However, the mechanism underlies hyperparathyroidism - related polyneuropathy was unclear.
2. Case 2: The deficiency of enzyme porphobilinogen deaminase (PBGD) in the heme pathway causes the over production of heme precursors (porphobilinogen and delta ALA). These precursors were neurotoxic hence resulted in acute axonal polyneuropathy.
3. Case 3: Acute and subacute axonal neuropathies could occur in the setting of chronic alcoholism, anorexia and post- bariatric surgery. In this case, the borderline serum thiamine level may not reflect the true thiamine level. Serum RBC transketolase is more reliable in detection of thiamine deficiency.

CONCLUSION

- Clinicians should have high index of suspicion when t reating acute flaccid paralysis with progressive disease and null response to immunotherapy as the neurology is reversible with treatment of the underlying cause.

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