Abstract

The author’s experiences with cases of periodic paralysis (PP) between 1975 and 2001, with review of literature regarding incidence, type, diagnosis, and treatment have been presented.

Hypokalaemic, hyperkalaemic, and normokalaemic periodic paralysis characterised by episodes of short-lived paralysis of skeletal muscles could be idiopathic (primary or familial periodic paralysis) or due to identifiable causes (secondary periodic paralysis). Serum level of potassium is abnormal during attacks in all types of periodic paralysis except in some cases of normokalaemic periodic paralysis. Both these groups of diseases are eminently treatable. Acetazolamide prevents paralytic attacks in both hypokalaemic and hyperkalaemic periodic paralysis. Oral potassium chloride, potassium – sparing diuretics, and dichlorphenamide prevent attacks only in hypokalaemic periodic paralysis. Thiazide diuretics prevent paralysis only in hyperkalaemic periodic paralysis. Oral or i.v. potassium chloride are used to relieve muscle weakness in hypokalaemic periodic paralysis. Thiazides, loop diuretics, i.v. calcium gluconate, i.v. glucose with insulin, and in refractory cases, i.v. normal saline relieve attack of hyperkalaemic periodic paralysis. Specific treatment for underlying disorders in secondary periodic paralysis must be instituted.

Key words

Serum potassium, Channelopathy, Gene mutation. (Thr. 704M, SCN 4A), Acetazolamide, Potassium chloride, Glucose-insulin and normal saline.

Introduction

Periodic paralysis (PP) is a group of disorders of different aetiologies, with episodic, short-lived, and hypo-reflexic skeletal muscle weakness, with or without myotonia but without sensory deficit and without loss of consciousness. Early in the course of disease, in primary or familial periodic paralysis, muscle strength is normal in-between the attacks. After many years of these attacks, inter-ictal weakness develops and may be progressive. These disorders are amenable to treatment and progressive weakness can be prevented or even reversed. This review is intended to focus on the classification, pathophysiology, clinical features, differential diagnosis, investigation, and treatment of this relatively rare but eminently treatable group of disorders.

Classification

These disorders have been conventionally divided into primary or familial periodic paralysis, and secondary periodic paralysis.

Primary or familial periodic paralysis is a group of disorders due to single gene mutation resulting in abnormalities of calcium, sodium, potassium, and chloride channels on muscle cell membrane. Hence, they are also known as channelopathies or membranopathies.

Secondary periodic paralysis may be due to demonstrably known causes (see Table I). In secondary periodic paralysis, even the inter-ictal level of potassium in serum is abnormal. History of use of ACE inhibitors, angiotensin-II-receptor–blockers, diuretics, or carbenoxolone (liquorice) gives a clue to diagnosis of secondary periodic paralysis. Clinical and/or biochemical features of chronic renal failure, thyrotoxicosis, paramyotonia congenita, or Andersen’s syndrome may be found in secondary periodic paralysis.

The serum potassium levels in both primary and secondary periodic paralysis are usually abnormal during attacks. If the level is below normal, the periodic paralysis is known as hypokalaemic type. If the level is higher than normal, it is termed as hyperkalaemic type. In normokalaemic type of
periodic paralysis, the serum potassium during attack may be normal or in lower or upper range of normal. In primary periodic paralysis, inter-ictal serum potassium is normal. In secondary periodic paralysis, the inter-ictal serum potassium may be abnormal.

Table I: Conventional classification of periodic paralysis.

Primary or familial periodic paralysis:
1. Hypokalaemic periodic paralysis
2. Hyperkalaemic periodic paralysis
All have autosomal dominant inheritance.

Secondary periodic paralysis:
1. Hypokalaemic periodic paralysis.
   a. Thyrotoxicosis
   b. Thiazide or loop-diuretic induced
   c. Potassium losing nephropathy
   d. Drug-induced: gentamicin, carbenicillin, amphotericin-B, degraded tetracyclines, vitamin B12, alcohol, carbonoxolone
   e. Primary or secondary hyperaldosteronism
   f. Acute human toxicity due to ingestion of barium carbonate as rodenticide
   g. Gastro-intestinal potassium loss
2. Hyperkalaemic periodic paralysis:
   a. Chronic renal failure
   b. High dose of ACE-inhibitor therapy, chronic renal failure or advanced diabetic nephropathy
   c. Potassium supplements if used with potassium-sparing diuretics (spironolactone, triamterene, amiloride) and/or ACE-inhibitors.
   d. Andersen's cardiomyopathy
      – Usually with hyperkalaemia, but occasionally with hypokalaemia or normokalaemia.
      – Associated with cardiac dysrhythmia and dysmorphic features (hypertelorism, low set ears, broad nose)
   e. Paramyotonia congenita-periodic paralysis occurs spontaneously or is precipitated by cold exposure
3. Potassium-aggravated myotonia

Increasing knowledge of molecular biology, genetics, and electrophysiology has resulted in improved understanding of disease mechanisms that underlie these neurological channelopathies. Recently, a new classification of primary periodic paralysis has been proposed. This is based on the specific abnormalities in ionic channels (calcium, sodium, chloride, and potassium channels) over the muscle cell-membrane (see table II).

Table II: Classification of primary periodic paralysis based on ion-channel abnormalities

1. Calcium channel disorders of muscle
   – Hypokalaemic periodic paralysis
2. Sodium channel disorders of muscle
   – Hyperkalaemic periodic paralysis
   – Paramyotonia congenita
   – Potassium-aggravated myotonia
   – Some cases of hypokalaemic periodic paralysis
3. Chloride channel disorders of muscle
   – Myotonia congenita
4. Disorders of potassium channel sub-unit
   – Some cases of hypokalaemic periodic paralysis
   – Some cases of hyperkalaemic periodic paralysis
   – Andersen's syndrome
5. Disorders of unknown pathogenic mechanism
   – Thyrotoxic periodic paralysis (perhaps a decrease in the activity of calcium pump).

Genetic factors in pathophysiology of familial or primary periodic paralysis (PPP)

Genetic mutation has been postulated as a basic cause of primary periodic paralysis. Mutation of Na+ channel gene at locus Thr. 704M has been detected in patients of hyperkalaemic periodic paralysis and of normokalaemic periodic paralysis. On this basis, normokalaemic periodic paralysis has been considered as a variant of hyperkalaemic periodic paralysis.

Twenty point mutations have been identified in the gene encoding a sub-unit of skeletal muscle sodium channel (SCN 4A) in patients with primary hyperkalaemic periodic paralysis and myotonic disorder. It is now believed that primary hyperkalaemic periodic paralysis is an autosomal
dominant skeletal muscle disorder caused by single mutation in the SCN4A gene, encoding human skeletal muscle voltage-gated Na+ channel. Bendahhou et al have identified one allele with two novel mutations occurring simultaneously in the SCN4A gene. These mutations have been found in two families of patients of hyperkalaemic primary periodic paralysis6-8. Hypokalaemic primary periodic paralysis is caused by mutation in a voltage-sensitive skeletal muscle calcium channel, although details of pathogenesis are incompletely understood1,3. Genetic studies have revealed mutations in the voltage-gated calcium channel alpha-1 subunit in families of hypokalaemic primary periodic paralysis. Electrophysiological studies on these mutants in different expression systems could not explain the pathophysiology of the disease6. In addition, several mutations (Arg 669 His, Arg 672 His, Arg 672 Gly and Arg 672 – Ser) in the voltage sensor of skeletal muscle sodium channel – alpha subunit (SCN4A gene) have been found in families with hypokalaemic periodic paralysis. The gating of both histidine mutants (Arg 669 His and Arg 672 His) can be modulated by changes of extra- or intra-cellular pH. The inactivation defects of Arg 669 His and Arg 672 His can be alleviated by low pH to a significant degree, suggesting that the decrease of pH in muscle cells (e.g., during muscle work) might lead to auto – compensation of functional defects. This may explain a delay or prevention of paralytic attacks by slight physical activity. Moreover, the histidine residues may be the targets for a potential therapeutic action by acetazolamide9. Phenotypic variation of Thr.704Met mutation, which was previously reported in patients with hyperkalaemic primary periodic paralysis, has been described in a family affected with paralysis periodica paramyotonica5. The inheritance is usually autosomal dominant, but in hypokalaemic type, 30% cases may be sporadic2. The pathophysiology of thyrotoxic periodic paralysis involves increased activity of Na+–K+–ATPase system. Thyroxine increases beta – adrenergic activity leading to augmented Na+–K+–ATPase activity10,11. Thyrotoxic periodic paralysis has autosomal dominant inheritance6.

Incidence

The periodic paralysis is a relatively rare disease in clinical practice. Between 1972-2001, the author has come across 12 cases of primary periodic paralysis and 27 cases of secondary periodic paralysis. Ten cases of primary periodic paralysis were of hypokalaemic type, one of hyperkalaemic type, and one of normokalaemic type. Eight cases of primary hypokalaemic periodic paralysis were males (between 14 to 45 years) and two were females (between 18 to 27 years). One case of normokalaemic primary periodic paralysis was a male of 13 years. One patient of hyperkalaemic primary periodic paralysis was a 20 years old female. In second order periodic paralysis, 12 were of hypokalaemic type and 15 were of hyperkalaemic type. Of these, 15 were males in the age group 16 to 62 years and 12 were females between 18 to 64 years. Mean age of ten male patients of primary hypokalaemic periodic paralysis was 22.87 years and mean age of two female patients of primary hypokalaemic periodic paralysis was 22.50 years. The values of two mean ages were not significantly different because the p-value is 0.96 of two-sample-t-test. Mean age of eight male patients of secondary hypokalaemic periodic paralysis was 45 years and mean age of four female patients of secondary hypokalaemic periodic paralysis was 31.75 years. The two-sample-means were not significantly different because the two-sample-t-test yielded the p-value of 0.27. Mean age of seven male patients of secondary hyperkalaemic periodic paralysis was 28.12 years. The two sample-t-test suggested that the two were not significantly different because the p-value was 0.80 for the test. In the secondary hypokalaemic periodic paralysis, evident causes were acute gastroenteritis, thyrotoxicosis, and heavy doses of thiazide or loop-diuretics. In the secondary periodic paralysis of hyperkalaemic type, the basic causes were acute tubular necrosis (due to septic
abortion, post-partum haemorrhage, and cholera), acute oliguric renal failure (due to acute glomerulonephritis), and chronic renal failure (chronic pyelonephritis and diabetic nephropathy).

Dhall et al. reported 14 cases of hypokalaemic periodic paralysis, of which 5 were of primary type, 4 were due to thyrotoxicosis, and 5 were due to gastrointestinal potassium loss. Agrawal et al. in 1993 reported 6 cases of hypokalaemic periodic paralysis, 2 of sporadic primary variety and 4 of secondary hypokalaemic periodic paralysis (2 due to thyrotoxicosis; 1 due to diarrhoea and vomiting, and 1 due to barium salt ingestion). Ram Krishnan et al. in 1993 reported 6 cases of hypokalaemic periodic paralysis, 2 of sporadic primary variety and 4 of secondary hypokalaemic periodic paralysis (2 due to thyrotoxicosis; 1 due to diarrhoea and vomiting, and 1 due to barium salt ingestion). Khuller et al. reported one atypical case of hypokalaemic periodic paralysis. Agrawal et al. have also described 3 cases of thyrotoxic hypokalaemic periodic paralysis in 1994.

Clinical approach to a case of periodic paralysis

Meticulous history, complete bedside clinical examination, simple laboratory investigations, ECG, and EMG will establish the diagnosis in most of the cases. A few patients may also require muscle biopsy.

History

a) History of muscle weakness: History of episodic short-lived paralysis of one, two, or all four limbs, without loss of consciousness or sphincter dysfunction is a strong pointer to the diagnosis of periodic paralysis. Weakness may start proximally and then spread distally. There may be history of localised paralysis in one limb or two, but later on other limbs are involved. Paralysis may last for 1 to 24 hours or several days and can occur at varying frequency ranging from daily to yearly. However, in few cases, the presentation may be with progressive and permanent muscular weakness even without a past history of repeated muscular paralysis at younger age.

In rare cases, with severe disease, respiratory muscles and muscles supplied by cranial nerves may be involved. Death may occur in these cases if not recognised and treated promptly.

b) Age: Age of onset is early in childhood in hyperkalaemic primary periodic paralysis, and paramyotonia congenita. Age of onset soon after puberty but earlier than 25-30 yrs suggests primary hypokalaemic periodic paralysis. Onset after age of 25 years nearly always points to secondary periodic paralysis.

c) Family history: There is usually a strong family history in primary periodic paralysis, though 33% cases of hypokalaemic primary periodic paralysis may be sporadic. In primary hypokalaemic periodic paralysis, if the children of patients did not suffer from attacks, the history of paralytic attacks in grand children should be elicited.

d) Timing: Periodic paralysis occurs typically on waking from sleep or on rest after exercise. It never occurs in the midst of vigorous exercise. This differentiates it from myasthenia gravis.

e) Intensity: Many patients in this series gave history of two kinds of attacks – mild and severe. During mild attacks, there was a feeling of tiredness and fatigue of muscles that usually disappeared in an hour. In severe attacks, the patients reported complete immobility.

f) History of administration of certain drugs: Patients should be questioned about administration of diuretics, ACE-inhibitors, angiotensin–receptor–blockers, carbenoxolone–sodium, gentamicin, carbenicillin, etc. History of gastroenteritis, oliguria or anuria, severe post-partum haemorrhage, or septic abortion (in case of females) should be elicited.

Clinical examination

In author’s experience most patients present during the inter-ictal period, and there is no positive physical finding in the primary periodic
paralysis. In secondary periodic paralysis, features of causative disorders like thyrotoxicosis, chronic renal failure, diabetic nephropathy, acute glomerulonephritis, or acute tubular necrosis may be present. In thyrotoxic periodic paralysis, the initial attack of periodic paralysis may occur before, during, or soon after the diagnosis of thyrotoxicosis.

In few patients, who are brought to the clinician during the attack, the weak muscles are flaccid and the tendon jerks are absent and Babinski’s sign is negative. There is no cloudiness of sensorium. No sensory deficit is present. Myotonia can be elicited in few cases of paramyotonia congenita with hyperkalaemic periodic paralysis. Myotonia may be marked in eyelids in the hyperkalaemic type.

Laboratory investigations\textsuperscript{1,2,11,15-17}

Serum potassium is, essentially, the most important laboratory investigation. In-between the attacks of paralysis, serum potassium is abnormal in secondary type of periodic paralysis, but is usually normal in primary periodic paralysis. During attack, serum potassium level may be high, low, or in upper or lower range of normal. Abnormal serum potassium level in absence of any other obvious cause in a patient with history of episodic short-lived paralysis of skeletal muscle is almost diagnostic of periodic paralysis. Random testing for serum potassium level may show periodic fluctuation in normokalaemic periodic paralysis.

Urinalysis, blood sugar, blood urea, serum creatinine, free-T\textsubscript{3}, free-T\textsubscript{4} and TSH (IRMA) should be ordered to exclude diabetic nephropathy, chronic or acute renal failure, and thyrotoxicosis respectively. In thyrotoxic periodic paralysis, the TSH level may be very low and only occasionally the T\textsubscript{3} and T\textsubscript{4} are high. Also inorganic phosphorus and magnesium are low in secondary hypokalaemic periodic paralysis\textsuperscript{17}.

CPK and serum myoglobin levels: Serum CPK is high in primary periodic paralysis during or just after attack. Serum myoglobin may be high.

ECG should always be carried out to corroborate the serum levels of potassium. In Andersen's cardio-dysrhythmic periodic paralysis, the ECG and Holter monitor reveal cardiac dysrhythmia.

EMG: In between attacks, there may be fibrillation and complex repetitive discharges, increased by cold and decreased by exercise (in hypokalaemic periodic paralysis). During attacks, EMG will show electrical silence, in both hyper- and hypokalaemic periodic paralysis.

Muscle biopsy is required in few cases with atypical presentation. In primary hypokalaemic periodic paralysis, there may be single or multiple centrally placed vacuoles. In primary hyperkalaemic periodic paralysis, vacuoles and tubular aggregates are found.

Provocative tests

These are done for diagnosis if patient presents in-between the attacks of paralysis and should be done in a hospital setting.

Oral glucose 5 gm/kg or i.v. glucose 3 gm/kg over one hour is administered. ECG monitoring is essential and serum potassium level is measured every 15 – 30 minutes and followed up to 12 hours after glucose administration. If the typical attack is precipitated, it nearly confirms hypokalaemic periodic paralysis. In this series, author does not prefer I.V. glucose with insulin as provocative test.

Oral potassium chloride 1 mEq to 2 mEq/kg is administered. If the attack of paralysis is provoked in 1.5 to 3 hours, it clinches the diagnosis of hyperkalaemic periodic paralysis or normokalaemic periodic paralysis. I.V. potassium loading test (potassium chloride 0.05 to 0.15 gm/kg) induces weakness in such cases\textsuperscript{1}, but the author prefers the oral test.

Ice-cold water test: A towel soaked in ice-cold water is put on the closed eyes for a minute or so. Patient is then asked to look up for two seconds and then to look down. The sclera can be seen above cornea (lid lag) in case of hyperkalaemic or normokalaemic periodic paralysis.
Epinephrine, nor-epinephrine, or corticosteroids parenterally may provoke an attack in hypokalaemic periodic paralysis.

Therapeutic tests: If i.v. frusemide or oral thiazide or i.v. normal-saline or i.v. 10 ml 20% calcium gluconate or i.m./s.c. salbutamol terminate the attack, and serum-\(K^+\) is within normal or upper or lower limit of normal, diagnosis of normokalaemic periodic paralysis can be considered established.

Another useful test DNA-analysis of appropriate gene is, however not yet available in our country.

Differential diagnosis

The differentiating features of various types of primary periodic paralysis are given in table III.

Other muscle diseases with ion-channel disorders (see table IV) can hardly be confused with primary periodic paralysis as episodic paralysis is absent and myotonia is a marked feature in the former.

**TREATMENT**

The treatment will vary according to the type of periodic paralysis – whether hypokalaemic, hyperkalaemic, or normokalaemic:

**Treatment of hyperkalaemic periodic paralysis**

1. Prophylaxis to prevent future attack

   Acetazolamide (Diamox tablet 250 mg) – \(\frac{1}{2}\) to 4 tablets per day orally

   Potassium–sparing diuretics, e.g., triamterene 25 to 100 mg OD or spironolactone 100 mg OD or amiloride 5 mg OD.

   Dichlorphenamide (DCP) another carbonic

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**Table III: Distinctive features of various types of primary periodic paralysis.**

<table>
<thead>
<tr>
<th></th>
<th>Hypokalaemic periodic paralysis</th>
<th>Hyperkalaemic periodic paralysis</th>
<th>Paramyotonia congenita</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age of onset</strong></td>
<td>Second decade</td>
<td>First decade</td>
<td>Early childhood</td>
</tr>
<tr>
<td><strong>Mode of inheritance</strong></td>
<td>Autosomal dominant</td>
<td>Autosomal dominant</td>
<td>Autosomal dominant</td>
</tr>
<tr>
<td><strong>Precipitating factors</strong></td>
<td>Rich carbohydrate meal, alcohol, rest after exercise</td>
<td>Rest after exercise, cold exposure</td>
<td>Spontaneously or after exposure to cold</td>
</tr>
<tr>
<td><strong>Episodic weakness</strong></td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Timing of attack</strong></td>
<td>Early morning, night</td>
<td>Day</td>
<td>Anytime</td>
</tr>
<tr>
<td><strong>Duration of attacks</strong></td>
<td>Hours to days (usually 2-12 hours)</td>
<td>Minutes to hours (usually 1-2 hours)</td>
<td>2-24 hours</td>
</tr>
<tr>
<td><strong>Severity of attacks</strong></td>
<td>Moderate to severe weakness</td>
<td>Mild to moderate weakness, may be local</td>
<td>Very mild</td>
</tr>
<tr>
<td><strong>Myotonia</strong></td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Effect of muscle cooling</strong></td>
<td>No change</td>
<td>Increased myotonia</td>
<td>Increased myotonia, then weakness</td>
</tr>
<tr>
<td><strong>Serum potassium during attack</strong></td>
<td>Low</td>
<td>Usually high, may be normal</td>
<td>Usually normal</td>
</tr>
<tr>
<td><strong>M: F ratio</strong></td>
<td>3 : 1</td>
<td>1 : 1</td>
<td>No sex predilection</td>
</tr>
<tr>
<td><strong>EMG during attack</strong></td>
<td>Electrical silence</td>
<td>Electrical silence</td>
<td>Myotonic discharges, hyper-irritability and after discharge. Weakened muscles show a dropping out of some motor unit potentials and reduced voltage and duration of others.</td>
</tr>
<tr>
<td><strong>Inter-ictal EMG</strong></td>
<td>Fibrillation and complex repetitive discharge increased by cold and decreased by exercise.</td>
<td>Myotonic discharges in some.</td>
<td></td>
</tr>
<tr>
<td><strong>Muscle biopsy</strong></td>
<td>Presence of single or centrally placed vacuoles</td>
<td>Vacuoles and tubular aggregate</td>
<td>No change or at most a few vacuoles</td>
</tr>
<tr>
<td><strong>Ionic channel disorder</strong></td>
<td>Calcium channel</td>
<td>Sodium channel</td>
<td>Sodium channel</td>
</tr>
</tbody>
</table>

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anhydrase inhibitor, has recently been advocated\textsuperscript{1, 11, 18}.

Side effects of acetazolamide are tingling sensation, hypersensitivity, and in long-term use renal stone formation. This is not so frequent with DCP. Caution should be exercised in pregnancy and breast feeding. It is contraindicated in COAD as acidosis precipitates respiratory failure.

Diuretics may be used to prevent an attack.

2. Treatment of an attack: Mild cases do not require drug therapy as these are brief and merely taking sweet drinks or sugar candies relieve the attack. In fact many patients volunteer this history that such foods abort their attacks. In more prolonged or severe attacks, thiazide diuretics and loop-diuretics (Frusemide, Bumetanide, etc.) are used in doses high enough to reduce serum potassium to normal levels. If the serum potassium level is very high, I.V. 20 ml of 20% calcium gluconate or I.V. drip of normal saline, or I.V. 10% glucose + insulin should be given. In case of failure or intolerance to diuretics, salbutamol may be tried by i.v. route for terminating an attack\textsuperscript{7}.

Table IV: Other muscle disorders due to ion-channel disorders.

<table>
<thead>
<tr>
<th>Age of onset</th>
<th>Sodium channel disorder (Sodium channel myotonia)</th>
<th>Chloride channel disorder (Congenital myotonia)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Episodic paralysis</td>
<td>Early childhood</td>
<td>Early childhood</td>
</tr>
<tr>
<td>Mode of inheritance</td>
<td>No</td>
<td>Autosomal dominant or autosomal recessive</td>
</tr>
<tr>
<td>Myotonia</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Effect of muscle cooling</td>
<td>Mild increase in myotonia</td>
<td>Increase in myotonia</td>
</tr>
</tbody>
</table>

2. Treatment of an attack\textsuperscript{1, 11}

Oral potassium chloride 0.2 to 0.4 mmol/kg every 30 minutes till paralysis recovers (1 mmol = 75 mg KCl) or 5-10 gm oral potassium chloride is given and repeated in 1 hour, provided renal function is normal.

If oral potassium chloride is not effective, then I.V. infusion of potassium chloride 0.1 mmol/kg in 10% mannitol (500 ml) is administered. (1 amp of potassium chloride contains 150 mg or 2 mmol). This I.V. administration is contraindicated in renal failure. Potassium chloride should not be given mixed with 5% glucose or normal saline as it will aggravate hypokalaemia and worsen the paralysis.

In an occasional patient, attack not responding to or worsened by acetazolamide, triamterene 25 to 100 mg/day or spironolactone 25 to 100 mg/day may be beneficial\textsuperscript{1}.

Treatment for normokalaemic periodic paralysis

The treatment is similar to hyperkalaemic periodic paralysis, e.g.

i) High carbohydrate-diet, e.g., sugar candies may be enough.

ii) Thiazides, e.g., chlorothalidone 250-1,000 mg/day may be needed.

iii) I.V. normal saline and calcium-gluconate.

iv) I.V. insulin and glucose.

Treatment of secondary periodic paralysis

Basic principles

Primary cause should be treated.
Offending drug should be stopped.
Potassium supplements should be given in hypokalaemic periodic paralysis. Loop diuretics, i.v. glucose+insulin, or calcium gluconate should be administered in hyperkalaemic periodic paralysis.

(a) Periodic paralysis due to thyrotoxicosis: As in this disorder there is hypokalaemia, treatment is done by administration of potassium chloride along with β-blockers and carbimazole (Neomercazol). Acetazolamide is not effective. In emergency situation, i.v. propranolol can be used19.

(b) Periodic paralysis due to acute barium toxicity: Treatment comprises of magnesium sulphate solution 2.5 gm i.v. in single bolus. In early cases gastric lavage with magnesium sulphate (2.5%) may be given. Ventilatory support may be needed. Of course, hypokalaemia has to be treated in the usual way by i.v. potassium chloride. Sodium sulphate may be used in place of magnesium sulphate11.

(c) Periodic paralysis due to paramyotonia congenita: There is usually hyperkalaemia (raised serum K+) and paralysis is precipitated by cold. Hence, the patient should be nursed in a warm room. Treatment consists of oral or I.V. glucose and oral thiazides.

(d) Andersen's syndrome1,20: Patient has to be admitted in ICU for cardiac monitoring and prompt treatment of cardiac dysrhythmias. As serum K+ level may be low, raised or normal treatment for hypokalaemia or hyperkalaemic is applied according to level of serum potassium1.

Treatment of associated disorders

Certain other conditions may occur in association with primary periodic paralysis and these should be treated. Daytime sleepiness21, (perhaps as a direct result of hyperkalaemia), fibromyalgia22, and malignant hyperthermia23 may be associated with some cases of hyperkalaemic periodic paralysis. Hyperinsulinaemia17 and rarely TSH-secreting pituitary tumour24 have been described in some cases of primary hypokalaemic periodic paralysis. All these associated conditions need appropriate specific treatment along with correction of hyper-or hypokalaemic states.

References


