Acute Movement Disorders

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Movement disorder is broadly categorised into two groups - hypokinesia (slowness or poverty of movement) which includes Parkinsons disease and related disorders and hyperkinesia (abnormal involuntary movement) which includes tremor, dystonia, athetosis, chorea, ballism, tics, myoclonus, akathisias and other dyskinesias. Most movement disorder reflects degenerative disorder which are gradual in onset and slowly progressive. However, these are some movement disorder which presents acutely. This article gives an overview of such movement disorders.

The movement disorder with acute onset can be broadly categorized into three groups:

A) Drug induced movement disorder
B) Paroxysmal dyskinesias
C) Episodic ataxias

A) Acute drug induced movement disorder

Large number of drugs cause movement disorder as their adverse reactions. The most common ones are dopamine receptor blockers, the antipsychotics (neuroleptics) and antiemetics. They can cause gradual onset movement disorder however can also cause abrupt onset acute dystonic reactions, akathisia and neuroleptic malignant syndrome.

1. Acute dystonic reactions:

This is one of the relative common drug induced disorder seen in 2-3% of patients on neuroleptics. With more severe neuroleptics this could be seen over 50% cases. Young people are particularly with more risk. Cranial, pharyngeal, cervical and axial muscles are more commonly involved and can produce oculogyric crisis, grimacing, jaw clinching, retrocollis, torticollis or opisthotonic posturing. Limbs are less commonly affected. All neuroleptics can produce this reaction. Atypical neuroleptics like resperidone, clozapine, olanzapine and quetiapine cause less risk. The risk is dose related and half of the cases occur within first 2 days of exposure and 90% within first 4 days. There can be diurnal variation with 80% occurring during evening. If untreated this may last for hours to days.

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The pathogenesis is not understood. However dopamine receptor blockade is most likely cause.

Treatment is remarkably affective. IV cholinergics like benztropine gives good recovery. Dose is 2 mg IV with 1.2mg orally twice daily for 1-2 weeks to prevent recurrence. Other drugs which can help are benzodizepine.

2) Akathisia

It is one of the common form of drug induced movement disorder. Subjectively patient suffer from a feeling of restlessness and inability to remain still. Objectively patient show increased motor activity, consisting of complex, semi purposeful, stereotypic and repetitive movements to calm down their urge to move. Acute akathisia develop in about 20 to 40% patients receiving dopamine receptor blocker. In 75% patients acute akathisia develops within 3 days after therapy initiation. There is also relationship between potency of neuroleptics, their dose and rate of dose increase and severity of akathisia. Acute acathisia may also be caused by serotonergic agents, SSRI or cocaine. Acute acathisia tends to continue as long as neuroleptic medication is maintained. Clozapine is relatively safe drug for psychosis without inducing akathisia. Anticholinergics, amantadine clonazepam and clonidine may reduce acute acathisia.

3) Neuroleptic malignant syndrome (NMS)

NMS is rare but life threatening adverse reaction of neuroleptics. It may occur in 0.2% of all patients exposed to neuroleptics. Potent neuroleptics like haloperidol or fluphenazine cause NMS more frequently but all antipsychotics including atypical ones may precipitate it. Rarely NMS can occur with metoclopramide, amoxapine and lithium. NMS can be triggered by rapid dopamine agonist withdrawal. NMS occurrence is highly unpredictable. It may occur at any time during neuroleptic treatment. It may occur even when previous neuroleptic exposures were tolerated. Likewise re-challenge may not trigger it again suggesting neuroleptic exposure alone may not be causative. Additional causative factor include increase ambient temperature, dehydration, catatonia, hyponatremia, exertion as well as pre existing brain disease. Younger patient especially men, seem to have higher risk of NMS.

NMS is believed to be due to D2-receptor mediated dopaminergic dysfunction in the hypothalamus and the striatum.

NMS presents with hyperthermia, rigidity , bradykinesia, reduced consciousness, delusions, agitations and autonomic dysfunction like tachycardia, tachypnea, diaphoresis. They may occasionally develop dystonia, chorea, myoclonus, seizure, ataxia and ocular fluttur. Elevated CPK and urinary myoglobin support diagnosis of NMS but are non specific.

Management of NMS depends on early intervention. If there is suspicion of NMS immediate withdrawal of the offending agent is the most important step in management of NMS. Intensive correction of fluid, metabolic imbalance and hyperthermia management is required. Drugs used are dantrolene and bromocriptine. Dantroline is given in dose of 10 mg/body weight/day bid or tid and bromocriptine is given in dose of 2.5 to 5mg tid and increased gradually till patient stabilises or if maximum dose of 60mg/day is reached. Other drugs like amentadine, levodopa, apomorphine and anticholinergics have been tried with variable results.

B Paroxysmal dyskinesias.

There are characterised by intermittent posturing of the limb which may be dystonic or choreoathetotic. These occur for brief period of time. They are divided into two main groups.

1) Paroxysmal kinesigenic dyskinesia (PKD)

Disorder usually occur in childhood or early adolescence and affect male 4 times more than females. It has autosomal dominant inheritance. The paroxysm consists of any combination of dystonia, chorea, athetosis and ballism. Rapid voluntary movement, startle reactions or hyperventilation trigger the paroxysm. The duration is limited to less than 5 minutes. The frequency may be up to 100 episodes per day. Between the episodes there is refractory period when the patient is normal. PKD is highly sensitive to anticonvulsants, like phenytoin, carbamazepine and valproate.

2) Paroxysmal non kinesogenic dyskinesia (PNKD)

PNKD usually manifest during childhood or early adolescence. Majority of cases are familial with AD inheritance. The clinical presentation is similar to PKD however paroxysms lasts for 10 minutes to several hours and their frequency is often as low as few episodes per month with only few patients suffering up to three paroxysms per day. It is not triggered by physical activity but may be primed by fatigue, stress, excitement alcohol or caffeine. It does not respond to anticonvulsants and medical therapy is generally unrewarding. Limited success has been achieved with anticholinergics, levodopa, acetazolamide, carbamazepine, haloperidol, gabapentin and benzodiazepines.

Symptomatic paroxysmal dyskinesias

Few patients develop paroxysmal dyskinesias due to large number of CNS disorders. These include multiple sclerosis, myelopathy, cerebral palsy, head trauma, cerebral infarcts, basal ganglia calcification, encephalitis, radiculopathy, hypoparathyroidism, thyrotoxicosis, hypoglycaemia, reflex sympathetic dystrophy. Most of the paroxysms are short lasting and most are triggered by external stimuli. Anticonvulsants are effective in most cases.

C) Episodic ataxias

In these disorders patient has ataxia at regular intervals. They may occur with or without trigger factors, with or without additional pathological findings during attacks or intervals in between. They can be caused due to number of condition however most frequently they are hereditary.

Hereditary episodic ataxia type-1 (EA-1)

It is characterised by brief attack of ataxia and often additional features including dysarthria and dystonic elements. There is neither vertigo nor dizziness Attacks last for upto 2 minutes and can be triggered by fatigue, excitement, stress and physical trauma. In between attacks myokymia and neuromyotonia may be present. Acetazolamide and anticonvulsants are usually not effective.

Hereditary episodic ataxia type -2 (EA-2)

Attacks are accompanied by vertigo, headache and malaise. They are triggered by exercise, fatigue, stress and sometimes carbohydrate or alcohol ingestion. The attack typically lasts for several hours. In between attack diplopia and nystagmus may be present. Treatment is acetazolamide.

Symptomatic episodic ataxias:

Intermittent ataxias can occur with various neurological disorders like Hartnup disease, pyruvate decarboxylase deficiency, maple syrup urine disease, multiple sclerosis and behcet's disease. In MS the attacks are brief and may respond to carbamazepine.

References:-

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