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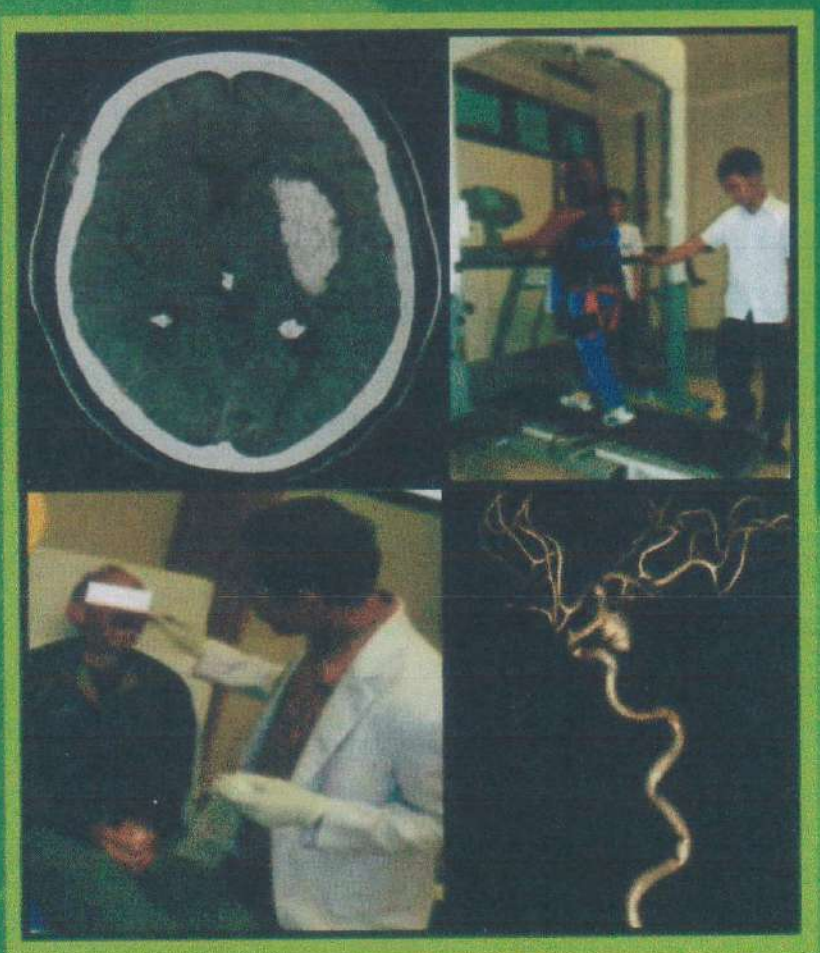
Dutch Foundation for Postgraduate Medical Course in Indonesia

Course on Neurology and Rehabilitation Medicine



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Movement Disorder After Stroke

Muhammad Hamdan

Introduction

Movement disorder caused by strokes are relatively common. They can present acutely or as a delayed sequel Many different types of hyperkinetics and hypokinetic movement disorders have been reported and can be seen after ischaemic and haemorrhagic stroke. Dystonia, chorea, tremor, parkinsonism, myoclonus, have all been described and occur at presentation of the stroke, in the delayed setting or as a progressive condition. Most are caused by lession in the basal ganglia or thalamus but can occur with strokes at many different location in the motor circuit. Many are self limiting but treatment may be required for symptoms control can present acutely or as a delayed sequel

Definition

Stroke was defined as the rapid development of signs of focal or global disturbance of cerebral function, lasting over 24 hours or leading to death, without any apparent cause. We used the definitions and guidelines for the diagnostic classification of stroke recommended by the World Health Organization (WHO), with the different subtypes based on schemes developed by the Pilot Bank of Stroke Data. Computed tomography (CT) or magnetic resonance imaging (MRI) had to show an ischaemic lesion or evidence of parenchymal, ventricular, or subarachnoid bleeding corresponding to the clinical picture.

We recognised five types of Movement Disorders in our patients:

- Chorea—defined as an arrhythmic involuntary movement, which intrudes in a sudden, brief, and non-repetitive fashion.
- Dystonia—defined as an abnormal movement characterised by sustained muscular contractions, frequently causing twisting and repetitive movements or abnormal posturing
- Tremor—defined as a rhythmic oscillation of a body.
- Myoclonus—sudden, involuntary jerking of a single muscle or a group of muscles
- Parkinsonism —defined as the presence of bradykinesia and at least one of the following symptoms: muscular rigidity, rest tremor, or postural instability.

Epidemiology

The frequency of post-stroke abnormal movements is unclear. Most reports are of isolated cases or relatively small series of cases compiled retrospectively from stroke registries. In a study of 56 patients with post-stroke abnormal movements, 3.7% of 1,500 stroke patients developed a movement disorder. A review of the hospital based Lausanne Stroke Registry identified a prevalence of 1% and an estimated incidence of 0.08% per year. Our study from stroke registries, we found 26 patient (37%) with post

stroke abnormal movement of 700 patient. Parkinsonism was the most common movement disorder in our study while hemichorea was most common in the second. Tremor was the next most common movement disorder.

Demographic

Twentysix of the 700 patients (3.7%) included in the Stroke Data Registry developed movement disorder after stroke. The average age (SD) of the included 26 patients (3.7%) (10 men, 16 women) was 68 years (range 24–88 years). Parkinsonism was the commonest movement disorder (34.6%), and the patients who had chorea were older 74,1 years than the patients with other movement disorder (table 1). Patients with chorea had the shortest time interval between diagnosis and Movement disorders onset 3,5 days (table 1) and patients with parkinsonism had the longest interval 110 days. In seven patients, the abnormal movements started on the first day of the stroke. The latest onset of an movement disorder was 8 months after stroke in a patient with parkinsonism.

Table 1. Demographic characteristics of the 26 patients with post-stroke abnormal movements

Abnormal Movement	Abnormal Disorder				
	Parkinsonism	Chorea	Tremor	Dystonia	Myclonus
No. of patients	9	8	5	3	1
Age in years	67	74	71	51	68
Sex (male/female)	4/5	3/5	2/3	1/2	0/1
Time in days (SD) between diagnosis of stroke and start of abnormal movement	110	3,5	12	16	10

Abnormal Movement, type of stroke and lesion location

A number of different locations within the brain have been identified as areas that result in abnormal movements when affected by stroke. The basal ganglia are most often implicated in post-stroke movement disorders. but can occur with strokes at many different location in the motor circuit. Tabel 2

Tabel 2. Abnormal Movement type of stroke and lesion location

Patient No.sex.age(years)	Abnormal Movement	Type of Stroke	Lesion location
1.F.75	Parkinsonism	Stroke infarct	Bilateral lenticular and pons infarcts
2.M.68	Parkinsonism	Stroke infarct	Right frontal and left parietal infarct
3.M.73	Parkinsonism	Stroke infarct	Left mesencephalic and pons infarct
4.M.81	Parkinsonism	Sroke infarct	Left caudocapsular infarct
5.M.64	Parkinsonism	Stroke infarct	Right subcortical frontal infarct
6.F.71	Parkinsonism	Stroke infarct	Bilateral lenticular infarcts
7.F.65	Parkinsonism	Stroke infarct	Bilateral temporal infarct
8.F.86	Parkinsonism	Stroke infarct	Lenticular infarcts and bilateral radiate corona
9.F.63	Parkinsonism	Stroke infarct	Right paramedian thalamic and lenticulocapsular infarcts
10.F.75	Chorea	Stroke infarct	Right paramedian thalamic infarct, left occipital calcification
11.F.88	Chorea	Stroke infarct	Left posterolateral thalamic lacunar infarct
12.M.76	Chorea	Stroke haemorrhagic	Left putaminocapsulo-thalamic haemorrhage
13.F.74	Chorea	Stroke infarct	Right thalamocapsular infarct
14.M.72	Chorea	Stroke infarct	Bilateral lenticular and right thalamic infarcts
15.F.82	Chorea	Stroke infarct	Right cerebellar and left thalamic infarct
16.F.70	Chorea	Stroke infarct	Right corona radiata and right lateral pons infarcts
17.M.56	Chorea	Stroke infarct	Right posterolateral thalamic and subthalamic infarct
18.F.72	Tremor	Stroke infarct	Right paramedian thalamo-mesencephalic infarct
19.F.73	Tremor	Stroke infarct	Right ventrolateral thalamic infarct
20.M.56	Tremor	Stroke infarct	Left putaminocapsular haematoma
21.M.79	Tremor	Stroke haemorrhagic	Right lenticular haematoma
22.F.75	Tremor	Stroke haemorrhagic	Hydrocephalus, subarachnoid haemorrhage
23.F.58	Dystonia	Stroke infarct	Left putaminal capsular infarct
24.F.72	Dystonia	Stroke infarct	Left lenticular lacunar infarct
25.M.24	Dystonia	Stroke infarct	Left globus pallidus infarct
26.F.68	Myclonus	Stroke infarct	Bilateral temporal infarct

Seven of the eight patients with hemichorea had the motor deficit on the same side the abnormal movement. The remaining once had a contralateral motor deficit. Two (2) (25%) patients with chorea improved partially six (6) (75% patient improved completely four the five patients with tremor showed motor deficit ipsilateral to the abnormal movement. 80 % the tremor disappeared completely in one patient (20%) partially. All patient with hemidystonia (100%) the motor deficit was ipsilateral to the involuntary movement. In two patients (66,6%) the dystonia disappeared completely and one patient partially (33,3%). The signs and symptoms of parkinsonism developed rapidly, starting unilaterally on the same side of the hemiparesis in seven cases and in two bilaterally with ipsilateral predominance to the motor deficit. At the start of levodopa therapy between one and four weeks after onset of parkinsonism patients, eight showed a moderate response and one did not respond. Parkinsonism was progressive in two patients. 23 patients had an infarct two patient parenchymal haemorrhage, and one patient subarachnoid haemorrhage. We found large and medium sized vessel atherothrombosis in three patients of 23 with infarct, small vessel occlusion (lacunes) in 18 patients, embolism of cardiac origin in 2, intracerebral haemorrhage 2 of whom had deep lesions.

Treatment

We describes the treatment of each movement disorder.

Parkinsonism

Patients with true vascular parkinsonism rarely respond to conventional dopaminergic therapy. Supportive therapy by the physiotherapists and occupational therapists should be arranged. Treatment for the risk factors for atherosclerotic diseases to arrest progression is recommended and includes anti-platelet agents, statins and anti-hypertensives. The caveat to this is that parkinsonism and idiopathic Parkinson's disease can co-exist, and a controlled trial of levodopa gradually increased up to a maximum of 600 mg/day (rarely up to 1,000 mg) for a minimum of 1 month should be given in order not to miss any dopaminergic responsiveness. Just as important in the apparent non-responders is to wean off the levodopa to confirm that no response, in fact, occurred and to stop unnecessary medication.

Hemichorea

Although these conditions spontaneously resolve, they do warrant urgent and vigorous short-term treatment in view of the distress and danger they incur. Non-pharmacological treatment includes good skin hygiene, high calorie nutritional support, fluids, appropriate mattress and padded bed rails. Pharmacological therapy comprises anti-dopaminergic therapy with typical and atypical neuroleptics and catecholamine-depleting agents. Typical neuroleptic agents including haloperidol, pimozide, perphenazine and fluphenazine work by blocking dopamine receptors and are the first line drug treatments. The atypical neuroleptic drugs olanzapine, quetiapine and sulpiride are less likely to cause drug-induced parkinsonism and tardive dyskinesia. Clozapine has been successful

in refractory cases, but can cause agranulocytosis. Tetrabenazine depletes presynaptic dopamine and blocks post-synaptic dopamine receptors. Reserpine depletes presynaptic stores of catecholamines and serotonin. Both are effective in treating hemiballismus but both can cause profound depression as well as hypotension and parkinsonism. Other drugs which have been used with some success include clonazepam and sodium valproate. Surgical intervention should be considered in drug-resistant cases, but they are contraindicated in the very frail and those with uncontrolled hypertension. Both stereotactic ventral intermediate thalamotomy and chronic thalamic stimulation have been effective.

Tremor

Tremor is particularly refractory to drug treatment. Rubral and palatal tremor may respond to clonazepam and sodium valproate. Dystonic tremor is treated as dystonia (see above). Propranolol is traditionally used in essential and thyrotoxic tremor but may help dampen tremor from all causes. In severe cases, functional neurosurgery (see below) may be the only useful treatment option

Dystonia

Treatment options may be used alone or in combination. Botulinium toxin injections have been a major breakthrough in the management of dystonia. Muscle contraction is reduced by direct injection into the overactive muscle, which blocks the release of acetylcholine. Other treatments include benzodiazepines, baclofen, anticholinergic drugs and dopamine-depleting/blocking agents. Clonazepam and diazepam treat focal, segmental and generalised dystonias. Higher doses are limited by drowsiness. Like botulinium toxin, the anti-cholinergic drugs block the action of the neurotransmitter acetylcholine thereby deactivating the muscle contractions. Trihexyphenidyl is the most common drug in this class, but may be more useful in younger patients due to the side-effects of confusion and constipation in the elderly. Tetrabenazine can be helpful, but may paradoxically cause dystonia. Combining trihexyphenidyl and tetrabenazine can be very effective in younger patients.

Myoclonus

The two most commonly used treatments are the GABA ergic drugs, clonazepam and sodium valproate. Other tried treatments include levetiracetam, piracetam, primidone and acetazolamide. Treatment should be started with a single agent although eventually several drugs in combination may be required. Clonazepam is effective in all types of myoclonus. Side-effects include sedation, vertigo, behavioural changes and tolerance and it is contraindicated in people with acute narrow-angle glaucoma and liver problems. Sodium valproate is effective in cortical and subcortical myoclonus. Side-effects include drowsiness, weight gain, tremor, nausea and alopecia. It is contraindicated in hepatic insufficiency. Piracetam is less sedating and may be as effective as levetiracetam. Both should be used with caution in renal impairment.

Primidone and acetazolamide can be tried but primidone causes drowsiness, confusion and falls, and acetazolamide necessitates electrolyte monitoring.

Prognosis

The prognosis for recovery of post-stroke movement disorders is generally good. If medication treatment is required, neuroleptics (such as haloperidol) or atypical agents (such as risperidone) are usually effective. However, because of the possibility of spontaneous resolution, the need for ongoing treatment should be reviewed on a regular basis.

Conclusion

Although rare, many different varieties of abnormal movement can be found after a stroke either acutely or as a delayed sequel. They can be hyperkinetic (most commonly hemichorea–hemiballismus) or hypokinetic (most commonly vascular parkinsonism). Most are caused by lesions in the basal ganglia or thalamus but can occur with strokes at many different locations in the motor circuit. Many are self-limiting but treatment may be required for symptom control.

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