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Movement disorders

Movement disorders and stroke

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INFO ARTICLE

Article history:
Received 31 March 2016
Received in revised form
30 April 2016
Accepted 8 July 2016
Available online 28 July 2016

Keywords:
Stroke
Movement disorders
Hemichorea–hemiballism
Dystonia

ABSTRACT

Stroke may be associated with different types of movement disorders, such as hyperkinetic syndromes (hemichorea–hemiballism, unilateral asterixis, limb-shaking, dystonia, tremor, myoclonus) and hypokinetic syndromes (especially vascular parkinsonism). However, movement disorders are rare and transient in acute stroke and, as a permanent consequence, are more often delayed. While ischemic and hemorrhagic strokes can happen at any level of the frontal–subcortical motor system, they can be explained most of the time by a dysfunction in the basal ganglia motor circuit. However, only brain MRI allows the involved structure(s) to be precisely located, and each syndrome is specific to the type of lesion. Treatment is above all symptomatic. Only limb-shaking syndrome requires urgent surgical treatment because of the low-perfusion hemodynamic state. The functional prognosis depends on the type of movement disorder.

1. Introduction

Focal brain lesions can induce several types of abnormal involuntary movements (AIMs), including hemichorea, hemiballism, dystonia, tremor, myoclonus, parkinsonism and asterixis [1,2]. The most frequent cause is stroke, but other disorders are sometimes responsible, such as tumors, trauma, anoxia, vascular malformations and multiple sclerosis [3]. AIMs may arise after 1–4% of strokes [3]. They may present immediately after the brain lesion (for example, as hemiballism during the acute phase of stroke) whereas the initial motor deficit may improve or rapidly recover. In other cases, AIMs (for example, dystonia) have appeared after a long delay (from a few months to years, especially in those of a younger age at the time of initial injury) of stable motor impairment [5,6]. Two types of stroke-related AIMs can be distinguished: transient (hemichorea–hemiballism, asterixis, episodic paroxysmal dyskinesia, often in the acute phase) and persistent (dystonia, tremor, vascular parkinsonism, vascular chorea). Whatever the timescales of their appearance or disappearance, AIMs may be the main symptoms of stroke. As a consequence, this can lead to a delay in diagnosis or even a lack of making a specific diagnosis. This problem is sometimes clinically evident when the AIM is intense and clearly present (hemiballism) as opposed to being mild and focused (asterixis). As any delay in diagnosis may have unhelpful consequences in terms of the patient's clinical and therapeutic management, this type of AIM should be considered a neurological emergency, where the patient is then addressed as quickly as possible in a stroke unit.

AIMs can be observed in patients with either ischemic or hemorrhagic stroke but also in those with cerebrovascular malformations and dural arteriovenous fistulas. In ischemic

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http://dx.doi.org/10.1016/j.neurol.2016.07.006
0035-3787/

stroke, the most common subtype is small-vessel disease with lacunar infarcts. AIMs are often unilateral and contralateral to the brain lesion, and sometimes bilateral, but rarely unilateral (for example, in cases of cerebral lesions) [7]. They may be attributed to lesions of various structures, particularly the striatopallidal complex, mesencephalon and thalamus and, more rarely, the cortex. If the vascular lesion is localized with well-defined boundaries, it will facilitate any anatomoclinical correlations. Computed tomography (CT) and magnetic resonance imaging (MRI) studies have produced many reports of clinicopathological correlations based on both single cases and small series of patients with lesions in the above-mentioned structures. This has enabled precise descriptions of the pathophysiological mechanisms behind various types of AIMs [1,8–10].

The present report is a review of the AIMs (hyperkinetic syndromes) observed in stroke with clinical classification based on the movement's progression and whether or not it is transient or persistent. However, 'vascular parkinsonism', a hypokinetic syndrome primarily characterized by lower-limb bradykinesia and sometimes a progressive supranuclear palsy or corticobasal syndrome, has been excluded, as this entity merits a chapter all on its own.

2. AIMs in transient stroke

AIMs arise immediately after a stroke and may regress spontaneously and progressively often within a few hours or days. This type of hyperkinetic movement disorder is seen less frequently, with a prevalence of 1–4%, in acute stroke [7,11]. It may sometimes be the only clearly observed clinical symptoms, which are often masked by the motor deficit. The following types of movement have been described: hemichorea–hemiballism, unilateral asterixis, transient paroxysmal dyskinesia (limb-shaking) and, to a lesser extent, myoclonus dystonia syndrome affecting the hands, stereotypies, akathisia and tics.

2.1. *Hemichorea–hemiballism syndrome*

This syndrome is among the most frequently seen post-stroke involuntary movement disorders [7,12]. The two eponymous terms are often used interchangeably, and refer to continuous and involuntary movements involving only one side of the body. Hemichorea consists of continuous random, anarchic and jerking movements involving both the distal and proximal muscles (but is sometimes localized more distally), whereas hemiballismus is characterized by violent irregular flinging movements of the limbs due to contractions of the proximal muscles. This is especially true after a stroke where hemiballism often evolves into hemichorea. Thus, it would seem more sensible to use the term 'hemiballism-hemichorea' to describe these symptoms. In any case, they should be considered two different entities (or two points on a spectrum) of the same underlying disease process.

Hemichorea–hemiballism is induced by either hemorrhagic or ischemic stroke in 50–60% of cases [13]. Previous studies have involved only a small number of patients, and have reported clinical outcomes and attempted to precisely localize the lesion responsible for post-stroke hemichorea [7,12–14]. In cases of other principal causes of hemichorea–hemiballism (abscesses, metastatic lesions, acquired immunodeficiency syndrome, levodopa therapy, Sydenham's chorea, neonatal anoxic brain injury, multiple sclerosis and central nervous system lupus), the mean age of onset was lower (35 years) than in the stroke subgroup (61 years) [13].

AIM onset occurred suddenly on the day of the stroke in 85% of cases [12], although they may worsen gradually over a period of several weeks or recommence after a latent period of several months [13]. They are observed on the side contralateral to the stroke [15]; bilateral symptoms appear only if bilateral basal ganglia lesions are observed on brain MRI. The upper and lower limbs are typically involved, although, in some cases, only a single part of the body is affected (neck, arm or leg). Other combinations of AIMs (dystonia and segmental myoclonus, athetosis, orofacial movements, parkinsonian tremor) are sometimes also observed, depending of the location of the lesions. As the patient's condition progresses, the AIM becomes intermittent, typically with spontaneous disappearance in more than half of patients. However, some patients may have persistent moderate or severe symptoms (including induced pain) with some level of disability, which will depend on both the intensity and duration of the AIM. Neurological examination reveals hypotonia in the affected limbs, a possible sensory deficit and mild motor weakness [12]. Neurobehavioral changes (logorrhea, euphoria) have also been described in a case of post-hematoma hemiballism of the left subthalamic nucleus [16].

In classic terms, hemichorea and hemiballism are both associated with lesions of the caudate nucleus and subthalamic nucleus, but also in association with lesions of the putamen and/or globus pallidus, striatum (caudate nucleus and putamen) and thalamus [1,2,12,15,17]. Less frequently, the stroke lesion may also involve the cortex in the superficial territory of the middle cerebral artery (parietal, insular and temporal cortex) [18].

The pathogenesis of hemichorea caused by contralateral lesions of the striatal neurons of the indirect striato-thalamocortical pathways may be explained by interruption of gamma-aminobutyric acid (GABA) transmission from the striatum to the external globus pallidus (GPE), which can then increase GPE neuronal activity and inhibit the subthalamic nucleus. Such inhibition would induce a loss of control of the internal globus pallidus (GPI) neurons which, in turn, can finally lead to loss of inhibition of the motor thalamus. Lesions of the subthalamic nucleus may also induce the same dysfunction, with a lack of inhibition of the motor thalamus. In cases of hemichorea-hemiballism caused by a cortical lesion, an excitatory neuronal circuit from part of the frontal or parietal cortex (the somatosensory cortex projecting into the caudate nucleus and putamen) must be interrupted to produce AIMs. The functional prognosis is significantly better in patients with cortical strokes compared with those who have subthalamic lesions [12]; in the former patients, AIMs are probably induced by transient hypoperfusion or functional 'disconnection' rather than destruction of the basal ganglia circuitry.

Hemichorea-hemiballism after stroke is uncommon whereas stroke in the above-mentioned arterial territory is rather