

BILATERAL BALLISM IN RENAL FAILURE – CASE REPORT

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Abstract: *Ballism is a rare movement disorder characterized by proximal, large-amplitude involuntary movements. We present the case of a 73 years old patient with history of repeated strokes and chronic renal failure who develops ballism. The article contains a review of the literature regarding this rare hyperkinetic disorder.*

Key words: *ballism, renal failure, phenomenology, treatment.*

1. Introduction

Ballism represents an non-rhythmic, repetitive, erratic, involuntary movement, mainly involving proximal parts of the limbs.

Ballism belong to the hyperkinetic movement disorders. Depending of the etiology, sometimes the ballism diminishes and the movements may change to other types of movements like chorea [1]. Ballism is usually unilateral and very rare bilateral.

The aim of this paper is to present a case report of a rare patient with bilateral ballism and renal failure.

2. Case study

We report the case of a 73 years old man with history of two prior ischemic strokes, hypertension, myocardial infarction, chronic renal failure on long-term hemodialysis therapy (three times per week), who was admitted in our clinic with altered consciousness and poor general

condition. Neurological exam at admission revealed coma (GCS 6 points), symmetrical pupils, right faciobrahial hemiparesis, decreased reflexes on the right side and bilateral Babinski sign. Two days after admission, he developed violent, arrhythmic involuntary movements involving proximal and distal muscle of both arms. The movements were continuous, non-rhythmic, non-suppressible and stereotyped. During sleep there were no such movements.

Dynamic CT scans revealed left fronto-temporal old infarction and left cortical and subcortical hypodensity suggestive for acute stroke. Haloperidol treatment improved the abnormal movements. In evolution, his condition gradually deteriorated with a decreasing level of consciousness. The patient presented hypotension, anuria, worsening of bilateral ballism and athetosis. The patient died one month after admission.

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3. Discussion

Anatomically, ballism results from different lesions of the subthalamic nucleus (STN). STN inhibits the activity of the globus pallidus.

On a large study conducted in 1961 by Dierssen and Giolino on 116 patients, in 53% of cases the lesions involved subthalamic nucleus, followed by corpus striatum (41%) and thalamus (28%) [5].

Destruction of the subthalamic nucleus due to different causes determines a reduction of excitatory glutaminergic inputs to the internal segment of the globus pallidus. In this way it will be a decrease of the normal inhibition of the ventral anterior and ventral posterior thalamic nuclei.

The thalamo-cortical activity is increased and the premotor areas are hyperactivated, producing ballistic movements [10].

In a study conducted by Krauss et al. on 16 patients, eleven of them presented hemiballism, three patients had monoballism and two patients had bilateral ballism [12]. Regarding the etiology, in most of the cases hemiballism has a vascular etiology [4]. This could be ischemic or hemorrhagic stroke. In these cases, the onset is sudden, the upper limb is more affected than the lower limb. Sometimes could occur complex movements with hemiballism associated with hemichorea and other neurological signs. Hemiballismus can occur when adjacent structures surrounding subthalamic nucleus are involved [4].

Bilateral ballism is a rare condition that can occur in different situations, both vascular and non-vascular. Non-vascular etiologies include drug use like oral contraceptives [7], lamotrigine [20], hyperthyroidism, multiple sclerosis, metabolic disturbances [9]. Subthalamic metastases or cerebral tumors, meningoencephalitis, tuberculosis, arteriovenous malformation, cerebral toxoplasmosis, head trauma and sepsis

may be other non-vascular causes of ballism [6], [11], [12].

Endocrine disorders, like thyrotoxicosis, may also develop hyperkinetic movement disorders like chorea or ballism [15]. Among metabolic causes of chorea or ballism, the most commonly reported is non-ketotic hyperglycemia in type II diabetes mellitus [2, 11]. Once the glycemic control is achieved, a gradual remission of the hyperkinesias has been observed. Other possible causes, relatively uncommon, include electrolyte disturbances like hyper or hyponatremia, hypomagnesemia, and hyper or hypocalcemia [2, 11].

In patients with bilateral basal ganglia lesions induced by chronic renal failure with azotemia, the most common clinical manifestations include parkinsonism, consciousness impairment, dysarthria, dysphagia and dyskinesia [18].

There are few cases reported in the literature of bilateral ballism associated with chronic renal failure. Ozben describes a case of a patient with diabetic mellitus and end-stage nephropathy [14]. Wang reports 3 patients with uremia and high serum creatinine levels who developed generalized dyskinesias and gait disturbances [18]. There have been described cases of paroxysmal bilateral ballism induced by hypoglycemia [16] and by non-ketotic hyperglycemia [3].

Decreased metabolism of glucose in the basal ganglia was demonstrated by Wang et al. using FDG-PET examination [19]. Abnormal dilatation of small vessels may be reversible and can explain complete resolution of the involuntary movements once the metabolic parameters have been corrected [13].

Neuroimaging like CT and MRI scans are the most useful tools to assess the lesions of the basal ganglia (putamen, subthalamic and caudate nucleus). The characteristic MRI findings include

hyperintense signal of the pallidum on T1, suggesting microhemorrhages at this level [11].

Review of literature shows different patterns of remissions: spontaneous remission of ballism secondary to cardiovascular disease and gradual remission in metabolic or endocrine disorders [17]. Symptomatic treatment with dopamine antagonist (haloperidol), benzodiazepines, tetrabenzine, atypical neuroleptics, or anticonvulsivants (topiramate, levetiracetam, valproate) may ameliorate hyperkinetic movements [8].

Neurosurgery could offer improvement, for instance stereotactic surgery, such as posteroventral pallidotomy or ventrolateral thalamotomy [9].

4. Conclusions

Bilateral ballism is a rare syndrome associated with stroke and chronic renal failure. Physiopathological mechanisms remain still poorly understood. Hypoperfusion and inflammation due to uremic toxins and ischemic consequences of the stroke may cause bilateral lesions of the basal ganglia leading to overactivation of thalamocortical activity and consequently to hyperkinetic movements.

References

- Adler, C.H., Ahlskog, J.E.: *Parkinson's Disease and Movement Disorders: Diagnosis and Treatment Guidelines for the Practicing Physician*. In: Humana Press, 2000, p. 329.
- Albanese, A., Jankovic, J.: *Hyperkinetic Movement Disorders: Differential Diagnosis and Treatment*. John Wiley & Sons, 2012, p. 175-181.
- Boughammoura-Bouatay, A., Chebel, S., Younes-Mhenni, S., et al.: *Biballism due to non-ketotic hyperglycaemia*. In: *Diabetes Metab*. 2008 Dec; 34(6 Pt 1): 617-9.
- Chaudhuri, R.K., Ondo, W.G.: *Handbook of Movement Disorders*. In: Current Medicine Group, 2009, p. 101.
- Dierssen, G., Gioino, G.: *Correlación anatómica del hemibalismo*. In: *Rev Clín Esp* 1961; 82: 283–305.
- Donaldson, I., Marsden, C.D., Schneider, S.A., Bhatia, K.P.: *Marsden's Book of Movement Disorders*. In: Oxford University Press, 2012. pg. 881-887.
- Driesen, J.J., Wolters, E.C.: *Bilateral ballism induced by oral contraceptives*. A case report. In: *J Neurol*. 1986 Nov; 233(6):379.
- Fahn, S., Jankovic, J., Hallett, M.: *Principles and Practice of Movement Disorders*. Elsevier Health Sciences, 2011, p. 401-403.
- Fernandez, H.H., Machado, A.G., Pandya, M. A.: *Practical Approach to Movement Disorders: Diagnosis and Medical and Surgical Management*. In: Demos Medical, 2007. p. 23-24.
- Guridi, J., Obeso, J.A.: *The subthalamic nucleus, hemiballismus and Parkinson's disease: reappraisal of a neurosurgical dogma*. In: *Brain*. 2001 Jan; 124(Pt 1):5-19.
- Jankovic, J., Tolosa, E.: *Parkinson's Disease and Movement Disorders*. Lippincott Williams & Wilkins, 2007, p. 241-242.
- Krauss, J.K., Borremans, J.J., Nobbe, F., et al.: *Ballism not related to vascular disease: A report of 16 patients and review of the literature*. In: *Parkinsonism Relat Disord*. 1996 Jan; 2(1):35-45.
- Lee, P.H., Shin, D.H., Kim, J.W., et al.: *Parkinsonism with basal ganglia lesions in a patient with uremia: Evidence of vasogenic edema*. In: *Parkinsonism Relat Disord*. 2006 Mar; 12(2):93-6.

14. Ozben, S., Oguz, E., Ozer, F., et al.: *Bilateral ballism in a patient with diabetes mellitus and chronic renal failure*. In: *J Clin Neurosci*. 2011 May; 18(5):731-2.
15. Ristic, A.J., Svetel, M., Dragasevic, N., et al.: *Bilateral chorea-ballism associated with hyperthyroidism*. In: *Mov Disord* 19:982, 2004.
16. Takahashi, S., Ohkawa, S.: *Paroxysmal bilateral ballism induced by hypoglycemia*. In: *Rinsho Shinkeigaku*. 2006 Apr; 46(4):278-80.
17. Wali, G.M., Khanpet, M.S., Mali, R.V.: *Acute movement disorder with bilateral basal ganglia lesions in diabetic uremia*. In: *Ann Indian Acad Neurol*. 2011 Jul;14(3):211-3.
18. Wang, H.C., Brown, P., Lees, A.J.: *Acute movement disorders with bilateral basal ganglia lesions in uremia*. In: *Mov Disord*. 1998 Nov; 13(6):952-7.
19. Wang, H.C., Hsu, J.L., Shen, Y.Y.: *Acute bilateral basal ganglia lesions in patients with diabetic uremia: an FDG-PET study*. In: *Clin Nucl Med*. 2004 Aug; 29(8):475-8.
20. Yetimalar, Y., Seckin, M., Secil, Y., Basoglu, M.: *Lamotrigine-induced bilateral ballism*. In: *Mov Disord* 2007; 22:1832-1833.