

CATALEPTIC POSTURES IN THALAMIC HEMORRHAGE

Case report

Gustavo Saposnik, Jorge Mauriño, Leonardo A. Gonzalez

ABSTRACT - We report a case of catalepsy associated with thalamic hemorrhage. A 72 year-old hypertensive woman had acute onset of right-sided weakness and speech disturbances. She was on anticoagulants because of aortic valve replacement. When postures were imposed, the patient maintained the left upper limb raised for several minutes, even in uncomfortable or bizarre positions. A CT scan of the head revealed a left thalamic hemorrhage. Cataleptic postures have been reported in few cases with acute stroke.

KEY WORDS: catalepsy, hemorrhage, stroke, thalamus, catatonia.

Posturas catalepticas y hemorragia talámica: reporte de caso

RESUMEN - Se reporta un caso de catalepsia asociado a una hemorragia talámica. Una mujer hipertensa de 72 años fue admitida por presentar en forma súbita debilidad en hemicuerpo derecho y alteración del lenguaje. Estaba en tratamiento anticoagulante por reemplazo valvular aórtico. Al examen, la paciente mantenía el miembro superior izquierdo elevado por varios minutos, aún en posiciones incómodas o bizarras. La TAC craneal evidenció un hematoma talámico izquierdo. Existen escasos reportes de posturas catalepticas en el contexto de ictus agudo.

PALABRAS CLAVE: catalepsia, ictus, hemorrágico, tálamo, catatonía.

Catalepsy is one of the features of the catatonic syndrome. The criteria for catatonia have been defined in the DSM IV¹⁻³. This syndrome may be due to general medical conditions such as, metabolic derangement, drug reactions, or CNS lesions. Catalepsy has been defined as a tendency to maintain postures induced by the examiner¹⁻³. There have been few reports of cataleptic postures in cases with acute ischemic stroke⁴⁻⁸. We describe isolated cataleptic posturing of the left upper limb in a patient with a left thalamic hemorrhage.

CASE

A 72 year-old right-handed woman had acute onset of right-sided weakness and speech disturbances while walking on the street. The patient had a past history of arterial hypertension and was on anticoagulants because of aortic valve replacement 14 years before. There was no history of psychiatric illness or dopaminergic blocker intake. On neurological examination, she had severe right-sided hemiparesis, hemisensory loss, hemianopia, and global aphasia. The National Institute of Health (NIH) stroke scale score was 22. When postures were imposed, such

as lifting of her arms, the patient maintained the left upper limb raised for several minutes. These postures remained unchanged after moving the upper extremities passively, or when other items of the neurological examination were tested. The imposed postures were present even in uncomfortable or bizarre positions (i.e.: raising and flexing the left arm, sustaining a pen or holding a hammer), or when unrelated conversation was brought up. We did not find parkinsonian signs, perseverative behaviors, or paratonia on the exam. The patient had no other features of the catatonic syndrome (i.e.: mutism, mannerisms, echopraxia, etc). On admission, INR was 4.5. A computed tomography (CT) scan of the head revealed a left thalamic hemorrhage, 50 cc in volume, with extension into the ventricles and the putamen (Fig 1). Cataleptic postures could be elicited only for the first 72 hours after stroke onset.

DISCUSSION

Catalepsy is an uncommon manifestation in stroke patients. In the few existing reports, most cases were described in ischemic infarction⁴⁻⁸.

In 1958, Denny-Brown and Chambers⁴ described

Department of Neurology, Stoke Unit, Hospital J.M. Ramos Mejia, Buenos Aires, Argentina.

Received 29 January 2001, received in final form 6 April 2001. Accepted 12 April 2001.

Gustavo Saposnik, MD - Department of Neurology, Hospital J.M. Ramos Mejia - Charcas 4431 4 "10" - 1425 Buenos Aires - Argentina. FAX: 54 11 4803-8674. E-mail: gsaposnik@yahoo.com

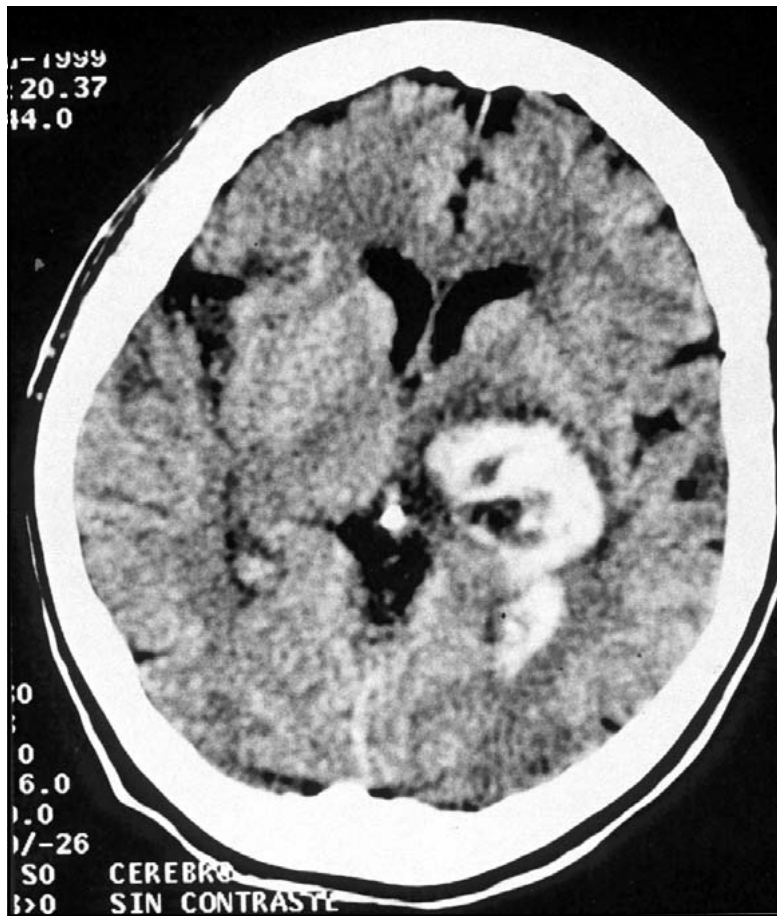


Fig 1. Tomographic computed scan with no contrast medium shows a left thalamic hemorrhage extending to the lateral ventricle and the posterior putamen.

cataleptic persistence of attitude in a 59 year-old woman with ischemic stroke, whose limbs "remained for many minutes in any posture passively applied to them". Saver et al.⁷ reported a case of a 75 year-old man with bilateral cataleptic postures more prominent on the hemiparetic side, after a right temporal-parietal-occipital infarction. This patient exhibited prominent cataleptic posturing in the left arm and to a lesser extent in the right upper extremity and left lower limb, sparing the right lower extremity. Later on, Saposnik et al.⁸ prospectively looked for cataleptic postures in 216 acute stroke patients. Catalepsy was found on the nonhemiparetic side in five subjects (2.3%), all of them with middle cerebral artery territory infarctions. This motor phenomenon was not seen in hemorrhagic stroke.

In 1993, Fukutake et al.⁹ reported one patient with right subcortical parietal hematoma exhibiting cataleptic posturing of the left upper limb. This motor phenomenon was neither observed in the right arm nor in the lower extremities. Cataleptic postures lasted longer than ten minutes, and disappeared twelve hours after the stroke onset. To our knowledge, this

was the only report of isolated catalepsy due to intracerebral hemorrhage in the literature.

In the current report, we describe cataleptic postures in isolation of other features of the catatonic syndrome³. Our patient had a thalamic hemorrhage secondary to excess anticoagulation. Other differential diagnoses were considered. We did not find features of athetosis or a dystonic limb because there were no such abnormal movements in the extremity. An akynetic-rigid or hypokinetic-hypometric syndromes were also excluded. There was no difficulty with the initiation, speed, or gain of the movements. Our patient did not exhibit the recurrent performance of an action, thus excluding motor perseveration.

In all previous reports of catalepsy after structural lesions, the most commonly involved areas included the frontal, parietal, or temporal lobes, or the basal ganglia^{4,7}. Carroll mentioned the role of D2 and GABA_A receptors in the catatonic syndrome. He hypothesized that GABA_A agonists may be effective in catatonia, while D2 antagonist may predispose to catalepsy¹⁰. Northoff et al. found a delayed latency in the cortical and motor response in patients

with catatonia taken Lorazepam¹¹. The authors suggest that catatonic patients have dysfunction in the inhibitory control of the motor cortical function with increased gaba-ergic sensitivity. They mentioned that disturbances in the cortico-limbic and thalamo-cortical circuits may be responsible for the catatonic postures¹². They also found a significant decreased blood flow in the right prefrontal and parietal cortex in patients with catatonia¹³.

In our patient, cataleptic postures occurred after a left thalamic hemorrhage for a brief period of time. Transient cognitive and motor phenomena after acute stroke may be more common than reported. The thalamus mediates motor functions by transmitting information from the basal ganglia and cerebellum to the frontal lobe. Partial recovery from the injury may explain the transient duration of the signs. However, because of the heterogeneity of the structures involved in most reports, it is difficult to establish the precise anatomical basis and the underlying mechanism of catalepsy.

Further studies are necessary to better understand the nature of this transient phenomenon.

REFERENCES

1. Taylor MA. Catatonia: a review of a behavioral neurologic syndrome. *Neuropsychiatr Neuropsychol Behav Neurol* 1990;3:48-72.
2. Gelenberg AJ. The catatonic syndrome. *Lancet* 1976;1:1339-1341.
3. American Psychiatric Association. Diagnostic and statistical manual of mental disorders (DSM-IV), fourth edition. Washington, DC: American Psychiatric Press, 1994.
4. Denny-Brown D, Chambers R. The parietal lobe and behavior. *Assoc Res Nerv Ment Dis Proc* 1958;36:35-117.
5. Tippen J, Dunner FJ. Biparietal infarctions in a patient with catatonia. *Am J Psychiatry* 1981;138:1386-1387.
6. Howard R. Catatonia following biparietal infarction with spontaneous recovery. *Postgrad Med J* 1989;65:316-7.
7. Saver JL, Greenstein P, Ronthal M, Mesulam M. Asymmetric catalepsy after right hemisphere stroke. *Mov Disord* 1993;8:69-73.
8. Saposnik G, Bueri JA, Rey RC, Sica R. Catalepsy after stroke. *Neurology* 1999;53:1132-1135.
9. Fukutake T, Hirayama K, Komatsu T. Transient unilateral catalepsy and right parietal damage. *Jpn J Psychiatry Neurol* 1993;47:647-50.
10. Carroll L. GABAa versus GABA_B hypothesis of catatonia. *Mov Disord* 1999;14:161.
11. Northoff G, Pfenning A, Krug M, et al. Delayed onset of late movement-related cortical potentials and abnormal response to lorazepam in catatonia. *Schizophr Res* 2000;44:193-211.
12. Northoff G, Koch A, Wenke J, et al. Catatonia as a psychomotor syndrome: a rating scale and extrapyramidal motor symptoms. *Mov disord* 1999;14:404-416.
13. Northoff G, Steinke R, Nagel Dezerwenka C, et al. Right lower prefronto-parietal cortical dysfunction in akinetic catatonia: a combined study of neuropsychology and regional cerebral blood flow. *Psychol Med* 2000;30:583-586.