

Depression with anosognosia following a left subcortical stroke

Iftah Biran, Anjan Chatterjee*

The Department of Neurology, Hospital of the University of Pennsylvania, University of Pennsylvania, 3 West Gates Bldg., 3400 Spruce Street, Philadelphia, PA 19104, USA

Received 30 September 2002; accepted 16 October 2002

Abstract

Controversy exists about whether depression following stroke represents a biologically mediated change or a psychological reaction to the deficits. We present a patient with an acute isolated personality change and depression following a lacunar infarct of the left internal capsule, who was unaware of his affective change. His anosognosia for the depression, which was abrupt and not accompanied by cognitive impairments, suggests that post stroke depression, at least in this case, is better explained by a biological than a psychological model.

© 2002 Elsevier Science B.V. All rights reserved.

Keywords: Affective disorders; Awareness; Neuropsychiatry; Post stroke depression; Lacunar strokes

1. Introduction

Depression after stroke is frequent, occurring in 20–50% of patients. It is more common with left than with right hemispheric strokes and when the stroke is located anteriorly, close to the frontal pole. Post-stroke depression (PSD) can also follow sub-cortical lesions, especially when left-sided. The clinical presentations of PSD vary from apathy to anxiety or even emotional catastrophic reaction [1]. Controversy exists about whether PSD is biochemically mediated by disruption of monoaminergic pathways or is a psychological reaction to the neurological deficit. Supporters of the monoaminergic hypothesis claim that PSD is very similar to biologically mediated endogenous depression. The clinical symptoms in PSD and endogenous depression are similar, as are biological markers such as the dexamethasone suppression test. Additionally, there is no clear correlation between the severity of depression and the degree of the neurological deficit [1,2]. By contrast, supporters of the reactive hypothesis claim that PSD is distinguishable from functional depression and that mood in subjects with PSD is linked to handicaps and disabilities [3]. We

report a patient who presented with the clinical picture of depression following a left subcortical stroke. He was unaware of his profound and abrupt depression. This case offers some insight into the question of whether PSD is biologically mediated or is a psychological reaction to the neurologic injury.

2. Case report

An 80-year-old right-handed man suffered an acute change in his personality. His family members noticed this change while driving to his sister's home. Normally talkative and engaging, he became withdrawn and seemed sadder. This change was sufficiently dramatic that his sister immediately commented on this difference. The patient's personality change persisted thereafter. He was passive, less socially involved and gave the impression of being sad to his family. The patient was not aware of this abrupt change and had no spontaneous complaints. He scored 25/30 on the Mini Mental State Exam, losing four points in trying to spell 'world' backwards and a point in trying to recall three objects. His language and visual-perceptual functions were intact and his praxis was normal. On bedside tasks assessing the frontal lobes such as verbal fluency, contrasting programs task, learning the Luria gestures

* Corresponding author. Tel.: +1-215-662-4265; fax: +1-215-349-8260

E-mail address: anjan@mail.med.upenn.edu (A. Chatterjee).

and the cross motor inhibition tasks, his performance was normal. His neurological exam demonstrated only minimal findings: cranial nerves were intact aside from minimal flattening of the left nasiolabial fold. Motor exam was only notable for mild bilateral and symmetric facilitatory paratonia. There were no pyramidal signs. His reflexes were diminished throughout. The sensory and cerebellar exams were normal. He had a slightly diminished right arm swing when walking. MRI of the head demonstrated an acute infarct of the genu and posterior limb of the left internal capsule (see Fig. 1). A diagnosis of depression following left lacunar stroke was made and the patient was started on Sertraline Hydrochloride. In the next follow-up, 2 months later, his family members reported a marked improvement. However, the patient who was not aware of his initial change, was also unaware of changes after treatment.

3. Depression assessment

The patient was assessed using open interviews and depression questionnaires. The clinical impression from

interviewing the patient, his wife and his son was of depression with minimal somatic or constitutional symptoms. His symptoms were consistent with major depression according to the DSM-IV TR criteria (depressed mood, anhedonia, change in sleeping pattern, psychomotor retardation, fatigue and impaired concentration). The patient and his wife filled out independently the Beck depression inventory (BDI) and the geriatric depression scale (GDS). Consistent with the clinical observations, the patient did not rate himself as depressed; however, his wife rated him as borderline depressed on the BDI, and depressed on the GDS [4] (Table 1).

4. Discussion

Our patient had an isolated and abrupt depression following a left subcortical infarct located in the internal capsule, mainly in its genu [5]. The stroke resulted probably from occlusion of a perforating branch of the internal carotid artery or the anterior choroidal artery as suggested by the sub-cortical vascular territories sup-

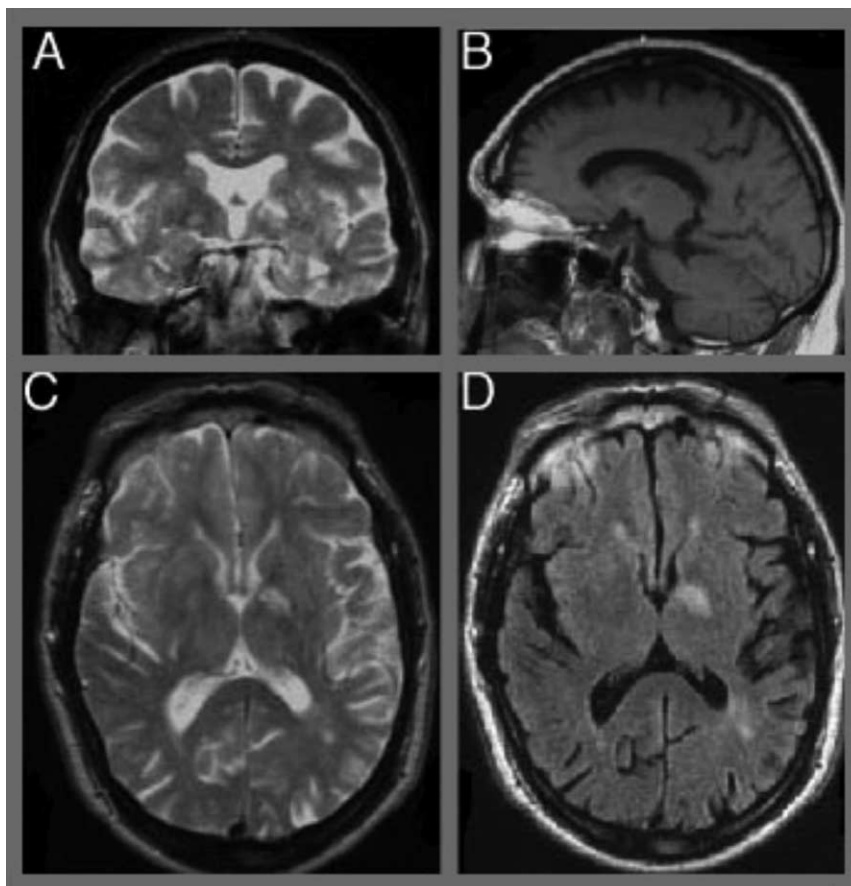


Fig. 1. MRI scan demonstrating diffuse atrophy, periventricular white matter disease and a left-sided hyper-intense area in the genu and posterior limb of the internal capsule (A, sagittal T1; B, coronal T2; C, axial FLAIR; D, axial T2).

Table 1
Depression assessment by patient and spouse

By subject	By spouse	Cut off for depression
GDS 2/30	15/30	≥ 10
BDI 0/21	9/21	≥ 9–10
DSM 2/9 (Psychomotor retardation, impaired concentration)	6/9 (Including both anhedonia and depressed mood)	5/9 (Including one of anhedonia or depressed mood)

GDS, geriatric depression scale; BDI, Beck depression inventory; DSM, diagnostic and statistical manual.

plied by these vessels [5]. The basal ganglia when damaged, especially on the left side, are implicated in depressive symptoms, thought to be due to disruption of frontal–subcortical circuits [6]. As fibers from three frontal–subcortical circuits (dorsolateral, orbitofrontal and anterior cingulate) coalesce in the genu of the internal capsule [7], affective symptoms are predicted in lesions to this structure. These symptoms are probably secondary to depletion in cortical biogenic amines due to pathway interruption [8,9]. Accordingly, affective symptoms can sometimes accompany lesions to the genu and posterior limb of the internal capsule [10,11] but these symptoms are usually accompanied by language, memory and executive symptoms not evident in our patient.

An unusual feature of our patient was his anosognosia for the depression, as confirmed by the discrepancy between his self-evaluation and his spouse's evaluation. Anosognosia for PSD is rare [12]. Models of anosognosia for hemiplegia propose that subjects only discover their deficit if they engage in activities that confront their disability. When patients have an intentional deficit and are disinclined to move their limb they are unlikely to discover their deficit [13,14]. To extend this reasoning to our patient, the absence of somatic or constitutional symptoms means that the patient is unlikely to be initially aware of a problem. Furthermore, his passivity and apathy make it unlikely that he would place himself in situations in which he would discover the change.

For two reasons, our case supports the biological rather than the psychological hypothesis of PSD. Firstly, psychological reactive mechanisms require time to evolve. In our case, the personality change was abrupt and the primary symptom that led to the discovery of the stroke. Secondly, our patient did not have other symptoms to which he might have reacted. Thus, we suggest that the capsular lesion in this case interrupted specific frontal–subcortical circuits leading to depletion in cortical monoamines resulting in depression without any accompanying cognitive impairment.

Acknowledgements

This work was supported in part by the American Physicians Fellowship for Medicine in Israel and NIH RO1 NS37539.

References

- [1] Starkstein S, Robinson RG. Stroke. In: Coffey CE, Cummings J, Lovell M, Pearlson GD, editors. *The American Psychiatric Press textbook of geriatric neuropsychiatry*, 2 ed. Washington, DC: American Psychiatric Press, 2000:601–20.
- [2] Starkstein S, Robinson RG. Affective disorders and cerebral vascular disease. *Br J Psychiatry* 1989;154:170–82.
- [3] Gainotti G, Azzoni A, Marra C. Frequency, phenomenology and anatomical–clinical correlates of major post-stroke depression. *Br J Psychiatry* 1999;175:163–7.
- [4] Burns A, Lawlor B, Craig S. *Assessment scales in old age psychiatry*, 1 ed. London, UK: Martin Dunitz, 1999.
- [5] Duvernoy HM. *The human brain: surface, three-dimensional sectional anatomy with MRI, and blood supply*, 2 ed. Wien, Austria: Springer, 1999.
- [6] Vataja R, Pohjasvaara T, Leppavuori A, et al. Magnetic resonance imaging correlates of depression after ischemic stroke. *Arch Gen Psychiatry* 2001;58:925–31.
- [7] Burruss JW, Hurley RA, Taber KH, Rauch RA, Norton RE, Hayman LA. Functional neuroanatomy of the frontal lobe circuits. *Radiology* 2000;214:227–30.
- [8] Morrison JH, Molliver ME, Grzanna R. Noradrenergic innervation of cerebral cortex: widespread effects of local cortical lesions. *Science* 1979;205:313–6.
- [9] Robinson RG, Kubos KL, Starr LB, Rao K, Price TR. Mood disorders in stroke patients. Importance of location of lesion. *Brain* 1984;107:81–93.
- [10] Starkstein SE, Fedoroff JP, Price TR, Leiguarda R, Robinson RG. Apathy following cerebrovascular lesions. *Stroke* 1993;24:1625–30.
- [11] Tatemichi TK, Desmond DW, Prohovnik I, et al. Confusion and memory loss from capsular genu infarction: a thalamocortical disconnection syndrome. *Neurology* 1992;42:1966–79.
- [12] Fedoroff PJ, Starkstein S, Parikh RM, Price TR, Robinson RG. Are depressive symptoms nonspecific in patients with acute stroke. *Am J Psychiatry* 1991;148:1172–6.
- [13] Chatterjee A, Mennemeier M. Anosognosia for hemiplegia: patient retrospections. *Cognit Neuropsychiatry* 1996;1:221–37.
- [14] Heilman KM. Anosognosia: possible neuropsychological mechanisms. In: Prigatano GP, Schacter DL, editors. *Awareness of deficit after brain injury*. New York: Oxford University Press, 1991:53–62.