Neurol Sci (2013) 34:999–1001
DOI 10.1007/s10072-012-1146-2

LETTER TO THE EDITOR

Frontal dementia related to thalamic stroke: a case report

Sabrina Realmuto · Valentina Arnao · Antonio Cinturino · Maria Antonietta Mazzola · Simona Talamanca · Marianna Riolo · Ignazio Cusmano · Chiara Cupidi · Tommaso Piccoli

Received: 17 February 2012 / Accepted: 21 June 2012 / Published online: 5 July 2012

Dear Editor,

The behavioral variant of frontotemporal dementia (bvFTD) is characterized by modifications of personality, social behavior and cognition and relies on a neurodegenerative process [1]. However, vascular lesions affecting subcortical structures, mainly the caudate nucleus and thalamus, may result in a clinical syndrome resembling bvFTD [2, 3].

We report a case of paramedian thalamic stroke mimicking frontotemporal dementia. A 58-year-old right-handed man was referred to our hospital because of behavioral and cognitive changes formerly diagnosed as bvFTD. His medical history was negative for previous illness and the patient neither smoked nor drank alcohol. In July 2010, the patient suddenly presented with loss of consciousness and was admitted to a nearby hospital. After a few days the decreased level of consciousness resolved and he developed amnesia and personality changes such as disinhibition, apathy and loss of self-activation. Toxicological exams, routine CSF analysis including total proteins, count cell, glucose and CSF culture were normal at admission. CT scan performed within twelve hours the onset of disturbances was also normal. The patient was diagnosed as affected by a conversion disorder. Because of the disturbances, the patient left his job and moved to live with his sister since he was not able to care for himself. After 6 months, the patient was submitted to a neuropsychological screening, showing an impairment of executive functions and amnesia. A diagnosis of frontotemporal dementia was supposed. The patient was referred to our Center in January 2011. The neurological examination was normal. Neuropsychological tests confirmed an alteration of executive functions as well as verbal and visual memory deficits. This was coupled with behavioral disturbances of apathy and depression. Brain MRI showed a mild frontotemporal atrophy with ventricular dilatation but also a left thalamic infarct in the paramedian territory (Fig. 1). The patient started therapy with paroxetine (20 mg/day) and memantine (10 mg/day). At 6-month and 1-year, follow-up neuropsychological test showed a further recovery of cognitive functions and behavioral disturbances (Table 1). The caregivers also reported a gradual improvement of disturbances since the onset. Brain CT confirmed thalamic infarction. We made a diagnosis of a thalamic stroke as the cause of dementia, because of the sudden onset and the gradual improvement of the clinical syndrome and the correspondent finding of an ischemic lesion. Routine blood tests, hemocoagulative screening and autoantibody pattern were normal. Electrocardiogram and carotid ultrasonography did not show any alterations. The transthoracic echocardiogram, the transcranial Doppler sonography and the transoesophageal echocardiography detected the patent foramen ovale (PFO) and the patient was started anticoagulant therapy. After 2 years, the

S. Realmuto (✉) · V. Arnao · A. Cinturino · M. A. Mazzola · S. Talamanca · M. Riolo · I. Cusmano · C. Cupidi · T. Piccoli
Dipartimento di Biomedicina Sperimentale e Neuroscienze Cliniche (BioNeC), Università degli Studi di Palermo, Via Gaetano La Loggia no 1, Palermo, Italy
e-mail: sabrinarealmuto@gmail.com

C. Cupidi
Centro Regionale di Neurogenetica, ASP Catanzaro, Via Arturo Perugini, Lamezia Terme, Italy

T. Piccoli
Department of Cognitive Neuroscience, Maastricht University, Maastricht, The Netherlands

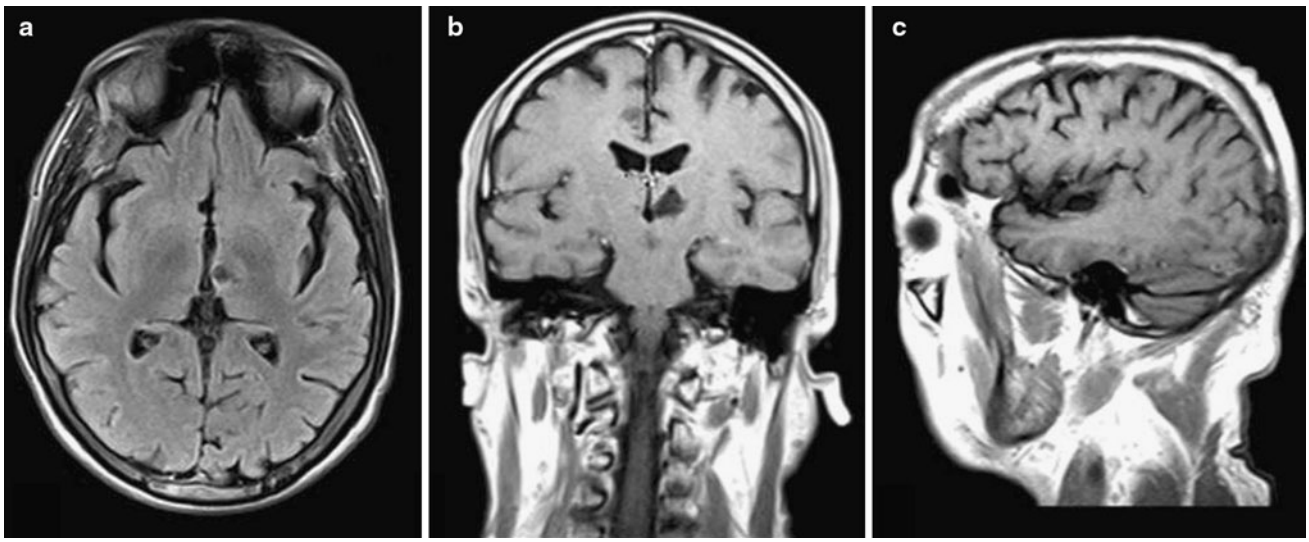

Fig. 1 Brain MRI: axial (**a**) fluid attenuated inversion recovery (FLAIR) and coronal (**b**) T1-weighted scans showed the ischemic lesion in the left paramedian thalamic nuclei; sagittal (**c**) T1-weighted image showed mild frontotemporal atrophy

Table 1 Raw score (adjusted score) of neuropsychological tests at basal examination (T0), at 6 months (T1) and at one-year follow-up (T2)

Test	T0	T1	T2	Cut-off
Mini mental state examination	22 (21.7)	27 (26.7)	25 (24.7)	≤24
Frontal assessment battery (FAB)	12 (13.41)	15 (16.41)	15 (16.41)	≤12
Trail making test A	110 (97)	90 (77)	87 (74)	>94
Trail making test B	235 (187)	220 (172)	216 (168)	>283
Visual search	51 (46.5)	44 (39.5)	45 (40.5)	≤31
Digit span forward	4 (4.5)	4 (4.5)	6 (6.5)	≤3.75
Corsi span	4 (4.25)	4 (4.25)	4 (4.25)	≤3.25
Rey auditory verbal learning task immediate recall	14 (14.7)	23 (23.7)	18 (18.7)	≤28.53
Rey auditory verbal learning task delayed recall	0 (0)	1 (1.2)	0 (0)	≤4.69
Story recall test	2.5 (4)	2 (3.5)	4 (5.5)	≤4.75
Letter fluency	12 (19)	8 (15)	7 (14)	≤16
Category fluency	22 (28)	27 (33)	23 (29)	≤24
Raven's color progressive matrices	23 (23.8)	31 (31.8)	25 (25.8)	≤18.96
Rey's complex figure copy	33 (33.5)	29 (29.5)	34 (34.5)	≤28.87
Rey's complex figure recall	5 (4.25)	10 (9.25)	4 (3.25)	≤9.46
Constructive apraxia	13 (12.5)	13 (12.5)	13 (12.5)	≤7.75
Cognitive estimation test (bizarreness score)	18 (6)	15 (−)	11 (3)	≤19 (≤4)
Aachener Aphasie test-denomination	118/120	116/120	119/120	–
Aachener Aphasie test-oral comprehension	48/60	58/60	55/60	–
Neuropsychiatric inventory	14	5	6	–
Activity daily living (ADL)	6/6	6/6	6/6	–
Instrumental activities of daily living (IADL)	2/5	3/5	4/5	–

patient has gradually recovered autonomy in activities of daily living even if he remained apathetic.

Stroke in the paramedian territory of thalamus explains about 35 % of all infarcts in this region and involves mainly the dorsomedian and intralaminar nuclei [4]. Frontal-like syndromes secondary to paramedian strokes are probably due to thalamo-frontal disconnection and behavioral changes consisting of personality abnormalities with disinhibited behavior associated with apathy and amnesia were reported [3]. Amnesia is also a frequent

sign after paramedian infarcts, but the role of the intralaminar and dorsomedial nuclei is still controversial [5, 6]. Subcortical structures are anatomically and functionally connected with frontal and temporal cortices and damage to those regions often causes frontal lobe dysfunctions. Thalamic vascular lesion should be looked for in differential diagnosis of bvFTD also in patients without apparent vascular risk factors. The reported patient may be diagnosed as cryptogenic stroke (CS), probably caused by PFO. The prevalence of PFO is nearly double in patients with CS with respect to the general population and the infarct has generally been attributed to a paradoxical embolism [7]. The accurate diagnosis of the vascular origin of frontal dementia in this case has had relevant clinical implications in terms of prognosis and pharmacological treatment.

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