

Neuropsychological deficits after bithalamic hemorrhages

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Abstract

Strategic lesions of the thalamus interfere with cognitive functions and produce complex neuropsychological symptoms. Bilateral, simultaneous thalamic hemorrhages are unusual causes of thalamic dementia.

We present clinical, neuropsychological and structural neuroimaging data of a 12-month follow-up period of a patient with bilateral thalamic hemorrhages.

After the operation of pancreatitis acuta hemorrhagico-necroticans, the patient developed coma. Computed tomography (CT) and magnetic resonance (MR) of the brain showed medially situated bithalamic hematomas. During the follow-up period, patient's level of consciousness has improved. Moderate dementia (MMSE 20/30) was found with severe temporal and spatial disorientation. Neuropsychological tests showed that attention and concentration were prominently impaired; there were severe verbal and less prominent, visual memory deficits, with anterograde and retrograde amnesia, accompanied by confabulations. Loss of cognitive flexibility and dysexecutive syndrome were also demonstrated. Dynamic apraxia, visual organization and visual construction deficit and impairment of categorial and phonemic fluency were noted. Language was only moderately impaired (anomia).

A year later, neuropsychological profile was similar with moderate improvement of retrograde amnesia, whereas anterograde deficits persisted.

Neuropsychological syndrome in our patient with bilateral thalamic hemorrhages was characteristic for subcortico-cortical cognitive deficit and was caused by disruption of the cortico-thalamic circuitry.

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1. Introduction

Strategic lesions of the thalamus due to infarction or hemorrhage may cause acute or persistent complex neuropsychological dysfunction and may lead to vascular dementia. The most prominent cognitive deficit and one of the most extensively studied is memory dysfunction [1]. Aphasia, neglect and anosognosia were also frequently reported [1–3].

Bilateral, simultaneous, symmetrical thalamic hemorrhages are unusual causes of thalamic dementia. Kumral and colleagues [4] studied 100 patients with thalamic hemorrhage, but none of them have had bilateral hemorrhages. Bilateral hemorrhages are mostly a consequence of systematic diseases and conditions.

We present clinical, neuropsychological and structural neuroimaging data of the 12-month follow-up period of a man with bilateral thalamic hemorrhages.

2. Case report

Sixty-year-old, right-handed, Serbian speaking civil engineer (16 years of education) was admitted to Emergency Unit suffering from pancreatitis acuta hemorrhagico-necroticans. After surgery, the patient developed right hemiparesis and coma (Glasgow Coma Scale 5). Computed tomography (CT) and magnetic resonance (MR) of the brain showed medially situated bithalamic hematomas with rupture into the third ventricle. MR angiography was normal. After 2 weeks level of consciousness had improved, but the patient remained indifferent, with loss of initiative. He had retrograde amnesia for 20 years, he could not recognize his wife and son. Also he had prominent anterograde amnesia. The patient did not show any interest and manifested

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no concern about the future. Right situated sensory deficit and moderate hemiparesis were observed.

The following neuropsychological tests were administered 1 month after the onset: Mini Mental State Examination (MMSE), Wechsler Adult Intelligence Scale-Revised (WAIS-R), Wechsler Memory Scale-Revised (WMS-R), Trail Making Test (TMT), Rey Auditory–Verbal Learning Test (RAVLT), Rey–Osterrieth Complex Figure Test (ROCFT), Wisconsin Card Sorting Test (WCST), Categorial and Verbal Fluency tests, Boston Diagnostic Aphasia Examination (BDAE), Boston Naming test (BNT), Hooper Visual Organization Test (HVOT), Clinical Tests for Praxia, Gnosia and Manual Dexterity.

Moderate dementia (MMSE 20/30) was found with severe temporal 0/5 and spacial 3/5 disorientation, and impaired short-time memory 0/3. Neuropsychological tests showed that attention and concentration were prominently impaired (WMS-R attention score 74), there were severe verbal long-term memory deficits (RAVLT scores were more than two standard deviations below standard for general population, with impaired verbal learning, evocation and recognition, accompanied by confabulations) and visual long-term memory deficits (ROCFT score was more than two standard deviations below standard for general population). General slowing of mental processing speed and loss of cognitive flexibility and dysexecutive syndrome (WCST categories 1, with perseverations) were demonstrated as well. Clearly reduced verbal fluency with impairment of categorial (animals 6) and phonemic fluency (S, K, L) were noted. Dynamic apraxia, visual organization and visual construction deficits were less prominent. Language was only moderately impaired with anomia (BNT scores were more than two standard deviations below standard for general population, with only 4 verbal phonemic paraphasic errors), with normal repetition, comprehension and fluency.

One year later, neuropsychological retests were administered. Neuropsychological profile was similar, anterograde memory deficits persisted, while retrograde amnesia showed moderate improvement. Two brain MRI studies were performed during follow-up period (after 1 month and after 6 months) showing bilateral lesions of the medially situated thalamic nucleus.

3. Discussion

Neuropsychological deficits due to bilateral thalamic infarctions had been reported previously [5,6]. Bilateral thalamic hemorrhages are very rare and neuropsychological outcome has not been studied yet. We present a patient with pancreatitis acuta hemorrhagico-necroticans, with developed bilateral, simultaneous, medial thalamic hemorrhages secondary to severe coagulopathy.

At the onset of stroke, coma was observed due to disturbance of the arousal system [4].

After 2 weeks, the patient's level of consciousness had improved, but he remained indifferent, with loss of initiative.

Persistent personality changes included apathy, lack of insight and lack of spontaneity. The patient did not show any interest and manifested no concern about the future. Similar behavioural manifestations had been reported previously with thalamic lesions, suggesting frontal lobe dysfunction (caused by disconnection of executive frontal circuits) [7]. The patient of Clarke and colleagues with thalamic infarction showed impaired emotional engagement with hypometabolism in the posterior cingulate cortex measures with positron emission tomography [8]. Behavioural changes were attributed to interruption of series of frontal–subcortical circuits.

Our patient had severe retrograde and anterograde amnesia, accompanied by confabulations. Neuropsychological tests showed that there were severe verbal long-term and visual long-term memory deficits as well. One year later, neuropsychological retests showed that anterograde memory deficits persisted, while retrograde amnesia showed moderate improvement. Diencephalic global amnesia is characterized by severe anterograde memory deficits. Retrograde amnesia tends to improve with time, whereas anterograde deficit persists [9]. Global amnesia after thalamic stroke mostly requires bilateral lesions [10]. Mediodorsal thalamic nucleus is a likely candidate to cause this type of amnesia “per se” [11], or because of its connections to cortical areas and to the hippocampal formation [12–14]. In recent years, the importance of major fiber tracts that connect thalamus to medial temporal lobe structures to mnemonic function is stressed [10]. Bleeding in thalamus caused hypoperfusion in the adjacent ipsilateral fronto-temporal deep temporal and insular cortex in patients who had SPECT [5,15,16]. Hemorrhage is seldom confined to the thalamus itself, however, and remote effects include pressure of the hemorrhagic mass and effect of surrounding edema on adjacent structures [6].

Neuropsychological tests showed that attention and concentration were prominently impaired. Loss of cognitive flexibility and dysexecutive syndrome were also demonstrated. Impairment of categorial and phonemic fluency and dynamic apraxia were noted. Frontal lobe dysfunction caused by disconnection of executive frontal circuits has been suggested as origin of these deficits [7]. Visual organization and visual construction deficits with fragmentary perception and damaged copy strategy also noted in our patient could be explained by right situated frontal lobe dysfunction [7].

The mechanism of thalamic aphasias is still controversial. Our patient had only moderately impaired language with anomia with preserved repetition, comprehension and fluency. Language disturbance after left sited thalamus lesion were reported by many authors suggesting that thalamus an important role in organization of language [17–21]. Other authors considered aphasia after thalamic stroke consequence of mass effect leading to direct compression of the cortex or compromise of the vascular supply to the cortex [22], diaschisis [23] or disconnection [24].

Strategic lesions of the thalamus due to infarction or hemorrhage may cause acute or persistent complex neuropsychological dysfunction [25,26]. Although it is not definitely confirmed, we do agree with the theory that this type of subcortical dementia is caused by disruption of the thalamo-cortical circuits.

4. Conclusion

Neuropsychological syndrome in our patient with bilateral thalamic hemorrhages was characteristic for subcortico-cortical cognitive deficit and could be caused by disruption of the cortico-thalamic circuits.

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