Bilateral hippocampal infarction and amnesia: A case report

Bilateralni hipokampalni infarkt i amnezija

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Abstract

Introduction. The hippocampus along with other structures of the medial temporal lobe plays an important role in the process of learning and memory consolidation. Bilateral hippocampal lesions lead to persistent anterograde amnesia while unilateral damage results in milder, content-specific forms of amnesia. Hippocampus may be affected by an acute or chronic pathologic process from a wide spectrum of neurological disorders. **Case report.** A 61-year-old female patient with a long history of hypertension, glucose intolerance, hypercholesterolemia and depression was hospitalized for acute anterograde amnesia, which led to repeated excessive drug taking. By further examinations that included laboratory tests, electroencephalography, Doppler sonography of intra- and extracranial vessels and neurovisualization methods [multislice computed tomography (MSCT) and magnetic resonance imaging (MRI) of the brain] bilateral hippocampal ischemia that occurred at different times was detected. Cognitive and neuropsychological evaluation revealed an isolated severe damage of episodic memory with the inability of retention of new information which persisted at the control examination three months later. The assumed mechanism of occurrence of ischemia in this case could be arterio-arterial embolism. **Conclusion.** Although ischemic stroke is one of the most common neurological diseases, ischemic stroke of the hippocampus is rare, the isolated bilateral presentation with clinical signs of severe amnestic syndrome in particular. Timely recognition and modern therapeutic approach could have a favorable impact on the recovery from severe neurological, cognitive deficit. It could be suggested that in patients with the clinical image of acute anterograde amnesia and vascular risk factors the MSCT examination of the brain with computed tomography perfusion and angiography is performed immediately upon hospitalization.

Apstrakt

Uvod. Hipokampus zajedno sa ostalim strukturama medijalnog temporalnog režnja ima značajnu ulogu u procesu učenja i konsolidacije pamćenja. Bilateralne lezije hipokampusa dovode do perzistentne anterogradne amnezije dok unilateralno oštećenje rezultuje blažim, sadržajno određenim oblicima amnezije. Hipokampus može biti pogođen akutnim i hroničnim patološkim procesom u sklopu širokog spektra neuroloških oboljenja. **Prikaz bolesnika.** Bolesnica, stara 61 godinu, sa istorijom dugogodišnje arterijske hipertenzije, intolerancije glukoze, hiperholesterolemije i depresije, hospitalizovana je zbog akutne anterogradne amnezije koja je dovela do ponavljanog prekomenog konzumiranja lekova. Daljim ispitivanjima koja su uključila laboratorijska ispitivanja, elektroencefalografiju, doplersonografiju intra- i ekstrakranijalnih krvnih sudova, neurovizuelizacione tehnike [multislajsnu kompjuterizovanu tomografiju (MSCT) i magnetsku rezonancu (MR) mozga] ustanovljena je bilateralna ishemija hipokampusa različitog vremena nastanka. Kognitivno i neuropsihološko ocenjivanje pokazalo je izolovano teško oštećenje epizodičnog pamćenja, sa nemogućnošću pamćenja novih informacija koje se održavalo i nakon tromesečne kontrole. Predpostavljeni mehanizam nastanka ishemije u ovom slučaju mogao bi biti arterijsko-arterijski embolizam. **Zaključak.** Iako je ishemički moždani udar jedna od najčešćih neuroloških bolesti, ishemički infarkt hipokampusa je redak, posebno izolovana bilateralna prezentacija sa kliničkom slikom teškog amnestičkog sindroma. Blagovremeno prepoznavanje i savremeni terapijski pristup bi mogli uticati povoljno na oporavak teškog neurološkog, kognitivnog deficita. Izdvaja se sugestija da se kod bolesnice sa ovakvom kliničkom slikom akutne anterogradne amnezije i vaskularnim faktorima rizika odmah pri prijemu uradi MSCT pregled mozga, po mogućstvu za CT perfuzijom i angiografijom.

Key words:
hippocampus; brain infarction; amnesia, anterograde; magnetic resonance imaging.

Ključne reči:
hipokampus; mozak, infarkt; amnezija, anterogradna; magnetna rezonanca, snimanje.

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Introduction

The role of the hippocampus (HC) in the process of learning and memory consolidation is important and undeniable. In conjunction with the surrounding limbic system structures the hippocampus participates in the creation of emotional behavior, endocrine stress regulation and has the potential for adult neurogenesis [1].

The HC is located in the limbic gyrus on the medial surface of the cerebral hemispheres and together with the amygdaloid complex, the surrounding entorhinal, perirhinal and parahippocampal cortex, with which it creates multiple connections, makes the medial temporal lobe (MTL) [2].

A key shift in understanding the role of the hippocampus happened with the case of Henry Molaison. In 1953, in an attempt to cure severe epilepsy, a Canadian neurosurgeon Scoville surgically removed anterior two-thirds of both temporal lobes: apical part of the temporal lobe, uncus and amygdala, *formatio hippocampi* and adjacent part of the parahippocampal gyrus [3].

The patient was cured of epilepsy but later it was established that he suffered from a form of memory impairment, anterograde amnesia, which was widely studied over the next 30 years on the case of this patient. He lost the ability to retain information from the recent past, immediately before and after the surgery, while information from the distant past were preserved, even more if they were more distant. The patient's short-term memory, cognitive and other intellectual abilities was mostly preserved [4]. In this case, as well as in many other ones, it was shown that the severe forms of amnesia are connected to bilateral hippocampal lesions, whereas unilateral damage results in milder, mostly content-specific determined forms of amnesia.

The hippocampus can be affected by acute and chronic pathological processes in a wide spectrum of neurological disorders.

vided, in greater or lesser extent, by the branch of anterior choroidal artery [8].

In the available medical literature we can find a scarce number of cases of hippocampal infarction, especially those that include its clinical presentation and magnetic resonance imaging (MRI) features [9–11].

In this paper we presented a patient with bilateral ischemic infarction of the hippocampus and a type of memory disorder. It is an extremely rare case in terms of stroke localization, as well as the etiology of bilateral hippocampal damage.

Case report

A 61-year-old female patient with the years-long medical history of hypertension, glucose intolerance, chronic thyroiditis, and depression, treated for two years, was admitted to the toxicology outpatient clinic in the emergency center of the Military Medical Academy for suspected acute self-poisoning with drugs.

The patient's family said that the previous evening she took a large number of tablets of propranolol, repeatedly, and that the next day she took more drugs uncritically. It was noted that she "acted strangely", she seemed confused and repeated the same questions and actions over and over again.

During examination the patient was conscious, afebrile, slightly disoriented in time and space, with slow heart rate, 50/min, and 96% oxygen saturation, measured using a pulse oximeter. Blood pressure was 175/80 mmHg with normal physical findings. Electrocardiogram (ECG) showed sinus rhythm, frequency 49/min, intermediate axis, QTc 422 ms with no changes in ST and T segment.

Laboratory analyses are shown in Table 1.

Clinical image and toxicological and chemical analysis on admission revealed overdose with drugs from the group of

Table 1

The values of laboratory and toxicological parameters that deviated from the normal

Parameters	Initially	Control	Normal
Glucose (mmol/L)	9.8	6.9	4.1–5.9
Sodium (mmol/L)	136	144	136–145
Potassium (mmol/L)	3.5	4.1	3.5–5.1
Cholesterol (mmol/L)	–	6.4	< 5.2
LDL holesterol (mmol/L)L	–	4.64	< 3.5
AST (U/L)	71	18	0–37
ALT (U/L)	150	25	10–49
Propranolol (mg/L)	0.04		0.02–0.3
Bromazepam (mg/L)	0.07		0.05–0.2
Fluoxetine (mg/L)	0.23		0.12–0.5

LDL – low density lipoprotein; AST – aspartat aminotransferase; ALT – alanine aminotrasferase.

Although acute ischemic cerebral infarction is one of the most common neurological diseases, ischemic stroke of the hippocampus is rare [5, 6]. This phenomenon is partly explained by less frequent rate of ischemic brain infarction in the posterior cerebral circulation, i.e. 15% of the total number. Isolated hippocampal infarction which does not involve the existence of ischemia in other areas of the posterior circulation is particularly rare [6, 7]. Vascularisation of the hippocampus is mainly provided by the posterior cerebral artery (PCA) through its branches, anterior, middle and posterior hippocampal arteries. Vascularisation of the front part, the head of the hippocampus, is also pro-

beta blockers, anxiolytics and serotonin reuptake inhibitors which was manifested in bradycardia, mild confusion and disorientation.

The patient was hospitalized for the application of nonspecific detoxication treatment, which led to normalization of the heart rate and toxicological parameters. During the treatment at the Clinic of Toxicology (National Poison Control Centre in the Military Medical Academy, Belgrade), the mild confusion was present in the form of constant repetition of the same questions as well as the temporal and spatial disorientation.

Neurological consultative examination showed a discrete pyramidal deficit of the right side and uncertainty regarding the events connected to the circumstances of admission to the hospital. Multislice computed tomography (MSCT) of the brain was indicated. It showed bilateral ischemic lesions of the hippocampus and parahippocampal gyrus. Left lesion was somewhat older, 50×20 mm in diameter, and right lesion in the subacute phase was 45×18 mm in diameter (Figure 1).
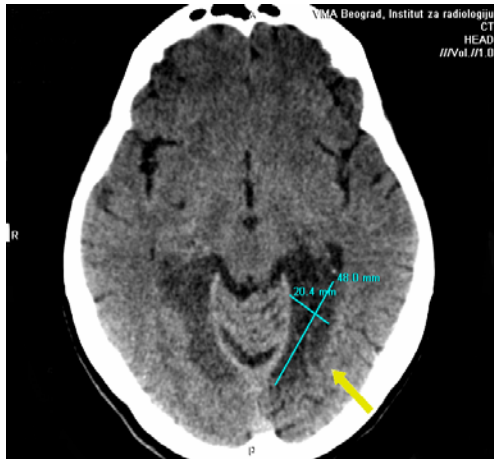

Fig. 1 – Axial noncontrast multislice computed tomography (MSCT) brain scan: bilateral areas of hypodensity in the hippocampus and parahippocampal gyrus demonstrate ishemic lesions. Left in the chronic, right in the subacute phase.

The patient was transferred to the Clinic of Neurology, Military Medical Academy.

The interviews with the patient revealed spatial and temporal disorientation regarding the date and the name of the hospital to which she was admitted. The patient was communicative, in normal mood, found filling crosswords in her spare time. She provided autobiographical data adequately, but she had no recollection of the events that occurred on the day of admission to the hospital, or the events which took place during her stay at the Clinic of Toxicology. A period of 3–4 days prior to the onset of the symptoms was also not available to the memory. National Institutes of Health Stroke Scale (NHSS) score was 2.

Control laboratory tests showed marginally elevated levels of serum glucose, cholesterol and LDL cholesterol, and normal HbA1c, coagulation factors, prothrombin time, C reactive protein, homocysteine, tumor markers, thyroid hormones and serum enzymes values. Ophthalmic examination and chest X-ray were normal. Cardiac examination confirmed arterial hypertension, which was corrected by antihypertensive therapy. Echocardiographic findings were normal, except the mild mitral regurgitation. Atrial fibrillation was excluded by holter ECG monitoring.

Electroencephalography (EEG) examination showed no presence of epileptic activity.

Doppler sonography of blood vessels of the neck showed the presence of fibrolipid plaques bilaterally in the region of bifurcation of common carotid arteries, with lumen reduction of about 50% in the first part of the right carotid artery. Left vertebral artery had a narrower lumen with reduced flow, with the characteristics of high resistance (RI 0.93).

Transcranial Doppler of cerebral blood vessels indicated high resistance in the vertebrobasilar territory. In all three blood vessels Doppler signal had a flattened tip and pulsatility indexes were above expected. Left anterior carotid artery (ACA) showed significantly higher flow rate, which indirectly suggested stenosis.

Contrast Transcranial Doppler (TCD) did not register the existence of spontaneous microemboli or the signs of right-to-left shunt.

MRI of the brain, performed on the sixth day after the onset of the symptoms, showed the area of hyperintensity in T2 fluid-attenuated inversion recovery (FLAIR) sequences with hypointensity in T1 sequences and signs of diffusion restrictions on diffusion weighted imaging (DWI) sequence in the region of the right hippocampus and parahippocampus, which corresponded to ischemia in the subacute phase. The signs of petechial hemorrhage were present in the specified zone cortically. Contralaterally in the same region, more extensive in posterior, a zone of encephalomalacia with hemosiderin deposits was observed representing the sequelae of chronic infarction lesion with hemorrhagic transformation. Multiple lacunar ischemic lesions were revealed within the deep cerebral white matter and subcortically bilaterally fronto-parietally (Figures 2a, 2b).

Angiography on a 3D (time of flight magnetic resonance angiography – TOF MRA) did not register the signal in the in-
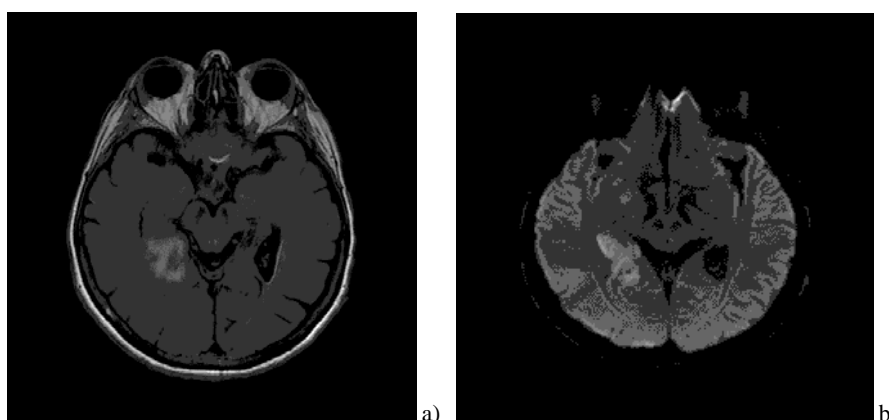

Fig. 2 – a) T2 fluid-attenuated inversion recovery (FLAIR) shows hyperintensity of the signal in the region of the hippocampus and parahippocampally on the right. On the left side hypointensity in the same region; b) DWI shows diffusion restriction in the hippocampus and parahippocampal gyrus on the right.

tracranial segment of the left vertebral artery, which corresponded to occlusion. In the right vertebral artery, at the transition from extracranial to intracranial segment atherosclerotic plaques could be seen, which caused stenosis of about 50%. Basilar artery with its branches was displayed normally. As an incidental finding, the existence of two aneurysms in the medial ophthalmic segment of the right *arteria cortis interna* (ACI), small in dimensions, was observed (Figure 3).
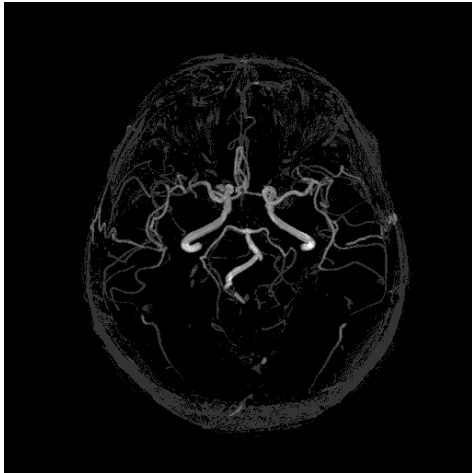

Fig. 3 –Angiography does not visualize the left AVwhile the right one is stenosed at the transition from the extra to intracranial AV segment.

Cognitive evaluation, performed on the fifth day after the onset of the symptoms, showed normal attention span and working memory, preserved vocabulary of previously acquired knowledge, preserved semantic memory, normal speech in expressive and receptive sense, preserved praxis, structural and visuospatial capability. There was mild to moderate executive dysfunction in terms of disrupted initiation and set shifting, as well as slightly reduced information processing speed. Severe anterograde amnesic syndrome, related to the domain of episodic memory, dominated. The ability of immediate memory was preserved within the average. In the area of declarative memory, verbal and visual learning severe deficit was registered, along with the inability to retain information. The patient was unable to recall any of the previously presented information. The effect of reminders was insufficient and recognition was disturbed.

The manifested form of cognitive impairment, with predominant and relatively limited focus on the syndrome of anterograde amnesia, suggested bilateral mediotemporal localization of the pathological process. Executive dysfunction and mild slowing of the information processing speed could be explained by chronic subcortical white matter vascular lesions with frontal distribution.

Cognitive examination was repeated after three months, revealing cognitive deficit maintained at the same intensity and the same pattern.

On cognitive screening test errors were registered predominantly in delayed recall tasks and phonemic fluency at both the initial and the control examination. Uncertainty regarding time and space orientation was more pronounced at the initial examination and to a lesser extent at the control examination. Detailed neuropsychological testing was then focused specifically on the domain of memory, executive functions and speed of information processing. The results of tests are shown in detail in Table 2.

Table 2

Cognitive screening and neuropsychological tests scores

Cognitive test	Initial evaluation	3-month follow up	Normative values (SD)
Screening test baterries			
MMSE	23	25	26.1 (3.1)
MoCA	20	22	27.4 (2.2)
Memory tests			
DSF	6	6	6 ± 1
DSB	5	5	5 ± 1
SRT			
SRT LTS	6	2	33.54 (18.30)
SRT CLTR	0	0	27.25 (21.61)
SRT DR	0	0	6.25 (2.80)
BVMT-R			
Trial 1	4	4	5.38 (2.2)
Trial 2	4	5	7.93 (2.07)
Trial 3	0**	2**	9.94 (2.00)
Total recall	8**	10**	22.35 (5.44)
Delayed Recall	0**	0**	8.48 (2.17)
Learning	0**	0**	3.76 (1.83)
Percent retained	0**	0**	90.66 (9.54)
Executive functions tests			
TMT A	52*	48*	33.22 (9.10)
TMT B	120*	119*	74.55 (19.55)
VFT	33	35	< 30
SDMT	43	47	37.71 (11.51)

MMSE – Minimental state examination [12]. MoCA – Montreal cognitive assessment [13]. DSF – Digit span forward; DSB – Digit span backward working memory; immediate memory [14]. SRT – Selective reminding test, version of 6 attempts, for estimate of verbal learning and memory [15, 16]. LTS – Long-term storage;CLTR – Consistent long-term retrievel; DR – Deley recall. BVMT-R – The brief visuospatial memory test – revised, for the evaluation of visuospatial learning [17]. TMT A, TMT B – Trial making test A and B provides information on visual search, scanning, speed of processing, mental flexibility, and executive functions [18]. VFT – Verbal fluency test executive functions [17]. SDMT – Symbol digit modalities test – speed of information processing [17].
* Scores that indicate mild to moderate damage
** Scores that indicate severe damage > than 3 standard deviation (SD).

The patient was treated with statins, antithrombotic, and antihypertensive therapy. An antidepressant and a small dose of an anxiolytic were reintroduced with the advice regarding taking of medication under supervision. Cognitive rehabilitation therapy was recommended.

Discussion

The paper by Szabo et al. [6] analized the patients with ischemic stroke in the vascular territory of the PCA, which accounted for 5.25% of all ischemic strokes. The hippocampus was affected in 21% of the cases, most commonly the lesion of one hippocampus, while only 5% of the patients suffered bilateral hippocampal infarction.

In all the cases of hippocampal ischemia extrahippocampal regions in the ipsilateral vascular territory of the PCA were affected, as the well as thalamopeduncular region, occipital lobe and lesions of the cerebellum. There were no isolated hippocampal lesions in any of the cases [19].

In the case of the presented patient a bilateral lesion of the hippocampus was identified. Ischemias occurred at different times. The left hippocampus, where the described ischemia was in a chronic phase, was affected first and afterwards the right hippocampus with subacute ischemia. In both cases the lesions appeared mainly in dorsal part of the hippocampus and the parahippocampal region. They were isolated, without other evident lesions in the posterior cerebral circulation outside the hippocampus.

This model of ischemia could be explained by distal vascular pathology, primarily by occlusion of the middle or posterior hippocampal artery, branches of the P2 segment of the PCA.

The pathogenetic mechanism of ischemia occurrence in the posterior circulation, according to the findings in studies, was mainly associated with cardiogenic embolism, arterio-arterial embolisation due to atherosclerosis of large blood vessels, the basilar and vertebral arteries [20, 21]. More recent findings, based on the studies that have used modern MRI techniques (angiography, DWI) suggest large-artery atherosclerosis as a leading cause, whereby three different scenarios can be distinguished: *in situ* atherothrombosis, arterio-arterial embolism and consequent occlusion of the ostium of perforating branches on the ground of the atherosclerosis of a larger arterial tree [5].

The estimated, more likely the mechanism of ischemia in the case of the presented patient, considering the existing occlusion of the left and the stenosis of the right AV, could be arterio-arterial embolism with remote ischemic lesion in the vascular region of the middle and posterior hippocampal artery, first on the left and then on the right. This is supported by the presence of hemosiderin deposits in the area of ischemia, caused by hemorrhagic transformation, which is more common in the case of the embolic stroke. Another possible reason is the occlusion of orifice of middle and posterior hippocampal artery on the ground of the already existing atherosclerotic stenosis of the PCA, the segment P2. Bilateral presentation in the latter case could be caused by anatomic variation of the arterial branching where the branches of both hippocampuses originate from the one arterial tree [22].

Differentially diagnosed isolated lesions of the hippocampus are often associated with another type of pathology such as herpes simplex, paraneoplastic limbic encephalitis, complex partial seizures, transient global amnesia and brain tumors [23, 24].

Ischemic etiology of hippocampal damage in the presented case is supported by the existence of vascular risk factors and the presence of atherosclerosis of extracranial and intracranial vessels.

A sudden outset of symptoms, the absence of clinical, laboratory signs of infection and the characteristic presentation of lesions in DWI and T2 FLAIR MRI sequences made the differential diagnosis of encephalitis unlikely. EEG examination showed normal brain activity during the existence of anterograde amnestic syndrome, which also excludes nonconvulsive status *epilepticus* as a potential cause.

Regarding the cognitive deficit, cases of persistent anterograde amnesia as sequelae of ischemic damage of the hippocampus were described in the literature and it was confirmed in the presented case.

In 1900 Bechterew described a patient with the characteristic form of amnesia. The post-mortem study revealed bilateral hippocampal infarction [25].

Later, in similar cases which were neuropsychologically analyzed in more detail, it was observed that unilateral infarctions in the vascular territory of PCA which include the hippocampus result in a milder form of amnesia compared to the bilateral, both in terms of duration as well as the degree and the type of memory damage [10, 11, 26]. Lesions of the left hippocampus are connected to difficulties in verbal content memorizing, while in the case of the right sided lesion remembering of visual content, orientation, spatial relationships and faces was more affected [6, 27]. These conclusions correlate to findings that emerged in the era of excessive surgery of severe epilepsy, at the beginning of the last century. They opened a new chapter in the understanding of the memory processes and the role of the hippocampus and the structures of medial temporal lobe.

Ischemia of the left hippocampus that occurred earlier in the presented patient did not lead to noticeable impairment of memory and everyday activities. The image of profuse amnestic syndrome developed only after the occurrence of ischemia of the right hippocampus.

Researches in the field of memory, especially in the last years of the previous century, led to significant findings. First of all, the existence of various types of memory, mediated by different mechanisms and engaging of the various neural structures, were determined. Declarative or explicit memory, consisting of episodic and semantic memory, refers to the conscious recollection of events and facts and depends on the integrity of the hippocampus, the structures of the medial temporal lobe, diencephalon and the basal forebrain nuclei [28].

Nondeclarative or implicit memory is independent of the limbic system structures and temporal neocortex and involves knowledge and skills acquired on the unconscious

level during the lifetime. It is believed that in the process of the implicit memory, motor learning, the key role is played by the basal ganglia, posterior neocortex and the cerebellum. Hence, it is understandable that in patients with severe amnesia implicit memory remains intact [14, 29].

In case of amnesia manifested in the presented patient the damage of episodic memory was severe and isolated while the semantic and implicit memory were preserved. This fully correlates with the lesion localization.

The representative neural structures of semantic memory are located outside the hippocampus and medial temporal region. Recent evidence indicate to the anterior and lateral temporal neocortex, mainly the left, as a key integrative region [14].

Dissociation regarding the form of amnesia where, in the presented patient, dominated the anetrograde over the retrograde, was described in cases of ischemic lesions of the hippocampus. The degree of retrograde amnesia depends on the size of the lesion [29]. Preservation of the more distant memory is explained by the fact that the recall of older information is carried out by other structures too, independently of the hippocampus.

According to the standard model of memory consolidation the hippocampus is initially dominant in memory traces formation. With the prolongation of the consolidation process the neocortical component in the memory traces gradually strengthens and over time they become independent of the hippocampus and medial temporal lobe [30]. According to the model of multiple memory traces the hippocampus is the mediator in the process of creating a memory trace. There is no prolonged consolidation, but with each recall of previous information new hippocampal/medial temporal lobe (HC/MTL) memory traces are formed, thus achieving their stronger representation. Therefore, more distant memories are less susceptible to damage in comparison to the more recent ones [31, 32].

Conclusion

In neurological practice ischemic stroke is frequently seen. Ischemia in the posterior cerebral circulation is a less common and isolated bilateral hippocampal damage with clinical signs of severe amnestic syndrome is an unusual manifestation which may initially lead to dilemmas in the differential diagnosis. In addition, these infarcts are not well presented by National Institutes of Health Stroke Scale score. All that can affect timely implementation of the therapy, primarily thrombolytic therapy, in which early recanalization can lead to a favorable outcome in terms of recovery from this severe functional neurological deficit. Hence, it could be suggested that to patients with the clinical image of acute anterograde amnesia and vascular risk factors multislice computed tomography examination of the brain with computed tomography perfusion and angiography, or, if possible, magnetic resonance imaging of the brain according to the protocol for stroke should be performed immediately at admission.

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