

UDK 616.127-005.8-055.2  
COBISS.SR-ID 222032140

ISSN 0350-2899. - God. 41, br. 1 (2016), str. 33-35.

## **BILATERAL PARAMEDIAN THALAMIC INFARCTIONS IN A YOUNG WOMAN WITH PATENT FORAMEN OVALE**

### **BILATERALNI PARAMEDIJALNI TALAMIČKI INFARKTI KOD MLADE ŽENE SA PERZISTENTNIM FORAMENOM OVALE**

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**Sažetak:** Uvod: Bilateralni talamički infarkti su retki i obično udruženi sa tipičnom kliničkom slikom koja, pored ostalog, uključuje i neuropsihološke promene. Prikaz slučaja: Prikazan je slučaj tridesetsedmogodišnje žene sa akutno nastalim diplopijama usled „skew” devijacije, centralnom desnostranom parezom mimične muskulature, levostranom hemihipestezijom, ataksijom, sa očuvanim nivoom svesti i bez ikakvih neuropsiholoških smetnji, izuzev blagog memorijskog deficita. Postavljena je dijagnoza bilateralnog talamičkog infarkta uzrokovanog kardioembolizacijom preko perzistentnog foramena ovale. Zaključak: U slučajevima bilateralnog talamičkog infarkta može se pretpostaviti postojanje retke anatomske varijante talamičke perfuzije poznate kao Percheronova arterija, jedinstvenog stabla koje se grana za irigaciju oba paramedijalne talamičke zone. Uzrok infarkta može biti kardioembolizacija kroz perzistentni foramen ovale, naročito kod mladih ljudi. Naš slučaj prikazuje kombinaciju dva specifična patološka stanja – perzistentnog foramena ovale i bilateralnog talamičkog infarkta. Klinička prezentacija u ovom slučaju je atipična za bilateralni paramedijalni infarkt.

**Ključne reči:** bilateralni talamički infarkt, „skew” devijacija, perzistentni foramen ovale, Percheronova arterija.

**Summary:** Background: Simultaneous bilateral thalamic infarctions are rare and in most cases associated with typical clinical pattern which, beside other things, include neuropsychological changes. Case report: We report a case of a 37- year-old woman with acute onset of diplopia from skew deviation, right-sided central facial nerve palsy, left hemihypesthesia, ataxia, with normal level of consciousness and without any neuropsychological disturbances except minor memory deficit. She was diagnosed with bilateral thalamic infarction due to the cardioembolisation via patent foramen ovale. Conclusion: In cases of bilateral thalamic infarction one can presume the existence of rare anatomic variant of thalamic perfusion commonly known as the artery of Percheron, single artery trunk that branches to irrigate both paramedian territories of thalamus. The cause of infarction can be cardioembolism through the patent foramen ovale, especially in young adults. Our case represents a combination of two specific pathological conditions – patent foramen ovale and bilateral thalamic infarction. Clinical presentation in this case was unusual for the bithalamic paramedian infarction.

**Key words:** bilateral thalamic infarction, skew deviation, patent foramen ovale, artery of Percheron

#### INTRODUCTION

Simultaneous bilateral thalamic infarctions are rare, representing approximately 0.6% of all ischaemic strokes [1]. The most common pattern (75%) on neuroradiological images are bilateral infarcts in the territory of paramedian artery or combined with other thalamic artery territories. Most of the patient with bilateral paramedian infarction have specific clinical

presentation with disorder of consciousness, memory dysfunctions, various types of vertical gaze palsy and psychic changes. The main cause of bilateral thalamic infarction was small artery-disease, followed by cardioembolism [1].

#### CASE REPORT

A 37 year-old woman, previously healthy, during regular activities on job suddenly became aware

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Rad primljen: 05. 9. 2015. Rad prihvaćen: 08. 12. 2015. Elektronska verzija objavljena: 28. 3. 2016.

of visual disturbances, saw double pictures and was not able to see left side of the field. She was alert the whole time, but was complaining of instability, speaking problems and some kind of transient hearing problem. Ex-professional basketball player, smoker; without previous illness and other known risk factors. She strictly denied previous stroke or any similar problems. On admission she was alert, orientated, all vital signs were within normal limits, except slight hypertension. Neurological findings were skew deviation, without hemianopsia, central facial palsy on the right side with deviation of tongue to the right, left hemihypesthesia, and truncal ataxia. She had no manifest motor deficit except discrete subjective feeling of weakness of the left arm. Glasgow Coma Scale (GCS) score was 15, National Institutes of Health Stroke Scale (NIHSS) score was 7, and Mini Mental Score Examination (MMSE) was 28. In the next two days she developed discrete right hemiparesis. Standard laboratory tests, prothrombin time, partial thromboplastin time, D-dimer were within normal limits. An EKG showed normal sinus rhythm.

A computed tomography (CT) with angiography (CTA) of the brain was normal. Duplex ultrasound imaging of carotid arteries revealed nonsignificant bilateral stenosis. Transcranial Doppler (TCD) of vertebrobasilar arteries showed mild hemodynamic changes in right vertebral artery. TCD of the circle of Willis was normal. Magnetic resonance imaging (MRI) of the brain with angiography (MRA) showed an increase in signal in the thalami bilaterally on T2 fluid-attenuated inversion recovery (FLAIR) sequence with the signs of restricted diffusion on the left side but without restriction on the right side suggesting the bilateral thalamic infarction (Fig.1). Despite the fact that it was not seen on CTA and MRA, the existence of Percheron's artery was not excluded. MR venography of the brain excluded cerebral veins thrombosis.

TCD bubble test was positive and transesophageal echocardiography (TEE) confirmed patent foramen ovale (PFO). Ultrasound of deep veins of the legs showed no abnormalities. Further laboratory findings revealed mild hyperlipoproteinemia, decreased level of folic acid and borderline elevated homocysteinlevel. Genetic screening revealed that the patient is homozygous for A1298C mutation in methylenetetrahydrofolate

reductase (MTHFR) gene. Laboratory tests of vasculitic diseases were negative.

The patient continued to do extremely well while in hospital without any episodes of alteration in consciousness, with gradually recovery of symptoms. Neurological findings remained with only slight diplopia. Mood, behavior and cognition appeared intact with just minor memory deficit, MMSE 28-29. She was discharged home on clopidogrel, folic acid and simvastatin therapies 12 days after the stroke onset. She was recommended for percutaneous closure of PFO and it was performed six months after the stroke.

At a seven-month follow-up patient report there were no complaints, no visual disturbances, sensory or motor deficit. Psychological testing revealed minor memory deficit (MMSE 30).

#### DISCUSSION

Bilateral thalamic infarctions are rare and associated with typical clinical patterns [1]. When they occur in the presence of normal brain and neck CTA, one rare anatomic variant of thalamic perfusion could be considered - commonly known as the artery of Percheron, single artery that branches from proximal segment of one of the posterior cerebral artery and irrigates both paramedian territories of thalamus [2]. Several short-numbered series and isolated case reports have been published about bilateral paramedian thalamic infarcts, but just a few of them associated with PFO [3-6].

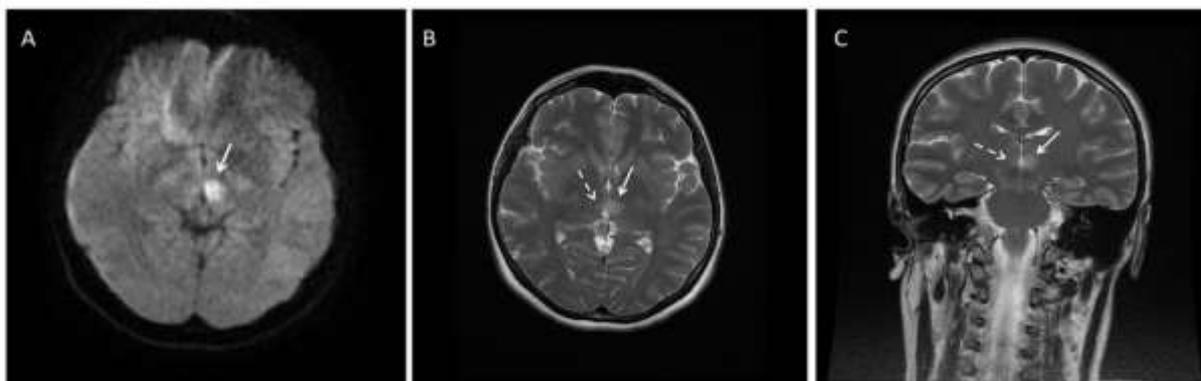
Patent foramen ovale occurs in up to 25% of the general population [7,8]. Several studies about association of PFO with ischaemic stroke in young people were made and they emphasized the fact that paradoxical embolism through a patent foramen ovale can be a possible cause of stroke in young adults [8-10]. Lechat et al found that PFO occurs in 40% of all young patients with stroke [8]. The same group of authors also found that in the group with no identifiable cause (cryptogenic stroke) which included 43 percent of cases, the most prevalent potential source of cardioembolism was patent foramen ovale (in 54%). Similar conclusions were obtained in a study by Webster et al (PFO was found in 50% of the stroke patients younger than 40 years) [10]. Study by Pezzini et al found a significant relation between cardioembolism and paramedian infarcts in young people [10].

Our case is interesting in the context of the combination of the two specific conditions -

paradoxical embolisation and presumed artery of Percheron. Clinical presentation was not usual for paramedian thalamic infarcts, the most prominent deficit was visual disturbance due to skew deviation. There was no consciousness deficit which is common for this type of stroke, nor personality or major cognitive changes. Although MRI of the brain did not show

restriction of diffusion on both paramedian areas, regarding clinical picture that revealed bilateral neurological deficit and anamnestic data of visual disturbances at a stroke onset without previous history of the disease, we are inclined to believe that this was simultaneous bilateral thalamic infarction.

Figure 1: A) Axial diffusion weighted MRI shows restriction of diffusion on the left thalamus (white arrow). B) Axial and C) coronal T2 weighted MRI shows the bilateral paramedian thalamic infarction, bigger one on the left side (solid white arrows) and small one on the right (dash white arrows).



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