

Karotid Arter Stentleme Sonrası Kontralateral Serebral Hiperperfüzyon Sendromu: Olgu Sunumu

Contralateral Cerebral Hyperperfusion Syndrome after Carotid Artery Stenting: Case Report

Serhan Yıldırım

Kocaeli Derince Eğitim ve Araştırma Hastanesi, Nöroloji Kliniği, Kocaeli, Türkiye

ÖZ

Üç hafta önce geçici iskemik atak geçiren 64 yaşında erkek hastada sağ karotid arter stentlendikten 2 saat sonra konuşma bozukluğu ile sağ kol ve bacakta güç kaybı gelişti. Çekilen beyin bilgisayarlı tomografide (BT) sol frontotemporal bölgede kontrast ekstrasvasyonu ve ödem izlendi. Kan basıncı 190/100 mmHg olan hastaya serebral hiperperfüzyon tanısı koyuldu. Beyin ödemi için mannitol, hipertansiyon için gliserol trinitrat infüzyonu yapıldı. Sonraki gün çekilen beyin BT'de kontrast ekstrasvasyonunun kaybolduğu ve ödemin gerilediği izlendi. Beşinci günde yapılan muayenede sadece hafif dizartrinin kaldığı izlendi.

Anahtar Kelimeler: hiperperfüzyon sendromu, karotid arter stentleme, karotis stenozu

ABSTRACT

64 year-old male patient with transient ischemic attack 3 weeks ago had speech disorder and weakness on right arm after 2 hours of right carotid artery stenting. Cerebral edema and contrast extravasation in left frontan and temporal lobes were seen in non-enhanced brain computerized tomography(CT). Patient's blood pressure was 190/100 mmHg. Patient was diagnosed as cerebral hyperperfusion syndrome. Mannitol infusion was given for cerebral edema. Blood pressure was decreased with glycerol trinitrate infusion. Contrast extravasation diasappeared and left frontal edema decreased in brain CT performed in the next day. There was only mild dysartria in neurological examination at 5th day.

Keywords: hyperperfusion syndrome, carotid artery stenting, carotid stenosis

İletişim / Correspondence:

Uzm. Dr. Serhan Yıldırım

Kocaeli Derince Eğitim ve Araştırma Hastanesi, Nöroloji Kliniği, Kocaeli, Türkiye

E-mail: serhan_yildirim@yahoo.com

Başvuru Tarihi: 02.12.2020

Kabul Tarihi: 06.05.2021

INTRODUCTION

Cerebral hyperperfusion syndrome (CHS) is a rare complication of carotid artery recanalization (1). This complication may occur after carotid endarterectomy (CEA) or carotid artery stenting (CAS) (2,3). Patients with CHS present with clinical findings such as headache, seizures and neurological deficit (hemiparesis, aphasia, loss of consciousness, etc). Most of the patients with CHS have mild clinical symptoms, while some patients may have severe and life-threatening symptoms. Diagnostic criterias include neurological findings after carotid recanalization with cerebral edema or haemorrhage on brain imaging, evidence of hyperperfusion on imaging studies (transcranial doppler or perfusion studies) or systolic blood pressure (SBP) >180 mmHg and exclusion of cerebral ischemia (4). Neurological and radiological findings of CHS after CAS or CEA appear in ipsilateral hemisphere. There isn't any data about CHS in only contralateral hemisphere without involvement of ipsilateral hemisphere. In here, we reported a patient with CHS in left cerebral hemisphere after stenting right internal carotid artery (ICA).

CASE

64 year-old male patient had a transient ischemic attack (TIA) with weakness in right arm 3 weeks before admission. He was treated with acetylsalicylic acid 100mg/day and klopidogrel 75mg/day after TIA. The patient didn't have any disease or drug usage in medical history. We spotted bilateral internal carotid artery stenosis (95% in right ICA origin, near-occlusion with thrombus in left ICA origin) in digital subtraction angiography (Figure 1-2). We decided to recanalize right ICA first due to thrombus over the lesion in left ICA. Right ICA was recanalized with predilatation balloon angioplasty (Abbott, USA, Viatract balloon 5x20mm), stenting (Medtronic, USA, Protege stent system 10-7x30mm). Distal protection device (Abbott, USA, Emboshield NAV6 embolic protection system) was used during the procedure (Figure 3-5). Intervention was finished without any complication. Patient had speech disorder and weakness on right arm after 2 hours of stenting. Motor aphasia and 4/5 muscle strength on right arm were spotted in neurological examination. Cerebral edema and contrast extravasation in left frontal lobe were

seen in non-enhanced brain computerized tomography (CT) (Figure 6). Patient's blood pressure was 190/100 mmHg. According to these findings, patient was diagnosed as CHS. Mannitol and glycerol trinitrate infusions were started to patient for cerebral edema and hypertension. Patient's blood pressure was decreased to 120/80 mmHg. Contrast extravasation disappeared and left frontal edema decreased in 24th hour brain CT (Figure 7). Patient was discharged at fifth day with mild dysarthria.

Figure 1: Right ICA



Figure 2: Left ICA



Figure 3 : Baloon angioplasty to right ICA



Figure 4: Right ICA after stenting



Figure 5 : Cerebral perfusion after stenting

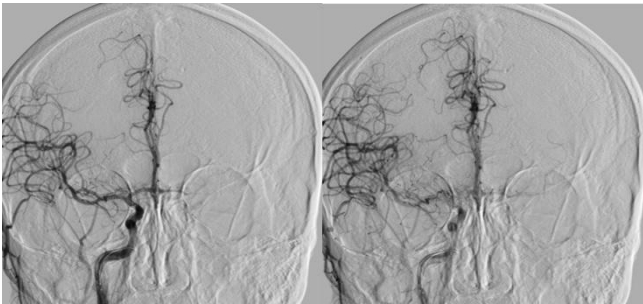


Figure 6: Brain CT after neurological worsening

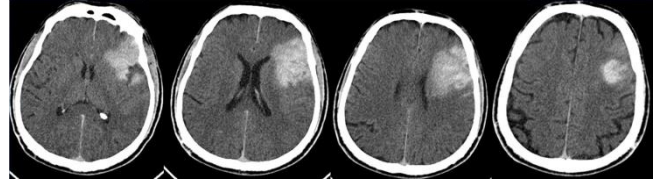


Figure 7: Brain CT at 24th hour

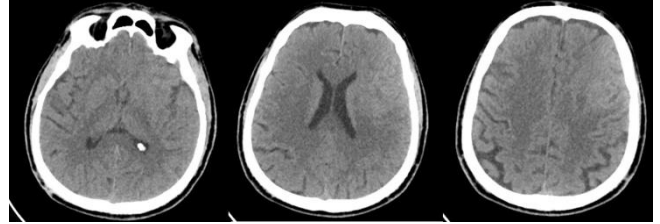
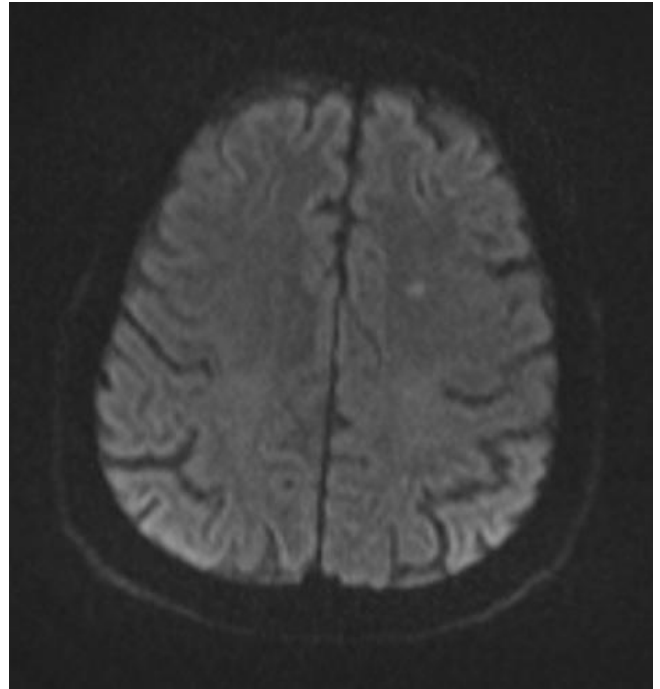


Figure 8: Lacunar infarction in diffusion MR performed 3 weeks ago



DISCUSSION

We reported a case with CHS in left cerebral hemisphere after stenting right ICA. Various studies found different CHS rates. In a review, Moulakakis et al reported incidence of CHS as 1,16% after carotid artery recanalization (5). In an other meta-analysis, CHS rate was found as 4,6% (6). In a prospective study, Abou-Chebl et al found CHS rate as 2,9% after CAS. Intracranial atherosclerotic stenosis >90%,

severe contralateral carotid stenosis and longstanding hypertension were found as three important factors increasing the risk of CHS after CAS (7). Additionally post-operative SBP over 150 mmHg increases the risk of CHS (8). Most accepted mechanism about CHS is impaired cerebral autoregulation. Cerebral autoregulation maintains the cerebral perfusion in acceptable range to blood flow and cerebral perfusion pressure changes. In chronic ischemic brain, arterioles and capillaries are vulnerable to bleeding after CAS due to increased cerebral perfusion pressure (1). Our patient had two of these factors (contralateral severe ICA stenosis and longstanding hypertension).

Clinical findings appear due to intracerebral haemorrhage (ICH) and vasogenic edema. Most common symptoms are headache (ipsilateral or bilateral), seizure, hemiparesis and loss of consciousness (4). In a meta-analysis, stroke occurred in 47% of CHS patients. Disability or death rate in CHS patients with stroke was found 54% (6). Appearance of clinical findings have peak within 12 hours after CAS (9). Our patient had motor aphasia and weakness on right arm occurred at second hour of CAS.

Intensive control of SBP decreases the risk of CHS after CAS. Postprocedural SBP should be <140/90 mmHg in CAS patients. SBP should be <120/80 mmHg in patients with high risk of CHS and ICH. High risk criteria were described as hypertension at baseline and treated carotid artery stenosis >90% (7). Blood pressure should be decreased in CHS syndrome. Labetalol or clonidine can be used for controlling the blood pressure, which do not increase cerebral blood flow (10). Mannitol treatment in CHS is controversial. It can be used in CHS but its benefit in long-term prognosis is unclear. Labetalol and clonidine aren't available in Turkey. That's why we decreased patient's blood pressure with glycerol trinitrat.

Most important feature of our patient is having CHS in only contralateral hemisphere. There isn't any information about this situation in literature. Poor collaterals and bilateral severe carotid artery stenosis increase the risk of CHS (6). Our patient had these two factors. But having these factors doesn't explain the CHS only in contralateral hemisphere. Our patient had a TIA from left cerebral hemisphere 3 weeks before intervention. Patient had lacunar infarction in left centrum semiovale in diffusion magnetic

resonance (MR) performed 3 weeks ago (Figure 8). There is a potential risk of CHS and cerebral hemorrhage if recanalization is done early in patients with large cerebral infarction (10). There may be a relationship between TIA and CHS, but there isn't any information in the literature. More studies are needed about this subject.

REFERENCES

1. Yen-Heng Lin, Hon-Man Liu. Update on cerebral hyperperfusion syndrome. *J Neuro Intervent Surg.* 2020;12:788–93.
2. M.M. Reigel, L.H. Hollier, T.M. Sundt Jr., D.G. Piepgras, F.W. Sharbrough, K.J. Cherry. Cerebral hyperperfusion syndrome: a cause of neurologic dysfunction after carotid endarterectomy. *J Vasc Surg.* 1987;5:628–34.
3. G.S. Tan, C.C. Phatouros. Cerebral hyperperfusion syndrome post-carotid artery stenting, *J. Med. Imaging Radiat Oncol.* 2009;53: 81–6.
4. van Mook WN, Rennenberg RJ, Schurink GW, van Oostenbrugge RJ, Mess WH, Hofman PA, et al. Cerebral hyperperfusion syndrome. *Lancet Neurol.* 2005; 4(12): 877–88.
5. Moulakakis KG, Mylonas SN, Sfyroeras GS, Andrikopoulos V. Hyperperfusion syndrome after carotid revascularization. *J Vasc Surg.* 2009;49:1060–8.
6. Huibers AE, Westerink J, de Vries EE, Hoskam A, den Ruijter HM, Moll FL, et al. Editor's Choice Cerebral Hyperperfusion Syndrome After Carotid Artery Stenting: A Systematic Review and Meta-analysis. *Eur J Vasc Endovasc Surg.* 2018;56:322-333
7. Abou-Chebl A, Yadav JS, Reginelli JP, Bajzer C, Bhatt D, Krieger DW. Intracranial hemorrhage and hyperperfusion syndrome following carotid artery stenting: risk factors, prevention, and treatment. *J Am Coll Cardiol.* 2004;43:1596–601.
8. Bouri S, Thapar A, Shalhoub J, Jayasooriya G, Fernando A, Franklin IJ, et al. Hypertension and the post-carotid endarterectomy cerebral hyperperfusion syndrome. *Eur J Vasc Endovasc Surg.* 2011;41:229–37.
9. Ogasawara K, Sakai N, Kuroiwa T, Hosoda K, Iihara K, Toyoda K, et al. Intracranial hemorrhage associated with cerebral hyperperfusion syndrome following carotid endarterectomy and carotid artery stenting: retrospective review of 4494 patients. *J Neurosurg.* 2007;107:1130–6.
10. Farooq MU, Goshgarian C, Min J, Gorelick PB. Pathophysiology and management of reperfusion injury and hyperperfusion syndrome after carotid endarterectomy and carotid artery stenting. *Exp & Trans Stroke Med.* 2016;8:7