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CASE REPORT

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Cerebral Hyperperfusion Syndrome After Carotid Stenting: A Case Report

Karotis Stentleme Sonrası Hiperperfüzyon Sendromu: Olgu Sunumu

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ABSTRACT

Hyperperfusion syndrome (HPS) is a rare complication of revascularization procedures performed for chronic stenosis of internal carotis artery, manifesting with a spectrum of clinical findings ranging from mild symptoms such as headache to severe neurological deficits. Here in, we present a case of HPS following carotid artery stenting. A 75-year-old male presented with significant symptomatic internal carotid artery (ICA) stenosis. Despite strict blood pressure control and maintenance of normotension following carotid artery stenting, delayed arterial filling was observed in the distal branches of the middle cerebral artery due to hyperperfusion. HPS can present a devastating course after carotid artery stenting. If not properly managed, it may lead to severe cerebral edema, intracerebral or subarachnoid hemorrhage, and death.

Keywords: Hyperperfusion syndrome, internal carotid artery, carotid stenosis.

ÖZ

Hiperperfüzyon sendromu (HPS), internal karotis arterin kronik stenozu için revaskülarizasyon prosedürlerinin nadir görülen bir komplikasyonudur. Baş ağrısı gibi hafif semptomlardan şiddetli nörolojik defisitlere kadar değişen klinik bulgulara neden olur. Bu yazıda karotis stentleme sonrası hiperperfüzyon sendromu gelişen olguyu sunmayı amaçladık. 75 yaş erkek hasta semptomatik internal karotis arterde ciddi darlık ile başvurdu. Karotis stentleme sonrasında sıkı kan basıncı takibine ve normotansif seyretmesine rağmen hiperperfüzyona bağlı orta serebral arter distal dallarında geç arteriyel dolum izlendi. HPS, karotis arter stentlemeden sonra gelişebilen yıkıcı bir tablo ile seyredebilmektedir. Doğru bir şekilde tedavi edilmezse, ağır beyin ödemi, intraserebral veya subaraknoid kanama ve ölüme yol açabilir.

Anahtar Kelimeler: Hiperperfüzyon sendromu, internal karotis arter, karotis stenozu.

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INTRODUCTION

Cerebral hyperperfusion syndrome (HPS) is a rare but potentially devastating complication of procedures such as carotid endarterectomy or carotid artery stenting (CAS).[1] Carotid stenosis greater than 90% is the main risk factor for the development of HPS, and other important risk factors include severe lesions of the contralateral carotid artery, poor collateral circulation, hypertension, and a recent history of stroke or the presence of an ischemic lesion. Although the incidence of HPS ranges from 14.1 to 56%, the presence of additional risk factors is associated with a higher risk of HPS.[2] Internal carotid stenting and angioplasty are increasing in use due to the widespread adoption of new devices; therefore, it is necessary to better understand their complications and take measures to minimize them.

CASE PRESENTATION

A 75-year-old male patient had been monitored at an external center for speech disturbance 1 month prior to presentation. His speech had completely recovered after 1 week. His medical history included deep vein thrombosis (DVT) and benign prostatic hyperplasia, and it was learned that he was using warfarin due to DVT. No pathology was observed in his neurological examination. External center diffusion MRI revealed multiple infarct areas in the left hemisphere. In cervical computed tomography (CT) angiography, approximately 90% stenosis was observed at the bulb level in the proximal cervical segment of the left internal carotid artery (ICA) (Figure 1). As the INR value of the patient using warfarin was 1.5, acetylsalicylic acid, clopidogrel, and enoxaparin sodium were initiated 3 days before the carotid stenting procedure.

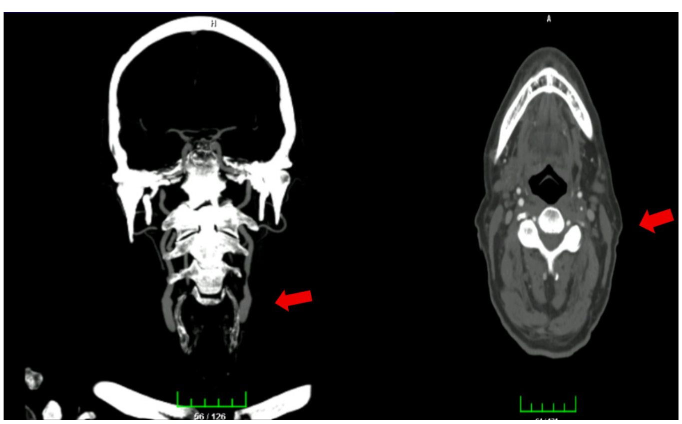

Figure 1. CT angiography image before carotid stenting; coronal and axial sections

MAIN POINTS

- Hyperperfusion syndrome (HPS) is a rare complication of revascularization procedures for chronic stenosis of the internal carotid artery.
- HPS may present with a devastating clinical picture that can develop after carotid artery stenting.
- If not properly treated, it can lead to severe cerebral edema, intracerebral or subarachnoid hemorrhage, and death.

Access to the femoral artery was obtained with a 6 French (F) introducer femoral sheath. Before carotid stenting, diagnostic angiography with bilateral ICA cervical and cranial imaging and posterior circulation imaging by injection from the left subclavian artery were performed, and the degree of stenosis was determined. 90% stenosis was observed in the cervical segment of the left ICA. Intervention was decided for symptomatic ICA stenosis. Before the carotid intervention, his blood pressure was 140/70 mmHg. Five thousand units of intra-arterial heparin were administered. Through the 6 F introducer femoral sheath, a 6 F guiding catheter (Envoy 6 Fr; Johnson & Johnson, Medtech, US) containing a hydrophilic wire was advanced into the left common carotid artery. The lesion was crossed with a 0.014 coronary guidewire. Predilatation balloon angioplasty was not performed. A 7x40 mm closed-cell self-expandable carotid artery stent was deployed at the lesion site (Wallstent 7x40mm, Boston Scientific, Marlborough). As more than 30% residual stenosis remained after stent deployment, postdilatation angioplasty was performed using a 4x20 balloon at 8 atm (Viatrac 14 Plus peripheral balloon dilatation catheter 4x20 mm; Abbott, US) (Figure 2).

Figure 2. Lateral angiographic image of the internal carotid artery before and after stenting

During the procedure, due to altered consciousness, severe dysarthria, and right motor strength evaluated as 4/5 in the neurological examination, repeat carotid and intracranial imaging was performed. In intracranial imaging, delayed arterial filling was observed in the distal branches of the middle cerebral artery (Figure 3).

After the procedure, since the intra-arterial (IA) blood pressure was measured at 170/90 mmHg, a total of 300 ml of nitrate was administered IA. After nitrate administration, the control blood pressure was 140/80 mmHg. Upon regulation of blood pressure, the procedure was terminated. Control brain CT and CT perfusion were performed. In the control CT, sulcal effacement and hyperdensity were observed in the left hemisphere. No significant finding was observed in CT perfusion. In transcranial Doppler studies, the pulsatility index was 1.78 in favor of increased intracranial pressure, and vasospasm was not observed. The normal pulsatility index range was 0.6–1.1. Blood flow was evaluated as normal in transcranial Doppler ultrasonography (Figure 4). Clopidogrel 75 mg and acetylsalicylic acid 100 mg were continued as antiplatelet therapy. 20% mannitol 4 times daily and 3% hypertonic solution twice daily were administered as anti-edema and hypertonic therapy with close monitoring. At discharge examination, the right upper extremity muscle strength was full, and he was discharged with mild dysarthria.

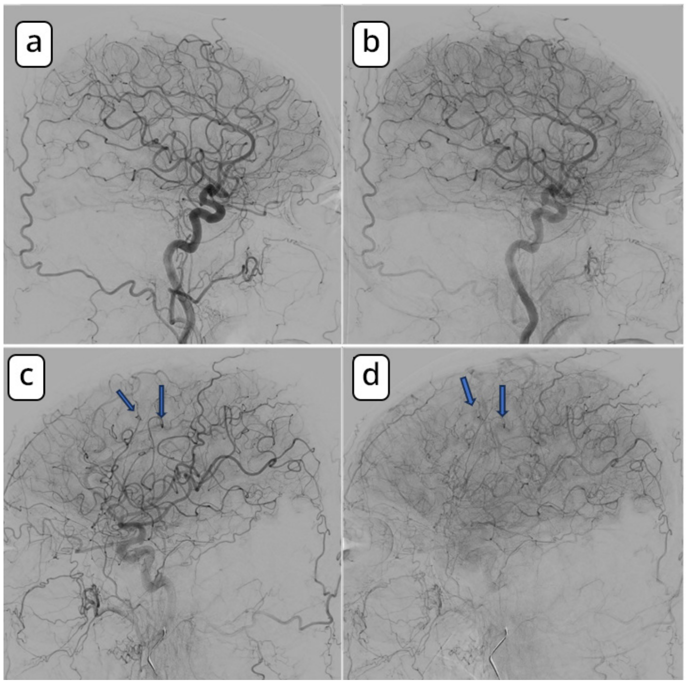

Figure 3 a-b. Lateral view arterial and capillary phase before carotid stenting **c-d:** Lateral view arterial and capillary phase after carotid stenting, showing delayed filling in distal branches.

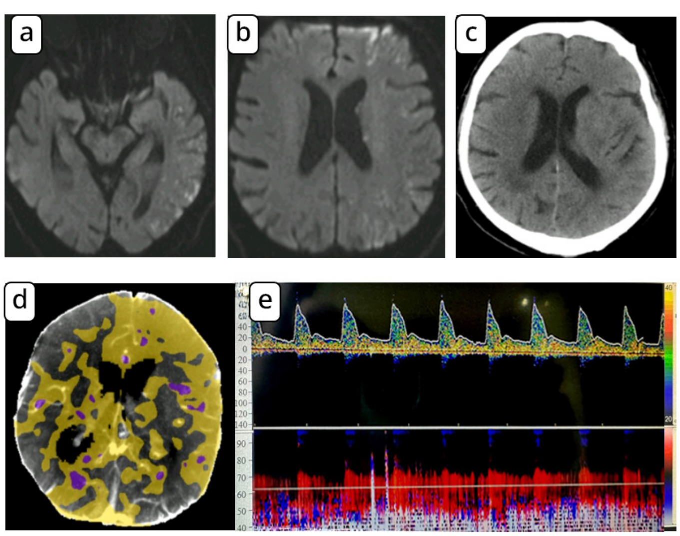

Figure 4 a-b. Diffusion MRI DWI sequence before carotid stenting **c.** Brain CT after carotid stenting; sulcal effacement and hyperdensity in the left hemisphere **d.** CT perfusion after carotid stenting **e.** Transcranial Doppler USG after carotid stenting, pulsatility index: 1.78

DISCUSSION

Hyperperfusion has been defined as a 100% increase in cerebral blood flow compared with the preoperative value or an increase of the postoperative asymmetry index ≥ 120% compared with the normal side. HPS is a rare complication of revascularization interventions performed for chronic stenosis of the extracranial and intracranial segments of the internal carotid artery and presents variable clinical manifestations ranging from mild symptoms such as headache to severe clinical pictures.[3-4]

The pathophysiological mechanisms of HPS are partially understood. Impairment in cerebral autoregulation and changes in cerebral hemodynamics after revascularization are considered the main mechanisms involved in the development of the syndrome. In patients with adequate collateral circulation, less damage occurs in arterioles and capillaries under chronic ischemia, and therefore, collateral circulation may provide protection against HPS.[5] Normal cerebral autoregulation maintains normal cerebral perfusion within an acceptable range (60–160 mm Hg) by constricting cerebral vessels in response to an abrupt increase in blood flow. In a chronically ischemic brain, when perfusion pressure suddenly increases, arterioles and capillaries are vulnerable to rupture and hemorrhage. This process explains the occurrence of HPS, particularly in patients with small vessel disease (as a result of chronic hypertension or diabetes mellitus) and normal systemic arterial pressure.[6]

HPS after CAS has been reported more frequently than after carotid endarterectomy, and disruption of the blood–brain barrier mediated by contrast agents is also thought to reinforce these mechanisms.[7]

In our case, neurological deterioration occurred due to increased cerebral perfusion pressure after carotid stenting. In accordance with our case, maximal arteriolar vasodilation, impaired cerebral autoregulation, and systemic hypertension, when combined, result in increased cerebral perfusion pressure and cerebral hyperperfusion. Two sentinel signs have been defined as indicators of maximal arteriolar vasodilation and impaired cerebral autoregulation; two angiographic findings suggestive of hyperperfusion after CAS may be observed. The first is early venous filling, which is visualized in the late arterial or capillary phase as early opacification of an anatomically normal vein draining the ischemic region located distal to the relevant vessel. This finding indicates early venous drainage due to increased perfusion in the treated area. The second finding is dense capillary blush observed in the treated ischemic area, which begins in the arterial phase, becomes more pronounced in the capillary phase compared with surrounding arterial areas, and persists until the venous phase. This situation is generally associated with the passage of contrast material early through the capillary bed to the draining vessels in the affected area, resulting in early venous filling.[8] Capillary blush is usually intense and evident, but as in our case, it may rarely appear in such a small amount that it may be overlooked. The prevention or treatment of HPS has not yet been determined, but several clinical studies have reported the effectiveness of staged angioplasty and strict blood pressure control in preventing HPS.[9]

The most common clinical finding is severe headache, while less common and more severe symptoms include neurological deficits, encephalopathy, and seizures.

The more abrupt restoration of high blood flow with carotid stenting may lead to a rapid increase in cerebral perfusion pressure and a higher likelihood of HPS. Patients undergoing CAS may experience more pronounced fluctuations in blood pressure during the procedure, which may increase the risk of HPS. The abrupt hemodynamic changes observed with CAS may further increase the risk of HPS by affecting the brain's ability to autoregulate cerebral blood flow. Risk factors for HPS have been reported as arterial hypertension, advanced age, contralateral ≥ 70% carotid stenosis, and recent symptomatic stenosis. Carotid stenting is frequently associated with long-term hemodynamic instability, and this may be considered a more significant risk factor for HPS in CAS compared with carotid endarterectomy.[10]

Hyperperfusion syndrome has a low incidence rate, but if it leads to intracranial hemorrhage, it has a high mortality rate of 26%.[11] Therefore, clinicians should evaluate the risk of HPS and consider the possibility of cerebral hyperperfusion after carotid stenting and angioplasty. In line with the existing literature, our case demonstrates a complex interaction of factors that affect the pathophysiology of HPS and have not yet been fully understood. This underscores the need for additional research to develop diagnostic and therapeutic strategies for this condition.

Informed Consent: Written informed consent was obtained from patient who participated in this study.

Author Contributions: Concept – A.Ö.Ö.; Design – A.Ö.Ö.; Supervision – H.B.A.; Resources – H.B.A.; Materials – H.B.A.; Data Collection and/or Processing – Ö.A.; Analysis and/or Interpretation – H.B.A.; Literature Search – H.B.A.; Writing Manuscript – H.B.A.; Critical Review – H.B.A.

Declaration of Interest: The authors have no conflicts of interest to declare.

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