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CASE REPORT

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Vasoconstriction and hyperperfusion syndrome after carotid artery stenting

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Abstract

Background: Common complications of carotid artery stenting (CAS) are cerebral ischemia and hyperperfusion syndrome. To date, only a few cases of late-onset cerebral vasoconstriction occurring several hours after CAS have been reported. However, there are no reports of developed vasoconstriction and hyperperfusion syndrome.

Case presentation: A 79-year-old man developed vasoconstriction 1 day after carotid artery stenting. Vasoconstriction improved immediately with glucocorticoid. However, a week later, the patient developed hyperperfusion syndrome.

Conclusions: Postoperative vasoconstriction and hyperperfusion syndrome is an unrecognized complication and surgeons should be aware of it.

Keywords: Vasoconstriction, Spasm, Carotid artery stenting, Hyperperfusion, Glucocorticoid

Background

Common complications of carotid artery stenting (CAS) are cerebral ischemia and hyperperfusion syndrome [1]. Surgeons mainly focus on intraoperative complications; however, the incidence of postoperative complications is high [2]. To date, only a few cases of late-onset cerebral vasoconstriction occurring several hours after CAS have been reported [3, 4]. However, there are no reports of developed vasoconstriction and hyperperfusion syndrome.

Case presentation

A 79-year-old man with right hemiplegia was admitted to the hospital. CAS was performed for the right internal carotid artery (ICA) stenosis 4 years ago, during which a left extracranial ICA stenosis was noted that progressed gradually. The patient's hypertension and dyslipidemia, which were risks for atherosclerosis, were controlled. Upon admission, infarctions in the left cerebral cortex were observed on magnetic resonance imaging (MRI) (Fig. 1A). Carotid duplex ultrasound of the left ICA revealed an equiluminant plaque and peak systolic velocity of 160 cm/s. Black-blood MRI showed high intensity and revealed an unstable high-volume plaque. With antiplatelet therapy, CAS was performed for severe stenosis (NASCET: 95%; Fig. 1B) using the Parodi system [5]. It was presumed that the patient was tolerant to ICA occlusion, because previous CAS with balloon occlusion device on the right ICA was performed without complications. After predilation (4×30 mm), a stent (Carotid WALLSTENT; Boston Scientific, Natick, MA, USA) was deployed (Fig. 1C). Right paralysis, aphasia, and loss of consciousness (Glasgow Coma Scale: E3V2M5) appeared postoperatively. MRI performed immediately after the surgery revealed new microcortical infarctions, but those were not serious and cannot explain the patient's condition. Moreover, no vascular abnormalities were observed. We suspected that the temporary blood flow interruption caused by the balloon device during the surgery would possibly explain the patient's condition. On the following day, the patient's state of consciousness did not improve.

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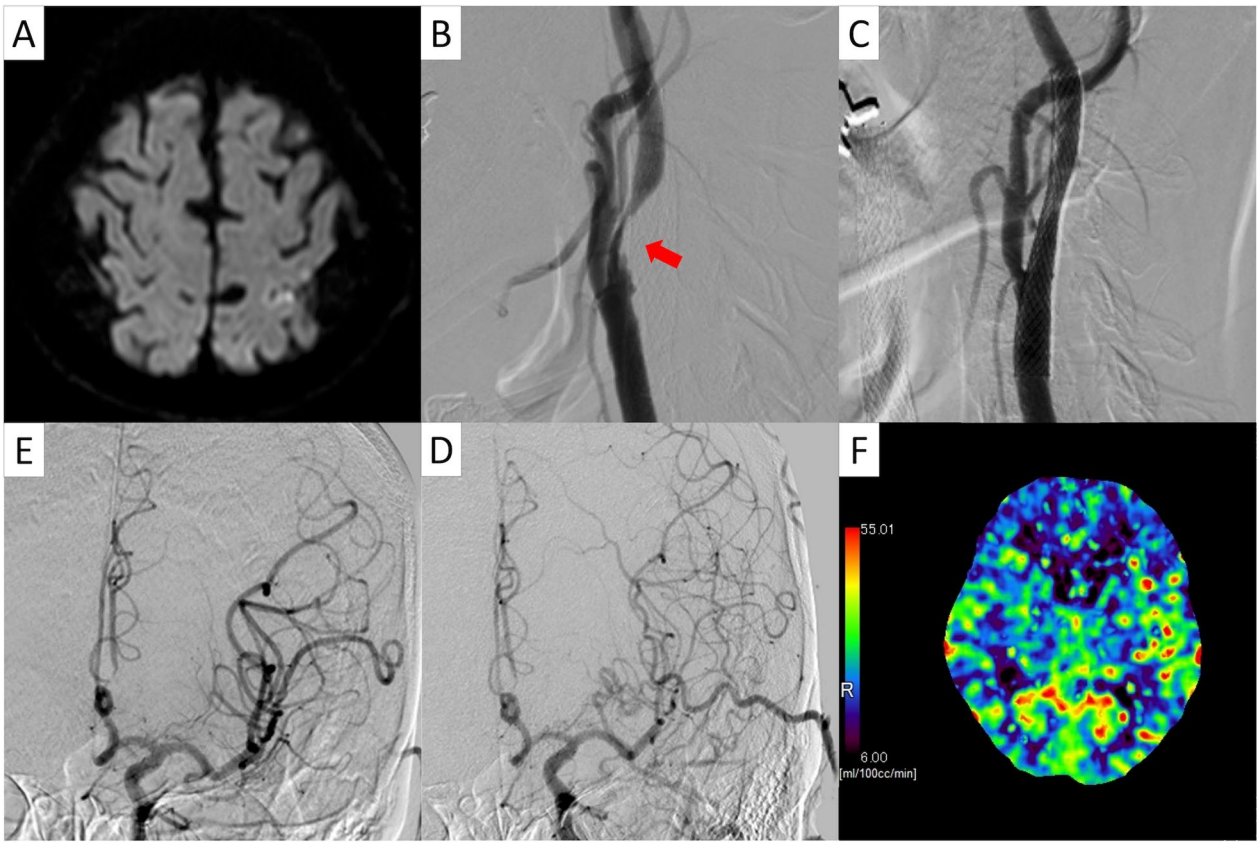

Fig. 1 **A** Diffusion-weighted imaging performed upon admission revealed multiple cortical cerebral infarctions. **B**, **C** Carotid artery stenting was performed for internal carotid artery stenosis (red arrows). **D** Preoperatively, atherosclerotic stenosis was observed in the left anterior cerebral artery. Digital subtraction angiography (DSA) was performed at the end of the surgery, and the results showed that the left middle cerebral artery (MCA) was dilated. **E** DSA performed 1 day after the surgery revealed a localized vasoconstriction mainly of the left MCA. **F** One week after the surgery, CT perfusion revealed hyperperfusion, with a cerebral blood flow contralateral ratio of greater than 1.3. CT perfusion was scanned using a multidetector CT scanner with 80 detector rows (Aquilion Prime, Canon, Japan) and DSA was performed with biplane equipment (Infinix Celeve-i INFX-8000 V, Canon, Japan). MRI scanner was 1.5 T scanner (Magnetom Avanto fit, Siemens Healthcare, Germany)

Computed tomography (CT) angiography, CT perfusion, and MRI revealed decreased blood flow in the left middle cerebral artery (MCA), and digital subtraction angiography (DSA) revealed vasoconstriction mainly in the left MCA (Fig. 1D, E). Considering an inflammatory pathology, methylprednisolone 500 mg/day was administered for 3 days, immediately after which the patient's symptoms and magnetic resonance angiography showed improvement. Blood pressure was controlled without restriction and systolic blood pressure was observed to be approximately 140 mmHg. Seven days after surgery, aphasia and right paralysis reappeared. CT angiography revealed hyperperfusion and the cerebral blood flow (CBF) contralateral ratio in the MCA territory was > 1.3 (Fig. 1F) [6]. Hyperperfusion improved after antihypertensive management; however, mild aphasia and right paralysis persisted. The patient was transferred to a rehabilitation hospital with a modified Rankin scale score of 4.

Discussion

There are several hypotheses of vasoconstriction mechanism after CAS and carotid endarterectomy (CEA). The first hypothesis was that debris or thrombus in stent expansion sites might have caused vasoconstriction due to ischemia or inflammatory conditions [3]. This is supported by the fact that CAS using covered stents caused less neurological complications [7] and the incidence of postoperative complications was high [2]. We suspected an inflammatory condition and administered methylprednisolone. As a result, vasoconstriction improved substantially. However, a pathological search is necessary to identify the effects of glucocorticoids and underlying cause. The

second hypothesis was the occurrence of reversible cerebral vasoconstriction syndrome (RCVS). Few reports showed the occurrence of RCVS after CEA, characterized by headache [8, 9]. However, we dismissed the occurrence of RCVS, because the patient did not suffer from headache and vasoconstriction on the image did not show segmental narrowing and dilatation, which is a characteristic of RCVS [10]. Moreover, we administrated methylprednisolone. In general, glucocorticoids are ineffective and can worsen RCVS [11]. The final hypothesis was that cerebral autoregulations became dysfunctional. The pathophysiological hallmark of hyperperfusion syndrome acutely increased the CBF in a hypoperfused brain with maximally dilated vessels. These vessels cannot control increased CBF due to autoregulatory mechanism loss [12]. It was reported that vasoconstriction and hyperperfusion syndrome were developed after CEA [8]. Although completely opposite conditions were observed in the same patient, it may have been the result of disturbances in cerebral autoregulations, which belong to the same spectrum. Chronic severe carotid artery stenosis is observed to cause disturbances in cerebral autoregulations, and relative hypertension in these arteries after CAS or CEA may play a role in developing vasoconstriction. Our patient did not strictly control blood pressure after vasoconstriction, but the patient's blood pressure was not too high. It is unclear as to why the patient developed hyperperfusion, but the disturbances in cerebral autoregulations may be associated.

This is the first case report on CAS complications, which include vasoconstriction and hyperperfusion syndrome. In general, surgeons know the risk of hyperperfusion syndrome. The operated side is hypoperfused even before the surgery, thus making it difficult to consider hypoperfusion as an unusual manifestation. Hence, surgeons should be aware of the vasoconstriction risk. Hyperperfusion syndrome and vasoconstriction represent two opposite ends of the pathophysiological condition, and the management of both complications is challenging. Thus, regional saturation of oxygen and transcranial Doppler ultrasonography should be performed, and treatment must be given in a timely manner.

There are no data showing the appropriate glucocorticoid treatment for vasoconstriction. Additional cases should be collected and assessed to better understand the pathology of this condition.

Conclusions

Postoperative vasoconstriction and hyperperfusion syndrome is an unrecognized complication and surgeons should be aware of it.

Abbreviations

CAS: Carotid artery stenting; CBF: Cerebral blood flow; CEA: Carotid endarterectomy; CT: Computed tomography; DSA: Digital subtraction angiography; ICA: Internal carotid artery; MCA: Middle cerebral artery; MRI: Magnetic resonance imaging; RCVS: Reversible cerebral vasoconstriction syndrome.

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Authors' contributions

YK: data acquisition, data analysis, and writing of the manuscript. TM, GO, KN and YS: data acquisition and revision of the manuscript. TS and AS: surgeons of the patient. All authors have read and approved the manuscript.

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Availability of data and materials

Additional data that support the findings of this study are available on request from the corresponding author.

Declarations

Ethics approval and consent to participate

The Ina Central Hospital ethics committee approved this case report. Informed consent was obtained in the form of opt-out on the website.

Consent for publication

The patient was dementia due to cerebral infarctions and consent from the patient was not obtained. The next of kin signed an informed consent to allow his data to be published. We obtained approval from the research ethics committee.

Competing interests

The authors declare that this research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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