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Report

A Case of Cerebral Infarction after Stellate Ganglion Block

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Cerebral vascular impairment is a rare complication of stellate ganglion block (SGB). We describe a patient in whom left leg paralysis caused by right anterior cerebral artery infarction developed about 30 minutes after left-sided SGB. Treatment with tissue plasminogen activator after 3 hours improved the disability. Cerebral angiography revealed latent stenosis of the right anterior cerebral artery. We suspected that acute hypotension caused by attenuated baroreflex control after SGB decreased cerebral arterial flow. The causal relationship between SGB and cerebral infarction was not clear in this patient.

Key Words: stellate ganglion block, cerebral infarction, baroreflex

Introduction

Patients with latent cerebral artery stenosis are at risk for cerebral ischemic events associated with systemic hypotension. Stellate ganglion block (SGB) is one type of sympathetic block used to manage pain in the face, neck, and chest. The autonomic nervous function is altered by SGB, potentially leading to cardiovascular complications such as hypertension or hypotension. We describe our experience with a case of cerebral infarction that developed after SGB.

Case

A 53-year-old man (height, 170 cm; body weight, 80 kg; BMI, 27) had continuous burning pain in the left side of the chest (Th2-3 region) for 1 month after herpes zoster infection. Oral analgesics (nonsteroidal anti-inflammatory drugs) were ineffective. He had hyperlipidemia and hypertension, for which he was not receiving medication. He was not a smoker and did not have diabetes or electrocardiographic abnormalities. He took oral aspirin for transient head heaviness about once a month. The frequency of headache had not changed recently, and the patient did not experience any symptoms of transient ischemic attack before or after the development of herpes zoster infection. The daily blood pressure was about 150/80 mmHg. One day after obtaining informed consent, left-sided SGB was performed with 6 ml of 1% mepivacaine via a C6 anterior approach. Horner's sign was confirmed, and slight pain relief was obtained. Fifteen minutes after the block, the systolic blood pressure reached 160 mmHg, and he returned home without any symptoms after about 10 minutes. After 1 week, control blood pressure was not measured before the second session of SGB with the same method and dose of 1% mepivacaine. Horner's sign was confirmed and slight pain relief was obtained, without any hypoesthesia around the neck. The systolic blood pressure was 160 mmHg, and he walked out of the room without unsteadiness 15 minutes after the injection. Subsequently, he complained of faintness accompanied by a cold sweat and difficulty in moving the left arm and legs in the outpatient ward. The blood pressure reached 208/108 mmHg, and the heart rate was 80/min at the emergency room after the accident. The blood pressure fell to 172/88 mmHg after 20 minutes. A neurologist was immediately

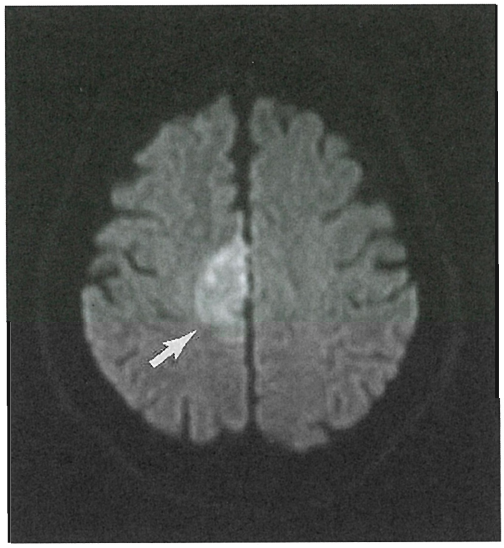

Fig. 1 The arrow shows right parietal cerebral infarction on MRI on the day of administration.

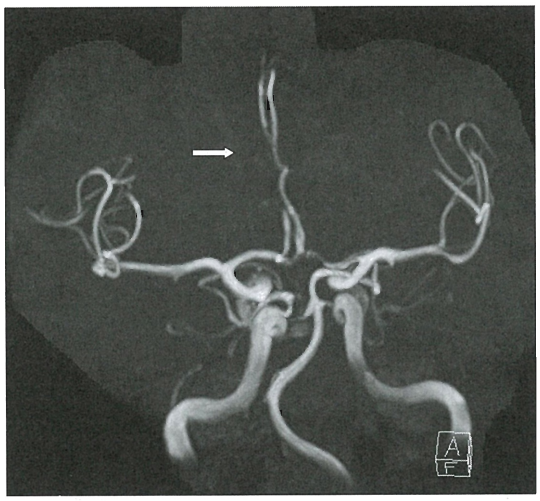

Fig. 2 The arrow shows right A2 stenosis on magnetic resonance imaging.

consulted, and cerebral magnetic resonance imaging (MRI) revealed infarction of the right parietal region of the brain (Fig. 1). The National Institute of Health Stroke Scale score (NIH stroke score) was 2 (left leg weakness). With the approval of the neurologist, treatment with tissue plasminogen activator was started 3 hours after SGB. Left leg paralysis improved on the next morning, and the NIH stroke score improved to 0-1, with slight weakness persisting in the left leg. Magnetic resonance angiography revealed latent stenosis of the right anterior cerebral artery (Fig. 2).

Discussion

Latent cerebral artery stenosis was suspected to be the cause of cerebral infarction in this patient. Treggiari et al. reported that SGB improved cerebral blood flow and reduced delayed ischemic neurologic deficit caused by cerebrovascular contraction in a patient with aneurysmal subarachnoid hemorrhage[1]. The direct effect of SGB on cerebral blood flow remains controversial[2)3)]. Recently, Kang et al demonstrated by magnetic resonance angiography that intracranial artery blood flow, excluding that in the ophthalmic artery, was unaffected by SGB in 19 healthy volunteers[4]. There was no description about SGB laterality.

Cardiovascular accidents after SGB include hypertension, hypotension, and cardiac arrest. Case reports documenting patients with abnormal hypertension after left-sided SGB concluded that accidental block of the glossopharyngeal nerve or vagal nerve decreased baroreceptor sensitivity, resulting in hypertension[5]. Yokota et al reported that the frequency of abnormal hypotension is higher after right-sided SGB, but abnormal hypertension is higher after left-sided SGB. These events occur predominantly in males and patients older than 50 years[6].

The Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification has 5 stroke subtype categories of cerebral infarction: 1) large vessel atherothrombotic, 2) cardioembolic, 3) small vessel, 4) stroke of other determined etiology, and 5) stroke of undetermined etiology[7]. According to this subtype diagnosis, cerebral infarction in our patient was classified as large vessel atherothromboembolic stroke, with latent ipsilateral anterior cerebral artery stenosis and without the risk of cardiac sources of emboli. Decreased cerebral blood flow caused by fluctuations in blood pressure might have triggered this thrombotic event. Some case reports have documented the occurrence of hypotension, bradycardia, and cardiac arrest after right-sided SGB, but not after left-sided SGB[8)9)]. These conditions were apparently caused by impaired sympathetic reinnervation of the sinus node[8]. Taneyama et al showed that either right- or left-sided

SGB significantly attenuated baroreflex sensitivity as assessed by head-up tilt testing in healthy volunteers. This impaired sensitivity was attributed to autonomic imbalance and complexity of heart rate and systolic blood pressure variability, persisting up to 60 minutes[10]. They strongly recommended bed rest for more than 60 minutes for all patients after SGB. Chaturvedi reported locked-in syndrome after right-sided SGB following accidental intra-arterial local anesthetic injection[11]. Despite negative aspiration of blood during SGB and no manifestations central nervous system stimulation immediately after SGB, we cannot exclude intra-arterial injection of mepivacaine as a possible cause. In our patient, we did not detect a fall in blood pressure after left-sided SGB.

Conclusion

The relation between left-sided SGB and right parietal cerebral infarction in our patient remains uncertain. However, our experience indicates that patients should rest in bed for at least 1 hour after SGB to avoid unexpected accidents.

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星状神経節ブロック後に脳梗塞を発症した1例

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脳血管障害は，星状神経節ブロックの合併症としてはまれである．我々は，左星状神経節ブロック約30分後に右前大脳動脈梗塞によると思われる左下肢不全麻痺を発症したが，3時間後に組織プラスミノーゲン活性化剤投与開始し症状改善を得られた症例を経験したので報告する．潜在性前大脳動脈狭窄に星状神経節ブロックによる圧反射減弱による血圧低下が脳血流低下を招いた可能性も考えられた．本症例の脳梗塞発症の原因および星状神経節ブロックとの因果関係は不明である．