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BRACHIAL PLEXUS AND SUPRACLAVICULAR NERVE INJURY CAUSED BY MANUAL CAROTID COMPRESSION FOR SPONTANEOUS CAROTID-CAVERNOUS SINUS FISTULA

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Komiyama M, Nakajima H, Nishikawa M, Yasui T. Brachial plexus and supraclavicular nerve injury caused by manual carotid compression for spontaneous carotid-cavernous sinus fistula. Surg Neurol 1999;52:306–9.

BACKGROUND
Manual carotid compression is an established treatment for a spontaneous carotid-cavernous sinus fistula unless emergency treatment is required for it.

CASE REPORT
A 63-year-old woman presented with a spontaneous carotid-cavernous sinus fistula. Manual carotid compression of 5 minutes duration, twice a day, for 10 days resulted in injury to the upper trunk (C 5–6) of the brachial plexus and the supraclavicular nerve (C 3–4), which subsequently resolved within a month after cessation of the procedure.

CONCLUSION
It is important to know the possible neurological complications of manual carotid compression. © 1999 by Elsevier Science Inc.

KEY WORDS
Brachial plexus injury, carotid-cavernous sinus fistula, carotid compression, complication.

Manual carotid or carotid-jugular compression is an established treatment for a spontaneous carotid-cavernous sinus fistula (CCF), unless emergency treatment is required for it [2,3,5–7]. The possible adverse effects of carotid compression may include hemodynamic or thromboembolic complications, seizure, vasovagal reactions, intracranial hemorrhage, retinal hemorrhage [7], the clinical deterioration known as "paradoxical worsening phenomenon" [8], vertebral artery occlusion [9], and temporary monocular blindness during the carotid compression [4]. We recently observed an unexpected neurological complication caused by mechanical injury to the brachial plexus and supraclavicular nerve during repeated manual compression in the treatment of a spontaneous CCF. The purpose of this communication is to present this rare but potentially dangerous complication of manual carotid compression.

CASE PRESENTATION

The patient, a 63-year-old woman, noticed periorbital edema and conjunctival injection on the left side, followed by diplopia 1 month before admission. The patient did not experience bruits or tinnitus at any time. She had a history of diabetes mellitus for 5 years, which had been controlled with medication. The patient had cataract surgery and implantation of an artificial lens in the left eye 12 years ago. At admission, the patient was alert and no neurological deficits were observed except for slight visual decline, abducent paresis (3/5), proptosis, chemosis, and conjunctival injection all on the left side. No bruit was audible. Intraocular pressure was 17 mmHg in the right eye and 28 mmHg in the left. Visual acuity was 0.4 in the right eye and 0.15 in the left. Proptosis was 11 mm in the right eye and 17 mm in the left.

Digital subtraction angiography demonstrated no abnormality on the right side. Left carotid injection showed the dural CCF on the left side (Figure 1**A**). Left cervical carotid bifurcation was normal. Main feeding arteries were the left meningohypophyseal

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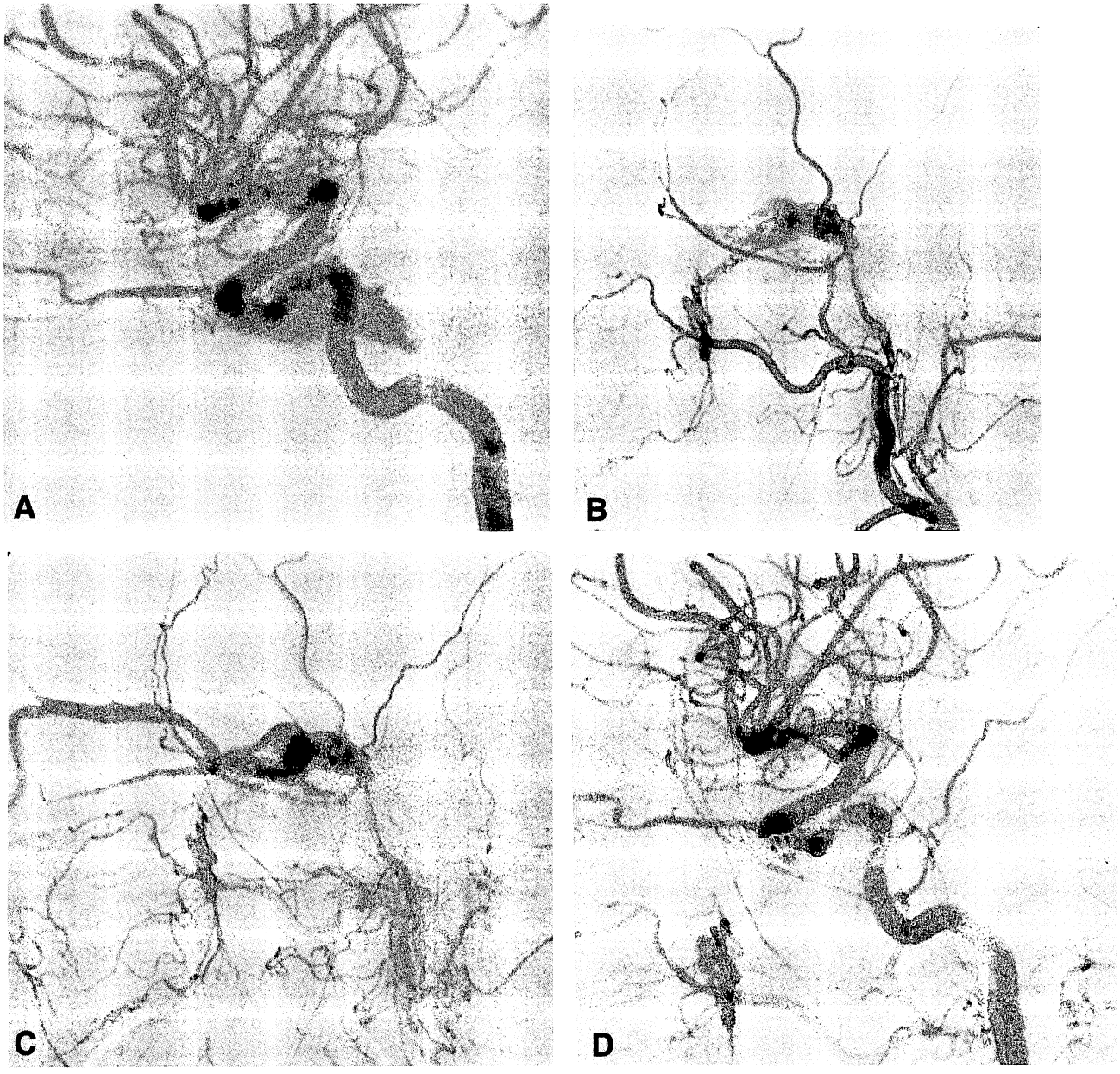

1 Left spontaneous carotid-cavernous sinus fistula. (**A**) Lateral view, pre-embolization. Left internal carotid injection shows arteriovenous shunts in left cavernous sinus. (**B**) Mid-arterial phase and (**C**) late arterial phase, pre-embolization. Left external carotid injection shows arteriovenous shunts and drainage solely to superior and inferior orbital veins. (**D**) Lateral view, at completion of transvenous coil embolization. Left common carotid injection shows no apparent arteriovenous shunts in cavernous sinus.

artery, middle meningeal artery, and artery of the foramen rotundum. There was no cortical drainage or posterior drainage. Drainage of the CCF was solely to the superior and inferior orbital veins (Figure 1 **B** and **C**). Thus, the diagnosis was a left spontaneous CCF, Barrow type D [1], with a sole anterior drainage.

Because there was no cortical drainage, acute visual decline or pathology in the left carotid bifurcation, we started manual carotid compression on the left side after obtaining informed consent to this treatment. One of the authors (MK) performed manual carotid compression for 5 minutes twice a day. Carotid compression was strong enough to occlude the carotid flow. The patient was instructed to do it in a similar fashion with her right hand. However, she complained of difficulty in identifying and compressing the carotid artery adequately; thus, carotid compression by the patient did not contribute to the treatment.

The patient complained of numbness in the left

supraclavicular region 3 days after the start of the carotid compression. Because we did not realize that this numbness was related to the carotid compression, manual compression was continued. One week after the patient's first complaint of numbness, the patient noted weakness of the left upper extremity (she was unable to elevate her arm) and a spreading area of numbness. Neurological examination revealed weakness (3/5) of the left C 5–6 motor territory without apparent sensory deficits and tenderness in the supraclavicular region where manual carotid compression had been performed. Numbness was spread over the left shoulder and supraclavicular regions, i.e., the territory of the left supraclavicular nerve (C 3–4). We immediately recognized that the manual carotid compression was causing brachial plexus and supraclavicular nerve injury and discontinued the treatment. Although the conjunctival injection and abducent paresis had improved to some extent, we decided to perform transvenous embolization because of the continued elevated intraocular pressure (26 mmHg) and inability to continue the carotid compression.

Under local anesthesia, a transfemoral, transvenous embolization was performed on the day after the cessation of the carotid compression. A microcatheter was navigated through the left inferior petrosal sinus (which did not fill on angiogram) into the left cavernous sinus. Interlocking detachable coils (Target Therapeutics, Fremont, CA) were placed first at the origin of the left superior orbital vein, and then at the presumed origin of the left sylvian vein, and finally in the main portion of the left cavernous sinus. Immediate closure of the spontaneous CCF was obtained (Figure 1**D**), but visual decline (left: 0.07) and deterioration of the abducent paresis (0/5) were apparent after coil occlusion, which might be attributable to the occlusion of the venous outflow of the orbital circulation and overpacking near the abducent nerve, respectively. Chemosis, conjunctival injection, and proptosis disappeared within several days. The patient's vision improved (left 0.4) within 2 weeks of treatment and abducent palsy disappeared in 3 months. Left intraocular pressure dropped to 18 mmHg. Paresis of the left upper extremity and paresthesia in the supraclavicular region began to improve a week after the cessation of carotid compression, and they recovered completely in a month without any special treatment.

Discussion

Carotid compression was first introduced for spontaneous pulsating exophthalmos in 1856 by Gioppi in Italy [cited in 6]. Today, unless treatment is urgent, carotid compression or carotid-jugular compression is the established treatment for spontaneous CCF and is less invasive than the other procedures [2,3,5–7]. A more aggressive approach is required if the patient develops acute visual decline, rapidly progressive proptosis, or intracranial hemorrhage, or when an angiogram shows cortical pial drainage [2,3]. Manual carotid compression is reported to be useful for about 30% of patients (7/23 patients) with spontaneous CCFs and there was no associated complication [3]. Possible adverse effects of carotid compression may include thromboembolic or hemodynamic complications in the territory of the ipsilateral carotid tree, seizure, vasovagal reactions resulting in cardiac arrhythmia, intracranial hemorrhage, retinal hemorrhage [7], clinical deterioration known as a "paradoxical worsening phenomenon" [8], vertebral artery occlusion with cerebellar and brain stem infarction [9], and temporary monocular blindness during the carotid compression [4]. Atherosclerotic change of the cervical carotid bifurcation should be examined with duplex sonogram or catheter angiography before the initiation of the manual carotid compression.

The brachial plexus is generally composed of roots C5 through T1. It is subdivided into 5 ventral rami, 3 trunks (upper, middle, and lower), 6 divisions (3 anterior and 3 posterior divisions), 3 cords (medial, lateral, and posterior cords), and individual nerves. The C5 and C6 ventral rami form the upper trunk in the supraclavicular region near the carotid bifurcation. Neurological examination of our patient showed that the upper trunk (and/or C5 and C6 ventral rami) of the brachial plexus and supraclavicular nerve (C3–4) were injured by manual compression.

There is no consensus on how often and how long carotid compression can be performed per day. Halbach et al [3] performed carotid-jugular compression initially for 10 seconds with a gradual increase to a maximum of 30 seconds per compression, several times per hour, but total occlusion time was not mentioned. Kuppersmith et al [5] recommended 5 minutes of compression every 2 hours for a week, and increased it to 20 minutes of compression every 2 hours. Miki et al [7] performed carotid compression over 2 hours in total, over 6 times a day. We did 5 minutes of carotid compression, twice a day. The longer the total duration of the carotid compression, the higher the risk of injury to the brachial plexus and superficial nerve, although this complication seems to be rare.

Our case clearly illustrates the possible compli-

cation of brachial plexus and supraclavicular nerve injury by manual carotid compression. Physicians who perform manual carotid compression should be aware of the possible complications and frequently ask the patient about numbness in the ipsilateral supraclavicular region or weakness of the upper extremity. As we saw in our patient, prognosis following the neurological injury by manual carotid compression is good if the procedure is discontinued at an early stage.

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COMMENTARY

This is an extremely rare complication of carotid compression in the neck for the treatment of a spontaneous dural CCF—I'm not sure it has ever been reported before.

It should be noted that the patient was unable to do the compression herself, and we can assume that the physician compressed the artery more strongly than the patient would be able to.

There has never been a double-blind study showing an advantage of carotid compression in the cure of these dural fistulas, which heal spontaneously in a substantial number of cases even without carotid compression. Therefore, considering the potential risks of carotid compression, I personally do not recommend it. I follow my patients conservatively until there is a good indication for treating them: progressive visual loss, increasing intraocular pressure, persistent diplopia, or pain.

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