

## Case Report

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# Spontaneous Recanalization from Traumatic Internal Carotid Artery Occlusion

The incidence of spontaneous recanalization after traumatic internal carotid artery occlusion is very rare. We have experienced a case of spontaneous recanalization after a traumatic internal carotid artery occlusion. A 5-year-old boy developed contra-lateral hemiparesis and dysphasia after a blunt injury on the head and neck. He had a complete left internal carotid artery occlusion which was diagnosed through angiography. We treated the patient with an antiplatelet agent and rehabilitation. Six months later, he regained motor power of right extremities, language ability, and revisualization of internal carotid artery on the follow-up magnetic angiography. We confirmed a recanalization of injured internal carotid artery on the conventional cerebral angiography which was performed one year later. We suggest conservative treatment with serial angiographic studies as a possible option of traumatic internal carotid artery occlusion even though there is hemodynamic instability.

**KEY WORDS :** Blunt injury · Carotid artery injury · Internal carotid artery dissection.

## INTRODUCTION

The occurrence of a blunt carotid injury (BCI) is very rare. Biffi<sup>1)</sup> reported that an incidence of BCI only constitutes 0.38% of all blunt traumas and up to 0.67% of motor vehicle accidents. Incidence of BCI is markedly lower with children than with adults. According to a report published by the US National pediatric trauma registry, 0.03% of internal carotid artery (ICA) occlusion developed among 57,695 patients with blunt trauma. This difference of incidence may be due to the increased vascular elasticity in children<sup>4)</sup>. The BCI carries a devastating outcome. Thromboembolic or hemodynamic infarction can be produced by BCI and symptoms vary in severity from transient ischemic attacks or focal neurological deficit to hemiplegia or coma. Morbidity and mortality have been reported to be 40-80% and 20-40% respectively<sup>8)</sup>.

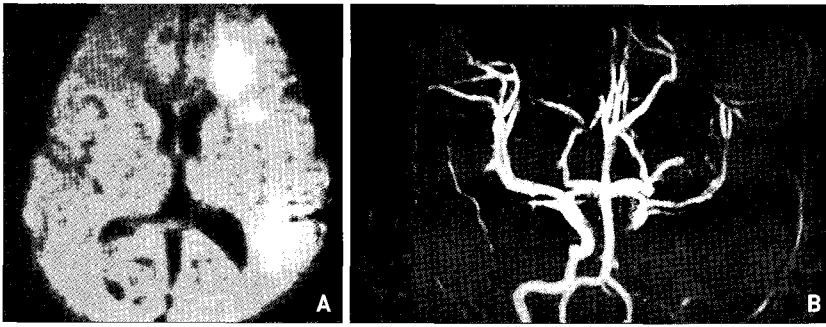
It is very difficult to make a treatment plan for complete ICA occlusion. There are several management options such as an antiplatelet agent, anticoagulation, stenting and / or bypass surgery. However, the optimal management remains controversial because the exact natural course of BCI has not been found yet. We report a rare case of traumatic cervical carotid artery occlusion that has been spontaneously recanalized after 6 months.

## CASE REPORT

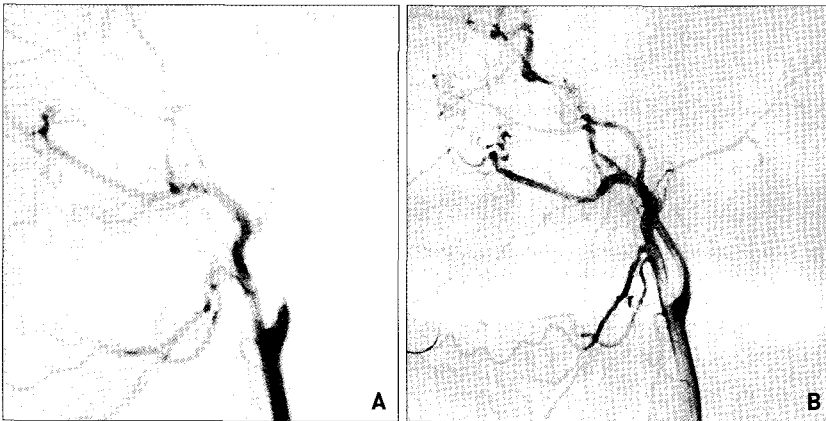
A previously healthy, 5-year-old boy was brought to the emergency room with a history of car accident. He had been crushed by an automobile, which was moving backward. His head had been run over by wheel. The physical examination revealed abrasions and skin defect on the left side of the face. His neurological examination was normal. The pupils were round, equal, and reactive. The optic discs were sharp. Motor strength was equal and symmetrical in all extremities. Computed tomographic (CT) scans did not reveal any definite intracerebral lesion.

Two days after the admission, his mental status was deteriorated to drowsy consciousness. Dysphasia and right hemiparesis gradually developed as well. Magnetic resonance imaging (MRI) revealed high signal intensity along the left middle cerebral artery and anterior cerebral artery borderzone on the diffusion weighted image, consistent findings of acute cerebral infarction. We could not identify the left ICA on the magnetic resonance angiography (MRA), which had been performed simultaneously (Fig. 1).

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**Fig. 1.** Brain magnetic resonance imaging and magnetic resonance angiography. There is acute infarction the border zone of left hemisphere (A) and decreased vascular flow on left middle and anterior cerebral artery (B).



**Fig. 2.** Cerebral angiography. Acutely tapered left proximal internal carotid artery (ICA) is demonstrated on left ICA angiography (A). It is definite evidence of traumatic ICA occlusion. 3 years later, the angiography shows that partially recanalization of left ICA and its blood supply on the left cerebral hemisphere (B).

We immediately began the process of adequate hydration and an antiplatelet therapy. Three days later, we performed cerebral angiography and it revealed complete occlusion of the left ICA with acutely tapering of proximal ICA (Fig. 2). We recommended bypass surgery, however, his parents refused the surgery. Then, we decided to treat medically with 50 mg of aspirin and rehabilitation therapy. Patient's neurological deficit had been gradually getting better. On the 14<sup>th</sup> admission day, he was able to walk independently, and say simple words and sentences. He was discharged from the hospital on the 16<sup>th</sup> hospital day.

After six months of rehabilitation, he regained motor power of right extremities and nearly-complete language ability. In order to decide on whether or not to perform cerebral revascularization, we did a follow-up paired CBF study using hexylmethylpropylene amineoxime (HMPAO), and acetazolamide and it showed marked improvement of vascular reserve capacity on the left hemisphere (Fig. 3). Then, we did magnetic resonance angiography (MRA) which showed reestablishment of the left ICA circulation (Fig. 4). We discontinued an antiplatelet therapy due to the possibility of unexpected complication from long term aspirin therapy

with children, such as Rey's syndrome.

One year later, he underwent cerebral angiography for establishing the next treatment plan. It showed that ICA had been recanalized and the recanalized portion was stenotic (lumen diameter less than 60% of normal ICA). There was suspected pseudoaneurysm in the petrous portion of the ICA and blood flow from injured ICA irrigated the left cerebral hemisphere properly (Fig. 2). No further treatments were given. He is now on regular check-ups and planning on taking a follow-up angiography.

## DISCUSSION

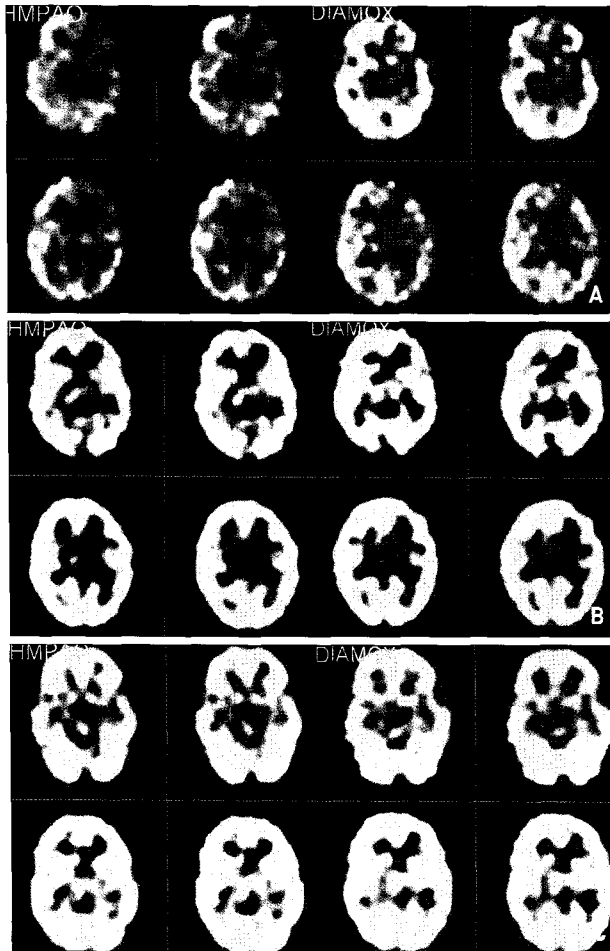
Blunt carotid injury, which can be caused by direct impact on the carotid artery or acute stretching, may cause tear of the carotid artery intima and can result in intimal dissection or subintimal hemorrhage, which may develop into complete or incomplete internal carotid artery occlusion.

Crissey<sup>3)</sup> described blunt carotid artery injury mechanism as follows;

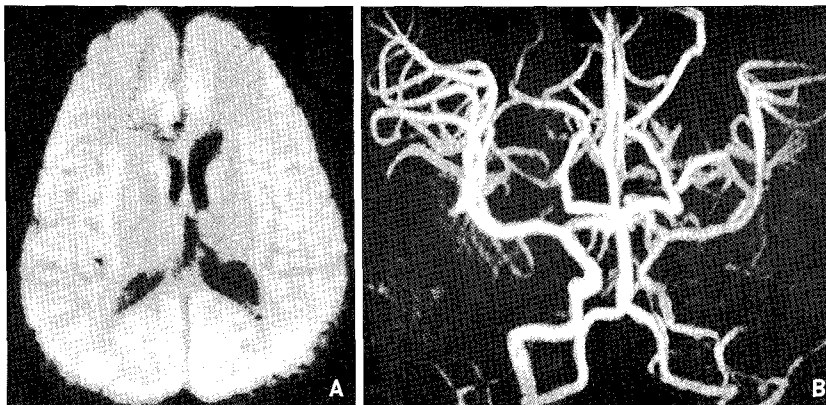
Type 1 : direct blow to the neck, Type 2 : stretching of the internal carotid artery with neck hyperextension or rotation, Type 3 : intra-oral injury, Type 4 : injury related with skull base fracture. In this case, initial findings of angiography showed that complete occlusion of ICA beginning 2 centimeters distal to the common carotid artery bifurcation, which was tapered distally and was suspected as arterial dissection (Crissey type 2). Although this finding was more compatible with direct blow or hyperextension with rotation rather than with carotid canal injury, final angiography showed that main lesion had located in the petrous bony segment indicating that injury had occurred in the carotid canal associated with basilar skull fracture (Crissey type 4). We believe that two above injury mechanisms were combined in this case.

As the incidence of traumatic ICA occlusion is low and the diagnosis of spontaneous recanalization needs conventional angiography, little is known about the natural course of traumatic ICA occlusion and its possible recanalization. Biffel et al.<sup>1)</sup> reported that spontaneous recanalization of traumatic ICA occlusion was very rare phenomenon, complete ICA occlusion was associated with a 44% of stroke

rate, and uniformly persisted on follow-up angiography. Spontaneous recanalization of nontraumatic ICA occlusion



**Fig. 3.** Single photon emission computed tomography (SPECT). The perfusion decrease in left internal carotid artery territory with decreased vascular reservoir (A). After 7 months later, Brain SPECT reveals that markedly improved state of rest perfusion and vascular reservoir compared with last study (B). 3 years later, Brain SPECT shows that improvement of perfusion on left cerebral hemisphere compared with last study (C). (HMPAO; hexylmethylpropylene amineoxime).



**Fig. 4.** After 7 months later, brain magnetic resonance imaging and magnetic resonance angiography show that no newly developed ischemic lesion (A). Improvement of vascular flow on left internal carotid artery is seen (B).

is not a rare phenomenon as compared to traumatic ICA occlusion. The incidence of recanalization has been reported to range anywhere between 17% and 67%<sup>6,9)</sup>. When considering the extent of occlusion and length of thrombotic propagation, we believe that the dissection is more severe than nontraumatic ICA dissection in patients suffering from traumatic ICA occlusion. This difference may relate with different incidence of recanalization between traumatic and nontraumatic ICA occlusion.

The mechanism of recanalization is unclear. Several mechanisms, including relieving of the acute vasospasm, distal embolization of an occlusive clot, and spontaneous intravascular lysis of the thrombus have been proposed to explain recanalization of nontraumatic ICA occlusion. Because there were no evidence of vasospasm or distal embolization on the angiography and MRI, autolysis of thrombus seems to be the most probable explanation for recanalization in this case. Good collateral flow to occluded proximal ICA may be another important factor for recanalization<sup>5,7)</sup>. In this case, angiography showed collateral flow from the posterior communicating artery to the proximal ICA. Second angiography revealed dilated multiple channels of vasa vasorum around stenotic portion of the recanalized ICA. The vasa vasorum can be a source of collateral circulation after vessel occlusion secondary to arterial dissection<sup>2)</sup>. As in the present case, we believe that these collaterals have been important factor to recanalize the occluded ICA. Since collaterals from the posterior circulation frequently persist in children, spontaneous recanalization of the occluded ICA may be more common in children than in adults.

The management of ICA occlusion remains controversial. To make a correct decision, we should consider the cause of ischemia (emboli or hemodynamic instability) first. In this case, there were hemodynamic infarctions resulting from completely occluded ICA and decreased vascular reservoir.

We had initially planned revascularization but we decided to treat with an antiplatelet agent because his symptoms had been gradually improving without any active treatment. In the case of traumatic ICA occlusion, if there is no clinical aggravation, conservative treatment with serial radiologic evaluation seems to be one of treatment options.

Although ICA occlusion had been recanalized spontaneously, it did not seem to be stable. Because there is a possibility of the emboli or the restenosis, the development of secondary

hemodynamic or embolic infarction should be considered even though hemodynamic stability was secured by spontaneous recanalization. Accordingly, if there is stenotic portion or pseudoaneurysm on the injured ICA, such as this case, serial assessment of injured ICA is recommended strongly.

## CONCLUSION

We report a case of spontaneous recanalization of traumatic ICA occlusion associated with basilar skull fracture. Internal carotid artery injury should be considered and careful evaluations of the carotid artery seem to be mandatory in patients of head trauma associated with skull fracture or blunt neck injury. In case of traumatic ICA occlusion, the possibility of spontaneous resolution should be considered even if there is a hemodynamic compromise. Anticoagulation with serial ICA assessment could be the one of treatment options in case without neurological deterioration.

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