A Case of Ischemic Optic Neuropathy Developed Eleven Days after an Aortic Arch Replacement

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We present a case of ischemic optic neuropathy (ION) developed 11 days after an aortic arch replacement in a 59 year-old male who had a history of untreated hypertension. Thoracic CT revealed severe stenosis of the right common carotid artery with poor blood flow. Aortic clamping time was 96 minutes, and selective cerebral perfusion time was 48 minutes. The lowest hemoglobin concentration of venous blood during cardiopulmonary bypass was 8.1 g/dl and the lowest arterial pressure was 60 mmHg. Due to pulmonary congestion, artificial ventilation was required until 11 post-surgical days. After removal of ventilator, the patient's consciousness was clear with no motor paralyses evident. However, the patient complained of blurred vision on that day. Bilateral papillae of the optic fund were pale. Atrophy of the papillae was also noted. Visual evoked potential was bilaterally flat suggesting bilateral optic nerve disturbance. The diagnosis of ION was made by ophthalmologist and neurologists. We speculated that low hemoglobin level during cardiopulmonary bypass was not the sole etiology of ION. Untreated hypertension, low blood flow through internal carotid artery and prolonged mechanical ventilation were also deteriorating factors of ION in this patient. We should be alert to prevent ION in such a complicated case.

Key words: ischemic optic neuropathy, aortic arch replacement, dissection of the thoracic aorta, cardiopulmonary bypass

INTRODUCTION

Ischemic optic neuropathy (ION) is one of important post hemorrhagic neurological complications. This ophthalmologic impairment sometimes develops after cardiopulmonary bypass. The incidence of ION has been reported to be between 0.06 and 1.3% after cardiac surgery using cardiopulmonary bypass [1, 2]. There is no reliable and effective treatment of this condition. Hence, measures to avoid ischemic optic neuropathy are important. Here we present a case of ION after cardiopulmonary bypass surgery followed by prolonged mechanical ventilation and discuss precipitation factors.

CASE PRESENTATION

The patient was a 59 year-old male. Sudden back pain developed one day in July in 2006. He visited a local clinic, where no abnormal findings on an electrocardiogram were present. Two hours later, he visited a hospital because of persistent back pain. At that hospital, dissection of the thoracic aorta was suspected on the computed tomography (CT). He was subsequently referred to our hospital.

The patient was a well nourished man, 166 cm in height and weighing 67 kg. Blood pressure on the right arm was 200/110 (systolic/diastolic) mmHg. No motor or sensory disturbances were found on neurological examinations.

He had a history of surgical treatment of duodenal ulcer. He had been prescribed medication for liver chirrhosis but not for hypertension. He did not have an experience of syncope.

Laboratory findings on admission were as follows: WBC 16,100/ml, RBC 464 x 10^4/ul, Hb 14.8 g/dl, Plt 13 **x** 10^4/ul. APTT 27 s, PTINR 0.98, D-dimer 11.8 μg/dl, BUN 11, creat 0.9 mg/dl, CPK 57 mg/dl, AST 312 IU/l, ALT 22 IU/l, r-GTP 109 IU/l, LDH 250 IU/l, glucose 142 mg/dl, Na^+ 143 mEq/l, K^+ 3.3 mEq/l, Cl-104 mEq/l.

As shown in Fig. 1, the thoracic CT revealed dissection of the thoracic aorta from the ascending part to the upper abdominal part at branching renal arteries. Entry of the dissection was located at a small curvature of the aortic arch near the aortic valves. Most of the major arteries such as the left common carotid artery, the left subclavicular artery, the mesenteric artery, superior mesenteric artery and the renal arteries, developed from the true lumen of the dissected aorta. Blood supply to the abdominal organ was good.

Fig. 2A shows a reconstructed image of the neck CT. Dissection of the aneurysm in the right brachial artery was also seen. The true lumen of the right common carotid artery was severely narrowed (almost 90% stenosis), and blood supply to the right internal common carotid artery was poor. These findings are more apparent in a coronal image of the neck CT (Fig. 2B). An echocariography revealed arterial regurgigation

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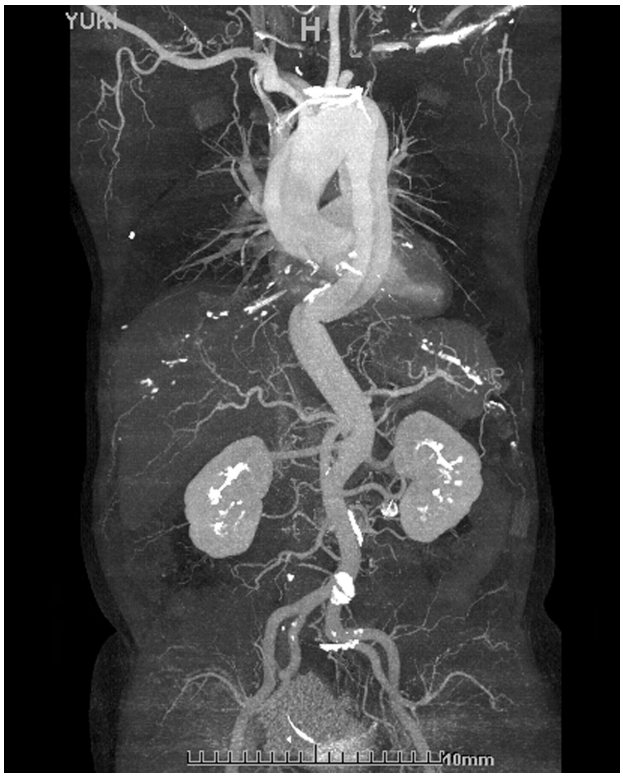

Fig. 1 Enhanced thoracic CT at the time of visit.

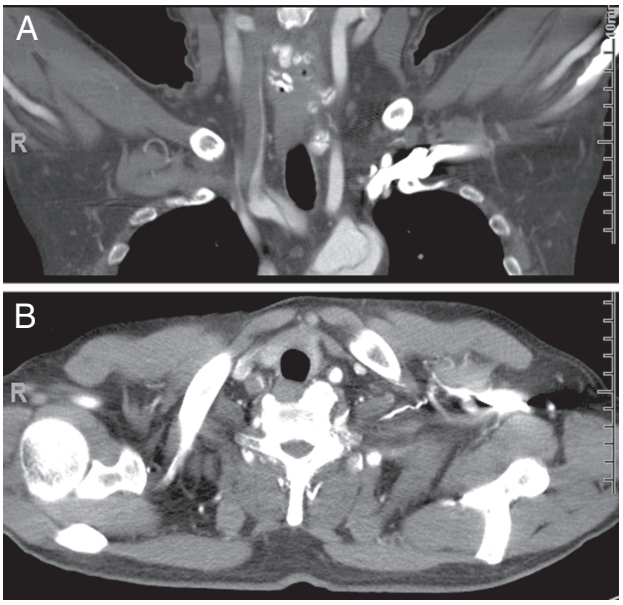

Fig. 2 Reconstructed image (A) and a coronal image of neck CT.

of third degree, but no pericardial fluid was found. From these findings, we diagnosed his condition to be DeBakey type I acute aortic dissection associated with 3rd grade arterial regurgitation. An urgent surgery was decided.

Since the aneurysm was located at the aortic arch, we chose selective cerebral perfusion for extracorporeal circulation. Blood from the extracorporeal circulator was supplied from the bilateral subclavian arteries and right femoral artery. Entry of the dissection was found at smaller curvature of ascending aorta and aortic arch. Approximately a half or the aortic arch was replaced with a blood vessel prosthesis.

Aortic clamping time was 96 minutes, and time of the selective cerebral perfusion was 48 minutes. During selective cerebral perfusion, flow rates through each of the arteries were as follows: 500 ml/min (right subclavian artery), 400 ml/min (left subclavian artery), 300 ml/min (left common carotid artery) and 800 ml/min (right femoral artery). Cerebrovascular hemoglobin oxygen saturation measured with near-infrared spectroscopy (INVOS 5400, Somanetics, USA) during the selective cerebral perfusion was around 60% at both sides, and the lowest was 42% (left) and 49% (right) respectively. Lowest rectal temperature during the surgery was 25°C, and hemoglobin concentration of venous blood during cardiopulmonary bypass was 8.1 g/dl. The lowest arterial pressure was 60 mmHg. Total blood loss was 1,803 ml, and urinary excretion during the surgery was 1,290 ml.

Generalized edema developed after the surgery, and it was treated with intravenous diuretics. Because of pulmonary congestion, artificial ventilation was required until 11 post-surgical days. Temporary blunted light reflex was observed 10 days after the surgery. After removal of the ventilator, his consciousness was clear and no motor paralyses were found. Eleven days after surgery, he complained of blurred vision. He was examined by a neurologist and an ophthalmologist on the day. Applanation intraocular pressure was 11−16 mmHg bilaterally. His fundi were normal.

Fig. 3 shows optic funds of the patient on postoperative day 29. There was slight arterial sclerosis of retinal arteries. Bilateral opic disk were pale. Atrophic optc disk without cupping was also noted. Visual evoked potential was bilaterally flat suggesting disturbance of bilateral optic nerves.

Results of an electroretinogram and fluorescein angiogram were normal.

His arm-to-retina circulation time was delayed 20 seconds.

Fig. 4 shows a reconstructed image of the head CT 14 days after surgery. Compression of the right carotid artery had been removed and good blood circulation was seen. An ultrasonic doppler measured the blood flow of the right internal carotid artery (ICA) to be 53.3/15.9/25.9 (max/min/mean) cm/s, that of the left ICA was 44.6/12.1/20.0 cm/s, that of the right vertebral artery (VA) was 40.1/11.4/19.0 cm/s, and that of the left VA was 46.0/8.8//18.4 cm/s.

Fig. 5 shows magnetic resonance angiogram of the brain arteries. Perfusion of the ophthalmicl artery was not disturbed. There were no findings suggestive of cerebral infarction or hemorrhage on brain MRI.

Two months later he was still totally blind. His pupils were 6 mm in diameter and did not react to light. Applanation intraocular pressure was 10 mmHg bilaterally.

From these findings the neurologist and ophthalmologist e diagnosed the condition to be ION.

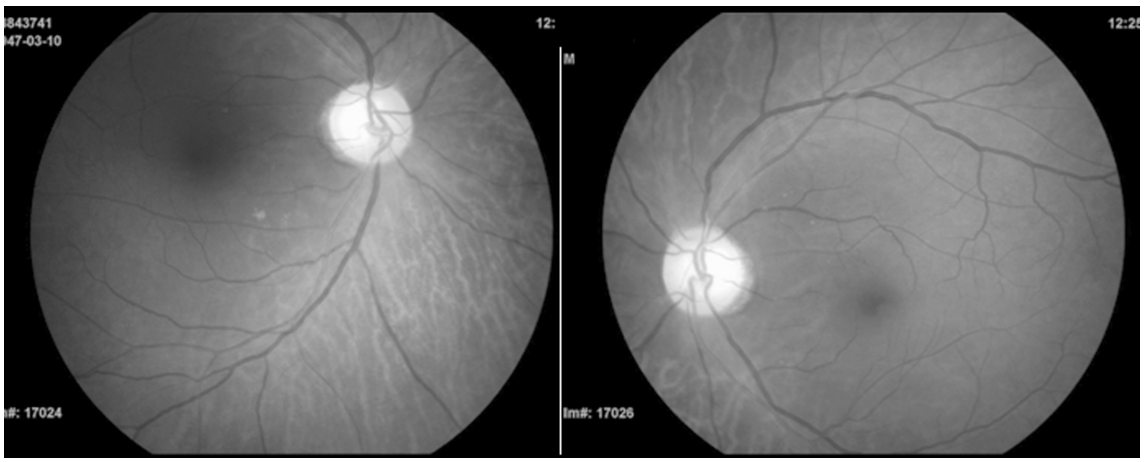

Fig. 3 Optic fund of the patient at the onset of ION.

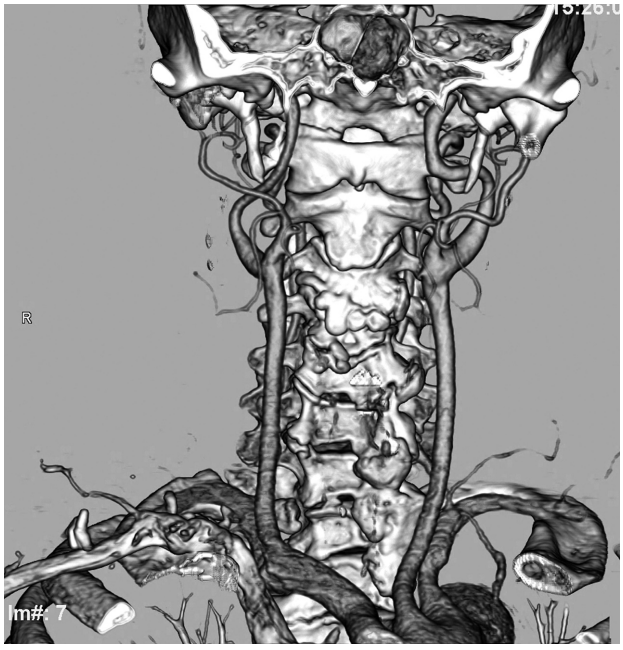

Fig. 4 Head CT at the onset of ION.

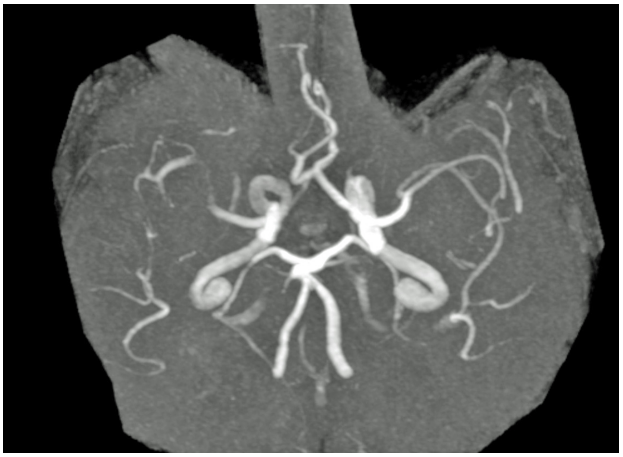

Fig. 5 Magnetic resonance angiogram of the brain arteries at the onset of ION.

DISCUSSION

ION is a rare complication of cardiovascular surgery using a cardiopulmonary bypass. Treatment options include the use of corticosteroid medications, reduction of intraocular pressure, and optic nerve fenestration [1], although most of them do not provide promising results. Thus, alert and protection of this condition is important. Busch *et al.* [3] suggested the following risk factors of postoperative of ION; history of glaucoma or other ophothalmological problems, prolonged cardiopulmonary bypass-time and myocardial ischemia, generalized edema during cardiopulmonary bypass, excessive hemodilution with low hemoglobin and hematociritt, hypo-or hypertension, systemic hypothermia, need for vasoactive medication. Among them relative drop in hemoglobin during the perioperative period is thought to be important [2]. Larkin, *et al.* [4] measured intraocular pressure in 24 patients undergoing elective cardiopulmonary bypass surgery. When the bypass circulation began, there was a rapid rise of the intraocular pressure, and this rise was maintained for about 25 minutes. A simultaneous drop in arterial perfusion pressure and packed cell volume occurred. They suggested that marked haemodilution is responsible for this effect, through increased ocular blood flow and low colloidal osmotic pressure.

In our case hemoglobin level fell from 14.8 to 8.1 g/dl and hematocrit level fell from 43 to 25.3% during cardiopulmonary bypass.

However, this anemic level is not unusual when comparing with other cardiopulmonary bypass operations, and thus anemia during cardiopulmonary bypass may not be the sole etiology of INO in our patient.

ION can be classified into arteretic and nonareteretic. Since pale rather than hyperemic optic disc swelling is a finding of arteritic ION, the major isch-

emic process of our patient may be the obstruction of the optic artery. Wong and Mitchell [5] described that hypertension as a major risk factor for the development of retinal vascular diseases, such as retinal vein and artery occlusion, and ischemic optic neuropathy. Thus untreated hypertension must be a precipitating factor for ION development in our case. Moreover, it was found that the true lumen of the right common carotid artery was considerably narrowed before surgery. Although many IONs were found at perioperative term, his visual disturbance was noted 11 days after surgery. During this time, generalized edema was developed and mechanical ventilation was performed. Both generalized edema and venous congestion are the other mechanisms and deteriorating factors for ION [6]. As known in spinal surgery, severe venous occlusion is necessary to develop ION in most cases. However, Kudo *et al*. [7] reported a case of anterior ION after massive fluid resuscitation.

Rizzo *et al*. [8] examined patient who awoke with profund bilateral visual loss after coronary bypass surgery. The optic disk appears normal in the phase of acute visual loss. Eight months later patient had bilateral optic atrophy wihtout cupping. The patient probably suffured intraoperative infarction of retrobulbar segments of both optic nerves, producing posterior ischemic optic neuropathy(PION). The use of the pump-oxygenator and anemia may have been a contributing factor in this patient.

PION results from infarction of the retrobulbar segments of optic nerve. It differs from the more prevalent and familiar anterior ischemic optic neuropathy(AION) in that the optic disk appears normal in the phase of acute visual loss. By contrast, disk edema is a constant finding in acute AION.

In our patient, his optic disk was normal in acute phase and atrophy in chronic phase, producing PION.

PION developed after cardiopulmonary bypass surgery. There was no severe ischemia or hypovolemic shock during surgery. History of hypertension and postoperative edema with prolonged mechanical ventilation may be additional factors for ION occurrence. We should be alert to maintain optic blood supply in patient with hypertension and lower cerebral circulation.

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