In the present report, a case complicated with Horner’s syndrome after off-pump coronary artery bypass (OPCAB) was presented. This case showed ptosis and miosis in the left eye promptly after OPCAB. No abnormal neurological findings other than Horner’s syndrome were observed in postoperative examinations including head magnetic resonance imaging (MRI), and this case was thought to have Horner’s syndrome as a complication after cardiac surgery through median sternotomy. (Ann Thorac Cardiovasc Surg 2006; 12: 113–5)

Key words: Horner’s syndrome, off-pump coronary artery bypass, peripheral nervous system complications

Introduction

Brachial plexus injury and Horner’s syndrome have been reported as peripheral nervous system complications after median sternotomy. Horner’s syndrome occurs in association with brachial plexus injury in many cases, and it is relatively rare that Horner’s syndrome is independently observed. In the present report, we presented a case complicated independently with Horner’s syndrome after off-pump coronary artery bypass (OPCAB), and discussed the case by using references.

Case Report

A 70-year-old female started to have exertional chest discomfort from the beginning of 2003. Coronary angiography (CAG) performed in July 2004 showed a triple vessel disease, and she was sent to our hospital to receive coronary artery bypass (CAB). Preoperative head CT or magnetic resonance imaging (MRI) showed no findings of cerebral infarction, and head MR angiogram indicated no significant stenosis in the right and left internal carotid artery or vertebral artery system. No neurological abnormalities were preoperatively observed. The operation was performed on October 26, 2004. After endotracheal intubation, a Swan-Ganz catheter was inserted from the right internal jugular vein. The intraoperative body position of the patient is shown in Fig. 1. The upper extremities were abducted and placed on triangle-shaped sponges. No pillow was placed under the back adjacent to the shoulder bones. When the internal thoracic artery was harvested, the chest wall was elevated using a Favaloro retractor. The quadruple OPCAB, using the right internal thoracic artery to the left anterior descending artery, the composite graft (the left internal thoracic artery and the left radial artery) to the obtuse marginal branch and the posterolateral branch, saphenous vein graft to the right coronary artery, was performed. Postoperative hemodynamics was stable, and an endotracheal tube was removed 4 hours after operation. She was transferred from the intensive care unit to a general ward one day after operation (day 1). After her eyes were closed for a long time on day 1, ptosis in the left eye was noticed on day 2. She was diagnosed with Horner’s syndrome by a neurological physician on day 7. Head MRI performed on day 10 showed no pathologic lesions. There were no neurological findings other than ptosis and miosis. We did not
treat her specifically for Horner’s syndrome. Patency of all bypass grafts was confirmed by angiography performed on day 16. She was discharged on day 20. When she visited our hospital as an outpatient on day 31, no ptosis or miosis was observed.

Discussion

It has been reported that the frequency of Horner’s syndrome as a complication after cardiac surgery is 0.2 to 7.7%, which may indicate that Horner’s syndrome is not a very rare complication. Some patients with Horner’s syndrome after cardiac surgery might be overlooked since a lot of patients did not show all the symptoms or lacked subjective symptoms.

Horner’s syndrome lacks function of the sympathetic nerve controlling the smooth muscle of eyes, and its prominent symptoms include ptosis, miosis, facial anhidrosis, and enophthalmos. The sympathetic pathway that reaches the eye is as follows. First-order neuron arises from the hypothalamus, and reaches the spinal cords at the level of C8 to Th2 through the mid brain, pons and medulla; the pathway then becomes the second-order neuron at the spinal cord, and reaches the superior cervical ganglion through the stellate ganglion; it then becomes the third-order neuron, and reaches the perspiratory gland and cutaneous vessels in the face and eyes. Therefore, sites of lesion and disease that cause Horner’s syndrome can be classified as follows. Infarct, hemorrhage and inflammation of the thalamus or brainstem, invasion and metastasis of a malignant tumor, and cervical injury are given as disorders of the first-order neuron; Pancoast’s syndrome, a mediastinal tumor and aortic aneurysm as those of the second-order neuron; and a cervical lymph node mass and thyroid tumor as those of the third-order neuron.

The causes of Horner’s syndrome after cardiac surgery are speculated to be the same as those of brachial plexus injury, since the sympathetic pathway that reaches the eye comes close to the peripheral nerve pathway of upper extremities.
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per extremities in the neck and superior chest. The following are given as causes of brachial plexus injury after surgery; injury associated with the first rib fracture,4) injury associated with internal jugular venous puncture on insertion of a catheter,7) and oppression and traction of the nerve associated with operation of thoracotomy or body position during surgery (especially the position of upper extremities).5,8-11) The relation of neurological disorder with harvesting of internal thoracic artery,5) and abduction of upper extremities10) has been particularly pointed out. Barbut et al.2) suggest that the patients with hypertension and diabetes tend to be at greater risk for developing Horner’s syndrome, and the syndrome may occur due to ischemia after the neurovascular refraction during thoracotomy in patients with severe arterial sclerosis. Since our case showed Horner’s syndrome in the left side, the causes had no relation with internal jugular venous puncture. Abduction of the left upper extremity, elevation of chest wall for harvesting of the left internal thoracic artery, and opening width of a retractor might be involved.

It has been suggested that Horner’s syndrome after cardiac surgery occurs with brachial plexus injury in many cases. As shown in Table 1, some investigators report that no brachial plexus injury patients are complicated with Horner’s syndrome,8) and others that not less than half of patients showing brachial plexus injury are complicated with Horner’s syndrome.5) Brachial plexus injury occurs with a high frequency as a complication after cardiac surgery, compared with Horner’s syndrome. This case was complicated independently with Horner’s syndrome.

In our institution, we always kept a slight bent position of the elbow without hyperabduction of the upper extremity, so that the brachial plexus was not excessively extended. The frequency of Horner’s syndrome and brachial plexus injury after cardiac surgery might be decreased through various devices, but could not be completely prevented.

Table 1. Reports on brachial plexus lesion and Horner’s syndrome after cardiac operation

<table>
<thead>
<tr>
<th>Authors/date/ref, no</th>
<th>Number of cases</th>
<th>Brachial plexus lesion</th>
<th>With Horner’s syndrome</th>
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<tr>
<td>Stangl R et al./1991(8)</td>
<td>201</td>
<td>13</td>
<td>0</td>
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<td>Lederman RJ et al./1982(1)</td>
<td>421</td>
<td>23</td>
<td>1</td>
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<tr>
<td>Vahl CF et al./1991(5)</td>
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<td>6+3</td>
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<td>Benecke R et al./1988(2)</td>
<td>1,585</td>
<td>22</td>
<td>12</td>
</tr>
</tbody>
</table>

References