A graft was utilized for vertebral reconstruction. Titanium plate was used for the fixation of cervical spine. The patient has done well afterwards, but in the second year of her follow-up a recurrent mass was diagnosed at the same location involving the RCA bulb/right ICA. So a third operation was undertaken, but this time the mass and carotid artery within were removed completely together. The carotid artery was reconstructed with No 6 mm polytetrafluoroethylene (PTFE) graft. Pathologic studies from the specimens found tumor cell scattered at several sites on the wall of the artery [Figure 2].

Chordoma is a rare bone neoplasm (1-4% of bone malignancies), which originates from the embryonic notochord.[1] Cervical chordoma, arising in the cervical spine, grows progressively into the soft tissue of the neck. The symptoms become evident by the compression of the chordoma to the adjacent structures. Surgical resection of the tumor is curative if completely removed, due to its low-grade malignancy and non-metastasis characteristics.[2] The two consecutive operations (with anterior and posterior approaches) before the recurrence operation, were devised to fully remove the tumor, for complete resection of the chordoma and reconstructing the cervical spine and carotid artery in our patient.

Carotid arteries may be complicated by the neighboring tissue tumors. These tumors may cause compression, kinking, flow restriction, turbulent jet flow, intimal ulcerations and micro thromboembolism into the carotid artery circulation.[3] Carotid artery involving by chordomas are rare cases, however, a few reports have been presented in the literature.[1] Recurrence rate of chordoma may reach up to 30%, predominantly from the local surgical area.[4] However, for this case in which the huge tumor spanned the whole neck from posterior to anterior, a two-staged operation was planned. Priority was given to the carotid artery reconstruction where the neurological findings were prominent. In the first stage, the chordoma was removed from the artery using deep periadventitial (white-line) dissection, relying on its low-grade; malignancy, potential for metastasis and invasive features. This case revealed that the recurrence is highly possible due to the chordoma cells remaining residual into the artery wall.

In our opinion the carotid artery complicated by chordomas should be replaced with grafts due to the high recurrence and infiltration capability of chordomas.

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References

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Oculomotor neuropathy following tetanus toxoid injection

Sir,

Anti-tetanus toxoid, a widely used prophylactic...
conduct in patients with open wounds, is considered to be very safe. However, there have been several reports of peripheral neuropathy occurring hours to weeks after its injection; the estimated incidence is 0.4 per million administered doses of the toxoid. These reports are consistent with neuropathy as a manifestation of immune complex disease. There are very few reports of cranial nerve palsy following tetanus toxoid vaccination; the nerves involved include the optic, facial and auditory nerves. Most of the reported cranial nerve palsies were reversible and developed within a few days of the vaccination.

A 16-year-old girl with no previous remarkable medical illnesses presented to our clinic complaining of blurred vision in her right eye that started a few hours after she had received an injection of anti-tetanus toxoid for a leg wound sustained in an injury. She also experienced mild right frontal headache. On the day following the injection she suffered double vision, especially when looking down and to the left. Her family noticed abnormal alignment of her eyes and mild drooping of the right eyelid.

On examination there were right eyelid ptosis and anisocoria (6 and 3 mm right and left pupil, respectively); the right pupil reacted weakly to light. This was associated with weak upward, downward and inward movement of the right eye.

The patient was admitted to look for the cause of her oculomotor nerve palsy. Laboratory results including a complete blood count, erythrocyte sedimentation rate, C-reactive protein and kidney- and liver function tests were all within the normal range. Magnetic resonance imaging (MRI) and MR angiography (MRA) returned no significant findings [Figure 1]. Although we recommended digital subtraction angiography (DSA) to rule out third cranial nerve compression by an internal carotid-posterior communicating artery aneurysm her family refused the procedure. Lumbar puncture revealed normal opening pressure; cerebrospinal fluid (CSF) findings were normal (2 lymphocytes/mm³, PMN 0, protein 38 mg/dl, glucose 75 mg/dl) and there was no oligoclonal band. We interpreted the clinical findings to be reflective of a rare toxic neuropathy secondary to tetanus toxoid injection and she was treated with intravenous methylprednisolone 500 mg twice a day for 7 days.

During her hospital stay she showed significant symptom improvement and her third nerve palsy cleared on day 7 of the treatment.

Compression of the subarachnoid segment of the oculomotor nerve is the most common cause of single complete third nerve palsy. Pathologies affecting other segments of the third nerve (nuclear, midbrain, cavernous orbital) are usually associated with long tracts, partial third cranial nerve palsy and/or the involvement of other cranial nerves. The most common lesion to affect the third cranial nerve in the subarachnoid space is compression by aneurysm, basal meningeal infection, neoplastic infiltration and miscellaneous inflammatory lesions. The absence of meningeal irritation signs and the normal imaging and CSF studies in our patient made a diagnosis of aneurysmal compression, neoplastic or inflammatory infiltration of the third nerve unlikely. Instead, the fact that her third nerve palsy developed shortly after tetanus toxoid injection made us suspect that her symptoms were related to the tetanus vaccination.

Our review of the literature disclosed few reports of cranial nerve palsy after the injection of tetanus toxoid [Table 1]. As in our patient, the cranial nerve palsy in the reported cases started a few days after injection and resolved within a few weeks either with or without treatment.

To our knowledge, this is the first case of third cranial nerve palsy following the injection of tetanus toxoid. The exact mechanism(s) by which tetanus toxoid produces cranial nerve palsy remains unknown, however, it may be the direct toxic effect of the vaccine or an immune-mediated reaction. Risk factors and patients susceptible to this complication are also unidentified. The administration of steroids appears to be very helpful to hasten recovery, as was the case in our patient.
Letters to Editor

Table 1: Reported cases of cranial nerve palsy following tetanus toxoid injection

<table>
<thead>
<tr>
<th>Author, year</th>
<th>Involved cranial nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kharoubi, 2005</td>
<td>Facial nerve</td>
</tr>
<tr>
<td>Burkhard et al., 2001</td>
<td>Optic nerve</td>
</tr>
<tr>
<td>Basek, 1958</td>
<td>Vagus nerve (recurrent laryngeal nerve)</td>
</tr>
<tr>
<td>Bauer et al., 1957</td>
<td>Vagus nerve (recurrent laryngeal nerve)</td>
</tr>
<tr>
<td>Cutter, 1936</td>
<td>Auditory nerve</td>
</tr>
<tr>
<td>Present report, 2006</td>
<td>Oculomotor nerve</td>
</tr>
</tbody>
</table>

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