obvious interface between the tumor, cord, and nerve roots. The pathological examinations confirmed the diagnosis of lipoma. Postoperatively motor weakness worsened and developed loss of anal tone, and urinary incontinence. The incontinence improved one month after the operation. Motor power in both the lower limbs improved from grade 2 to grade 4 after six months of rehabilitation.

Lipoma of conus medullaris without spinal dysraphism in an adult is a rare entity and only seven cases have been reported in the English literature till 2002. Many causes such as proliferation of adipose cells, deposition of fat in the connective tissue, metaplastic differentiation of persisting embryonic meninges, and abnormal embryologic development (dysraphism) have been hypothesized for the development of these tumors.

The clinical presentations of intradural spinal lipoma are mainly related to the mass effect. Most of these patients have the symptoms for more than two years before the diagnosis. MRI is the best diagnostic modality to evaluate spinal lipoma and delineate the adjacent neural structures. The fat component can be easily confirmed by using the fat-suppression images. Surgical intervention is indicated for the patients with progressive neurological symptoms. The goal of surgery is not only to remove the tumor, but also to preserve the neurological functions. With improvements in the neurosurgical techniques, use of carbon dioxide laser has been shown to have surgical precision, and maximal removal of the tumor with minimal surrounding trauma and improved hemostasis. Use of carbon dioxide has less electrical interference with the intraoperative evoked potential recordings. However, as there is often no clear-cut margin between lipoma and the cord, aggressive total removal of tumor is hard to achieve and is also not recommended. Partial surgical excision with wide laminectomy, laminoplasty or duroplasty also has been advocated to treat the intraspinal lipoma.

The management of lipoma of conus medullaris without spinal dysraphism remains a challenge.

Cheng-Ta Hsieh, Jui-Ming Sun
Department of Neurological Surgery, Tri-Service General Hospital, National Defense Medical Center, Surgery, Songshan Armed Forces General Hospital, Taipei, Taiwan, Republic of China.

E-mail: nogor@mail2000.com.tw

References

Accepted on 06-08-2009

Ruptured anterior communicating artery aneurysm presenting with monocular blindness

Sir,
Subarchnoid hemorrhage (SAH) is the common
Letters to Editor

presenting feature of anterior communicating artery (Acoma) aneurysms. The usual locations of the aneurysms presenting with visual deficits are cavernous, carotid-ophthalmic, supraclinoid, paraclinoid, posterior communicating.\(^1\) Only a few cases of Acoma aneurysms presenting with visual deficits have been reported in the literature.\(^1-3\)

A 45-year-old female patient presented in emergency with sudden onset headache, vomiting, and right eye vision loss. Vision loss was sudden and complete. On examination, she was conscious and well oriented. Her vision and pupil on left side was normal, whereas on right side there was no perception of light; right pupil was dilated and nonreacting. She also had neck stiffness. Cranial computed tomography (CT) scan showed diffuse SAH in anterior interhemispheric fissure, suprasellar cistern, and bilateral sylvian fissures [Figure 1a]. CT angiography showed a large pedunculated Acoma aneurysm 75 × 45 × 35 mm\(^3\) with fundus directed anteroinferiorly, towards right side with 3 mm long and narrow neck (2 mm); and right A1 segment was attenuated [Figure 1b]. She was subjected to pterional craniotomy and clipping of Acoma aneurysm. Intraoperatively, the Acoma aneurysm was found to be projecting into right optic nerve. There was dense adhesion between the aneurysm sac and the optic nerve with evidence of hemorrhage into the nerve. Gently aneurysm was dissected and was clipped with an asculap permanent 6.5 mm curved clip. There was no intraoperative rupture and no temporary clipping was used. Postoperative period was uneventful. Vision in the right eye did not improve. At one year follow-up, patient was fully conscious and oriented, but the vision in the right eye had still not improved.

Ruptured Acoma aneurysms generally present with SAH and associated focal neurological deficits. The various causes of visual deficits include: Compression of the optic apparatus,\(^1,4\) leaking of aneurysm inside the optic nerve or chiasma,\(^2\) perianeurysmal inflammatory changes,\(^3\) thromboembolism, and vitreous hemorrhage (terson’s syndrome).\(^4\) Only a few ruptured Acoma aneurysms with visual deficits such as monocular blindness have been reported till date. In most of the patients visual deficits were related to chiasmal field defects because of the proximity of Acoma complex to the optic chiasma rather than optic nerve. Isolated involvement of one optic nerve resulting in monocular blindness has not been reported before. In the present case, monocular blindness was probably due to the direction of the growth of fundus of large Acoma aneurysm and compression of the optic nerve and also due to the hemorrhage into the optic nerve. Distortion or traction and indirect pressure on the optic nerves at the margins of the optic foramina also play an important role.\(^3\) Other factor is interference with the blood supply of the optic nerve or chiasma either by occlusion or distortion of the perforating arteries arising from anterior cerebral-communicating complex. In large Acoma aneurysms, the intraaneurysmal thrombosis may occlude the origins of the branch vessels supplying the optic nerves and chiasma and thus causing ischemia. Sometimes, if the optic nerve is adherent to the fundus of the Acoma aneurysm there may also be damage from hemorrhage into the neural substance.\(^3\) This was the most likely cause of monocular blindness in our case. This was also confirmed intraoperatively as fundus of Acoma aneurysm was compressing the optic nerve with hemorrhagic staining of the optic nerve. Patients with unruptured aneurysms may present only with progressive visual loss, due to the compression of optic pathway by enlarging aneurysmal sac or other causes. Thus neurologists, neurosurgeons, and ophthalmologists should have high index of suspicion and should consider unruptured Acoma aneurysm as one of the differential diagnosis for monocular blindness.

Alok A. Umredkar, Navneet Singla, Sunil K. Gupta

Department of Neurosurgery, Postgraduate Institute of Medical Education and Research, Chandigarh, India.

E-mail: alokumar@yahoo.co.in

DOI: 10.4103/0028-3886.59498

References


Figure 1: (a) Noncontrast computed tomography scan showing diffuse subarchnoid hemorrhage in interhemispheric, suprasellar, and bilateral sylvian cistern; (b) Computed tomography angiography showing a large Acoma aneurysm with fundus directed anteroinferiorly and towards right side with attenuated right A1 segment
Letters to Editor


Accepted on 02-07-2009


Intramedullary Ewing's sarcoma of the spinal cord associated with hydrocephalus

Sir,

Ewing's sarcoma is an uncommon malignant tumor and accounts for approximately 10% of the primary malignant bone tumors. [1] Spinal cord metastases is an extremely rare and only two cases have been reported. [2,3] We report a third patient.

A 28-year-old man presented with progressive weakness of both lower extremities of fifteen days duration and recurrent headaches, vomiting, fever and bladder of four days duration and bowel incontinence of one day duration. Neurological examination showed bilateral papilledema and abducens nerve paralysis. Flaccid areflexic weakness (motor power 0/5) in both the lower with sensory level at T11 for all the modalities of sensations. Cranial magnetic resonance imaging (MRI) showed communicating type of hydrocephalus. Spinal MRI showed abnormal signal focus (14.3 cm) at the level of T12 to L3 vertebrae [Figure 1a-c].

External ventricular drainage and exploration and decompression of spinal canal from T11 to L3 were performed. During the operation, spinal exploration found sclerotin of vertebra bodies from T11 to L3 as normal. The tumor was flesh and soft, with no clear boundary and peplom. The gross tumor was resected under micromanipulation. Both hematoxylin and eosin (H and E) and immunohistochemistry staining [Figure 2] made the diagnosis of Ewing's sarcoma. The patient recovered well. But the sarcoma recurred and metastasis nine months after the operation, and there was no more opportunity to treat the disease.

Ewing's sarcoma is a kind of osseous undifferentiated reticulocytic sarcoma, which was firstly reported in 1921 by James Ewing. It represents the second most common primary bone malignancy in childhood and adolescence, with an estimated annual incidence of 0.6 per million population. [4]

It was depicted that the Ewing's sarcoma was originated from immature marrow reticulum tissue, while more and more immunohistochemistry, electron microscope and cytogene studies on sarcoma indicate the tumor originated from neural ectoderm. [2,5] In this report, the tumor was originated from nerve tissue in the spinal cord. Neither clinical and MRI examination nor the operation indicated any relationship between tumor and sclerotin, and the spondylous was intact. Initial symptoms of intramedullary Ewing's sarcoma were completely similar to common tumor in the spinal cord, which included decreasing sensation or absence first and followed by motor dysfunction and difficulty of urination and defecation. However, intramedullary Ewing's sarcoma had an acute onset and a short course of disease, and a progressive aggravation of nerve damage, which caused the complete flaccid paralysis.

Figure 1: (a) T1-weighted sagittal magnetic resonance image (MRI) of the thoracolumbar spine (T12 to L3) reveals a well-defined lesion, hypointense to the spinal cord, located in the substance of the spinal cord; (b) T2-weighted sagittal MRI shows that the lesion is hyperintense compared with the spinal cord; (c) GD-DTPA enhancement scanning showed moderate heterogeneous enhancement and no abnormal signal in canalis spinalis from T12 to L3.