Case Report

**Recurrent posterior fossa meningioma in a young child: A case report**

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Abstract
16 years old boy - Mizanur Rahman presented with recurrent posterior fossa meningioma within 2 years of initial surgery. Meningiomas are generally benign slowly growing, circumscribed neoplasm arising from arachonoid cap cells with secondary attachment to dura. Meningioma constitute about 15-20% of all intracranial tumours. It commonly occurs in the forth to sixth decades of life. Females have meningiomas more often then males, having a ratio of 3:2. Posterior fossa meningiomas are uncommon in adult and very rare in childhood. Only 1.5% of meningioma occur in children. Microscopic total removal is the goal of surgery to avoid recurrence free survival. However recurrent meningiomas are common following subtotal or partial removal of tumour and usually from primary site. Here author presents the case that had recurred not from the primary site but from a regional area within short period of time interval. Both the primary and recurrent tumour were successfully managed by microsurgical approaches.

Key words
Meningioma, recurrent meningioma, posterior fossa.

Introduction
The meningioma is the neurosurgeon's "friend" and often his most enduring challenge. Meningioma is the second most common intracranial neoplasm following glioma. It is arachinooidal cap cell origin but attached with dura. Commonest locations are in the vicinity of arachinooidal granulation tissue and arachinooidal villi such as convexity and parasagittal and sphenoidal ridge location. Female are more commonly affected then male because of hormonal influence. Childhood meningioma are associated with neurofibromatosis or previous irradiation. The more age more chance of development of meningioma.

It also carries the possibility of "cure" in approximately 80% of cases. The posterior cranial fossa, the largest and deepest of the three cranial fossa containing the 10 pairs of cranial nerves, brain stem, cerebellum, vertebral and basilar artery and major cerebellar arteries. It is bounded infront by dorsum selle and clivus, behind by the squamous part of occipital bone and on each side petrous and mastoid part of temporal bone. Posterior fossa is penetrated by jugular foramen, internal acoustic meatus, hypoglossal canal. It is bounded above by tentorial cerebelli.

It communicates above by tentorial hiatus and below with spinal canal by foramen magnum. In the posterior fossa, meningioma can be arise from tentorial surface, suboccipital surface of cerebellum, cerebellopontine angle, pterocivclical junction, lateral petrosal bone, jugular foramen, foramen magnum, and fourth ventricle. Meningioma is well demarcated round or oval and frequently multiloculated. They are firm and pink and vary in consistency from soft to rock hard. Although the etiology of meningioma is still unknown, however tumour has arisen from repeated trauma, radiation and viral infection. The role of female hormone- progesterone, play a role in the genesis of meningioma, thus explaining the higher incidence of meningioma in women that accelerated growth during pregnancy. Chromosomal abberetions are regularly seen in meningiomas, meningioma cells often lose one copy of chromose. Neurofibromatosis

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and 2 (NF-1 & NF-2) may be associated with meningioma\textsuperscript{1,3,4}.

Posterior fossa meningioma is usually associated with obstructive hydrocephalus. Clinical symptoms most often related to hydrocephalus, long tract sign from brain stem compression, cerebellar syndrome and lower cranial neuropathy due to stretching and or ischemia of nerves\textsuperscript{1,4}.

Approach to the posterior fossa depends upon the site of pathology. Common approaches are:
1. Midline suboccipital craniectomy/craniotomy.
2. Lateral retrosigmoid suboccipital craniectomy.
3. Far lateral transcondylar approach.

When surgical resection is incomplete, meningioma will continuously grow unless it stop spontaneously or by any kind of therapeutics. The term recurrence should be reserved for those meningioma in which macroscopic total removal were achieved\textsuperscript{1-3}.

Several factors are responsible for the recurrence: Incomplete surgical removal, stimulation of tumour growth by successive operation and modification of histological aspects with dedifferentiation. CT scan detectable recurrence would take 6 months for very rapidly growing meningioma, 6 years of moderately growing meningioma and 11 years in very slowly growing meningioma\textsuperscript{4}.

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16 years old Mizanur Rahman presented to us with history of posterior fossa surgery 2 years back. Before his first surgery he had headache, vomiting, visual blurring. At that time MRI of brain revealed midline tentorial meningioma with obstructive hydrocephalus.

External ventricular drain(EVD): External ventricular drainage (EVD) was applied for management of HCP and Midline suboccipital craniotomy and microscopic gross total removal of meningioma was done in same sitting. Peroperative tumour found hard, gritty, nonsuckable, difficult to remove enmass and attached with undersurface of tent. Hence it was removed in piece meal following detached from the undersurface of tent.

Postoperatively patient recovers well and EVD was removed on 5\textsuperscript{th} postoperative day.

Histopathology revealed transitional variety of meningioma. After surgery his symptoms resolved within short time.

The patient had only one follow up in early postoperative period where he was found quiet normal except some visual blurring.
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For last 6 months his visual blurring progressively increased and leaded to bilateral blindness. He also developed left cerebellar syndrome in the form of left sided ataxia, intensity tremor, nystygus, dysarthria and dysphagia. He had left sided deafness but no facial weakness.

Repeat MRI of brain was done and found huge cerebellopontine angle menigioma with obstructive hydrocephalus.

This time he underwent lateral retrosigmoid suboccipital craniectomy and microsurgical gross total removal was done. Peroperative tumour found attached with petrosal dura beneath the internal acoustic meatal opening. It was tough, gritty, hard, less vascular, difficult to remove by microseissor. Hence it was removed by metzenbaum and BP blade number 11.

Postoperatively he had no facial weakness. His dysphagia, dysarthria, headache, ataxia reduced. He developed meningitis on 5th postoperative day which was successfully managed by intravenous vancomycin. His vision did not improved significantly because of secondary optic atrophy. His postoperative CT scan of brain revealed no evidence of residual tumour, little heamorrhagic contusion of middle cerebellar peduncle and resolving HCP.

Histopathology reported transitional variety of menigioma again.

Discussion

Meningioma is the 2nd intracranial neoplasm. Posterior fossa menionioma although rare, but the recurrence is very rare. Clinically it present with headache, vomiting, ataxia and visual blurring. As menigioma is slow growing so it causes compression and distortion of 4th ventricle, hence patient develops obstructive hydrocephalus. Clinically it is manifested by headache, vomiting and visual blurring. On funduscopy examination papilloedema is the commonest findings of raised I.C.P. Here we got secondary optic atrophy which was the consequence of severe papilloedema. Brainstem feature and cerebellar syndrome was due to compression of brain stem and cerebellum by the menigioma.4

Before going to manage the posterior fossa menigioma there are obvious role of management of HCP. Hydrocephalus can be managed by various way-such as VP shunt, Endoscopic 3rd ventriculostomy, External ventricular drainage (EVD). Commonly we are doing VP shunt and EVD. However endoscopic 3rd ventriculostomy is a new and advanced technique in our country. We did this procedure in some of our patient with good results. We couldnot do endoscopic 3rd ventriculostomy in this patient because at that time it was not available in 2004. Hence we managed him by EVD.

Posterior fossa menigioma can arise from cerebellar convexity dura, tentorial dura, petrosal dura, clival dura and tela choroiade of 4th ventricle. In the C.P angle menigioma it can arise either infront of IAM opening or suprameatal or inframeatal or postmeatal origin. It clinically presents with hearing problem, facial weakness, facial numbness, dysphagia, dyarthria, dysphonia due to lower cranial nerve palsy1.

Management of this C.P angle menigioma is same as traditional retrosigmoid suboccipital craniectomy and tumour removal by microsurgical technique. Commonest problem encounter was bleeding and potential injury to lower cranial and facial nerves.
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Simpson classify the extent of meningioma removal into 5 grades:-

- Grade 1: Complete removal with resection of dura and abnormal bone.
- Grade 2: Complete removal with coagulation of dural attachment.
- Grade 3: Complete removal without coagulation of dural attachment.
- Grade 4: Partial removal (>10% preserved).
- Grade 5: Biopsy.

The rate of meningioma recurrence depends upon extent of tumour resection and tumour histopathological grading. The more radical the operation less chance of recurrence. In Simpson's analysis the recurrence rate following Grade 1 surgery was 9% compared to 19% in Grade 2, 29% for Grade 3 and 44% for Grade 4.

Mushrooming, lobulated meningioma more likely to recur then round ones. The mean average doubling time is 205 days (50 days to 500 days) as noted by Jaaskelainen et al, hence it is not surprising to have high rate of recurrence. Multivariate analysis showed that risk factors of recurrence included coagulation of dural insertion, invasion of bone and soft consistency of tumour.

Conclusion

Rate of growth is fundamental factor for development of recurrence. Benign meningioma recur locally after 5 to 10 years. If a meningioma doubles in 1000 days it would take 11 years to be visible on CT scan.

Here we have discussed the recurrent posterior fossa meningioma in an unusual age. Recurrent developed not from the primary site but from regional area of posterior fossa called CP angle. We could managed successfully both the tumour by microsurgically which was documented by postoperative CT scan following each surgery where gross total tumour removal was evident in the scan. As patient presented with secondary optic atrophy before 2nd operation hence his vision did not improved following surgery but his other neurological feature did improved significantly without facial weakness and other sensory-motor deficit.

Reference