*Original Article*

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# Cerebellopontine Angle Epidermoid : A Clinical Study

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**Abstract**

*Objective* : Aim of this study is to correlate final outcome with the extent of surgical removal of cerebellopontine angle epidermoid, in term total or near total resection, recurrence and morbidity. Being a benign nature, neurosurgically shown a great concern because of close proximity and adherence to the cranial nerves, intracranial vessels and brain stem. *Methods*: We use our data from 21 consecutive patients of cerebellopontine epidermoid that were surgically treated in our institute from 1995-2012 were reviewed and the final outcome were assessed with emphasis on outcome after surgery, long term follow up results and anatomic relationship between the tumor and surrounding neurovascular structures. *Results*: The average age of patients were 33.14 years at onset of symptom and 36.33 years at the time of operation. The mean duration from onset of symptoms to surgery was 3.1 years. Of 21, 12 were female and 9 were male. Headache was present in 9 patients most common presentation followed by hearing impairmentin 7 patients. Most of them confined to cerebellopontine angle, hypodense non-enhancing lesion in 8 patients, 3 have supratentorial extension. Retromastoid approach in 14 and supine in 12 patients rest all in sitting position. Total removal done in 16 patients. After total removal transient cranial nerve palsy in 9 patients which were improved in 7 patients. Cerebrospinal fluid leak in 2 patients improved by repeated lumber puncture. Pulmonary embolism in1 patient. 18 were followed over mean period of 60 month, range from 1-11 years. 3 had recurrence ,two in near total excision, one in sub-total excision and 2 had persistent cranial nerve paresis. *Conclusion* : -Approach needed to be conservative in whom capsule is adherent to brain stem and neurovascular structures. Aggressive removal had transient but significant short term cranial nerve and brain stem dysfunction. Total removal of cerebellopontine angle epidermoids does not result in significantly increased long term morbidity and mortality and should be the goal of surgical treatment .

**Keywords:** Epidermoid Tumor ; Cerebellopontine and Reteromastoid Approach.

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## Introduction

Epidermoid cysts, also known as pearly tumors[39] or cholesteatomas are uncommon congenital lesions usually manifests at 3rd-4th decade[4] account for 0.2-1% of all intracranial tumors.[1,4,5] Cerebellopontine angle epidermoids constitute 40% of all CPA epidermoids and 6% of all CPA tumors.[2]

Their close proximity to exquisitely sensitive the brainstem, cranial nerves and vascular structures, tendency to adhere them pose difficulty in complete removal mean while partial removal risks recurrence and subsequent surgery.[3]

We summarize our experience with 21 patients of CPA epidermoids over a period of 15 years and correlate the final outcome with extent of surgical resection.

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## Material and Methods

Twenty-one patients were operated for CPA epidermoidtumor in one unit, Bombay hospital institute of medical sciences during last 15 years (1997-2012).

Inclusion criteria was cerebellopontine angle epidermoids and exclusion were epidermoids in other intracranial location and other cerebellopontine angle lesions.

Variables were total, near or subtotal removal of tumor.

Predictor of variables was cranial nerve palsy, Brain stem involvement and vascular damages.

Outcome of variable were depended upon recurrence, morbidity and mortality.

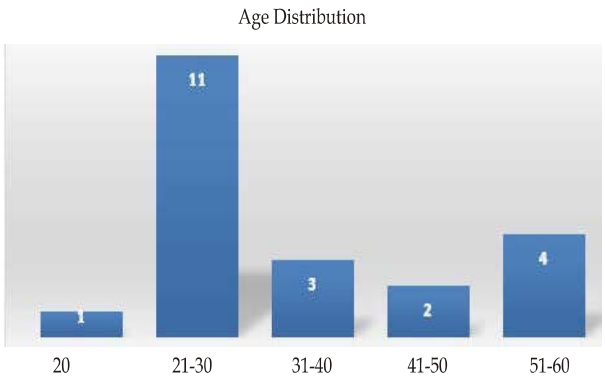


**Fig. 1**: Age (in years) distribution of patients

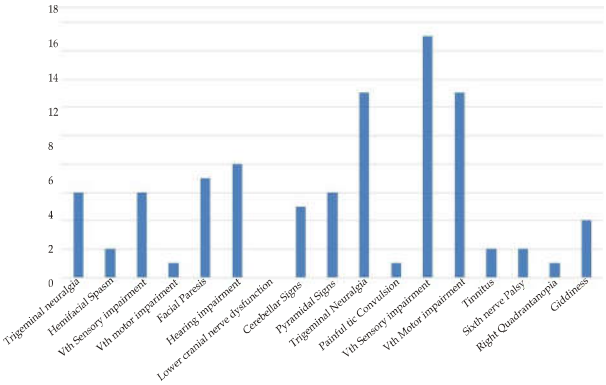


**Fig. 3**: Histogram showing frequency of clinical features

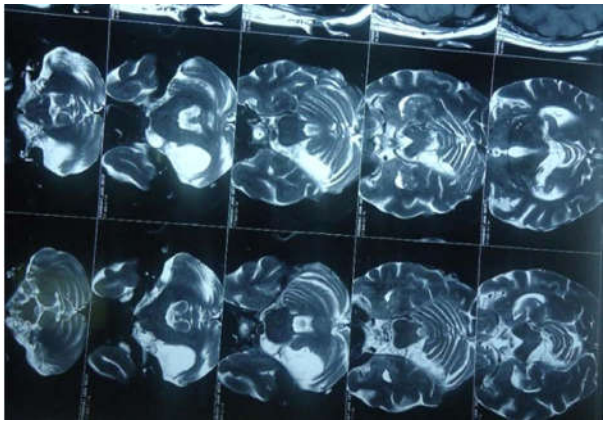

**Fig. 5**: Showing hypodense and non-enhancing lesion confined to the cerebellopontine angle on FLAR-MRI.

*Results*

The age group varied from 18-54 years, with most of them were between (20-40) (Fig1)

*Clinical Features*

The mean duration of symptoms was 3.1 years,12 patients(58%)were present within 1 year, ranges of duration of symptoms were from 2 month -15 years. (Table 1)

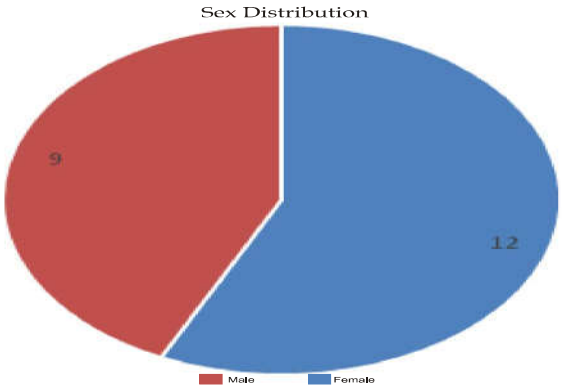


**Fig. 2**: Sex distribution of patients.

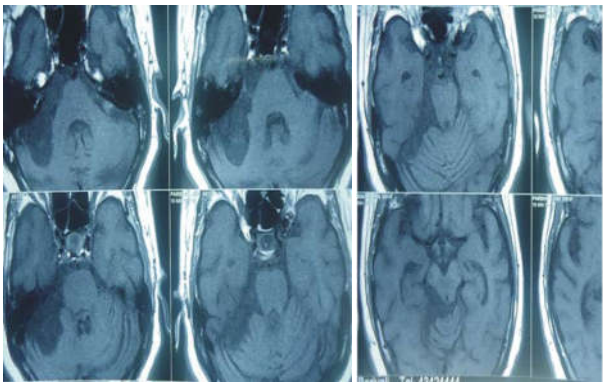

**Fig. 4**: showing hypodense and non-enhancing lesion confined to the cerebellopontine angle on MRI.

Only 4 patients (19%) reported symptoms lasting longer than 5 years. The most common symptom was headache (47%) followed by hearing impairment (38.5%) and in 28.5% patients had ataxia. Trigeminal neuralgia and seizure were presents in 23.8 %. Hemifacial spasm was present in 9 %. Rarely presents with cerebrospinal fluid rhinorrhea, facial paresis, giddiness and loss of consciouness in 4.7% cases. Table 2;3;4. Findings of neurological examination are presented in tabe 2;3;4. Preoperative Signs and symptoms of patients with CPA Epidermoids

Radiological examination included computed tomography(CT SCAN) and Magnetic resonance imaging(MRI).

**Table 1**: comparing age distribution, sex distribution, strength of study, duration of symptoms current study with sillar studies

| Reference | No. of Patients | M/F | Mean Age (y) | Duration of Symptoms (mo) |
|---|---|---|---|---|
| Gagliardi et al,[4] 1980 | 8 | 4/4 | 41.7 | 69 |
| Fisher et al,[23] 1984 | 6 | 3/3 | 38.6 | 112.6 |
| Berger et al, [2] 1985 | 13 | 7/6 | 47 | 4.3 |
| Sabin et al,[25] 1987 | 20 | 12/8 | 42 | 54 |
| Salazar et al,[15] 1987 | 17 | 10/7 | 15–60 | 45.6 |
| Morard et al,[24] 1988 | 6 | 4/2 | 42 | 66.8 |
| Rubin et al,[31] 1989 | 7 | 4/3 | 40.7 | 202.2 |
| de Souza et al,[22] 1989 | 30 | 19/11 | 27 | NA |
| Yamakawa et al,[5] 1989 | 15 | 9/6 | 33 | 94 |
| Altschuler et al,[14] 1990 | 11 | NA/NA | NA | 44.4 |
| Lunardi et al,[13] 1990 | 17 | 8/9 | 40 | 84 |
| Vinchon et al,[11] 1995 | 9 | 6/3 | 40.7 | 47 |
| Samii et al,[10] 1996 | 40 | NA/NA | 42 | 36 |
| Mohanty et al,[29] 1997 | 25 | 15/10 | NA | 35 |
| Mallucci et al,[19] 1999 | 12 | 2/10 | 42 | 24 |
| Kobata et al,[18] 2002 | 30 | 7/23 | 37.8 | 137 |
| Lakhdar et al,[20] 2002 | 10 | 6/4 | 31.7 | 31.2 |
| Schroeder et al,[26] 2004 | 8 | 5/3 | 36 | NA |
| Our series | 21 | 9/12 | 39.33 | 37.2 |

**Table 2**: Showing frequency of symptoms

| Clinical Features | Number of patients |
|---|---|
| Trigeminal neuralgia | 6 |
| Hemifacial spasm | 2 |
| Vth sensory impairment | 6 |
| Vth motor impairment | 1 |
| Facial paresis | 7 |
| Hearing impairment | 8 |
| Lower cranial nerve dysfunction | Nil |
| Cerebellar signs | 5 |
| Pyramidal signs | 6 |
| Trigeminal neuralgia | 13 |
| Painful tic convulsion | 1 |
| Vth sensory impairment | 17 |
| Vth motor impairment | 13 |
| Tinnitus | 2 |
| Sixth cranial nerve palsy | 2 |
| Right quadrantanopia | 1 |
| Giddiness | 4 |

**Table 3**: Showing Preoperative Signs and Symptoms of Patients with CPA Epidermoids

| Reference | Headache | Cerebellar Signs | Pyramidal Signs | Seizures | Hydrocephalus |
|---|---|---|---|---|---|
| NA, not available. | | | | | |
| Gagliardi et al,[4] 1980 | 2 | 4 | 2 | 1 | NA |
| Sabin et al,[25] 1987 | 6 | 17 | 10 | 3 | NA |
| Salazar et al,[15] 1987 | 9 | 10 | 8 | 2 | 5 |
| Morard et al, [24] 1988 | 1 | 2 | 0 | 0 | 0 |
| Rubin et al,[31] 1989 | 3 | 4 | 0 | 0 | 0 |
| de Souza et al,[22] 1989 | 16 | 12 | 4 | 3 | NA |
| Yamakawa et al,[5] 1989 | 9 | 10 | 4 | 0 | NA |
| Vinchon et al,[11] 1995 | 2 | 1 | 0 | 0 | 1 |
| Samii et al,[10] 1996 | NA | 7 | 0 | 1 | NA |
| Mohanty et al,[29] 1997 | NA | 16 | 5 | 2 | 13 |
| Talacchi et al,[12] 1998 | 7 | 10 | 4 | 2 | 4 |
| Mallucci et al,[19] 1999 | 0 | 4 | 0 | 0 | 1 |
| Kobata et al,[18] 2002 | 1 | 1 | 0 | 0 | NA |
| Lakhdar et al,[20] 2002 | 8 | 7 | 1 | 0 | 4 |
| Schroeder et al,[26] 2004 | 3 | 2 | 1 | 2 | NA |
| Safavi-Abbasi et al, 2005 | 3 | 3 | 3 | 0 | 1 |
| Our series,2012 | 10 | 5 | 1 | 5 | 4 |

**Table 4**: Showing frequency of neurological findings.

| Reference | VIII | | | VII | V | IX, X | III, IV, VI | II | II |
|---|---|---|---|---|---|---|---|---|---|
| | Hearing Impairment | Dizziness | Tinnitus | | | | Diplopia | | |
| NA, not available. | | | | | | | | | |
| Gagliardi et al, [4] 1980 | 4 | NA | NA | 5 | 4 | 1 | 2 | 2 | 0 |
| Fisher et al, [23] 1984 | 3 | 3 | NA | 3 | 6 | 1 | 2 | 0 | 0 |
| Sabin et al,[25] 1987 | 8 | 9 | 5 | 12 | 13 | 10 | 6 | 0 | NA |
| Salazar et al,[15] 1987 | 8 | 6 | 2 | 0 | 8 | 7 | 6 | 0 | NA |
| Morard et al,[24] 1988 | 3 | 0 | 1 | 3 | 4 | 0 | 0 | 0 | 0 |
| Rubin et al,[31] 1989 | 5 | NA | 1 | 4 | 6 | 1 | 2 | 1 | 1 |
| de Souza et al,[22] 1989 | 15 | NA | 7 | 16 | 11 | 5 | 6 | 8 | 0 |
| Yasargil et al,[30] 1989 | 12 | 6 | 0 | 9 | 13 | 5 | 7 | 1 | 1 |
| Yamakawa et al, [5] 1989 | 10 | 2 | 9 | 12 | 11 | 7 | 4 | 0 | 4 |
| Vinchon et al, [11] 1995 | 5 | 2 | 3 | 1 | 1 | 0 | 1 | 0 | 0 |
| Vinchon et al,[11] 1995 | 22 | 16 | 5 | 7 | 17 | 4 | 4 | 2 | 1 |
| Mohanty et al,[29] 1997 | 12 | 0 | 0 | 13 | 17 | 4 | 0 | 6 | 0 |
| Talacchi et al,[12] 1998 | 7 | 3 | 2 | 9 | 5 | 4 | 7 | 1 | 1 |
| Mallucci et al, [19] 1999 | 6 | NA | 0 | 4 | 5 | 1 | 1 | 0 | 0 |
| Kobata et al,[18] 2002 | 2 | 0 | 2 | 2 | 29 | 1 | 1 | 0 | 0 |
| Lakhdar et al,[20] 2002 | 0 | 6 | 1 | 4 | 2 | 2 | 0 | 5 | 0 |
| Schroeder et al,[26] 2004 | 0 | 0 | 1 | 1 | 3 | 0 | 1 | 0 | 0 |
| Safavi- Abbasi, 2005 | 4 | 1 | 2 | 4 | 6 | 1 | 4 | 0 | 3 |
| Our series | 8 | 4 | 2 | 7 | 6 | 0 | 2 | 0 | 0 |

**Table 5**: Cerebellopontine Angle Epidermoids: Radiology

| **Procedure** | | |
|---|---|---|
| Computed tomography | Nature | |
| | Hypodense Non-Enhancing | 21 |
| | Location | |
| | Confined To Cp Angle | 11 |
| | With Cpa,Extension To Prepontine and Quadrigeminal Cistern | 7 |
| | Along With Cpa, Supratentorial Extension | 3 |
| | Hydrocephalus | 4 |
| Magnetic resonance | Hypointense On T1 Weighted And Hyperintense On T2 Weighted Images | 2 |
| | Diffusion Restriction ( Done In 8 Patients) | 8 |
| | Flair (Done In 8 Patients) | 8 |
| | Gadolinium Enhancement | 0 |

**Table 6** : Complications of Cerebellopontine Angle Epidermoid Surgery

| **Reference** | **CSF Leak** | **AsepticMeningitis** | **Septic Meningitis Abscess** | **Hematoma** | **VenousThrombosis** | **Long Tract and Cerebellar Deficit** | **Epilepsy** | **Hydrocephalus** | **CN Worsening** | **No. of Deaths** |
|---|---|---|---|---|---|---|---|---|---|---|
| Fisher et al,[23] 1984 | – | – | – | – | 1 | – | – | – | NA | – |
| Berger et al,[2] 1985 | – | 2 | 2 | – | – | NA | – | 2 | NA | – |
| Sabin et al, [25] 1987 | 1 | 3 | 1 | – | 2 | 2 | 1 | 3 (2 shunts and 1 EVD) | 3 | 1 |
| Salazar et al, [15] 1987 | NA | NA | NA | NA | NA | 2 | 1 | 5 (VP shunt) | NA | 11 |
| Morard et al,[24] 1988 | – | 1 | – | – | – | – | – | – | NA | 1 |
| Rubin et al, [31] 1989 | – | 1 | – | – | – | – | – | – | 5 | 5 |
| de Souza et al, [22] 1989 | 3 | – | 3 | – | – | – | – | 12 (shunted prior to surgery ) | 5 | 2 |
| Lunardi et al, [13] 1990 | – | 3 | – | – | – | – | – | 3 | NA | 2 |
| Vinchon et al,[11] 1995 | – | – | 2 | 1 | 1 | – | 1 | – | 6 | 2 |
| Samii et al,[10] 1996 | 2 | 1 | – | – | – | – | – | 1 | 19 | 1 |
| Mohanty et al,[29] 1997 | – | 2 | – | – | – | 2 | – | 1 | 11 | 2 |
| Talacchi et al,[12] 1998 | – | – | – | – | – | 1 | – | – | 11 | – |
| Mallucci et al,[19] 1999 | 1 | – | – | – | – | – | – | – | 2 | - |
| Kobata et al,[18] 2002 | 1 | 2 | 1 | – | – | 1 | – | – | NA | – |
| Lakhdar et al,[20]2002 | 3 | 1 | 1 | – | – | – | – | 2 (VP shunt) | NA | - |
| Schroeder et al, [26] 2004 | – | – | – | – | – | – | – | – | 5 | – |
| Safavi-Abbasi 2005 | 1 | – | – | – | – | – | – | 1 (EVD) | 6 | - |
| Our series 2012 | 1 | 1 | - | - | - | 2 | - | 4(improved after tumour remova l) | 2 | 0 |

*Includes new onset of cranial nerve deficit, remaining or worsening of an existing deficit, and unchanged deficits.

**Table 7**: CP Angle Epdermoids: outcome and follow-up

| Outcome at discharge [n=21] | No. of patients |
|---|---|
| Better than pre-operative state | 18 |
| Same as preoperative state | 2 |
| Persistent cranial nerve deficits | 2 |
| Follow-up [n=21,mean duration=60 mnths, range= 1-11yrs] | |
| Persistent cranial nerve paresis | 2 |
| Able to resume to previous occupation | 16 |

Out Come And Follow Up

CT of brain with intravenous contrast was performed for all patients.

The lesion was hypodense and non-enhancing lesion confined to the cerebellopontine angle only in 52% (11 patients) while 33% (7) along with cerebellopontine angle extent to prepontine and parasellar region. There was supratentorial extension in rest 14.28% (3 patients). Hydrocephalus was present in19% (4 patients); 9.5% (2 patients) had obstructive type, other 2 had non-communicating type hydrocephalus (Table 5).

MRI was done in all patients, but newer sequences like FLAIR, (Table 5) Diffusion were done in 38% (8 patients).

Diffusion restriction presents in 8 patients which help to differentiating it from arachnoid cyst. Proton density sequences done in 3 patients help to differentiate from Cerebrospinal fluid .Newest sequence FLAIR done in 8 patients help to differentiate from cerebrospinal fluid , as epidermoids are heterogenous. No contrast enhancement seen in any patients.

*Tumor Location*

52% (11 patients) (Table 5) was located in CPA in another 33%(7 cases) along with CPA extension to prepontine & parasellar region and 14.28%(3 cases) also extends to supratentorial compartment,as we know that epidermoids have tendency to spread in subarachnoid spaces then migrate to laterally.

*Operative Approach*

All tumors in this series were surgically excised using established microneurosurgical techniques.

Using retromastoid approach radical(Total) removal was possible in 76% (16 patients), near total excision was done in 19% (4 patients) of which 3 was approached through standard retromastoid while in 1 case we used sub-temporal approach, as the temporal component was compressing the mid brain. The capsule was densely adhere to the midbrain, AICA, PICA,[5,7,8,9,10,11] cranial nerves so it was carefully coagulated and left.

In 1 case subtotal(20% of tumor left with capsule) excision was done, previously operated elsewhere present to us as symptomatic recurrence had severe adhesions of arachnoid and capsule to brain stem, anterior inferior cerebellar artery(AICA), posterior inferior cerebellar artery(PICA),[5,6,7,8,9] cranial nerves.

All the operations were performed in single stage except one which was present to us as a recurrent/ residual previously operated elsewhere.

The most common approach used was retromastoid craniectomy 95% and in one patient combined approach (subtemporal+retromastoid) was used as tumor extent to supratentorial through tentorial hiatus.

In one patient temporal component was more than CPA, so sub-temporal approach was used.

*Operative Technique*: After dural opening & assessment of thepertinent anatomy, the tumor capsule is immediately entered, after optimum removal of characteristically soft cyst contents gain 'working space' & decrease manipulation of neighbouring neurovascular structure. After piecemeal reduction of the mass, the capsule is teased free of adjacent neurovascular structures. In majority of cases, a plane of dissection can be developed between capsule and the overlying arachnoid. Occasionaly a granulamatous reaction is present, which causes dense adherence of capsule to the neurovascular structure. If capsule is severely adherent to brainstem or neurovascular structure then capsule should be left behind.

Single stage operation were possible even in extensive tumor growth. As these tumors grow, spread, invaginate and expand the subarachnoid spaces. Subsequently, as removal of tumor progresses, an artificial "surgical channel" was created.

*Complication*

Table 6 early post operative complication occurs within 24 hours. 5th Cranialnerve involvement in 4 patients, total excision was done in 3 patients and subtotal excision was don in 1 patient.6 cranial nerve paresis in 2 patients oftotalexcision.

New onset 7 C.N. paresis in 6 patients, 3 in total, 2 in near-total and 1 in sub-total excision.

CSF leak present in 2 patients from operative site which was improved by repeated lumber puncture. pulmonary embolism in 1 patients improved subsequently.

Mean follow up is 60 months, range is between 1-11 years.

18 patients (Table 7) were in better than pre operative status while 2 patients had persistent 7th cranial nerve paresis(House-Brackman grade-2).

Sensorineural hearing loss was same as that of pre-operative status .

No symptomatic recurrence in any patients while MRI suggestive of recurrence in 2 patients who underwent near total excision that is 50% of near total excision.

There is no recurrence in patients who underwent total excision.

**Discussion**

Epidermoids are benign intradural extradural tumor commonly located in cerebellopontine angle, sellar, parasellar, inter hemispheric and interventricular region.[6,7,8] Rarely may be intracerebral or in pineal region.[9,10,11]. They can extend both in supratentorial and infratentorial compartment along subarachnoid spaces.[12] Recently, Dias and Walker et al. explained etiology for epidermoid is gastrulation dysembryogenesis with secondary disruption of neural tube closure during 3rd-5th week of gastrulation.[15,16] The lateral preference of most of epidermoids is possibly due to proliferation of multipotential embryonic cell rests or the transplantation of epithelial cell rests carried laterally with the migrating otic vesicle or developing neurovasculature.[17] Rarely intracranial epidermoids may be induced iatrogenically induced by repeated percutaneuosly subdural taps.[18,38]

They grow by accumulation of keratin and cholesterol which are breakdown products created by the desquamation of epithelial cells. As the cyst slowly increases its volume,it simply grows into the available subarachnoid spaces i.e. cisterns, fissures, sulcus and ventricles as the soft and pliable nature of cyst.[13]

Epidermoids have an extremely slow linear growth rate.[13] hence duration of symptom is often prolonged and the patients present late in the course of the illness.[14] Mean duration in our study is 3.1 years while in akar et al had 4.6 years.[4,14]

Symptoms and signs attributed toirritative effects created by physical presence of space occupying lesion[19,20] and displacement of neurovascular structure.

In our series headache was most common(Table 3) presentation, followed by hearing impairment (all had mild-moderate sensorineural hearing loss). In our series 38% have impaired hearing which almost similar to samii et al. 2005. The incidence of diminished hearing increases when formal testings are performed.[22] The development of trigeminal neuralgia and hemifacial spasm can be most likely explained by local irritation[21], seizure because of proximity to parasellar and temporal lobe. In our series, facial paresis was 33.33%(Table 2)(Table 4) i.e. 2nd most common cranial nerve involved after 8 cranial nerve. Hydrocephalus present in 4 of our patients, 2 non-communicating and 2 obstructive hydrocephalus. communicating hydrocephalus developed after one or more particularly intense periods of meningitis or post operative if cyst contents egress from operative field.[23] Non communicating hydrocephalus is also a common occurance in natural history of epidermoids because of their characteristic to lie along the cerebrospinal fluid outflow pathways.[23] Currently the MRI is investigation of choice particularly the diffusion weighted and FLAIR (Fluid Attenuated Inversion Recovery), CISS (steady state) and FIESTA quite often helpful to differentiate epidermoids and cerebrospinal fluid within arachnoid cyst.[24] Diffusion help in delineating borders of epidermoid. In our series all patients present with typical ct scan findings, shows homogenous non-enhancing hypodense lesion in subarachnoid space without surrounding oedema, Hounsfield units show epidermoids have more fat density (-30 to -40) then cerebrospinal fluid density (5 HU).[2]

*Surgical Consideration*: Retrosigmoid approach allows goodvisualization and resection of lesions involving the cerebellopontine angle without requiring excessive cerebellar retraction.[25,26,27] It has most often been used to resect epidermoids in posterior fossa.[3,22,23,25,28,29] Even a large tumors extending beyond the cerebellopontine angle can be managed with this approach.[25,29] Usually,

the selection of surgical approach depends on surgical upon the surgeons experience and preference. we mostly used supine with the head turn position. As advised by Berger and Wilson, a subtemporal approach can be used for lesion extends supratentorial into middle cranial fossa.[31] We used combined approach in one patient.

When only partial resection is possible, careful severance and coagulation of the tumor capsule may reduce the risk of regrowth and symptom recurrence.[23,29] Epidermoid extension and radical removal. Rate of radical removal have ranged from 0-97%.[22,23,25,26,31,32,33,34,35] Our resection rate is about 76%. The extent of resection influenced by the tumor capsule adhesion to surrounding neurovascular structure but also by its pattern of extension.

*Management of Recurrence:* As we had come across with one recurrent tumor who was symptomatic extend beyond its original site[3,34], operated 3 years earlier. Scars and adherences related to a previous surgery can constitute a further obstacle to achieve complete removal. To diagnose recurrence FAST-FLAIR imaging sequence of MRI was superior.[36]

To avoid recurrences and evaluate the extent of resection of lesion of cerebellopontine angle must be inspected as a final operative step. we evaluate the extent of resection on immediate and follow up post operative MRI[3,7]

**Conclusion:**

Despite improvement in post-operative outcome, cerebellopontine angle epidermoids remain challenging entity. The cranial nerve fifth and seventh may recover after decompression whether the symptom related to eighth cranial nerve dysfunction will improve remains uncertain.[30] When complete excision of the cyst cannot be obtained, its remnants should be severed cautiously and coagulated thoroughly to reduce the risk of recurrence as much as possible.[30] Total removal of cerebellopontine epidermoids does not results in significantly increased long term morbidity and mortality, should be the goal of surgical treatment.

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