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Intracranial epidermoid cyst in a single Mexican institution, experience of over 16-years

Sergio Moreno-Jimenez,[1] Martha Lilia Tena-Suck,[2] Miguel Angel Collado-Ortiz,[3]
Manuel Castillejos-López,[4] Diego Alvarado-Moreno,[2] Mirelle Kramis Holland[1]

ABSTRACT

This study details our series of intracranial epidermoid cysts. A historical cohort of 110 epidermoid cysts that were excised from intracranial location over a 16 year period was reviewed. The incidence of epidermoid cysts, clinico- pathological examinations and cyst recurrence rate were noted.

110 cases were included in this study. 60 were males and 52 were females. The range aged was 15-74 years, mean 36,36 ± 13,45 for males and 34, 20 ± 12,17 for female. Their locations included; supratentorial region (n=20), cerebello pontine angle (n=46), Middle ear (n=34), intraventricular and intraparenchymal (n=3), and one in cistern magnum, pineal region, retoclival, medular and mesencephalic region. Surgical exeresis was performed in all cases, partial in 25(26%) cases and total in 71 cases. 74% of the patients had a good prognosis and just 10(10.4%) cases had bad one. There was correlation between tumor size papillomatosis (p = 0.002), inflammation, and granuloma formation p = 0.000 respectively), between older patients and dyskeratosis (p = 0.008) and with the presence of dystrophic calcifications and longer follow-up (p = 0.013). Aseptic meningitis occurred postoperatively in 4 cases, recurrence of tumor was in 22 (20%) cases, two patients died and the rest of the cases had a satisfactory outcome.

Conclusions: Recurrent leakage of the irritating cyst contents and subsequent chemical inflammatory response may be responsible for recurrence, high-density on CT scans and the cystic nature.

Ked words: Brain tumors, Epidermoid Cyst, Epidemiology, Clinicopathological correlation.

Epidermoid cyst (EC) is considerate as a congenital tumor which develops from ectodermal remains arising between 3th and 5th weeks of gestation, of embryonic development if squamous epithelial remnants are included in the neural tube when the neural tube separates from the ectoderm.[1] Intracranial EC are benign tumors account for only 0.2 to 1.8% of all intracranial tumors they grow slowly and they are presented between ages from 20 to 40 years.[2] EC are rare disontogenetic tumoral lesions in the cranial compartment and are usually located in relation to the cranial sutures; the most common location for epidermoid cysts is the cerebellopontine angle cistern (40%–50%), where they are the third most common mass overall cerebellopontine angle and cistern–internal auditory canal.[3] Epidermoid cysts also occur in the fourth ventricle (17%) and the sellar and/or parasellar regions (10%–15%). Less common locations include the cerebral hemispheres or brainstem. Ten percent of epidermoid cysts are extradural, located in the skull or spine. All are located off the midline.[4,5]

Most are asymptomatic but may occasionally present mass effect, cranial neuropathy or seizures, the prognosis is favorable, but the preoperative diagnosis is often difficult.[1,5,6]

Histologically, subcutaneous epidermal cysts and intracranial epidermoid cysts are pathologically identical.[7] The cyst interior is filled with "unpleasant" soft, waxy, or flaky keratohyalin material that results from the progressive desquamation of the cyst wall.

1 Unit of Radioneurosurgery. National Institute of Neurology and Neurosurgery, Mexico City.
2 Department of Neuropathology. National Institute of Neurology and Neurosurgery, Mexico City.
3 Service of Neurology. National Institute of Neurology and Neurosurgery, México City.
4 Service of Epidemiology. National Institute of respiratory disease, Mexico City.

Correspondence: Martha Tena-Suck. Departamento de Neuropatología Instituto Nacional de Neurología y Neurocirugía ìManuel Velasco Suárezî, Av Insurgentes Sur no. 3788 Col. La fama, Delegación Tlalpan, CP 14269. E-mail: tenasuck@yahoo.com.mx
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The authors review brain epidermoid cysts, epidemiologic and pathologic findings, and radiological appearance in a single Mexican institution.

MATERIALS AND METHODS

Case selections

EC were retrieved from the files of the Pathology Laboratory of Neuropathology of the National Institute of Neurology and Neurosurgery in Mexico City. Clinical information and follow-up data were available in 110 cases. Age, gender, location and size of tumor, familiar history, traumatic and infection history, surgical exeresis, postoperative complications, recurrence and death were also recorded.

Histology

All tumors analyzed in the study were obtained from the first surgery. Surgically removed specimens were fixed immediately in 10% formalin and subsequently embedded in paraffin. Standard hematoxilin and eosin sections and PAS stains were used for diagnosis. Histopathological finding showed multiple layers of squamous epithelium lining and laminar keratinization consistent with a benign epidermoid cyst, sometimes, the cyst contained high cellular area with enlarged pleomorphsm and hyperchromatic cells. The following histological features were recorded: presence or absence of inflammatory cells, gliosis, clef cholesterol, laminar keratin and brain invasion, as well as, papilomatosis and epithelial atrophy for each case.

Statistical analysis

Statistical analyses were performed using the SPSS software (version 16; Chicago, IL, USA). Clinical characteristic, radiological imaging and histological features were correlated between them. Descriptive statistics (mean, standard deviation, and frequency distribution) was determined for continuous and categorical variables. Age, tumor size, time of symptoms presentations and follow-up were mainly expressed as mean ± SD (standard deviation). Percentages were compared with the χ^2 test, Fisher's exact test and Phi, Spearman, Person correlation and one ANOVA were also obtained. Tumor localization and gender were correlated between them. A P-value of ≤ 0.05 was considered to be statistically significant.

RESULTS

One hundred and ten cases cases were included in this study. Clinical data and tumor localization are seen in Table I. 60 (55%) were female (f) and 50 (46%) were male (m)($p = 0.324$). In relation to gender and age, women had a mean age of 34.20 ± 12.178 years and the average male was 36.3 ± 13.4576 (women were younger than men. 31.70 ± 6.920 years *($p = 0.077$).*

The time of presentation of symptoms was 4.43 ± 14.431 mo to females and 13 821 ± 3.6 mo for males ($p = 0.376$) (women had a shorter onset of symptoms than men). The mean follow up time was 25.72 ± 9.7 for women and 25.24 ± 8.7mo for men ($p = 0.383$) (women longer follow-up than men).

In relation to the size of the tumor and with the gender, women had a mean of 30.53 ± 8.12 mm and men of 31.70 ± 6.9 mm ($p = 415$), men were slightly greater size than women and was not statistically significant.

The surgical findings and outcome in relationship with tumors location are seen in Table II. There was no relationship between type of surgery and gender. Men had more complications (n=14) than women (n=10) was not significant. Second surgery was carried out equally to men and women. 4 cases showed aseptic meningitis, 3 of them were women and 1 man, as the performance follow-up they had a good prognostic (50 women and 39 men), surgical complications occurred in 9 patients and worsening in 12 (same for both genders).

The histologic findings in relation to clinical data are observed in Table III. Histological examination showed stratified squamous epithelium (Figures 1a and b), hyperplasia and inflammation were observed in 27 cases (17 women and 10 men ($p = 0.096$) atrophy in 37 cases (16 m and 21 f, $p = 0.068$)(Figure 1c), papilomatosis (Figure 1d), and dyskeratosis in 20 (12m and 8 men ($p = 0.052$), lamellar keratinizing squamous epithelium in 13 cases, 6 women and 7 men ($p = 0.593$). There was a statistical difference when compared with epithelial hyperplasia tumor location, in APC ($p = 0.023$), supratentorial ($p = 0$ .012) and ear canal ($p = 0.006$). There were statistically difference significative in relation to gender and hyperplasia ($p = 0.023$). Location APC with dyskeratosis ($p = 037$), supratentorial granulomas ($p = 0.000$), ear canal with inflammation ($p = 0.006$) and with rupture of the cyst ($p = 0.026$). Histological changes in relation to the clinical

Table I. Tumor localization and it relation with patient's clinical data

	Age(yr)	*Tumor size (mm)*	*Time of symptoms Presentation (mo)*	*Follow-up (mo)*
Gender				
Female	34,20 ± 12,17	30.53 ± 8.1	14.43 ± 4.43	25.72 ± 9.7
Males	36,36 ± 13,45	36.36 ± 13.45	13.82 ± 3.6	25.24 ± 4.7
Pontocerebellous	17-69	21-54	8-21	12-45
Angle (n=46)	(36.09 ± 12.7)	(32.15 ± 7.55	(12.48 ± 2.15)	(24.46 ± 7.8)
			p=0.000	
Supratentorial (n=20)	15-48	22-43	9-22	12-34
	(33.10 ± 9.49	(33.10 ± 6.47)	(13.10 ± 2.15)	(23.95 ± 7.1)
Meddle ear (n=34)	15-74	16-44	7-23	12-54
	(36.21 ± 14.13)	(26.74 ± 6.5)	(17.82 ± 4.7)	(29.26 ± 11.08)
		p=0.000	**p=0.000**	**p=004**
Intraparenchymal(n=3)	18-29	32-43	12-15	12-24
	(23.67 ± 5.5)	(39.33 ± 6.3)	(13.0 ± 7.9)	(16.33 ± 6.65)
Intraventricular (n=3)	19-27	33-45	10-13	14-32
	(22.0 ± 4.35)	(40.36 ± 4.42)	(11.67 ± 1.5)	(22.33 ± 9.0)
		p=0.000		
Pineal (n=1)	48y	35	6	14
Clivus (n=1)	24y	34	12	12
Spinal (n=1)	54y	23	12	32
Mesencephalic (n=1)	54y	36	9	32
Recurrence (n=22)	20-60	21-44	2-22	12-23
	(37.59 ± 11.46)	(30.16 ± 7.9)	(14.23 ± 4.1)	(24.73 ± 8.8)
Dead (n=2)	27-41	23-32	12-12	12-21
	(34-0 ± 9.8)	(27.50 ± 3.3)		(16.50 ± 6.3)

Age, tumor side, time of presentation of the symptoms and follow-up and were obtained by minimum-maximal data and mean and standard derivation). Descriptive statistics (frequency distribution) were determined for continuous and categorical variables. *p* value vas obtained by Phi, Person and Spearman correlations.

findings assumes that hyperplasia was present in larger tumors (ANOVA $p = 0.002$) (Figures 2a and 2b).

Epithelial atrophy was associated with lower tumor size and a shorter time of evolution of symptoms. As well as, papillomatoses was correlated with older patients, larger size and shorter development (ANOVA, $p = 0.001$ respectively). Dyskeratosis was associated with older patients ($p = 0.008$), and smaller tumors ($p = 0.004$). Inflammation was associated with patients with older age, less time monitoring and symptoms as well as with longer follow-up. However, there was not statistically significant correlation between tumor size and histologically features ($p = 0.000$). Aseptic meningitis was present in 4 patients (2h and 2m), two of them died and 2 relapsed. There was correlation between time of presentation of symptoms with the location of the tumor in APC and ear canal ($p = 0.000$, respectively). The intraventricular location of tumors showed higher tumor size and with a longer follow-up ($p = 0.000$, respectively). Figure 3 showed the correlation between recurrence and histological changes.

DISCUSSION

Epidermoid tumors are rare intracranial tumors. They are commonly found in the cerebellopontine angle and the suprasellar region.[1] However, they are also known to occur in other locations often spread into several intracranial compartments.[1,5,6] EC appears like as a thin-walled sac containing mucoid material, which can be aspirated easily, contains keratin, cellular debris and cholesterol, and the lining of the cyst is composed of simple stratified squamous epithelium supported by an outer layer of collagenous of variable thickness.[7] Progressive exfoliation of keratinaceous material (laminar keratinizing squamous epithelium), towards the interior of the cyst produces the lamellar character of the contents.[7] Cholesterol crystals

Table II. Showed the relation between surgery, exeresis and tumor localization

Tumor Localization	*Surgery*	*Exeresis*	*Complications*	*Prognosis*
Pontocerebellous Angle (n=46)	Lobectomy 1 Craniotomy 29 Descompresion 13 Exeresis 3 (p=000)	total 28 partial 18	Infection 7 Hemorrhage 2 Fistula 3 Neumoencephalum 2	Good 41 Complicacions 3 Deterioration 2
Supratentorial (n=20)	Lobectomy 2 Craniotomy 14 Decompression 3 Exeresis 1	total 17 partial 3	Infection 5 2º surgery 6	Good 14 Complications 4 Deterioration 2
Meddle ear (n=34)	Craniotomy 11 Decompression 4 Exeresis 2 Mastedotomy 17 (p=000)	total 31 partial 3	Infections 4 2º surgery 9 Meningitis 2	Good 26 Complications 2 Deterioration 7
Intraparenchymal (n=3)	Lobectomy 2 Craniotomy 1	total 28 partial 18	No	Good 41 Complications 3 deterioration 2
Intraventricular (n=3)	Craniotomy 3	total 2 partial 1	Infections 1	Good 3
Pineal (n=1)	exeresis	total	No	Good
Clivus (n=1)	craniotomy	total	No	Good
Spinal (n=1)	exeresis	total	2º surgery	Good 2, bad 1
Mesencephalic (n=1)	Decompression	total	No	Good
Aseptip meningitis	craniotomy	partial	2 surgery	deterioration 3

have been claimed to be rare and when present are thought to be the results of cholesterol accumulation derived from the cell membranes of an antecedent hemorrhage of infiltration of inflammatory cells. In the middle ear EC is called cholesteatoma and is characterized by the presence of keratinizing squamous epithelium in this cavity with highly invasive properties causing bone destruction and it may lead to complications. Epithelial atrophy (78%), epithelial acanthosis (88%), hyperplasia of the basal layer (88%) and formation of epithelial cones (62%) can be seen. Histopathological variables presented no statistical significant difference in both age groups.[8]

In our cases we found discrete differences between gender and histologic findings, women showed more hyperplasia and granulomas that men but men showed more calcification. In relation to the location of the tumor hyperplasia was observed higher in the MPA and the ear canal than in other areas, atrophy was observed in intraventricular location.

Also we can observe in the benign epidermoid cyst calcifications[9] and malignant transformation**,** these may be due to a chronic inflammatory stimulation caused by repeated cystic rupture.[10] In our cases rupture was present in 7 cases, most often in women than in men and was associated with inflammation. Although there were more calcifications in men tumors than in women and those tumors that were broken and APC were present in the ear canal.

Generally, the diagnostics of the EC is made by CT-scan and MRI. On CT scans, most epidermoid cysts are well-defined hypoatenuated masses that resemble CSF and do not enhance. A calcification is present in 10%–25% of cases. Most epidermoid cysts are isointense or slightly hyperintense to CSF on both T1- and T2-weighted MR images. They do not suppress completely on FLAIR images and restrict (show high signal intensity) on diffusion-weighted images.[5,6]

The major differential consideration for the epidermoid cyst is an arachnoids cyst. Arachnoids cysts are isointense to CSF at all sequences, including FLAIR. Other epidermoid cyst mimics include dermoid cyst, neurocisticercosis, and cystic neoplasm.[11,12] Dermoid cysts are typically located along the midline and resemble fat. Neurocisticercosis cysts often enhance and demonstrate surrounding edema or gliosis.[3,5,6]

Table III. Histological findings and correlation ship between clinical data

	Age(ys)	*Tumor size (mm)*	*Time of symptoms Presentation (mo)*	*Follow-up (mo)*
Hyperplasia	16-65 (35.29 ± 11.21)	21-54 (32.42 ± 7.5) **P=0.002**	7-23 (14.35 ± 3.8)	12-45 (27.21 ± 8.7) P=0.065
Atrophy	15-75 (33.77 ± 13.0)	16-45 (30.61 ± 13)	6-23 (13.64 ± 4.2)	12-54 (24.98 ± 9.3)
Papillomatosis	18-74 (35.8 ± 13.6)	16-43 (27.2 ± 7.5) **p=0.001**	8-23 (14.6 ± 14.0)	12-54 (25.83 ± 9.3)
Dyskeratosis	20-74 (41.7 ± 15.4) **p=0.008**	16-43 (27.89 ± 7.3) **p=0.004**	6-23 (13.9 ± 4.0)	12-54 (25.86 ± 10.4)
Laminar keratinization	15-67 (30.7 ± 14.5)	21-45 (30.1 ± 7.8)	10-22 (14.0 ± 4.0)	12-35 (24.71 ± 8.8)
Inflammation	15-74 (37.53 ± 16.0)	16-43 (24.66 ± 6.0) **p=0.000**	9-23 14.72 ± 4.7)	12-46 25.44 ± 8.9)
Granuloma	20-74 (35.9 ± 16.9)	16-32 (22.2 ± 3.7) **p=0.000**	10-22 (14.8 ± 4.6)	12-54 (28.36 ± 13.3)
Cyts rupture	15-57 (31.09±13.8)	16-43 (±	9-22 (13.09 ± 4.3)	12-54 (25.6 ± 13.6)
Cholesterol	25-54 (32.7±10.0)	23-43 (33.0 ± 8.0)	12-21 (14.57 ± 14.2)	13-45 (28.43 ± 10.1)
Dystrophic Calcifications	15-63 (29.42±12.6)	21-44 (30.3 ± 7.7)	7-22 (16.9 ± 5.0) **p=0.013**	12-46 (28.0 ± 10.7)

Age, tumor side, time of presentation of the symptoms and follow-up and were obtained by minimum-maximal data and mean and standard derivation). Descriptive statistics (frequency distribution) were determined for continuous and categorical variables. *p* value vas obtained by Phi, Person and Spearman correlations.

Clinically ECs behave like benign, slow growing lesion; symptomatology can be very diverse and basically depends on the location, tumor size and compression of normal cerebral structures, female predominate.[1,12]

Vestibular symptoms such as vertigo, gait ataxia, and nystagmus masquerading as acute vestibular neuritis or central vertigo and trigeminal neuralgia, and signs of posterior fossa tumor have been described in cerebellopontine angle EC.[12-14] EC located in the fourth ventricle are exceedingly rare, and the patients usually present with headaches, vertigo, and/or disequilibrium, gait disturbances and cerebellar signs.[1] Intrasellar epidermoid cyst presenting as pituitary apoplexy[15,16] and conductive hearing loss, hypoacusia and vertigo are presenting in case of cholestatoma.[17]

Age of presentation of EC varies according to the series studied; mean age is 40 years.[1,10,15,18] The duration of the disease at admission varied between a few days and 30 years.[1] The duration of symptoms until the diagnosis is less in pediatric population (2 days to 2 months) than in adults (2 mounts to 12 years).[17]

Recurrence is reported in EC, between 10 weeks to 18 mounts after surgery.[1] Shiefer, et al[11] performed a retrospective review of 24 patients with epidermoid tumors of the CPA surgically treated and they observed a rate of recurrence of 23% in tumors considered totally removed and 27% in those near/sub totally removed. Of the 6 patients with recurrences, 5 underwent a second operation. The mean duration of follow-up time was 4.2 years. Recurrent leakage of the irritating cyst contents and subsequent chemical inflammatory response may be responsible for the high-density on CT scans and the cystic nature. A correct histological diagnosis is important because, in contrast to typical ECs and other lesions. Early

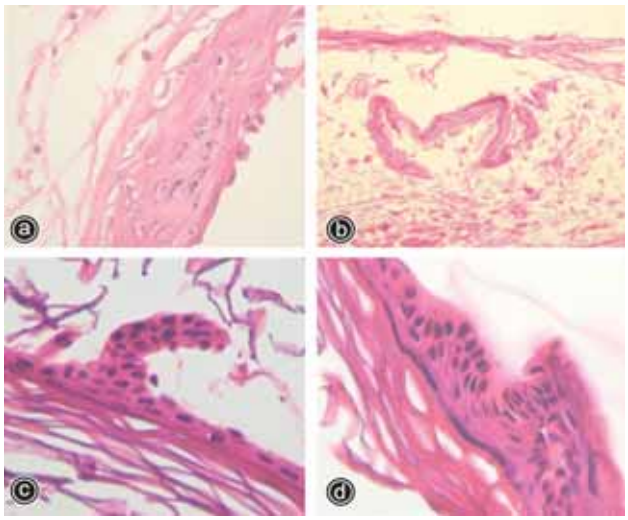

Figure 1. Histopathological features: a) Squamous epithelium, showed a close-up of the squamous epithelial (H&E ×400); b) observed the laminar keratinization of squamous epithelium and atrophy changes of the same (H&E ×200); c) papillary formation in squamous epithelium (H&E ×200) and d) hypertrophy changes (H&E ×400). For color images from this paper see Annex 2.

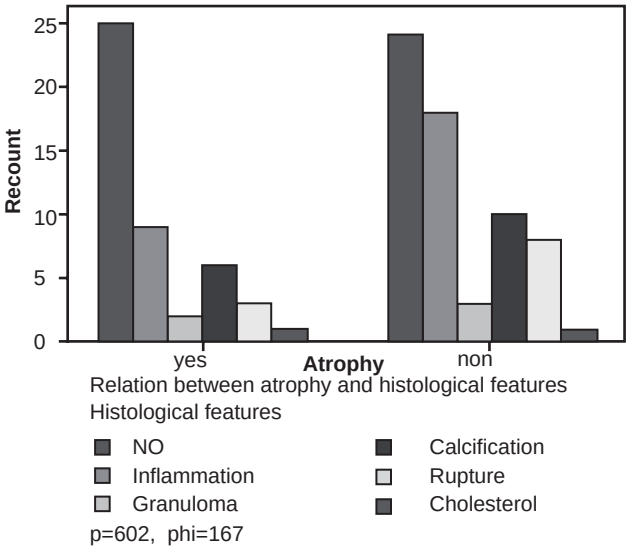

Figure 3. Histogram showed the relation between recurrence and histological features in a) and in b).

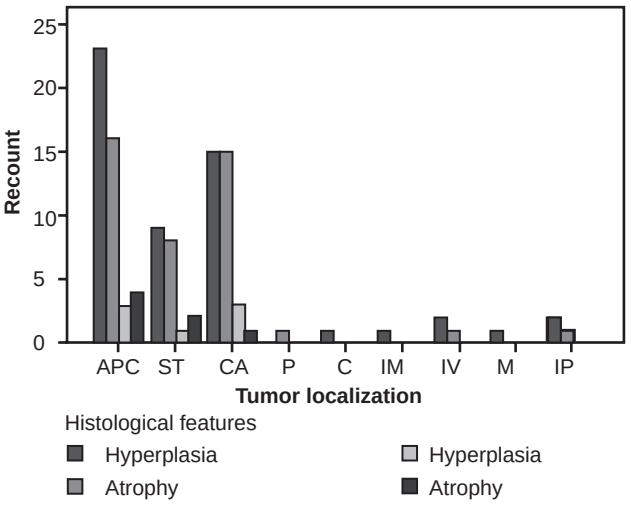

Figure 2. Histogram showed the relation between gender and histological changes in a) and in b).

recurrence of an epidermoid, even in the absence of overt evidence of infection, should produce a high index of suspicion that there may be a low-grade infective cause. Surgical excision coupled with antibiotics resulted in a symptomatic and radiological recovery.[19]

Roy K, et al[1] reported 5/28 patients died, in 3 cases had a worsening of the cerebellar functions and aggravation of cranial nerve deficits were presented in 2 patients.

Cho, et al series[1] reported a followed up over a mean duration of 5 years and 10 months, the author reported a mean time of follow-up of 48.35 ± 47.65 mo for males and for females 48.50 ± 33.2 mo (p.495 and Phi = 0.002).

Radical surgical removal of epidermoid cysts should be attempted, but a less aggressive surgical strategy should be considered if there is strong adherence to the surrounding brain tissue, particularly in eloquent areas. Since this tumor growth slowly some authors do not recommend total removal. However, total exeresis have a favourable outcome with minimal or no mortality.[18]

Surgery is the only effective treatment, and radical resection of the entire cyst, whenever possible, generally succeeds in achieving a cure. Much has been said about the type of surgery and the best surgical approach.[11,12]

Because surgery is the only effective treatment for these lesions, radical resection should be performed in all cases to avoid tumor recurrence; however, because the cyst capsule can adhere firmly to vital structures and its radical removal can be dangerous, subtotal resection may be a wise option in selected cases. They

involve multiple cranial nerves and vascular structures. Complete resection is frequently possible with minimal new cranial nerve deficits.[20] Multiple cranial nerves, seizures, facial weakness, headaches were neurological complication by tumor removal. Total resection of the tumor is the optimum treatment. Postoperative complications included chemical meningitis in 7/28 in Roy K, et al serie.[1] Several series studies have been reported that the most of the patients have had satisfactory outcome.[1,11,12] Chemical meningitis presumably due to leakage of content from EC may be confused with septic meningitis.

Is well known that tumor size is an important factor to completely tumor exeresis, however, there is little literature on this subject. Kaylie DM, et al reported the average tumor size was 3.9 cm.[20]

In addition, the aetiology of epidermoid cysts is not clear. The aetiology is likely related to the time and locus of sequestration of ectodermal tissue during foetal development.[21] Acquired epidermoid cysts may develop as a result of trauma,[22] or vascular anomalies, infectious agents, inflammatory response,[14] or association between this tumoral lesion and a tissue inflammatory reaction due to foreign bodies,[23] with perforation of the dura and brain parenchymal involvement,[24] associated with iatrogenic puncture[25] and secondary to mucocele surgery,[26] etc.

CONCLUSIONS

ECs are benign tumors with good prognosis, they have a low incidence of recurrence, however minimal histologic findings may vary in relation to gender, tumor size and location.

REFERENCES

1. Roy K, Bhattacharyya AK, Tripathy P, Bhattacharyya MK, Das B. Intracranial epidermoid a 10-year study. J Indian Med Assoc 2008;106:450-3.
2. Yaşargil MG, Abernathey CD, Sarioglu AC. Microneurosurgical treatment of intracranial dermoid and epidermoid tumors. Neurosurgery 1989;24:561-7.
3. Dutt SN, Mirza S, Chavda SV, Irving RM. Radiologic differentiation of intracranial epidermoids from arachnoid cysts. Otol Neurotol 2002;23:84–92.
4. Katzman GL. Epidermoid cyst. En: Diagnostic imaging: brain. Salt Lake City, Utah: Amirsys, 2004; I-7-16.
5. McLendon RE, Tien RD. Tumors and tumor-like lesions of maldevelopmental origin. In: Russell and Rubinstein's pathology of tumor OsbornAG. Miscellaneous tumors, cysts, and metastases. En: Diagnostic neuroradiology. St Louis, Mo: Mosby, 1994; 631–649.
6. Osborn AG, Preece MT. Intracranial cysts: radiologic-pathologic correlation and imaging approach. Radiology 2006;239:650-64.
7. Suzuki C, Maeda M, Matsumine A, Matsubara T, Taki W, Maier SE, Takeda K. Apparent diffusion coefficient of subcutaneous epidermal cysts in the head and neck comparison with intracranial epidermoid cysts. Acad Radiol 2007;14:1020-8.
8. Alves AL, Pereira CS, Ribeiro Fde A, Fregnani JH. Analysis of histopathological aspects in acquired middle ear cholesteatoma. Braz J Otorhinolaryngol 2008;74:835-41.
9. Praveen KS, Devi BI. Calcified epidermoid cyst of the anterior interhemispheric fissure. Br J Neurosurg 2009; 23:90-1.
10. Maffazzoni DR, Barbosa-Coutinho LM, Chemalle Ide M, Maciel E. Carcinoma originating in an intracranial epidermal cyst. Case report. Arq Neuropsiquiatr 1986;44:391-4.
11. Schiefer TK, Link MJ. Epidermoids of the cerebellopontine angle: a 20-year experience. Surg Neurol 2008;70:584-90.
12. Li F, Zhu S, Liu Y, Chen G, Chi L, Qu F. Hyperdense intracranial epidermoid cysts: a study of 15 cases. Acta Neurochir (Wien) 2007; 149:31-9.
13. Han IB, Huh R, Chung SS, Kim OJ. Cerebellopontine angle epidermoid tumors presenting with bilateral gaze nystagmus. Br J Neurosurg 2008;22:441-4.
14. Guttal KS, Naikmasur VG, Joshi SK, Bathi RJ. Trigeminal neuralgia secondary to epidermoid cyst at the cerebellopontine angle: case report and brief overview. Odontology 2009;97:54-6.
15. Rao VJ, James RA, Mitra D. Imaging characteristics of common suprasellar lesions with emphasis on MRI findings. Clin Radiol 2008; 63:939-47.
16. Tuna H, Torun F, Torun AN, Erdogan A. Intrasellar epidermoid cyst presenting as pituitary apoplexy. J Clin Neurosci 2008;15:1154-6.
17. Robertson G, Mills R. Findings at exploratory tympanotomy for conductive hearing loss. Laryngol Otol 2009;123:1087-9.
18. Lopes M, Capelle L, Duffau H, Kujas M, Sichez JP, Van Effenterre R, Faillot T, Bitar A, Fohanno D. Surgery of intracranial epidermoid cysts. Report of 44 patients and review of the literature. Neurochirurgie 2002;48:5-13.
19. Rutherford SA, Leach PA, King AT. Early recurrence of an intracranial epidermoid cyst due to low-grade infection: case report. Skull Base 2006;16:109-16.
20. Kaylie DM, Warren FM 3rd, Haynes DS, Jackson CG. Neurotologic management of intracranial epidermoid tumors. Laryngoscope 2005;115:1082-6.
21. Kaido T, Okazaki A, Kurokawa S, Tsukamoto M. Pathogenesis of intraparenchymal epidermoid cyst in the brain: a case report and review of the literature. Surg Neurol 2003;59:211-6.
22. Lee VS, Provenzale JM, Fuchs HE, Osumi A, McLendon RE. Post-traumatic epidermoid cyst: CT appearance. J Comput Assist Tomogr 1995;19:153-5.
23. Prat Acín R, Galeano I. Giant occipital intradiploic epidermoid cyst associated with iatrogenic puncture. Acta Neurochir (Wien) 2008;150:413-4.

24. Cho JH, Jung TY, Kim IY, Jung S, Kang SS, Kim SH. A giant intradiploic epidermoid cyst with perforation of the dura and brain parenchymal involvement. Clin Neurol Neurosurg 2007;109:368-73.
25. Mejdoubi M, Lagarde S, Ponsot A, Gigaud M. Intracranial epidermoid cyst secondary to mucocele surgery. J Radiol 2009;90:233-5.
26. Locatelli M, Alimehmeti R, Rampini P, Prada F. Intradiploic frontal epidermoid cyst in a patient with repeated head injuries: is there a causative relationship. Acta Neurochir (Wien) 2006;148:1107-10.