REVIEW

Myoepithelioma of the Orbital Apex and Middle Cranial Fossa: Case Report and Review of the Literature

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ABSTRACT

Myoepitheliomas are rare tumours that originate from glandular tissues such as the parotid or salivary glands, and less commonly from soft tissues of the head, neck, and other parts of the body. Intraorbital myoepitheliomas generally arise from the lacrimal gland. Intracranial myoepitheliomas are rare. We report a myoepithelioma of the orbital apex that did not originate from the lacrimal gland. It extended to the middle cranial fossa from the orbital apex and involved the dura and adjacent bone. A diagnostic biopsy via a lateral orbitotomy preceded resection. We review the natural course and histopathology of myoepithelial neoplasms, the surgical nuances of approaching an orbital apex tumour with maximal functional preservation, and the optimal management practices of these rare lesions.

Keywords: Middle cranial fossa, myoepithelioma, orbital neoplasm, orbital osteotomy

INTRODUCTION

Myoepitheliomas are rare neoplasms that arise from myoepithelial cells normally found in the epithelium of glandular structures such as sweat, lacrimal, mammary, or salivary glands.^{1–5} They may also arise from non-glandular soft tissues and are hence classified into two groups based on origin; those arising from glandular tissues and those from soft tissue.^{1,3,4} The first description of a myoepithelial neoplasm is attributed to Sheldon, who in 1943 described a myoepithelioma of the parotid gland.⁶

Myoepithelial tumours may occur in the scalp, orbit, sella region, cerebellopontine angle, lacrimal and salivary glands, sinuses, nasopharynx, masticator space, palate, and larynx (Table 1).^{1,4,5,7–13}

Other sites where myoepithelial neoplasms have been reported include the trunk, viscera, and limbs.^{1,4} At a genetic level, Ewing sarcoma breakpoint region (ESRW-1) locus and pleomorphic adenoma gene (PLAG-1) locus mutations may be seen; the ESRW-1 mutation is more commonly seen in myoepithelial tumours of soft tissue origin and the PLAG-1 mutation in myoepithelial tumours of glandular origin.^{14–19} In general, these are slow growing, benign, or low-grade tumours, but malignant lesions, known as myoepithelial carcinomas, are also reported.^{1,3–5,11,13,20,21} Myoepithelial carcinomas are more aggressive tumours but are even rarer than the low-grade myoepithelial neoplasm. They tend to invade surrounding tissues and reach a large size at presentation. Histologically, they are characterised by

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TABLE 1 Summary of all studies on orbital and intra-cranial myoepitheliomas.

Author/Year	N	Age	Origin/Location	Treatment	Outcome
Bolzoni/2005	1	46	Lacrimal gland	GTR	No recurrence at 22 months
Font/1992	1	23	Lacrimal gland	GTR	No recurrence at 12 years
Grossniklaus/1997	1	76	Lacrimal gland	GTR	No recurrence at 10 months
Heathcote/1990	1	"Middle-aged"	Lacrimal gland	GTR	No recurrence at 15 months
Herrera/1990	1	68	Lacrimal gland	Unknown	Unknown
Iiida/2001	1	77	Lacrimal gland	Unknown	Unknown
Okudela/2000	1	34	Lacrimal gland	GTR	No recurrence at 3 years
Herlihy/2009	1	3 months	Orbit not involving lacrimal gland	Observation	Unknown
Waldrop/2001	1	11	Thigh primary, metastatic to orbit	STR, Chemotherapy	Patient died at 4 months
Tran/2009	2	55, 24	Paranasal sinuses with extension to orbit	1. GTR with orbital exenteration 2. GTR	1. Deceased at 13 months 2. Deceased at 1 month
Amin/2002	1	76	Parotid gland, temporal bone	GTR	No recurrence at 20 months
Fritchie/2012	1	20	Parietal-occipital bone	GTR	No recurrence at 6 months
Erdogan/2007	1	47	Convexity dura	STR × 2, XRT	No recurrence at 4 months
Bhonsteadt/2012	1	14	Hip and lung primary, metastasis to clivus	STR, Chemotherapy, proton beam therapy	Died at 14 months
Hornick/2003	5	N/A	Scalp	Not reported	Not reported
Nieder/2005	1	34	Sellar region	STR, XRT, chemotherapy	Died at 20 months
Hampton/1997	5	17, 61, 66, 81, 85	Sellar region (4), parotid with sellar extension (1)	1. TSP resection, XRT 2. TSP resection, XRT 3. STR, XRT 4. GTR 5. TSP resection	1. No recurrence at 2 years 2. Unknown 3. Unknown 4. Doing well 5. Deceased
Chimelli/2000	1	44	Sellar, suprasellar extension	TSP resection	Not reported
Curry/1981	1	44	Middle ear, CP angle	GTR	Unknown
Rodriguez/2004	1	53	CP angle, skull base	Posterior fossa craniotomy and resection	Not reported

CP = cerebellar-pontine; GTR = gross total resection; STR = subtotal resection; TSP = transphenoidal approach; XRT = radiation therapy.

cellular atypia, >7 mitoses per 10 high-power fields, and coagulative necrosis.^{4,5,11,21}

Intraorbital myoepitheliomas almost always arise from the lacrimal gland.^{2,3,21–26} There are two reports in the literature of orbital myoepitheliomas not associated with the lacrimal gland.^{2,27} Similarly, intracranial myoepitheliomas are rare and there are no reports of these lesions involving the orbital apex and middle cranial fossa (Table 1). This report details such a lesion and we describe the histopathology and management strategy. Surgery is the mainstay of treatment and the proximity to orbital apex neural and vascular structures necessitates a careful approach to mitigate complications.

CASE REPORT

This 17-year-old right-handed Caucasian female presented with a 3-month history of progressive proptosis of the right eye. She denied headache, blurry vision, or diplopia. Cranial computed tomography (CT) and magnetic resonance imaging (MRI) demonstrated a lobulated, 4.7 × 2.4 × 1.5 cm slightly heterogenous, avidly enhancing, expansile mass at the orbital apex that extended to the adjacent anterior

middle cranial fossa through the superior orbital fissure (SOF) into the extraconal posterior orbit (Figure 1a). The mass displaced the superior rectus and levator palpebrae muscles inferiorly, the optic nerve medially, and the temporal lobe posteriorly. It encased the contents of the SOF and insinuated itself into the optico-carotid recess. The lacrimal gland was notably not involved (Figure 1b). Remodelling and erosion of the adjacent orbital and sphenoid bones, including the anterior clinoid process, were noted (Figure 1c) and a hyperostotic bone spur was observed from the orbital roof. Prominent flow voids along the periphery of the lesion suggested hypervascularity (Figure 1c) and enhancement of the anterior temporal dura suggested dural involvement (Figure 1d). A right superior-lateral orbitotomy approach was chosen for biopsy as the optic apparatus precluded a medial endoscopic approach and a transcranial approach would have been significantly more invasive. Biopsy demonstrated a possible low-grade chondromyxoid tumour. Further review suggested a diagnosis of myoepithelioma, as the tumour was positive for S100, keratin, smooth muscle actin (SMA), and p63; infrequent cells expressing epithelial membrane antigen (EMA) were noted and the tumour was negative for desmin.

FIGURE 1 (a) Axial post-gadolinium T1-weighted MRI depicting an avidly enhancing mass lesion that occupies the posterior orbit, orbital apex and middle cranial fossa. (b) Axial T2-weighted MRI demonstrating multiple flow-voids representing feeding vessels along the periphery of the tumour (arrowheads) as well as a lack of continuity with the lacrimal gland (arrow), suggesting a non-glandular origin. (c) Axial non-contrast cranial CT demonstrating erosion of the anterior clinoid process and sphenoid ridge by the tumour. Also noted is heterotopic ossification along the posterior-medial tumour margin (arrowheads). (d) Axial post-gadolinium T1-weighted MRI demonstrated enhancement of the temporal dura suggesting infiltration of the adjacent dura by tumour. (e) Coronal post-gadolinium T1-weighted MRI demonstrating significant displacement of the contents of the orbital apex.

In light of these biopsy results, the decision was made to surgically remove the lesion as safely as possible. Due to the lesion's location away from the cranial convexity along the middle cranial fossa and orbit, the proximity of the right supraclinoid internal carotid artery and middle cerebral artery, the avid tumour enhancement, and the flow-voids visible on MR within the lesion (Figure 1b), we performed a preoperative cerebral angiogram to evaluate the lesion's

vascularity and embolize feeding vessels. No significant tumour blush was demonstrated, and although small feeding vessels from the M1 segment of the middle cerebral artery were observed, they were not of sufficient calibre to allow safe embolisation. Two days following angiogram, a pterional craniotomy with orbital osteotomy was performed. A complete resection of the tumour and the involved dura of the middle cranial fossa was completed.

FIGURE 2 Axial post-gadolinium T1-weighted image 6 months post-resection. No evidence of residual tumour could be identified.

Postoperatively, the patient demonstrated a right side superior orbital apex syndrome and decreased visual acuity along with a right inferior temporal hemianopsia. Right eye extraocular movements were limited to 10% abduction, 50% supraduction, 90% adduction, and 90% infraduction, representing severe abducent nerve neuropraxia, partial oculomotor nerve neurapraxia, and a superior rectus muscle injury. In addition, a V1 sensory deficit including corneal anaesthesia was noted. These deficits improved gradually. Follow-up imaging at 9 months demonstrates no evidence of recurrence nor metastasis (Figure 2). At her last eye examination 10 months after surgery, she was noted to have a right inferotemporal hemianopsia, minimal limitation of the right eye in adduction along with diplopia in primary gaze, and a persistent right afferent pupillary defect. Her visual acuity had improved to 20/25, her corneal sensation had returned, and the rest of her ocular motility returned back to baseline.

DISCUSSION

Cranial base myoepitheliomas generally arise from ectopic glandular tissue or embryological remnants of vestigial glandular structures.^{7,8,10,28} When seen in the vicinity of the sella, they are purported to originate from salivary gland rests found in the posterior pituitary lobe that communicate with the oropharyngeal remnants of Rathke's pouch.^{7,10,28} In the middle cranial fossa, they are thought to arise from ectopic salivary gland tissue in the middle ear or along the developing chorda tympani.^{8,12} They may also have a dural origin or be entirely contained within bone.^{17,29,30} Fritchie et al. reported a myoepithelioma arising from the parieto-occipital bone with a ESRW-1 mutation¹⁷ and a myoepithelial neoplasm of the clivus is reported as well.²⁹

Intraorbital myoepitheliomas are thought to arise from the lacrimal gland in most cases, although in one report it was thought to arise from the soft tissues of

the lower eye lid.^{2,3,21–25,27} In the case described here, the tumour did not have any continuity with the lacrimal gland neither on preoperative imaging (Figure 1) nor at the time of surgery and there was no involvement of the eyelid. Rather, it appears to have arisen from the sphenoid ridge and extended into the orbit via the SOF. On the cranial side, it involved the dura but remained extra-axial with regards to the brain. The radiographic features, patient's age, and clinical presentation suggested a neoplastic process. The differential diagnosis included rhabdomyosarcoma, meningioma, schwannoma, orbital lymphoma, a venous-lymphatic malformation, or chloroma (myeloid sarcoma). With this differential of lesions requiring a spectrum of highly aggressive to more conservative management dependent on pathology, the need for preoperative biopsy was clear. Given the varied origin of myoepitheliomas, immunohistochemistry is key to accurate diagnosis and complements routine histological stains. Myoepitheliomas are positive for keratin and EMA in conjunction with S100 protein and myogenic markers such as SMA and calponin.^{1,4,5,11} Other markers often found in myoepitheliomas include glial fibrillary acidic protein (GFAP) and p63.^{1,4,5} Desmin positivity may be seen in 14% of myoepitheliomas.^{1,4,5}

Myoepitheliomas and their malignant counterpart, myoepithelial carcinomas, are neoplasms whose phenotype recapitulates normal myoepithelial cells. Neoplastic myoepithelial cells are morphologically heterogeneous and their histological features are not well recognised. Myoepitheliomas are lobulated tumours composed of cords or nests of epithelioid, ovoid, or spindle cells with variable reticular architecture and a chordomyxoid, collagenous, or hyaloid stroma.^{1,3–5,11,20} They may acquire a heterogeneous phenotype, in which case they are designated "mixed-cell tumours" or "pleomorphic adenomas"; these tumours have a significant epithelial (ductal) differentiation. Pure myoepitheliomas and mixed tumours lie on a morphological continuum, with overlap in histological appearance and clinical behaviour.^{3–5,11}

Our tumour demonstrated a mixture of epithelioid (round), spindle, and plasmacytoid (plump) cells (Figure 3b and c) imbedded in a hyalinised, myxoid background (Figure 3d) with a mixed architectural pattern lacking ductal differentiation (Figure 3a), all features consistent with myoepithelioma. Mild atypia, uniform nuclei, and low mitotic index indicated a benign tumour. The tumour demonstrated strong positivity for S100 and keratin, focal positivity for smooth muscle actin, p63, and EMA, and was negative for desmin. No histological features suggestive of carcinomatous differentiation were noted and fluorescence in situ hybridisation (FISH) analysis did not reveal any chromosomal abnormalities.

FIGURE 3 (a, b, c, d) Mixture of epithelioid, spindle and plasmacytoid cells (b, c) imbedded in a hyalinised, myxoid background (d) with a mixed architectural pattern lacking ductal differentiation (a), all features consistent with myoepithelioma. Mild atypia, uniform nuclei, and a low mitotic index indicate a benign tumour. *Note:* Figure 3 of this article is available in colour online at www.informahealthcare.com/op.

There are conflicting reports regarding the optimal treatment strategy for myoepithelial neoplasms. For localised tumours, wide surgical excision is the mainstay of therapy with adjuvant radiotherapy.^{1,18,29,31} Myoepithelial carcinomas may be characterised by nodal spread and these may be addressed surgically as well.³² Chemotherapy has no effectiveness against these tumours.^{1,18,28,29,31} The tumour in this case was well circumscribed, had a soft tissue density, and was partially surrounded by reactive bone. In addition, it was hypervascular, suggesting that a preoperative angiogram may delineate the possibility of embolisation prior to surgery, even though it was not successful in this case.

The location of the tumour at the orbital apex placed it in close proximity to the neurovascular structures exiting the cavernous sinus and entering the orbit through the SOF and optic canal. Surgical access to the lateral aspect of the SOF may be obtained via a lateral orbitotomy; but this limits the exposure of the neurovascular structures traversing within and around the annulus of Zinn and does not provide safe access to the optic canal. The pterional craniotomy provided excellent exposure of the roof and lateral wall of the orbit and SOF and was supplemented by an orbital osteotomy allowing decompression of the periorbital all the way back to the SOF and optic canal,

thus decompressing the optic nerve. In addition, following dural opening, the falciform ligament may be sectioned, completing the decompression of the optic nerve as it enters the subarachnoid space.

The extent of resection may predict the possibility of recurrence. In their series of 101 cases, the largest in the literature to date, Hornick and Fletcher reported a local recurrence of 18% for myoepithelioma and benign mixed-cell tumours, and 42% for myoepithelial carcinomas.⁴ A third of myoepithelial carcinomas will metastasize.^{4,5} These patterns may be more pronounced in paediatric patients.¹ Hence all myoepithelial tumours warrant close surveillance of the primary site following resection and a diagnosis of myoepithelial carcinoma should prompt a metastatic workup, including a CT of the chest, abdomen, and pelvis, and a bone scan.¹⁷

The literature supports the mainstay of treatment of intracranial and intraorbital myoepitheliomas is gross total resection. Radiation therapy has been reported as an adjunctive therapy following subtotal resection or in cases of recurrence; however, its efficacy has not been well defined. In our review, there are a total of six patients in the literature with intracranial myoepithelioma treated with radiation therapy. One patient underwent two subtotal resections of a dural convexity myoepithelioma followed

by radiotherapy and remained recurrence free at the end of a 4-month follow-up.³⁰ Another underwent transphenoidal resection followed by radiation therapy and remained recurrence free at 2 years.¹⁰ Two additional patients underwent subtotal resection and radiation therapy; however, their follow-up was not documented.¹⁰ Two others underwent subtotal resection, chemotherapy, and radiation therapy for a metastatic clival lesion and solitary sellar lesion; however, both patients died from their disease at 14 and 20 months, respectively.^{28,29} Of the three patients in the literature treated with chemotherapy, none have survived beyond 20 months.^{26,28,29}

CONCLUSION

Myoepithelial neoplasms are rare tumours that can arise from salivary glands or soft tissue structures. Intracranial myoepitheliomas are rare and when they involve the cranial base or orbital apex, surgical approaches require careful planning to minimize morbidity. Although commonly benign and slow growing, myoepitheliomas may invade bone and dura and in rare instances differentiate into frankly malignant lesions with metastatic potential. Surgical excision is the mainstay of therapy, with radiation therapy reserved for unresectable or recurrent lesions. Chemotherapy has not been effective in the management of myoepithelial neoplasms, but is reserved for unresectable, progressive, or metastatic myoepithelial carcinomas. A metastatic workup and long-term radiographic follow-up for all myoepithelial tumours is recommended.

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