

Review

Chondromyxoid Fibroma of the Mastoid: A Rare Entity with Comprehensive Literature Review

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Chondromyxoid fibroma (CMF) is the least commonly occurring bone tumor of cartilaginous origin. It is usually situated in the metaphysis of long bones of the lower limbs. Localization of the tumor in the skull is extremely rare. The definitive diagnosis is challenging and depends on radiological and histological examinations. To the best of our knowledge, only 14 cases of CMF involving the temporal bone have been reported to date, 7 of which were within the mastoid. The most common clinical symptom is headache; however, these symptoms vary greatly according to site, size, and extension of the lesion. Surgical removal is the treatment of choice. A literature review of the diagnostic challenges, histological difficulties in differential diagnosis, imaging, clinical features, and recommended modalities of treatment have been discussed in the present case.

KEYWORDS: Chondromyxoid fibroma, differential diagnosis, mastoid neoplasm, skull base lesions, temporal bone tumor

INTRODUCTION

Chondromyxoid fibroma (CMF) is a rare, slow-growing, benign tumor of chondroplastic origin that was first reported as a distinct entity by Jaffe and Lichtenstein in 1948^[1]. It represents less than 5% of all bone tumors^[2]. Involvement of the metaphysis of the long bones, particularly those around the knee joint, is considered to be the most common site occurrence of this lesion. The overall incidence of CMF in the craniofacial bone ranges from 2% to 5%, while isolated temporal bone involvement is particularly rare^[3]. In the skull, CMF develops from embryonic cell rests that are entrapped at the suture lines during endochondral ossification^[4,5]. No predisposing factors have been described; however, it is believed that this tumor has a genetic origin. The genetic mechanism underlying the pathogenesis of CMF, despite extensive target gene analyses, remains poorly understood. Recurrent cytogenetic findings have implicated gene(s) on chromosome 6. Associations with recurrent rearrangements of chromosome bands 6p23-25, 6q12-15, and 6q23-27 have been reported^[6]. Although radiological images show characteristic features, an ultimate diagnosis cannot be made with imaging alone and biopsy is necessary for histopathological examination. Since this lesion is so rare in the temporal bone, we have shared our case and the management strategy we employed based on a review of the literature.

METHODS

A chondromyxoid fibroma in the mastoid part of temporal bone was reported. The clinical presentation and key radiographic and histopathological features of the tumor were discussed. Also, the literature was reviewed to further characterize this pathology and to assess management strategies.

A literature review in PubMed, Scopus, the Cochrane library, and Embase was conducted using the search terms chondromyxoid fibroma in combination with temporal bone and/or mastoid and/or skull base. A total of 131 reports were retrieved and their abstracts were screened. Non-English reports and lesions outside the temporal bone were excluded. Also, other histopathological findings that were not consistent with CMF were excluded from further reading.

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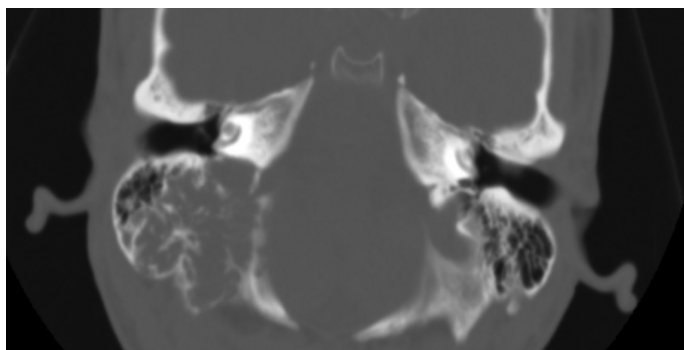


Figure 1. Axial CT scan shows typical bony erosion of the mastoid with central amorphous calcifications. The arrow indicates partial erosion of the bony plate overlying the posterior fossa dura.

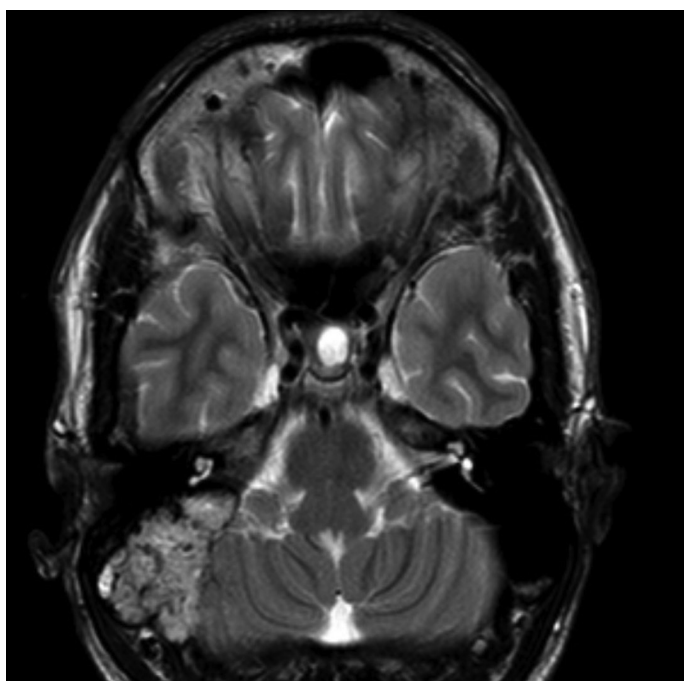


Figure 2. An MRI scan shows a large lobulated mass in the right temporal bone with a typical hypointense signal on T1 and heterogeneously hyperintense signal on T2.

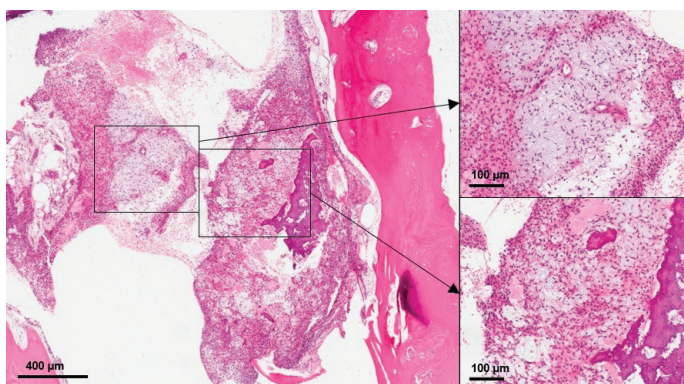


Figure 3. Histopathological microscopic examination shows a variable cellular lesion located within preexisting bone structures. The lesion has a vaguely lobular pattern (Figure 3, left side). At higher magnification, it was seen that the lesion is composed of stellate or spindle-shaped cells suspended in a myxoid background (Figure 3, top right). The centers of the lobules are hypocellular with hypercellular peripheries. The tumor cells exhibit no cellular atypia, pleomorphism, or mitotic activity. Focally coarse calcifications are present (Figure 3, bottom right).

RESULTS

An otherwise healthy 21-year-old man was referred to our tertiary medical center for the evaluation of an incidental computed tomography (CT) finding within his right temporal region following blunt head trauma. He was employed as a construction worker with an irrelevant past medical and surgical history. He had no prior otological complaints and presented normal findings on otolaryngological and neurologic physical examination. His pure tone and speech audiometry revealed normal thresholds for age and sex.

The CT scan demonstrated a soft tissue lesion infiltrating almost the entire right mastoid with the destruction of bony septa and a thin bony cortex around the lesion, except for partial erosion toward the cerebellum. The posterior fossa dura appeared intact. Magnetic resonance imaging (MRI) displayed a 4.7 cm×2.6 cm×3.6 cm lobulated mass in the right mastoid part of the temporal bone, extending anteriorly to the jugular foramen and posteriorly to the bony plate overlying the posterior fossa dura, which was eroded without evidence of intracranial extension (Figure 1). The lesion had a diffuse hypointense signal on T1-weighted images and a heterogeneous hyperintense signal on T2-weighted imaging, with significant post-gadolinium diffuse contrast enhancement (Figure 2). Because of its heterogeneous nature and the involvement of the jugular vein, the radiological report mentioned a possible glomus jugulare tumor. However, magnetic resonance angiography showed the tumor to be avascular. Since a neoplastic process was mentioned in the differential diagnosis, surgical access to obtain a tissue diagnosis was recommended.

The patient was taken to the operating room and mastoidectomy and surgical debulking were performed via a retro-auricular approach. An irregular, firm, and bloody lesion was excised intraoperatively for histopathological evaluation (Figure 3).

Microscopic examination revealed a distinct lobular architecture, with cellular areas of chondrocytes and stellate cells at the periphery of the lobules alternating with central myxoid areas that were less cellular and showed cystic degeneration. The cells ranged from spindled to stellate, containing round to ovoid normochromatic nuclei with indistinct to eosinophilic cytoplasm. There were no areas of necrosis and no discernible mitotic activity. These features are consistent with the histopathological diagnosis of chondromyxoid fibroma.

Our patient recovered well after the debulking procedure. After he was completely recovered, he was counseled about CMF in the temporal bone being a benign lesion that required further surgery. We advised complete resection but the patient refused further treatment. Therefore, as the second-best option, periodic follow-ups have been planned.

Our review identified only 14 previous case reports of CMF in the temporal bone, 7 of which were within the mastoid. All published cases were studied, a summary of which is shown in Table 1. This includes patient demographics, clinical presentation, radiological findings, and management strategies.

An analysis of the 14 reports with our case revealed a male to female ratio of 60%-40%. The vast majority (67%) of patients were affected by CMF on the left temporal bone. The mastoid was by far the most commonly affected site at 53%. The second most popular site was

Table 1. Summary of case reports including patients' demographics, clinical and radiological characteristics, and management strategy

Study	Age(y)	Sex	Presentation	Side	Lesion site & size	Management strategy
Tarhan et al. ^[2] 2000	44	F	Left side facial pain	Left	2.5 x 2 x 1.5 cm, in the temporal bone, compressing the temporal lobe	Complete resection
Haberal et al. ^[4] 2001	45	F	Left side facial pain & numbness	Left	2.5x2.5x1.5 cm in the anterior portion of the left tympanic temporal bone	Complete macroscopic resection after recurrence
LeMay et al. ^[5] 1997	22	M	Headaches & mild hearing loss	Left	6 x 4.5 x 3 cm, from the superior & medial portion of the mastoid	Complete resection via craniotomy
Oh et al. ^[20] 2013	38	F	left-sided hearing loss	Left	4.1-cm mass within the mastoid bone	Complete excision with facial nerve skeletonization
Kitamura et al. ^[21] 1989	48	M	Left aural fullness, tinnitus & dizziness	Left	Mastoid, extending to the occipital bone, foramen magnum & jugular foramen	Incomplete resection due to bleeding. Followed by revision one year latter
Maruyama et al. ^[22] 1994	67	M	Headache, & facial nerve paralysis	Right	Petrous apex & extended into the posterior fossa & the jugular foramen	Incomplete resection due to jugular foramen involvement
Frank et al. ^[24] 1987	26	M	Diplopia	Left	Petrous apex, extending into sphenoid sinus, & cavernous sinus area	Resection via sub-temporal approach; V1 sacrificed
Patino-Cordoba et al. ^[25] 1998	20	M	Hearing loss	Left	Mastoid with inferior extension	Resection via infratemporal fossa approach, with neck dissection
Suzuki et al. ^[26] 1999	49	M	Visual disturbance	Left	Squamous temporal bone	Preoperative embolization & resection
Otto et al. ^[27] 2007	58	F	Acute-onset vertigo & syncope	Right	1.7 x 1.3 x 1.5 cm. filling the mastoid with erosion of the posterior fossa plate	Complete resection via mastoidectomy
Thompson et al. ^[28] 2009	33	F	Progressive facial nerve paralysis	Left	Mastoid & protruded out of the stylomastoid foramen	Complete resection with interposition of a sural nerve graft
Wang et al. ^[29] 2011	31	M	Headache	Right	2.5 x 2 x 1.5 cm in the petrous apex	Complete resection via craniotomy
Sharma et al. ^[30] 2012	12	F	Headache with left earache	Left	4 x 3.7 x 4.4 cm in the squamous part of temporal bone	Complete excision
Gupta et al. ^[31] 2012	42	M	otalgia	Right	1.6 x 1.2 cm eroding the mastoid, facial nerve canal & sigmoid plate	Biopsy via transmastoid approach & definitive resection was scheduled
Our case	21	M	Incidental finding	Right	4.7 x 2.6 x 3.6 cm in the mastoid, & extended into the posterior fossa & the jugular foramen	Biopsy via transmastoid approach

F: female; M: male; V1: ophthalmic branch of the trigeminal nerve

equally shared between the petrous and squamous portion of the temporal bone at 20% each, while only 7% of CMF were noted in the tympanic part of temporal bone. The mean age of the patients at the time of surgery was 37 years (range: 12-67 years).

DISCUSSION

Chondromyxoid fibroma is the least common neoplasm of cartilaginous origin and represents less than 0.5% of all bone tumors. There is a slight predilection for males with peak incidence being in the second and third decades of life ^[7, 8].

The clinical presentation varies according to the size, site, and extension of the lesion. In cases with skull involvement, it presents clinically as headache, bony swelling, neuralgia, facial pain, hearing loss, otalgia, convulsions, diplopia, exophthalmos, and facial nerve paralysis (Table 1).

Although the radiographic features of such lesions are characteristic, due to its rarity, CMF is not usually at the top of a radiologist's list of possible diagnoses. X-ray shows a radiolucent lesion with well-defined margins ^[9]. CT scan findings of CMF demonstrate a relatively homogenous, well-circumscribed, osteolytic lesion with a wavy bony

Table 2. Differential diagnosis of chondromyxoid fibroma

<i>Chondromyxoid fibroma CMF</i>	<ul style="list-style-type: none"> • Hypocellular lobules of spindle-shaped or stellate cells that proliferate within an abundant intercellular matrix, which can be either myxoid or chondroid in nature. • Lobules are separated by a highly cellular tissue which contains many spindle-shaped cells and fibrous bands with a variable number and size of multinucleated giant cells. • Absent anaplastic changes. • Immunohistochemical analysis shows positivity for vimentin and the S100 protein. 	
	Histopathological similarities	Differentiation
<i>Chondrosarcoma</i>	<ul style="list-style-type: none"> • Lobular growth pattern • Contains myxoid areas • As high cellularity as CMF • Immunohistochemical analysis also positive for vimentin and the S100 protein 	<ul style="list-style-type: none"> • Presence of bony trabeculae • Absent fibrous component • Well-differentiated hyaline matrix • Invasive growth pattern • Abundant mitosis • The tumor cells are arranged in cords surrounded by myxoid stroma
<i>Chondroblastoma</i>	<ul style="list-style-type: none"> • Multinucleated giant cells • Chondroid matrix • Immunohistochemical analysis also express vimentin and S100 protein 	<ul style="list-style-type: none"> • Fetal chondroblasts are characteristic. They are round and consist of abundant cytoplasm, with distinct cytoplasmic membranes. Nuclei are also round and are located in the center of the cytoplasm (a “fried egg” appearance)
<i>Chordoma</i>	<ul style="list-style-type: none"> • Chondroid form of Chordoma • Myxoid matrix 	<ul style="list-style-type: none"> • Infiltrative margin • Physaliferous cells with an eosinophilic foamy cytoplasm with numerous vacuoles which are organized into masses or along trabeculae • Immunohistochemistry is positive for epithelial antigens (cytokeratin, epithelial membrane antigen)
<i>Giant cell tumor</i>	<ul style="list-style-type: none"> • Multinucleated giant cells 	<ul style="list-style-type: none"> • Osteoclastic giant cells with a peculiar spatial arrangement with stromal fragments • Lack of chondroid differentiation
<i>Fibrous dysplasia</i>	<ul style="list-style-type: none"> • When myxoid changes occur in fibrous dysplasia 	<ul style="list-style-type: none"> • Irregularly shaped trabeculae of immature woven bone • Variably cellular background with loosely arranged fibrous stroma
<i>Osteosarcoma</i>	<ul style="list-style-type: none"> • Chondromyxoma-like and chondroblastic variant of low-grade osteosarcoma contain very large CMF-like areas 	<ul style="list-style-type: none"> • Presence of osteoid production • Presence of mitoses and nuclear atypia
<i>Enchondroma</i>	<ul style="list-style-type: none"> • Lobular growth pattern 	<ul style="list-style-type: none"> • Well-differentiated hyaline cartilage

CMF: Chondromyxoid fibroma

margin and foci of calcification^[4, 10]. Intratumoral calcification is reported to be closely related to craniofacial CMF than CMF in long bones^[2, 5]. Consistent with these results, our case also exhibited matrix calcification that was apparent on CT. MRI is used to determine the extension of the lesion to the dura and the intracranial space. Chondromyxoid fibromas are typically hypointense (low signal) on T1-weighted MRI and heterogeneously hyperintense (high signal) on T2-weighted imaging. The lesion is markedly enhanced after gadolinium contrast administration^[11–13].

In the case presented, all the radiological features were characteristic of CMF. Nevertheless, a definite diagnosis could not be made and rhabdomyosarcoma could not be excluded. Therefore, biopsy was necessary to achieve a definitive diagnosis.

The differential diagnosis of CMF based on histopathological criteria includes several chondroid lesions such as chondroblastoma, enchondroma, chondrosarcoma, chordoma, giant cell tumor, and fibrous dysplasia^[14] (Table 2). The World Health Organization defines CMF as a benign tumor characterized by lobules of spindle-shaped or stellate cells that proliferate within an abundant intercellular matrix that can be myxoid, chondroid or fibrous in nature. The lobules are separated by areas of tissue that are highly cellular and contain many spindle-shaped or round cells associated with a variable number of multinucleated giant cells of different sizes^[15]. It is crucial to differentiate between CMF and chondrosarcoma because the management and prognosis differ significantly. It was reported that about 22% of CMF cases were misdiagnosed as chondrosarcoma^[16]. Chondroblastoma is also benign cartilaginous tumor that can mimic CMF, howev-

er, histopathologically it lacks stellate cells^[17]. Immunohistochemical analysis is not helpful in differentiating chondrosarcoma, chondroblastoma, and CMF since they all express vimentin and S100 protein^[11, 17]. Conversely, immunohistochemical analysis is useful in diagnosing the chondroid form of a chordoma, as the expression of cytokeratin and epithelial membrane antigen is positive in chordoma and negative in CMF. Chordomas can also be distinguished from CMF by the anatomical location, as they are mostly located in the midline and display more bone destruction with extraosseous extension^[18]. Fibrous dysplasia has characteristic radiological and histopathological features, however, myxoid changes might occur, which may result in another diagnostic challenge. Chondromyxoma-like low-grade osteosarcoma can mimic CMF, which may also be a challenge for the cytologist to identify. Nevertheless, osteosarcomas often show lobular arrangement of cartilaginous areas and the presence of osteoid. Lastly, giant cell tumor was considered in the differential diagnosis. However, it lacks chondroid differentiation and contains numerous clustered giant cells^[19].

Apart from its rarity and unexpected diagnosis, another histopathological diagnostic challenge is the excision of a small biopsy specimen that contains only part of the characteristic features of CMF. Therefore, the ultimate diagnosis is usually reached after complete surgical excision. The surgical approach in skull base lesions is to drill out the tumor, removing as much as possible. The risks of post-operative neurological deficit have to be weighed against benefits of complete excision^[4, 9, 11].

With regard to the current review, follow-up data was only available in less than half of the cases, ranging from 3 months as reported by LeMay et al.^[5] and Morimura et al.^[13] to 4 years as described by Oh et al.^[20]. Interestingly, a 1-year post-operative follow-up showed recurrence and required revision surgery^[4].

The surgical challenges seem to depend on the site, size, and extension of the lesion. Though complete surgical excision was described in 12 reports of CMF of the temporal bone, 6 of these patients experienced major post-operative complications. These complications included hearing loss, hoarseness, sacrifice of the ophthalmic branch of the trigeminal nerve, sacrifice of the facial nerve, and recurrence. Biopsy was carried out with definitive resection planned in more than one report, similar to the presented case. Incomplete resection due to encountering a challenging surgical situation was also mentioned. Unfortunately, the follow-up of these cases were not reported^[21, 22].

Although malignant change of CMF is very low, radiotherapy does appear to increase the incidence^[16, 23]. Nevertheless, some authors use radiotherapy for incomplete resection or recurrence after surgical excision, particularly for skull base lesions^[11, 12, 17, 21].

CONCLUSION

Chondromyxoid fibroma is a benign but potentially aggressive tumor. Involvement of the skull base, particularly the temporal bone, is extremely rare and therefore it is difficult to establish treatment protocols that are universally applicable. Although CMF has a very characteristic radiological appearance, histological differentiation from other lesions with chondroid origin necessitates biopsy. Whenever possible, complete resection is recommended, since radiother-

apy for inoperable or recurrent lesions carries the risk of malignant transformation.

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