Primary Myoepithelioma of Bone
A Report of 8 Cases

Pawel Kurzawa, MD, PhD, Susan Kattapuram, MD,† Francis J. Hornicek, MD, PhD,‡ Cristina R. Antonescu, MD,§ Andrew E. Rosenberg, MD,‖ and G. Petur Nielsen, MD¶*

Abstract: The clinical and pathologic features of 8 primary myoepitheliomas of bone were analyzed. There were 5 female and 3 male patients who ranged in age from 16 to 49 (mean, 33.5) years. Three tumors arose in the ilium, 2 in the tibia, and 1 each in the maxilla, sacrum, and L1 vertebral body. Microscopically, the tumors had a solid, lobulated, reticular, or storiform growth pattern and were predominantly composed of spindle-shaped cells arranged in intersecting fascicles with eosinophilic cytoplasm. The round to polygonal epithelioid cells were arranged randomly or formed small clusters and contained variable amounts of eosinophilic or clear cytoplasm. Immunohistochemically, all the tumors were positive for vimentin and S100 protein, and 7 were positive for epithelial membrane antigen. No tumors were positive for keratin (AE1.3/CAM5.2). Smooth muscle actin was positive in 3 tumors and negative in 4, whereas desmin was negative in all 7 tumors tested. Nuclear staining for p63 was negative in 3 tested tumors. Staining for GFAP and CD34 was performed on 4 and 5 tumors, respectively, and all showed no expression. Fluorescence in situ hybridization for *EWSR1* rearrangement was performed in 7 tumors. Five tumors (71%) showed the presence of *EWSR1* gene rearrangement, and 2 were negative. Cytogenetic studies conducted on 1 tumor showed 46,XY,t(1;22)(q21;q12) associated with *EWSR1-PBX1* fusion. Surgical procedures included curettage in 3 patients, resection in 3 patients, and 2 patients only had an open biopsy. Follow-up information was available for 4 patients; all remain free of disease with no recurrence. Although experience with primary myoepithelioma of bone is limited, histologically, banal tumors appear to behave in a benign manner, and conservative surgery appears to be sufficient treatment. Immunohistochemical and molecular analyses are helpful in their accurate identification.

From the *Department of Pathology, Greater Poland Cancer Centre, Poznan, Poland; Departments of †Radiology; ‡Orthopedics; ¶Pathology, Massachusetts General Hospital, Boston, MA; §Department of Pathology, Memorial Sloan-Kettering Cancer Center, New York, NY; and ‖Department of Pathology, University of Miami Hospital, Miami, FL.

A.E.R. and G.P.N are co last authors.

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Correspondence: G. Petur Nielsen, MD, Department of Pathology, Massachusetts General Hospital, 55Fruit street, Boston, MA 02114 (e-mail: gnielsen@partners.org).

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Myoepithelial tumors are composed of neoplastic cells that have a myoepithelial phenotype; in some cases these tumors may also contain a second population of cells that are purely epithelial. Normal myoepithelial cells exhibit the structural and functional characteristics of both epithelial and mesenchymal cells, and the expression of these traits in myoepithelial neoplasms underlies the diverse morphologic and biological spectrum of these tumors.[1,2]

The characteristics of myoepithelial cells include the secretion of mucins such as the acid glycosaminoglycans (hyaluronic acid, heparin sulfate, chondroitin-4-sulfate, chondroitin-6-sulfate), the basement membrane constituents elastin and tenascin, the intermediate filament vimentin, the contractile filaments actin and myosin, as well as calponin and S100 protein.[3–7] The epithelial component of myoepithelial cells is reflected in their expression of keratin and/or epithelial membrane antigen and in the presence of tonofilaments ultrastructurally.

The composition of myoepithelial tumors and their location forms the basis of their classification; tumors with distinct epithelial and mesenchymal phenotypes are known as pleomorphic adenoma (mixed tumor) in salivary glands and mixed tumor (chondroid syringoma) in the skin and other locations, whereas neoplasms composed of cells that have a predominant mesenchymal phenotype, regardless of the site of origin, are known as myoepithelioma.[8–11] These tumors arise in salivary glands, skin, upper airway, lung, gastrointestinal tract, breast, soft tissue, and other unusual sites including bone.[12–15]

Myoepithelioma of bone is rare; only 9 cases have been reported, and they have arisen in the head and neck region and the appendicular and axial skeleton.[16–26] Because of their rarity, unusual morphology, and intraosseous origin, they have frequently caused diagnostic difficulties. To increase our understanding of the biological features of these tumors we report herein our experience with 8 primary intraosseous myoepitheliomas.

MATERIALS AND METHODS

The cases were retrieved from the files at the Massachusetts General Hospital and the consultation files of

TABLE 1. Panel of Antibodies Used in This Study

Antigen	Clone	Dilution	Antigen Retrieval	Source
CK AE1.3/CAM5.2	AE1.3/CAM5.2	1:160	Protease 1	Covance, CA
CD34	QBEnd/10	Prediluted	CC1 short (Ventana)	Ventana Medical System, AZ
S100	None (polyclonal antibody)	Prediluted	No	Ventana Medical System
SMA	1A4	Prediluted	CC1 short (Ventana)	Ventana Medical System
Calponin	CALP	1:180	CC1 Mild (Ventana)	Dako Corp., CA
CD163	10D6	1:150	CC1 Mild (Ventana)	Leica Microsystems, IL
EMA	E29	Prediluted	CC1 Standard (Ventana)	Ventana Medical System
Vimentin	V9	Prediluted	CC1 Mild (Ventana)	Ventana Medical System
CD99	013	1:60	CC1 Short (Ventana)	Covance
LCA	RP2/18	Prediluted	CC1 Short (Ventana)	Ventana Medical System
HMB45	HMB45	1:100	CC1 Standard (Ventana)	Dako Corp
Mel-A	A103	Prediluted	CC1 Standard (Ventana)	Ventana Medical System
Desmin	DE-R-11	Prediluted	Protease 1	Ventana Medical System
p63	4A4	1:200	CC1 Mild (Ventana)	Neo Markers, CA
Caldesmon	h-cD	1:2	CC1 Standard (Ventana)	Biogenex, CA
GFAP	EP672Y	Prediluted	CC1 Standard (Ventana)	Ventana Medical System

2 of the authors (A.E.R., G.P.N.). Intraosseous origin of the tumor was confirmed by review of the radiographic images (5 cases) or the radiology reports (3 cases). Clinical data were obtained from the medical records, referring pathologist, or from the patient's treating physician. Two tumors (cases 7 and 8) were previously included in the study by Antonescu et al.[27]

The diagnosis of myoepithelioma by light microscopy was based on the criteria delineated by Hornick and Fletcher.[13] Immunohistochemical analysis was performed on all 8 tumors utilizing standard techniques; the antibodies, clones, dilutions, pretreatment conditions, and sources are listed in Table 1.

The hematoxylin and eosin-stained sections (ranging from 2 to 32 with an average of 15 hematoxylin and eosin-stained sections per case) were examined, and the neoplasms were assessed for their architecture, margin of growth, cell morphology, mitotic activity, and necrosis.

Fluorescence in situ hybridization (FISH) for *EWSR1* rearrangement was attempted in 7 tumors. It was performed on interphase nuclei from paraffin-embedded 4-μm-thick sections. We applied custom probes using a bacterial artificial chromosome (BAC) probe for EWSR1 (RP11-77M13; RP11-965D15; RP11-945M21; RP11-155B12; RP11-551L12; RP11-794O14), covering and flanking *EWSR1* in 22q12.

BAC clones were chosen according to the USCS genome browser (http://genome.UCSC.edu). The BAC clones were obtained from BACPAC sources of Children's Hospital of Oakland Research Institute (Oakland, CA) (http://bacpac.chori.org). DNA from individual BACs was isolated according to the manufacturer's instructions, labeled with different fluorochromes in a nick translation reaction, denatured, and hybridized to pretreated slides. Slides were then incubated, washed, and mounted with DAPI in an antifade solution, as previously described. The genomic location of each BAC set was verified by hybridizing them to normal metaphase chromosomes. Two hundred successive nuclei were examined using a Zeiss fluorescence microscope (Zeiss Axioplan, Oberkochen, Germany), controlled by Isis 5 software (Metasystems). A positive score was interpreted when at least 20% of the nuclei showed a breakapart signal. Nuclei with incomplete set of signals were omitted from the score.

One tumor (#7) was sent for cytogenetic karyotype analysis and processed according to standard techniques.

TABLE 2. Summary of Clinicopathologic, Immunohistochemical, and Molecular Features

No	Age	Sex	Site	Extra-osseous Extension	Size (mm)	Surgical Procedure	Follow-up Time (mo)	Symptoms	S100	EMA	Vim	CD34	Keratin AE1.3/Cam5.2	SMA	Desmin	P63	GFAP	FISH
1	31	F	Tibia	No	NA	Open biopsy	NA	NA	Pos	Pos	Pos	Neg	Neg	Neg	Neg	ND	ND	Pos
2	42	F	Maxilla	No	25 × 20 × 5	Resection	NA	Pain, discomfort	Pos	Pos	Pos	Neg	Neg	ND	ND	ND	ND	Neg
3	21	M	Ilium	No	NA	Open biopsy	NED (4)	NA	Pos	Pos	Pos	ND	Neg	Neg	Neg	Neg	Neg	TF
4	37	M	Ilium	Yes	57 × 43 × 40	Resection	NA	Pain	Pos	Pos	Pos	Neg	Neg	Pos	Neg	Neg	Neg	Pos
5	27	F	Sacrum	No	34 × 28	Biopsy and curettage	NA	Pain	Pos	Pos	Pos	Neg	Neg	Neg	Neg	ND	Neg	Neg
6	16	F	Tibia	No	34 × 17 × 5	Curettage	NED (16)	Discomfort	Pos	Pos	Pos	Neg	Neg	Pos	Neg	ND	ND	Pos
7	49	M	Ilium	Yes	15 × 4 × 1	Curettage	NED (21)	Pain	Pos	Pos	Pos	ND	Neg	Pos	Neg	ND	ND	Pos
8	45	F	L1	No	40 × 32 × 20	Resection	NED (35)	Pain	Pos	Neg	Pos	ND	Neg	Neg	Neg	Neg	Neg	Pos
Total									8/8	7/8	8/8	5/5	0/8	3/7	0/7	0/3	0/4	5/7

Case #7 was also sent for cytogenetics, which showed t(1;22) with *EWSR1-PBX1* fusion.
NED indicates no evidence of disease; NA, not available; ND, not done; TF, technical failure.

RESULTS

Clinical Findings

The age of the patients at the time of diagnosis ranged from 16 to 49 (mean, 33.5) years and included 5 female and 3 male patients. Presenting clinical symptoms was known in 6 patients who reported pain of variable duration (4 mo to 2.5 y). Three tumors arose in the ilium, 2 in the tibia, and 1 each in the maxilla, sacrum, and L1 vertebral body (Table 2).

The surgical procedures included curettage in 3 patients, tumor resection in 3 patients, and 2 patients had only an open biopsy.

Clinical follow-up information was available for 4 patients and ranged from 4 to 35 (mean, 19) months. No patient developed metastases, and the tumors that were curetted or resected did not recur within the follow-up period.

Imaging studies for our review were available for 5 patients. Radiographically, the tumors were centered in bone, were radiolucent, had well-circumscribed sclerotic margins, and in some cases they were expansile and induced a thick layer of periosteal new bone (Figs. 1, 2). In 2 patients (cases 4 and 7) (Figs. 3, 4) the neoplasms eroded the cortex, remained confined by the periosteum, but "pushed" into the adjacent soft tissues. On a computed tomographic scan the tumors were homogenous in density and exhibited well-defined margins. Magnetic resonance imaging revealed that the tumors had a hypointense signal on T1-weighted images and a heterogenous signal intensity on T2-weighted sequences. They enhanced uniformly with gadolinium.

Macroscopic Features

Grossly the tumors were described as mucoid or cartilaginous-like in appearance, and their cut surfaces were white, red-gray, or pink and focally hemorrhagic (Fig. 5).

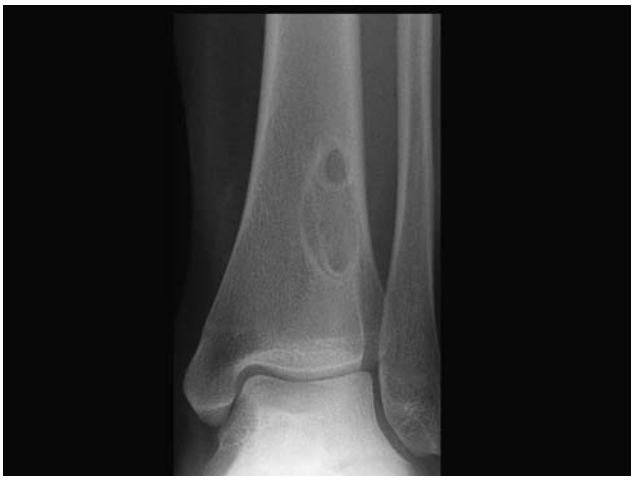

FIGURE 1. Myoepithelioma presents as eccentrically located lucent lesion along the distal anterior cortex of the tibia. The margins are well defined and sclerotic, characteristic of a benign tumor.

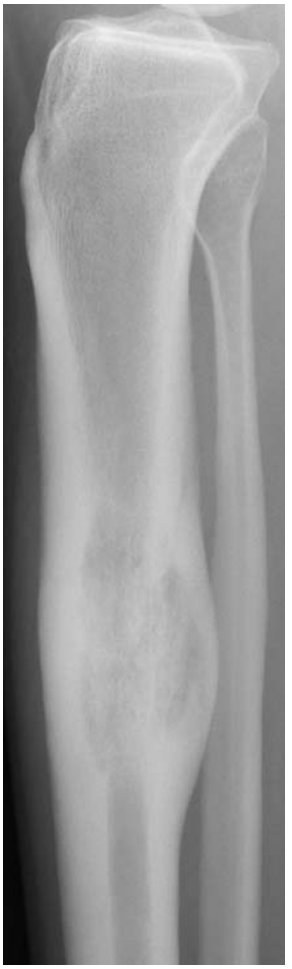

FIGURE 2. Lateral view of the lower leg shows an expansile myoepithelioma in the mid shaft of the tibia. The mass is mixed lytic and radiodense, has fairly well-circumscribed margins, and has induced a thick layer of periosteal new bone formation both proximally and distally.

Microscopic Features

The tumors were well circumscribed and did not demonstrate an infiltrative growth pattern with regard to the preexisting bone or soft tissues. In 2 patients the tumors eroded the cortex and bulged into the adjacent soft tissues with a "pushing" margin (Fig. 6).

Architecturally the tumors showed solid, lobulated, reticular, or storiform growth patterns (Figs. 7, 8). They were composed of spindle cells, which constituted 30% to 90% of the tumor cell population, and epithelioid cells. The spindle cells had elongate nuclei with tapered ends and eosinophilic cytoplasm and were arranged in intersecting fascicles of varying length (Fig. 9). In 2 tumors the nuclei had conspicuous nucleoli and cytoplasmic pseudoinclusions. The round to polygonal epithelioid cells were arranged randomly or in small clusters and contained round nuclei and variable amounts of eosinophilic cytoplasm (Fig. 10). Neoplastic cells with clear cytoplasm were present in 3 tumors (Fig. 11), and so-called

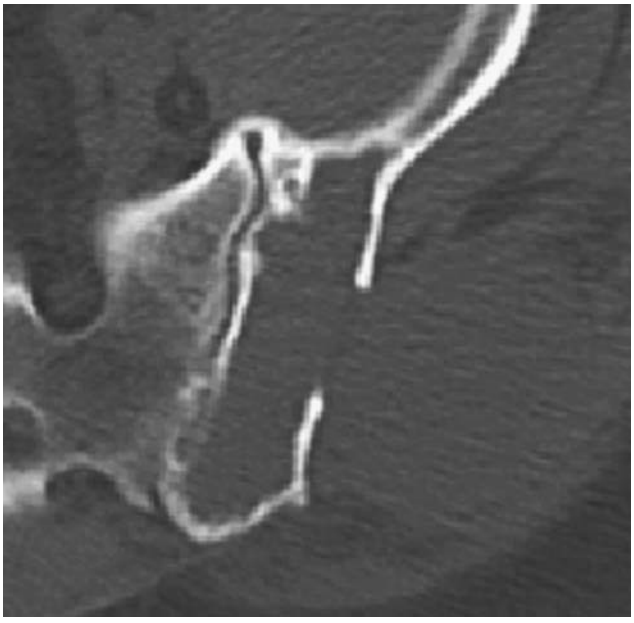

FIGURE 3. Axial computed tomographic scan shows a soft tissue density mass arising in the posterior ilium that erodes the cortex and extends in the soft tissues forming a large component in the gluteal region. The bone margins are well defined.

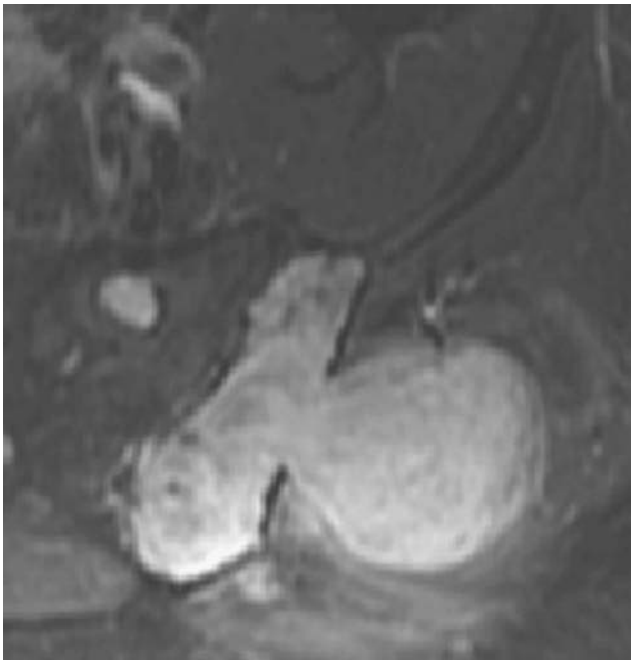

FIGURE 4. Axial T1-weighted image with fat saturation after gadolinium administration shows that the mass is enhancing and replaces the marrow in the posterior ilium. Note the sharp demarcation anteriorly. The soft tissue component also is uniformly enhancing with minimal surrounding edema and inflammation.

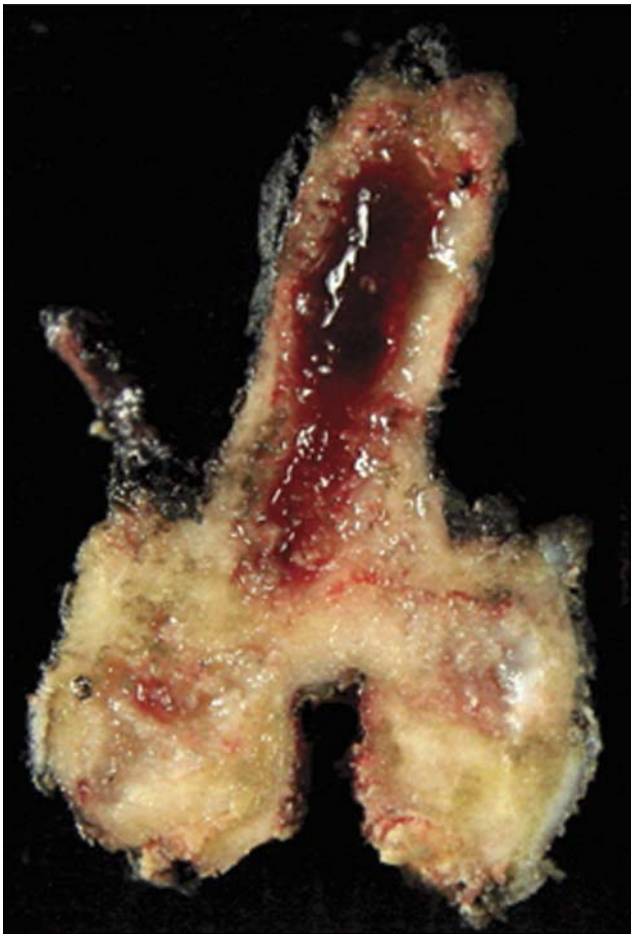

FIGURE 5. Resected myoepithelioma centered in the posterior element of L1 demonstrates a hemorrhagic, mucoid tumor expanding the spinous process.

"hyaline" cells with eccentric densely eosinophilic cytoplasm formed a minor component in 2 tumors. One neoplasm was composed mainly of small round cells arranged in strands, cords, or nests embedded in extracellular myxoid stroma. Nuclear pleomorphism was minimal, and focal necrosis was seen in 1 tumor (Fig. 12).

In 2 tumors (cases 4 and 7) the cells demonstrated mild cytologic atypia manifest by slight nuclear enlargement, vesicular chromatin, and visible nucleoli.

Mitotic activity was low, and mitotic counts identified 0/10 HPF in 3 tumors, 1/10 HPF in 4 tumors, and 3/10 HPF in 1 tumor (Fig. 9). All mitotic figures were structurally normal.

The extracellular matrix was collagenous, hyalinized, or myxoid. No epithelial differentiation or cartilaginous areas were identified in any tumor.

Immunohistochemical Findings

All of the tumors were positive for vimentin and S100 protein, and 7 were positive for EMA (Figs. 13–15). One tumor (case 8) was negative for EMA; however, it demonstrated characteristic morphology, and FISH

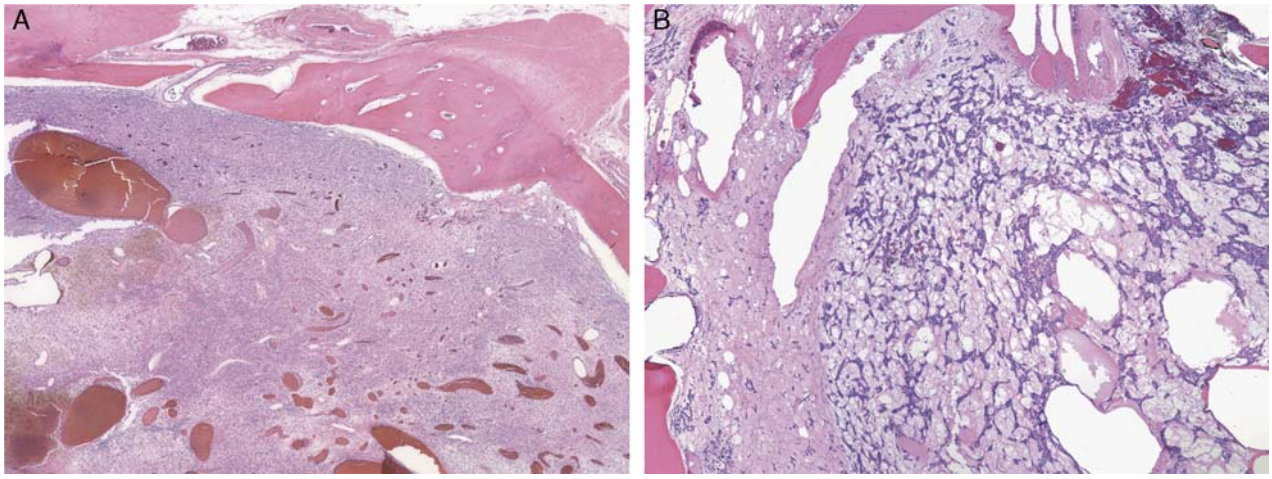

FIGURE 6. A and B, The myoepitheliomas have well-defined margins with the cortex and cancellous bone.

confirmed *EWSR1* rearrangement. None of the tumors were positive for keratin (AE1.3/CAM5.2). Smooth muscle actin was positive in 3 tumors and negative in 4, whereas desmin was negative in all 7 tumors tested. Nuclear staining for p63 was negative in 3 tested tumors, and GFAP and CD34 were negative in 4 and 5 tumors, respectively. One tumor showed negative staining for HMB45, melan-A, LCA, CD99, and CD163 (Table 2).

Fluorescence In Situ Hybridization

FISH for *EWSR1* rearrangement was performed in 7 tumors (Fig. 16). One tumor was excluded because of technical failure. Five tumors (71%) showed the presence of *EWSR1* gene rearrangement, and 2 tumors were negative.

Cytogenetic Studies

Cytogenetic studies conducted in 1 tumor (case 7) showed 46,XY,t(1;22)(q21;q12) associated with *EWSR1-PBX1* fusion.

DISCUSSION

The histologic classification of myoepithelial tumors is based on their location, cell composition, and biological behavior. Myoepithelial tumors containing an epithelial component by light microscopy are known as pleomorphic adenoma, mixed tumor, and chondroid syringoma, depending on their site of origin. Neoplasms composed solely of myoepithelial cells are known as myoepithelioma when benign and myoepithelial carcinoma or malignant myoepithelioma when malignant.

Myoepitheliomas are rare and are usually found in locations such as the salivary glands (most common), skin, upper airway, gastrointestinal tract, lung, breast, soft tissue, and, rarely, bone.[14] Because they have biphenotypic differentiation—mesenchymal and epithelial—myoepithelial tumors are composed of cells that may be spindle, stellate, epithelioid, or plasmacytoid and are arranged in solid, reticular, and plexiform patterns with a myxoid, chondroid, or collagenous stroma.[10]

Genetic studies have shown that myoepithelial cells containing tumors harbor a variety of genetic abnor-

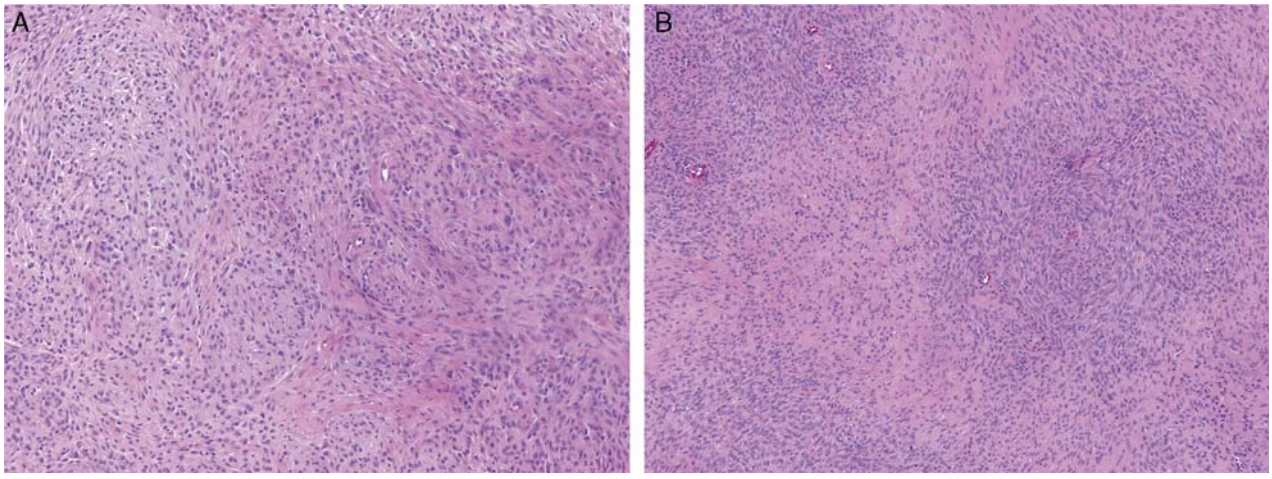

FIGURE 7. A and B, The plump spindle-shaped tumor cells are arranged in intersecting fascicles.

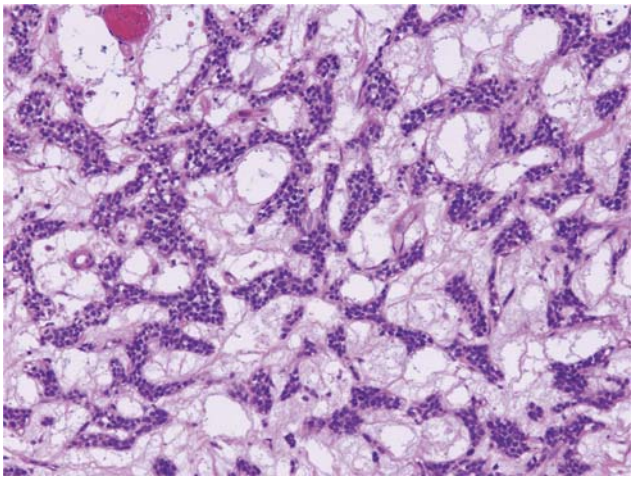

FIGURE 8. The tumor involving the posterior element of L1 (case 8) is composed of aggregates of small round and oval cells arranged in cords and nests in a myxoid stroma.

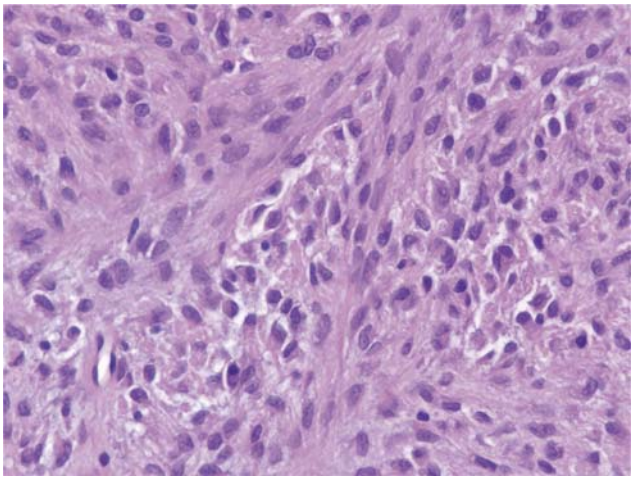

FIGURE 10. In some tumors the spindle cells intermingle with epithelioid and plasmacytoid cells.

malities. Pleomorphic adenomas of the salivary gland, for example, are characterized by the deregulated expression of *PLAG1* or *HMGA2*.[28–30] Although some soft tissue myoepitheliomas may demonstrate *PLAG1* rearrangement,[31] many soft tissue myoepitheliomas and mixed tumors of the skin have an *EWSR1* gene rearrangement, which is not present in mixed tumors of salivary glands.[12,27,32–34]

Myoepithelioma of bone is rare; 9 cases have been reported in the literature (Table 3).[16,18,20,27] Combining our cases with those previously described, men and women are affected at a ratio 5:4, and the age range is 14 to 55 (mean, 32) years.

Clinically, patients present with painful or asymptomatic masses that range in size from 3 to 12 cm. Roentgenograms have demonstrated that benign intraosseous myoepitheliomas are characteristically radiolucent with well-defined margins. Malignant tumors have been described as being multicystic and expansile and may erode the cortex and induce periosteal new bone formation; there is no information regarding their margins.[16,18,20,27]

Computed tomographic scans show well-circumscribed, homogenous, unilocular tumors in the benign ones; 1 locally aggressive tumor was expansile, it eroded the cortex, and did not demonstrate any significant periosteal reaction. One histologically malignant tumor destroyed the cortex and extended into the adjacent soft tissue.[16] The magnetic resonance imaging findings have largely been described in benign tumors, and they are usually hypointense on T1-weighted images and show a heterogenous signal intensity on T2-weighted images.

Overall, it seems that the imaging features of myoepithelioma portray a slowly enlarging tumor that elicits reactive changes in the surrounding bone and may extend into the soft tissues in a minority of cases.

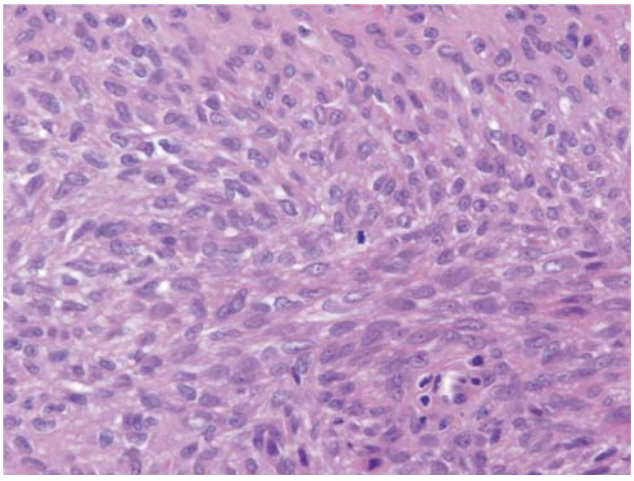

FIGURE 9. The spindle cells have nuclei that contain fine chromatin and small nucleoli. Note the mitotic figure (center).

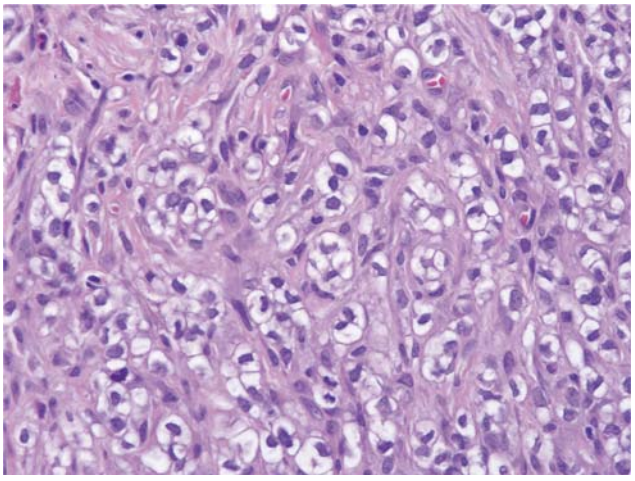

FIGURE 11. Myoepithelioma may contain nests of polyhedral cells with clear cytoplasm.

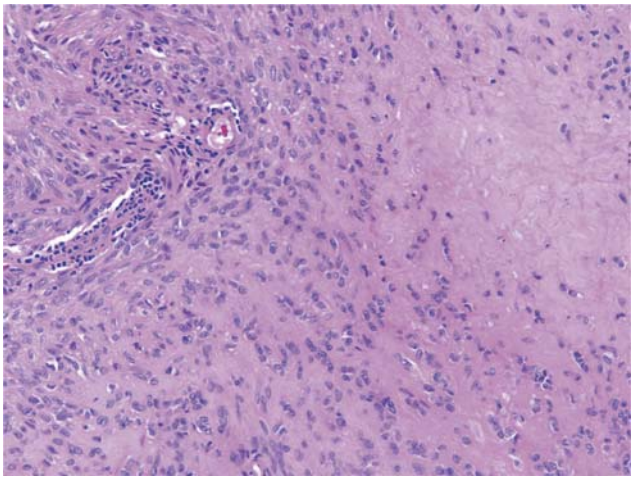

FIGURE 12. Myoepithelioma with focal cell dropout (necrosis).

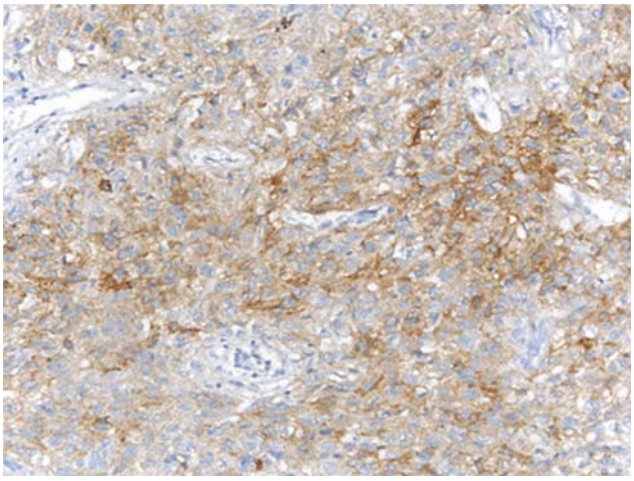

FIGURE 14. Many tumor cells are EMA positive.

Grossly, the tumors have been tan-white in appearance. The benign myoepitheliomas of bone have consisted of epithelioid, plasmacytoid, polygonal, or spindle cells embedded in a stroma described as chondromyxoid or hyalinized. The cells were arranged in nests, cords, strands, or clusters and did not show significant atypia, pleomorphism, or necrosis, and the mitotic rate was < 2 mitoses/10 HPF. The malignant tumor consisted of spindle cells intermingled with epithelioid eosinophilic cells. The cells were mildly pleomorphic with occasional mitoses (< 2 mitoses/10 HPF); however, there were multiple areas of necrosis.[16]

Immunohistochemically, soft tissue myoepitheliomas are usually positive for cytokeratin (93%) (CK AE1/AE3, Pan-K, CK CAM5.2) or EMA (63%) and S100 protein (87%).[13] Myoepitheliomas of bone reported in the literature were all positive for cytokeratin (CK AE1/AE3 or CK MNF116), S100 protein (except for 1 case), GFAP, and SMA (in 2 cases). In contrast, in our study, all of the tumors in our series were negative for keratin, but expressed vimentin, S100 protein, and EMA. We have no explanation for this difference in antigen expression. In the largest study on myoepithelioma of soft tissue by Hornick and Fletcher,[13] all tumors stained for cytokeratins and/or EMA; however, only 68% were positive for Pan-K, 77% for AE1/AE3, 51% for CK8/18, 32% for CK14, and 63% for EMA. In our study all tumors except 1 were positive for EMA, and the only negative tumor was FISH positive; additionally, the one tumor studied cytogenetically showed t(1;22) with *EWSR1-PBX1* fusion—characteristic of myoepithelioma. Accordingly, we feel that the characteristic light microscopic appearance of the tumors and the fact that all were EMA and/or FISH positive support our contention that the tumors are myoepitheliomas.

Molecular analysis of 66 myoepithelial tumors (including 4 myoepitheliomas of bones by Antonescu et al[27]) demonstrated *EWSR1* rearrangement in 45% cases of deep-seated and osseous myoepitheliomas with fusion partners being *PBX1*, *ZNF 444*, or *POU5F1*. The

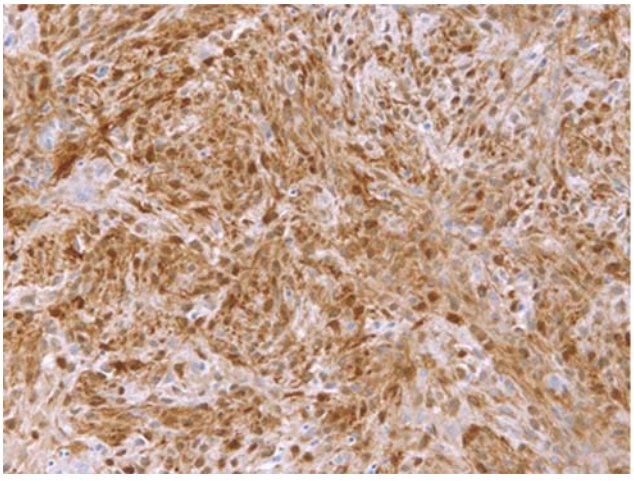

FIGURE 13. Immunohistochemical stain shows diffuse expression of S100 protein by the tumor cells.

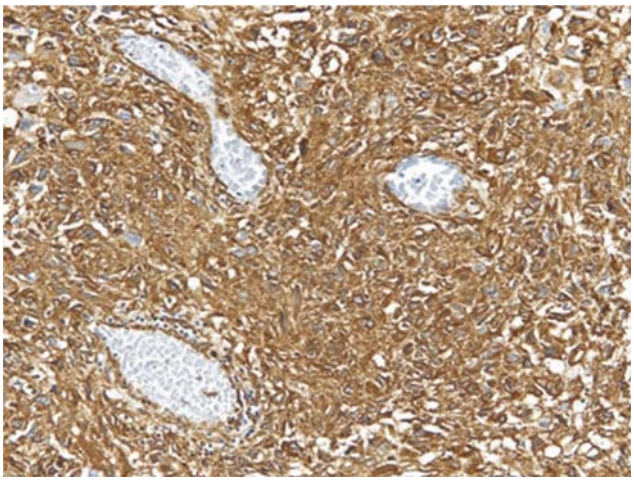

FIGURE 15. Tumor cells are strongly positive for smooth muscle actin.

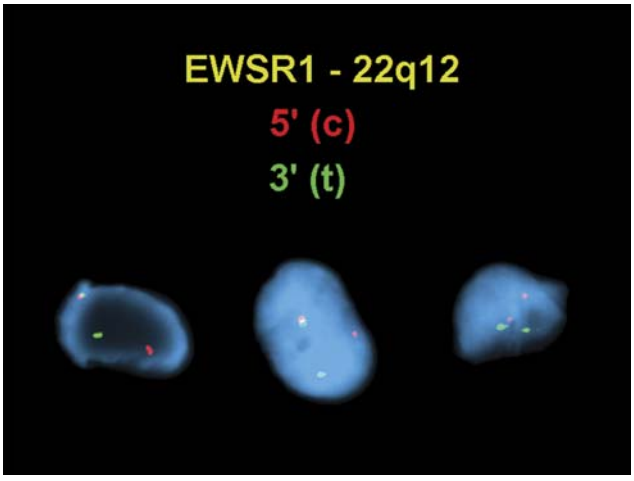

FIGURE 16. Tumor cells with *EWSR1* gene rearrangement identified by FISH utilizing a breakapart signal.

EWSR1-rearranged tumors were composed mainly of sheets of small blue cells with scant cytoplasm and ill-defined cell borders or of epithelioid cells with eccentric eosinophilic or clear cytoplasm or tumors composed of spindle or ovoid cells embedded in sclerotic stroma. The *EWSR1*-negative tumors contained ductal and glandular elements and were located superficially with predilection for the skin and subcutaneous tissue.

In our series, FISH using a breakapart probe for *EWSR1* identified rearrangement in 5 of 7 tumors. Two *EWSR1*-positive tumors were composed of an admixture of spindle, epithelioid, and clear cells. Two tumors were composed predominantly of spindle cells with some epithelioid cells, and 1 tumor was composed predominantly of small round cells. The 2 *EWSR1*-negative tumors were composed predominantly of spindle-shaped cells with a small component of epithelioid cells.

The morphologic features that distinguish benign myoepitheliomas of bone from malignant variants are not well established. In the soft tissues, the features of at least moderate cytologic atypia (prominent nucleoli, coarse chromatin), invasive growth pattern, and high mitotic activity (over 7 mitoses/10 HPF) are considered to be indicative of malignancy.[13]

TABLE 3. Previously Reported Cases of Myoepitheliomas of Bone

References	Age (y)	Sex	Site	Outcome
Ferretti et al[20]	14	F	Palate	NA
	18	M	Palate	18 mo, NED
	14	M	Palate	NA
Alberghini et al[16]	55	M	Femur	Lung metastases
Cuesta et al[18]	54	F	Maxilla	3 y, NED
Antonescu et al[27]	49	M	Iliac bone	NED
	45	F	Vertebral body L1	NED
	16	F	Fibula	NED
	23	M	Humerus	NED

NA indicates not available; NED, no evidence of disease.

Among the cases reported in the literature, 6 patients with intraosseous myoepithelioma did not have any evidence of disease within their follow-up period, which was 16 months to 3 years. The one patient with the tumor containing necrosis developed lung metastases 1 year after surgery, and the follow-up data of 2 patients were not available. Surgical management consisted of tumor resection or curettage. In our cases, 6 tumors were histologically benign, and 2 were atypical in that they demonstrated mild cytologic atypia and a mitotic rate of 1 mitosis/10 HPF. Because clinical follow-up information in our series is limited we cannot be certain of the relationship between morphology and biological behavior, although we suspect that all of our tumors do not have the capacity to disseminate. We believe that tumors that do not show worrisome histologic features can be adequately treated by curettage or limited en bloc resections. However, further studies and longer follow-up is needed in order to adequately evaluate their clinical behavior.

The differential diagnosis of intraosseous myoepithelioma includes a variety of neoplasms such as chondrosarcoma with secondary myxoid changes, extraskeletal myxoid chondrosarcoma, chondromyxoid fibroma, and desmoplastic fibroma.

Unlike myoepithelioma, chondrosarcoma with secondary myxoid changes is composed of round to stellate neoplastic cells having a cord-like growth pattern and enmeshed in pure myxoid stroma. Although chondrosarcomas with secondary myxoid changes are S100 protein positive, they do not stain for epithelial markers and do not demonstrate *EWSR1* rearrangement.

Extraskeletal myxoid chondrosarcoma has rarely been reported to arise within bone.[35] In contrast to extraskeletal myxoid chondrosarcoma, myoepithelioma is usually composed of an admixture of spindle and epithelioid cells, the latter usually more epithelioid when seen in extraskeletal myxoid chondrosarcoma. Occasionally this distinction can, however, be difficult. Both can express S100 protein; however, expression of epithelial and muscle markers are rarely seen in extraskeletal myxoid chondrosarcoma.

Chondromyxoid fibroma has a lobulated growth pattern with the lobules having a hypocellular center with increased cellularity toward the periphery. The cells are stellate or spindle-shaped and are embedded in abundant extracellular myxoid matrix. Although immunohistochemically the cells can express S100 protein, they do not stain for epithelial markers and are negative for chromosome 22 translocations.

Desmoplastic fibroma is composed of fascicles of spindle-shaped cells embedded in collagenous matrix. The cells do not stain for S100 protein or epithelial markers and lack chromosomal abnormalities involving chromosome 22.

In conclusion, primary myoepithelioma of bone is rare. Their morphology is variable, and immunohistochemical and molecular analyses can help distinguish them from tumors that are in the histologic differential diagnosis. Although the experience is limited, histologically

banal tumors appear to behave in a benign manner, and conservative surgical removal appears to be sufficient treatment.

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