Locally Aggressive Fibrous Dysplasia Mimicking Malignancy: A Report of Four Cases and Review of the Literature

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Abstract

Background Fibrous dysplasia is a benign fibroosseous bone tumor that accounts for 5% to 10% of benign bone tumors. It can present as monostotic fibrous dysplasia (70% to 80%), polyostotic fibrous dysplasia (20% to 30%), McCune-Albright syndrome (2% to 3%), or Mazabraud's syndrome in rare cases. Bone lesions in fibrous dysplasia arise in the medullary canal and usually are confined to the bone. Cortical destruction and extension into soft tissue usually indicates malignant transformation or secondary aneurysmal bone cyst formation. Locally aggressive fibrous dysplasia with cortical destruction and extension into soft tissue in the absence of

these two possibilities is extremely rare. It is important for the treating physician to distinguish this entity from more aggressive or malignant tumors to avoid overtreating the patient for a benign condition or inattention to a malignant tumor.

Case Descriptions We report four unusual cases of fibrous dysplasia with an aggressive radiographic appearance. They occurred in the rib (1), ilium (2), and distal femur (1). Two patients had pain and two had swelling. Radiologically, all were associated with cortical destruction and an associated soft tissue mass, and initially they were interpreted as potentially malignant. Three patients underwent biopsy and one patient did not have a biopsy. Histopathologic analysis by an experienced bone pathologist confirmed fibrous dysplasia in all patients. Two patients were treated surgically; one patient with zoledronic acid and one patient currently is being followed by observation alone.

Literature Review There are only a few reports in the literature that describe the locally aggressive variant of fibrous dysplasia that presents with pain and progressive swelling clinically and with cortical destruction and soft tissue extension on imaging which suggest malignancy. We could not find any article that describes the use of bisphosphonates in such lesions or the response to bisphosphonates clinically, on laboratory parameters or imaging. To our knowledge, this is the largest case report published regarding locally aggressive fibrous dysplasia arising outside the craniofacial skeleton.

Clinical Relevance The locally aggressive variant of fibrous dysplasia may be confused with a malignant tumor or malignant degeneration of fibrous dysplasia. It is important to properly evaluate these lesions to ensure that a proper diagnosis is made, especially with respect to a malignant versus benign mass.

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Each author certifies that his or her institution approved the reporting of this case report, that all investigations were conducted in conformity with ethical principles of research, and that informed consent for participation in the study was obtained.

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Introduction

Fibrous dysplasia is a benign fibroosseous tumor in which fibrous tissue with metaplastic immature woven bone replaces normal lamellar bone. It accounts for 5% to 10% of all benign bone tumors. Fibrous dysplasia can present as monostotic fibrous dysplasia (70% to 80%), polyostotic fibrous dysplasia (20% to 30%), McCune-Albright syndrome (2% to 3%), or as Mazabraud's syndrome (fibrous dysplasia of bone with associated intramuscular myxoma). The lesions in fibrous dysplasia arise in the medullary canal in the metaphyseal and diaphyseal regions (rarely the epiphysis) and usually are confined to bone. They sometimes cause cortical erosion, endosteal scalloping, bone expansion, and thinning of the cortex. Sudden appearance of pain associated with swelling in a patient with fibrous dysplasia usually indicates either pathologic fracture or in rare instances malignant transformation [11]. Radiographically, malignant transformation is associated with cortical destruction and an adjacent soft tissue mass [2]. These features also are observed in primary bone sarcomas and metastatic lesions. In a patient with fibrous dysplasia, the presence of cortical destruction and a soft tissue mass usually indicates either malignant transformation or secondary aneurysmal bone cyst formation [9, 21].

Locally aggressive fibrous dysplasia with cortical destruction and extension into soft tissue in the absence of these two possibilities is extremely rare. There are few published reports systematically describing the clinical and radiologic features of locally aggressive fibrous dysplasia (Table 1). Such lesions are extremely rare and create a diagnostic dilemma. It is important therefore, for all physicians treating patients with fibrous dysplasia to be aware of this variant and its clinical and radiologic presentation, and to be able to differentiate it from a malignant tumor to

avoid overtreating a benign condition or inattention to a malignant tumor.

We report the cases of four patients with locally aggressive fibrous dysplasia who originally were thought to have malignant bone tumors owing to clinical and imaging features, but careful histologic review revealed no evidence of malignancy. We highlight the clinical and imaging features in four cases and review the literature to emphasize the importance of clinical, pathologic, and radiologic correlation in determining the correct diagnosis.

Case Reports

Patient 1

A 62-year-old woman presented with radiating pain and tenderness, most severe over the left ilium without swelling. Radiographs showed an expansile radiolucent lesion in the left ilium. The initial CT scan and MR images of the pelvis showed a geographic radiolucent lesion of the left ilium without a soft tissue mass. An image-guided core biopsy revealed fibrous dysplasia. Treatment with tramadol followed by three doses of intravenous pamidronate resulted in substantial pain relief. The lesion was stable clinically and on imaging. After 1½ years, the pain and tenderness spontaneously recurred. New radiographs showed a larger, expansile radiolucent lesion with an ill-defined cortical outline (Fig. 1A). The new MR images showed progression of disease with cortical destruction and an associated soft tissue mass medially. The soft tissue mass also contained a rim-enhancing, predominantly cystic component (Fig. 1B–C). Multiple cores obtained by CT-guided biopsy were examined by an experienced bone pathologist (AR) who confirmed fibrous dysplasia with no malignant features (Fig. 1D). Treatment with tramadol offered partial pain relief but her

Table 1. Reported cases of locally aggressive fibrous dysplasia outside the craniofacial skeleton

Study	Demographics age (years), sex	Location	Clinical presentation	Treatment
Latham et al. [9]	26, Female	Proximal humerus	Pain, soft tissue mass, paresthesia	Excision and prosthetic reconstruction
Yao et al. [21] (3 cases)	23, Male	Distal femur;	Pain, soft tissue mass	Curettage and bone grafting
	38, Female	Distal humerus;		
	47, Male	Distal humerus		
Dorfman et al. [3] (2 cases)	18, Male	Rib;	Swelling	Not known
	33, Male	Proximal tibia		
Hermann & Garcia [7]	56, Male	Rib	Pain, swelling	Not known
Kashima et al. [8] (3 cases)	60, Male	Rib	Not known	Not known
	72, Female	Rib		
Zídková et al. [22]	Not known	Pelvis	Not known	Not known

Fig. 1A–D (A) An AP radiograph of the pelvis shows the expansile radiolucent lesion involving the superior part of left ilium with cortical destruction medially and a thin rim of cortex remaining superiorly (white arrow). (B) The coronal short tau inversion recovery MR image shows a heterogenous lesion with medial soft tissue extension (white arrow). The homogenous hyperintensity in the lower

part suggests cystic change. (C) An axial T1 postcontrast fat-suppressed MR image shows diffuse enhancement of the soft tissue mass (white arrow). The cyst seen in the coronal image did not enhance. (D) Histologic analysis of the biopsy specimen reveals typical irregular woven bone trabeculae without malignant features (Stain, hematoxylin and eosin; Original magnification, $\times 60$).

pain subsided spontaneously after biopsy and therefore she was not considered for surgery or bisphosphonate treatment. She continued to have a stable lesion with serial clinical and radiographic followups for 20 months. Serial urine N-telopeptide levels have been stable between 75 and 85 nmol NTX/mmol creatinine. We would consider bisphosphonate therapy again if she becomes symptomatic.

Patient 2

A 70-year-old woman was referred with an incidental lesion in the left ilium discovered during bone scintigraphy for a painful stiff knee arthroplasty. There was increased radiotracer uptake in the left ilium that corresponded to an ill-defined radiolucent lesion on radiograph (Fig. 2A). The patient had no pain or tenderness but did have an area of fullness and a palpable mass. MR images revealed an expansile lesion involving the superior left ilium with an associated extraosseous soft tissue mass medially (Fig. 2B). CT scans showed a mineralized matrix reminiscent of cartilage with a periosteal shell that was focally disrupted (Fig. 2C–D). Given these findings, particularly the

extraosseous soft tissue mass and mineralized matrix, chondrosarcoma was suspected. Considering the heterogenous nature of chondrosarcoma and the relative ease of surgical resection, primary surgical excision without biopsy was planned to avoid sampling error and soft tissue contamination. The patient underwent a partial Type I pelvic resection. The pathology report confirmed fibrous dysplasia with focal secondary aneurysmal bone cyst formation, but no malignant features. No local recurrence has been seen with close surveillance clinically and with radiographs for 4½ years.

Patient 3

A 65-year-old man with McCune-Albright syndrome, originally diagnosed when he was 31 years old, was referred with a 4-year history of an enlarging 5th rib lesion, initially found on routine surveillance CT. Since the original diagnosis of McCune-Albright syndrome, he had been asymptomatic and the fibrous dysplasia had been stable on serial CT and bone scans. A chest radiograph was

Fig. 2A–D (A) An AP radiograph of the pelvis shows an expansile radiolucent lesion with cortical destruction extending superiorly (white arrow). (B) A T1-weighted sagittal MR image shows the soft tissue extension of the lesion arising from the superior part of the left

ilium (white arrow). (C) An axial CT image shows the expansile lesion and thinned out cortex with areas of complete destruction on the lateral cortex, and an (D) axial image at a different level shows matrix calcification in the lesion.

normal. New MR images showed an expansile radiolucent lesion of the left 5th rib with cortical destruction and an associated intrathoracic extrapleural soft tissue mass that enhanced diffusely with gadolinium (Fig. 3A). New CT scans revealed mineralized matrix in the soft tissue mass (Fig. 3B–C). Bone scintigraphy revealed intense radiotracer uptake in this lesion, and in multiple other osseous lesions (Fig. 3D). A CT-guided needle biopsy of the 5th rib lesion confirmed fibrous dysplasia. Considering the size, interval growth, proximity to the heart and great vessels, and possibility of a sampling error on biopsy, he underwent wide resection of the 5th rib including parts of the 4th and 6th ribs and reconstruction with Prolene[®] mesh (Somerville, NJ, USA) and methylmethacrylate. The final pathology analysis confirmed fibrous dysplasia, with no evidence of malignancy (Fig. 3E). At 3½ years of followup, there has been no recurrence.

Patient 4

A 41-year-old woman was referred with an aggressive-appearing lesion in the left distal femur. A diagnosis of McCune-Albright syndrome was made when she was 34 years old. She had been treated with pamidronate, risedronate, and zoledronic acid before presentation and had

been monitored by biochemical markers and serial MRI for 6 years. There was no disease progression after that and the therapy was stopped. Although she was asymptomatic for 3 years after cessation of therapy, severe generalized pain and tenderness in multiple bones developed spontaneously and she presented for further evaluation and treatment. She presented with pain, tenderness, and swelling in the left lower thigh. Radiographs revealed an expansile radiolucent lesion with posterior cortical thinning (Fig. 4A–B). New MR images revealed an enlarged, heterogeneous and predominantly hyperintense signal marrow-replacement abnormality on T2 pulse-weighted sequences in the distal ⅓ of the femur. There was cortical destruction and an extraosseous soft tissue mass posteriorly (Fig. 4C–E). A biopsy confirmed fibrous dysplasia and no malignant features (Fig. 4F). Bone scintigraphy revealed increased radiotracer uptake (Fig. 4G) in multiple lesions that was similar to previous examinations, and PET imaging showed increased 2-[18F] fluoro-2-deoxy-D-glucose (FDG) avidity. She was treated with seven infusions of zoledronic acid, 4 mg every 6 weeks. She had complete resolution of pain from 9–10/10, erythema and warmth and a significant reduction in soft tissue swelling. Serum C terminal telopeptide decreased from 725 pg/mL to normal. Bone-specific alkaline phosphatase decreased from 163 to 65 mcg/L. Pyridinoline cross-links and N-telopeptide became normal. The lesion became stable on serial

Fig. 3A–E (A) A coronal contrast-enhanced T1-weighted MR image reveals a hyperintense heterogenous mass extending into the chest from the chest wall. (B) A sagittal CT image shows destruction of the cortex and the soft tissue extension of the rib lesion. (C) An axial CT image shows complete destruction of the cortex medially with a ground glass appearance of the matrix. (D) The bone scan shows

multiple sites of uptake in this patient with McCune-Albright syndrome with the most intense uptake in the rib lesion. (E) Histologic analysis of the excised specimen shows the characteristic appearance of fibrous dysplasia without malignant features (Stain, hematoxylin and eosin; original magnification, $\times 60$).

radiographs, repeat bone scan, MRI, positron emission tomography and CT. Her intermittent pain is being treated with naproxen with good clinical response. The fibrous dysplasia lesions have been stable on routine surveillance for more than 9 years.

Discussion

The bone lesions in patients with typical fibrous dysplasia arise in the medullary canal and rarely extend outside the bone. They sometimes cause endosteal scalloping, bone

Fig. 4A–G (A) An AP radiograph of the left distal femur shows an expansile lesion and cortical thinning with ground glass matrix. There also is involvement of the tibia in this patient with McCune-Albright syndrome. (B) The lateral view shows extension in the soft tissue. (C) A coronal T1-weighted fat-suppressed postcontrast image shows a heterogenous hyperintense expansile mass with diffuse enhancement throughout. (D) The axial T2-weighted fat suppressed image shows complete destruction of the posterior cortex and soft tissue extension mimicking a malignant lesion. (E) A sagittal T1-weighted fat suppressed postcontrast image shows the posterior soft tissue mass with contrast enhancement. (F) Histologic analysis of the biopsy specimen shows the characteristic fibrous dysplasia with irregular osseous trabeculae in a bland fibrous background that lacks osteoblastic rimming (Stain, hematoxylin and eosin; original magnification, $\times 40$). (G) A bone scan revealed multiple sites of increased uptake in this patient with McCune-Albright syndrome with the most intense uptake in the left distal femur.

Fig. 4A–G continued

expansion, cortical erosion, and cortical thinning. Rarely these lesions may present as an exophytic growth arising in the bone without medullary involvement which has been described as an exophytic variant of fibrous dysplasia or fibrous dysplasia protuberans [6, 16, 18, 20]. In contrast, the locally aggressive fibrous dysplasia variant exhibits cortical destruction and tumor extension into the adjacent soft tissue (Table 1). It is important that the clinician be familiar with this variant to avoid missing a malignancy or overtreating a benign condition, a situation that occurred in two patients in our series.

The incidence of locally aggressive fibrous dysplasia is not known. Numerous of the previously published reports of locally aggressive fibrous dysplasia described occurrence in the craniofacial skeleton, especially the maxilla and mandible in young patients [4, 5, 8, 10, 12, 14, 17]. Outside this location, locally aggressive fibrous dysplasia has been reported in the proximal and distal humerus, rib, pelvis, distal femur, and proximal tibia (Table 1), mostly in skeletally mature patients. There is no gender predominance with this disease [3, 7–9, 21, 22]. Most of the reported cases of locally aggressive fibrous dysplasia have been associated with a previously diagnosed fibrous dysplasia lesion [3, 7–9, 21, 22]. Typically, fibrous dysplasia occurs sporadically and is associated with a missense mutation in the *GNAS1* gene on chromosome 20 [15]. It usually becomes inactive after skeletal maturity but with locally aggressive fibrous dysplasia, what causes the lesion to grow after skeletal maturity and develop aggressive features (cortical destruction and soft tissue mass) is not known and the etiology remains unclear.

The pathologic features of locally aggressive fibrous dysplasia are identical to those of fibrous dysplasia. On gross examination, the lesion in locally aggressive fibrous dysplasia appears tan-white with associated cortical destruction and a soft tissue mass. Histologically, all the reported cases of locally aggressive fibrous dysplasia have typical fibrous dysplasia histologic features showing irregular osseous trabeculae of purposeless woven bone spicules in a bland fibrous background that lacks osteoblastic rimming [1]. Neither nuclear atypia nor mitotic activity have been reported with locally aggressive fibrous dysplasia. Based on some reports [3, 7–9, 21, 22], that include patients with asymptomatic lesions found incidentally on imaging done for other reasons and others who present with pain and/or a mass, the clinical features of locally aggressive fibrous dysplasia are variable. For patients with fibrous dysplasia, sudden appearance of pain or swelling is highly suggestive of pathologic fracture or malignant transformation. Locally aggressive fibrous dysplasia also has been associated with deformity or chronic pressure symptoms in the craniofacial region [14, 17].

Laboratory studies do not help differentiate locally aggressive fibrous dysplasia from typical fibrous dysplasia.

However, as in fibrous dysplasia, the disease activity, extent, and response to treatment could be monitored with serum and urine markers like alkaline phosphatase, osteocalcin, N-telopeptide of collagen, deoxypyridinoline cross links, and hydroxyproline.

Radiologically, locally aggressive fibrous dysplasia appears as expansile lytic lesions with ground glass matrix and coarse internal septations. The lesion extends outside the boundary of the native bone and may appear as an exophytic growth. Cortical destruction and a soft tissue mass with mineralization may be visible on radiographs but they are better seen with CT and MRI. In contrast to typical fibrous dysplasia where radiographs often are diagnostic, the presence of cortical destruction and a soft tissue mass in locally aggressive fibrous dysplasia may lead to diagnostic confusion. Patients with a bone lesion who present with cortical destruction and a soft tissue mass are considered to have a malignant diagnosis unless proven otherwise by histologic analysis. In a patient with a preexisting fibrous dysplasia lesion, cortical destruction and a soft tissue mass often suggest either malignant transformation or secondary aneurysmal bone cyst formation. In such cases, advanced imaging is usually necessary.

The MR appearance of locally aggressive fibrous dysplasia is variable. The lesion is heterogenous; hypointense on T1 and hyperintense or, in some lesions, hypointense on T2 images. The degree and type of contrast enhancement are variable. The MR images may show cortical destruction but MRI is the most sensitive in detecting the soft tissue extension. Unlike conventional MRI, gadolinium-enhanced dynamic subtraction MRI may be helpful in differentiating malignant tumors from benign and inflammatory lesions [17]. Overall, the MRI features are nonspecific and cannot differentiate malignancy from locally aggressive fibrous dysplasia. CT scans of the lesions show an expansile lesion with ground glass appearance and matrix calcification. CT is superior to MRI in evaluating endosteal scalloping, matrix calcification, cortical thinning, presence of a cortical shell, and cortical destruction. The gadolinium-enhanced subtraction MRI and CT may be helpful in surgical planning and for followup [17].

A bone scan sometimes is helpful to look for multicentricity if that is suspected, but neither bone scan nor positron emission tomography CT is helpful in distinguishing locally aggressive fibrous dysplasia from a malignant neoplasm. Therefore, unlike the typical fibrous dysplasia where a biopsy is seldom necessary, a biopsy is necessary to distinguish locally aggressive fibrous dysplasia from other neoplasms. The most important indication for a biopsy is to exclude primary sarcoma, metastatic carcinoma, and malignant transformation in a preexisting fibrous dysplasia lesion and to differentiate secondary aneurysmal bone cyst formation.

The radiologic differential diagnosis of locally aggressive fibrous dysplasia is broad and includes benign aggressive and malignant lesions. In a preexisting fibrous dysplasia lesion, the most important differentials include malignant transformation or secondary aneurysmal bone cyst formation. Owing to considerable overlap in imaging features of locally aggressive fibrous dysplasia and fibrous dysplasia with malignant transformation, a biopsy is necessary for definitive diagnosis when atypical features are present. The most important pathologic differential diagnosis is the low-grade, central (intramedullary) osteosarcoma which is strikingly similar to locally aggressive fibrous dysplasia [8, 13, 19]. Low-grade central osteosarcoma is a rare intramedullary well-differentiated tumor with clinical, imaging, and histologic features similar to those of fibrous dysplasia. The presence of nuclear atypia, hypercellularity, and the absence of a typical woven bone pattern observed on microscopy and positive immunohistochemistry for CDK4, MDM2, and GNAS mutations indicate a low-grade intramedullary osteosarcoma. Absence of prominent osteoblastic rimming of woven bone is characteristic of locally aggressive fibrous dysplasia.

The ideal treatment of locally aggressive fibrous dysplasia is not known because of the limited number of cases reported. The treatment options include observation for stable asymptomatic lesions, pain medications (NSAIDs/opioids), and/or bisphosphonates for symptomatic lesions [10, 16]. Pain medications offered incomplete relief but bisphosphonates offered better pain relief and halted disease progression in our patients. Impending pathologic fractures may warrant prophylactic curetting, bone grafting, and internal fixation. The aggressive and destructive lesions may require resection and reconstruction to control pain and improve function. The clinical course of locally aggressive fibrous dysplasia is unpredictable. Some lesions may become stable with or without medical treatment whereas others may progress despite medical therapy [17]. Schofield [14] reported local recurrence after surgical treatment of a patient with locally aggressive fibrous dysplasia but distant metastasis or malignant transformation of locally aggressive fibrous dysplasia has not been reported. Since the natural history of locally aggressive fibrous dysplasia is unknown, it is prudent to follow these patients indefinitely with clinical examinations and serial imaging.

We described a rare, more aggressive variant of fibrous dysplasia that presents clinically with pain and swelling and with cortical destruction, and soft tissue extension on imaging. It mimics malignant transformation of fibrous dysplasia, primary bone sarcomas or metastatic lesions, but histologically it is benign and identical to typical fibrous dysplasia. These lesions require careful evaluation by an experienced team of clinicians, radiologists, and pathologists to ensure that they are not overtreated as a malignancy.

or misdiagnosed as an aneurysmal degeneration of fibrous dysplasia. The exact behavior of this variant in the long term remains uncertain.

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