Chondromyxoid Fibroma of the Radial Shaft Treated With Nonvascularized Fibular Autograft

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Abstract

Chondromyxoid fibroma is a benign yet highly recurrent neoplasm of bone, usually found in the metaphyseal segments of long bones. We present the case of an unusual diaphyseal chondromyxoid fibroma of the radius and review the literature regarding these rare chondroid neoplasms.

Chondromyxoid fibroma (CMF) is an extremely rare, benign lesion of bone, representing less than 1% of all benign and malignant tumors of bone.[1,2] Predominantly a lesion of adolescence and young adulthood, CMF has cartilaginous origins but prominent features of both myxomatous and fibrous tissues.[3] It has a strong tendency to occur in the metaphyseal area of long bones and a predilection for the lower extremities, particularly the proximal tibia.[3] Although CMF has been described in almost every bone in the body,[1,3] its presence in the diaphyseal region of the long bones of the upper extremity is exceedingly rare. Some 95% of CMFs in long bones are found in the metaphyseal region.[2] To our knowledge, up until now this lesion has yet to be described in the diaphysis of the humerus or radius. Here we report the case of a CMF presenting in the radial diaphysis and treated with wide resection and bridging autologous fibula grafting.

Case Report

A right-hand–dominant man in his early 40s presented for evaluation of left forearm pain 2 days after sustaining minor trauma in a fall. He indicated he had had intermittent pain and swelling in the forearm for about 1 year preceding the fall but had not had the forearm evaluated by a physician. He had no history of trauma to the extremity before the fall.

Physical examination (gross inspection) revealed a slight prominence on the left arm. The patient had full range of motion (ROM) to flexion and extension at the elbow and the wrist. The only discomforting motions were full supination and pronation, as well as resisted flexion of the elbow. Neurovascular assessment and the rest of the musculoskeletal examination were unremarkable, and there were no skin changes. The patient indicated no family history of neoplastic disorders or any recent constitutional symptoms.

Baseline laboratory studies were obtained during the initial phase of evaluation. Hemoglobin, hematocrit, and white blood cell count were normal. C-reactive protein was marginally elevated (2.21 mg/dL; normal, <0.8 mg/dL), and erythrocyte sedimentation rate was 17 mm/h (normal, <20 mm/h).

Radiographs at presentation showed a well-circumscribed, geographic, expansile lesion in the midshaft portion of the radius (Figure 1). There was evidence of cortical thinning with disruption consistent with either a pathologic fracture or a cortical breakthrough of the lesion. No discernable matrix was present within the lesion, and there seemed to be no pronounced periosteal reaction. The lesion appeared to be centrally based, without extension beyond a well-defined sclerotic rim of bone.

The patient was immobilized in a sugar-tong splint for comfort during additional imaging studies—a chest radiograph and computed tomography (CT) scans of the lesion and chest.

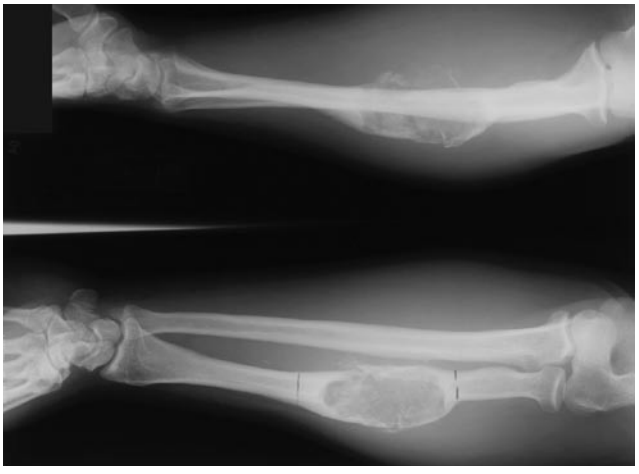

Figure 1. Anteroposterior and lateral radiographs of lesion at initial presentation. Note expansile nature and sclerotic rim of bone at proximal and distal extent of lesion.

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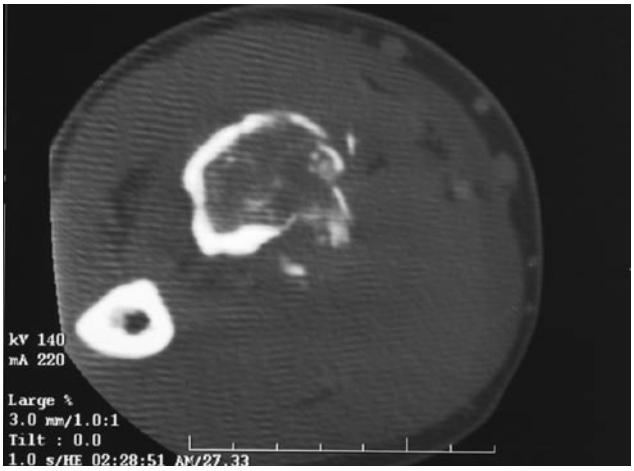

Figure 2. Computed tomography scan of lesion with intralesional calcification. Cortical disruption is apparent.

A magnetic resonance imaging (MRI) study of the lesion was obtained to evaluate the overlying soft tissues. The CT scan of the chest was normal; the CT scan of the lesion (Figure 2) showed intralesional calcification and an area of cortical disruption; and the MRI scan showed a mildly heterogenous signal with no soft-tissue extension of surrounding edema.

An incisional biopsy was performed to obtain tissue for pathologic evaluation. Meticulous hemostasis was achieved, obviating the need for drain placement. An open biopsy technique was used to minimize the likelihood of an incorrect diagnosis secondary to limited sample size. Pathologically, the biopsy tissue sent was found to be CMF. Tissue sent for culture was negative. Because of the high risk for local recurrence associated with intralesional curettage and bone grafting, we proceeded with wide resection of the lesion and structural fibular autografting.

The entire length of the radius was exposed through an anterior approach,[4] and the lesion was exposed with a cuff of normal tissue at its periphery. The lesion, though expansile, did not invade the surrounding tissue layers and was resected en bloc with wide margins. Lesion dimensions were 10×2×3 cm (inclusive of 0.5 cm of normal cortical bone at either end of the lesion); the lesion encompassed a large portion of the central third of the radial shaft.

The lesion had lobulated areas—hypocellular, myxoid lobules interspersed with areas that were more hypercellular. The hypocellular areas contained abundant myxoid matrices punctuated by stellate cells with indistinct cytoplasmic borders, and the hypercellular areas contained angulated cells in a predominantly chondroid matrix. Occasional giant cells were identified in the hypercellular areas. Representative microphotographs (Figures 3, 4) show the pathology. The preceding changes were seen mostly toward the periphery of the overall lesion; the center of the lesion consisted predominantly of aneurysmal bone cyst (ABC)–like changes, as well as fibrosis. Hemosiderin-laden macrophages were found adjacent to the ABC-like areas, suggesting remote hemorrhage. The surrounding lamellar bone showed changes of bone regeneration. The lesion did not penetrate the cortex; all inked margins were negative for tumor.

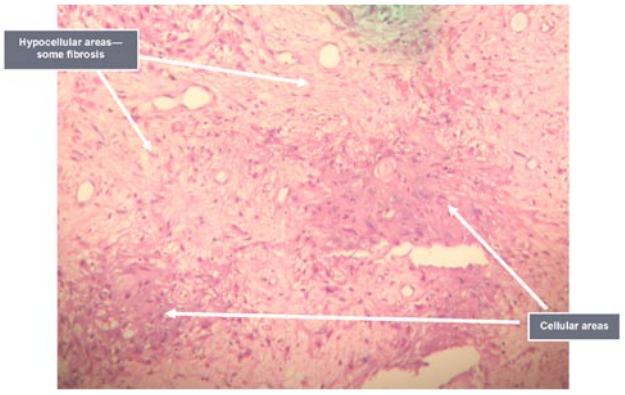

Figure 3. Hypercellular areas contain angulated cells in a predominantly chondroid matrix; occasional giant cells are identified in hypercellular areas (hematoxylin-eosin, original magnification ×400).

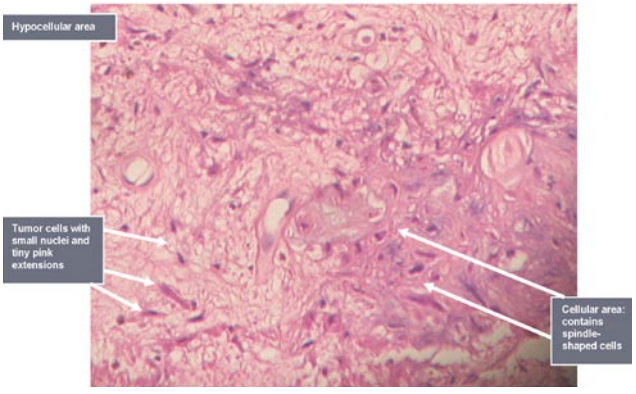

Figure 4. Hypocellular areas contain abundant myxoid matrix punctuated by stellate cells with indistinct cytoplasmic borders (hematoxylin-eosin, original magnification ×400).

Two of the authors harvested the midportion of the right fibula along with a nutrient vessel for the purpose of microvascular anastomosis, using a second set of instruments, as the lesion was excised from the arm. The autograft was positioned to bridge the cortical gap in the radius and secured with a 3.2-mm LCP Locking Compression Plate (Synthes, West Chester, Pa). Tight cortical apposition, forearm rotation, and appropriate radiocarpal orientation and alignment were confirmed with fluoroscopic imaging.

After fixation was achieved, the vessel dissected as the nutrient vessel of the fibula to be anastomosed with a branch of the radial artery was found to be of insufficient length to proceed with the vascularization portion of the grafting procedure. We decided to proceed with nonvascularized fibular autografting. The procedure ended with wound irrigation and layered closure.

In the immediate postoperative period, the patient was immobilized in a sugar-tong splint. At initial follow-up, he was switched to a Munster-style cast to limit forearm rotation. Three weeks later, a short-arm cast was applied, and it was maintained for another 6 weeks. At just more than 3 months after surgery, a radiograph showed evidence of bridging callus at the proximal and distal osteotomy sites.

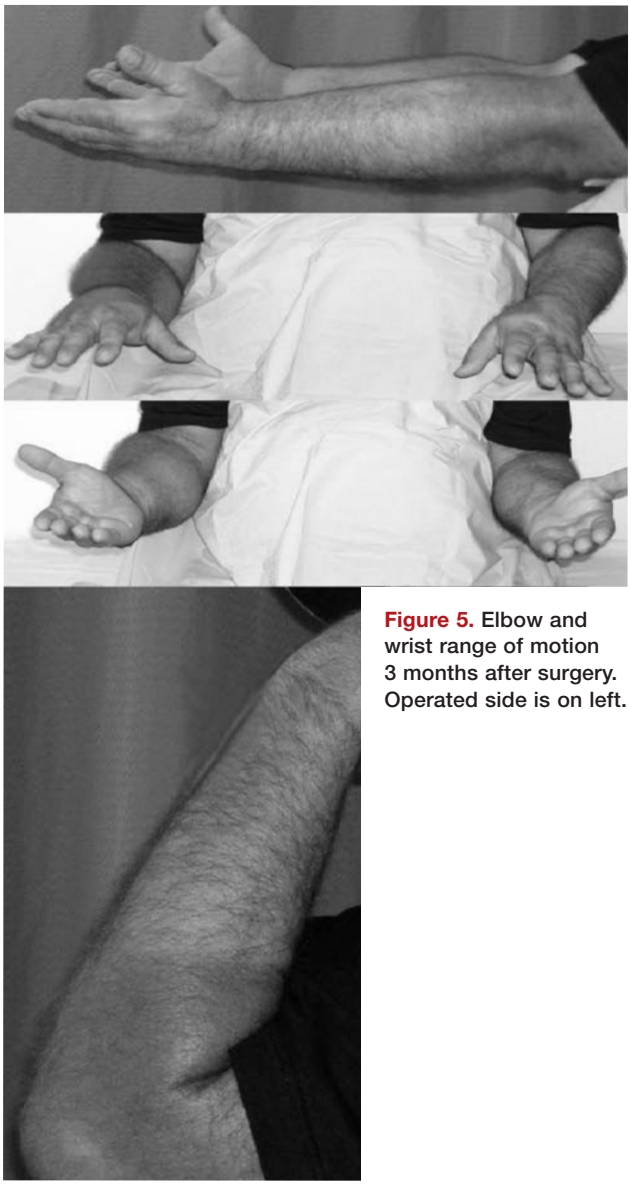

Figure 5. Elbow and wrist range of motion 3 months after surgery. Operated side is on left.

Three months after surgery, the patient had excellent ROM, without pain at the osteotomy site (Figure 5), from full extension to 115° of flexion. He lacked 10° to 15° of terminal supination and pronation and had occasional numbness along the dorsal radial border of the wrist and into the thenar eminence. He was allowed to proceed with a cock-up wrist splint at this point, with restrictions against heavy lifting.

By 7 months after surgery, the patient had advanced with occupational therapy to full ROM of the forearm and had returned to work. A follow-up radiograph showed osseous bridging at both osteotomy sites with graft incorporation. At most recent follow-up, 12 months after surgery, the patient showed no radiographic evidence of recurrence and had excellent osseous bridging of osteotomy sites (Figure 6).

Discussion

CMF was first described as a clinical entity by Jaffe and Lichtenstein[5] in 1948. In their series, they reviewed cases previously encountered while investigating chondrosarcomas. The

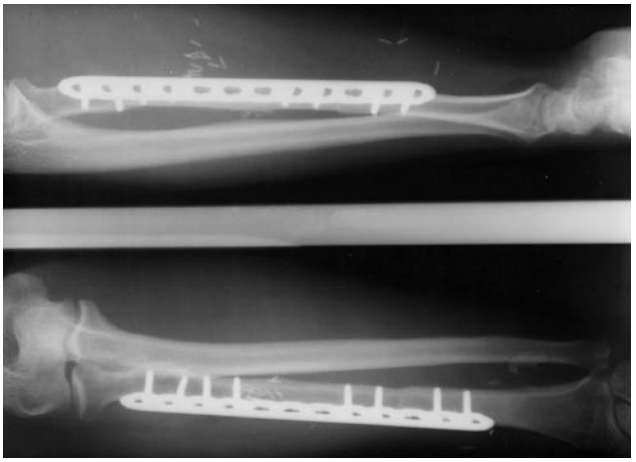

Figure 6. Anteroposterior and lateral radiographs 12 months after fibula autograft reconstruction. Note cortical bridging at both osteotomy sites and maintenance of radial length.

usual clinical presentation was that of mild, intermittent pain over several months. Although their numbers were small (8 patients), their clinical description of the lesion and its key features was remarkably accurate and remains largely true today.

CMF predominantly affects young people, usually those in the second or third decade of life.[1-3,6,7] The male-to-female ratio is roughly 1.5 to 1. CMF is the least common benign tumor derived from cartilage.[8-10] It has been well described that the lesion has a predilection for the long bones, particularly those of the lower extremity, with the proximal tibia and distal femur having the highest incidence rates.[10,11] There have been a significant number of CMFs occurring in the small bones of the hands and feet and in the axial skeleton, the spine, and the craniofacial osseous structures.

As noted, the anatomical location of the lesion is most commonly the lower extremity, particularly the proximal end of the tibia.[1,3,4] Less common anatomical sites are the sacrum,[12] the thoracic or lumbar spine,[13] the craniofacial bones,[14] and the apophysis of long bones in skeletally immature people.[15] Adler[8] reported a CMF associated with an ABC of the metadiaphyseal region of the radius. Although hypervascularity is not a common feature of CMF,[13] ABCs can be engrafted on primary CMFs.[16] Wilson and colleagues[11] evaluated the radiographic features of 38 CMFs, only 1 of which was confined solely to the diaphysis of a long bone. In their review of the literature (356 reported CMFs up until then), only 9 lesions in series with at least 20 cases occurred in the radius. On the basis of our review of the series reported, none has specifically documented a case within the diaphyseal portion of the radius.

Radiologically, the overlying cortex is thinned but is usually intact, and there is no periosteal reaction.[8,11] The tumor generally conforms to the affected bone, is ovoid, and tends not to be invasive[6]; is eccentric in location and has a well-defined sclerotic margin[10]; and is in a metaphyseal location.[7] Marin and colleagues[10] reported on MRI findings that CMFs are heterogeneous depending on the degree of vascularity of the fibrous areas and presence of cystic areas within the lesion.

Diagnostic Considerations

The differential diagnosis of CMF is broad and varied: chondroblastoma, unicameral bone cyst, enchondroma, ABC, giant cell tumor (GCT), and chondrosarcoma. Because of the expansile nature of the lesion, the calcified matrix, and cortical destruction, chondrosarcoma was highest on our list of differential diagnoses. Certain clinical and radiographic variables distinguish CMF from the other lesions mentioned, but pathologic confirmation is vital in making the definitive diagnosis.

An enchondroma is an intramedullary lesion with a calcified matrix that does not typically expand the cortex and histologically consists of calcified hyaline cartilage.[17] Unicameral bone cysts are typically not seen after skeletal maturity; they yield a pure fluid signal on MRI and usually do not expand the cortex beyond the width of the adjacent physis. ABCs typically occur in people younger than 20 and cause pain and swelling[18]; by virtue of hemorrhagic fluid cavities, they give a "fluid-fluid" level on MRI.

Chondroblastomas and GCTs are similar in radiographic presentation, except that chondroblastomas occur in the skeletally immature and GCTs in the skeletally mature.[7,18] Although these lesions share the upper tibia and the distal femur as their most common sites of clinical presentation with CMFs, chondroblastomas and GCTs are epiphyseal lesions,[7] typically with eccentric nonsclerotic borders.[18] Histologically, chondroblastomas are relatively hypocellular and show "chicken-wire" calcification[19]; GCTs have many multinucleated giant cells of the same size.[18]

Chondrosarcoma is the most worrisome of lesions in the differential with CMF, but it tends to calcify more readily and to be associated with periosteal reaction and development of Codman triangles. Although thorough knowledge of the radiographic and clinical presentation of CMFs and of those lesions within the differential can lead one to the diagnosis, pathologic correlation is necessary, as malignant transformation has been known to occur. Histologically, chondrosarcomas have a well-differentiated hyaline matrix, lack a fibrous component, and demonstrate cellularity that is almost exclusively chondroblasts.[14]

The gross appearance of our patient's lesion is not dissimilar to what other authors have described. CMF is often glistening, lobulated, and bluish white.[9] Others have reported that it is gray and gelatinous.[20] Regardless of the gross appearance, histologic evaluation is critical to diagnosis and treatment. Thus, as was done in this case, a frozen section is obtained before definitive treatment.

Moreover, histologic evaluation is needed to determine appropriate treatment because the radiographic characteristics of CMF can so closely resemble those of malignant processes.[6] The World Health Organization defined CMF as a "benign tumor characterized by lobulated areas of spindle-shaped or stellate cells with abundant myxoid or chondroid intercellular material separated by zones of more cellular tissue rich in spindle-shaped or round cells with a varying number of multinucleated giant cells of different sizes."[21,22] In a series by Wu and colleagues,[1] 4 distinct matrix patterns were identified: macroglobular, microglobular, mixed, and no pattern. Desai and colleagues[23] described the histologic appearance as bluish myxochondroid lobules with increased peripheral cellularity and occasional giant cells. Although karyotype analysis was not performed in this case, it is certainly of interest to review the literature on this aspect of the diagnosis of the lesion (see Box above).

Cytogenetic Aspects

Several investigators citing cytogenetic studies have indicated varied genetic abnormalities associated with CMFs. Although there appear to be clonal abnormalities involving the short arm of chromosome 6 in CMF, chondrosarcomas also often have abnormalities of this chromosome. Halbert and colleagues[24] were among the first to report on chromosome 6 abnormalities in CMF; there was an uneven reciprocal translocation involving the 6q arm. Previous reports described translocations between chromosomes 2 and 5[25] and between chromosomes 2 and 13.[26] Safar and colleagues[27] reported on the recurrent nature of chromosome 6q aberrations in CMFs but suggested that location and type of genetic abnormality vary. As new technologies arise and the field of molecular genetics develops, diagnosis may progress beyond the need for tissue biopsy.

Treatment Options

Surgical curettage plus bone grafting is an accepted treatment for CMF[6,28] but has been suggested to have a higher recurrence rate.[12,20,29] En bloc wide excision was used in our case to minimize risk for recurrence, alleviate the potential for fracture of the thin cortical rim after intralesional curettage, and achieve rigid internal fixation and structural support to permit early motion and rehabilitation. As it happened, a structural defect was created by virtue of the segmental bone loss secondary to tumor resection, and vascularized bone grafting is indicated in such cases.[30]

Vascularized fibular bone grafting was first described by Taylor and colleagues[31] in 1975. Compared with nonvascularized grafting, vascularized grafting results in faster union, preserves circulation, and improves stress response.[30,32] These advantages must be weighed against the potential adverse outcomes associated with disrupting the vascularity of the extremity, particularly when the viability of the distal structures is entirely dependent on the vessel used for the graft anastomosis.[32] With regard to treatment of distal radius resections with nonvascularized fibular grafts, results have been favorable.[33,34] Although union may occur sooner in vascularized graft, higher nonunion rates have not been observed in nonvascularized grafts in the presence of adequate stabilization.[35] In our patient's case, the decision to proceed with vascularized autologous fibular bone grafting was based on patient preference and on wanting to avoid the potential disease transmission risks of allograft and to maximize the likelihood of early union and return to function.

A preoperative Allen test[36] was performed to confirm adequate collateral blood supply to the fingers through the ulnar artery in anticipation of potential surgical modification of the radial artery. After the graft was stabilized with a spanning locking plate, the vascular pedicle of the fibula was found to be of insufficient length to perform a tension-free end-to-end anastomosis with the perforating branch to the brachioradialis that had been isolated for that purpose during the exposure. Because the patient had a normal Allen test, a direct end-to-end anastomosis with the radial artery itself was briefly considered, but it was not technically feasible because of the lumen diameter mismatch between the donor radial artery and the recipient fibular vascular pedicle at the level of the midforearm. An end-to-side anastomosis into the radial artery was also contemplated, but it would have introduced risks for bleeding and thrombosis with potential distal embolization to the radial digits—risks we thought were too high. We were comfortable proceeding with a nonvascularized fibular autograft because previously reported success rates were still reasonably high.[33,34]

Adjuvant treatment, such as radiation therapy, is contraindicated in primary resection because it has been associated with increased risk for malignant transformation.[13,14,16] Radiotherapy is recommended in recurrent disease, which is not uncommon in bony lesions but is very rare in soft tissue.[20]

Conclusions

CMF is an extremely rare cartilaginous primary lesion of bone, and its obscure presentation pattern makes diagnosis difficult, particularly when the anatomical location is atypical. The diagnostic challenge begins with determining the potential of the lesion to be a malignant process and proceeds with determining the most stable treatment method. The risks of aggressive surgical resection must be weighed against the likelihood of recurrence if excision is inadequate. Our patient's case is unique in 2 ways. First, the unusual anatomical location (in the diaphyseal portion of the radius) led to suspicion of many other pathologic processes; CMF was not part of the initial differential diagnosis. Second, a nonvascularized fibula graft was used for structure stability without compromising distal extremity vascularity. Although early union was apparent on radiographs at most recent follow-up, long-term clinicoradiographic evaluation for this lesion should be done every 3 months for 3 years, every 6 months for 2 years, and on a yearly basis thereafter to track ultimate incorporation and rule out recurrence of the lesion. We hope this report offers further understanding of this rare lesion.

Authors' Disclosure Statement

The authors report no actual or potential conflict of interest in relation to this article.

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