

Aneurysmal Bone Cyst: Concept, Controversy, Clinical Presentation, and Imaging

Mark J. Kransdorf^{1,2} and Donald E. Sweet³

The so-called aneurysmal bone cyst...is neither a cyst nor a neoplasm; rather it is probably a periosteal to intraosseous arteriovenous malformation, not uncommonly seen in association with other well known benign and even malignant lesions.

Mirra [1]

Dr. Copeland: The term aneurysmal bone cyst, then, represents a state of mind when used in classifying the end result of a variety of disease processes?

Dr. Jaffe: Yes.

Jaffe [2]

Concept

The origin of the term "aneurysmal bone cyst" stems from two cases reported by Jaffe and Lichtenstein [3] in their article on unicameral bone cysts in 1942. In that report, they noted two "peculiar blood-containing cysts of large size," which they described as aneurysmal cysts. In a subsequent paper, Jaffe chose the name "aneurysmal bone cyst" as the descriptive term for this lesion, with the word "aneurysmal" to emphasize the "blown-out," distended contour of the affected bone, and the words "bone cyst" to underscore that when the lesion is entered through a thin shell of bone, it appears largely as a blood-filled cavity [4] (Fig. 1). As originally described by Jaffe and Lichtenstein in 1942, and in following articles by each [3-6], aneurysmal bone cyst was sufficiently characteristic to identify it as a distinctive radiologic-pathologic entity. However, its nature has remained unclear. In both

the original and an ensuing paper on the subject, Jaffe postulated that aneurysmal bone cyst may be a secondary phenomenon due to a hemorrhagic "blow-out" in a preexisting lesion, which may be destroyed in the process [3, 4]. Lichtenstein also suggested a vascular origin but postulated that the lesion was the result of a "local circulatory disturbance," noting that although "the precise basis for this vascular disturbance is not readily discernible... it could conceivably be thrombosis of a sizable vein, or perhaps an anomalous arteriovenous communication" [5, 6].

The tendency toward formation of blood-filled spaces (or telangiectasia) is observed in many highly vascularized tumors such as giant cell tumor, osteoblastoma, angioma, chondroblastoma, telangiectatic osteosarcoma, and traumatized (fractured) bone cyst, and less commonly in other diverse lesions such as fibrous dysplasia, nonossifying fibroma (fibroxanthoma), and chondromyxoid fibroma [7-11]. This phenomenon has led increasing numbers of orthopedic pathologists to regard aneurysmal bone cyst as a secondary vascular phenomenon superimposed on a preexisting lesion. The primary antecedent lesion, presumably through involuntarily change within its recognizable structure, initiates an intraosseous "arteriovenous malformation and thereby creates, via hemodynamic forces, a secondary reactive lesion of bone, which we know as an aneurysmal bone cyst" [7]. This process further modifies and may obliterate the original morphology, obscuring the true nature of the preceding lesion [1]. Thus, conceptually, it appears that aneurysmal bone cyst

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¹Department of Radiology, Saint Mary's Hospital, Richmond, VA 23226. Address correspondence to M. J. Kransdorf.

²Department of Radiologic Pathology, Armed Forces Institute of Pathology, Washington, DC 20306-6000.

³Department of Orthopedic Pathology, Armed Forces Institute of Pathology, Washington, DC 20306-6000.

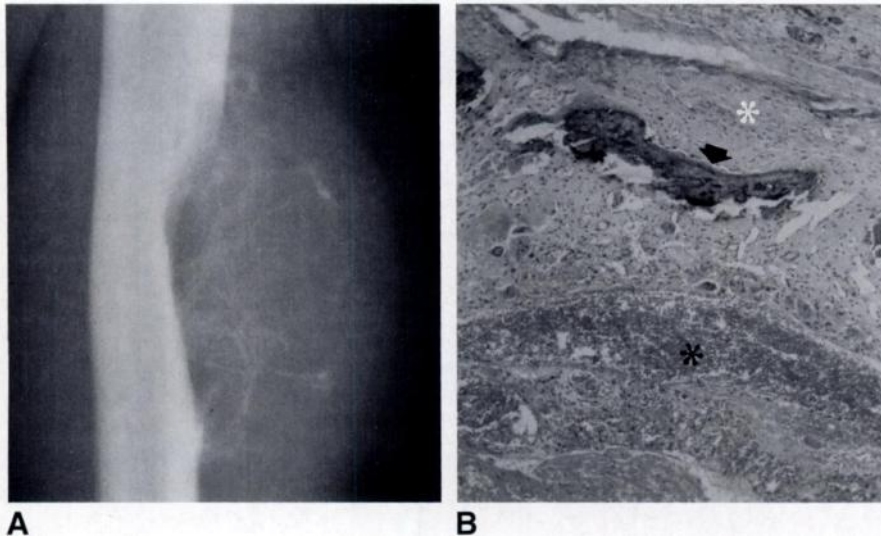


Fig. 1.—Aneurysmal bone cyst of humerus in 20-year-old man.

A, Radiograph of humerus shows subperiosteal lesion in mid-diaphysis. Bone has expanded, remodeled contour, with "ballooned-out" appearance. Note delicate trabeculated appearance.

B, Photomicrograph of solid portion of lesion's shell shows prominent vascular channels (black asterisk), with fibrous tissue (white asterisk), and reactive bone (arrow). (H and E, original magnification $\times 50$)

is indicative of a pathophysiologic change, rather than a unique entity. This was probably most succinctly expressed by Jaffe and Copeland in 1963, during a discourse at the Eighth Annual Clinical Conference on Cancer [2], in which they agreed the "term aneurysmal bone cyst...represents a state of mind when used in classifying the end result of a variety of disease processes."

The concept of aneurysmal bone cyst as a secondary phenomenon occurring in a preexisting lesion has been validated

by multiple investigators; in approximately one third (29–35%) of cases, the preexisting lesion can be identified [7–10]. The most common of these is giant cell tumor, which accounts for 19–39% of those cases in which the preceding lesion is found [7–10]. Other common precursor lesions include osteoblastoma, angioma, and chondroblastoma (Fig. 2). Less common lesions include fibrous dysplasia, fibroxanthoma (nonossifying fibroma), chondromyxoid fibroma, solitary bone cyst, fibrous histiocytoma, eosinophilic granuloma, radiation ostei-

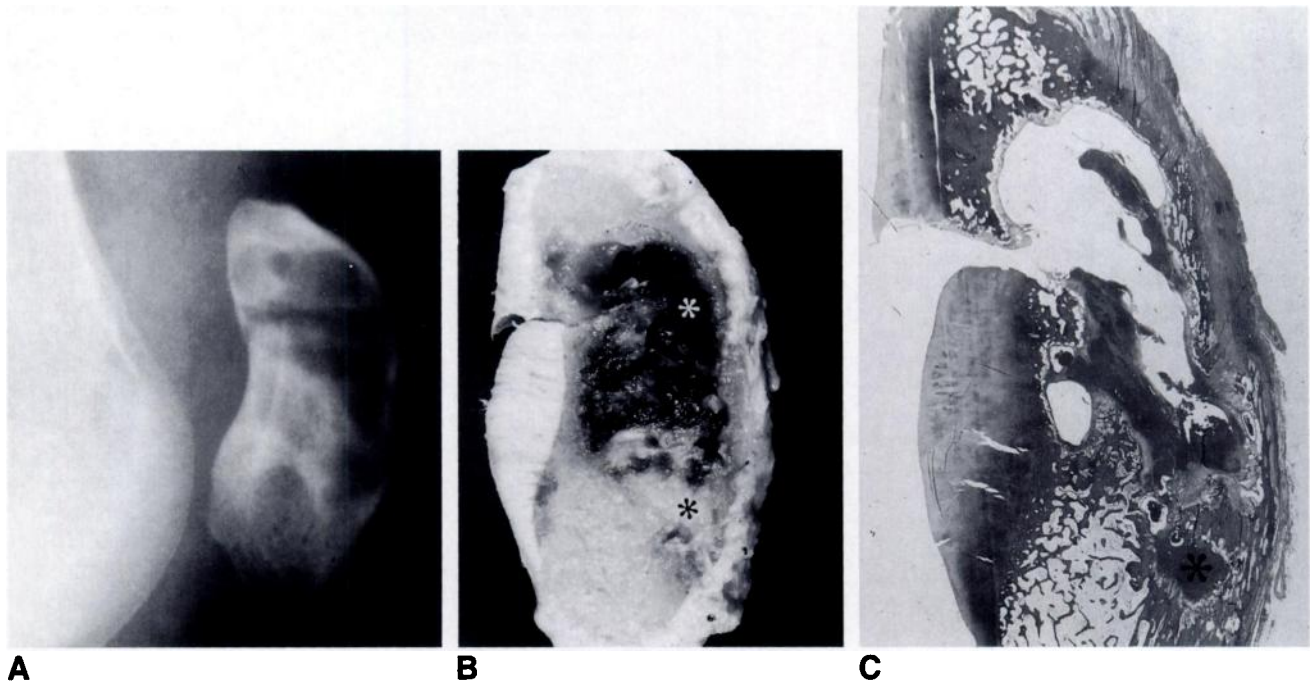


Fig. 2.—Cystic chondroblastoma of patella in 23-year-old man.

A, Lateral radiograph shows pathologic fracture through geographic lytic lesion in mid and upper patella. Lesion has well-defined, minimally sclerotic border.

B, Gross photograph shows lesion is predominantly hemorrhagic (white asterisk) with small residual solid tumor nodule (black asterisk) in inferior aspect of lesion.

C, Photomicrograph of macroscopic section shows a similar configuration. Residual tumor nodule (asterisk) is again noted. (H and E, original magnification $\times 1.0$)

tis, osteosarcoma, trauma (including fracture), fibrosarcoma, and even metastatic carcinoma [1, 7–13] (Fig. 3). Other investigators have proposed two forms of aneurysmal bone cyst, those in which the lesion is secondary to an identifiable precursor, and a “primary aneurysmal bone cyst” in which no preexisting lesion is identified [10].

Aneurysmal bone cyst has been likened to a “blood-filled sponge” [6], composed of blood-filled, anastomosing, cavernomatous spaces, separated by a cystlike wall composed of fibroblasts, myofibroblasts, osteoclastlike giant cells, osteoid, and woven bone [1, 14] (Fig. 4B). In approximately one third of cases, a characteristic reticulated lacy chondroidlike material, also described as a calcified matrix with a chondroid aura, is seen [1, 15]. Mirra notes this is “strongly suggestive, if not pathognomonic, of repair” in an aneurysmal bone cyst. This supports the concept that aneurysmal bone cyst may be primarily a reparative process, probably the result of “trauma or tumor induced anomalous vascular process” [1].

Controversy

Much of the controversy surrounding this lesion may result from a change in how the lesion was defined by Lichtenstein in 1953 [6]. The radiographs of the original two cases described in 1942 and subsequent independent case descriptions by Jaffe and Lichtenstein in 1950, when reviewed, suggest that the original concept of aneurysmal bone cyst was of a juxtacortical (cortical or subperiosteal) lesion [3–6] (Fig. 1). By written description, these authors describe the lesion as “ballooned-out,” resembling a “coarse soap bubble” and “located eccentrically...to involve only one side of the shaft” [3–6] (Fig. 1). Review of the contemporary literature showed accepted synonyms for the lesion included subperiosteal giant cell tumor, benign bone aneurysm, aneurysmal giant cell tumor, and ossi-

fying subperiosteal hematoma [5, 6, 16]. The association of trauma was noted by both Jaffe and Lichtenstein [4, 5], although it was dismissed as coincidental. Thompson [16], reporting nine cases of subperiosteal giant cell tumor from the Mayo Clinic, noted a history of trauma in 71% of patients, supporting the possible posttraumatic hemorrhagic vascular nature of the lesion as originally described.

Lichtenstein, in 1953 [6], reported “further observations” in an additional nine cases of aneurysmal bone cyst. He again emphasized the lesion’s expanded contour and eccentric location but also noted a case in the proximal part of the ulna, commenting that “an occasional lesion in a slender tubular bone like the ulna that has not yet attained large size may also show symmetrical, rather than eccentric, expansion.” Lichtenstein [17] subsequently reported his observations on 50 cases in 1957 (including 33 unpublished cases), again combining both juxtacortical and intramedullary lesions. By including these latter cases, Lichtenstein combined the originally described cases, which were juxtacortical or on the surface of a bone and which may have been primary posttraumatic lesions, with those of intramedullary origin, which more likely reflect secondary hemorrhage or vascular malformations within preexisting lesions. Thus, although the pathophysiologic change is a common denominator, conceptually the lesions’ origins are probably different.

The concept that the radiologic–pathologic entity designated aneurysmal bone cyst is the result of a specific pathophysiologic change is generally accepted. The fact that not all series have identified an association with preexisting lesions may be partially explained by the likely different pathogenesis of “subperiosteal” and “intramedullary” lesions, by the possible complete obliteration of diagnostic morphology in the latter group, and by the difficulty in identifying the presence of diagnostic morphology in the scant residual por-

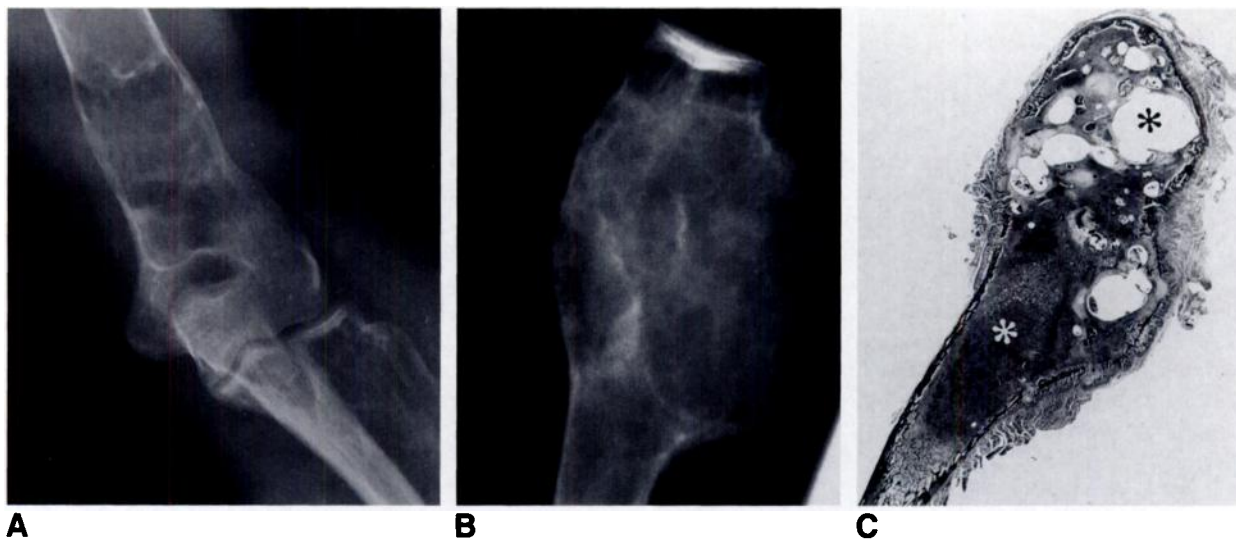


Fig. 3.—Polyostotic fibrous dysplasia of upper extremity, with secondary cystic change.

A, Anteroposterior radiograph of elbow shows central lytic lesion in diaphysis and metaphysis of distal part of humerus. A second lesion with an expanded, remodeled contour is visible in proximal part of radius. No matrix identified.

B, Specimen radiograph of resected proximal part of radius shows expanded contour, with thin trabeculations, which represent areas of subperiosteal bone reinforcement.

C, Macrosection shows hemorrhage (white asterisk) and cystic change (black asterisk) within fibrous dysplasia. Areas of fibrous dysplasia are well seen in more distal portion on lesion. (H and E, original magnification $\times 1.0$)

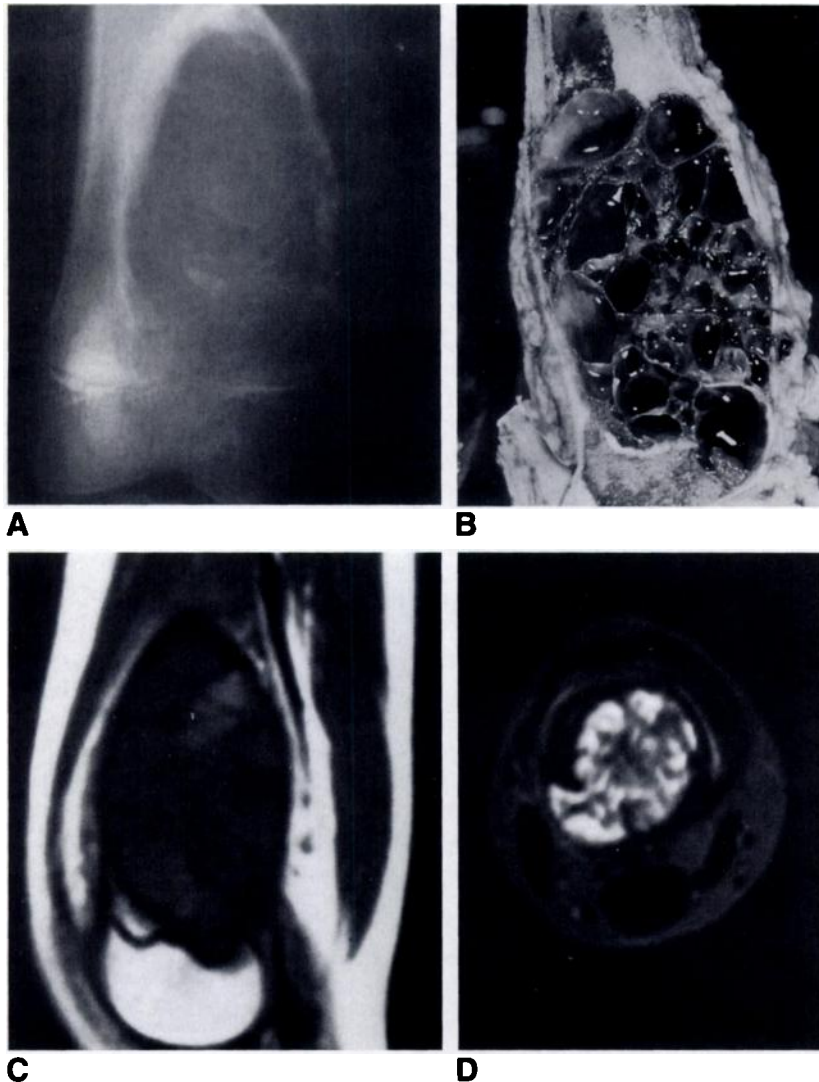


Fig. 4.—Intraosseous aneurysmal bone cyst in distal part of femur of 11-year-old girl, likely secondary to preexisting lesion.

A, Anteroposterior radiograph of distal part of femur shows well-defined geographic lytic lesion, eccentrically located in metaphysis.

B, Corresponding gross photograph shows multiple cysts and small amount of solid material within lesion. Gross appearance likened to "blood-filled sponge."

C and D, Sagittal T1-weighted (**C**) (600/20) and axial T2-weighted (**D**) (2500/100) spin echo MR images show multiple cystic areas within lesion.

tions of the lesion. Ruiter et al. [18], in a review of 105 aneurysmal bone cysts, did not find histologic characteristics of other bone lesions, although they did find regions in a few lesions resembling fibrous dysplasia, benign osteoblastoma, and nonossifying fibroma. Similar results were noted by Tillman et al. [19], in a review of 95 cases. Although areas similar to fibrous dysplasia were found in some lesions, no clear antecedent lesions were identified.

What of those "aneurysmal bone cysts" in which a preexisting lesion is not conclusively identified? Can they reasonably be assumed to represent a further progression of this pathophysiologic process resulting in only reactive and non-neoplastic tissue? Examination of giant cell tumor, the most common lesion associated with aneurysmal bone cyst, indicates about 15% of all giant cell tumors will have areas of cystic change that resemble aneurysmal bone cyst [9]. Although only 1.7–10.6% of giant cell tumors occur in children and adolescents [15, 20, 21], approximately 75% of giant cell tumors in the skeletally immature will have such areas of cystic change. These range from small cystic areas in essentially solid lesions to predominantly cystic tumors

with only a small solid component [15]. Accordingly, progression of such a lesion to one with features of aneurysmal bone cyst alone, with no identifiable precursor, would seem logical. The prevalence of aneurysmal bone cysts in children and the increased frequency of cystic and telangiectatic change in lesions such as giant cell tumor in this age group seem more than coincidental.

Observation of the apparent varying origins of aneurysmal bone cyst, as well as the large number of lesions to which the term has been misapplied, has prompted the members of the Department of Orthopedic Pathology at the Armed Forces Institute of Pathology (AFIP) to redefine their use of this designation. Accordingly, for research and retrieval purposes, juxtacortical lesions that are compatible with Jaffe and Lichtenstein's original description are coded as cystic subperiosteal giant cell tumors or subperiosteal myositis ossificans; central lesions thought to reflect secondary change in preexisting lesions are identified by their parent lesion. Thus, an aneurysmal bone cyst arising in association with a giant cell tumor would be designated as a cystic giant cell tumor, or giant cell tumor with secondary aneurysmal

bone cyst change, rather than a secondary aneurysmal bone cyst. This emphasizes the underlying lesion and is paramount in establishing the appropriate natural history and prognosis. AFIP members suspect that many cases designated as a primary aneurysmal bone cyst in which no distinct preexisting lesion can be identified may represent giant cell tumors of bone (Fig. 4). Historically, aneurysmal bone cyst was considered to be a variant of giant cell tumor. The microscopic appearances of the two lesions are sometimes strikingly similar, and they are occasionally indistinguishable [22]. In such cases, the conventional distinction is often based on the patient's age, the lesion's location, and radiologic features [22]. Identification of the metaphysis as the location for giant cell tumor in the skeletally immature, as well as the large percentage of such tumors with cystic change in this age group, further underscores the morphologic and pathophysiologic similarity of these lesions and makes the validity of this distinction questionable [15]. Occasionally, intramedullary lesions have the morphologic features of an "aneurysmal bone cyst" alone, which members of the AFIP suggest reflects cystic change in preexisting angiomas or secondary telangiectatic change in antecedent bone cysts. Such lesions have been coded as angiomatous bone cyst or atypical bone cyst, reflecting Jaffe's original use of the term.

The term "solid aneurysmal bone cyst," coined by Sanerkin et al. [23] in 1983 describes a lesion that contains the characteristic reticulated, lacy, chondroidlike material seen in conventional aneurysmal bone cyst, but without the typical vascular and cystlike cavities. In a recent review of 238 patients with primary aneurysmal bone cyst in the Mayo Clinic files [22], about 5% were the "solid" variant. This corresponds with file results at the Rizzoli Institute, in which 15 (7.5%) of 200 cases were the solid variant of aneurysmal bone cyst [24]. Histologically, lesions described as solid aneurysmal bone cyst (or conventional aneurysmal bone cyst) may be confused with malignant tumor; particularly a low-grade osteosarcoma [1].

The similarity of giant cell reparative granuloma of the jaw to the solid portions of aneurysmal bone cyst was noted by Dahlin and McLeod [14]. More recently, the solid aneurysmal bone cyst has been equated to the giant cell reparative granuloma in long bone [25], as well as the giant cell reparative granuloma of the small bones of the hands and feet [22]. There appear to be no differences in the clinical or radiologic presentation in patients with classic aneurysmal bone cyst or the solid variant [22, 24]. As with conventional aneurysmal bone cyst, care must be exercised in the morphologic assessment of these lesions to distinguish those most likely representing responses to intraosseous hemorrhage from true hemorrhagic neoplasms.

Clinical Presentation and Treatment

In general, the clinical presentation of patients with secondary aneurysmal bone cyst will mirror those of the associated preexisting lesion [9]. The vast majority (approximately 80%) of patients with primary aneurysmal bone cystlike findings are less than 20 years old. The lesion is rare in children under 5 [1, 7–10, 14, 18, 22, 26]. Females are affected slightly more often than males [1, 14, 18, 22]. More than half

of all such lesions occur in long bones [1, 14, 27, 28]. Spinal lesions account for approximately 12–30% of cases [1, 14, 26, 27]. The pelvis accounts for about half of all flat bone lesions [22]. Most patients have pain and/or swelling, with symptoms usually present for less than 6 months [22]. Vergel De Dios [22] reported multiple bone involvement in 20 (8.4%) of 238 patients with primary aneurysmal bone cyst, 95% of which involved contiguous spine lesions. Pathologic fracture is not uncommon, especially in the spine.

In long bones, the lesion is usually eccentric and metaphyseal [29, 30]. Epiphyseal lesions are usually intramedullary and associated with chondroblastoma or giant cell tumor [1]. An associated lamellated periosteal reaction may be seen in 63% [22, 29]. Spine involvement is typically in the posterior elements, although extension into the vertebral body is common (75%) [14, 29]. Involvement localized to the vertebral body is rare [1], and associated scoliosis and kyphosis are present in more than 10% of patients [1]. Spinal lesions commonly will cause compression of the spinal cord or nerves [14].

The natural history of aneurysmal bone cyst has been described as evolving through four radiologic stages: initial, active, stabilization, and healing [31, 32]. In the initial phase, the lesion is characterized by a well-defined area of osteolysis with discrete elevation of the periosteum. This is followed by a growth phase, in which the lesion grows rapidly with progressive "blown-out" radiologic appearance. The growth phase is succeeded by a period of stabilization, in which the characteristic "soap bubble appearance" develops, as a result of maturation of the bony shell. Final healing results in progressive calcification and ossification, with the lesion transformed into a dense bony mass.

Conventional treatment has been directed at the surgical removal of the entire lesion, or of as much as possible [14]. Recurrence is seen in 10–44% of cases [6, 14, 18, 22], with 90% recurring within 2 years [22]. Appropriate treatment of "aneurysmal bone cyst" requires realizing that it has a specific pathophysiologic origin, and identifying the preexisting lesion, if possible, is essential, especially for intramedullary lesions. If no coexistent lesion is identified, lesions are usually treated with curettage and bone grafting, with more aggressive treatment reserved for recurrent lesions [1]. If a more aggressive lesion is present, treatment must be directed toward the more aggressive component [1]. Clearly an osteosarcoma with superimposed secondary cystic change must be treated as an osteosarcoma, and giant cell tumor with features typical of a secondary aneurysmal bone cyst would be expected more likely to recur locally [1].

Recently, embolotherapy has been used to treat vascular bone tumors to limit blood loss at surgery or as definitive therapy when surgery is not feasible [29, 33, 34]. Transcatheter selective arterial embolization has proved useful in this regard, either by itself or in combination with surgery. The goal of embolization is occlusion of the vascular supply of the lesion without interfering with the vascularity of surrounding tissue or structures [33, 34]. Various materials have been used, depending on the size of the vessels involved and the therapeutic indications [33].

De Cristofaro et al. [33] reported recurrence in only two of

19 cases treated by embolization: 14 patients with aneurysmal bone cyst and five patients with angioma of bone. In five cases, embolization could not be performed; in one cervical lesion, the vascular supply of the lesion could not be separated from that of the spinal cord, and in one scapular and three pelvic lesions, a recognizable feeding artery could not be identified. One of the recurrent lesions became stable after a second embolization. Successful embolization of an aneurysmal bone cyst will result in progressive ossification within 2–4 months of initial embolization, almost always beginning peripherally [33], not unlike that seen in callus or myositis ossificans. Complete ossification may require 8–12 months or longer [33].

Imaging

The imaging appearance of aneurysmal bone cyst will reflect the underlying pathophysiologic change. Bonakdarpour et al. [10] noted that of 26 cases of aneurysmal bone cyst in

which a preexisting lesion could be identified, 21 (81%) had the radiologic appearance typical of the primary lesion.

Radiographs typically will show an eccentric, lytic lesion with an expanded, remodeled "blown-out" or "ballooned" bony contour of the host bone [14, 26, 29, 30]. The appearance of an expanded contour is the result of bone production by the periosteum, stimulated directly or indirectly by underlying pathophysiologic change. Lesions frequently show a delicate trabeculated appearance (Figs. 1 and 5). This is nonspecific but almost always represents reinforcing internal ridges of periosteum and should not be mistaken for bony septa within the lesion [5, 35]. The outer shell of bone may be partially absent [26]. Usually eccentric in large tubular bones, lesions in the short tubular bones may appear more central, filling the entire shaft of the bone [26]. Cortically centered lesions comprise about 12–18% of cases, whereas purely surface lesions are less common and comprise about 7–8% of cases [22, 27]. These juxtacortical lesions are typical of those initially described by Jaffe and Lichtenstein [3–6].

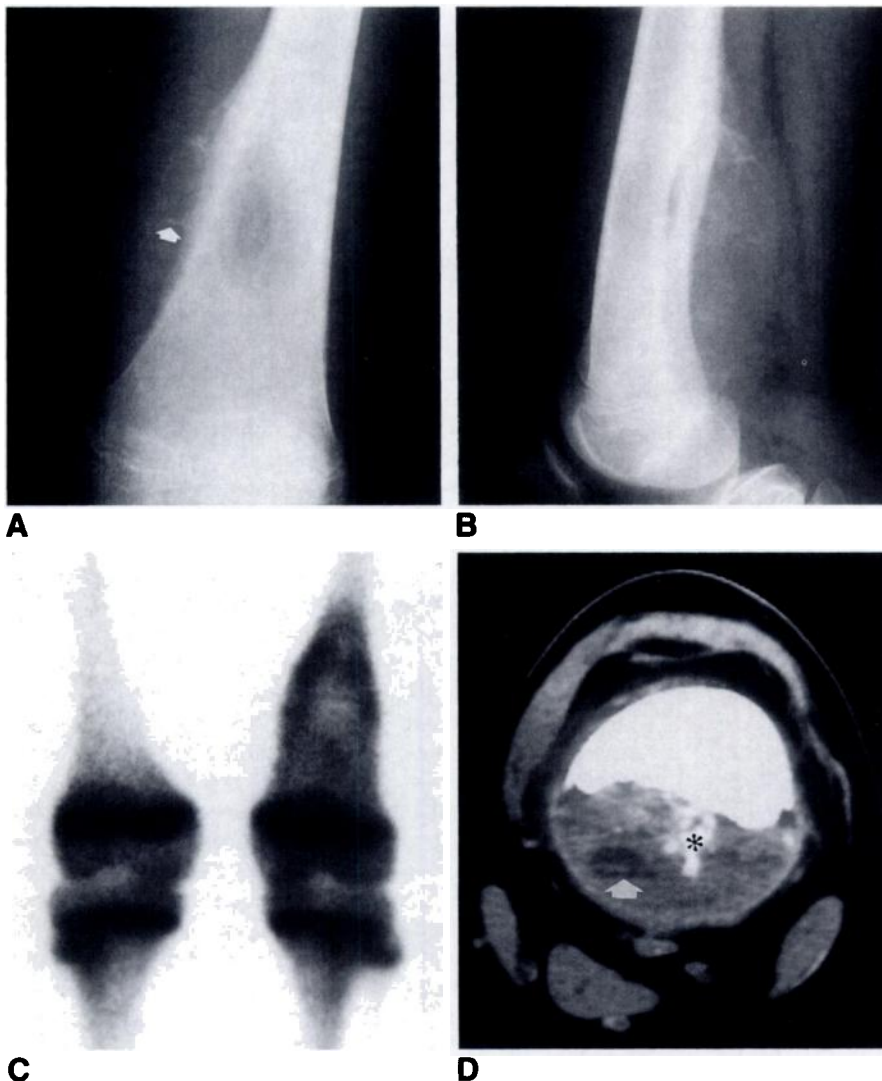


Fig. 5.—Aneurysmal bone cyst (subperiosteal cystic giant cell tumor) in distal part of femur of 10-year-old girl.

A and B, Anteroposterior (A) and lateral (B) radiographs of distal femur show a lytic lesion along posterior aspect of distal part of femur. Lesion centered on posterior cortex. A few areas of subperiosteal reinforcement (arrow) are visible.

C, Delayed static image from bone scintigraphy shows increased accumulation of radionuclide at periphery of lesion with relative decreased uptake centrally.

D, Axial CT scan photographed at soft-tissue window shows posterior cortical location of lesion. Note fluid-fluid level (arrow) and flocculent opacities within lesion (asterisk), most likely representing mineralized chondroidlike material.

Aneurysmal bone cysts are usually well defined, with geographic margins [26]. Marginal sclerosis is seen in 32% of cases; margins are sharp without sclerosis in 64% and geographic, but poorly defined, in 14% [22]. Radiographs may rarely show flocculent densities within the lesion, which may mimic chondroid matrix [28] (Fig. 5). These densities represent mineralized chondroidlike material within the lesion, and although this is present histologically in approximately one third of cases, it is seen on radiographs and CT scans only when abundant [28]. Gold and Mirra noted it in two (13%) of 15 aneurysmal bone cysts [28] and Vergel De Dios et al. [22] noted faint mineralization in 22 (16%) of 138 cases.

CT scanning can be used to define the lesion and is especially valuable for those lesions that are located in areas in which the bony anatomy is complex and that cannot be adequately evaluated with plain films. CT scanning will show an interrupted cortex in many instances, although in these instances the adjacent soft-tissue mass is smooth, sharp, and well defined, indicative of an intact periosteum [30]. Hudson [30] reported the findings on 17 CT scans of aneurysmal bone cyst, noting fluid-fluid levels in six cases (35%) (Figs. 4 and 5). Hudson emphasized the need to view such scans with a narrow window setting, to identify small differences in fluid attenuation, and to allow time for the fluid to settle to create the fluid levels [30]. In one case in which fluid-fluid levels were observed and then not seen when the patient was repositioned, 10 minutes was adequate to allow them to reform [30]. The dependent layer will show increased attenuation [29].

MR imaging will typically show a well-defined lesion, often with lobulated contours [36, 37]. Internal septa with multiple fluid-fluid levels may be seen, although adjacent loculi may have markedly different imaging characteristics [36, 37]. The lesion, as well as internal septa, may show a thin, well-defined rim of decreased signal intensity, thought to be due to fibrous tissue [37] (Fig. 4). In those regions in which fluid-fluid levels are identified, increased signal on T1-weighted images has been reported in the dependent fluid and in the nondependent fluid, presumably due to methemoglobin [29, 38, 39]. Fluid-fluid levels are less commonly seen on T2-weighted images [29]. Fluid-fluid levels are a nonspecific finding and, when present, reflect only the underlying pathophysiology. They are more readily seen on MR images than on CT scans [38].

Bone scintigraphy will show increased uptake of radionuclide in all cases [40]. Hudson [40] reported the scintigraphic findings in 25 cases and showed that scintigraphy accurately reflected the extent of osseous involvement in approximately 88% of cases (22 of 25), with only three showing an extended pattern of radionuclide uptake beyond the tumor margin. In 16 cases (64%), the increased uptake was localized to the periphery of the lesion, with less activity at the center; the remaining lesions showed a diffuse, more or less homogeneous pattern of radionuclide uptake [40] (Fig. 5). Although the hypothesis that the pattern of peripheral uptake corresponds to new bone formation is tempting, Hudson [40] was unable to correlate histologic features with scintigraphic findings. Arteriography shows similar findings with hypervascularity, which is marked in approximately 75% of cases [41, 42]. Most of the hypervascularity is peripheral, with associated areas of decreased vascularity centrally.

As with all bone lesions, radiologic evaluation of an aneurysmal bone cyst begins with the plain radiograph. The next step in the imaging evaluation is determined by the radiologist's ability to establish a diagnosis, or a differential diagnosis, based on the lesion's radiologic appearance, its location, and the patient's age [43].

Summary

The aneurysmal bone cyst is the result of a specific pathophysiologic change, which is probably the result of trauma or a tumor-induced anomalous vascular process. In approximately one third of cases, the preexisting lesion can be clearly identified. The most common of these is the giant cell tumor, which accounts for 19–39% of cases in which the preceding lesion is found. Other common precursor lesions include osteoblastoma, angioma, and chondroblastoma. Less common lesions include fibrous dysplasia, fibroxanthoma (nonossifying fibroma), chondromyxoid fibroma, solitary bone cyst, fibrous histiocytoma, eosinophilic granuloma, and even osteosarcoma.

Interestingly, some of the controversy surrounding this lesion may be the result of a change in how the lesion was defined by Lichtenstein in 1953, when intramedullary lesions were added to the previously described juxtacortical (superficial) lesions. Members of the AFIP have suggested that many of the intramedullary lesions in which no previous lesion can be identified may represent giant cell tumors of bone. Their similarity to proved giant cell tumors in skeletally immature patients can be striking and seems more than coincidental.

Appropriate treatment of an aneurysmal bone cyst requires the realization that it results from a specific pathophysiologic process, and identification of the preexisting lesion, if possible, is essential. Clearly an osteosarcoma with superimposed secondary aneurysmal bone cyst change must be treated as an osteosarcoma, and giant cell tumor with secondary features of aneurysmal bone cyst would be expected to be more likely to recur locally.

The vast majority (approximately 80%) of patients presenting with aneurysmal bone cystlike findings are less than 20 years old. More than half of all such lesions occur in long bones, with approximately 12–30% of cases occurring in the spine. The pelvis accounts for about half of all flat bone lesions. Most patients present with pain and/or swelling, with symptoms usually present for less than 6 months.

The imaging appearance of aneurysmal bone cyst reflects the underlying pathophysiologic change. Radiographs show an eccentric, lytic lesion with an expanded, remodeled "blown-out" or "ballooned" bony contour of the host bone, frequently with a delicate trabeculated appearance. Radiographs may rarely show flocculent densities within the lesion, which may mimic chondroid matrix. CT scanning will define the lesion and is especially valuable for those lesions located in areas in which the bony anatomy is complex, and which are not adequately evaluated by plain films. Fluid-fluid levels are common and may be seen on CT scans and MR images. In addition, MR imaging typically will show a well-defined lesion with a lobulated contour and internal septations. Bone scintigraphy and arteriography usually reflect the vascular nature of the lesion.

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