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DIFFERENTIAL DIAGNOSIS OF GIANT CELL-CONTAINING BONE LESIONS
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Giant cell tumor (GCT)

Definition: A GCT is a primary neoplasm of bone composed of round to oval mononuclear cells, which "form" giant cells. The tumors are usually situated at the end of a long bone in skeletally mature patients. [1]

A GCT of the bone mainly affects patients between 20 and 40 years of age, with a slight predominance for females; occurrence in patients under 15 years of age is rare (2-6). The epiphyses of the long bones are the preferred site, particularly in the distal femur, proximal tibia and distal radius, in this order of frequency [1–6]. About half of the cases are found around the knee, and the tumor has also been found in the sacrum, humerus and vertebrae. A GCT of the small bones in the hand and foot is relatively rare [7, 8]. Roentenographically, a GCT typically presents as a purely lytic lesion in the epiphyseal area of the long bones, with eccentric and expansile translucency. The margin is usually irregular without marginal sclerosis and no periosteal reaction is noted. In patients with soft tissue recurrences or lung metastases, egg-shell like peripheral ossification can be seen.

On gross examination, the tumor frequently shows a brownish area of necrosis with a xanthogranulomatous yellowish-white appearance, and cystic changes with blood retention are also frequently observed.

The histological diagnosis of a GCT depends on the cytologic qualification of the mononuclear stromal cells. The mononuclear cells proliferate in a compact reticular pattern with a relatively ill-defined cytoplasmic border, frequently showing hand-in-hand cohesiveness to each other. Multinucleated giant cells are usually numerous and, in typical areas, tend to be evenly distributed. (Pic. 1). There can be up to 50 nuclei in giant cells, and they frequently share the same cytologic features as seen in mononuclear stromal cells. Mitotic findings are frequent but atypical mitoses are absent in the typical histology of the tumor. An aneurysmal bone cyst-like change has also been reported, focally or diffusely. Secondary reactive changes including necrosis and xanthogranulomatous changes are

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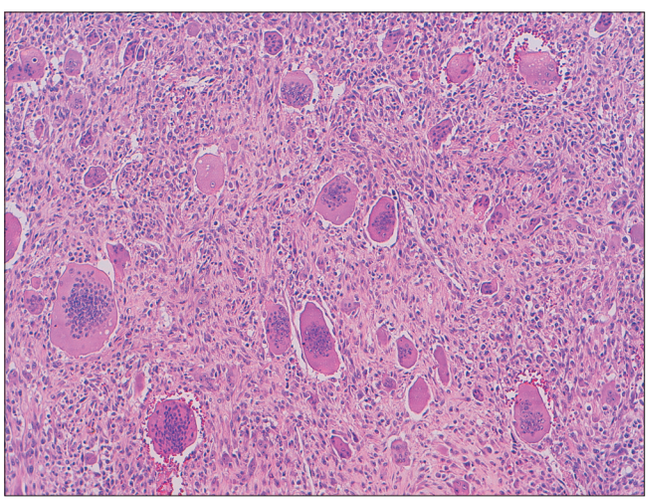
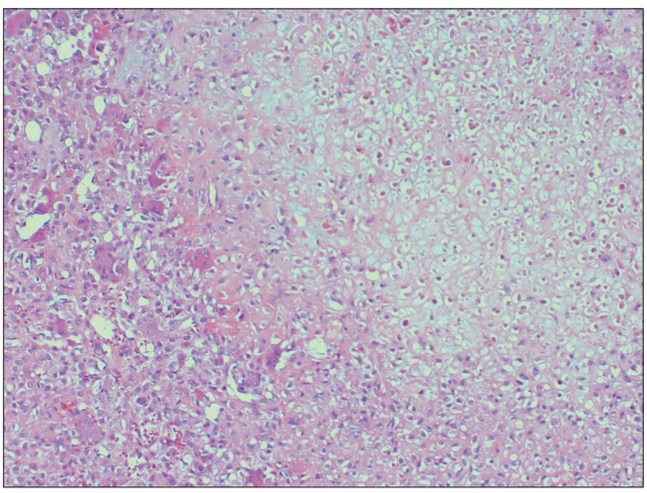


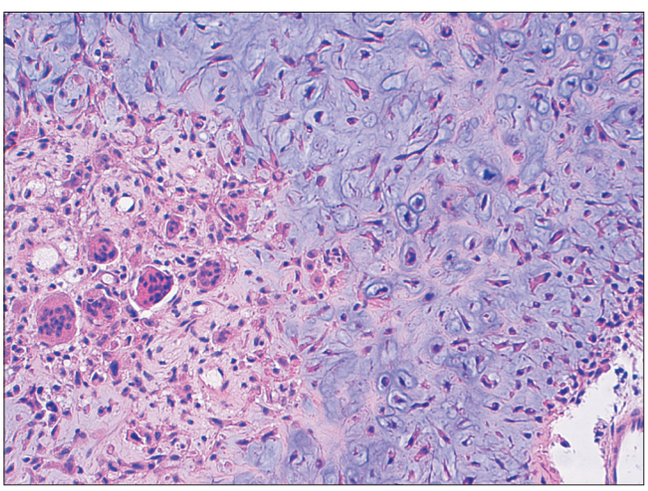
Pic. 1. Typical appearance of a benign giant cell tumor. Giant cells with varying numbers of nuclei are arranged more or less uniformly within backgriund of mononuclear cells

frequent. Mononuclear cells can mimic a fibrous histiocytoma in foci. In some instances, an osteoblastic reaction with osteoid deposition may be observed,and in the areas of soft tissue extension,a bony shell can be seen.

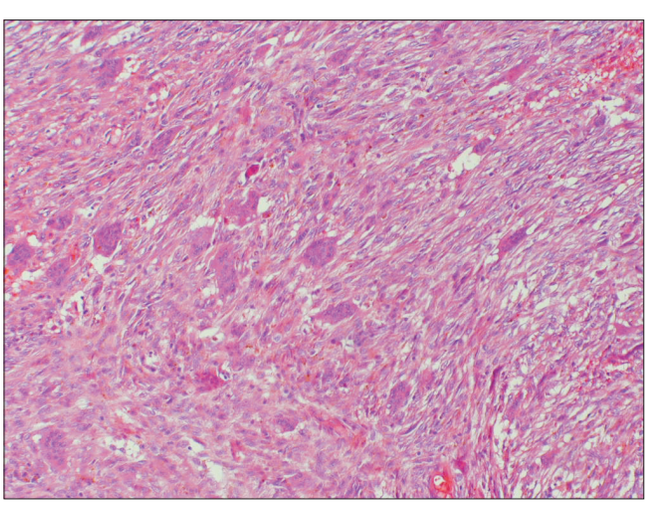
The differential diagnosis for a GCT of the bone must consider various types of benign and malignant bone lesions. Chondroblastomas (Chbls) usually occur from 10 to 20 years of age,and have specific roentgenologic features; mononuclear cells of a chbl are characterized by an indented nucleus simulating histiocytic cells in Langerhans cell granulomatosis and by a distinctive cytoplasmic border. Characteristic calcification and cartilaginous qualities can be observed in the stroma (Pic. 2). A chondromyxoid fibroma can contain a cellular area with numerous multinucleated giant cells between nodules of the myxoid portion, having a typically lobular arrangement under low-powermagnification (Pic. 3). A Metaphyseal fibrous defect (Non ossifying fibroma) shows an extremely characteristic roentgenologic appearance of eccentric translucency with prominent marginal sclerosis in the metaphysis of the long bones. Histologically, spindle cells proliferate in a storiform pattern with numerous foam cells and multinucleated giant cells are



Pic. 2. Mononuclear cells in chondroblastoma have a well defined cytoplasm, some nuclei have a cleaved appearance; some dark lines, outlining the cytoplasmic boundaries represent calcifications

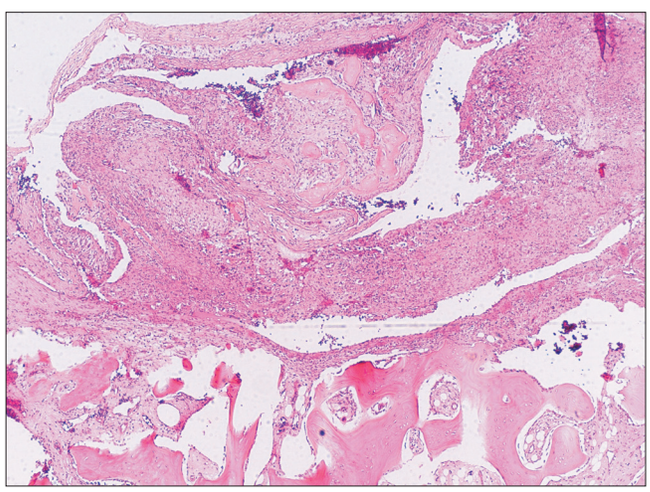


Pic. 3. High-power appearance of a single lobule of chondromyxoid fibroma. The background is myxoid and the cells are spindle- to stellateshaped. An occasional benign giant cells are part of the structure

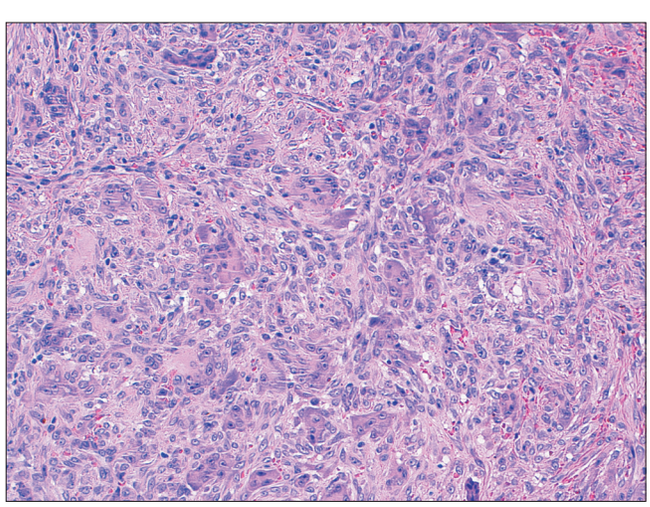


Pic. 4. Typical appearance of metaphyseal fibrous defect. Cell-rich fibroblastic tissue disposed in somewhat whorled bundles. Giant cells are sparser than in the average giant cell tumor

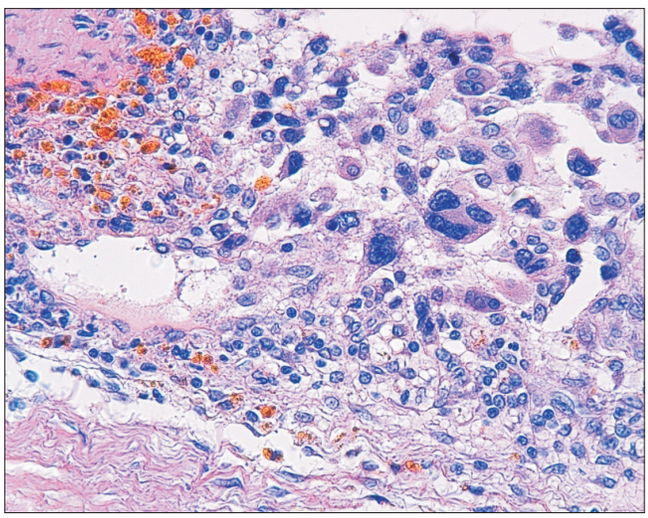
present (Pic. 4). Aneurysmal bone cysts (Pic. 5) and giant cell reactions .(giant cell reparative granuloma, a solid variant of an aneurismal bone cyst) tend to affect the diaphysis or metaphysis of the long bones in adolescents, show reactive reparative change with hemorrhage, necrosis, granulation tissue and frequent ossification (Pic. 6). Zonal architecture with an irregular distribution of multinucleated giant cells is frequent. Multinucleated giant cells may be numerous in telangiectatic Ogs (Pic. 7). Highly anaplastic malignant cells, however, produce neoplastic osteoid, and usually cause no differential problem. Giant cell proliferation may be prominent in bone lesions in hyperparathyroidism, which usually involves the metaphysis or diaphysis in multiple bones with systemic skeletal change, including subperiosteal bone absorption in the hand and foot (Pic. 8). Age distribution, location, and roentgenologic features of an eosinophilic granuloma (Langherans cell histiocytosis, histiocytosis



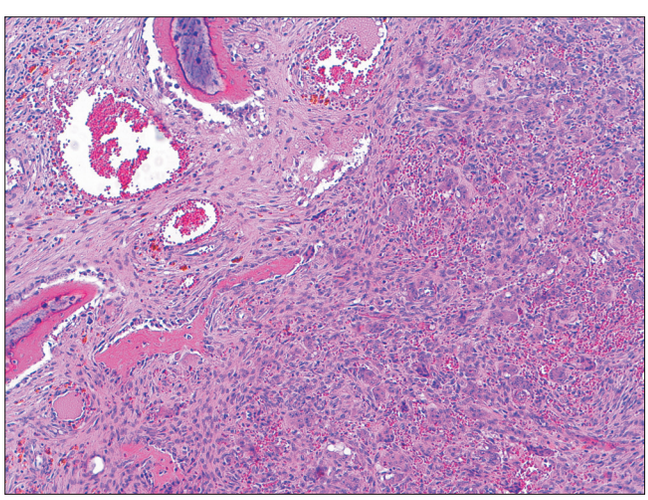
Pic.5. Typical image of the aneurysmal bone cyst. Septa separating spaces contain loosely arranged spindle cells and scattering of benign giant cells



Pic. 6. In giant cell reparative granuloma benign giant cells are present, but the histologic pattern is dominated by fibrobladtic cells



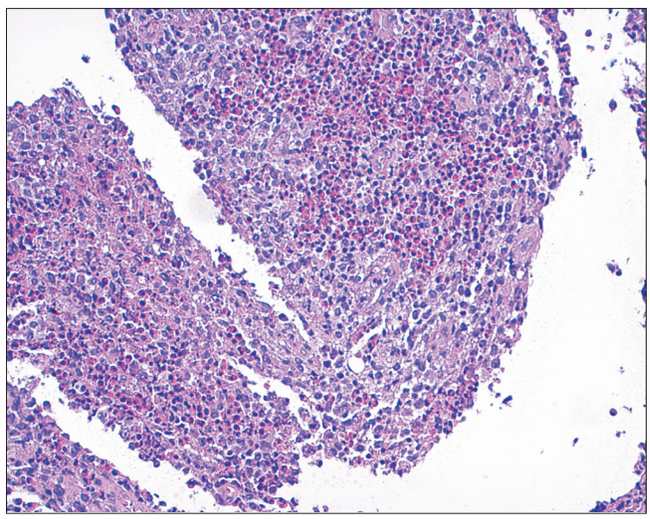
Pic. 7. In telangiectatic osteosarcoma spaces are separated by septa. At low magnification, the lesion cannot be differentiated from aneurismal bone cyst. At high power, the pleomorphic nature of the cells within the septa is obviousc



Pic. 8. Parathyroidizm is a serologic diagnoses, but characteristic appearance of «brown tumor» is a trabecular bone with osteoblastic rimming, surrounded by a fibrous stroma with scattered multinucleated giant cells

X, Langherans cell granulomatosis) are different from those of GCTs of the bone. Under low magnification, an eosinophilic granuloma shows a patchy aggregation of mononuclear histiocytic cells with an irregular distribution of multinucleated giant cells, and the histiocytic cells have coffee bean-like indented nuclei (Pic. 9).

Complete removal of the lesion, is the treatment of choice for GCTs of the bone. Local recurrence has been reported in 20–50% of cases treated with simple curettage and bone grafting, mainly in the first 3 years after surgery [1–5]. Formulating a prognosis in patients with GCTs of the bone is generally difficult, even with meticulous evaluation of the clinical, roentgenological and pathological features of the primary tumor, including findings of vascular invasion; the type of surgical removal may be



Pic. 9. Characteristic cells of histiocytosis X dominate in the picture, clusters of eosinophils are often seen. Multinucleated histiocytes and giant cells are scattered throughout

the most significant factor in recurrence [1– 6, 9–11].

Giant cell tumors of the bone with conventional bland histologic features rarely metastasize to the lung or to other bones [1, 3, 4, 12–21]. The reported incidence of pulmonary metastasis ranges from 1 to 9% and is generally below 2% in larger series. The mean interval between the initial diagnosis and lung metastases ranges from 3 to 5 years [3, 13, 18, 19], and pulmonary metastasis may appear even more than 20 years after the initial treatment of the primary lesion [21]. Many authors have reported difficulty in predicting the development of lung metastases, and that the clinical, demographic, roentgenological,and histological findings of ordinary GCTs of the bone were not reliable predictors of the incidence of pulmonary metastases. However, Siebenrock et al. [18] suggested that local recurrence and a primary lesion at the distal radius were associated with an increased risk of lung metastases. Since pulmonary metastases are extremely rare in cases not having undergone previous surgery on a primary tumor, the number of operations needed to control the primary lesion may be a significant factor in the development of pulmonary metastases(18). Several authors have suggested that GCTs in the hand and foot,which occur in young patients, are frequently multifocal, and have a higher risk of local recurrence and pulmonary metastasis [7, 22]. Biscaglia et al. [8] did not observe multicentricity or pulmonary metastases in GCTs of the bone in the hand and foot. Vascular invasion can be seen in GCTs in the bone, especially in the periphery of the lesion, but cannot predict the development of lung metastasis [1, 11, 18, 23]. The prognostic significance of DNA flow cytometry has not been demonstrated [9, 10]. Masui et al. [24] reported that p53 is prognostically significant. Surgical resection is the preferred treatment for pulmonary metastases in a patient with a conventional GCT [18]. The prognosis of patients with lung metastases from GCTs is generally

favorable, and even spontaneous regression has been reported [12, 18].

The histogenesis of GCTs of the bone is still unknown. Recently, the presence of EWS/FLI-1 hybrid transcripts resulting from at [11, 22] translocation, one of the key features of Ewing's sarcoma, was observed in cases of GCTs of the bone by Scotlandi et al. [25].

Two rare malignant problems related to GCT have to be considered:

Malignancy in GCT [26, 27]
GC-rich Ogs [28]

Malignancy in GCT [26]: In studying the malignant phenomena in GCT of the bone, it is important to use clear definitions. «*Malignant GCT*» is a nonspecific term which has been used in the past to describe giant cell tumors with different degrees of anaplasia, giant cell-rich Ogs, malignant fibrous histiocytomas containing multinucleated giant cells, locally aggressive GCTs of the bone, metastasizing benign GCTs and GCTs with concomitant sarcoma either de novo or after definitive treatment [28]. We use the term **Malignancy in GCT** and distinguish **primary** and **secondary malignancies** according to Hutter et al. [29] and Dahlin et al. [30]. A **primary malignancy in GCT**(**PMGCT**)is a lesion in which there are areas of synchronous high-grade sarcomatous growth next to areas of benign GCT.A **secondary malignancy in GCT**(**SMGCT**) is a metachronous high-grade sarcomatous growth superimposed on a previous, biopsy-verified, benign GCT which had been treated either surgically or by radiotherapy. The two types of secondary malignant development, postsurgical and radiotherapy-induced, are believed to have different etiologies but cannot be distinguished from each other on the basis of radiographic and histologic presentation. Sakkers et al. [31] proposed a noteworthy theory regarding the malignant transformation of GCTs treated by curettage and bone grafting; the theory stated that, in this situation, the reparative proliferative changes which occur at the border of an area of dead bone could serve as the nidus for the formation of a malignant tumor. A comparable transformation has been described in bone infarcts. In fact, in all postsurgical SMGCTs, both in the Rizzoli experience [26] and in other well-documented cases in the literature, bone grafts were used in the treatment of GCTs.

Studies have attempted to define a minimum latent interval between radiotherapy and secondary malignancy,but since these minimum intervals are arbitrary, we decided to consider the following two groups of patients with SMGCTs: patients who received previous radiotherapy at the site of the malignancy and patients who did not receive previous radiotherapy. In the first group, the interval between radiotherapy and diagnosis of the malignancy was 1,7–15 years.(Average 9 years).

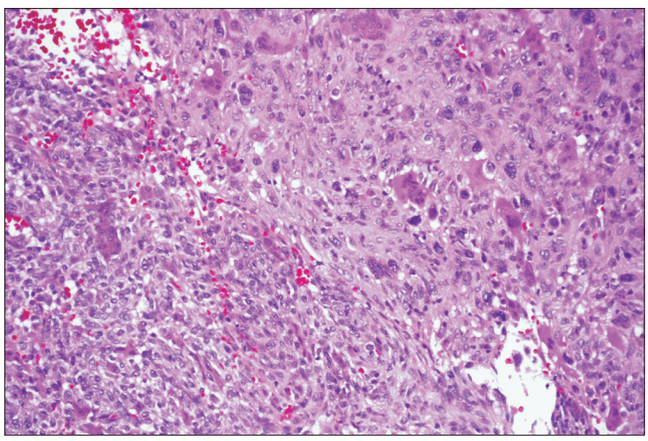
Previous studies found that patients with PMGCTs are older on average than patients with benign giant cell lesions. The current study shows the same tendency, but we emphasize that two of the five patients with PMGCTs were diagnosed in the second decade of life. Patients with SMGCTs after radiotherapy were younger on average at the time of malignancy than were patients with postsurgical SMGCTs. Four of the six patients with post radiation SMGCTs were within the normal age range for patients with a benign recurrence of GCTs. All patients with PMGCTs and five of the six patients with SMGCTs after surgery were male. In the post radiation SMGCT group, the male:female ratio was 1:1, similar to the normal ratio for GCTs of the bone. We cannot explain the high rate of male patients in the current study. In the Mayo Clinic study(27,32), a slight female predominance was found.

As in other studies, we found that the clinical presentation characteristics of PMGCTs and SMGCTs were comparable to those of classic GCTs. In the post radiation SMGCT group, more pelvic bone tumors (50%) were reported, as lesions in this area were more likely to be treated with radiotherapy.

The typical radiographic appearance of PMGCTs was that of a GCT of the bone,and, in most cases, it was impossible to distinguish a PMGCT from a benign lesion on plain films. SMGCT had a much more malignant appearance on plain films, but some cases of SMGCTs were also indistinguishable from benign recurrences of GCTs . In many cases, no computed tomography or magnetic resonance imaging data were available.

Our diagnoses indicated that all malignancies were high-grade sarcomas. In the literature, osteosarcomas, fibrosarcomas, and malignant fibrous histiocytomas have been reported. Whereas Rock et al.(32) found 3 times more fibrosarcomas than ostesarcomas among SMGCT cases, we diagnosed more osteosarcomas among both primary malignancies (4 out of 5 cases) and secondary malignancies (9 out of 12 cases). The other sarcomas encountered in the Rizzoli series were malignant fibrous histiocytomas (one PMGCT and one postsurgical SMGCT) and fibrosarcomas (two post radiation SMGCTs). PMGCTs should be differentiated from GC-rich Ogs,but, as in the mononuclear cells of GC-rich Ogs,the cytologic evidence of malignancy in certain areas can be very subtle;differentiation between these two lesions is sometimes difficult, and its clinical importance is probably limited. (Pic. 10). More important is the fact that both types of lesions can be difficult to differentiate from benign GCTs. The diagnosis of PMGCTs is difficult because they contain areas of benign GCT;therefore,a biopsy may not initially detect the malignant portion.

In the Rizzoli study,in patients with postsurgical SMGCTs ,the average interval between the diagnosis of a benign GCT and that of sarcoma was 18 years, much longer than the average interval observed in patients who received previous radiotherapy (9 years). Different time intervals have been reported in the literature in patients with both types of SMGCTs. Malignant transformation occurred 1.8–36 years after surgery alone and 4–42 years



Pic.10. In malignant giant cell tumor sarcoma like arrears are admixed with typically benign areas of giant cell tumor

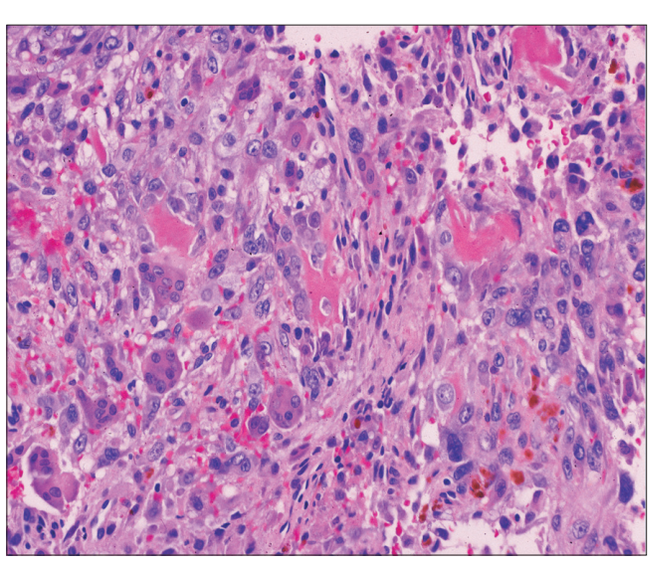
after radiotherapy. In cases of secondary malignancies in which there is a very short interval between the diagnosis of a GCT and malignancy, it is possible that the original lesion was already malignant. Secondary malignancies usually occur > 3 years after the diagnosis of an initial benign GCT. Although benign recurrences of GCTs can occur after long latent periods, most recurrences take place < 2 years after the initial treatment. Thus, in the event of a recurrence > 3 years after the initial GCT, the physician should have a heightened awareness of the possibility of malignant transformation. Current imaging techniques and/or histologic material should lead to a correct diagnosis in these cases.

The treatment of choice for patients in the current study was influenced by several factors and was different for all individuals. Therefore due to the various factors and the small number of patients in the study, our results do not allow us to draw conclusions about the best treatment methods. Early ablative surgery is usually indicated, but the role of chemotherapy is unclear. Antract et al. [33] studied the treatment and outcome of patients with malignant GCTs, but their inclusion criteria differed from ours. They reported a better one-year survival rate after surgery combined with chemotherapy as compared to surgery alone; however, the five-year survival rates and actuarial survival curves showed no statistical differences.

The prognoses for both PMGCTs and SMGCTs are poor. In the current study, PMGCTs demonstrated a better outcome than SMGCTs (especially post radiation SMGCTs). This result could be related to the finding that there were more unfavorable tumor locations in the post radiation SMGCT group. However, because of the small number of cases and some short follow-ups in the PMGCT group, no statistical analysis of survival was carried out. Nascimento et al. [34] reported relatively good outcomes for patients with primary malignancies and stated that these patients had a better prognosis than those with secondary malignancies, but Antract et al. [33] observed equally poor outcomes in both groups.

Malignancies in GCTs are extremely rare events, representing 1.8% of all cases of GCTs of the bone in the current study. These malignancies can be either primary or secondary. Because the prognosis of these sarcomas is very poor, it is imperative that they be recognized at an early stage so that they can be treated adequately by aggressive surgery and, in some cases, chemotherapy. A PMGCT is typically difficult to distinguish from a GCT of the bone, as the clinical and radiographic presentation of a PMGCT can mimic that of a benign lesion. As a result, the malignancy can initially go undetected if a frozen section or biopsy shows only areas of a classic GCT. Suspicion of an SMGCT should increase when recurrence occurs > 3 years after the diagnosis of a GCT of the bone which was treated with surgery or radiotherapy.

GC-rich Ogs [28–35]: Multinucleated giant cells morphologically indistinguishable from those seen in GCTs of the bone can, at times, dominate the histologic appearance of Ogs. This phenomenon has been mentioned in major bone tumor textbooks;however, only a few small series have been published in the literature .In a study by Troup and co-authors [36] evaluating the relationship between GCTs of the bone and Ogs, numerous osteoclast-type giant cells were found in 13% (53) of the 403 osteosarcomas examined. Eight of the 53 were initially diagnosed as GCTs (Pic. 11). Bathurst and co-authors [37] described 9 cases of osteoclast-rich Ogs defined as undifferentiated sarcomas with an overabundance of osteoclasts and a paucity of tumor osteoid. All involved the long bones,and 8 were in the diaphysis or metaphysis. The remaining tumor was in the femoral condyle but was not subarticular. The authors suggested an association between osteoclast-rich Ogs and telangiectatic Ogs. Mirra [38] has indicated that, in



Pic. 11. At low magnification, giant cell rich osteosarcoma have microscopic appearance similar of a giant cell tumor. Delicate network of osteoid and cellular atypia help to make a proper diagnoses

roughly 1–2% of Ogs, giant cells are present in «diffuse and massive quantities», therefore simulating a giant cell tumor of the bone. The authors stressed the importance of recognizing stromal cell anaplasia and the location of the lesion when differentiating giant cell tumor-like Ogs from GCTs.

The criteria defining GC-rich Ogs include the following histologic features [28]: on low magnification, the distribution and number of multinucleated GCs simulated a giant cell tumor of the bone,while, on high magnification, the stromal cells were cytologically malignant and produced osteoid. These histologic criteria are similar to those emphasized by Mirra.

We identified 7 cases out of 1.786 Ogs in the Mayo Clinic files and 17 cases from our consultation (out of approximately 30.000 cases). We therefore confirm that GC-rich Ogs is rare, comprising $<$ 1% of all Ogs.

The most important consideration in the differential diagnosis of GC – rich Ogs is the GCT. If the tumor involves a long bone, the location within the bone can be very helpful in determining the differential diagnosis. The majority of GCT tumors are centered in the epiphysis or the metaepiphyseal end of the long bones. In a study by Fain et al. at the Mayo Clinic, only 14 out of 1682 (0.8%)

GCTs of the long bones were located exclusively in the metadiaphysis or diaphysis. Sanerkin did not find a single tumor in the diaphysis or metaphysic in a series of 86 GCTs. Therefore, a GC- rich nonepiphyseal tumor in the long bones is much more likely to be a GC-rich Ogs than a GCT (if an aneurysmal bone cyst can be excluded histologically), even if there is minimal to moderate stromal anaplasia and osteoid production. We do not, however, use location as a defining criterion since 7 of the long bone lesions in our series were found in a typical site for GCTs. Moreover, the location in the bone is not helpful in the flat bones where there is more overlap in distribution.

The age of the patient is generally not very helpful in differentiating GCTs from Ogs. We found [28] a broad age range with a somewhat older average age than either GCT or Ogs. However, a giant cell-rich lesion in the pediatric population is more likely to be an Ogs since GCTs rarely occur in the immature skeleton.

Radiographic findings may or may not aid in differentiating Ogs from GCTs. Some large GCTs are destructive and poorly marginated, suggesting malignancy. However, only rarely do they contain significant mineralization. Therefore, lesions showing radiographic evidence of heavy mineralization are usually not GCTs.

The most important histologic feature to recognize when making a diagnosis of GC rich Ogs is the presence of cytologic atypia [28]. In 16 (67%) of our cases, stromal cell anaplasia was obvious. However, this can be very subjective, particularly when only subtle to moderate anaplasia is present. The degree of atypia can also vary within an individual tumor making the diagnosis difficult when the amount of biopsy tissue is limited. One must also distinguish degenerative atypia from malignant anaplasia. The most problematic group is that with subtle anaplasia located in a common site for GCTs. Four of our cases fell into this category. Three of these 4 patients died as a result of their tumor within an average period of 20 months and the fourth was lost to follow-up. Since the degree of anaplasia does not appear to be related to biologic behavior, we consider all GC-rich Ogs to be high grade tumors. Moreover, this clinical course also does not correspond to the relatively favorable prognosis and protracted course associated with benign metastasizing GCTs.

In the Mayo Clinic experience of 16 patients with benign metastasizing GCTs of the bone, the overall mortality rate directly due to GCTs and their metastases was 19% over an average time of 9 years.

In general, GCTs do not produce matrix. Some tumors occasionally contain foci of reactive bone formation,especially if there is extension into the soft tissue. This type of osteoid matrix typically shows prominent osteoblastic activity. The pattern of osteoid production by the malignant cells in Ogs is variable. However, it is usually intimately associated with stromal cells and architecturally more irregular than reactive trabeculae. While osteoid production was identified in all the tumors in this series, it was minimal or only focally present in 9 tumors, 5 of which were located in a common site for a GCT. Of these 5, only 1 showed subtle anaplasia in a patient who died 2 years following an initial diagnosis with pulmonary metastases.

In all 9 tumors, we felt that the pattern of osteoid production was more in keeping with what one sees in Ogs, not in reactive bone. Admittedly, at times, the distinction may be very difficult.

Permeation of cancellous and/or cortical bone can be seen in both GCTs and Ogs. However, entrapped host bony trabeculae tightly surrounded by a tumor is a feature often seen in Ogs, not in GCT. Spindling of tumor cells is also found in GCT and Ogs, but spindle cells in GCTs do not demonstrate cytologic pleomorphism. Mitotic figures cannot be used to rule out GCTs since most are mitotically active and, at times, they may be quite numerous. The presence of atypical mitotic figures favors malignancy, but by itself is not diagnostic. Whereas they were fairly easy to detect in tumors with obvious anaplasia, they were rare to absent and therefore difficult to detect in the more problematic tumors with less anaplasia.

Other considerations in the histologic differential diagnosis include malignant fibrous histiocytomas, fibrosarcomas, telangiectatic Ogs, and malignancy in GCTs. Malignant fibrous histiocytomas and fibrosarcomas with giant cells do not tipically contain as many giant cells or osteoid production when compared with GC-rich Ogs. Moreover, the stromal cells in GC-rich Ogs are not diffusely spindle shaped as one would expect in spindle cell sarcoma. Telangiectatic Ogs can also be extremely rich

in giant cells. However, the multicystic appearance along with septae and lacunae are not present in GC-rich Ogs in which the architecture is more solid and compact.

Malignancy in GCTs should be included in the differential diagnosis when the tumor is located in a common location for GCTs. As has been defined previously, it must demonstrate zones of a typical benign GCT in the malignant neoplasm under appraisal or in tissue previously obtained from the same neoplasm. None of the tumors in this series showed a convincing synchronous or metachronous presence of sarcoma along with the GCT.

However, since GC-rich Ogs and malignancy in GCTs are both high grade sarcomas which behave in an aggressive fashion, the distinction may be purely academic.

Of the12 patients treated by incomplete surgical excision, all developed locally recurrent tumors.Of these, 50% developed metastases and died as a result of their tumor. This suggests that surgical treatment for this tumor should be the same as for the common types of Ogs. Chemotherapy in the form of adjuvant or neoadjuvant and/or radiotherapy were employed in too few patients for an analysis of significant results.

In conclusion, GC-rich Ogs is a rare histologic variant of Ogs. The most important consideration in the differential diagnosis is a GCT of the bone. When a GC-rich tumor is located in an unusual site for GCTs, there is a high probability that it is a GC-rich Ogs. In any location,the identification of nuclear anaplasia and osteoid production by the stromal cells remains the cornerstone for reaching a diagnosis. In some cases in which the cytologic atypia is more subtle, only follow-up information may lead to a correct diagnosis.

Recently molecular techniques researched on biomarkers and GCT behavior. An update on molecular mechanisms in GCT were reported at the 101st Annual Meeting,USCAP, Companion Meeting of SBSTP(39).

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ДИФФЕРЕНЦИАЛЬНАЯ ДИАГНОСТИКА ГИГАНТОКЛЕТОЧНЫХ ОПУХОЛЕЙ КОСТЕЙ

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Для многих патологов диагностика заболеваний костей остается малоизученным разделом. Костная патология требует знания специальной терминологии, особого приготовления образцов ткани с применением декальцинации, обязательного сопоставления морфологической картины с данными лучевых методов исследования, включающих рентгенографию, компьютерную и магнитно-резонансную томографию, а также мультидисциплинарный подход с участием хирурга-ортопеда, лучевого диагноста, патолога и, при опухолевых процессах, онколога. Гигантоклеточная пролиферация в той или иной мере является составной частью опухолевого или репатативного процесса в кости. Правильной оценке характера данного явления посвящается обзор с подробным описанием наиболее часто встречающихся опухолей, составной частью структуры которых является гигантская клетка. Это прежде всего гигантоклеточная опухоль, хондробластома, хондромиксоидная фиброма, гигантоклеточная реакция при гигантоклеточной репаративной гранулеме и метафизарном фиброзном дефекте, лангергансоклеточном гистиоцитозе, аневризмальной кисте кости, паратиреоидной остеодистрофии, а также злокачественных процессах, таких как гигантоклеточная остеосаркома, телангиэктатическая остеосаркома и злокачественная гигантоклеточная опухоль. В обзор включены основные классические диагностические признаки «гигантоклеточных новообразований кости» с учетом рентгенологического, макро- и микроскопического исследований.