

Abstract

Aneurysmal bone cyst is a benign lesion occurring most often in excentric position in the long bone metaphysis, pelvis and spine. It may cause pain and swelling which becomes obvious in cases of blowing tumors. In the long bones, pathological fracture is mostly produced in central blowing aneurysmal bone cyst, and in the spine, vertebral fracture. A standard radiograph may be sufficient for diagnosis, and magnetic resonance imaging is also performed for differential diagnosis. A biopsy is mandatory as aneurysmal bone cyst may be secondary to a malignant lesion as telangiectasic osteosarcoma. The most accepted etiopathogenic theory is the one of a reactional process following a venous malformation. The neoplastic theory is now evoked since the translocation t(16;17)(q22;p13) has been discovered that is recurrent in primary aneurysmal bone cysts. Most often a surgical treatment is needed. As aneurysmal bone cyst is a benign condition, radiotherapy has to be avoided. A lot of minimal invasive surgical techniques are nowadays available and have shown their efficiency.

Keywords

Aneurysmal bone cyst • Pseudotumor • Bone cyst • Benign bone lesion

Introduction

Aneurysmal bone cysts are lesions which look like a bone tumor but they are not. Bone cysts are a common example of this. However there are a lot of bone lesions that can

simulate a bone tumor from the clinical, image or pathological point of view.

Aneurysmal bone cyst (ABC) is not really a tumor but rather a pseudotumor. It is a lytic bone lesion located most often in metaphyseal position in the long bones, in the pelvis or the spine. ABC may have different aspects.

Classical ABC: This is a primitive bone lesion, lytic, in metaphyseal position, with multiple cavities separated by septa. The cavities are filled with blood and aggregates of solid tissues (Fig. 23.1).

Other possible precursors are osteblastoma, non ossifying fibroma, chondromyxoid fibroma, fibrous histiocytoma, or eosinophilic granuloma. It may also be secondary to a malignant lesion such telangiectasic osteosarcoma (Fig. 23.2), angiosarcoma, chondrosarcoma or fibrosarcoma.

Multiple cavities and septa are present. The biopsy should demonstrate malignancy.

Secondary ABC: This may be secondary to a traumatism or another pre-existing lesion such a simple bone cyst, fibrous dysplasia, or brown tumor of primitive hyperparathyroidism. It may be secondary to another benign tumor, most often a giant cell tumor.

Solid variant ABC or giant cell resorption granuloma: These ABC are more compact and do not contain cavities.

Soft tissue ABC may develop in the muscles, perivascular areas, the susclavicular or inguinal spaces without bone involvement. The radiographic aspect may evoke myositis ossificans.

Epidemiology

Incidence: ABC is a rare lesion. Incidence is about 1.4 per million people each year. ABC represents 1% of bone tumors.

Age: ABC may occur at every age but mainly during the two first decades of life and becomes rare after 30 years of age (Fig. 23.3).

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Fig. 23.1 Classical ABC involving a long bone metaphysis, with multiple cavities separated by septa

Location

ABC is generally a single lesion, but rare multiple lesions have been described. ABC may involve adjacent bone, which is often the case at the spine level with progression of the tumor to a rib or another vertebra by the articular apophysis. The most frequent location is the long bones of the lower limbs, next the upper limbs, and finally the axial skeleton and flat bones (Fig. 23.4). Location at hands and feet are quite rare and limited to the tubular bones.

Long Bones

ABC is usually excentric in metaphysis (Fig. 23.1) or in the metaphyso-diaphysis and ABC may rarely be located in the diaphysis. ABC is never primarily epiphyseal but may involve secondarily the epiphysis by extension from the metaphysis.

Spine

Vertebral ABC usually first involves the posterior arch of the vertebra (Fig. 23.5). Most often, it progresses to the

vertebral body (71% of cases) Fig. 23.6. Transmission to a rib or an adjacent vertebra is possible. Articular cartilage does not really constitute a barrier at the difference of the intervertebral disc. Weakening of the vertebra may lead to a flattening (vertebra plana).

For most authors, the lumbar location is the most frequent whereas for others, it is the thoracic and cervical location.

Pelvis

The pelvic location is very frequent (11.6%) (Fig. 23.7). ABC is usually initially located in the obturator ring and secondarily may involve the acetabulum or iliac wing.

Sacrum

The sacral involvement is often anterior and posterior (Fig. 23.5). Several sacral vertebrae may be involved. Extension may occur by the sacral to the iliac wing and to the whole pelvis.

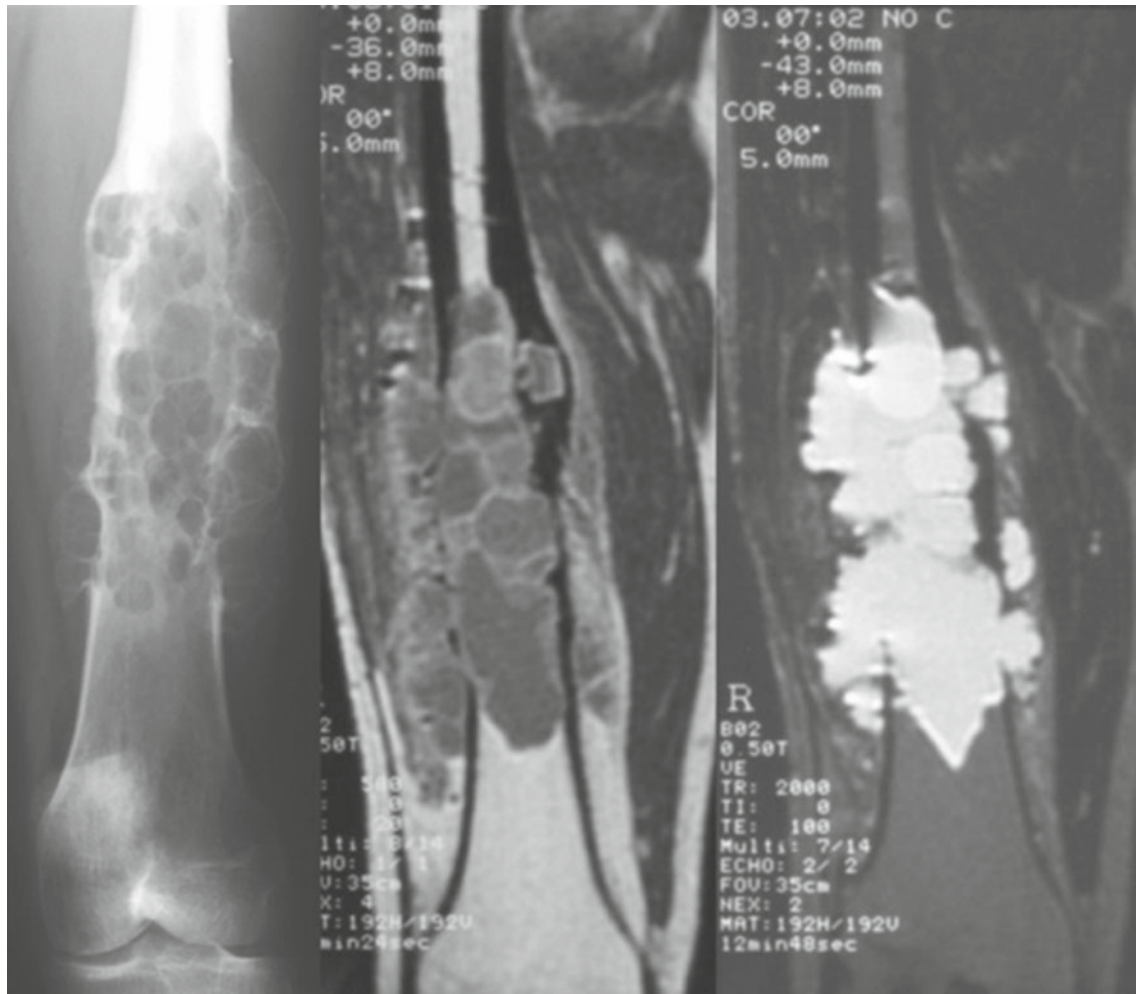


Fig. 23.2 Telangiectastic osteosarcoma may mimic ABC appearance

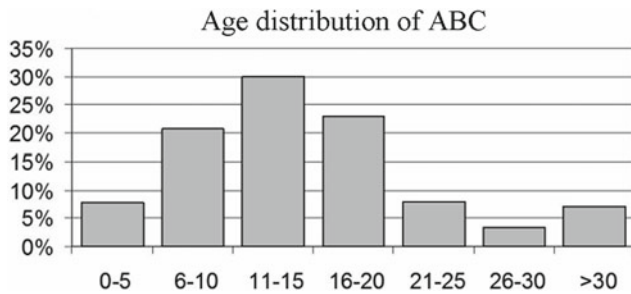


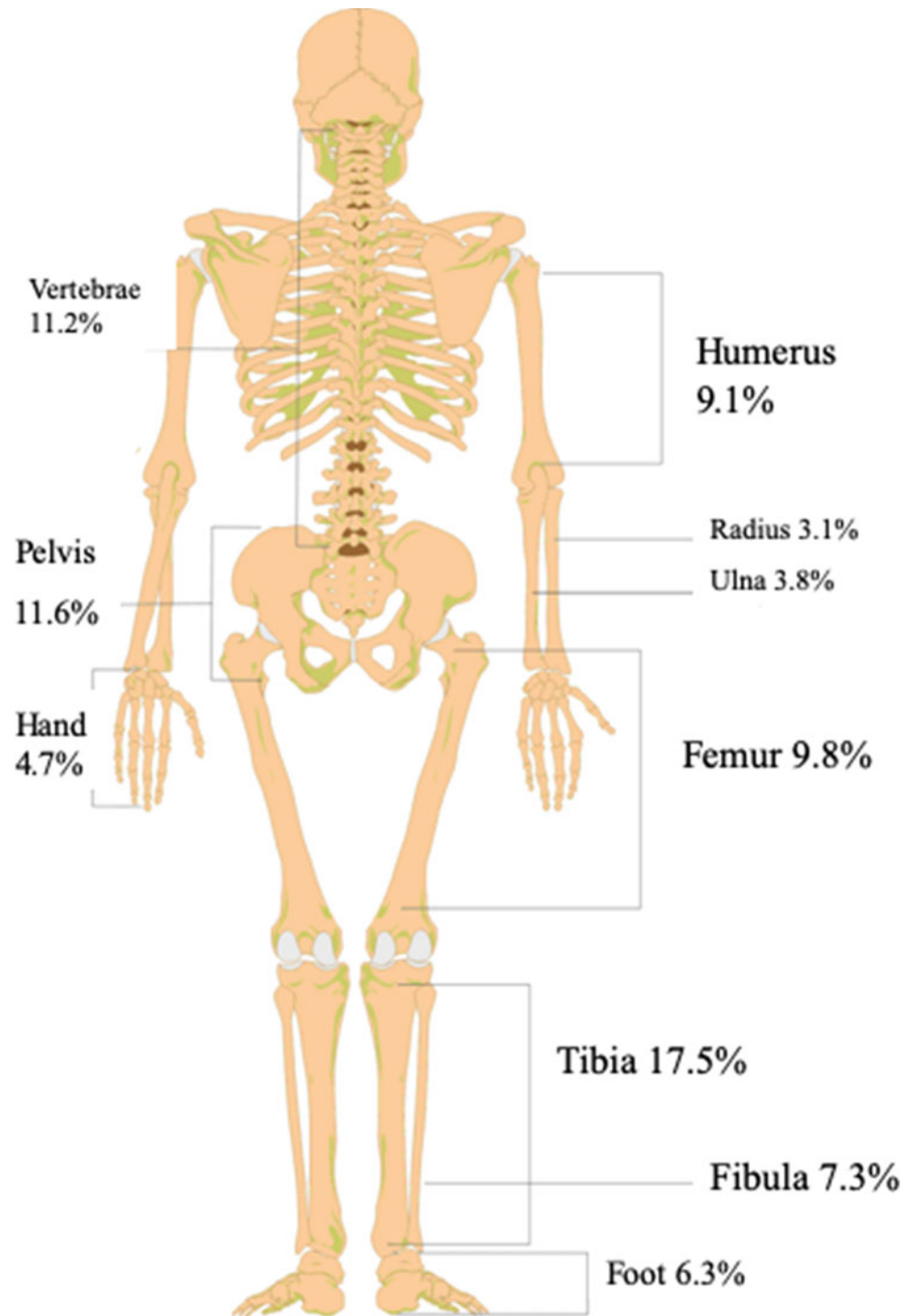
Fig. 23.3 Age distribution of ABC

Etiopathogeny

Several theories have been proposed for the pathogenesis of ABC [1]. The most widespread theory considers ABC as a reactionary process secondary to an intraosseous subperiosteal hemorrhage. This hemorrhage should be secondary to a local

circulatory abnormality with increased venous pressure and dilatation of the local vascular network. This venous malformation may be primitive or secondary. Szendroi et al. found venous abnormalities but no arteriovenous fistulae based on an angiographic study of 20 ABC cases. ABC should be a reactionary tissue to this hemorrhage with osteoclast activation. This theory may also explain the secondary ABC as some tumors may lead to a circulatory perturbation and may contain some area histologically comparable with ABC. Recent studies propose a purely neoplastic etiology to ABC. Panoutsakopoulos et al. demonstrated a chromosomal translocation $t(16;17)(q22;p13)$ recurrently found in primary ABC. Other authors have confirmed that translocation 17p13 is a frequent chromosomal aberration in primary ABC. Oliveira showed that translocation 17p13 locates USP6 oncogen under the regulatory influence of the very active CDH11 promotor. Pathogenesis of most primary ABC should be a up-regulation of the transcription of USP6. On the contrary secondary ABCs do not have this chromosomal aberration.

Fig. 23.4 Bone distribution of ABC



A hereditary factor has been evoked by some authors. DiCaprio et al. reported a case of ABC involving T12 vertebra in a father and another involving L1 in his daughter. Power et al. reported a case of ABC in two monozygotic twins but at different locations.

Clinical Features

The main symptoms associated with ABC are pain and swelling. Pain comes from microfissures due to cortical weakening by the ABC. Swelling may be important and due

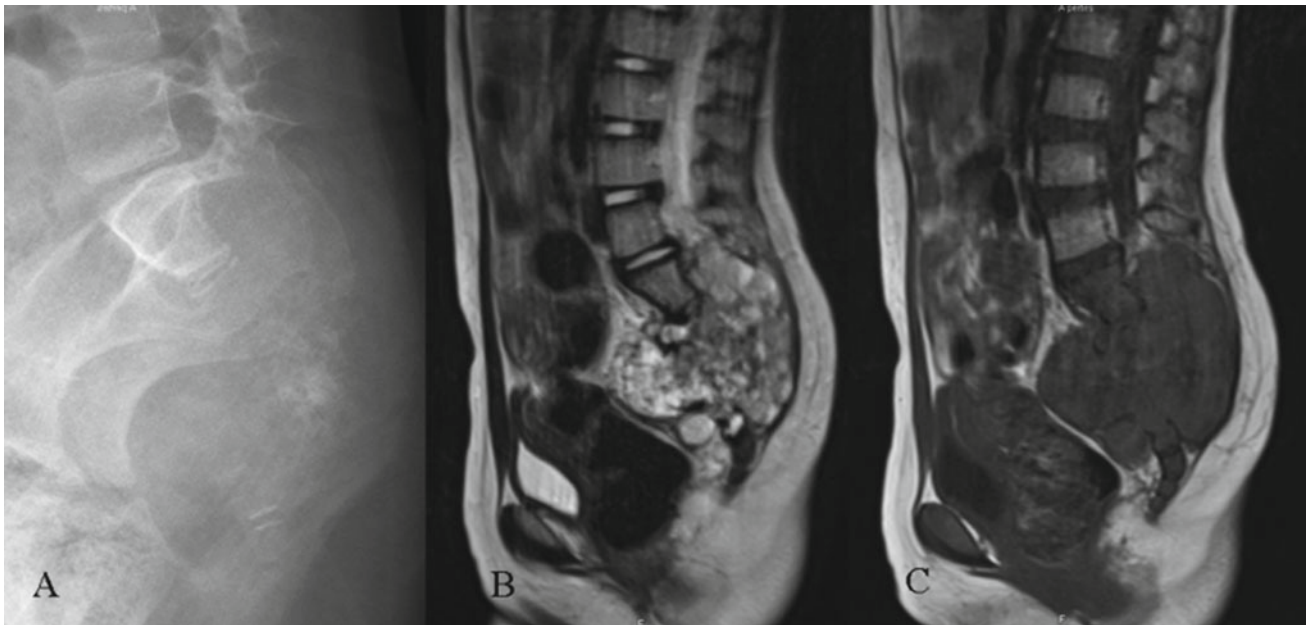


Fig. 23.5 Sacral ABC in a 7-year-old girl; the sacral involvement is often anterior and posterior; several sacral vertebrae are involved

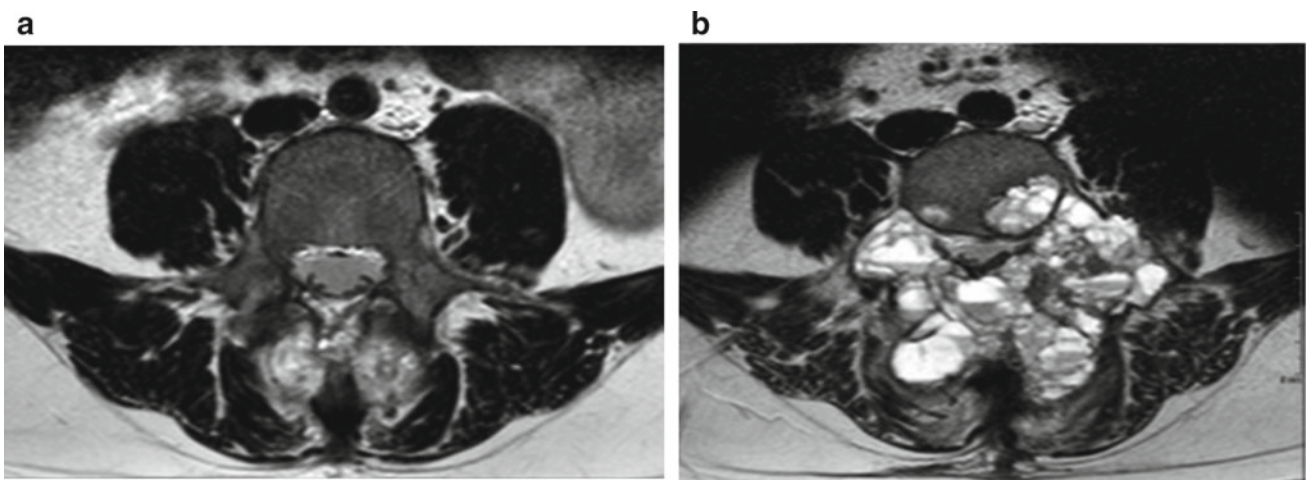


Fig. 23.6 ABC of the fourth lumbar vertebra in a 13-year-old child: **A** Initial lesion is limited to the posterior arch (spinous process); **B** six months later, the lytic process has expanded to the vertebral body

to the blowing aspect of the lesion. Sometimes symptoms appear or worsen during pregnancy. Pathological fractures are quite rare at long bones and more frequent for central ABC than for excentric ABC. Pathological fractures are more frequent at the spinal column. At that location, pain may cause segmental stiffness with scoliosis or torticollis and neurological impairment can appear (45% of cases).

Imaging

Standard radiograph. Maturation stages of ABC. The ABC evolution shows several maturation stages with radiographic typical aspects [2] (Fig. 23.8).

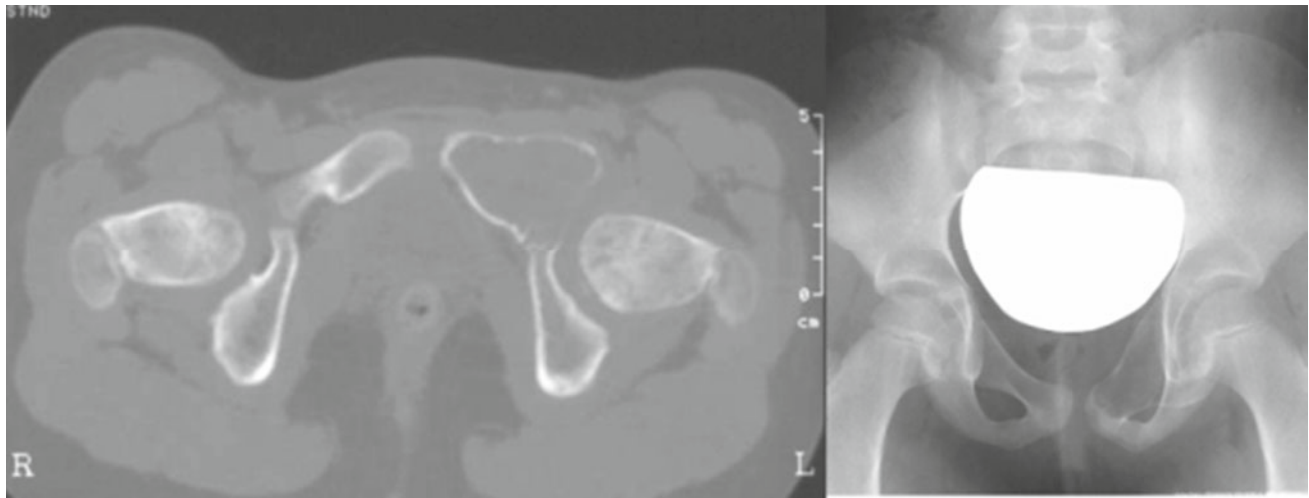


Fig. 23.7 Pelvis ABC

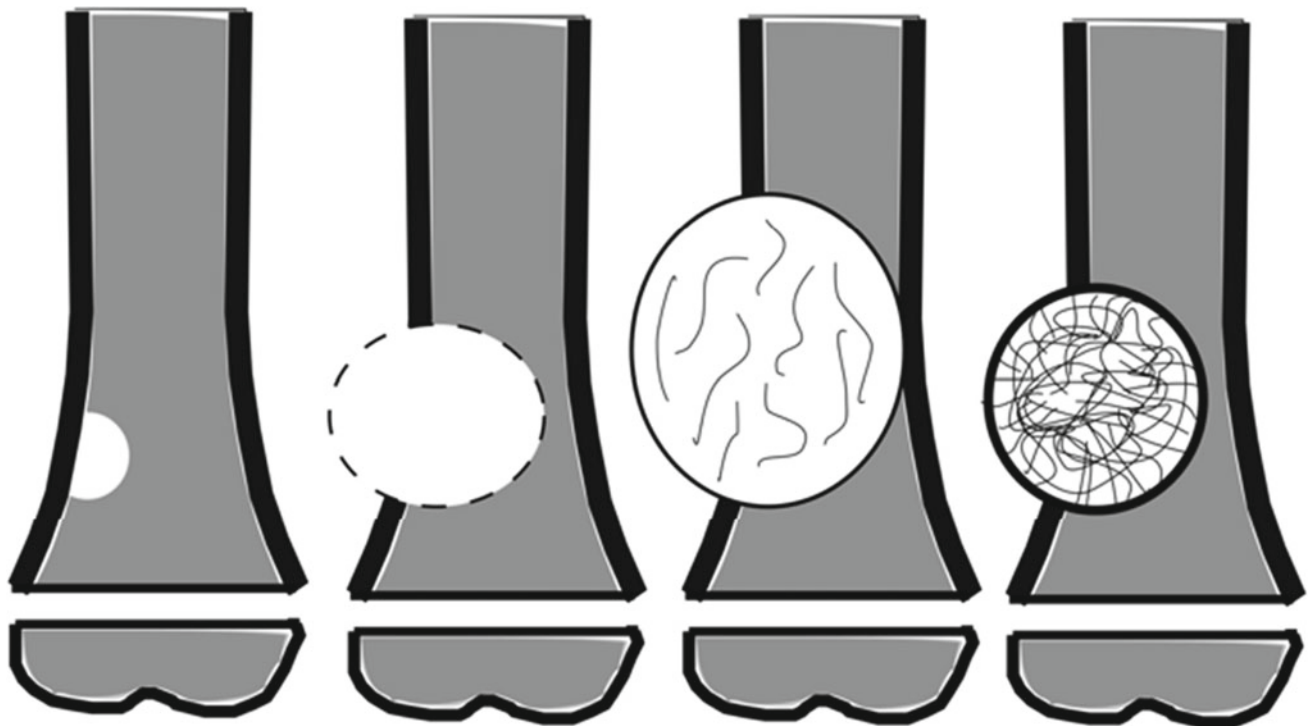


Fig. 23.8 Four evolutive stages of ABC: initial lytic phase, active expansion phase, stabilization phase and healing phase

Early lytic phase: a lytic well-defined area appears often excentric or subperiosteal.

Active expansion phase: it is the typical blowing aspect of ABC that is entered in an aggressive stage (stage 3 of Enneking) (Fig. 23.9). At this stage ABC is difficult to distinguish from a malignant lesion. The periosteum may be lifted with Codman's triangle but no peripheral shell is obvious. The limit between ABC and soft tissue is not clear.

Stabilization phase: a peripheral bony shell appears as well as internal septa giving a "soap bubble" aspect. The periosteum has produced bone that delimits ABC with a thin border. Codman's triangle is often visible at the diaphyseal side of the periosteal lifting.

Late healing phase: progressive ossification of ABC results in a bony dense irregular lesion. The peripheral shell and the septa are enlarged. The border is more clearly



Fig. 23.9 ABC in an active expansion phase; the periosteum is lifted to soft tissue but is not yet ossified; Codman triangles are present at diaphyseal side; at this stage, ABC looks like a malignant tumor

defined. ABC enters into a latent phase (stage 1 of Enneking). At this stage recurrence is not possible.

ABC is usually discovered at expansion or stabilization phases (stage 2 or 3). Healing is usually obtained after treatment but spontaneous healings have been reported (45).

Radiographic Classification of ABC

In 1985, Capanna et al. proposed an interesting classification for the juxtaepiphyseal ABC of long bones (Fig. 8.1.11):

Type I: central ABC, not blowing, metaphyseal or metaphyso-diaphyseal.

Type II: central ABC extending to the whole breadth of the bone, with the blowing aspect. It is usually observed at metaphysis or metaphyso-diaphysis of small diameter long bones (fibula, radius, ulna) or at the flat bones.

Type III: excentric ABC, intraosseous, often metaphyseal.

Type IV: subperiosteal ABC, extraosseous, often diaphyseal (rare).

Type V: subperiosteal ABC intra- and extra-osseous, often metaphysodiaphyseal.

The “fallen fragment sign” is never present in the ABC following a fracture, at the difference of simple bone cyst.

Angiography

This radiographic study shows usually venous malformation with persistence of the contrast inside ABC. No arterial malformation or arteriovenous fistula is observed. This is usually performed for therapeutic or pre-operative embolization but not for diagnosis.

Tomodensitometry

Fluid-fluid levels may be visible at CT-scanner and are due to blood sedimentation (serum is separated from blood cells). CT-scanner may show the limit of the blowing expansion and may show the possible cortical destruction.

Magnetic Resonance Imaging

The liquid component is high signal T2 and low signal T1. T2-weighted MRI mainly shows fluid component and fluid-fluid levels. T1-weighted MRI better shows bony cortex. Fluid-fluid levels are present in 66–84% of the cases but presence of fluid-fluid levels is not specific to ABC. It is only due to the presence of liquids with different densities. In case of blood, the cellular component has a more important density than the plasma, so the plasma constitutes the upper layer. These levels may change their orientation following the patient position.

Other lesions such as fibrous dysplasia, simple bone cyst, malignant fibrous histiocytoma, osteosarcoma may also present fluid-fluid levels.

In ABC fluid-fluid levels are mainly present at the expansion or stabilization stages but absent at the early stages. The presence of multiple cavities separated by septa is much more constant (100% of cases).

Technetium Bone Scan

The bone scan will show hyperfixation at the border of ABC whereas the center will fix moderately or not at all.

Differential Diagnosis

The differential diagnosis has to be made with other metaphyseal lesions:

Simple Bone Cyst

The simple bone cyst is more often central [3]. It involves mainly the proximal humerus or proximal femur. It is filled with serum and no blood, except in cases of fracture or microfracture. Fluid-fluid levels are possible but rarer. Septa are absent, except after fracture. Epiphyseal involvement is not frequent. Evolution is slower. The blowing aspect is rare but possible after multiple fractures and a fibrous dysplasia lesion involvement of the callus by the cyst.

Eosinophilic Granuloma

Isolated eosinophilic granuloma could be confused with ABC. Its borders are often irregular and blurred. In the multiple disease, several osteolytic lesions are present (Langerhans histiocytosis).

Non Ossifying Fibroma

Non ossifying fibroma or cortical defect is the most frequent benign tumor. The cortex is the point of birth of the lesion. It is surrounded by a dense line and is multilocular with multiple septa separating lobules. The diagnosis is usually easy.

Fibrous Dysplasia

This may mimic ABC. The borders are often blurred and radiolucency is typically veiled by a “ground glass” opacity due to the delicate trabeculae of woven bone. The MRI will show hyposignal T1 and T2 and should distinguish the two lesions.

Chondromyxoid Fibroma

This is usually also located in the metaphysis and occurs within the same age range. It is often excentric. Its borders are polycyclic and well demarcated. MRI shows no fluid-fluid levels and no septa.

Giant Cell Tumor

Giant cell tumor is rare in open-physis patients as it is usually a tumor of adults. It is located in the metaphysis and can extend to the epiphysis.

Telangiectasic Osteosarcoma

Radiographic and MRI features may be very similar compared with ABC [4]. Fluid-fluid levels are frequent as well as the septa. It is due to this difficult distinction that a biopsy is mandatory to give the diagnosis of ABC.

Pathology

Gross Anatomy

The tumor is surrounded by an intact periosteum and soft tissues are not invaded. In case of opening of an ABC, hemorrhage may occur with venous blood that can persist as long as the time of the surgery. ABC is filled with a spongy tissue constituted by cavities surrounded by thick septa. The cavities are filled with blood sometimes clotted. In the solid variant only fleshy tissue is present.

Histological Aspect

Three main components are present in ABC (Fig. 23.10) [2].

Cellular Component

The cellular component includes stromal cells and giant cells. Giant cells are easily detected as they contain several nuclei. Stromal cells are mononuclear, with a round or oval nucleus and a scarce or absent intercellular matrix.

Fibrillar Component

The fibrillar component is comprised of fibroblasts and collagen. Fibroblasts are elongated cells characterized by an oval nucleus and a spindle cytoplasm, embedded into a collagenous extracellular matrix. Dense collagen is occasionally present, with abundance of enlarged thick collagenous fibers.

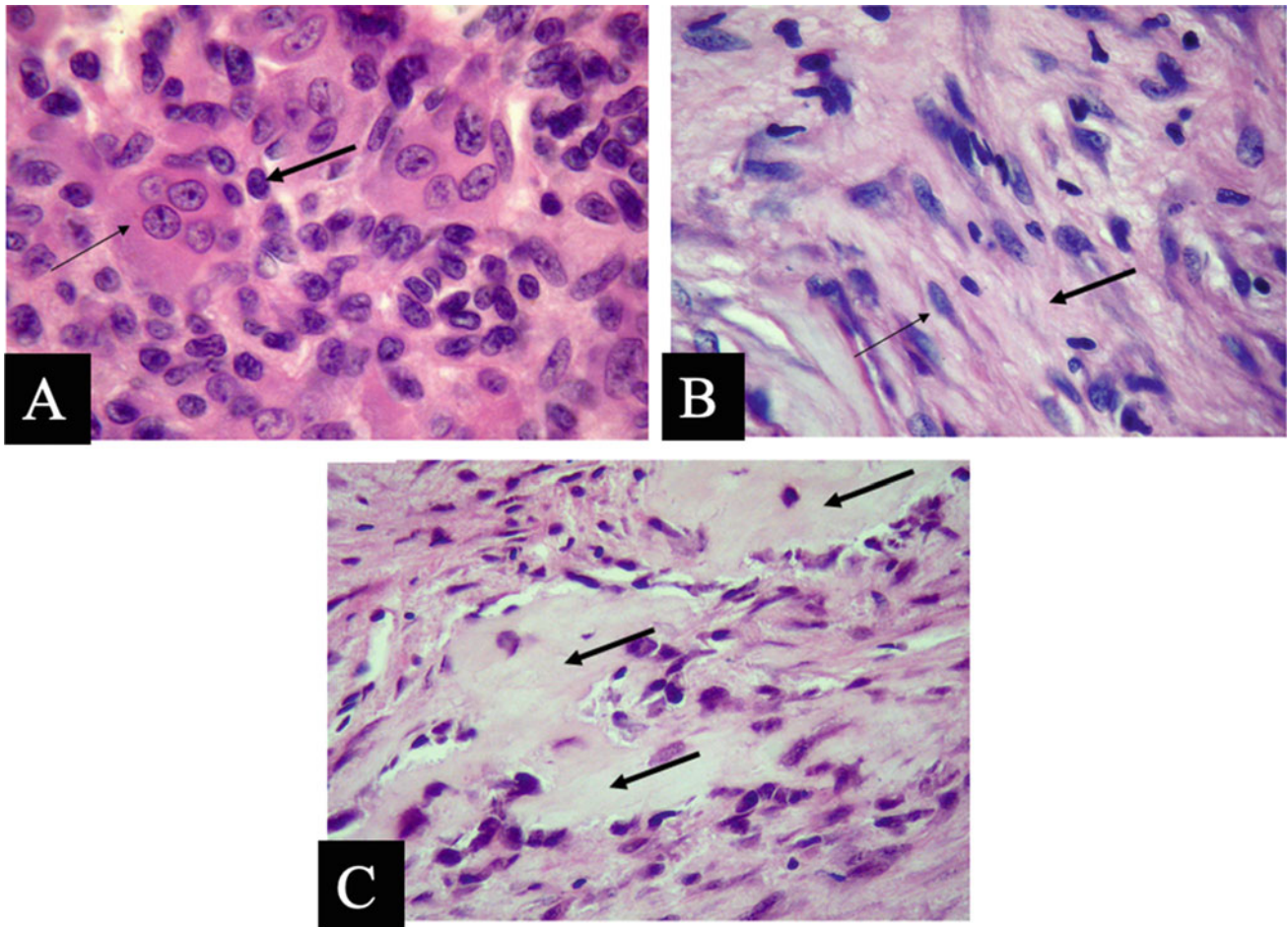


Fig. 23.10 Three histological components of ABC: **A** cellular component: stromal cell (small large arrow) and giant cells (large small arrow); **B** fibrillar component: fibroblasts (small arrow) and collagen (large arrow); **C** osteoid component: osteoid tissue (arrow) and osteoblasts

Osteoid Component

The osteoid component is made of organic bone matrix deposited by osteoblasts.

- **Immunostaining:** A cluster of differentiation 68 (CD68) is a glycoprotein that binds to low-density lipoprotein and is expressed only on macrophages. Commercially available antibodies (anti-CD68) are available to detect macrophage (giant cells and stromal cells). Proliferating cell nuclear antigen (PCNA) is an antigen that is expressed in the nucleus during the DNA synthesis phase of the cell cycle. Commercially available antibodies (anti-PCNA) are able to detect proliferating cells.

Natural History

Spontaneous Healing

Cases of spontaneous healing have been reported. These cases occur most often in adults and in the pelvic location. Healing after biopsy has also been described (Fig. 23.11).

Fracture Risk

The fracture risk is minimal for ABC of long bones by comparison with simple bone cysts. It is the central blowing ABC that may lead to a fracture. In the spine, vertebral fracture is more frequent.

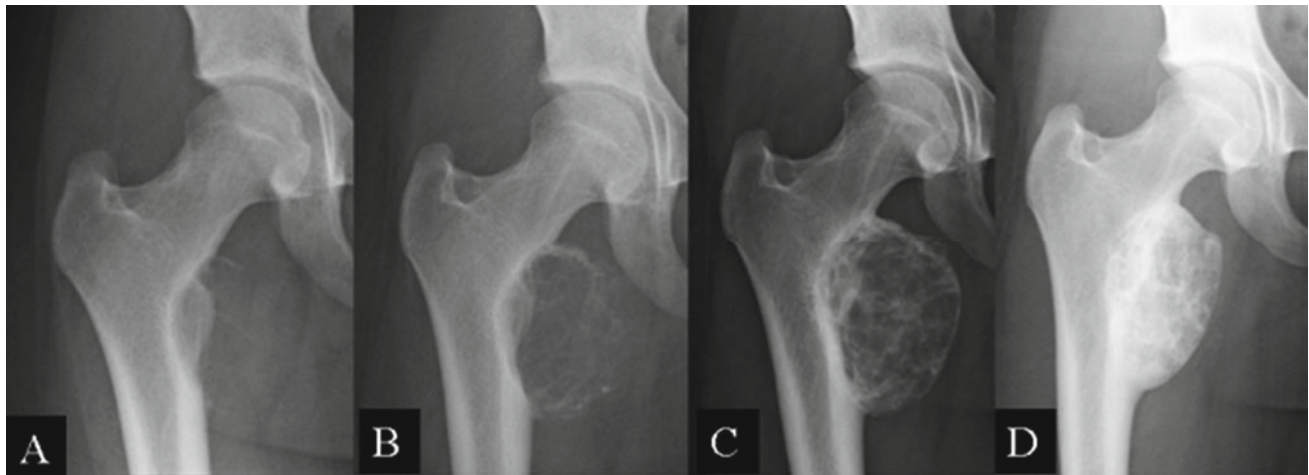


Fig. 23.11 Evolution of an ABC

Malignant Transformation

Cases of malignant transformation have been reported [5]. Brindley et al. reported two cases of malignant transformation to telangiectasic osteosarcoma and to fibroblastic osteosarcoma five years and 12 year after curettage of an ABC. Kyriakos et al. reported a case of transformation to Pleomorphic osteosarcoma after multiple curettage of an ABC. Anract et al. reported a case of malignant transformation to a malignant fibrous histiocytoma at the site of a previously treated ABC.

A case of pelvic ABC has been described with lung, liver and kidney metastasis.

Growth Perturbation

In the growing child, ABC may alter the bone growth. When the ABC is close to the growth plate (juxtaepiphyseal ABC) it may invade it (23% of the cases). In 60% of the cases of invasion, a premature epiphysiodesis occurs with leg length discrepancy or axial deviation.

Treatment

First of all, it must be kept in mind that ABC is a benign lesion. To avoid confusion with a malignant tumor, a biopsy is mandatory.

Radiotherapy

Radiotherapy has been shown to be efficient in the treatment of ABC but numerous complications may occur, the most

severe being the risk of radio-induced sarcoma. This sarcoma appears two to 28 years after irradiation. Other reported complications are vertebral fractures with possible neurological impairment, growth plate destruction, gonadal lesions, and femoral head necrosis.

This treatment is only used in exceptional cases where surgical access is very difficult.

Selective Embolization

Embolization may be used as single treatment or as pre-operative treatment to decrease post-operative bleeding.

Cases of healing after embolization used as a unique treatment have been reported for ABC involving cervical vertebra, thoracic vertebrae or the sacrum [11]. De Cristofaro et al. obtained a healing in 17 cases out of 19 (89%). Ossification occurred very slowly and after more than one year. Embolization has to be repeated (two to four times). The main danger is embolization of vital arteries with ischemia of visceral or nervous structures (spinal cord). Somesthetic evoked potential are mandatory during the procedure. Sometimes embolization may not be performed as no afferent artery is evident.

Ethibloc® Injection

Ethibloc is an emulsion of zein (a corn protein), alcohol, oleum papaveris, propylene glycol, and a contrast medium. It induces fibrosis and secondary ossification of ABC. It is introduced into the cyst under fluoroscopy or CT-scanner. George et al. obtained a complete healing in 58% and a partial healing in 35.5% of cases in a series of 33 patients [6]. Following injection an inflammatory response occurs

with pain and sometimes fever. The major risk of Ethibloc is the risk of multiple pulmonary embolisms if the fibrosing agent goes intramedullary. Topouchian et al. reported a high complication rate in his series: severe pulmonary embolism (7%), early aseptic fistulization needing surgical debridement and curettage (27%), transient inflammatory reaction with temperature (33%). A case of fatal embolism in the vertebrobasilar system has been described after Ethibloc injection into a cervical vertebra. For Mascard and Adamsbaum, Ethibloc injection is a safe, efficient and non invasive treatment for ABC if precautions are respected. ABC opacification has to be performed and in case of venous drainage the use of Ethibloc is contra-indicated. The filling of ABC has to be slow and not complete. A preventive antipyretic treatment has to be given. In view of the possible complications numerous centers have forsaken this technique.

Alcohol

An alternative to Ethibloc is absolute alcohol which has good fibrosing properties. It is very cheap and easily available. It is used at 95% or 100% strength. It acts by injuring the vascular endothelium and by denaturing the blood proteins leading to thrombosis. Injections are performed by transcortical puncture under general anesthesia and fluoroscopy with two or three 18 or 19 gauges needles depending on the size of the cyst. Sclerotherapy is performed with 5 or 10 ml of alcohol in each needles without increase of the pressure. The results seems to be comparable to Ethibloc without local inflammatory reaction.

Cryotherapy

Cryotherapy may be used alone or as adjuvant treatment to decrease the recurrence rate. The surgical technique consists of curettage of the cyst followed by one or more cycles of freeze-thaw by introducing liquid nitrogen into the cyst. It is also possible to produce a liquid nitrogen spray with a machine. A temperature less than -50°C in the cavity is considered as lethal for tumoral residual cells. Post-operative complications such as pathological fracture or soft tissue healing problems are possible.

Surgical Curettage

In the literature review, Schreuder et al. reported the following recurrence rate after surgical treatment (all recurrences occurring in the two post-operative years): 14.2% for curettage and radiotherapy

- 30.8% for curettage and bone grafting
- 12.8% for curettage and cryotherapy
- 7.4% for marginal resection
- 0% after wide resection
- 11.4% for radiotherapy alone.

Only a wide resection may guarantee the healing without recurrence but at what price? To avoid surgical aggressiveness, numerous minimal invasive techniques are now available [6–9].

Percutaneous Injections

This is demineralized bone matrix mixed with bone marrow (Fig. 23.12). This treatment is interesting because is it non invasive. The bone matrix has to be demineralized to acquire induction power. Bone marrow aspirated at the iliac crest may bring osteoblast progenitor cells. Muschler et al. recommends aspirating no more than 2 ml of bone marrow at the same puncture site to maximize the number of osteoblast progenitor cells and avoid contamination by peripheral blood. The osteogenic power of bone marrow is increased by mixing it with the demineralized bone matrix (synergetic effect). The particle size of demineralized matrix has been decreased to be able to inject it with a normal syringe without any surgical approach.

Calcitonin and Methylprednisolone

Numerous cases of healing after percutaneous injections of calcitonin and methylprednisolone have been reported. Calcitonin acts as osteoclastic inhibitor whereas corticoid acts as angiostatic. Szendroi et al. recommend injections of calcitonin alone in case of hypovascularized ABC. They obtained healing in six out of seven cases. The recurring cases were hypervascularized ABCs.

Calcium Sulfate

An injectable type of calcium sulfate cement has been used by Clayer. The cement is fully resorbed in eight weeks. The first response is a bony peripheral shell followed by a progressive ossification of the cavity. Two patients out of 15 had a recurrence.

Sclerotherapy by Polidocanol Injection

Intralesional 3% polidocanol administration may be used percutaneously under fluoroscopy. In the Rastogi series, the

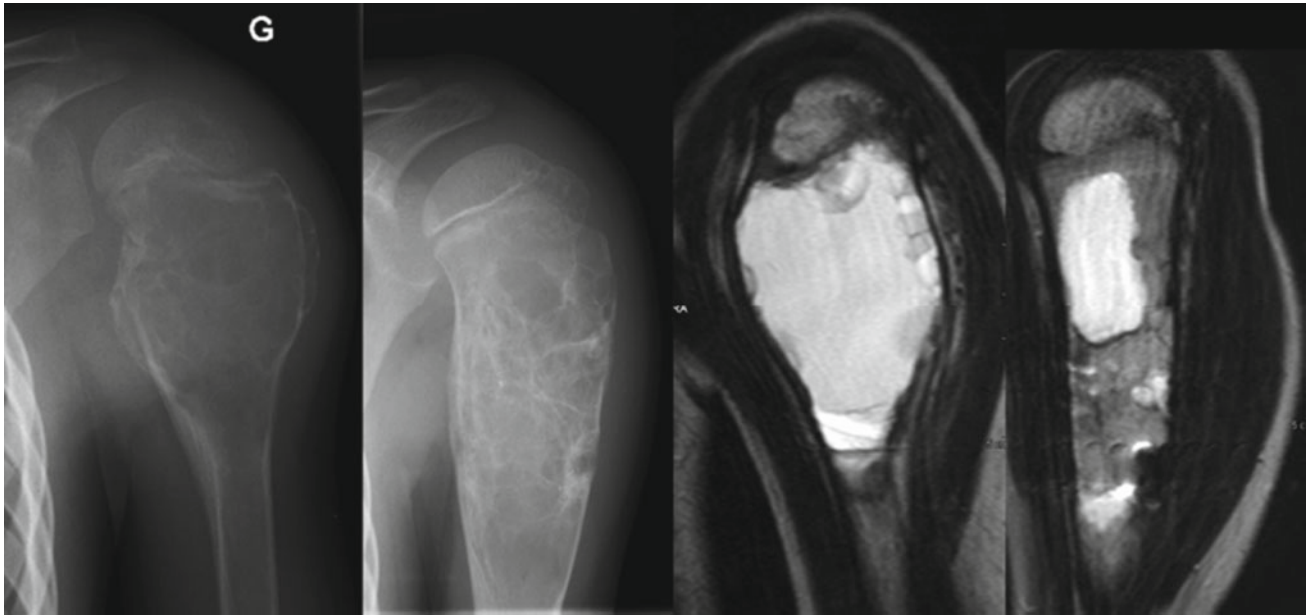


Fig. 23.12 Percutaneous injection of demineralized bone matrix mixed with bone marrow: **A** voluminous ABC of proximal humerus in a 7-year-old boy; a swelling is visible and the child has pain; right: two years after percutaneous injection of demineralized bone matrix

mixed with bone marrow, swelling has disappeared and the child is asymptomatic; ABC has become latent, only small cavities are persisting

mean number of injections was three. This technique may be used for surgically inaccessible sites [10].

Factors for Recurrence After Treatment

Different factors may be associated with a higher recurrence rate after treatment.

Young Patients

Patient less than 15 years of age with open growth plate have more recurrence risk. Cottalorda et al. in a retrospective review found that the recurrence rate was not increased in children of less than five years of age.

Male Sex

The male sex could be associated with greater recurrence.

Location

Central locations are prone to recurrence (Capanna types I and II)

Stage

Aggressive (Enneking stage 3) or active ABC (stage 2) may recur after treatment whereas latent ABC (stage 1) do not recur.

Histology

Mitotic index of 7 or more is associated with a important recurrence rate (Fig. 23.13). Freiberg et al. did not observe this correlation.

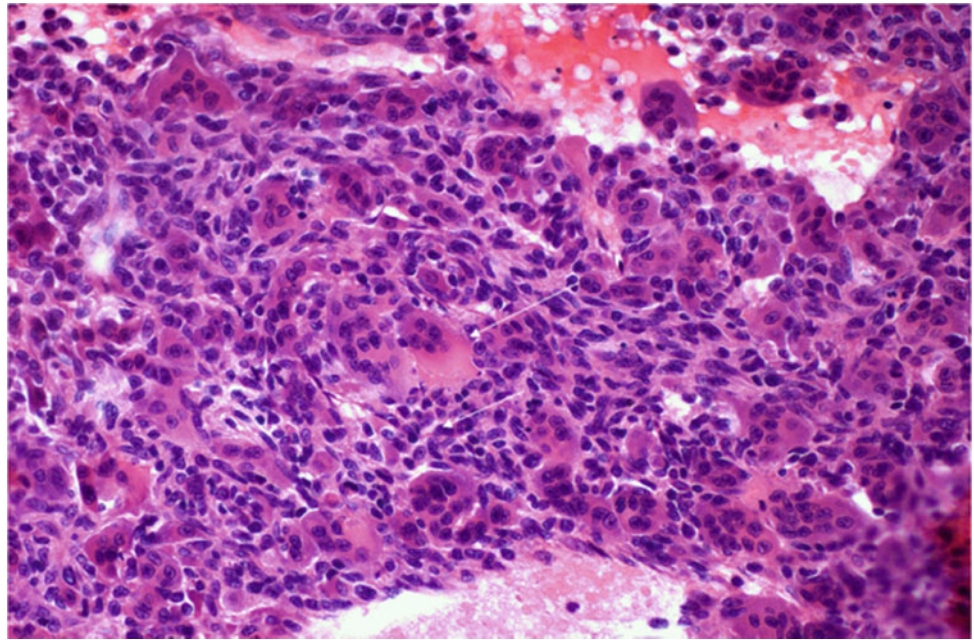
The cellular component is a factor of bad prognosis if important, whereas osteoid and fibrillar components are factors of good prognosis. A healing index may be calculated by histomorphometry. It corresponds to addition of osteoid (O) and fibrillar (F) components divided by the cellular component (C). If the ratio $(O + F)/C$ is equal or more than 1:2 ABC is prone to heal [2].

Abundant immunostaining with CD68 (macrophage marker) is a factor of bad prognosis.

Conclusion

Aneurysmal bone cyst is a benign lesion that develops commonly in children and young adults. The usual symptoms are pain and swelling in cases of blowing tumors.

Fig. 23.13 Prognostic factors: typical picture of a ABC prone to recur after treatment; it is mainly compound of cellular component (giant cells and stromal cells (white arrows); osteoid tissue and fibrous tissue are scarce



Diagnosis may be probable with a standard radiograph but magnetic resonance imaging is more useful in the differential diagnosis. A biopsy is mandatory as aneurysmal bone cyst may be secondary to a malignant lesion such as the telangiectatic osteosarcoma. Cases of spontaneous healing after biopsy have been reported but remain rare. Most of the cases require surgical treatment. As it is a benign lesion, invasive treatment and radiotherapy has to be avoided. Numerous minimal invasive treatments are now available.

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