Pathological fractures in children can occur as a result of a variety of conditions, ranging from metabolic diseases and infection to tumours. Fractures through benign and malignant bone tumours should be recognised and managed appropriately by the treating orthopaedic surgeon. The most common benign bone tumours that cause pathological fractures in children are unicameral bone cysts, aneurysmal bone cysts, non-ossifying fibromas and fibrous dysplasia. Although pathological fractures through a primary bone malignancy are rare, these should be recognised quickly in order to achieve better outcomes. A thorough history, physical examination and review of plain radiographs are crucial to determine the cause and guide treatment. In most benign cases the fracture will heal and the lesion can be addressed at the time of the fracture, or after the fracture is healed. A step-wise and multidisciplinary approach is necessary in caring for paediatric patients with malignancies. Pathological fractures do not have to be treated by amputation; these fractures can heal and limb salvage can be performed when indicated.

Introduction

A pathological fracture should be suspected in a paediatric patient when there is a fracture associated with minimal trauma, when the location of the fracture is unusual or when an abnormal process in the bone is seen in the radiographs. Intrinsic processes, such as changes in the mineral density of the bone from bone tumours (both benign and malignant), diseases like osteogenesis imperfecta, or infection; and extrinsic processes, such as internal fixation, biopsy tracts and radiation, can cause changes to the normal biomechanics of bone. The altered strength of the bone and the load applied are the factors that will determine the risk of a pathological fracture. Fracture-risk indices based on lesion size alone also do not take into account the compensatory remodeling of the host bone. The downside is additional radiation exposure by the CT scan of the lesion and its contralateral bone for comparison. When used in conditions that affect several bones, such as osteogenesis imperfecta, Ollier disease or polyostotic fibrous dysplasia, the prediction will not be as reliable as it is in normal bone. Leong et al prospectively analysed the protocol described by Snyder et al and, although they were unable to evaluate the sensitivity of the quantitative CT-based predictions of fracture risk, they were able to evaluate prospectively the specificity of the protocol as compared with the criteria predicated on the size of the lesion as measured on plain radiographs. This avoided surgery that would most likely have been unnecessary in 30 patients predicted to be at high risk for fracture on the basis of the lesion size, but not.

Characteristics such as pain, size of the lesion (> 2.5 cm in width or > 3.5 cm in length) and cortical destruction (≥ 50%) have been found not to be independently predictive of fracture risk. Snyder et al used CT with ratios of bending and torsional rigidities in order to predict fractures in benign skeletal lesions, by analysing changes in both the material properties of the bone and the cross-sectional geometry of the lesion, and comparing with the contralateral unaffected bone. This allows quantification of the mechanical properties of the bone at the lesion site and determination of the reduction in the load-carrying capacity of the bone. Fracture-risk indices based on lesion size alone also do not take into account the compensatory remodeling of the host bone. The downside is additional radiation exposure by the CT scan of the lesion and its contralateral bone for comparison. When used in conditions that affect several bones, such as osteogenesis imperfecta, Ollier disease or polyostotic fibrous dysplasia, the prediction will not be as reliable as it is in normal bone. Leong et al prospectively analysed the protocol described by Snyder et al and, although they were unable to evaluate the sensitivity of the quantitative CT-based predictions of fracture risk, they were able to evaluate prospectively the specificity of the protocol as compared with the criteria predicated on the size of the lesion as measured on plain radiographs. This avoided surgery that would most likely have been unnecessary in 30 patients predicted to be at high risk for fracture on the basis of the lesion size, but not.
on the CT protocol. They suggested that the CT method should be used as an objective tool that can help patients and physicians to come to the best decision, which also depends on the patient’s activity level and individual preferences. Pireau et al. found the bone cyst index (BCI), measured on T1-weighted MRI, to be a reliable intra- and interobserver tool to predict pathological fractures in unicameral bone cysts (UBCs), when compared with the bone cyst diameter and the cortical thickness. The BCI is obtained by dividing the cyst area by the diameter of the bone cyst diameter and the cortical thickness. The BCI is measured on T1-weighted MRI, to be a reliable intra- and interobserver tool to predict pathological fractures in unicameral bone cysts (UBCs), when compared with the bone cyst diameter and the cortical thickness. The BCI is obtained by dividing the cyst area by the diameter of the bone.11 Although some can heal spontaneously, the majority will become inactive or latent and persist after the natural history of a UBC is to stabilise in size, and, with the natural growth of the bone, drift away from the diaphysis.11 Although some can heal spontaneously, the majority will become inactive or latent and persist after 12 years of age.11 Approximately 75% of patients with a UBC present with a pathological fracture. A UBC is usually the most common cause of pathological fracture in children with a bone lesion.12,13 The most common sites of pathological fractures due to UBC are the proximal humerus followed by the proximal femur.13,14

The priority for treatment of a UBC is to treat the fracture first and then the lesion. Fracture treatment is achieved with simple immobilisation of the extremity for four to six weeks (Fig. 1). This applies to fractures in non-weight-bearing areas that are minimally displaced and stable. The majority of the fractures heal, but the UBC will persist in between 20% and 50% of cases.13 When the fracture is unstable or is in a weight-bearing bone, there may be need to proceed to surgery sooner, for both fracture fixation and treatment of the cyst.

Although cysts located in the proximal femur are more likely to heal with a fracture than cysts in the proximal humerus,13 UBCs of the proximal femur can cause varus deformity and even avascular necrosis if not treated properly. Dormans and Pilli described a classification system to guide treatment of lytic lesions of the femoral neck depending on the location and size of the lesion. This classification can be used for UBCs, as well as for other benign

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**Table I.** Common predisposing benign and malignant lesions by age (adapted with permission from Arkader A, Dormans JP. Pathologic fractures associated with tumors and unique conditions of the musculoskeletal system. In: Beaty JH, Ska ggs DL, Flynn JM, Waters K, eds. Rock-wood and Wilkins’ fractures in children. Seventh ed. Philadelphia: Lippincott Williams & Wilkins, 2010:120–191)

| Age (yrs) | Benign lesions | Malignant lesions |
|----------|----------------|------------------|
| 0 to 5   | Osteomyelitis   | Metastatic tumours (neuroblastoma, Wilm’s tumour) |
|          | Eosinophilic granuloma | Leukaemia |
|          | Hand-Schuller-Christian disease | Ewing sarcoma, Fibrosarcoma |
|          | Osteochondroma | Eosinophilic granuloma/ Letterer-Siwe disease |
|          | Fibrous dysplasia | |
|          | Enchondroma/Ollier disease | |
|          | Neurofibromatosis/Congenital pseudarthrosis of the tibia | |
| 5 to 10  | Unicameral bone cyst (UBC) | Leukaemia |
|          | Aneurysmal bone cyst (ABC) | Osteogenic sarcoma, Ewing sarcoma |
|          | Nonossifying fibroma (NOF) | |
| 10 to 20 | UBC              | Lymphoma, Osteogenic sarcoma, Ewing sarcoma |
|          | ABC              | |
|          | NOF              | |
|          | Osteochondroma   | |
|          | Fibrous dysplasia | |
|          | Chondroblastoma | |
|          | Giant cell tumour | |
|          | Osteoid osteoma  | |

**Initial evaluation**

In children, most pathological fractures are due to benign bone tumour or tumour-like conditions, metabolic diseases and infection. A malignancy, however, can sometimes be the cause and should always be kept in mind.

A detailed history and thorough physical exam is paramount in the evaluation of any patient with a pathological fracture. Certain bone tumours are more common at specific ages (Table I). A detailed pain profile of symptoms prior to the fracture should also be obtained in order to further characterise the lesion. Plain radiographs are the initial imaging modality of choice. At least two orthogonal views of the affected bone should be obtained. The location of the lesion in the bone, its size, growth pattern, matrix, periosteal reaction and the number of lesions are all important factors in the differential diagnosis of bone lesions.

Almost all lesions will require biopsy at some point. Determining the best time to biopsy can sometimes be a difficult task, and it will be specific to different lesions, as some can wait until the fracture is healed, some can be simultaneous with fracture care and others are required for diagnostic purposes. When a lesion appears malignant on radiographs, the biopsy should not be delayed and should be performed by the treating surgeon, with proper training in orthopaedic oncology and who will be responsible for the final care of the patient, since a poorly performed biopsy can lead to catastrophic consequences. Cultures should always be obtained along with the biopsy to rule out an infectious process.
lytic lesions. We have updated this classification to include lesions of the proximal femur that extend into the diaphysis, where we recommend diaphyseal fixation (Fig. 2).

The objective of treatment is to eradicate the cyst and prevent future fractures and deformities, especially in active cysts adjacent to the physis. Current treatment options for UBCs typically include a combination of some of the following: decompression or mechanical disruption of cyst wall, injection (steroids, bone marrow aspirate, demineralised bone matrix or bone-substitutes) and fixation for structural support when in a weight-bearing bone.

There is a current trend towards agreement that percutaneous techniques that create mechanical disruption of the cyst membrane, concomitant migration of bone marrow cells for osteoinduction, and the use of calcium sulphate substitute for osteoconduction yield better outcomes when compared with other treatments. The technique consisting of percutaneous intramedullary decompression, curettage and grafting with calcium sulphate pellets, as described by Dormans et al.,17 yielded higher rates of healing when compared with previously published data. Hou et al16 described patients treated with minimally invasive curettage, ethanol cauterisation, disruption of the cystic boundary, insertion of a synthetic calcium sulphate bone-graft substitute, and placement of a cannulated screw to provide drainage, and reported that these patients had the highest rate of healing and shortest time to union when compared with three other techniques. Canavese et al18 compared patients treated with one of autologous bone marrow injection, methylprednisolone injection or percutaneous curettage alone for treatment of UBC, and described that percutaneous curettage alone had better results (healing in 70%) when compared with the other two treatment types (41% and 21% for methylprednisolone and bone marrow aspirate, respectively). Wright et al19 performed a randomised clinical trial comparing rates of healing in UBC between those treated with intralesional injections of bone marrow aspirate and those treated with methylprednisolone. They reported that 16 of 38 UBCs (42%) treated with methylprednisolone healed, compared with nine of 39 (23%) of those treated with bone marrow aspirate only; these rates lower than previously published studies on both treatments (100% by Lokiec et al20 67% by Yandow et al21 and 60% by Scaglietti et al22). Although scraping the cyst was part of the trial protocol, the completeness of this step was not reported. A recent report on the long-term (seven-year) follow-up of 23 of the patients in the

Fig. 1

Radiographs of a 16-year-old male patient, a) at presentation, showing a pathological fracture of the left humerus through a unicameral bone cyst, b) at two months after treatment in a sling with interval healing, c) and d) after percutaneous curettage and bone grafting, respectively, and e) at two years post-operatively, showing a healed cyst.
study by Wright et al¹⁹ demonstrated that although the distance from physeal scar had increased (p < 0.0001), reduction of the cyst area and overall cyst healing had not changed since completion of the trial (p = 0.06 and p = 0.5, respectively).²³ Although 78% (18 of 23) of the growth plates were closed, none of the cysts had completely healed.

While there is an apparent high healing rate, there is still a rate of recurrence/persistence that ranges from 8% to 30% with these percutaneous techniques.¹⁶,¹⁸,²⁴ These patients have a slightly higher chance of pathological fracture and are typically submitted to a repeat procedure until healing. The success rate increased to 94% after a repeat surgery, reaching a 100% healing rate in patients undergoing more than two repeated surgeries.²⁴

**Non-ossifying fibroma**

Non-ossifying fibromas (NOFs), also known as fibrous cortical defects or nonosteogenic fibromas, are the most common benign bone lesion in children. It is estimated that between 30% and 40% of people aged < 20 years have a NOF, and these are mostly asymptomatic.²⁵,²⁶ The radiological appearance of an NOF is a lytic, well-defined lobulated lesion, located eccentrically in the metaphysis. Multi-locular appearance, ridges in the cortical wall, sclerotic scalloped borders and erosion of the cortex are frequent findings.²⁵,²⁷ There is no periosteal reaction in the absence of a pathological fracture. Most are in the distal ends of long bones. A frequent location is the distal femur at the origin of the medial head of the gastrocnemius. The natural history of NOF is to gradually disappear with age; they are expected to start regressing within years after its recognition, and usually the disappearance may take several years.²⁸ In a few cases a NOF can cause pain and swelling, especially associated with athletic activity.

NOFs are typically not associated with pathological fractures²⁷,²⁹; however, when fractures do occur, it is usually in larger NOFs and almost always in the lower extremity.¹³,²⁷ Ortiz et al¹³ reported that of the 17 patients with pathological fractures through a NOF in his series, 16 of the fractures were in the lower extremity. Arata et al²⁷ reported that if a NOF involved > 50% of the transverse diameter of the bone, or if it measured > 33 mm in length, there was an increased risk of pathological fracture. However, Easley and Kneisl³ showed that 59% of their cases of large NOF exceeded these threshold measurements and also did not fracture. They suggested that the majority of patients with large NOFs can be monitored without intervention, as there is evidence to support spontaneous resolution of the majority of these lesions.³ Small NOFs can also have pathological fractures, or even microfractures and stress fractures. Shimal et al³⁰ described five cases of smaller NOFs associated with fatigue-type stress fractures of the involved bone and lesion, stating that the NOF introduced an element of insufficiency to those stress fractures. They also suggested that the two conditions were inter-related since NOFs are said to occur at the site of insertion of a tendon or ligament exactly where abnormal muscular forces initiating a stress fracture are maximally applied.³⁰

Treatment of a pathological fracture of an NOF generally follows the same principles used in the treatment of UBCs. The order of priority is the fracture first, and then the lesion, if necessary. Fractures in NOFs are known to have the best
outcome of all pathological fractures. The fracture usually will heal with conservative treatment consisting of closed reduction, if necessary, and casting, since most are in the lower extremity. Fractures will usually heal in four to six weeks, but sometimes can take up to 12 weeks. The NOF will also heal in almost all cases, although it can take much longer to resolve completely, thus treatment of the lesion is not usually recommended when the fracture is already healed in an asymptomatic patient. Cases that require surgical treatment at the time of fracture are rare, these are usually the ones that cannot be adequately closed, reduced and immobilised. In these cases the NOFs should be curetted and bone grafted at the same time an open reduction and internal fixation is performed. A period of protected weightbearing is typically warranted until the fracture heals.

Fractures that were treated by conservative means and healed but have persistent pain are also atypical and can be treated with elective biopsy, curettage and bone grafting alone. Enlargement of the lesion is not expected after the fracture heals, and a different process should be suspected if this occurs.

**Aneurysmal bone cyst**

Aneurysmal bone cyst (ABC) is a benign but locally aggressive tumour. The prevalence of primary ABC is about 2% of all benign tumours of bone. A total of 80% of cases will be diagnosed in patients younger than 20 years of age. Usually patients will present with local pain that has been present for several weeks or months, or a pathological fracture. Rapid growth of the lesion can occur, mimicking a malignancy. On radiographs, an ABC is an expansile, lytic lesion that elevates the periosteum, contained by a thin shell of cortical bone. It can have well-defined margins or a permeative appearance, resembling a malignancy. Common locations include the femur, tibia, fibula, humerus and the spine. MRI, typically, will show a fluid-fluid level, depicting the multi-loculated cavities filled with fluid. ABCs can sometimes be secondary to other primary benign lesions or malignant bone tumours.

The natural history of ABCs was described by Dabska and Buraczewski based on radiology. They divided the lesions into four phases: 1) the initial phase, described as osteolysis of the marginal part of the bone; 2) the growth phase, characterised by the progressive destruction of bone; 3) the stabilisation phase, defined by the classic ABC appearance: an expansile lesion with a distinct bony shell and osseous septations; and 4) the healing phase, where progressive ossification of the lesion is obvious and results in a bony mass with a somewhat irregular structure. The real incidence of spontaneous healing is difficult to assess as there are no series evaluating the natural history of ABCs without treatment. In most cases, when an ABC is diagnosed, surgical treatment is recommended. Approximately 36% of patients will present with a pathological fracture, which are usually in active lesions.

**Fibrous dysplasia**

Fibrous dysplasia (FD) is a benign non-hereditary condition that causes destruction of normal bone with fibrous tissue and small, woven spicules of bone. It represents between 5% and 7% of benign bone tumours. The lesions of FD develop during skeletal formation and growth and have an inconsistent evolution. Clinical presentation may occur at any age, however there is an increase in the incidence around 10 years of age. FD can be monostotic or, less commonly, polyostotic. Polyostotic FD can be associated with endocrine dysfunction and café-au-lait spots (McCune-Albright syndrome) or muscular myxomas (Mazabraud syndrome). Both of these syndromes are rare. In a very small subset of patients (0.5%) these lesions can undergo malignant change, typically about 15 years after diagnosis. The radiological appearance of FD varies from the classical lytic, ground-glass appearance and distinct sharp rim on the inner borders to more mature lesions, where there is increased thickness of the reactive rim and the lesion appears denser. Polyostotic lesions are larger and can be associated with deformity, such as coxa vara, shepherd’s crook deformity of the proximal femur and bowing of the tibia. FD can occur in the epiphysis, metaphysis or diaphysis of the bone, with a predilection for the long bones, ribs and craniofacial bones. Lesions are typically discovered due to local pain and/or swelling at the site of the lesion, incidentally or due to a pathological fracture. The natural history of these monostotic lesions is of a significant risk of fracture in the face of limited disease in the proximal femur, whereas its tendency to progress is restricted to a minority of cases. Long-term outcome in non-progressive cases is usually satisfactory, regardless of treatment. Polyostotic FD has a more
reserved prognosis as it is associated with increased deformity and higher number of pathological micro- and macrofractures, even though there is a tendency of stabilisation of the deformities after adolescence.

Pathological fractures can occur in 50% of the patients with monostotic disease, especially in the proximal femur. Lesions that are more likely to remain stable present as heterogeneous lesions in the upper third of the femur or in the midshaft of the tibia. These lesions may convey a significant risk for fracture in the femur, but not in the tibia. Conversely, cervico-diaphyseal lesions in the femur, distal medullary lesions in the tibia, and cystic lesions at any site, may correspond to lesions with a greater tendency to extend and cause secondary events beyond fracture, including deformity, limb shortening, and development of aneurysmal bone cysts. Pathological fractures in FD can be treated conservatively in most monostotic cases, especially if the location is not in the lower limbs and there is no deformity in the affected bone. Femoral diaphysis fractures in younger patients can be treated with traction and subsequent casting. FD will persist after the fracture. The lesion can be addressed later, but a whole body bone scan is recommended to exclude the possibility of polyostotic FD. Lesions that are characteristic and pose no risk of further deformity, especially in the upper extremities can be treated with close observation, but a biopsy to confirm the diagnosis is advised. Polystotic FD patients should see an endocrinologist to rule out associated syndromes. In fractures that require surgical treatment: biopsy, curettage and bone grafting with cortical graft should be performed, as well as internal fixation when warranted. If the lesion of the proximal femur is small it can be treated with cannulated screws or compression screw and side plate, however, the surgeon should be aware that in moderate to severe cases this frequently can lead to a shepherd crook deformity, malunion or fracture below the plate. Due to the abnormal nature of the bone, it may be difficult to get sufficient fixation in the bone with screws and plates alone, especially in cases when a deformity is present. Curettage and use of cortical graft over cancellous graft along with open reduction and internal fixation with an intramedullary rod with fixation is highly recommended to avoid deformity (Fig. 3). Guille et al reported that 18 of 27 patients (66.6%) with fibrous dysplasia and Shepherd’s crook deformity treated surgically by several different methods required repeat operation or casting as a result of recurrence or microfractures, while Yang et al reported no progression in all 14 patients treated surgically by the four-step procedure for the lesion, valgus osteotomy for correction of the deformity and intramedullary nail with neck cross pinning. 

Malignant bone tumours

Patients presenting with a pathological fracture of a primary bone malignancy are rare. These patients encompass approximately between 5% and 10% primary malignancies of bone. Osteosarcoma and Ewing
sarcoma are the most common primary bone malignan-
cies in childhood. Failure to recognise these fractures as
being pathological can lead to inappropriate treatment
and potentially worse outcomes.45

Fractures may occur from minimal trauma or spontane-
ously due to the loss of bone matrix from the high cellular-
ity of the tumour, or secondary to biopsy.43,46 The
incidence of osteosarcoma presenting with a pathological
fracture ranges from 5% to 13%.43,47 Of 397 patients with
Ewing sarcoma, Fuchs et al46 reported a pathological frac-
ture in 35 patients (8.8%). Of these, 14 fractures (40%)
were sustained either before or after initial treatment, while
the remainder sustained a fracture subsequent to radia-
tion.46 Larger, proximal, diaphyseal, lytic and telangiectatic
or fibroblastic osteosarcoma subtypes have been found to
be more prone to fracture.44,47,48

Standard initial management of malignant bone
tumours includes neoadjuvant chemotherapy, after appro-
priate biopsy and staging studies have been performed.
Plain radiographs and MRI of the entire bone are
paramount to assess the primary tumour, evaluate for a
soft-tissue mass, proximity to neurovascular structures and
to rule out skip metastases. A whole body nuclear medi-
cine bone scan is necessary to rule out bony metastases
and a CT of the lung to rule out pulmonary metastases.

Patients presenting with a fracture require immobili-
sation during this initial phase of treatment. Cast
immobilisation, traction or external fixation can be used
to stabilise the fracture,49 based on the location of the
lesion and type of fracture (Fig. 4). The use of external
fixator pins or traction pins requires careful planning to
ensure the pins are placed away from the tumour to
reduce the risk of potential pin site and/or soft-tissue
seeding. Protected weightbearing or cast immobilisa-
tion after bone biopsy may also be necessary. Fre-
quently, malignant tumours have a large soft-tissue
component and this can be sampled.

Pathological fractures have shown potential to heal
while on neoadjuvant chemotherapy. Jaffe et al50
described 13 patients with pathological fractures, 11 of
which healed while on chemotherapy. Scully et al51
showed that fractures that healed had increased necrosis
after final histopathologic analysis. Fractures may also
occur while on chemotherapy perhaps due to increased
tumour necrosis.

Limb salvage surgery is the preferred treatment for
malignant bone tumours in paediatric patients at most
institutions; however, amputation still plays a role, espe-
cially when a satisfactory surgical margin cannot be
obtained, or the residual limb will be non-functional.52
Several studies have demonstrated no adverse effect on local recurrence and overall survival in patients undergoing limb salvage surgery compared with those undergoing amputation.\textsuperscript{53-55}

The question of whether a pathological fracture affects disease-free and overall survival has been reviewed in many studies. A fracture through a primary bone tumour will result in the formation of a haematoma that may contaminate adjacent tissue.\textsuperscript{43} Some authors had theorised that this may also lead to distant haematogenous spread of the tumour, due to damage to the microcirculation.\textsuperscript{56} Earlier reports had advocated for amputation in patients with pathological fractures due to this theorised risk.\textsuperscript{57} In Abudu et al’s\textsuperscript{43} retrospective series of 40 patients with pathological fractures of osteosarcoma, there was not a significant difference in overall survival between patients treated with limb salvage versus those treated with amputation. Moradi et al\textsuperscript{58} also found no difference in the disease-free and overall survival outcomes when amputation was compared with limb salvage in 447 patients treated for a pathological fracture of a malignant bone tumour.

Scully et al\textsuperscript{51} investigated osteosarcoma patients with (n = 52) and without pathological fracture (n = 55) in a multicentre study. A pathological fracture was an independent multivariate risk factor for local recurrence and a univariate risk factor for decreased survival.\textsuperscript{51} The degree of displacement of the fracture and whether it occurred prior to presentation or during treatment had no effect on outcomes. However, not all patients received neoadjuvant chemotherapy, as this study reviewed patients from over a 30-year follow-up period.

Bacci et al\textsuperscript{44} showed no difference in disease-free survival and overall survival in a comparison of a cohort of 46 patients with a pathological fracture of an osteosarcoma to a larger group without a fracture. All patients in this study received neoadjuvant chemotherapy. There was also no difference between those that underwent an amputation compared with those who had limb salvage surgery.

Bramer et al\textsuperscript{48} in 2007 reviewed outcomes after pathological fracture at a single institution. In 484 patients with osteosarcoma, rates of local recurrence were similar between groups with or without a fracture; however, overall survival was worse among the fracture group. More aggressive tumours had an increased risk for fracture, and perhaps already carry a worse prognosis, and hence were likely to have a worse outcome. In 156 patients with Ewing sarcoma, no difference in local recurrence or overall survival was found. Another study by Ferguson et al\textsuperscript{47} also found lowered overall five-year survival among patients with a pathological fracture in osteosarcoma, compared with those without a fracture (41% versus 60%). Limb sparing surgery can be performed in patients with a pathological fracture, where appropriate, although more aggressive tumours, which may be more prone to fracture, carry a worse overall outcome in patients with osteosarcoma.

Fractures can also occur after limb sparing surgery, most commonly after allograft reconstruction, but also in patients with Ewing sarcoma after radiation treatment. Reports of failure ranges from 16% to 60%.\textsuperscript{59-62}

Late fractures in patients with Ewing sarcoma treated with radiation for local control can occur due to the effect radiation has on bone healing potential. Of a total of 35 patients with pathological fractures, Fuchs et al\textsuperscript{46} described the development of a late pathological fracture in 21 (60%) at a mean of four years after treatment with radiation. Wagner et al\textsuperscript{63} found a trend towards a relationship between external beam radiation > 40 Gy and incidence of pathological fracture, although small numbers meant that statistical significance was not reached. One-third of the patients with a late fracture had active disease or a secondary neoplasm, reinforcing the need for continued surveillance of these patients with malignancies.

**Summary**

Pathological fractures can occur due to benign and malignant conditions. A thorough history, physical examination and review of plain radiographs are crucial to determine the cause and guide treatment. In most benign cases the fracture will heal and the lesion can be addressed at the time of the fracture or after the fracture is healed. A step-wise and multidisciplinary approach is necessary in caring for paediatric patients with malignancies. Pathological fractures do not have to be treated with amputation; these fractures can heal and limb salvage can be performed when indicated.

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