

Fixation Principles for Pathologic Fractures in Metastatic Disease



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KEYWORDS

• Fixation • Pathologic • Fractures • Bone tumors • Metastases

KEY POINTS

- Primary malignant bone tumors must be ruled out before operative intervention for pathologic fracture.
- Pathologic bone has impaired healing and requires additional fixation strategies.
- Adjuvant therapies are critical in the management of pathologic fractures.

INTRODUCTION

Pathologic fractures occur in response to altered bone physiology, resulting in compromised mechanical properties owing to an underlying lesion. The root cause can be either benign or malignant, primary (ie, bone sarcoma) or secondary (ie, metastatic disease). These entities require different treatments, and the consequences of a missed diagnosis can be devastating; therefore, proper evaluation of the lesion is essential before surgery. The broad differential diagnosis includes metastases, benign bone tumors and tumor-like conditions (eg, fibrous dysplasia, aneurysmal bone cyst, giant cell tumor), bone sarcomas (eg, osteosarcoma, Ewing's sarcoma, secondary sarcoma), and lymphoproliferative diseases (eg, myeloma and lymphoma). Of these, metastasis is the leading cause of pathologic fracture and 500 times more common than primary bone sarcoma.¹ This article will, therefore, focus mainly on pathologic fractures secondary to metastatic disease; however, a diagnosis of primary bone sarcoma must be excluded before intervention.

An estimated 1.9 million people will be diagnosed with cancer in 2022. More than half of

these diagnoses will involve cancers that metastasize to bone, the most common being breast, prostate, lung, renal, and thyroid carcinomas.² Overall, the skeleton is the third most common site of metastatic disease after the lungs and liver, and the most frequent sites for metastasis include the spine, pelvis, proximal femur and proximal humerus.^{3,4} In the United States, the cumulative incidence of bone metastases after the date of diagnosis is 2.9% at 30 days, 4.8% at 1 year, 5.6% at 2 years, 6.9% at 5 years, and 8.4% at 10 years.⁵ An estimated 3% to 8% of patients with metastatic cancer will experience pathologic fractures at some point in their lifetime.^{6,7}

In some patients, pathologic fracture may be the presenting symptom and lead to the diagnosis of metastatic cancer. A database study in 2016 found roughly 5% of patients hospitalized with bone metastases first presented with pathologic fractures.⁸ Skeletal-related events (SREs)—including pathologic fracture, spinal cord compression, and malignant hypercalcemia—were present at diagnosis in 10% to 23% of breast, lung, and prostate cancers.⁹ Pathologic fractures are also associated with increased morbidity and mortality. Patients with metastatic

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breast cancer and a pathologic fracture have a 32% increased risk of death compared with those without fracture.¹⁰ In metastatic prostate cancer, SRE led to an increase in 1-year mortality from 4.7 to 6.6.⁶ This increase in mortality is likely related to both the biology of cancer as well as complications associated with surgical treatment. Many studies have shown that the prophylactic stabilization of impending long bone fractures results in better patient outcomes including shorter hospital stays,¹¹ lower health care costs,¹² earlier mobilization,^{13,14} and lower morbidity.^{6,10,15,16} As such, early identification of bone lesions at risk of fracture and subsequent prophylactic stabilization can improve patient care.

Pathophysiology

Pathologic fractures differ from nonpathologic fractures in location, mechanism of injury, pattern, and healing potential. Pathologic fractures more frequently occur in the common location of metastases (eg, the spine, pelvis, proximal femur, and proximal humerus),^{3,4} whereas nonpathologic fractures frequently occur in the long bones (eg, femur, tibia/fibula, radius/ulna) and hand.¹⁷ Bone lesions can either be characterized as blastic, lytic, or mixed based on the lesion's radiographic destruction or production of bone, respectively. Although lytic lesions are at highest risk of fracture, both blastic and lytic lesions reduce the load-bearing capabilities of the bone with altered elastic modulus, compressive strength, and tensile yield strain.¹⁸ The altered mechanics make bone more susceptible to fracturing under lower energy loads than might be expected. Pathologic fractures also differ in their fracture patterns: pathologic fractures typically present with transverse or short oblique fracture patterns as compared with fracture patterns commonly more commonly seen in normal bone under higher loading conditions, such as butterfly fragments or comminution.

Pathologic fractures also exhibit altered physiology that inhibits healing. Metastatic bone lesions have a disorganized bone matrix with an imbalance between osteoblasts and osteoclasts. This imbalance is mediated by several factors produced or induced by the tumor cells: Receptor activator of nuclear factor kappa-B ligand, prostaglandin-E, transforming growth factor- α and β , epidermal growth factor, tumor necrosis factor- α , interleukins 1 and 6, parathyroid hormone-related peptide, insulin growth factor, endothelin-1, and platelet-derived growth factors.¹⁹ Healing is also further impaired by adjuvant therapies, such as radiation

treatments, which are associated with increased numbers of osteoclasts compared with osteoblasts, disrupted vascular supply, and bone marrow fibrosis.²⁰ This dual inhibition of normal bone formation results in union rates as low as 35% and subjects patients to the complications of nonunion.²¹

Biopsy Principles

When a pathologic fracture is identified through a lesion of unknown origin, a systematic workup must be performed to determine the diagnosis. Although metastasis is the most common cause of bone lesions in adults older 40 years, an unknown lesion should always be approached with a high level of suspicion for a possible primary bone tumor. Inappropriate or incomplete work-up with a misdiagnosis can have devastating complications including local dissemination or systemic progression. Inaccurate diagnosis can not only require additional treatments but also lead to loss of limb or even premature death.²²⁻²⁴

Initial workup should include a thorough history, physical examination, and orthogonal radiographs, both of the fracture site and the involved bone in its entirety. The radiographs should provide insight into the nature of the lesion and the fracture pattern. For an isolated lesion in a patient with no prior diagnosis of cancer, a bone scan and CT of the chest, abdomen, and pelvis should be obtained to evaluate for other lesions and a possible primary tumor. A CT of the affected area may also be useful to aid in preoperative planning, especially for pelvic and spine lesions. MRI is indicated to determine the location and extent of primary malignant bone tumors but is not necessary for the evaluation of pathologic fractures related to metastatic disease.

Following imaging, a biopsy is always needed to confirm the diagnosis in the setting of an unknown primary lesion. Biopsies are necessary for any lesion in a patient without a known primary cancer, any solitary lesion in a patient with a known cancer but no history of metastases, and any lesions concerning a primary bone tumor on imaging. Patients with multiple metastases of a biopsy-proven primary cancer do not require biopsy. The goal of the biopsy is to obtain diagnostic tissue in the least invasive manner possible while avoiding contamination or complicating future planned procedures. Improper biopsy technique can result in altered patient outcomes in 8.5% to 10.1% of cases with a 4.5% to 5.5% rate of potentially avoidable amputation.^{25,26} Appropriate biopsy requires adherence to the following guiding principles.^{27,28}

- The biopsy track is considered contaminated and, in cases of primary bone tumors, will need to be resected with the tumor. Therefore, the incision should be made longitudinally and in line with the planned incision for definitive surgical resection.
- Care should be taken to avoid creating unnecessary tissue planes and to limit exposure to neurovascular structures and uninvolved muscles.
- Strict hemostasis should be maintained, as bleeding or hematoma can contaminate surrounding tissues.
- If a drain is needed to prevent hematoma formation, it should be placed to exit in line with the future surgical resection incision so that it can also be excised during the definitive surgery.
- When possible, the biopsy should be performed by an experienced multidisciplinary sarcoma team, as the risk of complications for a biopsy performed by a nontertiary sarcoma team is five times greater than a biopsy performed by a dedicated sarcoma team.^{25,26}

There are 3 different types of biopsies: fine-needle aspiration (FNA), core-needle biopsy, and open biopsy. FNA is not reliable in solid tumors such as sarcomas, because these samples demonstrate cytology but cannot accurately characterize the histologic structure. Historically, the gold standard for bone biopsies is open surgical biopsy because larger tissue samples can be obtained.²⁹ However, studies have demonstrated similar diagnostic accuracy for percutaneous biopsy with low misdiagnosis rates (3%).^{30,31} In addition, image-guided core needle biopsy is often preferred to incisional biopsy given lower risk of complications (0%–10% vs up to 16%), decreased contamination of surrounding tissue, and lower costs.^{25,26,29} Finally, percutaneous, image-guided biopsy can be used to target areas of the tumor that seem radiographically suspicious. The type of biopsy modality should ultimately be a shared decision between surgeon and interventional radiology team with the goal of accurate diagnosis and minimal morbidity.

Surgical Treatment

The treatment of pathologic fractures relies on many of the same techniques as fractures in disease-free bone; however, there are important differences as well. Communication with medical

oncology to understand the prognosis of the patient is critical. Patients with metastatic cancer are late-stage by definition; while some have short life expectancies and multiple medical comorbidities, others may live for a decade or longer. Patients in advanced stages of disease may prefer nonoperative management of their fractures to avoid the associated morbidity and mortality of surgery. More commonly, surgery is indicated to relieve pain and restore function. In these situations, the focus should be on optimizing short-term rather than long-term outcomes; for example, it is preferable to achieve immediate stability using metal and cement-based constructs than longevity with a biologic reconstruction that will require either a prolonged recovery or carry a risk of early failure. The patient's prognosis and risks, as well as their personal goals and priorities, should be weighed in deciding whether to proceed with surgery and what operation to perform.

The timing of surgery is also an important consideration. Although traditional fracture management recommends expeditious fixation, surgery may need to be delayed in patients with pathologic fractures. Patients without known osseous metastases require a workup, as described previously. Those with an established diagnosis may be undergoing chemotherapy, which would ideally be completed or held before surgery. In general, femur fractures are fixed as soon as possible (ie, within days), whereas fractures in locations such as the humerus, tibia, or acetabulum can be deferred until the patient is optimized (ie, within weeks). Coordination with the medical and radiation oncology teams is thus essential in determining the timing of surgery.

The technical goals of pathologic fracture surgery remain the same as for nonpathologic fracture fixation: restoration of limb length, alignment, and rotation. In addition, emphasis should be placed on early return to weight bearing, attention to impaired bone biology and healing, as well as longevity of the construct appropriate for the patient's life expectancy. Additional fixation strategies such as locking plates/screws and incorporation of polymethyl methacrylate (PMMA) bone cement should be considered whereby appropriate.^{32,33} Locking plates, commonly used for poor bone quality such as in osteoporotic bone, provide improved fixation and limit screw pullout by functioning as fixed-angle devices.³⁴ These systems have been used successfully in oncologic reconstructions with hardware failure rates reported as 0% to 8% and union 4.75 months after fixation.^{35–37} Of note, locking screw technology is only needed

when bone is not sufficiently supportive, so it is not necessary for cement or healthy cortices. The implant itself should bridge the lesion and bypass it by at least 2 cortical widths. Lastly, PMMA is a well-established adjuvant for fixation which may assist by filling large osseous voids, providing axial and rotational stability, and improving the pullout strength of screws.^{38–40}

The type of fixation used depends on the lesion and its location. Bone metastases are generally managed with intramedullary nails (IMN), plate and screw constructs (ORIF), and arthroplasty techniques, with advantages and disadvantages to each. IMNs offer smaller incisions, stabilization of the entire affected bone, and immediate weight bearing. Although highly effective for long bone diaphyseal lesions, they are not appropriate for most periarticular tumors. Open approaches for ORIF provide direct visualization of the tumor for curettage, reduction, and cement application; however, they require larger incisions and soft tissue dissection with a more limited ability to prophylactically protect the whole affected bone. When applied in conjunction with curettage and cement, they can also support immediate weight-bearing for impending fractures in the lower extremity and impending or completed fractures in the upper extremity. Arthroplasty is also an excellent option, particularly for periarticular lesions. This strategy offers immediate stability independent of healing and reduced risk of local disease progression, but can sometimes involve a more extensive surgery in terms of dissection, operative time, and blood loss, as well as bearing an increased risk of infection.^{41,42}

In general, epiphyseal and articular fractures are treated via prosthesis, metaphyseal fractures via plate or prosthesis, and diaphyseal via plate or IMN.^{32,42,43} Curettage and PMMA is most common with plating but can also be performed in conjunction with IMNs. Fixation options for the 2 most commonly affected bones in the appendicular skeleton, the humerus and the femur, are outlined later in discussion. Of note, these should be considered guidelines only, as each fracture requires an individualized approach.

Humerus

- Humeral head: Shoulder replacement with hemiarthroplasty, total shoulder arthroplasty, reverse total shoulder arthroplasty, or proximal humerus replacement.
- Surgical neck to the proximal-third shaft: Plate or IMN.

- Diaphysis: IMN or plate.
- Distal: Parallel plating or distal humeral replacement with total elbow arthroplasty.

Femur

- Femoral head and neck: Hemiarthroplasty or total hip arthroplasty.
- Intertrochanteric or subtrochanteric: IMN, calcar-replacing arthroplasty (hemi vs total), or proximal femoral replacement (hemi vs total).
- Diaphyseal: IMN or plate.
- Distal third: plate, IMN, distal femoral replacement.

Adjuvant treatments

Management of pathologic fractures also relies on adjuvant treatments, such as chemotherapy, radiation, bisphosphonates, and embolization, to treat the primary tumor and reduce disease progression.

Chemotherapy

The need for chemotherapy depends on the primary tumor. If given preoperatively, cytotoxic chemotherapy should be held one to 2 weeks before surgery to allow blood counts to recover. Glucocorticoids should be reduced or tapered before surgery to optimize wound healing.⁴⁴ Most immunotherapies can be continued perioperatively.⁴⁵ There are no definitive recommendations for hormone therapy; however, the increased risk of venous thromboembolisms warrants consideration.⁴⁶ Postoperatively cytotoxic chemotherapy and glucocorticoids should be held for approximately 2 weeks until the incision is healed. Any adjustments to a patient's chemotherapy regimen should be discussed first with the patient's medical oncology team.

Radiation Therapy

Radiation therapy, like chemotherapy, may be administered pre or postoperatively for radiation-sensitive cancers. For metastatic disease, radiation is typically used for pain control rather than control of local disease. Preoperative radiation is generally chosen for cases requiring improved pain control, reduction in the size of the lesions, and decreased likelihood of tumor seeding during surgery. Postoperative radiation may also be chosen for pain control and to reduce the rate of local disease progression. Therapy is typically started 2 to 4 weeks after surgery to allow for soft tissue healing.⁴⁷ Radiation can also be used as a palliative pain relief measure for patients who are too ill to undergo surgery.

There is a lack of definitive consensus regarding the timing of radiation therapy management of pathologic fractures. The American Society for Therapeutic Radiology and Oncology does not offer guidelines for timing, but does support the use of radiation therapy for pain control with metastases.⁴⁸ The conflicting evidence and guidelines emphasize the importance of discussion with the patient's medical and radiation oncology teams to individualize decision-making.

Bisphosphonates

Bisphosphonates, which inhibit osteoclastic bone resorption, can provide another adjunct for managing osseous metastases. Their mechanism of action addresses the underlying imbalance between osteoblast-mediated bone formation and osteoclast-mediated bone resorption seen in pathologic fractures.⁴⁹ Bisphosphonates alone have shown some improvement in pain relief of bony metastases in 1 out of 6 patients treated, but there is insufficient evidence to support their use as first-line therapy.⁵⁰

When used in conjunction with radiation therapy, bisphosphonates can reduce skeletal-related events from bony metastases.⁵¹ After the stabilization of complete or impending pathologic fractures, bisphosphonates and radiation therapy can also reduce local tumor progression and improve pain.⁵² Timing of bisphosphonate administration is usually begun 2-weeks postoperatively, which has not been shown to delay radiographic or clinical healing.⁵³

Embolization

Some hyper-vascular metastatic tumors, such as renal, thyroid, and hepatocellular cancer, may require preoperative embolization. The goal of embolization is to devascularize the tumor before surgical resection, which helps reduce intraoperative blood loss and need for transfusions.⁵⁴ Embolization is successful in up to 75% of cases and can obliterate greater than 70% of a tumor's vascularity before surgery. Generally, patients should proceed to surgery within 48 to 72 hours after embolization, before revascularization begins to occur.⁵⁵

Complications

Complications of surgical fixation of pathologic fractures include local progression, nonunion, hardware failure, infections, venous thromboembolisms (VTEs), and bone cement implantation syndrome (BCIS).

Local Progression

Tumor resection can be an important part of strategies designed to reduce local progression in the management of pathologic fractures. The overall rate of local progression can approach 25% but is tumor- and location-dependent.⁵⁶ For example, renal cell cancer is relatively resistant to chemotherapy and radiation; it also tends to be expansile with a soft tissue component and highly vascular. One study investigating the intramedullary fixation of pathologic fractures showed renal cell cancer, in addition to age, was an independent risk factor associated with disease progression.⁵⁷ For these reasons, while other cancers can be managed with intralesional stabilization and radiation, renal cell metastases may require more aggressive management, such as wide resection, to achieve recurrence-free survival.³³ As such, surgical management of pathologic fractures secondary to renal cell metastases often favors resection and replacement over bony fixation.

Hardware Failure and Nonunion

Pathologic bone exhibits impaired healing resulting in high rates of nonunion and hardware failure. Though data are limited, union rate after metastatic pathologic fracture has been reported as low as 35%.²⁰ Healing can be further inhibited by chemo or radiation therapy. Chemotherapy causes bone marrow suppression, reducing the number of myeloid cells, and radiation therapy inhibits chondrogenesis. Both cell types are prerequisites for fracture healing.⁵⁸ Nonunions place greater strain on the hardware and can lead to early implant failure. One study of patients with femoral metastases who underwent intramedullary nailing for impending or complete pathologic fractures found implant breakage occurred in 8%. Factors associated with implant failure were complete pathologic fractures and prior radiation therapy.⁵⁹ A study of pathologic fractures owing to multiple myeloma found hardware failure occurred in 12.5% of cases, all of which occurred outside of the radiation field. This detail suggests that another risk factor for failure may be not including the full operative bed and implant in the radiation field.⁶⁰

The decision to stabilize (ie, with an intramedullary device or plate) versus reconstruct (ie, resect and replace with an endoprosthesis) a metastatic lesion is based on many factors, including tumor histology, size, and location, as well as patient characteristics. A large number of variables limit research on this topic, with only a small

case series comparing intramedullary stabilization to endoprosthetic reconstruction.⁶¹ Reconstruction is typically more invasive and carries the risk for arthroplasty-related complications such as infection, dislocation, and aseptic loosening; however, advantages include improved local control and no potential for nonunion. Although osteosynthesis using long plates combined with intramedullary fixation has gained popularity in trauma practices, the success of this technique depends on the healing potential of the bone, making it less useful in treating pathologic fractures for the reasons described above. If nonunion occurs, resection and reconstruction with an endoprosthesis (either arthroplasty or intercalary) are preferred over repeated attempts to achieve fracture healing.

Infections

Postoperative infection is a significant concern, with an incidence as high as 10% to 15% in orthopedic oncology patients.⁶² This elevated risk is owing to several factors including patient and surgery characteristics. Patients with cancer can exhibit impaired healing owing to chemotherapy-induced immunodeficiency and/or poor soft tissues owing to radiation, as well as other comorbidities such as diabetes, obesity, and smoking. The prevalence is also high owing to the long duration of oncologic orthopedic surgeries, large incisions, and areas of potential dead space following tumor resection. Inpatient surgery and blood transfusions have also been associated with increased risks of infections.^{62–64}

Optimizing the patient's comorbidities and nutritional status preoperatively, as well as the timing of adjuvant therapy, are important for the prevention of infection. Several drains are commonly used to minimize dead space and fluid collections. Other intraoperative prophylactic strategies range from the use of antibiotic cement and silver-coated prostheses to betadine irrigation and vancomycin powder, but data are limited and no standard of care has been established. As such, the use of these strategies must be extrapolated from the joints' literature as there are little supporting data in the orthopedic oncology literature. Perioperative prophylactic antibiotics are also critical. Unlike the standard clinic practice to administer antibiotics for 24 hours postoperatively, many patients undergoing oncologic surgery are given antibiotics for longer postoperative periods. This practice stems from previous research showing extended postoperative antibiotics could reduce the risk of infection from 13% to 8% in patient undergoing endoprosthetic reconstruction.⁶⁴

More recently, an international randomized controlled trial compared the infection rate of patients undergoing endoprosthetic reconstruction for a primary bone tumor or a soft tissue sarcoma who received one versus 5 days of prophylactic intravenous antibiotics and found no significant difference in infection rate between groups. This study focused only on endoprosthetic reconstruction surgeries and did not evaluate the use of prophylactic oral antibiotics.⁶⁵ As such, there are still no generalized guidelines regarding the duration of prophylactic antibiotics for orthopedic oncology patients.

Venous Thromboembolisms

At baseline, patients with cancer are at an increased risk of both arterial (2%–5%) and venous (4%–20%) thrombotic events compared with the general population.⁶⁶ Surgery increases this risk, as oncologic patients undergoing a procedure have a twofold higher risk of deep vein thrombosis and threefold higher risk of fatal pulmonary embolism compared with patients without cancer.⁶⁷ One study found patients with long-bone metastases who undergo surgery have up to a 6% chance of developing VTE following long bone surgery.⁶⁸ These elevated risks are still present even when patients are prophylaxed with a course of low molecular weight heparin (LMWH).⁶⁹ Perioperative chemotherapy and reduced mobility can further contribute to these risks, highlighting the importance of anticoagulation for oncologic patients undergoing surgical fixation.⁷⁰ Current anticoagulation guidelines from the American Society of Clinical Oncology recommend thromboprophylaxis with a direct oral anticoagulant (DOAC), such as apixaban or rivaroxaban, or LMWH for patients undergoing major cancer surgery. This prophylaxis should start before surgery and continue for at least 7 to 10 days after surgery.⁷¹ There are no specific recommendations between DOACs and LMWH. Although DOACs have a nonsignificant trend toward better efficacy owing to the oral route, LMWH is associated with lower rates of bleeding complications.⁷²

Another consideration in the incidence of VTE is the use of tranexamic acid (TXA), an antifibrinolytic agent that may be administered either via IV or topically and has proven efficacious in decreasing perioperative blood loss and need for transfusion in patients with total hip and knee arthroplasty.^{73,74} Even in patients with known risk factors for postoperative VTE, such as prior VTE or prothrombotic medical comorbidities, TXA has not been associated with

increased risk for postoperative VTE.^{75,76} The current literature to support of the use of TXA in orthopedic oncology is much more limited. One single-center retrospective study of patients undergoing endoprosthetic reconstruction showed that patients who received topical TXA had reduced perioperative blood loss and transfusion rates without an increase in VTE occurrences.⁷⁷ While these results are promising, additional prospective studies are necessary to fully examine the safety of TXA in oncologic patients.

Bone Cement Implantation Syndrome

Bone cement implantation syndrome (BCIS) is a constellation of symptoms that include hypoxia and hypotension shortly after pressurizing cement within the bone. The pathophysiology is not fully understood, but BCIS has been described more consistently within the hip arthroplasty literature. One study demonstrated that up to 75% of oncologic patients who underwent femoral cemented arthroplasty experienced BCIS. Patients older 60 years who had lung cancer also exhibited an increased risk of BCIS.⁷⁸ As cement is often necessary to achieve fixation, surgeons can minimize the risk of BCIS by use of a low-viscosity cement mixed under vacuum without pressurization, lavaging the intramedullary canal before the insertion of prosthesis, using the shortest stem necessary, and inserting the stem slowly.^{79,80} Communication with anesthesia colleagues and careful monitoring are also key to the recognition of BCIS and initiation of supportive care when necessary. *Pulmonary complications.*

Both intramedullary stabilization and insertion of long cemented stems can lead to pulmonary compromise, especially in patients with poor reserve at baseline. Instrumentation of the canal during nailing increases intramedullary pressure, causing the intravasation of bone marrow content and dissemination into the pulmonary circulation. This disruption of the blood flow through the lungs can result in hypoxia, heart failure, and a systemic inflammatory response.⁸¹ Reaming before nail insertion has been shown to increase intramedullary pressure compared with unreamed nailing. Utilization of long stems in arthroplasty reconstructions can also have this effect, with the added risk of bone cement implantation syndrome (BCIS). The mechanism of BCIS is poorly understood, but the physiologic result can include hypoxia, hypotension, pulmonary hypertension, arrhythmias, and cardiac arrest. Clinically, the effect can range from mild hypoxia and confusion postoperatively to

intraoperative death.⁸² Lastly, reaming through a metastatic lesion may theoretically seem to promote iatrogenic spread of cancer to the lungs; however, the metastatic process depends more on the biological capability of the cancer cells than their physical distribution. A recent retrospective study demonstrated the surgical fixation of pathologic fractures using IMN does not significantly increase the incidence of new metastatic disease to the lungs compared with arthroplasty and ORIF techniques in pathologic fractures secondary to breast, prostate, renal, and lung metastases.⁸³

The risk of pulmonary complications can be mitigated by the use of venting, that is, drilling a hole into the distal cortex of the canal, which decreases intramedullary pressure by 50% in cadaveric studies.⁸⁴ While venting also has the potential to increase the spread of tumor cells to the surrounding tissues, this very rarely has clinical relevance. Suction devices, such as the Reamer-Irrigator-Aspirator (RIA), also reduce pressure and embolization during reaming, though the benefit is less clear. A prospective, multi-center trial examining the use of the RIA in the fixation of isolated nonpathologic femur fractures demonstrated a modest reduction of embolic debris during the reaming and nail insertion segments of the operative procedure in patients who underwent nailing with a RIA; however, the study was unable to correlate that reduction with any changes in physiologic clinical measures.⁸⁵ Most importantly, gentle reaming, slow implant insertion, and careful communication with anesthesia are critical to minimize the risk of cardiopulmonary compromise.

SUMMARY

Surgical management of pathologic fractures requires an accurate diagnosis of primary malignancy, which often involves a biopsy. Although the most common cause is metastatic disease, the possibility of a primary bone tumor should always be considered. Pathologic fractures have impaired healing potential with high rates of nonunion and hardware failure. The surgical plan should, therefore, include additional fixation strategies to mitigate these risks and may include locking plate systems, PMMA cement, and arthroplasty. Adjuvant therapy, such as chemotherapy and radiation, may also be used whereby appropriate to reduce local recurrence and improve pain. Optimizing treatment involves the careful coordination of a multidisciplinary team.

CLINICS CARE POINTS

- Accurate diagnosis of the primary malignancy resulting in a pathologic fracture is essential before surgical fixation. The most common cause in adults older 40 years is metastatic disease.
- Diagnosis usually requires biopsy, which should adhere to strict technique guidelines to avoid adverse patient outcomes.
- Pathologic bone has impaired healing and requires additional fixation strategies, such as PMMA cement.
- Adjuvant treatments, such as chemotherapy and radiation, should be coordinated by the medical and radiation oncology teams, respectively.
- Patients with pathologic fractures are at increased risk of postoperative complications compared with those with nonpathologic fractures.

DISCLOSURE

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