Metastatic Tumors of the Spine: Diagnosis and Treatment

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Abstract

Metastatic disease of the spine occurs in as many as 70% of patients with disseminated cancer and may result in vertebral collapse, spinal instability, and progressive neurologic compromise. Today, magnetic resonance imaging is the most effective means of differentiating benign from malignant causation of vertebral collapse, based on the imaging patterns and extent of marrow ablation. The more rapid the onset of the neurologic deficit, the worse the prognosis for recovery, no matter what treatment is instituted. The majority of vertebral lesions requiring decompression and stabilization emanate from the vertebral body and are best managed by anterior decompression and stabilization alone. With posterior element destruction, spinal subluxation through the involved segment, or involvement of the lumbar spine, a combination of both anterior and posterior stabilization is required. The author's preference is to perform anterior vertebral replacement with methylmethacrylate incorporating a Knodt distraction rod. This construct affords instantaneous stability that is not adversely affected by postoperative irradiation. Many devices can provide adequate posterior stabilization, but the author prefers to use Luque rods with sublaminar wire fixation. In a series of 77 patients with major neurologic compromise treated with this technique, 62% showed improvement by at least two Frankel grades, compared with fewer than 5% who improved after laminectomy decompression with or without irradiation. Nineteen of the 77 patients remained alive more than 4 years postoperatively.

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The spine is the most common site for skeletal metastases, irrespective of the primary tumor involved. The vertebral body typically is affected first because of its rich blood supply and sinusoidal vascular distribution. However, the initial radiographic finding often is destruction of a less well vascularized pedicle. This paradox is explainable by the fact that between 30% and 50% of a vertebral body must be destroyed before any changes can be recognized radiographically, unless there is a blastic or sclerotic reaction. In contrast, minimal lysis of pedicular bone can be appreciated because the cortex of the pedicle tends to be involved early and because the pedicle can be seen well in cross section on conventional anteroposterior radiographs.

Approximately 70% of patients who die of cancer have evidence of vertebral metastases apparent on careful postmortem examination. Three fourths of these lesions originate from carcinoma of the breast, prostate, kidney, or lung or from myeloma or lymphoma. However, vertebral metastases often are asymptomatic and may be discovered only on routine bone scans. When symptoms do develop, they are a consequence of one or more of the following: (1) an enlarging mass within the vertebral body, which may break through the cortex and invade paravertebral soft tissues; (2) compression or invasion of adjacent nerve roots; (3) compression of the spinal cord; (4) development of a pathologic fracture secondary to vertebral destruction; and (5) development of spinal instability from such a fracture, particularly when associated with lytic destructive changes in the posterior elements.

Spinal cord and/or nerve-root compression occurs in approximately 5% of patients with widespread cancer. The most common cause of this compression is the extrusion of tumor tissue and detritus of bone or disk into the spinal canal following the partial collapse of a vertebral body that has been infiltrated and weakened by a metastatic deposit.

Radiographic Findings

Plain radiographs of a symptomatic patient typically will demonstrate either an anterior compression deformity with secondary kyphosis (Fig. 1) or a more uniform vertebral collapse usually associated with posterior column destruction and focal spinal instability (Fig. 2). Of course, either of these bony deformities can also result from osteopenic changes unrelated to malignancy, due to a variety of causes. Primary vertebral neoplasms or indolent vertebral osteomyelitis also may progress to cause vertebral collapse and a lesion difficult to differentiate

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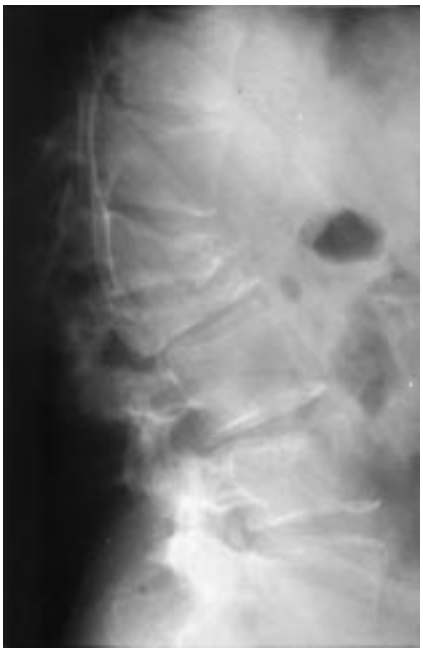


Fig. 1 Radiograph of a 66-year-old woman with known breast cancer and scintigraphically demonstrable metastases to T-11 and T-12. Although the wedge compression fractures demonstrated presumably are secondary to metastases, their appearance on plain radiography is indistinguishable from that of benign pathologic fractures secondary to osteoporosis.

from metastatic disease. Even patients with known metastatic disease of the spine may develop collapse or instability at other spinal levels due to nonmalignant causes.

All of these processes initially present as back pain of sudden or insidious onset, with or without neurologic compromise. A history of progressive quadriparesis or even of specific radiculopathy is of minimal benefit in helping to differentiate among the various potential causes of spinal deformity. The oft-quoted maxim that sudden fracture myelopathy invariably is the result of acute trauma has been repeatedly proved invalid, just as the concept that acute trauma never results in gradual or progressive neurologic compromise has been proved wrong.

Other Diagnostic Studies

The availability today of a variety of imaging modalities has enhanced our ability to differentiate between benign and malignant spinal deformity on the basis of distribution of abnormalities in the spine as well as specific patterns of focal bony destruction. Technetium-99m scintigraphy often will demonstrate multiple sites of radioisotope uptake in other vertebrae, long bones, ribs, or the skull typical of generalized skeletal metastases, even when a patient's symptoms and plain radiographs suggest isolated involvement of a single spinal level (Fig. 3).

The most helpful and sensitive study, however, has been magnetic resonance (MR) imaging, because this technique most effectively delineates the extent and pattern of marrow involvement within an affected vertebra. Characteristically, the malignant pathologic fracture occurs because virtually the entire vertebral body has been infiltrated by tumor. The tumor spreads initially through the hematopoietic tissue and only later progressively destroys bone. In contrast, benign compression fractures occur because the bone substance itself has been lost or weakened, with hematopoietic tissue remaining relatively intact. In both instances, the disk remains unaffected, thus helping to differentiate either lesion from osteomyelitis (Fig. 4).

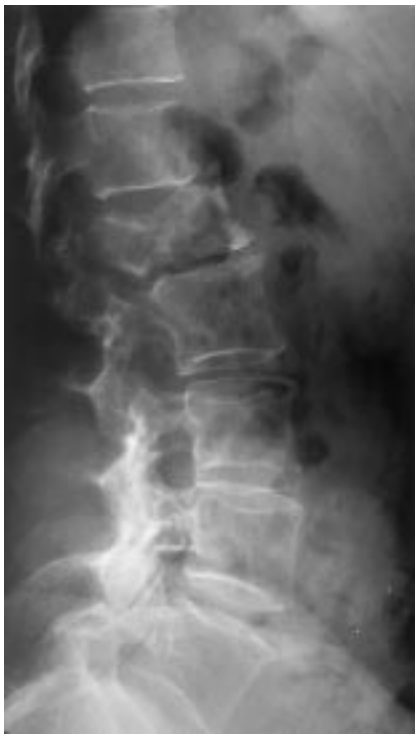


Fig. 2 Spontaneous fracture of L-1 from known metastatic breast cancer. Osteolysis of all three columns of the spine resulted in symmetrical vertebral collapse and focal instability.

An MR image of a benign compression fracture typically reveals preservation of the normal marrow signal, although there may be displacement of the marrow along vectors created by the compression deformity. This phenomenon is particularly apparent in the T1-weighted image, where the combination of the hematopoietic tissue, edema, and bleeding increases the focal water signal and the consequent intensity of that signal (Fig. 5)

An acute benign compression fracture of the superior endplate typically causes temporary linear striation of the marrow distribution in the rest of the vertebra, particularly on T1 imaging. This finding usually occurs in a uniform pattern and is reversible as fracture healing occurs.[1] The T2-weighted image shows bone-marrow signal intensity in the fractured bone similar to that in the rest of the vertebral body.

In contrast, the MR imaging of a compression fracture secondary to metastatic malignancy reveals total or subtotal replacement of the normal bone by tumor. This is reflected by a decreased-signal-intensity (darker) image on T1-weighted images (Fig. 6) and increased intensity on T2 images. There may be incomplete replacement of marrow, but its pattern will be irregular,

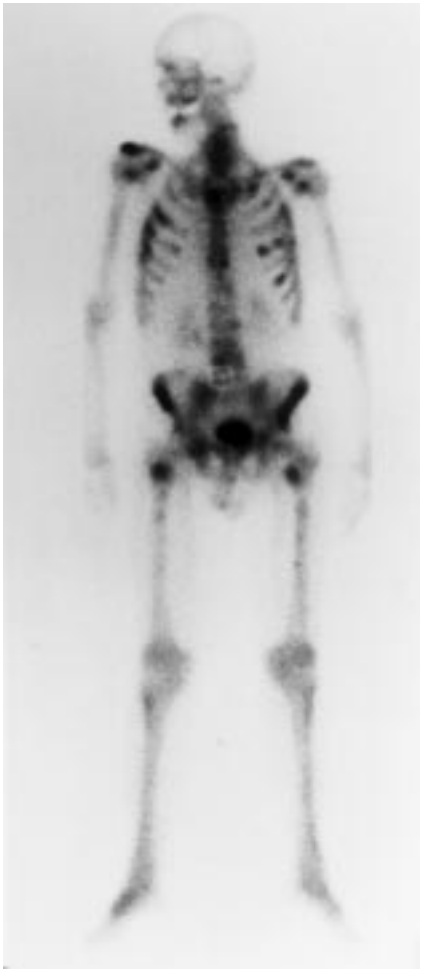


Fig. 3 Anterior whole-body radionuclide image of a patient with prostatic carcinoma reveals multiple foci of increased tracer deposition in the shoulders, ribs, lumbar spine, pelvis, and proximal femora.

reflecting focal destruction rather than uniform compression of hematopoietic tissue and fat.

Although MR imaging has a high level of sensitivity, its specificity may become blurred when an acute benign fracture is associated with marked edema and bleeding into the marrow space. The T1 signal may mimic the typical tumor pattern (Fig. 7). Bulging of the partially collapsed vertebral body and diffuse marrow signal changes extending into the pedicles may be strongly suggestive of tumor infiltration. In these instances, or in any situation in which an occult symptomatic vertebral metastasis is suspected, early biopsy of the lesion is warranted.

Computed tomography (CT)-directed needle biopsy is accurate and safe and has virtually replaced open or percutaneous trocar biopsy in most centers. In the event of an equivocal or nondiagnostic specimen, the CT-directed biopsy should be repeated at different areas of the affected vertebra before resorting to open biopsy techniques.

Clinical Course

Once the presence of spinal metastases has been established, treatment options can be considered. As already noted, it is common for vertebral metastases to be asymptomatic and to be diagnosed only with the use of routine bone scintigraphy. Such a finding may prompt the oncologist to alter the patient's chemotherapy or hormonal manipulation, but no specific additional measures are indicated. If spinal pain develops, it is essential to clarify whether it is attributable to tumor destruction or to local phenomena such as osteoporosis or arthritis, particularly because corticosteroids or chemotherapy given as part of systemic cancer treatment may result in marked osteopenia (Fig. 1). Insufficiency fractures of the spine due to local irradiation may appear years after treatment has been completed. Debilitated cancer patients who are receiving chemotherapy typically become chronically pancytopenic and are at increased risk for hematogenous osteomyelitis involving the spine (Fig. 4).

When spinal metastases truly are the source of pain, that pain is usually of gradual onset, is relentlessly

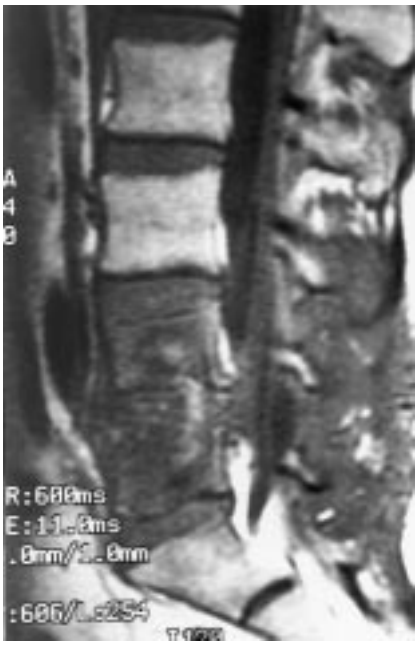


Fig. 4 Sagittal MR image of the lumbar spine of a 66-year-old man receiving chemotherapy for metastatic prostatic carcinoma. Spontaneous hematogenous osteomyelitis developed at L4-5.

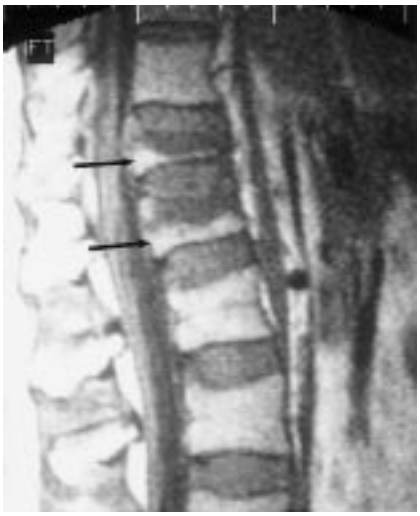


Fig. 5 Sagittal T1-weighted MR image shows two benign compression fractures with incomplete bone marrow replacement and peripheral low-signal-intensity band (arrows).

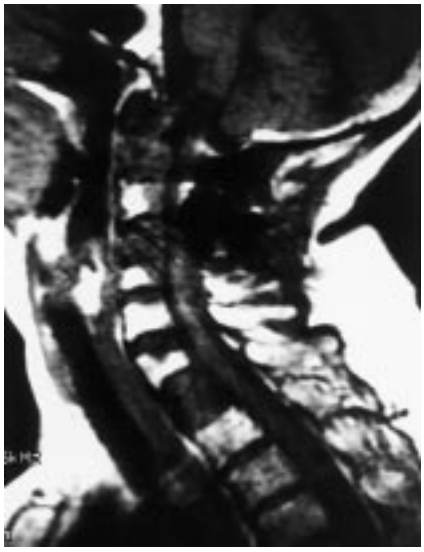


Fig. 6 Sagittal T1-weighted MR image of the cervical spine of a 69-year-old woman with widely metastatic breast carcinoma. Multiple foci of abnormal replacement of the marrow signal are particularly apparent in the C-1, C-2, C-4, and C-8 vertebral bodies.

progressive over weeks or months, is worse at night, and is unassociated with significant elevations of white blood cell count or sedimentation rate. This type of pain has been attributed to stretching of the periosteum by direct pressure of the expanding tumor or to microfractures occurring sequentially within weakened bone. Another potential source of pain is from compression of the ventral aspect of the dura, which is richly innervated with nociceptor fibers. Such pain can occur before there is evidence of neurologic involvement. Pain can also result from invasion of paravertebral structures, sometimes producing neurologic symptoms from involvement of the lumbosacral plexus.

Not infrequently, the patient will localize the pain at a level below the actual metastatic lesion. This may lead the unsuspecting physician to attribute initial symptoms to arthritis or disk disease and to continue conservative and ineffective treatment in the face of progressive neurologic compromise. The presence of radicular pain may help to locate the level of vertebral involvement.

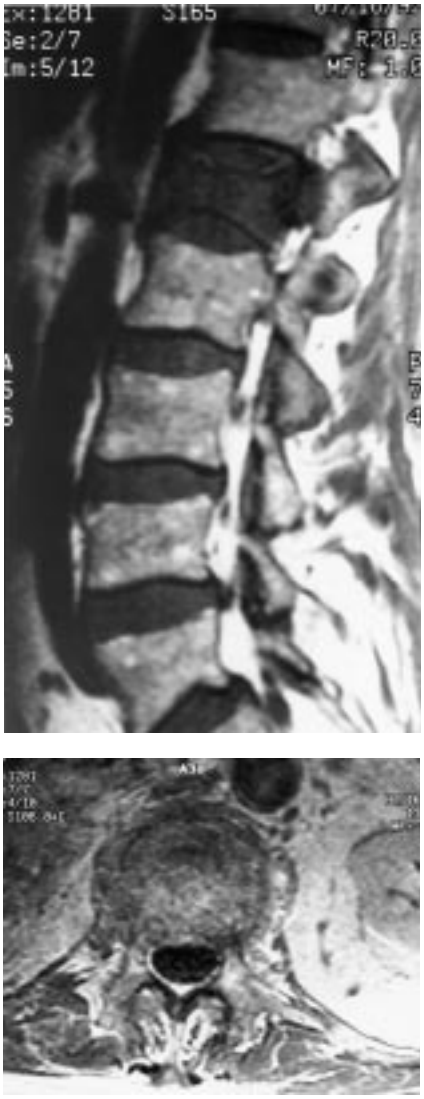


Fig. 7 Images of a 72-year-old woman with sudden onset of severe thoracolumbar pain without trauma. **Top,** Sagittal T1-weighted image shows marked homogeneously decreased signal intensity with posterior bulging of the vertebral cortex into the canal. **Bottom,** Axial T1-weighted image shows that abnormal signal changes extend into both pedicles. Both T2-weighted images were interpreted as suggestive of tumor infiltration of the vertebral body, but biopsy revealed only osteoporosis.

Approximately 50% of patients with thoracic cord impingement complain of radicular pain before they develop symptoms of cord involvement. Such pain often is described as "girdle pain," particularly with lesions at T-9 or below, and may not be recognized as reflective of intercostal root irritation.[2]

With more central neural involvement, motor deficits usually precede sensory changes because of the typically anterior location of cord compression. Loss of sphincter control is thought to be a late phenomenon, and usually occurs only in patients with profound cord involvement. However, cauda equina involvement can occur acutely or subtly in patients with involvement of the conus medullaris. Sphincter function should be carefully and sequentially evaluated. The sensory level often is not a reliable indicator of the level of cord compression, commonly being recorded several segments below the site of fracture or tumor extrusion into the spinal canal.

The rapidity of onset of muscle weakness has considerable bearing on the prognosis. Constans et al[3] reported that 166 of 600 patients (28%) had an acute onset with a delay of less than 48 hours between the manifestation of initial symptoms and the appearance of maximal neurologic compromise. These patients had the worst prognosis for recovery, no matter what treatment was rendered. Patients with a slower evolution of neurologic compromise, indicating in most instances a slower growth rate of the metastasis and a sparing of the anterior spinal artery, had a decidedly better prognosis. Tarlov and Herz[4] demonstrated experimentally that even major neurologic compromise caused by gradual cord compression was reversible for a longer period than was compromise due to an acute cord lesion. Conversely, a

sudden onset of paralysis is almost invariably associated with a poor prognosis, probably primarily attributable to vascular compromise.

Nonoperative Treatment

The philosophy of treatment for vertebral metastases has changed considerably in the past two decades. With improvement in chemotherapy and hormonal manipulation, many patients with bony metastases now survive for long periods without premorbid involvement of vital organs. Consequently, progressive vertebral metastases are often apparent in patients with a prolonged life expectancy, and the prospect of ultimate spinal instability and neurologic compromise becomes of increasing concern.

Most patients with spinal metastases do not develop progressive spinal instability or neurologic involvement and can be treated successfully with systemic chemotherapy, local irradiation, or temporary bracing. Primary tumor types vary in radiosensitivity after metastasis (Table 1). Even those who sustain a pathologic compression fracture of one or more vertebral bodies often can be treated effectively with temporary bed rest and soft bracing, as is done for pathologic compression fractures due to osteoporosis. In my experience, approximately 80% of patients with spinal metastases can be treated effectively with one of these nonoperative modalities.[2,5]

When metastases are causing minimal bone destruction and pain appears to be the result of periosteal expansion or reaction within the bone to tumor, radiation therapy alone often is the ideal means of achieving relief. If the tumor extends into the epidural space, causing early neurologic compromise, radiation therapy usually leads to recovery unless the cord or nerve roots are compressed by fragments of bone or disk detritus. Radiation therapy also should be the primary treatment modality in patients with an anticipated survival of 4 months or less or with vertebral-body lesions affecting multiple levels of the spine.

Table 1
Radiosensitivity of Common Metastases

High sensitivity
Myeloma
Lymphoma
Moderate sensitivity
Colon
Breast
Prostate gland
Lung
Squamous cell
Low sensitivity
Renal
Thyroid
Melanoma
Metastatic sarcoma

The threshold for radiation complications, including myelopathy, radiation osteitis, interference with wound healing, and interference with graft incorporation consistently appears to be between 3,000 and 3,500 cGy. Because the control of local tumor recurrence in the spine does not seem to improve with doses in excess of 3,000 cGy, it is generally recommended that local irradiation be limited to this dose level. In any case, adjunctive irradiation should be postponed for a minimum of 3 to 4 weeks after any operative intervention to limit interference with wound healing and graft incorporation.

Operative Management

The principal indications for operative intervention are progressive neurologic compromise and intractable mechanical spine pain unresponsive or unlikely to be responsive to irradiation or bracing. Decompression is particularly indicated when cord or root compression is due to retropulsed bone or disk fragments or when spinal instability or malalignment causes neural compromise. Other specific indications include radioinsensitive tumors, recurrence of cord compression following adequate local irradiation, and presumed metastases when the primary tumor is occult.

Two decades ago, "operative intervention" usually meant laminectomy decompression. The results of this procedure for the management of advanced spinal metastases were dismal. The majority of patients with neurologic compromise did not improve. Instead, progressive spinal deformity and instability frequently developed as a result of, rather than in spite of, the decompression. In a large retrospective series, Gilbert et al[6] demonstrated that radiation therapy alone was as effective as decompressive laminectomy (with or without radiation) in the treatment of epidural cord compression. After either treatment, fewer than 50% of patients regained the ability to walk.

It was only after the evolution of anterior spinal decompression and stabilization techniques that the clinical results showed dramatic improvement.[2,7] In the vast majority of patients, tumor originates from the vertebral body or soft tissue anterior to the spinal cord and cannot be decompressed adequately from a posterior laminectomy approach. When the entire vertebral body (both anterior and middle columns) becomes weakened by tumor lysis, the vertebral body begins to collapse, and the bending moment of the spine shifts posteriorly. As this worsens, the compression load on the remaining vertebral body increases geometrically, leading to a progressive kyphotic deformity and ultimately to extrusion of tumor tissue, disk, and

bony detritus posteriorly into the spinal canal (Fig. 8).

Ordinarily the posterior elements (posterior column) are minimally involved, and posterior tensile stability remains intact. In such a situation, overall spinal stability can be restored entirely through an anterior approach. However, if tumor destruction of the posterior elements (particularly the pedicles) is advanced, the greatly increased tensile loads posteriorly cannot be resisted. Typically, a forward-shearing deformity will develop (Fig. 2), further compromising the spinal canal and necessitating both anterior and posterior decompression and stabilization.

If the previously mentioned indications for operative intervention are present, the surgeon must consider separately the issues of decompression and stabilization. For any given patient with spinal cord or cauda equina compromise, decompression should be recommended as soon as a clear-cut motor deficit is apparent, but only if that deficit correlates with a demonstrable focus of spinal canal intrusion by tumor or bony debris. In my experience, neither systemic corticosteroids nor emergency local irradiation is beneficial in such circumstances.

The rare syndrome of progressive sensory loss in the absence of motor deficit may respond to local irradiation, particularly if a peridural tumor mass is apparent without major spinal instability or bony debris within the canal. However, the surgeon must be aware of the fact that numbness and paresthesias, particularly if peripheral, more often are attributable to the neurotoxic effect of certain chemotherapeutic agents.

One must also be wary of attributing progressive motor compromise to irradiation-induced transverse myelitis unless a gadolinium-enhanced MR imaging study clearly demonstrates changes consistent with that diagnosis. In my experience, it is far more likely for progressive motor deficits to be caused by gradual spinal instability or local tumor recurrence than by the late effects of irradiation. Patients with intractable pain secondary to spinal instability who do not have neurologic compromise do not require emergency operative intervention. Such patients may enjoy sufficient relief from external bracing, rendering spinal stabilization unnecessary. If elective surgery is required, chemotherapy must be discontinued early enough to allow correction of anemia and recovery of white blood cell and platelet counts.

Spinal canal compromise from posterior extrusion of the vertebral body can be decompressed only from an anterior approach. Combined anterior and posterior cord compression (so-called napkin-ring compression) usually must be relieved by both anterior and posterior approaches (Fig. 9). If the posterior column structures remain functionally intact, at least in the cervical and thoracic spine, restoration of stability can be achieved by anterior vertebral reconstruction alone. If all three columns are severely weakened, combined anterior and posterior stabilization is essential. The only exception to this general rule pertains to the lumbar spine. Because of its lordotic curvature and the extent of weight-bearing torque and lateral bending forces to which it is subjected, I believe that both anterior and posterior stabilization are necessary in all instances in which spinal decompression is required (Fig. 10).

The surgeon should strive to achieve instantaneous and rigid intraoperative stability and should not depend on gradual incorporation of bone grafts to restore late local rigidity. There is abundant evidence that, with rare exceptions, bone grafts will not be incorporated

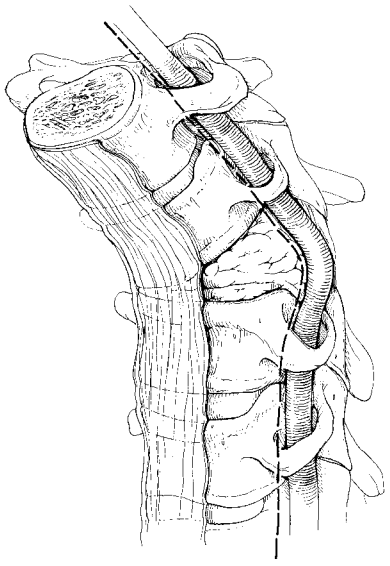


Fig. 8 Replacement of the vertebral body by tumor results in collapse of the body, increasing kyphosis, and extrusion of tumor and bone fragments into the epidural space.

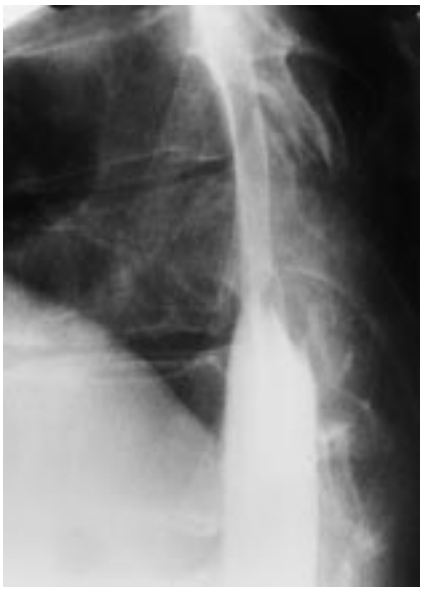
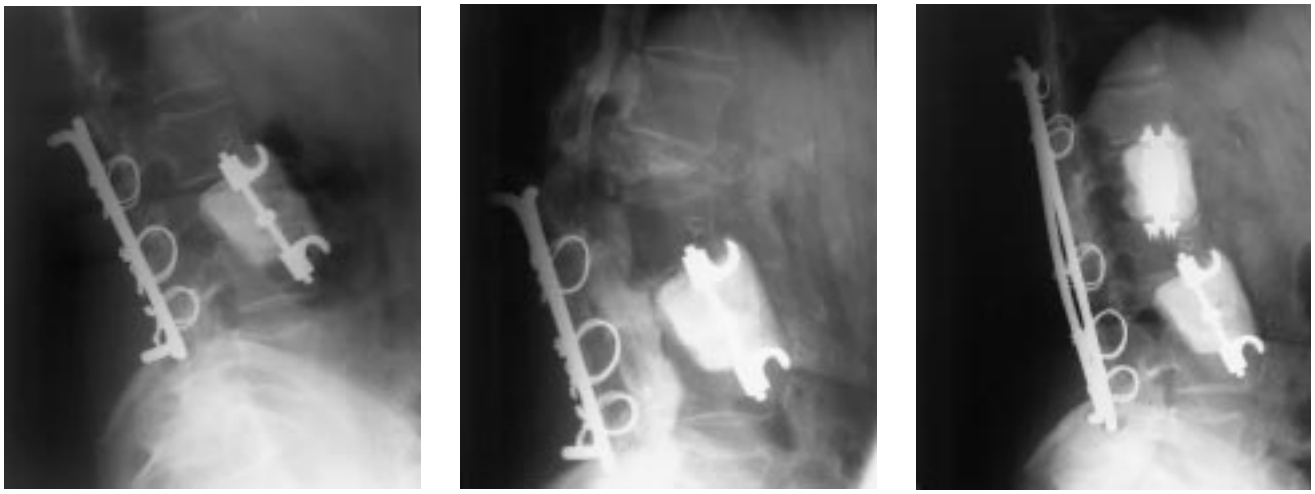


Fig. 9 Unusual "napkin-ring" constriction of the cord caused by a metastatic tumor within the spinal canal growing around the dura and compressing the cord circumferentially. In such cases both anterior and posterior decompression and stabilization are usually necessary.



A B C

Fig. 10 Radiographs of a 65-year-old woman with multiple myeloma, progressive tumor infiltration, and collapse of the L-3 vertebral body. **A,** The patient presented with a rapidly progressive cauda equina syndrome (Frankel grade C) despite 4,500 cGy of local irradiation. After anterior L-3 vertebrectomy and replacement by methylmethacrylate incorporating a Knodt rod, a posterior four-level stabilization was accomplished with Luque rods and sublaminar wire fixation. The patient enjoyed a complete neurologic recovery. **B,** Six years later, a new compression fracture appeared at L-1, again associated with a progressive cauda equina syndrome. **C,** The L-1 vertebral body was replaced using methylmethacrylate incorporating a Rezinian vertebral distractor. The original Luque rods were replaced with longer rods and sublaminar wiring spanning seven levels. Pathologic examination of the resected L-1 vertebral body revealed that it had collapsed because of radiation osteitis, not myeloma.

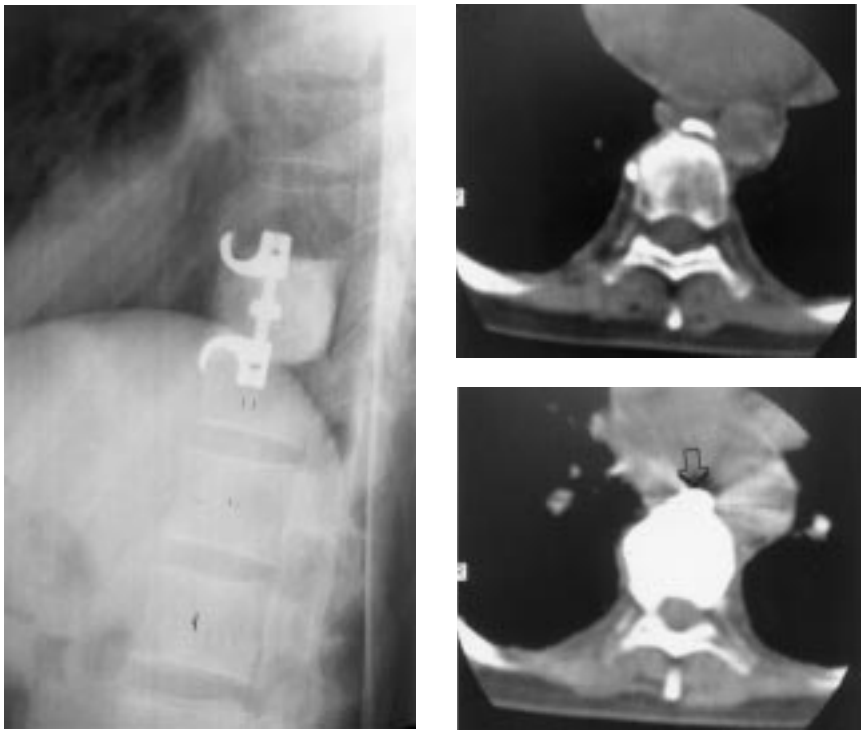
in the face of postoperative irradiation of the affected area. For these reasons, I advocate the technique of replacing the resected vertebral body with methylmethacrylate, polymerizing in situ, and incorporating a distraction-fixation device that secures the cement mass into the adjacent normal vertebral endplates.

In my hands, the most effective device is the Knodt distraction rod with hooks (Zimmer), which jacks open the collapsed vertebral space to its appropriate height and can be buried entirely within the long axis of the spine. This fixation construct does not protrude beyond the vertebral bodies, thus protecting adjacent soft tissues from injury (Fig. 11). The combination of the methylmethacrylate and the Knodt rod very effectively resists compression and torque loads in the cervical and thoracic spine but requires adjunctive posterior stabilization devices in the lumbar spine.

The Rezinian distraction device functions in a similar manner and also does not extend beyond the confines of the vertebral bodies. However, in my experience, it offers no advantages over the Knodt rod and is many times more expensive. The distraction hook-rod system is similar in concept to the Knodt rod but is much bulkier and extends into the perivertebral soft tissues, causing a risk of soft-tissue erosion.

Alternative anterior-fixation devices that depend on screw fixation across the vertebral bodies are more complicated to insert, protrude well outside the vertebral column, and are subject to a higher incidence of failure because their means of screw fixation to the vertebral bodies is at right angles to the axial compression load on the spine.

If posterior fixation is necessary, a variety of devices are available. Their selection should be based on the severity of posterior bony destruction demonstrable in any given patient. Most commonly, patients with a metastatic malignant neoplasm extensive enough to require posterior stabilization have advanced lysis of one or more pedicles (in addition to the vertebral body), which precludes secure fixation by pedicle screw-and-rod systems. Distraction or compression rods with hooks may be used but have the disadvantage of focusing the fixation stress at only a few levels where progressive tumor lysis may cause late instability. For this reason, I have usually chosen to use Luque rods with sublaminar (not spinous process) wire fixation three levels above and three below the span of laminectomy decompression. On occasion, when the strength of laminar bone at any level is suspect, combining the sublaminar wires with methylmethacrylate may help to reduce the tendency of an individual wire to cut through soft bone at that level (Fig. 12).



A B

Fig. 11 Images of a patient with metastatic breast carcinoma 5½ years after a midthoracic vertebrectomy and anterior stabilization with a Knodt rod and methylmethacrylate. **A,** Lateral radiograph demonstrates that the height of the vertebral space has been reconstituted fully and remains so without evidence of displacement of the construct despite the absence of posterior stabilization. **B,** CT scans. **Top,** Section through the vertebral body just above the cement construct. Note that the tip of the Knodt rod hook protrudes slightly in front of the anterior longitudinal ligament. **Bottom,** Section through the methylmethacrylate reconstruction. Despite the diffraction artifact from the metal rod (arrow), the normal dimensions of the spinal canal can be appreciated.

Operative Technique

The technique of anterior decompression and stabilization of the thoracic spine is illustrated in Figure 13. Before undertaking the procedure, the surgeon should attempt to anticipate how aggressive the tumor appears radiographically and how vascular the lesion is likely to be. Large osteolytic lesions with minimal host bony response are likely to be extremely vascular, particularly if the primary malignant neoplasm is myeloma or metastatic hypernephroma. Such lesions should be embolized preoperatively. Olerud et al[8] have described the indications and technique for this procedure in detail. In essence, using standard arteriographic techniques, the major feeder vessels supplying the tumor focus are catheterized, and a thickened paste made of moistened and morcellized absorbable gelatin sponge (Gelfoam) is injected, which effectively obstructs blood flow.

Anterior stabilization of the thoracic spine requires a thoracotomy, with exposure of the pericardium, one lung, and the great vessels. A double-lumen endotracheal tube may be employed, permitting collapse of the ipsilateral lung for improved exposure. A chest tube is required postoperatively for a period of 48 to 72 hours for pleural drainage and lung reexpansion. Occasionally, overnight intubation will be expedient, particularly for the patient who is moderately debilitated, has chest wall or pleural metastases that interfere with ideal ventilation, or shows evidence of pleural metastases.

The thoracotomy incision is made one level higher than the highest affected vertebra, and the rib at that level is removed. The vertebral bodies are easily visualized through the thin overlying parietal pleura. By transecting but not removing one or two additional ribs below the incision, it is possible to expose multiple vertebrae above or below the tumor focus. By incising the posterolateral crura of the diaphragm and then approaching the lumbar spine retroperitoneally, we have been able to expose from T-8 to L-4 through the same thoracotomy incision with a single rib resected.

The parietal pleura is incised, elevated, and reflected to expose the segmental vessels (Fig. 13, A). These are ligated and transected as close to the aorta as possible, thus minimizing disturbance of the paravertebral anastomoses. In more than 60 such approaches, I have seen no evidence clinically of cord vascular compromise after division of up to nine vessels on one side; some surgeons, however, feel that spinal evoked potential monitoring is essential as the vessels are sequentially ligated. After division of these vessels, the aorta can be retracted carefully, facilitating exposure of the entire anterior aspect of the vertebral bodies involved (Fig. 13, B). Careful blunt dissection is continued subperiosteally to expose the lateral aspect of the affected vertebra on the opposite side.

All remnants of the affected vertebra should be resected, together with

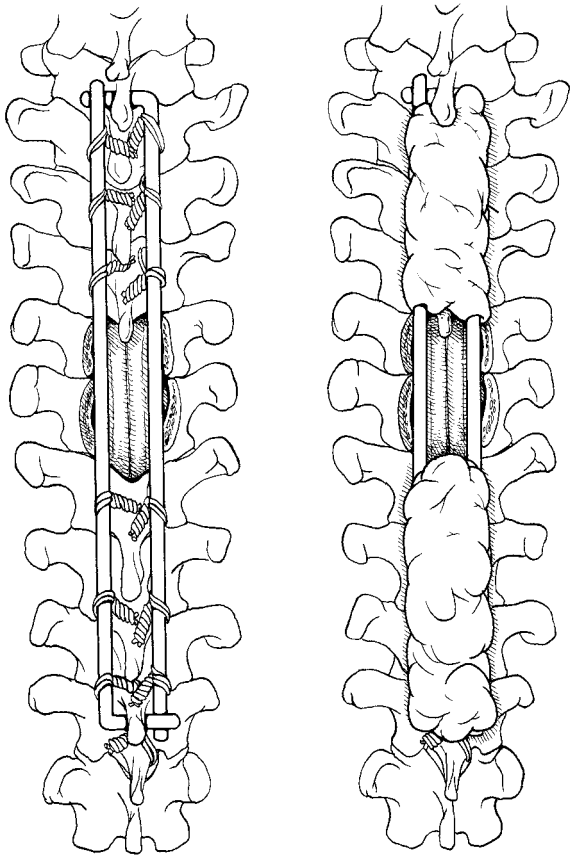


Fig. 12 For posterior stabilization, the Luque rods are cut to appropriate lengths, interdigitated along the laminar sulcus, and secured by doubled 16-gauge wires at each level **(left).** Stability above and below the laminectomy can be enhanced by packing methylmethacrylate into the areas of wire-rod fixation **(right).** This forms a rigid construct that allows sublaminar wire fixation at any single level to reinforce every other level.

all tumor tissue. Only by performing a complete vertebrectomy can the surgeon be sure of removing every bit of debris forced into the spinal canal by the posterior vector of the kyphotic deformity. The anterior two thirds of the vertebra can be removed rapidly with a gouge and rongeur (Fig. 13, C). When only a thin shell of bone and tumor tissue remains in front of the spinal canal, an angled curet is used to avoid inadvertent penetration of the dura or damage to the cord and nerve roots (Fig. 13, D). Great care is taken to decompress the canal completely, using the angled curet to undercut the posterior corners of the intact vertebrae above and below the level of resection.

After complete decompression, a high-speed bur is used to cut a well into the intact vertebral endplates of sufficient depth and width to seat the Knodt rod and hooks (Fig. 13, E). As the rod is twisted, the hooks will become seated firmly into the vertebrae, and the kyphotic angulation will be corrected (Fig. 13, F).

A malleable retractor is placed across the back of the defect to protect the dura from the heat of polymerization and, more important, from compression by the expanding cement mass. Methylmethacrylate then is packed about the rod and hooks and into the defects in the vertebral endplates (Fig. 13, G). Before polymerization is complete, all excess cement is removed from outside the confines of the vertebral bodies. A CT scan of the vertebral construct should show that the cross-sectional diameter of the acrylic-metal construct is nearly identical to that of the normal vertebra, with no encroachment of cement into the spinal canal (Fig. 11, B). In patients who have a good prognosis for prolonged survival and who will not require further irradiation, cancellous autogenous bone or allograft may be packed around the vertebral construct to enhance the likelihood of bony arthrodesis.

The decompression-stabilization procedure in the cervical spine is much simpler than that in the thoracic spine, because an essentially avascular interval is used for the approach between the sternomastoid and carotid sheath laterally and the strap muscles, trachea, and esophagus medially. Ordinarily, the only vascular structure requiring ligation and transection is the middle thyroid vein. The technique for vertebrectomy and distraction-stabilization is similar to that described for the thoracic spine and has been discussed extensively elsewhere.[2,9]

In my experience, the lumbar spine is the least common location for metastatic lesions requiring anterior decompression. This is fortunate, since it is also the area where anterior exposure is most difficult, at least for the L-4, L-5, and S-1 vertebral bodies. Anterior stabilization is also most problematic for these lower lumbar levels. Exposure is best accomplished through a flank incision, paralleling the inferior costal margin. Dissection is retroperitoneal, with the transversalis fascia and abdominal contents being displaced medially until the ureter, vena cava, aorta, and iliac vessels are encountered. In patients who have previously undergone local irradiation, it may be very difficult to mobilize the great vessels overlying the L-4 and L-5 vertebral bodies, and great care must be taken to avoid tearing the vena cava. This approach has also been described extensively elsewhere.[2] As already noted, because

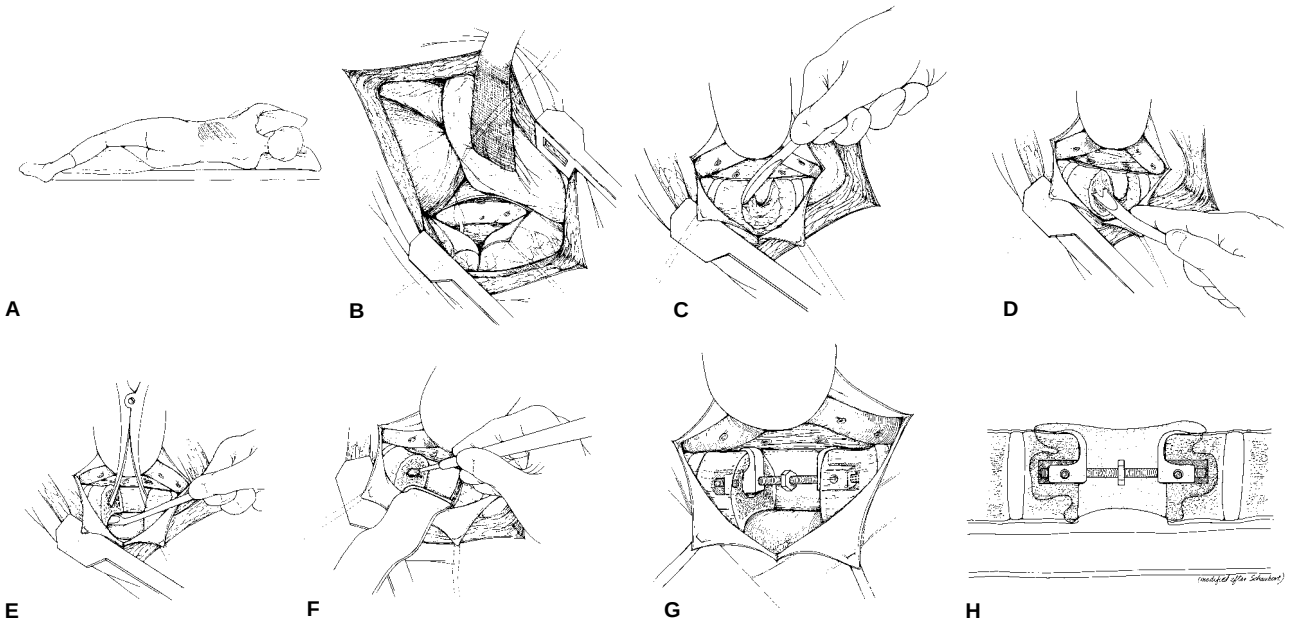


Fig. 13 Technique for anterior decompression and stabilization of the thoracic spine. **A,** Decompression is accomplished by means of a thoracotomy with the patient in the lateral decubitus position. **B,** The aorta is retracted gently, the segmental vessels are ligated and transected, and the affected vertebral body is easily approached. The presence of a prominent paravertebral extrapleural tumor mass will often assist in locating the focus of destruction. **C,** Most of the tumor and bone-disk debris can be removed with a small periosteal elevator. **D,** As the level of the posterior cortical margin is approached, further decompression is achieved with an angled gouge. All material adherent to the adjacent vertebral body is removed. **E,** The vertebral space is recreated with a lamina spreader. A small angled curet is used to complete decompression of the spinal canal and to round off the edges of the posterior cortices of adjacent vertebrae. **F,** The endplates of the adjacent vertebrae are undercut with a high-speed bur to allow the ends of the Knodt rod and the bodies of its hooks to be buried within the vertebral bone. **G,** The Knodt rod has been positioned within the resected space. Twisting distracts its hooks, and their bodies become firmly impacted within the adjacent vertebral bone. Only the tips of the hooks extend anterior to the vertebral cortex. **H,** The defect is filled with methylmethacrylate that polymerizes in situ, incorporating the rod and hooks. To avoid compression of the cord, a malleable retractor is placed between the expanding mass and the spinal canal.

of the lordotic configuration of the lumbar spine and because of the torque and lateral bending moments encountered there, I advocate a combination of anterior decompression-stabilization and posterior stabilization for all lumbar spinal metastases requiring surgical treatment.

Results

It is essential to discuss, at least briefly, the overall results for the treatment of patients with spinal instability and neurologic compromise from metastatic malignancy. Only by such an assessment can the reader determine for himself or herself whether the aggressive techniques described here for selected instances of cord and root decompression and for spinal stabilization seem justified.

Frankel et al[10] established a classification system for quantitating neurologic compromise (Table 2). With the use of this system the extent of sensory and motor dysfunction can be conveniently discussed and the results of various treatment regimens can be compared. Although the Frankel classification relates primarily to acute traumatic, rather than gradually progressive, spinal cord compromise, it is nevertheless useful as a means of comparing the efficacy of different techniques for treating metastatic spine disease.

Using this system, Nather and Bose[11] reported that fewer than 5% of patients with Frankel grade A, B, or C lesions recovered normal (grade E) or near-normal (grade D) function after laminectomy decompression. By comparison, in my series of 77 patients treated by the techniques of anterior decompression described herein, 62% improved to the level of either grade D or grade E.[5] Of 14 patients with complete paraplegia or quadriplegia (grade A), eight improved at least two grades, and six regained the ability to walk and have normal bowel and bladder function.[2] The mean postoperative survival period for patients with breast metastases, myeloma, and lymphoma was

approximately 28 months. At the other extreme, patients with lung cancer metastases had a mean postoperative survival period of only 8 months. Nineteen patients survived for more than 4 years postoperatively. Twelve had had major neurologic compromise preoperatively, and all 12 had improved by at least two grades postoperatively. As expected, the long-term survivors had primary malignant conditions with good prognoses for survival, including breast carcinoma in ten patients and multiple myeloma in six.

Table 2
Frankel Classification System for Neurologic Compromise

Grade A	Complete motor and sensory loss
Grade B	Complete motor loss; incomplete sensory loss
Grade C	Some motor function below the level of involvement; incomplete sensory loss
Grade D	Useful motor function below the level of involvement; incomplete sensory loss
Grade E	Normal motor and sensory function

Ten of the 19 survivors required additional operations for the sequelae of other bony metastases, including four with distant spinal metastases and two with late local recurrence. Two patients suffered posterior wound sloughs through previously irradiated tissues. There were no wound-healing problems with anterior spine approaches. My experience seems comparable with that of other clinical investigators who used similar decompression and stabilization techniques.[12-17]

Based on these results, I believe that patients with major neurologic compromise or intractable mechanical spine pain from vertebral collapse or instability should be considered for decompression and stabilization. The majority can be treated with the anterior approach alone. However, my enthusiasm for this procedure must not be construed as an advocacy for surgical management of all spinal metastases. Most patients do not continue to suffer severe pain after vertebral collapse once they have completed an initial period of rest and a course of local irradiation. Most do not experience significant neurologic compromise, and many with spinal involvement, even when associated with severe local pain or neurologic compromise, do not enjoy a sufficiently long life expectancy to warrant operative intervention of this magnitude.

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