10 Kummell's Disease

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Introduction

Kummell's disease is defined as delayed post-traumatic vertebral collapse in an osteoporotic spine. It was first described in 1891 by Dr Hermann Kümmell, a German surgeon who reported six patients with a clinical entity characterized by the development of gradual collapse of the vertebra and dynamic instability, resulting in a progressive painful kyphosis and even paraparesis following an asymptomatic period of months or years after a minor spinal trauma.[1] He hypothesized that "the nutrition of the affected vertebral bodies is injured," leading to avascular necrosis and delayed collapse of the vertebral bodies.[2,3] Subsequent authors, however, questioned the existence of the delayed collapse; they thought that the fracture was missed initially due to the poor quality of the radiographic studies. Hence, they hypothesized the condition to be a pseudarthrosis or non-union of an osteoporotic vertebral fracture (OVF) that is seen as a transverse intravertebral gas/cleft/fluid on plain radiographs and computed tomography (CT) scans.[4,5] However, to date, the pathogenesis remains unclear.

Even though the prevalence of Kummell's disease was low in the early 1900s, it's incidence and prevalence are steadily increasing due to an increase in the aged and osteoporotic population. Currently the incidence in literature varies from 7 to 37% of all vertebral compression fractures (VCFs).[6,7] This chapter reviews the pathogenesis, diagnosis, and management of Kummell's disease.

Etiopathogenesis

Even though the condition was described long ago, there is no clear consensus on its exact pathogenesis even today. Kummell in his earlier reports suggested an inflammatory pathology initiated by the initial traumatic event, which finally led to a delayed vertebral collapse.[5,8] Subsequently, many different hypotheses have been proposed, but the theory of avascular necrosis remains the most prevailing one. Risk factors commonly associated with osteonecrosis of the vertebral body include osteoporosis, steroid medication, radiotherapy, hemoglobinopathies, vasculitides, alcoholism, pancreatitis, cirrhosis, diabetes mellitus, sarcoidosis, Cushing's disorder, Gaucher's disease, and trauma.[9] Even though the presence of osteonecrosis has been proven histopathologically, this cannot establish the causal hypothesis as the osseous necrosis might be a consequence of the vertebral crush following the initial trauma.[10]

Maldague et al described the association of intravertebral vacuum cleft (IVC) with Kummell's disease in 1978.[11] However, it is unclear which is the initial event in the formation of the intravertebral cleft. Some authors have postulated that Kummell's disease is secondary to ischaemic osteonecrosis because they have not found a compression fracture on radiographs taken immediately after the trauma.[3,12] Currently, it is postulated that in elderly individuals who develop an OVF after an initial traumatic event, if there is an impaired healing of the fracture either due to impaired vascular proliferation or due to a vascular insult (secondary to the stretching of segmental artery or fat micro-embolism of the medullary vessels due to the fracture), it leads to osteonecrosis of the vertebral body. The dynamic flexion-extension forces along the fracture plane then cause a nounion of the fracture and the gap gets filled up with a gas (90% nitrogen) from the extracellular fluid.[9] The gas can later get replaced by the fluid due to a position-dependent dynamic process.[13] Some authors have also reported the coexistence of intradiscal gas and IVC, and also hypothesized that the intravertebral gas may be due to the accumulation of gas from the adjacent disc space.[14]

Clinical Features

There are two subsets of patients who present with Kummell's disease. The first subset of patients are those who present with symptoms and signs suggestive of delayed vertebral collapse, i.e., a history of trivial trauma and axial back pain but with an absent or negative initial plain radiographic examination. This is followed by an asymptomatic period of weeks to months following which they present with axial mechanical back pain (aggravated by an upright posture and relieved by lying down), radicular pain, gibbous deformity, and neurological symptoms due to a dynamic, with or without static compression of the spinal cord or cauda equina.[3,12] The second subset of patients are those who initially present with an acute OVF with a positive plain radiographic evidence and are managed conservatively but the fracture fails to unite even after up to approximately 6 months of conservative treatment and the patient has a persistent axial back pain which gets aggravated by an upright posture and relieved by lying down, suggesting a nonunion of the VCF. These symptoms can be associated with the neurological symptoms similar to that of the first subset of patients.[15–17]

Imaging

Intravertebral cleft sign (aka Kummell's sign) is an important finding in Kummell's disease and it has a specificity of 99%, a sensitivity of 85%, and a positive predictive value of 91%.[10] As mentioned earlier, the osteonecrosis leads to nonunion of the fracture and the void gets filled with gas (90% nitrogen) from the extracellular fluid leading to the formation of Kummell's sign and the gas gets replaced by fluid by a time-dependent dynamic process (i.e., prolonged supine positioning).[9]

Plain Radiographs

Serial radiographs aid in the diagnosis of Kummell's disease and they demonstrate the intravertebral cleft as a transverse, linear-to-semilinear, and radio-opaque shadow on anteroposterior radiographs in the central body or adjacent to the endplate.[5,18] This sign can be better appreciated by performing an extension lateral view or supine bolster cross-table lateral view and comparing them with a flexion lateral view or a standing lateral view, respectively.[5] Often in extension, the cleft opens up like a crocodile's mouth and the mouth closes on flexion of the spine.

Computed Tomography

CT shows a better bony morphology of the vertebra and the fractured fragments than plain radiographs, and the diagnostic rate of Kummell's disease increases by 10-fold when compared to plain radiographs.[10,18] The sensitivity further rises when a dynamic (flexion-extension) CT is used.[5]

Magnetic Resonance Imaging

The intravertebral cleft, if filled with gas (vacuum sign), has low signal intensity on T1- and T2-sequences. If the gas is replaced by fluid, there will be a low signal intensity on T1-sequence and high signal intensity on T2- and fat-suppressed sequences (TIRM/STIR). On a contrast-enhanced T1 MR, the cleft area appears nonenhanced with enhancement seen in the surrounding vertebral body. MRI can also demonstrate a double-line sign on T2-sagittal sequence characterized by a hypointense line corresponding to the vacuum cleft surrounded by a hyperintense signal corresponding to the intraosseous oedema.[6]

Bone Scintigraphy

Even though its findings are nonspecific, bone scintigraphy is considered one of the sensitive tools for the early diagnosis of osteonecrosis in Kummell's disease when the plain radiographs are normal. Like the MRI, bone scintigraphy helps in determining the chronicity of the lesion. However, with the routine use of MRI, bone scintigraphy has lost its reputation in the diagnosis of these lesions. But some authors believe that the activity on bone scintigraphy is more accurate in evaluating old fractures (>3–4 months) when compared to the edema

on MRI, as such lesions may respond to cement augmentation procedures.[8,19]

Classification

Li et al[20] have proposed a three-stage classification for Kummell's disease based on plain radiographic and MRI findings (**Table 10.1**). The sequential stages of the disease include:

- **Stage 1:** This stage is characterized by an absent or less than 20% of anterior vertebral wedging without Kummell's sign and dynamic mobility on plain radiographs. MRI, however, shows Kummell's sign with an inhomogeneous fluid signal on the MR T2-sequence (**Fig. 10.1a**). Clinically the patient may be asymptomatic or may have an axial back pain.
- **Stage 2**: This stage is characterized by an anterior vertebral wedging greater than 20% with Kummell's sign and dynamic mobility on plain radiographs and an intact posterior vertebral wall. The MRI shows Kummell's sign with homogeneous or inhomogeneous fluid signal on the T2-sequence (**Fig. 10.1b**). Clinically, the patients have axial back pain, radiculopathy, and a kyphotic deformity.
- **Stage 3:** This stage is characterized by a severe anterior vertebral wedging with Kummell's sign and dynamic mobility on plain radiographs associated with the breakage of the posterior wall. MRI shows a retropulsed posterior wall fragment causing cord or cauda equina compression and Kummell's sign with homogeneous or inhomogeneous fluid signal on the MR T2-sequence (**Fig. 10.1c**). Clinically, the patients have axial back pain, radiculopathy, and a kyphotic deformity and an associated neurological deficit due to cord or root compression.

Management Options

There is no effective conservative treatment for Kummell's disease. Surgical treatment is recommended for patients presenting with

Table 10.1 Various stages of Kummell's disease, as proposed by Li et al[20]

S.no	Stage	Plain radiographic features	MRI features	Treatment
1	I	Absent or <20% anterior vertebral wedging without Kummel sign and dynamic mobility	Kummell sign present	Percutaneous cement augmentation
1	II	Anterior vertebral wedging greater than 20% with Kummell sign and dynamic mobility	Kummell sign present	Percutaneous cement augmentation
3	III	Severe anterior vertebral wedging with Kummell's sign and dynamic mobility on plain radiographs associated with the breakage of the posterior wall	Kummell sign and retropulsed posterior wall fragment causing cord or cauda equina compression	Stand-alone cement augmentation with egg shelling technique or Egg shelling cement augmentation + posterior stabilization or Transpedicular vertebral body augmentation + posterior stabilization

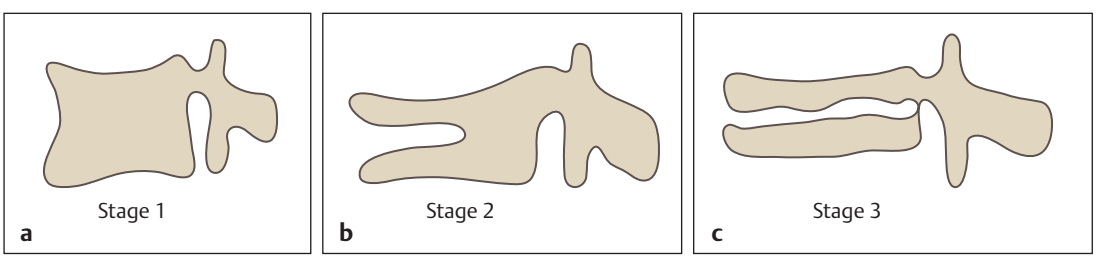

Fig. 10.1 (a–c) Images showing stage 1, stage 2, and stage 3 of Kummell's disease.[18]

severe pain, deformity, or neurological deficit.[21] However, anti-osteoporotic treatment along with calcium and vitamin D supplementation must be started for all patients who suffer from OVFs. They should be encouraged to consume a healthy diet and regular weight-bearing aerobic conditioning exercises to prevent secondary osteoporosis. The surgical approach depends on the presence or absence of neurological deficit.[20,22]

Stages 1 and 2

These are usually not associated with neurological deficit and can be managed by percutaneous cement augmentation techniques (vertebroplasty or kyphoplasty) (**Fig. 10.2**).[20]

Stage 3

Kummell's Disease without Neurological Deficit

The goal of surgery in these patients is to provide adequate stability for pain relief and to permit early mobilization. Stability may be provided by anterior column reconstruction alone with percutaneous cement augmentation procedures such as vertebroplasty or kyphoplasty performed by a standard transpedicular technique or by using an egg-shelling technique. Egg-shelling technique is adopted when there are posterior vertebral wall defects or an endplate violation that occurs during balloon inflation. A small amount of doughy-consistent cement is injected under fluoroscopy control into the cleft and balloons are inflated and left in place until the cement outside hardens. Then the balloons can be deflated and a routine low-pressure cement injection can be done under fluoroscopy guidance (**Fig. 10.3**).[23]

Some surgeons have reported persistent back pain even after vertebroplasty in patients with Kummell's disease and have attributed it to inadequate stability due to lack of cement interdigitation with the bone.[24,25] Others have reported recurrent back pain and progressive vertebral collapse, a few weeks/months following cement augmentation.[26,27] These surgeons recommend additional posterior instrumented stabilization along with cement augmentation of the fractured vertebra to provide better pain relief and more consistent long-term outcomes.[28]

There are many retrospective level 3 and 4 studies evaluating the role of percutaneous cement augmentation procedures (vertebroplasty or balloon kyphoplasty) in patients with Kummell's disease without a neurological deficit (**Table 10.2**).[29–34] Retrospective

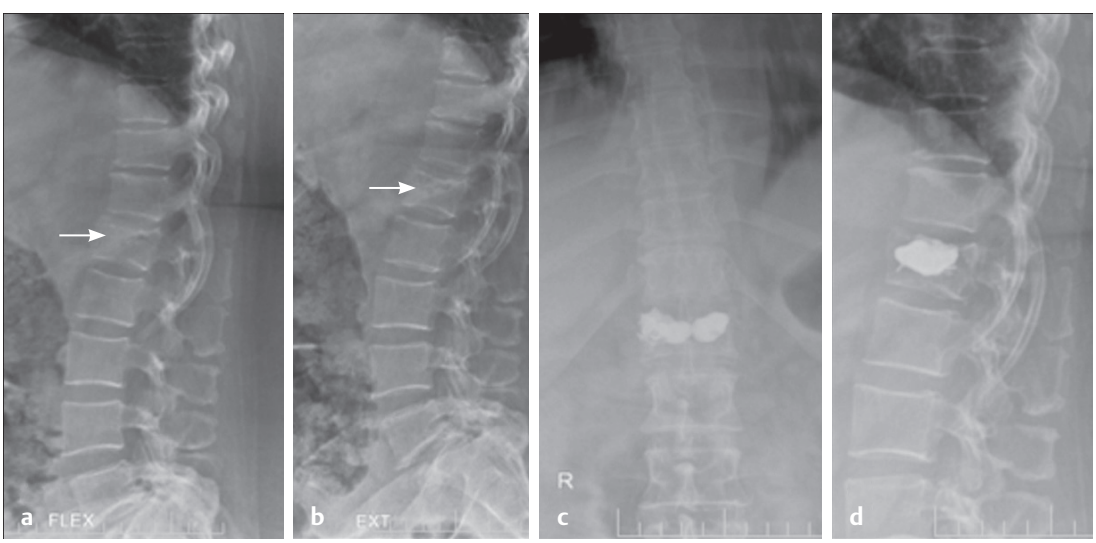

Fig. 10.2 (a–d) Images showing preoperative stage 2 Kummell's disease of the L1 vertebra (arrow indicating the vacuum sign) **(a)** lateral flexion and **(b)** extension radiographs, **(c)** postop anteroposterior and **(d)** lateral radiographs after cement augmentation with balloon kyphoplasty procedure.

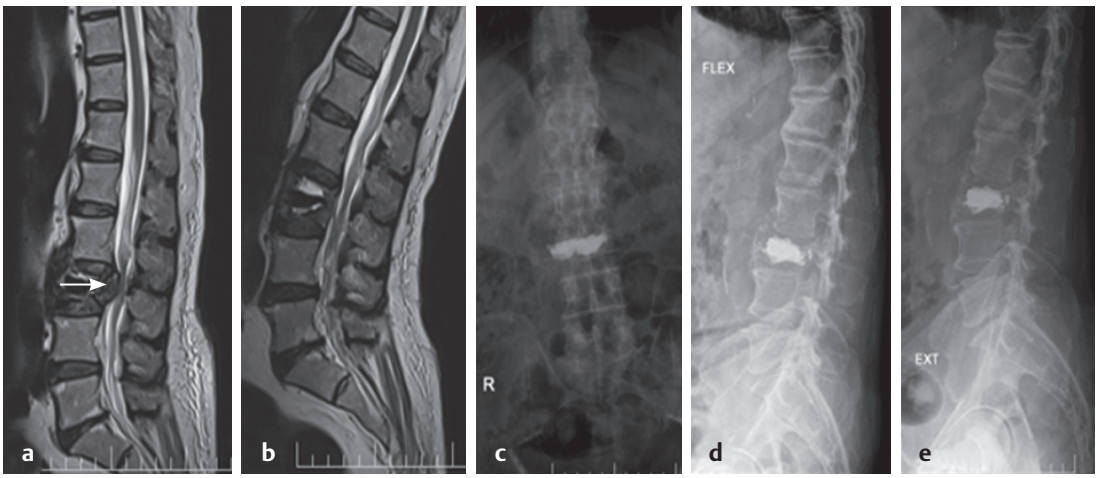

Fig. 10.3 (a–e) Image showing a 70-year-old patient with stage 3 Kummell's disease of L3 vertebra and an intact neurology with preoperative **(a)** T2-weighted sagittal flexion MRI (arrow indicating the retropulsed fragment) and **(b)** T2-weighted sagittal extension MRI. **(c)** Postop anteroposterior, **(d)** lateral flexion, and **(e)** extension radiographs after cement augmentation using egg-shelling balloon kyphoplasty procedure.

cohort studies (level 3 evidence) comparing stand-alone cement augmentation to cement augmentation supplemented by posterior stabilization did not find significant differences among both these procedures at final follow-up (2 years). These stand-alone cement augmentation procedures had less blood loss, less surgical time, and less postoperative complications when compared to the cement augmentation supplemented by posterior stabilization.[29,35] The patient population that may benefit from additional stabilization in Kummell's stages 1, 2, and 3 has not been clearly defined, and so the decision to perform additional posterior stabilization in stages 1, 2, and 3 without neurological deficit depends on the treating surgeon's preference and is made on a case-to-case basis.

Kummell's Disease with Neurological Deficit

The goals of surgery in patients with Kummell's disease with a neurologic deficit are decompression of the neural structures, stabilization of the pathological spinal segments, and restoration of the weight-bearing mechanics of the anterior spinal column.

- Static compression of the neural structures occurs due to either retropulsion of the posterior wall of the vertebral body or epidural soft tissue at the site of vertebral pseudarthrosis. Decompression is achieved indirectly via laminectomy or directly by performing a vertebrectomy either from an anterior or posterolateral approach.
- Stabilization of the spine is essential to relieve dynamic spinal cord compression that occurs due to micromotion at the pseudarthrosis site, to provide pain relief, and to prevent progression of kyphosis. Stabilization can be done anteriorly or posteriorly. Posterior fixation is preferred because it is more rigid, allows three-column purchase and is easy to extend up or down by one or more levels. Longer constructs that enhance stability are usually preferred. Alternatively, one may use cement-augmented pedicle screws to enhance pullout strength to permit shorter constructs. Anterior screws have poor purchase as they are inserted into the cancellous, osteoporotic bone of the vertebral body.
- Reconstruction of the anterior column is essential to restore the weight-bearing mechanics of the spine to provide adequate stability to prevent failure of fixation in the long term. Anterior reconstruction is achieved by inserting a cage filled with bone graft into the vertebrectomy defect or by cement augmentation

Table 10.2 Studies managing Kummell's disease without neurological deficit with stand-alone vertebroplasty

Sl. No	Study	Level of evidence	Sample size	Duration of Kummell's	Surgery performed	Pain relief	Early complications	Late complications	Conclusion
1	Chen et al[30]	4 (retrospective case series)	27 pts	At least 3 months after failure of conservative management	Vertebroplasty (via uni-pedicular approach)	Mean reduction of VAS score by 40 points (100 pt score) on first postoperative day	No major perioperative complication	Two patients developed adjacent segment fractures	Stand-alone vertebroplasty is an effective treatment in managing pseudoarthrotic clefts without neurological deficit
2	Chen et al[29]	3 (retrospective cohort)	54 pts	2–9 months	Group 1 (kyphoplasty): 31 Group 2 (short segment stabilization plus vertebroplasty): 23	Both groups had a significant reduction of VAS score at early postop and final follow-up period	Asymptomatic cement leakages in both groups (intradiscal, paravertebral, and intra canal)	Implant failure due to loosening of screws in one patient of group 2	Balloon kyphoplasty shows similar results as that of short segment stabilization plus vertebroplasty in patients with Kummell's disease without neurological deficit
3	Wang et al[31]	4 (retrospective case series)	12 pts	1 month to 2 years	Kyphoplasty	There was a significant improvement in VAS scores at early postop and final follow-up periods	Asymptomatic cement leakage – 3 pts	None	Kyphoplasty is safe and effective in the management of Kummell's disease
4	Xia et al[32]	4 (retrospective case series)	50 pts	1–12 months	Kyphoplasty	Significant reduction of VAS score in the early postop and final follow-up periods	Asymptomatic cement leakages in 8 pts	None	Kyphoplasty is effective for relieving pain in Kummell's disease with minimal complications
5	Huang et al[33]	4 (retrospective case series)	18 pt	1 month–3 years	Balloon kyphoplasty in hyperextension	Significant relief of pain in the early postop and final follow-up period	Asymptomatic cement leakage in one	None	Kyphoplasty is safe and effective in providing pain relief in patients with Kummell's disease without neurological deficit
6	Kim et al[34]	4 (retrospective case series)	32 pts	Not mentioned	Vertebroplasty	Significant relief of pain in the early postop and final follow-up period	Asymptomatic cement leakage in 4 pts	None	Vertebroplasty is effective for reducing the pain associated with avascular necrosis of vertebral body

of the vertebra. Inserting a cage can be a problem, as it often erodes the weak, osteoporotic vertebral endplates and subsides into the vertebral body, resulting in non-union and recurrent deformity. An alternative procedure is cement augmentation of the vertebral body. However, in patients where the posterior vertebral wall is deficient, cement can leak into the epidural space. The egg-shelling technique has been described to limit this problem.[23] Zhang et al have described a posterior transpedicular subtraction and disc osteotomy to compress the vertebral body across the pseudarthrosis site.[36] The compacted (shortened) vertebral body with bone-to-bone contact has little room for further vertebral collapse and offers on-table stability. Li et al have described a transpedicular reconstruction of the vertebral body using intracorporeal bone graft and a titanium spacer into the pseudarthrosis gap.[22]

Surgery for Kummell's stage 3 with neurological deficit can be performed via anterior, posterior, or combined approaches.[20,37–39]

Anterior surgery permits decompression, anterior column reconstruction, and instrumented stabilization to be performed through a single approach. However, anterior surgery in elderly individuals with multiple medical comorbidities is often fraught with perioperative complications. Since most fractures occur at the thoracolumbar junction, anterior surgery often involves taking down the diaphragm which may be dangerous in patients with preexisting poor pulmonary reserve.[37] Besides, as mentioned earlier, anterior cages tend to subside and anterior screws have poor purchase in weak, osteoporotic vertebral bone.

A combined surgery involves anterior decompression and reconstruction followed by posterior stabilization using pedicle screws to enhance construct rigidity and pullout strength. However, this involves two different approaches with longer surgical time, more blood loss, a higher infection rate, and the associated complications of both the approaches. Kashii et al have shown that combined approaches have a significantly higher morbidity compared to posterior approach alone in elderly individuals with OVFs.[39] Currently, a combined approach is employed mostly within the lumbar spine where insertion of a cage following vertebrectomy is difficult because of the limitations imposed by the lumbar nerve roots that cannot be sacrificed.

Currently, the posterior-alone approach is preferred in most patients with pathology in thoracic spine and the thoracolumbar junction. Posterior stabilization is performed using pedicle screws, hooks, and/ or sublaminar wires to restore adequate stability and maximize pullout strength. Using cement-augmented screws to improve pullout strength, the fixation can be limited to one or two levels cephalad or caudad to the Kummell's lesion. Since the neurological deficit is mainly due to dynamic compression which is largely relieved by strong posterior fixation, adequate decompression is usually achieved indirectly with a laminectomy (**Fig. 10.4**). Direct decompression, if necessary can be performed via posterolateral transpedicular or costotransversectomy portal. Anterior reconstruction can also be satisfactorily achieved from posteriorly with any of the options mentioned earlier in the chapter. A posterior vertebral column resection procedure is associated with increased surgical time and blood loss that is usually not well-tolerated in elderly frail patients[40] (**Fig. 10.5**).

In a retrospective cohort study comparing direct decompression by an anterior only procedure, direct decompression by a posterior-only procedure, and an indirect decompression with limited laminectomy at the involved level and a posterior short segment stabilization supplemented with cement augmentation of the involved vertebra showed no clinical differences in pain score, neurological scores, and activities of daily living scores among all three procedures.[39] As the neurological deficit in patients with Kummell's disease is predominantly due to the dynamic instability, the authors feel that direct decompression of neural elements is not always necessary and a less invasive procedure with a short-segment stabilization, limited laminectomy, and anterior augmentation by vertebroplasty or an intracorporeal bone grafting could provide promising results.

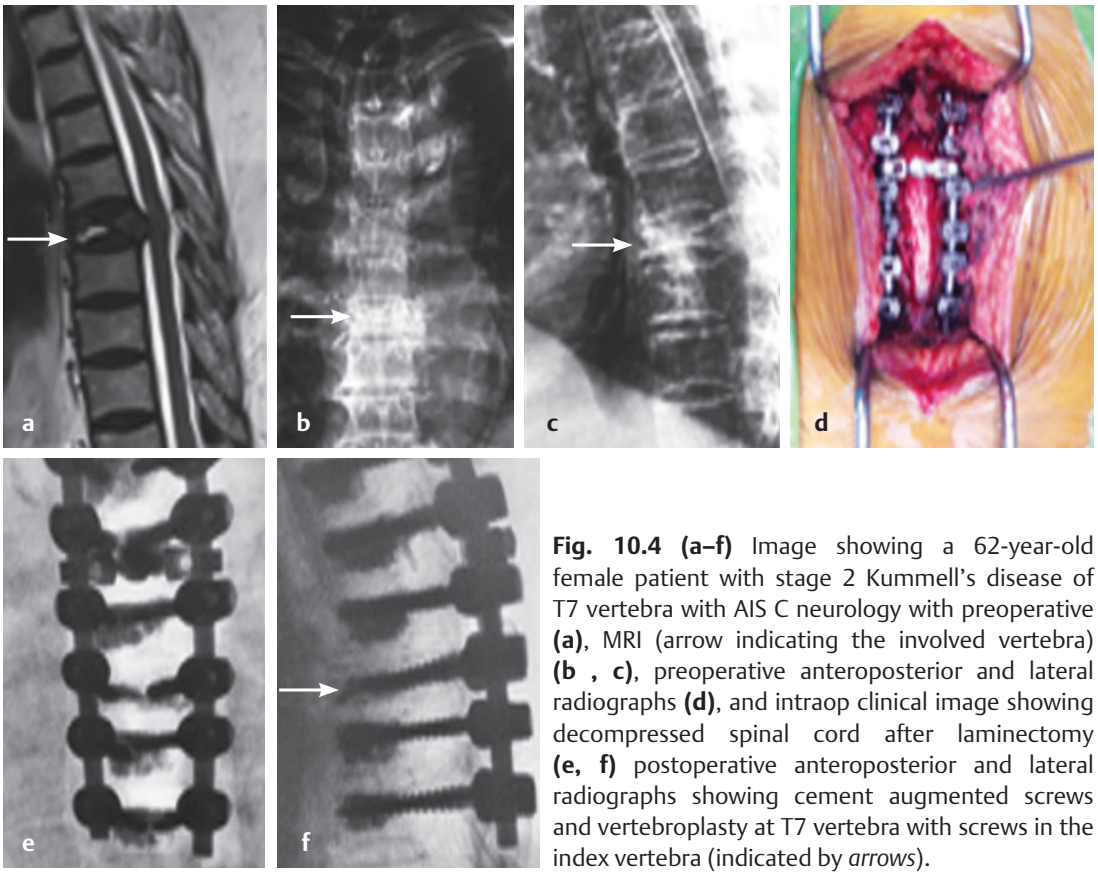

Fig. 10.4 (a–f) Image showing a 62-year-old female patient with stage 2 Kummell's disease of T7 vertebra with AIS C neurology with preoperative **(a)**, MRI (arrow indicating the involved vertebra) **(b , c)**, preoperative anteroposterior and lateral radiographs **(d)**, and intraop clinical image showing decompressed spinal cord after laminectomy **(e, f)** postoperative anteroposterior and lateral radiographs showing cement augmented screws and vertebroplasty at T7 vertebra with screws in the index vertebra (indicated by *arrows*).

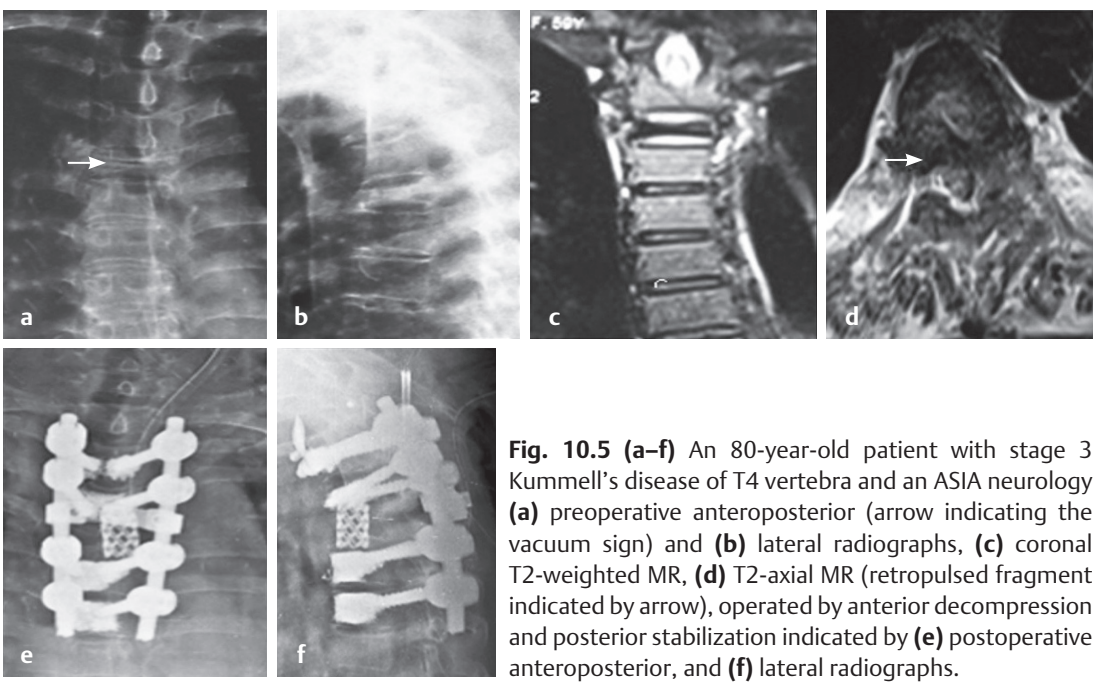

Fig. 10.5 (a–f) An 80-year-old patient with stage 3 Kummell's disease of T4 vertebra and an ASIA neurology **(a)** preoperative anteroposterior (arrow indicating the vacuum sign) and **(b)** lateral radiographs, **(c)** coronal T2-weighted MR, **(d)** T2-axial MR (retropulsed fragment indicated by arrow), operated by anterior decompression and posterior stabilization indicated by **(e)** postoperative anteroposterior, and **(f)** lateral radiographs.

Conclusion

Kummell's disease reflects delayed post-traumatic collapse of an osteoporotic vertebral body. Patients typically present with mechanical back pain and pain radiating anteriorly along the chest wall or abdomen. Pain typically aggravates with movement. Neurological deficit may occur in a small percentage of patients, primarily due to dynamic spinal cord compression. The diagnosis is best made on CT scans and MRI, where the Kummell's lesion is seen as a horizontal or semilunar cleft filled with gas or fluid.

Management of Kummell's lesions is primarily surgical. For stages 1 and 2 lesions, cement augmentation by vertebroplasty or kyphoplasty is the procedure of choice. For stage 3 lesions without neurological deficit, posterior stabilization and anterior reconstruction with cement augmentation is preferred. Alternative methods of anterior reconstruction include insertion of an interbody cage, intracorporeal bone grafting, and posterior column shortening. For stage 3 lesions with neurological deficit, additional laminectomy or corpectomy may be necessary to treat the static spinal cord compression.

Key Points

- A rise in the aged and osteoporotic population has led to an increase in the incidence and prevalence of Kummell's disease.
- Kummell's disease is essentially a non-union of the osteoporotic vertebral body that results in delayed collapse.
- Patients with Kummell's disease typically present with severe axial back pain and pain radiating anteriorly along the chest wall. The pain is aggravated with movement. Other clinical presentations include kyphotic deformity and/or neurological deficit.
- Li et al classified Kummell's disease into three types based on the plain radiographic and MRI findings.
- For stages 1 and 2, cement augmentation with vertebroplasty or kyphoplasty is the preferred treatment.
- For stage 3 without neurological deficit, posterior stabilization with cement augmentation is recommended.
- Dynamic spinal cord compression in patients with Kummell's disease is treated with stabilization. Static compression can be dealt with directly via vertebrectomy or indirectly via laminectomy.
- A less invasive procedure such as an indirect decompression along with a short segment posterior stabilization with an intracorporeal bone grafting or vertebroplasty of the involved vertebra gives similar clinical results like a relatively morbid anterior-only or a combined procedure in patients with Kummell's disease who have neurological deficit.

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