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Clinical Report

Spine Fracture with Neurological Deficit in Osteoporosis

M. H. Heggeness

Center for Spinal Disorders, Baylor College of Medicine, Houston, Texas, USA

Abstract. The literature suggests that spinal fractures acquired after minimal trauma in osteoporotic patients cause neurological problems only extremely rarely. This report describes 9 patients with severe osteoporosis in whom minimal trauma led to a fracture of the thoracic or lumbar spine causing significant neurological deficit. All patients presented originally with isolated back pain after minimal trauma. Initial radiographs documented what appeared to be 'benign' compression fractures with minimal loss of vertebral height. Over the following 1–12 weeks all patients described a gradual onset of severe radicular pain which was subsequently associated with profound lower extremity weakness in 7 cases. Repeat radiographs revealed advanced collapse of the fractured level. CT and MRI images revealed violation of the posterior cortex of the vertebrae with retropulsion of bone into the spinal canal. In 2 cases, two simultaneous fractures of this kind were noted; in both of these cases the fractures occurred at adjacent levels. The upper lumbar spine was most frequently involved. This relatively small series suggests that caution should be used in the assessment of benign-appearing compression fractures in osteoporotic patients. The delayed appearance of neurological signs and symptoms may lead to a late or missed diagnosis. The onset of back pain in an elderly patient quite frequently represents the acquisition of a fracture; subsequent complaints of leg pain or lower extremity dysfunction may indicate progression of such a fracture, with compromise of the neutral elements.

Correspondence and offprint requests to: M. H. Heggeness, MD, PhD, Department of Orthopaedic Surgery, Center for Spinal Disorders, Baylor College of Medicine, 6550 Fannin Suite 2501, Houston, TX 77030, USA.

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Introduction

Osteoporotic spine fractures are a major source of morbidity in the elderly [1–6]. While the severe morbidity associated with osteoporotic hip fractures is well known [5,7,8], the epidemiology and clinical significance of vertebral fractures is not well established [1,3,9]. Some osteoporotic vertebral fractures are asymptomatic, but many such fractures acquired after minimal trauma do cause profound pain and morbidity. Such fractures also contribute to problems of deformity [1,2,9].

The literature suggests that while the incidence of a vertebral fracture in the aging population is very high, such fractures have been noted to cause neurological deficits extremely rarely [1,2,10]. Indeed, previous literature from North America on this phenomenon consists entirely of isolated case reports [11–13]. Interestingly, the Japanese authors Shikata et al. [14] and Kaneda et al. [15] have described respectively, 7 and 22 osteoporotic patients who had neuroological deficits secondary to vertebral fractures acquired after minimal trauma. Both groups of authors report excellent results following surgical treatment of these injuries. This literature has suggested that such injuries may be more common in the Oriental population than in North America. Both Shikata and Kaneda advocated operative treatment of these fractures and all of their patients were so managed. This retrospective report describes 9 Caucasian patients with osteoporosis, in whom minimal trauma led to a fracture of the lower thoratic or lumbar spine which resulted in significant neurological deficit.

Three of these patients were managed operatively with anterior decompression and instrumentation; 6 were managed non-operatively. All patients recovered from their neurological deficits.

Materials and Methods

This is a retrospective study. All patients presented to the author's orthopaedic surgery practice at Baylor College of Medicine Center during an 18-month period. Definitive management of all patients was conducted by the author.

Bone mineral density measurements were obtained using a Hologic 2000 dual-photon instrument.

Clinical Histories

Demographic data was presented in Table 1. Of the 9 patients 3 were male and 6 were female. Ages varied from 49 to 93 years. Etiological risk factors for osteoporosis included severe alcoholism ($n = 2$), chronic corticosteroid use ($n = 1$) and Waldenström's macroglobulinemia ($n = 1$). The remaining patients, had no identifiable risk factors ($n = 5$). Osteoporosis was documented in all patients on the basis of bone mineral density measurement greater than 2 standard deviations below age-matched values or documented multiple (previous) vertebral fracture.

Only 3 patients recalled a specific incident of trauma. In 2 cases this consisted of a fall from the standing position onto a carpeted floor. Another patient related

Table 1. Demiographic data

Case	Age/ sex	Osteoporotic risk factors	Mechanism of injury	Fractured level	Delay from injury to onset of lower extremity symptoms	Neurological deficit	Treatment	Follow-up period (months)	Functional status
1	73 F	None	Atraumatic	T9	6 weeks	Profound lower extremity weakness bilaterally (unable to resist gravity with any lower extremity motors). Sensory level at T9	Anterior surgical decomposition and fusion with instrumentation	21	Neurologically intact, fully ambulatory
2	82 F	None	Atraumatic	L4	3 weeks	Severe L4 and L5 radiculopathy with ankle and toe weakness	Orthoplast jacket	21	Neurologically intact, fully ambulatory
3	70 F	Rheumatoid arthrisis, chronic corticosteroid use	Fall onto carpeted floor	L2, L3	2 weeks	Severe, diffuse right leg pain. Profound weakness of right leg (unable to resist gravity with any motors)	Anterior surgical decompression and fusion with instrumentation	19	Neurologically intact, ambulatory with assistance
4	78 F	None	Atraumatic	L1, L2	2 weeks	Severe left leg pain. Objective weakness in left hip flexors (3/5), quadriceps (4/5) and all ankle motors (3/5)	Intravenous corticosteroids and bedrest	15	Neurologically intact, but non-ambulatory
5	66 F	Alcoholism, cirrhosis and ascites	Atraumatic	L1	11 weeks	Severe bilateral leg pain. Bilateral hip flexor weakness (4/5)	Orthoplast jacket	15	Neurologically intact, fully ambulatory
6	49 M	Alcoholism	Lifting suitcase	L3	10 days (patient experienced alcohol withdrawal seizure in the interim)	Severe radicular pain in an L3 distribution. Profound right hip flexor weakness (2/5)	Anterior surgical decompression and fusion	14	Neurologically intact, fully ambulatory
7	78 M	Waldenström's macro-globulinemia	Atraumatic	L2	8 weeks	Severe bilateral leg pain. Mild (4/5) weakness in hip flexors bilaterally and in right quadriceps	Orthoplast jacket	19	Neurologically intact, fully ambulatory
8	93 F	None	Atraumatic	L2	6 weeks	Severe bilateral leg pain, subjective weakness (motor exam. objectively intact). Decreased sensation in both lower extremities with L2 sensory level	Intravenous corticosteriods, orthoplast jacket	19	Neurologically intact, fully ambulatory
9	77 M	None	Fall onto carpeted floor	L1	10 days	Bilateral pain. Weakness in the left hip flexors (4/5) and left quadriceps (3/5)	Orthoplast jacket	10	Neurologically intact with return of strength and abatement of pain; however, no longer ambulatory

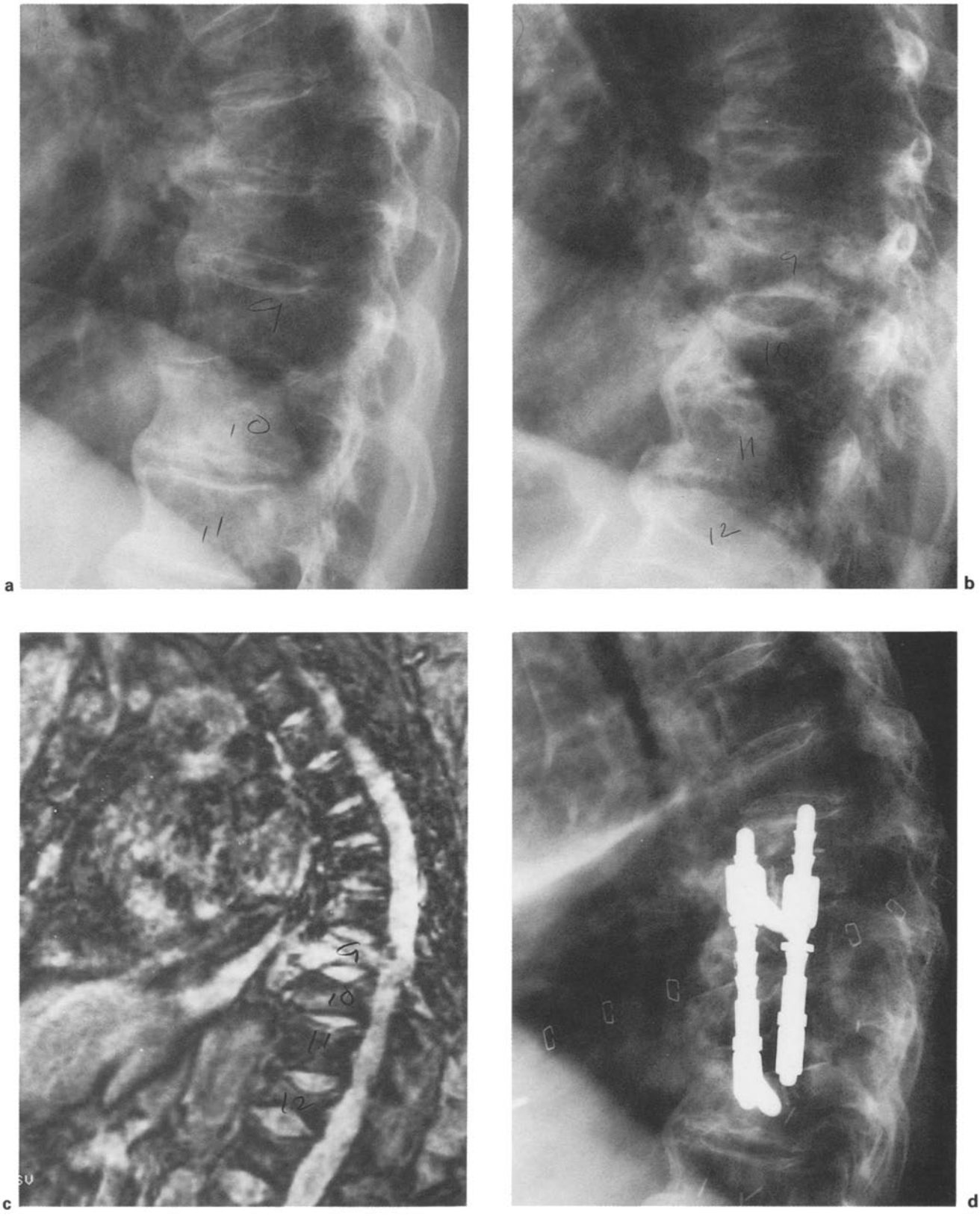

Fig. 1. Patient 1. **a** Lateral thoracic spine radiograph on initial presentation. Note mild collapse of the superior and inferior endplates of T9. **b** Lateral thoracic spine radiograph obtained 3 months following injury. Note the collapse of T9. **c** MRT scan of the thoracic spine 3 months following intitial injury. Note a significant quantity of bone retropulsed into the canal. Several other (non-acute) endplate fractures are noted through the spine. **d** Lateral radiograph of the thoracic spine following anterior spinal decompression, fusion and instrumentation with a Kostuik–Harrington device.

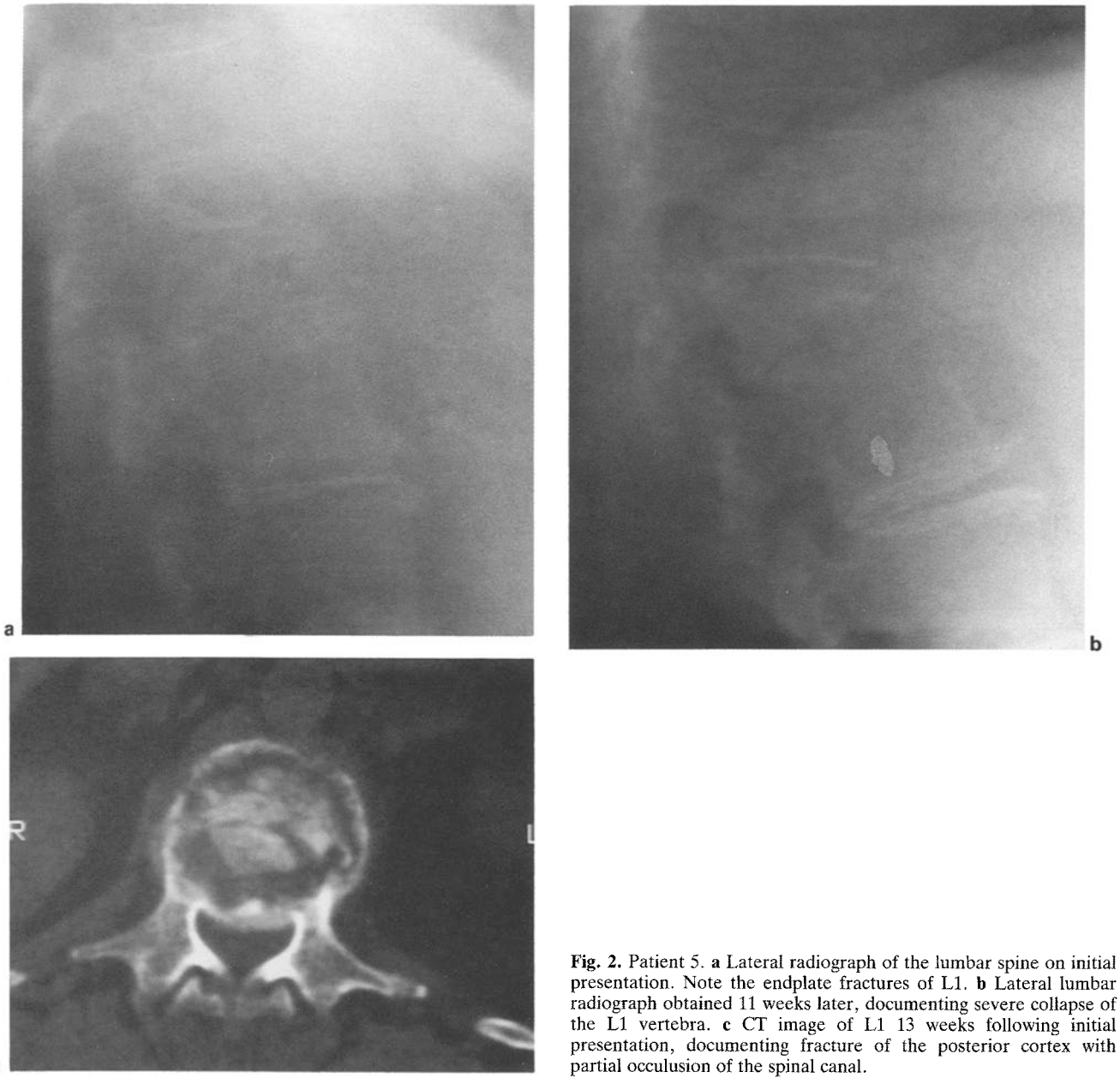

Fig. 2. Patient 5. **a** Lateral radiograph of the lumbar spine on initial presentation. Note the endplate fractures of L1. **b** Lateral lumbar radiograph obtained 11 weeks later, documenting severe collapse of the L1 vertebra. **c** CT image of L1 13 weeks following initial presentation, documenting fracture of the posterior cortex with partial occulusion of the spinal canal.

the abrupt onset of back pain to bending over to lift a suitcase. The majority of fractures occurred in the upper lumbar spine; interestingly four of the fractures occurred at L2. In 2 patients, two acute fractures each causing spinal canal encroachment were documented.

All patients described isolated back pain as their initial complaint. Compression fractures were documented in all 9 patients on initial presentation (Figs. 1–3).

Lower extremity pain and weakness developed after an interval of from 10 days to 12 weeks. All patients described the insidious onset of radicular-type lower extremity pain which in every case preceded the onset of motor weakness. Although the degree of motor deficit noted in these patients varied widely, none of 9 patients was able to walk because of lower extremity pain and/or weakness. No patient lost bowel or bladder control.

Repeat radiographs on all patients revealed progressive collapse of the fractured levels. CT and MRI images (Figs. 1–3) revealed violation of the posterior cortex of the vertebral bodies with retropulsion of bone into the spinal canal. In all cases the spinal canal appeared to be compromised by 30%–50% compared with the cross-sectional area of the vertebral canal above the level of the injury.

All patients in this series were investigated extensively to rule out metastatic disease to the spine and underwent a bone scan, serum protein electrophoresis and extensive blood chemistry tests. No evidence of malignancy was found in any patient, nor has any

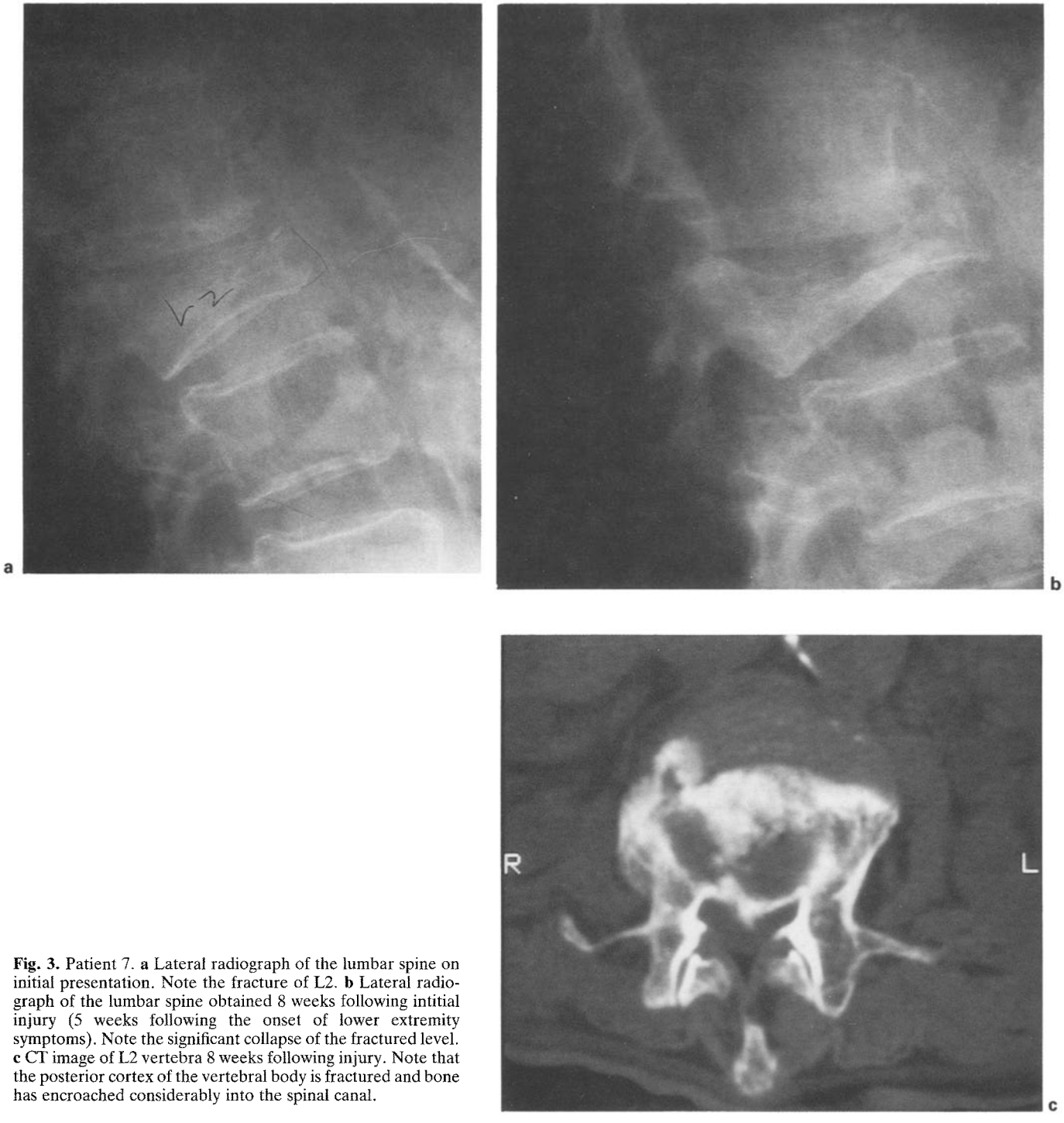

Fig. 3. Patient 7. **a** Lateral radiograph of the lumbar spine on initial presentation. Note the fracture of L2. **b** Lateral radiograph of the lumbar spine obtained 8 weeks following intitial injury (5 weeks following the onset of lower extremity symptoms). Note the significant collapse of the fractured level. **c** CT image of L2 vertebra 8 weeks following injury. Note that the posterior cortex of the vertebral body is fractured and bone has encroached considerably into the spinal canal.

problem of malignancy developed in the subsequent care of these patients (10–21 months).

Treatment decisions on these debilitated patients were difficult because of limited literature on these injuries. On the basis of very profound and evolving deficits, 3 patients were treated surgically by anterior retroperitoneal or transthoracic vertebral corpectomy, spinal canal decompression, fusion, and intrumentation [16]. Such extensive surgical procedures involved considerable risk for these older debilitated patients, and the surgical insertion of the rods was made technically challenging because of the severely osteoporotic bone. Nonetheless, all 3 patients treated surgically did extremely well with full return of neurological function. All 3 patients so treated remained ambulatory at last follow-up, although 1 patient with severe rheumatoid arthritis and corticosteroid myopathy requires ambulatory assistance.

The remaining 6 patients were managed non-operatively with an interval of bedrest followed by careful,

supervised return to activity while immobilized in a custom-fitted Orthoplast jacket. Non-operative management was recommended for patients with significant associated medical problems which were judged to make surgical treatment an unacceptable risk, and for those patients in whom the motor deficits were relatively minor. These patients with 'minor' deficits were judged to be capable of ambulation were it not for the radicular pain. All of these patients have resolved their complaints of radicular pain and recovered essentially all of their objective neurological function, usually over an interval of 1–3 months. Unfortunately, 2 of the 6 patients so managed are now unable to walk: 1 is severely involved with Parkinson's disease with advanced dementia; the other is noted to be extremely weak systemically and was in fact only very minimally ambulatory prior to her fracture.

Discussion

Understanding of the clinical problem of osteoporotic spinal fractures has been hampered in the past by the lack of a standardized nomenclature of these fractures [17]. The natural history of the fracture process is not well understood, despite some excellent investigations [1,2,18,20].

A critical feature of any spinal fracture is the integrity of the posterior cortex of the vertebral body. Any fracture which involves the posterior cortex of the vertebral body creates a potential for retropulsion of bone into the spinal canal and subsequent neurological injury [21]. It should be noted, however, that the spinal canal may accommodate a substantial amount of encroachment before any neurological deficits result [21–23]. It is probable that the morphology,mechanical behavior, and clinical significance of these fractures may be better understood in the future if they are more critically studied prospectively by CT and MRI techniques, as well as by electrodiagnostic techniques.

The posterior cortex and posterior annulus, referred to as the 'middle column' of the spine, is regarded as critical to the stability of the spine. In his classic review of 412 thoracolumbar fractures, Denis [21] concluded that fractures which spare the posterior cortex of the body are unlikely to undergo collapse or displacement. It is important to note, however, that patients with osteoporosis were specifically excluded from his series. It is possible, on the basis of the present clinical series, that Denis's guidelines do not apply to osteoporotic patients. Unfortunately, the status of the posterior cortex of the vertebral body at the time of the original fracture event is unknown in these patients, as none underwent CT or MRI imaging acutely. The example of these 9 patients suggests the possibility that in osteoporotic patients a fracture of the anterior body of the vertebra may in some cases lead to a delayed failure of the posterior cortex, and subsequently to compromise of the neural elements.

All of the patients presented originally with isolated back pain after minimal trauma. Initial radiographs documented what appeared to be 'benign' compression fractures without severe loss of vertebral height. Over the 1–12 weeks following injury, all 9 patients described the gradual onset of severe radicular pain associated with lower extremity motor weakness. Repeat radiographs revealed advanced collapse of the fractured level. CT or MRI images revealed encroachment of bone into the spinal canal. A consistent feature of all patients in this series is the delay between injury and the development of neurological signs and symptoms.

A unique feature of these patients is the universal complaint of radicular pain. Radicular pain is not normally seen as a consequence of thoracolumbar spine fracture, the common presenting neurological signs and symptoms of an acute spinal fracture being weakness ans sensory loss [11,21–24]. It is possible that the radicular pain experienced by these patients may be related to a local vascular or inflammatory process, or other phenomena associated with a local healing process within the spinal canal.

The patients described here were offered surgery only if severe motor deficits were objectively documented, and the patients themselves were judged to be medically sound. Brace immobilization of patients with mild deficits can result in excellent clinical results, although no definitive guidelines for the management of these patients can be drawn from this limited retrospective study.

This small retrospective series suggest that caution should be exercised in the management of any compression fracture in an osteoprorotic patient. Complaints of increasing back pain and/or lower extremity dysfunction may be on the basis of progression of such a fracture with resulting neural compromise. The author's experience suggests that this phenomenon may be more common than has previously been appreciated. Nonetheless, these injuries can lead to devastating neurological deficits and major morbidity. It is hoped that increased awareness of this phenomenon may lead to more timely diagnosis and improved outcome.

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