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Short Communication

Spinal Stabilisation in Plasma Cell Disorders

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Five patients with plasma cell disorders and vertebral body lesions who presented with severe localised back pain associated with spinal instability are described. They underwent a total of six surgical spinal stabilisation procedures with excellent symptomatic relief. Spinal stabilisation was performed at presentation in 1 patient with a cervical spine lesion, and at sites of previously irradiated disease or recurrent disease in the other 4 patients. Stabilisation was achieved using various internal fixation techniques, and in 2 cases external fixation was obtained with a halo brace. The procedures were performed with minimal operative morbidity, and resulted in good symptom control. Spinal instability should be considered as a cause of severe localised back pain exacerbated by movement in patients with plasma cell disorders and vertebral body lesions. Operative spinal stabilisation should be considered as part of their management.

Key words: plasmacytoma, myeloma, spinal instability
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INTRODUCTION

BONE DEMINERALISATION and localised lytic disease leading to vertebral body collapse are major causes of morbidity in multiple myeloma. Bone pain is the most common presenting symptom of vertebral body disease in both multiple myeloma and solitary plasmacytoma [1, 2]. Tumour infiltration into bone may cause bone pain in the absence of structural abnormalities of the vertebral column. Pain, which may be exacerbated by movement, may also be caused by loss of structural integrity, so called "instability pain". In addition, a proportion of patients with instability pain will have neurological problems such as radicular pain or paraparesis.

Radiotherapy with or without decompressive surgery is the primary treatment for localised destructive vertebral lesions presenting with neurological complications. Whether chemotherapy is required following radiotherapy depends upon the documentation of more widespread disease after staging. Severe pain due to spinal instability may occur at presentation as a primary event, later as a secondary event following radiotherapy or associated with tumour recurrence. Operative spinal stabilisation may then be required.

We report our experience of 5 patients with plasma cell disorders who presented to a single centre and underwent six decompressive/stabilisation surgical procedures as treatment for destructive vertebral body lesions associated with spinal instability. The role of this rarely reported procedure in the overall management of plasma cell disorders associated with unstable vertebral body lesions is discussed.

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CASE REPORTS

Case 1

A 73-year-old man presented in May 1989 with low back pain. Radiographs demonstrated collapse of L4 and subsequent biopsy was consistent with plasmacytoma. Further staging confirmed the lesion to be a solitary plasmacytoma (Table 1). He re-presented 28 months later with worsening low back pain. X-ray computerised tomographic (CT) imaging showed destruction of L4 with nerve root compression. The intractable pain was thought to be due to instability at the L4/L5 level. An L4 laminectomy with tumour decompression and spinal stabilisation was performed. A 6 × 17 mm Hartshill rectangle was attached at two levels above and below L4 by Hartshill wires passed through the transverse processes. A posterior intertransverse bone graft was positioned between the bodies of L3 and L5. No radiotherapy was given postoperatively, but the patient received six cycles of monthly oral melphalan and prednisolone. A lumbar brace was worn for 3 months postoperatively. The patient remains fully mobile, pain free with no neurological deficit and no evidence of disease progression 18 months following surgery.

Case 2

A 65-year-old man presented in November 1988 with back pain and leg weakness. Radiographs showed collapse of T2 and a myelogram confirmed spinal cord compression at this level. A T2 laminectomy was performed and biopsy was consistent with a plasmacytoma. Subsequent staging showed no evidence of multiple myeloma (Table 1). He was treated with local radiotherapy (Table 1). He re-presented 26 months later with collapse

Table 1. *Details of staging investigations and initial presenting features*

Patient no.	Age	Sex	Initial presenting factors PP	BJ	SS	BM (% plasma cells)	IF	Level of spinal lesion	Diagnosis	Initial treatment
1	73	M	IgG 9.2 g/l	-ve	-ve	< 2	n/a	L4	Solitary plasmacytoma	10 Gy L3–L5
2	65	M	-ve	-ve	-ve	< 2	n/a	T2	Solitary plasmacytoma	30 Gy C1–T4
3	65	F	IgGk 5 g/l	-ve	-ve	< 2	Polyclonal	C1, C2	Solitary plasmacytoma	25 Gy to base of skull and cervical spine
4	78	F	IgA 15 g/l	n/a	-ve	15	Monoclonal	T1	Myeloma	20 Gy D7–D11 melphalan and prednisolone
5	53	M	IgGk 2 g/l	-ve	-ve	< 5	Polyclonal	Skull base	Solitary plasmacytoma	40 Gy to skull base

PP, paraprotein; BJ, Bence–Jones protein urea; SS, skeletal survey; BM, bone marrow; IF, immunofluorescent studies performed on bone marrow cytospin preparations; n/a, not available.

of T10 and received 20 Gy radiotherapy in five fractions to T5–T12. Repeat staging again showed no evidence of multiple myeloma. Forty-nine months from diagnosis, magnetic resonance imaging (MRI) (Figure 1a) and then CT (Figure 1b) of the lumbar spine was performed because of recurrent pain. CT and MRI demonstrated destruction of the bodies of T11 and T12 with displacement but no compression of the spinal cord. Severe localised pain at T11/T12 was elicited on movement and spinal instability was considered to be contributing significantly to the aetiology of the pain. Surgical decompression of T11 and a stabilisation procedure was performed. A Hartshill rectangle (14 × 6 mm) was fixed to the spine at two levels above and below T11/T12 by Hartshill wires passed through the transverse processes and an interspinous bone graft performed. Postoperatively, 34 Gy radiotherapy in 19 fractions was given to a field from T9–L1 to maximise the dose to the vertebral body and spare the spinal cord, part of which had been included in the previous irradiation portal. Sixteen months postoperatively the patient is mobile, pain-free and has no neurological deficit.

Case 3

A 65-year-old woman presented in June 1993 with severe neck pain on movement of the cervical spine and an unsteady gait. A CT scan showed a destructive lesion at the base of the skull involving the petrous bone, clivus, C1 and C2. The upper cervical spine was thought to be unstable. A CT-guided biopsy of the mass showed a plasmacytoma. Subsequent staging showed no evidence of multiple myeloma (Table 1). The upper cervical spine was immobilised in a neck halo whilst 25 Gy radiotherapy in 10 fractions was given to the cervical spine. Following radiotherapy, the occipito-cervical spine was stabilised operatively. At operation, a large plasmacytoma was seen to have eroded the posterior rim of the foramen magnum and destroyed the arch of C1 and C2. A Ransford loop was secured to the occipital bone with heavy gauge Hartshill wire and to the laminae of C4, C5 and C6 with sublaminar soft wire. A neck brace was worn for 3 months postoperatively. A further 25 Gy of radiotherapy was given to the base of skull and cervical spine 1 month postoperatively. The patient remains alive and well 13 months from diagnosis. No chemotherapy has been given to date and there has been no evidence of disease progression. Simple non-opiate analgesia is required for pain control.

Case 4

A 78-year-old woman presented in March 1986 with paraplegia and collapse of T10. Further investigation was consistent with multiple myeloma, Salmon and Durie Stage IIA. Treatment with local radiotherapy resulted in full neurological recovery and was followed by 8 months of treatment with cyclical oral melphalan and prednisolone to serological plateau. The patient re-presented 5 and a half years later with a bilateral T1 radiculopathy and collapse of T1 vertebral body. Twenty G-rays radiotherapy in five fractions was given to the C5–T1 region. Three months later the patient developed severe back pain and CT showed destruction of the body of T1 with expansion and destruction of the left pedicle and lamina. A myelogram showed a complete block at the C7–T1 level. Severe localised pain was exacerbated by movement and thought to be due to spinal instability associated with tumour recurrence. A partial C7 vertebretectomy with decompression was performed through an anterior surgical approach. Spinal fusion by bone grafting was performed. Postoperatively, a collar brace was worn for 3 months. Six further monthly cycles of oral melphalan and prednisolone were given. The patient remained pain free for 18 months until there was a large soft tissue recurrence of the plasmacytoma at C7, T1 and T2. Further palliative radiotherapy was given. The patient currently requires opiates for pain control and is receiving further chemotherapy.

Case 5

In September 1985, following a fall and head injury, a 53-year-old man had a craniotomy and a skull base lesion was biopsied that histologically was consistent with a plasmacytoma. Subsequent staging confirmed this to be a solitary lesion (Table 1) and he received 40 Gy radiotherapy to the skull base. In May 1988 and again in July 1990 six cycles of monthly melphalan and prednisolone were given when the paraprotein titre rose. On both occasions, however, restaging failed to show evidence of disease elsewhere. Five and a half years after the initial presentation he developed mid-thoracic back pain and paraparesis. Radiographs showed collapse of T3 and 30 Gy radiotherapy was given in 10 fractions to T2–T4 with complete resolution of his neurological signs. Eight months later, however, he developed severe back pain and nerve root irritation on movement. Instability at the T3 level was thought to be a major

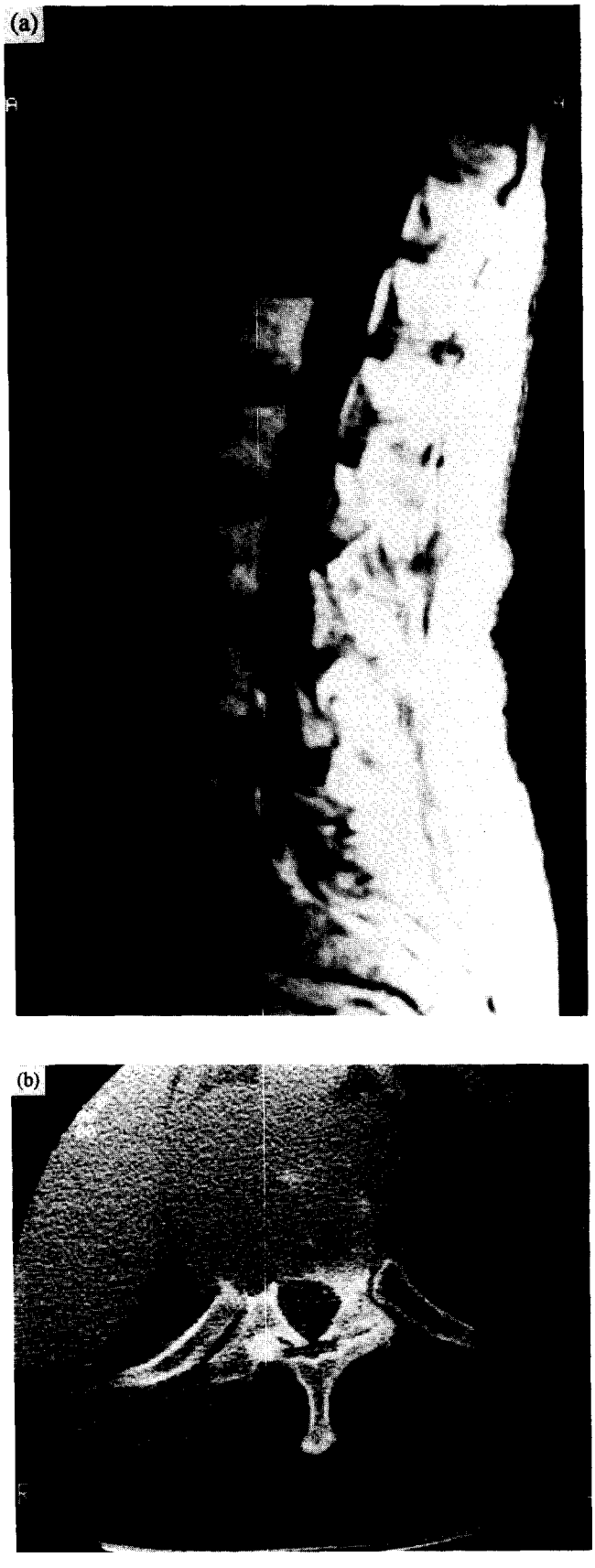

Figure 1. (a) Sagittal T1 weighted magnetic resonance image of lumbar spine (case 2) showing tumour in T11 and T12 vertebral bodies with associated collapse and displacement of spinal cord. (b) Axial X-ray computerised tomographic image of body of T12 showing extensive destruction of body and pedicles of T12 by tumour.

contributing factor to the pain and posterior spinal stabilisation performed. A 6 × 18 mm Hartshill rectangle was fixed at two levels above and below T3/T4 by Hartshill wires passed through the transverse processes, and bone grafting was performed. The patient was pain-free postoperatively and mobilised fully. No further chemotherapy was given at this point.

Eighteen months later the patient re-presented with severe neck pain which was exacerbated by movement. CT demonstrated a destructive lesion of C2 with forward subluxation of C1. The upper cervical spine was thought to be unstable and was immobilised in a halo brace whilst 27 Gy radiotherapy was given to the upper cervical spine. A posterior occipito-cervical stabilisation procedure was then performed. A Ransford loop was wired to the occiput with heavy gauge Hartshill wires bilaterally and secured to the laminae of C3, C4 and C5 with sublaminar soft wire. An interspinous bone graft was performed. The patient was mobilised in a hard collar and required simple analgesia only postoperatively. Restaging showed evidence of bone marrow involvement by myeloma for the first time and a rising IgGk paraprotein. Six further cycles of oral melphalan and prednisolone were given and the patient is currently mobile and pain-free with no neurological deficit 11 months postoperatively.

DISCUSSION

Bone pain is a presenting feature in approximately 70% of patients with multiple myleoma [1], and in the majority of patients with spinal plasmacytomas [2–6]. Neurological complications, either radicular pain or paraparesis, as a presenting feature of plasmacytoma of the spine are also frequent [3, 4]. Radiotherapy is the treatment of choice for localised bone pain due to myelomatous bone disease, and is often the primary therapy in patients presenting with localised back pain due to a solitary plasmacytoma of the spine. For patients presenting with severe and/or rapidly progressive neurological problems, such as cord compression due to vertebral collapse, a surgical decompression procedure is usually undertaken. In a proportion of patients spinal pain is largely or wholly due to spinal instability. In the context of this study, spinal instability was defined as severe back pain localised to a particular level which was exacerbated by movement and associated with a destructive vertebral lesion on imaging.

In the patients reported here, spinal stabilisation was performed for pain relief, to relieve or prevent the development of neurological complications and to maintain spinal integrity. In 4 cases, stabilisation was performed because of pain arising from instability alone or because of symptoms arising due to tumour recurrence and associated instability in previously irradiated sites. In case 3 the stabilisation procedure formed part of planned primary therapy with radiotherapy for treatment of a large destructive plasmacytoma. The second stabilisation procedure in case 5 was performed because of cervical spine instability associated with a new vertebral lesion.

In these cases the procedures were associated with minimal morbidity and excellent symptom control. Surgery was followed in all cases by chemotherapy, radiotherapy or a combination of both. The symptom control achieved is, therefore, attributable to a combination of spinal stabilisation with chemoradiotherapy. Follow-up postsurgery ranges from 29 to 13 months (median 18 months). As this study is a retrospective analysis, pain was not assessed formally. However, following spinal stabilisation 3 patients are pain-free requiring no analgesia, 1 patient requires non-opiate analgesia and 1 patient requires opiates to control pain due to local tumour recurrence at the site of surgery (case

4). 3 patients have shown no evidence of disease progression 13 to 18 months postsurgery and have excellent performance scores (ECOG score 0–1). One patient has relapsed locally (case 4) (ECOG score 3), and the other developed multiple myeloma (case 5) (ECOG score 2).

Spinal stabilisation in plasma cell disorders has not been widely reported. Kivioja and asssociates [7] reported 8 patients with plasma cell disorders who underwent, as part of primary therapy, tumour decompression combined with a variety of stabilisation procedures [7]. These included the insertion of Harrington rods in 5 patients, and the use of an external halo fixator, cementation and bone grafting in 3 others. Loftus and asssociates reported 6 patients with solitary plasmacytomas of the spine who presented with back pain and variable neurological symptoms who all underwent laminectomy and spinal fixation/fusion as part of planned primary surgical therapy followed by radiotherapy [8]. In these 6 cases the spinal stabilisation procedure was performed using Harrington rods and bone grafting. Loftus and asssociates advocated performing a spinal fusion procedure in combination with laminectomy in all patients presenting with spinal plasmacytomas and neurological symptoms, in order to prevent possible further collapse during subsequent radiotherapy or chemotherapy [8]. However, McLain and Weinstein reported no progressive neurological deficit in their series of patients with solitary spinal plasmacytoma treated with radiotherapy, and suggested that spinal stabilisation was only necessary in patients with signs of instability and pain with progressive deformity and neurological compromise [3].

In the 2 patients with upper cervical vertebral body lesions (cases 3 and 5), temporary spinal stabilisation was achieved with a halo vest whilst radiotherapy was given prior to definitive occipita-cervical fusion. The use of a halo vest as a temporary form of spinal stabilisation has been described in only 3 previous cases [7, 9], whilst occipita-cervical fusion, to our knowledge has only previously been described by Lofvenberg [10].

This series illustrates the importance of recognising back pain due to spinal instability in patients with plasma cell disorders and destructive vertebral body lesions. Operative spinal stabilisation when combined with chemo- or radiotherapy produced excellent symptom control irrespective of whether surgery was performed as part of planned primary therapy or for recurrent disease in previously irradiated sites. Spinal instability can be diagnosed clinically by typical spinal pain on movement and also predicted by imaging with CT and MRI. Operative spinal stabilisation should be considered as an integral part of the management of patients with plasma cell disorders, destructive vertebral body lesions and spinal instability.

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