CASE REPORT

Osteoradionecrosis of the lumbar spine
25 years after radiotherapy

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Osteoradionecrosis is a rare but recognised complication of radiotherapy. Cases have been described in the cervical spine following treatment for head and neck malignancies up to 25 years after administration of radiotherapy. We present a rare case of osteoradionecrosis affecting the L5 and S1 vertebral bodies in a 58-year-old woman who presented with low back pain 25 years after undergoing a hysterectomy with adjuvant radiotherapy for cancer of the cervix.

Case report

A 58-year-old woman presented with a two-month history of lower lumbar pain. There was no history of trauma or weight loss. She had undergone a hysterectomy with adjuvant radiotherapy for cervical cancer 25 years earlier. On examination she was tender over the lower lumbar spine, with increased pain on extension and axial rotation. A radiograph of her lumbar spine taken before referral showed a loss of height of the vertebral body of L5, which had been interpreted as an old crush fracture.

On MRI of the lumbar spine there was a heterogenous T2 marrow signal and a reduced T1 marrow signal in the collapsed L5 vertebra and the anterior portion of the body of S1 (Fig. 1). The loss of normal fat signal within these vertebrae suggested neoplasia and prompted CT investigation. Sagittal images showed moderate collapse of L5, with abnormal bone texture and patchy trabecular thickening and sclerosis (Fig. 2). There was irregularity in the superior endplate of L5 but no vertebral expansion. The appearances were considered non-specific, with a differential diagnosis of spondylodiscitis, trauma, Paget’s disease or neoplasia. Inflammatory markers, a myeloma screen and full blood count were all within normal limits. A biopsy of the L5 vertebra was performed under flat-panel CT imaging control using an 11G Jamshidi needle (Cook Ireland Ltd, Limerick, Ireland). Two solid bone cores were obtained. Histological examination showed no evidence of neoplasia (Fig. 3). It was concluded that the diagnosis was osteoradionecrosis secondary to the pelvic radiotherapy 25 years earlier. The patient was reassured and managed symptomatically.

Discussion

Soon after the discovery of X-rays their harmful effects began to be reported. Hall-Edwards was the first to describe these effects on his own hands, and in 1926 Ewing first used the term ‘radiation osteitis’. Osteoradionecrosis is now well recognised as a complication of therapeutic radiation, with potentially severe consequences.

The effects of radiation on the mature skeleton range from osteopenia to necrosis depending on the location, dose and fractionation of the beam. The effects of high-dose radiation on the surrounding soft tissues are also thought to be of key significance in the development of osteoradionecrosis. Radiotherapy produces a hypoxic, hypocellular and hypovascular environment in which the basic metabolic demands for cellular survival cannot be met. The bone becomes devitalised and this can potentially result in the surrounding soft tissues breaking down to form an open wound. Exposure of bone to radiation primarily damages osteoblasts, resulting in reduced production of bone matrix. Unopposed...
osteoelasis leads to progressive osteopenia and atrophy in the affected area,\(^6\) which becomes prone to insufficiency-type fractures after minor trauma, as seen in this case.

Osteoradionecrosis is most commonly encountered in the treatment of head and neck cancer, and affects the mandible and maxilla in up to 22% and 11% of patients, respectively.\(^7\) Osteoradionecrosis of the spine is rare, with only one case affecting the lumbar spine reported to date.\(^8\) There are three reports of osteonecrosis affecting the cervical spine,\(^1,5,10\) and all these patients had undergone previous radiotherapy for head and neck malignancies. Donovan et al.\(^1\) described three cases of osteoradionecrosis affecting the cervical spine presenting up to 25 years after radiotherapy. In one of these a brachytherapy implant was placed just anterior to the arch of C1 in combination with radiotherapy. Two of the cases required reconstructive surgery because of large destructive lesions causing progressive deformity and neurological impairment. All of the cases were treated with antibiotics and hyperbaric oxygen therapy. In reports by van Wyk, Sharma and Tranter\(^9\) and Mut et al.,\(^10\) both patients presented with evolving neurology and an epidural mass which appeared malignant on MRI. Biopsies after decompressive surgery showed no evidence of infection or neoplasia with hypocellular bone lacking osteoblasts. Sato et al.\(^8\) reported a case affecting the lumbar spine 18 months after intra-operative radiotherapy.
for pancreatic cancer. At follow-up the patient complained of back pain and was subsequently found to have a localised lytic lesion in the anterior border of L2. The patient underwent MRI and CT scanning to exclude a metastatic deposit. The results of the imaging were in keeping with a benign pathology and subsequent histological analysis demonstrated empty lacunae and fibrosis of marrow spaces with neovascularisation in keeping with osteoradionecrosis. Osteoradionecrosis affecting the acetabulum and head of femur has also been reported following radiotherapy to the pelvis. In the series presented by Deleeuw and Pottenger progressive radiological changes were not noted in the hip joint up to 14 years after radiotherapy, including bone that was not thought to be initially involved. Massin and Duparc noted the high rate of aseptic loosening in total hip replacements where patients had previously undergone pelvic irradiation.

The case presented here represents a milder form of osteoradionecrosis compared to previous reports affecting the cervical spine. There was no evidence of infection or gross structural deformity as described in other reported cases. This may reflect the higher dose and closer proximity of the cervical spine to structures in the neck treated with radiotherapy. The late presentation of this case is comparable with other late cases described in the literature. The imaging features in this case of partial vertebral collapse with endplate irregularity and sclerosis are non-specific and in keeping with a previous destructive process within the L5 and S1 endplates with secondary bone healing. The most common cause for this type of appearance would be infectious spondylodiscitis; however, the marked fatty (yellow) marrow replacement signal seen on the T1-weighted MRI sequences (Fig. 1b) would not be explained by disc/vertebral infection. The appearances are also atypical for vertebral Paget’s disease with the lack of vertebral body expansion. In the absence of histological features or clinical features to suggest old infection, these appearances are consistent with chronic osteoradionecrosis in this clinical context.

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References