Epidural compression of the cauda equina caused by vertebral osteoblastic metastasis of prostatic carcinoma: resolution by hormonal therapy

Keiichiro Susuki, Shunsuke Matsumoto, Norikazu Kitagawa, Hiroyasu Shinohara, Osamu Hasegawa, Yoshiyuki Kuroiwa

Abstract
A 59 year old man with prostatic carcinoma developed epidural compression of the cauda equina caused by bony expansion from a vertebral osteoblastic metastasis. For medical reasons he could not undergo radiation or surgery. Hormonal therapy alone relieved his low back pain and restored ambulation and urinary function. Postmyelography CT showed that the bony expansion from the vertebra had completely disappeared after treatment. This is the first report of remarkable improvement due to hormonal therapy alone.

Keywords: prostatic carcinoma; osteoblastic metastasis; epidural compression; hormonal therapy

Compression of the spinal cord and cauda equina is an important neurological complication of prostatic carcinoma. Direct tumour extension from a vertebral metastasis is the most common mechanism. Epidural compression caused by bony expansion from a vertebral osteoblastic metastasis, a rare occurrence, is thought to be an absolute indication for surgical decompression. We describe a case of epidural compression of the cauda equina due to such an uncommon condition, which was treated successfully with hormonal therapy alone.

Case report
A previously healthy 59 year old man developed low back pain in September 1997, and the pain gradually worsened. He began to notice paraesthesia in both legs in February 1998. One month later, he developed weakness and severe paraesthesia in both legs and could not walk.

In August 1998, he was admitted to our hospital, at which time anaemia was apparent. There was no neurological abnormality in the cranial nerves or upper limbs. Atrophy of the right lower limb was apparent. Muscle tone was decreased to grade 3–4 (Medical Research Council) power in the right lower limb and grade 4–5 in the left. Deep tendon reflexes were pathologically depressed in both lower limbs, and the Lasègue sign was positive on both sides. Hypaesthesia was present below L1 area on his right side and the S1 area on the left side, but the saddle area was normal. Although bowel dysfunction was not apparent, he developed urinary retention several days after admission.

Haematological investigations disclosed anaemia (Hb 7.3 g/dl) and thrombocytopenia (84 000/µl). Serological investigations showed an increased alkaline phosphatase of 5301 U/l, and lactic dehydrogenase of 751 U/l. Cerebrospinal fluid had increased protein, 63 mg/dl, but normal cellularity. Plain radiography showed multiple osteoblastic lesions involving the thoracic and lumbar vertebral bodies, and pelvis. A nuclear bone scan showed multiple hot spots in the skull, vertebrae, ribs, humeri, and femora, consistent with multiple bone metastases. Myelography showed narrowing of the vertebral canal in the body of Th11, L2. Postmyelography spinal CT showed an epidural mass in the body of Th11 and bony expansion from the body of L2 into the vertebral canal (figure).
Rectal palpation disclosed a hard, enlarged prostate. Serum prostate specific antigen was raised to 2300 ng/ml. Prostatic biopsy confirmed the diagnosis of a poorly differentiated adenocarcinoma. Pathological examination of the bone marrow by iliac aspiration and biopsy showed metastases from the prostatic carcinoma.

Unfortunately, as the patient’s general condition deteriorated due to incidental severe pneumonia, he could not undergo surgical decompression or radiation therapy. Corticosteroids also could not be used. Hormonal therapy, that combined an antiandrogen (oral chloromadinone acetate, 100 mg daily) with luteinising hormone-releasing hormone (LH-RH) analogue (leuprolrelin acetate, 3.75 mg subcutaneous injection once every 4 weeks) was started. This regimen was continued throughout the observation period. His low back pain and paraesthesia in both legs resolved within 6 weeks. Eight weeks later, his serum prostate specific antigen dropped to 15 ng/ml. Follow up myelography and postmyelography spinal CT 11 weeks after the start of hormonal therapy showed that both the epidural mass in the body of Th11 and the bony expansion from the body of L2 had disappeared (figure). By that time, muscle power in his right lower limb had improved to grade 4–5, and urinary retention had disappeared. Fifteen weeks later, he was discharged from the hospital and could walk with the aid of a cane.

Discussion

In men, prostatic carcinoma is the second most frequent cause of spinal cord compression after lung carcinoma. It occurs in about 7% of men with prostatic carcinoma and usually is the result of metastasis to the vertebral column. The tumour erodes through the bony structures and presses on the dura. As the tumour enlarges in the vertebral canal, both the cord and its blood supply are compressed. There may also be compression secondary to the collapse and displacement of a bony element. Epidural compression by bony expansion from a vertebral osteoblastic metastasis is a rare occurrence, only five cases having been reported as accompanying prostatic carcinoma.5 6 The mechanism by which new bone is formed is unclear. Reactive new bone formation may occur periosteally as a response to endosteal cortical bone destruction, or the tumour breaking through the cortex may induce new bone formation. Spinal cord compression secondary to metastasis from prostatic carcinoma is incurable, the goal in treatment being palliation. Preservation or restoration of ambulation and bladder function and pain relief are the measures of successful treatment. The mainstays of treatment are corticosteroids, hormonal therapy, radiation therapy, and surgical decompression. Hormonal therapy is an important treatment for spinal cord compression, if the patient has not yet received it. In the Massachusetts General Hospital series, two patients were treated successfully with orchietomy alone. Total androgen blockade, combining antiandrogens with LH-RH analogue has been used for advanced prostatic carcinoma.

During the initial 1 to 2 week period of administration of LH-RH analogue alone, a transient increase in serum testosterone concentration can lead to exacerbation of clinical symptoms, including spinal cord compression.8 The addition of an antiandrogen neutralises the influence of the transient increase in serum androgens and thus makes fully acceptable the use of LH-RH analogues as an advantageous substitute for surgical castration and estrogens in the treatment of prostatic carcinoma.9

Epidural compression caused by bony expansion of the vertebra secondary to osteoblastic metastasis is thought to be an absolute indication for surgical decompression.10 Four patients in the past received surgical decompression, combined with radiation therapy and/or hormonal therapy,10 two of whom became ambulatory;7 the others died. One patient who developed spinal cord compression during hormonal therapy received radiation therapy without improvement. Because of incidental severe pneumonia, neither surgical decompression nor radiation could be used for our patient. He was treated successfully solely with hormonal therapy. To our knowledge, this is the first report of remarkable clinical and radiological improvement based on hormonal therapy alone. The mechanism remains unclear. Total androgen blockade causes significant hypogonadism and results in high bone turnover with bone loss.10 This may account for resolution of the new bone formation in our patient.

Compression of spinal cord or cauda equina is an oncological emergency. Early diagnosis is of the utmost importance. Aggressive treatment to reverse compression usually is justified for prostatic carcinoma with often a slow natural history. Our case suggests that hormonal therapy may be of value in treatment of malignant spinal cord or cauda equina compression caused by bone disease of prostatic carcinoma in patients not suitable for radiation or surgery.

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