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Multiple myeloma of the thoracic spine developed at the previous trauma site: case report

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Abstract The precise nature of the underlying plasma cell dyscrasias is not well understood. Even today, no causative factor has been found for this disease. It has been suggested that, several factors may act together in the development of this disease. This is a report of a 62-year-old man who initially had a T7 compression fracture and developed a multiple myeloma two years later at that site and at the adjacent vertebral level (T7–T8), respectively. Although the notion that trauma would initiate such a malignancy is speculative, the preceding trauma may be a causative factor in plasma cell dyscrasias.

Keywords Multiple Myeloma · Trauma · Spine

Introduction

Plasma cell dyscrasias are the major malignancies of plasma cells [2, 6, 7]. These disorders are classified into various specific disease including monoclonal gammopathy of undetermined significance (MGUS), solitary plasmocytoma, and multiple myeloma (MM) [6].

In approximately 5–10% of patients with plasma cell cytomias, the disease may be manifested as a solitary plasmocytoma of the bone, and frequently involves the vertebral column [4, 10]. The incidence of vertebral compression fractures is 60% in patients with multiple myeloma and spinal cord compression accompanies 10–15% of them [14]. MM is characterised by proliferation of a single clone of plasma cells derived from B-cells. It

represents approximately 1% of all cancers and 2% of all cancer deaths [4, 6].

Although advances in cellular biology and molecular genetics in the past decade have provided clinicians with powerful tools to better understand the underlying causes of MM, no definite cause for this disease has been identified to date. However, it has been found that there is some possible relations between plasma cell dyscrasias and a decline in the immune system, genetic factors, certain occupations, exposure to certain chemicals, and radiation [2, 5–7, 13, 15, 16]. Most recently, Kaposi's sarcoma-associated herpes virus have been identified in patients with multiple myeloma [3].

Multiple myeloma generally presents an insidious onset and due to complex clinical and radiological

findings, the diagnosis is often delayed. Besides, magnetic resonance imaging (MRI) of the bone marrow in MM patients does not always enable the recognition of vertebrae that will develop compression fracture [8].

Although medical literature contains no definite evidence, preceding trauma may be a causative factor in the development of a plasma cell dyscrasia. We present a 62-year-old man with a T7 traumatic compression fracture who developed a multiple myeloma at the same level about two years later.

Case report

A 62-year-old man, who had been diagnosed as having nonsurgical T7 compression fracture after falling out onto concrete floor two years ago, presented with a one-month history of fatigue, and neurologic examination revealed minimal paraparesis. His medical records indicated that he had been clinically stable, and followed up conservatively at a local hospital. Previous MRI, performed two years earlier reviewed and interpreted as T7 compression fracture (Fig. 1a) with no bone marrow involvement. New MRI showed a large mass, which involves T7 and T8 vertebral bodies, displacing spinal cord posteriorly with extraosseous soft tissues extension (Fig. 1b). Blood examination revealed anaemia, and erythrocyte sedimentation rate was markedly elevated.

Laboratory investigations showed increased plasma beta2-microglobulin at 0.2 mg/dl (normal range 0.00–0.16 mg/dl). Serum immunoelectrophoresis revealed the monoclonal component of immunoglobulin G (IgG) protein and lambda-light chain. Bone marrow biopsy revealed 30% plasma cells infiltration. Abnormal proteinuria was not detected. We subtotally removed the tumour and destroyed vertebral bodies from T7 to T8 following reconstruction of the thoracic spine with a corpectomy cage (Ulrich GmbH & Co. KG, Ulm, Germany) and autologous rib graft via an anterolateral approach (Fig. 1c). Postoperative course was uneventful and the patient's symptoms gradually improved. Systemic chemotherapy was started and radiotherapy was planned later because of the possibility of fusion failure. Histological examination confirmed the tumour was a plasma cell cytoma (Fig. 2).

Discussion

The relationship between the trauma and development of plasma cell dyscrasias at the injury site has not been established yet. Hussein et al. [6] reported eight cases who had plasma cell neoplasms originating from previously traumatised sites. Mongkonsritragoon et al. [11] reported a case of primary plasmocytoma, which developed at the electrical injury site. Suna et al. [15]

Fig. 1 Sagittal T2W MR image (a) taken 2 years earlier demonstrating T7 compression fracture. No bone marrow involvement is noted. New sagittal T2W MR image (b) revealing osteolysis of T7 and T8 vertebral bodies, a mass formation displacing the spinal cord posteriorly, and extraosseous soft tissues extension. Early postoperative coronal reconstructed CT (c) showing the fusion material and also rib graft

Fig. 2 Photomicrograph showing atypical plasma cells with abundant cytoplasm, and the nuclei are situated eccentrically (H&E, original magnification $\times 400$)

described an intracerebral plasmocytoma in a patient who had experienced severe head injury 20 years ago.

The mechanisms by which plasma cell cytomias develop at the previous trauma sites are of interest. Trauma is postulated to cause plasma cell cytoma via various mechanisms [6, 9, 11–13, 15]. Rosenblum et al. [13] reported a patient with multiple myeloma developing a subcutaneous plasmocytoma due to a local trauma localised at the previous sites of intravenous and haemodialysis catheters. It has been generally postulated that the trauma can cause the release of the cytokines

leading to the proliferation of plasma and stromal cells in the bone [6, 13, 16]. On the other hand, continued inflammation due to local trauma may be responsible for the development of plasma cell dyscrasias by overstimulation of plasma cells [6, 9, 12].

If this patient would have been assessed thoroughly at the first admission and/or follow-up examination, myeloma might have been recognised. De facto, if a patient is suspected to have multiple myeloma; all blood and urine tests should be performed. Although diagnosis of MM requires the detection of a monoclonal gammopathy in the serum or urine, in some clinical forms of MM such as nonsecretory multiple myeloma (NSMM), no gammopathy can be demonstrated [1]. In addition, if the bone survey shows diffuse or multifocal osteolytic lesions, bone marrow biopsy can be performed. However, there is no absolute indication to perform a needle biopsy for every vertebral compression fractures in elderly patients.

It is important to remember that in most cases, individuals who develop multiple myeloma have no clear risk factors. Although the relationship between trauma and plasma cell cytoma is not clear, preceding trauma may be a causative factor in the development of this disease.

In conclusion, especially elderly patients presenting with traumatic vertebral fracture should be evaluated periodically and systematically, and considering the uncertain radiological and clinical criteria in the early stages, serious measures should be taken for the differential diagnosis and suitable treatment.

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