CASE REPORT

Plasma cell myeloma infiltrating the dental pulp: An interesting finding

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ABSTRACT

Plasma cell myeloma (PCM) is a clonal neoplastic proliferation of terminally differentiated B lymphocytes (plasma cells/myeloma cells) that involves the skeletal system in a multifocal fashion. Even though jaw involvement has been reported in as many as 30% of cases, myeloma cells infiltrating into the pulpal tissue is extremely rare. Here, we present a case of PCM in which myeloma cells are seen infiltrating into the pulpal tissue of 46.

Key words: Dental pulp, multiple myeloma, plasma cell myeloma

INTRODUCTION

Plasma cell myeloma (PCM) is a neoplastic proliferation of plasma cells within the bone marrow, characterized by plasma cell infiltrate of the bone marrow, osteolytic bone lesions and the presence of monoclonal protein in the serum or urine. Extraosseous involvement of the disease is less common.[1] Also referred to as multiple myeloma (MM), PCM is the second most common hematologic malignancy, having an indolent course and poor prognosis.[2] PCM belongs to a broad group of disorders called plasma cell dyscrasias each having a distinct clinical presentation. In case of PCM the atypical plasma cells are manifested in the bone marrow throughout the course of disease. However as the disease progresses, the malignant plasma cells can be seen in the peripheral blood and other organs like spleen, liver, etc.[2] This is considered as an indicator of a more aggressive form of disease called plasma cell leukemia.[2]

Infiltration of the atypical plasma cells of PCM into the pulpal tissue is a rare phenomenon. To the best of our knowledge, there is no documented case of PCM involving pulpal tissue in the English literature. Hence, we report the first case of PCM where atypical plasma cells were seen in dental pulp. Since peripheral blood involvement and soft tissue infiltrations occur in the late stages of the disease, we also suggest that pulpal infiltration in PCM may be considered as a prognostic indicator of the terminal phase of the disease.

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CASE REPORT

A 55-year-old male patient reported to our department with complaints of pain and numbness on the right side of mandible of 3 months duration. His medical history revealed that he was under treatment for PCM, which was diagnosed a year back (M-band positive in protein immunoelectrophoresis). Radiographs showed multiple punched out radiolucencies on skull and spinal X-rays [Figure 1a and b].

On examination there was a diffuse swelling on the right side of mandibular body seen in relation to 45, 46 and 47 regions. Mucosa over the swelling was normal. It was bony hard on palpation with slight buccal and lingual cortical expansion. The right molar (46) was caries exposed with grade III mobility [Figure 1c]. Radiograph showed altered trabecular pattern in the periapical region of 46 [Figure 1d]. The teeth was extracted and tissue from the adjoining socket area was reviewed for microscopic examination.

Histopathologically, the tissue showed proliferating sheets of atypical plasma cells. Most of the cells had an abundant basophilic cytoplasm and an eccentrically placed nucleus. In between these cells were cells with centrally placed nuclei and basophilic cytoplasm, the plasmablasts. Prominent nucleoli were also seen [Figure 2a and b].

The pulpal cavity of the extracted 46 after decalcification and hematoxylin and eosin (H and E) staining showed infiltration with atypical plasma cells with eccentrically situated nuclei and mild degree of nuclear pleomorphism [Figure 2c-e]. Immunohistochemical staining (IHC) for kappa and lambda light chains were done. The plasma cells showed strong cytoplasmic positivity for kappa-stain and were negative for lambda light chain which is consistent with clonal plasma cell dyscrasia [Figure 3a and b].

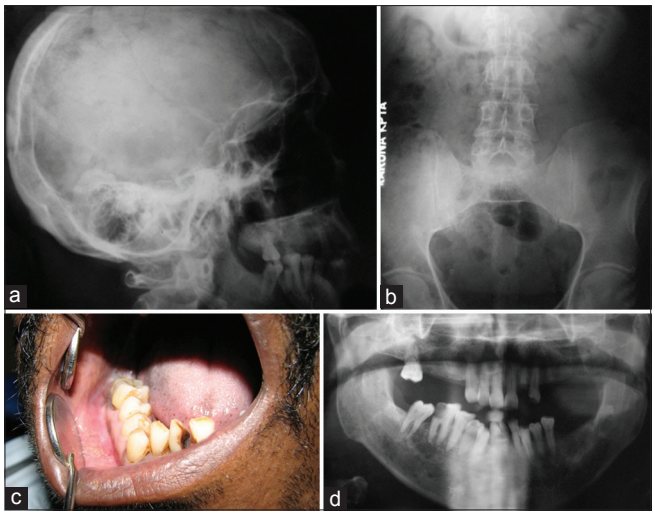

Figure 1: (a and b) Radiographs showing multiple punched out radiolucencies on skull and spinal X-rays respectively. (c) Intraoral view of the lesion. (d) Orthopantomogram (OPG) shows periapical radiolucency in relation to 46 with loss of trabeculation

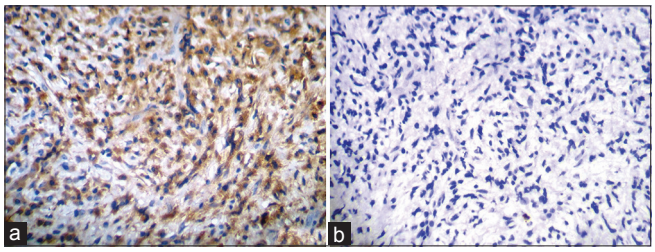

Figure 3: (a) Immunohistochemical staining for immunoglobulin kappa showed strong positive cytoplasmic kappa-staining plasma cells (IHC stain, ×400). (b) IHC for immunoglobulin lambda showed negativity for lambda light chain (IHC stain, ×400)

DISCUSSION

PCM represents 1% of all malignancies and between 8 and 20% of all bone malignancies.[3] It is the second most common hematological malignancy and most common plasma cell dyscrasia.[2]

The precise etiology of PCM is unknown.[4] PCM is a disease of older adults; median age of onset is 70 years.[5] PCM is somewhat more prevalent in men than in women. In the oral cavity, mandible is far more frequently involved than maxilla. Exposure to radioactivity is also seen to be a risk factor for the disease. If people have a sibling or parent with PCM, their chance of developing it is nearly four times that of the general population. People working in petroleum industry may have an increased risk of this cancer. Obesity is also recognized as a risk factor for PCM.[5]

Pathophysiology:[6] Exogenous stimuli induces cytogenetic changes in B-lymphocytes that transform into malignant plasma cells. These malignant plasma cells secrete monoclonal immunoglobulin M-proteins or para-proteins. Unregulated proliferation of monoclonal plasma cells into the bone

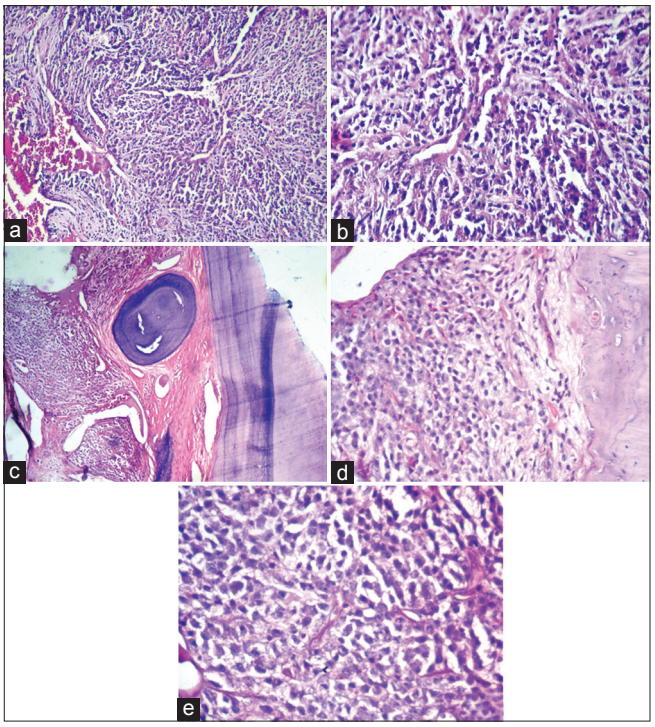

Figure 2: (a) Photomicrograph showing proliferating sheets of atypical plasma cells. (H&E stain, ×100). (b) Higher magnification of the figure 2a. (H&E stain, ×400). (c) Photomicrograph showing pulpal tissue which is infiltrated by atypical plasma cells.(H&E stain, ×100). (d and e) Higher magnification of the figure 2c. (H&E stain, ×400)

marrow leads to adhesion of these cells to stromal cells and secretion of cytokines.These cytokines stimulate the growth of PCM cells and inhibit apoptosis. The excessive secretion of these cytokines stimulates osteoclastic activity and bone resorption, resulting in osteopenia, multiple small lytic bone lesions. Excessive numbers of MM cells can collect in the spine, pelvis, skull, ribs, sternum and proximal appendages; forming masses or tumors known as plasmacytomas.[6]

Clinical manifestations results from the infiltration of plasma cells into bone marrow and secretion of Bence-Jones protein into blood and urine.[6] PCM is clinically manifested by low blood counts, painful lytic bone lesions and fractures and susceptibility to renal insufficiency and infections.[7] Lytic lesions of the entire skeleton are a major manifestation of PCM and result from the infiltration of bone marrow by the myeloma cells. These lytic lesions are commonly observed in the pelvis, spine, ribs, sternum and skull bones.[8]

Oral manifestations in PCM are generally restricted to the jaw bones and manifest as lytic bone lesions similar to ones seen elsewhere in the skeleton. Oral manifestations may eventually arise in 30%[9] of cases and include facial, oral, or dental pain; numbness and paresthesia; swelling, soft tissue epulides, mobility of teeth, hemorrhage, pathologic fractures, amyloid deposition and radiolucencies.[3,10] The present case is unique as the pulpal infiltration of myeloma cells is rarely seen and so, this is probably the first reported case of this nature.

In the present case, the radiographic picture of a well-defined periapical radiolucency involving the distal root of caries exposed right mandibular molar suggested two possibilities, either a periapical lesion of the caries tooth or an infiltration of myeloma cells. Histopathology confirmed the presence of malignant plasma cells in the periapical region. The myeloma cells were also seen to be infiltrating into the pulpal tissue.

There could be two possible routes through which the myeloma cells would have reached the dental pulp. The first possibility could be a direct spread of the cells from the adjacent periapical bony lesion.[2] If this was the case, then such an infiltration would have resulted in some degree of root resorption as well as more widespread bone destruction. In addition, the radiographic presentation would have been that of an ill-defined lesion with ragged borders. However, in this case the exact outline of the root could be traced.The lesion was more or less well-defined and confined to the periapical region. Therefore in our case it can be said with certainty that the source of myeloma cells in the pulpal tissue was not from the periapical lesion.

For a major course of the disease the atypical plasma cells in PCM are confined to the skeletal system. Appearance of these cells in the peripheral vascular system indicates transformation to an aggressive lesion and possibly represents terminal stages of the disease which is then termed as plasma cell leukemia.[2] Once the myeloma cells enter the peripheral circulation, they can infiltrate various organs and soft tissues. Therefore, infiltration of organs like spleen, liver and other soft tissues is relatively common in the later stages of the disease.[2] Kim *et al*., reported a 40% increase in blood flow in moderately inflamed pulp.[11] This increased blood flow accompanied by the movement of fluid from the capillaries into the interstitial tissue results in hyperemic changes in the pulp which allow the myeloma cells to move into pulp; a second possibility.[12] In our case the tooth was caries exposed and therefore the pulpal tissue would show signs of inflammation. The increased blood flow resulting from this inflammation, as suggested by Kim *et al*., could have resulted in greater movement of myeloma cells through the pulp. At some point these malignant plasma cells could have moved out from the pulpal circulation into the pulp tissue proper and began to proliferate there.

It can safely be assumed that the myeloma cells in the dental pulp had reached there from the peripheral circulation. This indicates that the disease in this case had progressed to the terminal stages (plasma cell leukemia). This knowledge could help in reviewing the prognosis of the patient as well as suggesting appropriate modification to his treatment.

The present case shows that pulpal infiltration by malignant plasma cells can take place in PCM. It highlights the need for dental practitioners to be aware of such a possibility. It also suggests the possibility of dental pulp involvement being considered as a clinical prognostic indicator in PCM. Since there is paucity of literature regarding the infiltration of atypical plasma cells into the pulpal tissue, investigations on larger samples is necessary for gaining insight to any kind of hypothesis.

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