A case of plasma cell myeloma involving the middle phalanx of the ring finger is reported. This was a case of a non-secretory myeloma; however, monoclonal immunoglobulins were demonstrated by immunohistochemical studies. Plasma cell myeloma with phalangeal involvement is extremely rare; our literature search disclosed only two well-documented cases. Plasma cell myeloma may occur in the hand, so it should be considered in the pre-biopsy differential diagnosis when bone lesions radiologically consistent with myeloma are encountered.

CASE REPORT

A 58-year-old male presented to the hospital Accident & Emergency department with an injury to his left ring finger while playing golf. Initial radiographs showed an undisplaced fracture through a cystic area in the middle phalanx of the ring finger. He was treated with strapping for three weeks, with a diagnosis of fracture through an enchondroma of the middle phalanx. On a subsequent visit to our clinic, the finger was found to be swollen and radiographs showed an expanding lytic lesion with erosion of the bone of the middle phalanx. (fig 1, 2). The patient initially underwent biopsy of the lesion with inconclusive histology. Skeletal radiological survey was normal. Three-phase bone scan showed a lytic lesion, with some increased activity around its periphery in the region of the middle phalanx. Routine haematological tests were normal. Twenty-four hour urine testing revealed the presence of Bence-Jones proteins and urine electrophoresis showed monoclonal free lambda chains. Urine para-protein was 0.8g/l, and urine total protein was 1.150 g/l. Serum electrophoresis showed IgG at 5.41 g/l (range 6-11), IgA at 22 g/l (range 0.8-4) and IgM at 0.25 g/l (range 0.5-2). The patient subsequently underwent amputation of the affected finger and the specimen was sent for histopathological analysis. The histology was reported as ‘Plasmacytoma’ with clear margins and immunocytoLOGY revealed that cells were positive for Lambda light chains. Microscopically, the tissue obtained in a case of solitary myeloma typically includes a mixture of well and poorly differentiated plasma cells. Bone marrow aspirate showed plasmacytosis in the marrow with 30% Lambda positive cells. The patient was referred to the haematologist who commenced him on a course of Vincristine, Adriamycin and Dexamethasone (VAD). At three-year follow-up review, the patient was asymptomatic with no local or widespread recurrence.
DISCUSSION

Multiple myeloma is the malignant proliferation of plasma cells involving more than 10% of the bone marrow. The multiple myeloma cells produce monoclonal immunoglobulins that may be identified on serum or urine electrophoresis (3). The diagnostic classification and staging of plasma cell dyscrasias proposed by Durie and Salmon (2) is still most widely used worldwide. An alternative modified classification system called British Columbia Cancer Association (BCCA) was proposed (6). According to this classification for the diagnosis of multiple myeloma two of the following should be present: presence of a paraprotein in urine or serum, lytic bone lesion and bone marrow plasma cell infiltration in excess of 10%. For solitary plasmacytoma the diagnostic criterion is a single area of bone destruction due to clonal plasma cells, bone marrow plasma infiltration less than 10% and absence of any other bony lytic area on skeletal survey. Accordingly our case was a case of multiple myeloma with phalangeal involvement and 30% plasmocytosis with lambda light chain. Lytic lesions in multiple myeloma are usually found in the vertebral column, ribs, skull, pelvis and axial bones (1). Bone lesions sparing the axial skeleton and found in bones of the hand have been reported to be an unusual finding in the course of multiple myeloma (4, 5). Our case was put on chemotherapy as a first line of treatment as it has been shown to give good cost effective remission (7, 8). To date, three years after diagnosis, our patient is asymptomatic with no local or widespread recurrence.
REFFERENCES


