Case report

SOLITARY PLASMACYTOMA: Report of a Rare Occurrence in the Mandible

[1]Okwuosa CU, [2] Nwoga M C,[3] Otakhoigbogie U[4], Nwokike I C, [3] Okechi CU,[3] Nwankwo KC[4]

[1]Department of Oral Pathology and Oral Medicine, University of Nigeria Teaching Hospital [2] Department of Oral Pathology and Oral Medicine, Faculty of Dentistry, University of Nigeria [3]Department of Oral and Maxillofacial Surgery, Faculty of Dentistry, University of Nigeria [4]Department of Radiation oncology, University of Nigeria Teaching Hospital

ABSTRACT

BACKGROUND: Very little has been reported regarding plasmacytomas in the oral and maxillofacial region and studies cited were mostly in foreign literature apart from two cases from Nigeria

Objective: The dearth of publications in African literature informed the need to report a rare case of solitary plasmacytoma of the mandible in a 52-year-old male patient

CASE REPORT: A 52-year-old male patient that presented at our institution with an eight-month history of painless swelling of the left mandible associated with difficulty in chewing. Histology diagnosed Non-Hodgkin lymphoma but immunohistochemical studies revealed tumour cells with strong expression of CD138 and confirmed a diagnosis of solitary plasmacytoma (SPB). The patient was treated with palliative radiotherapy 30Gy in 10 fractions over two weeks.

CONCLUSION: SPB of jaw most commonly presents clinically as a painless swelling, and radiographically as multilocular radiolucency with ill-defined borders. Diagnosis of the SPB depends on the microscopic evidence of plasma cell proliferation and absence of any other bone involvement. Ancillary techniques such as immunohistochemistry play an important role in distinguishing SPB from other haematological diseases

Keywords: extramedullary plasmacytoma, multiple myeloma, solitary plasmacytoma

Correspondence address:

Dr Nwoga, M. C.
Department of Oral Pathology and Oral Medicine,
Faculty of Dentistry, University of Nigeria.
+2348037234573
mark.nwoga@unn.edu.ng

INTRODUCTION

Plasmacytomas are lymphoproliferative neoplasms which are characterized by monoclonal neoplastic proliferation of plasma cells.[1,2] They are histopathologically indistinguishable from multiple myeloma and may signify a disease continuum.[3,4] It was first cited by Unna in 1891 and defined by Schridde in 1905. The Revised European-American International Lymphoma Study Group classified these lesions under B-cell peripheral lymphomas.[5] Plasma cell dyscrasias account for about 2.6–3.3/100,000 population.[1,6]

The three clinical entities of plasma cell dyscrasias include multiple myeloma (MM), solitary plasmacytoma of the bone (SPB) and extramedullary plasmacytoma (EMP). MM is the commonest neoplasm among the 3 entities.[5,7]

SPB and EMP are rare localized forms with a variable tendency to progress to systemic multifocal multiple myeloma after the initial diagnosis.[11,12,13] Both lesions are characterized by absence of systemic involvement attributed to myeloma.[14]

The development of SPB is restricted to a lone medullary bone which accounts for 3%–7% of all plasma cell dyscrasias.[10,11] SPB primarily affects the axial skeleton, especially vertebrae and secondarily long bones. However, jaw involvement is very rare, and when it does, it shows a predilection for mandible than maxilla. Most of the cases reported in the literature are in the bone marrow-rich areas of the posterior mandible and also retromolar trigone.[12,13] SPB is a distinct entity that may herald the first manifestation of ensuing MM.[14]

Tissue biopsy for histopathology remains the gold standard since urine and serum paraproteins are very low or undetectable in most cases of SPB.[15,16] It is vital to evaluate the patient for disseminated disease through an extensive workup consisting of thorough clinical examination, skeletal survey, complete blood picture, renal function tests, serum calcium profile, bone marrow biopsy and urine/serum electrophoresis.[17,18]

Very little has been reported regarding plasmacytomas in the oral and maxillofacial region and studies cited were mostly in the foreign literature apart from two cases from Nigeria.[16,19] The dearth of publications from the African literature informed the need to report a rare case of solitary plasmacytoma of the mandible in a 52-year-old male patient that presented at our institution, suggesting the utmost importance of early diagnosis by the clinician.

CASE REPORT:

A 52-year-old male with no relevant medical history presented to the Oral Surgery Clinic of the Teaching Hospital on account of an eight-month history of painless swelling of the left mandible associated with difficulty in chewing (Figure 1). There was no fever, no drenching night sweat and no weight loss. The jaw swelling was non-tender but mildly warm on palpation, and no significant lymphadenopathy was noted. Lesion measured 8 x 6 cm, and was firm, ulcerated, bled spontaneously and discharged bloody exudates. There was associated bone destruction with root resorption of the teeth and mobility of 36, 37 and 38.

Histologic diagnosis was non-Hodgkin's lymphoma (Diffuse Large B-cell Lymphoma), (Figure 2). To rule out myeloma as a differential, investigations showed no evidence of hypercalcemia and Bence-Jones proteins. Skeletal survey showed no other bony lesions. Bone marrow biopsy and examination showed no obvious morphological alteration, and was reported as within normal histologic limits. Confirmatory Immunohistochemical studies revealed tumour cells formed by mildly atypical and fairly matured plasma cells which showed lambda light chain restriction. The tumour cells exhibited strong uniform expression of CD138 (Figure 3) but were completely negative with CD20, CD79a, CD56 and cyclin D1. The Ki-67 index was less than 5% overall. The histologic features were reported as those of plasmacytoma.

The patient was treated with palliative radiotherapy 30Gy in 10 fractions over two weeks. The tumour shrank and the swelling disappeared after the radiotherapy. He was placed on chymoral and Dexamethaxone for 10 days by the oncologists. He developed post-radiation oral complications including oral mucositis, tooth mobility, and reduced salivation with an unstimulated salivary flow rate (whole saliva) of 0.1ml/min. Clinical examination revealed Miller's grade II mobility of the lower left first molar (tooth 36). A diagnosis of post-radiation hypo-salivation (xerostomia), and chronic periodontitis of tooth 36 was made. He was started on cevimeline 30mg, three times daily for one month, and counseled to maintain good oral hygiene, lemon juice was recommended to stimulate residual salivary gland activity.

Nine months post radiation therapy, the patient developed severe continuous hiccups, loss of appetite. He was managed at peripheral hospitals and was referred to the Teaching hospital five months later when he was already cachexic. An abdominopelvic ultrasound scan showed multiple intra-abdominal gut masses, small bowel masses, suprapubic mass, and epigastric mass. Chest Radiograph was free of any significant finding.

Laboratory haematological investigations indicated normal ranges of Hb, PCV, platelet count, and WBC (Total). The Differential white cell count showed Neutrophil (58.7%), Lymphocytes (33.8%), Eosinophils 0.0%, Monocytes 7.5%, Basophils 0.0%, ESR 105/hour (Westergreen: 0 - 10mm/1st hour). There was elevated monocyte count and ESR. Electrolytes, urea and creatinine were within normal ranges, liver function test showed values within normal ranges for bilirubin (conjugated), Alanine aminotransferase, Aspartate aminotransferase, and alkaline phosphatase. However total bilirubin was high 1.9mg/dl (normal range 0.1-1.5mg/dl).

Endoscopic biopsy of one of the masses was not diagnostic and no further attempt was made for a repeat biopsy because of the poor health condition of the patient. The patient was managed palliatively thereafter. He expired 17 months after the initial diagnosis and treatment.

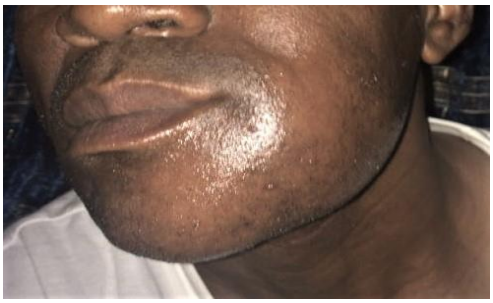

Figure 1: Clinical presentation of the mandibular swelling diagnosed by histology as Solitary plasmacytoma of the mandible.

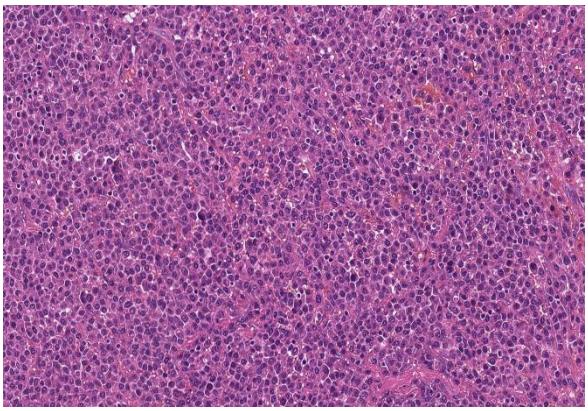

Figure 2a: Low power view of solitary plasmacytoma showing sheets of moderately and poorly differentiated plasma cells (H&E X100).

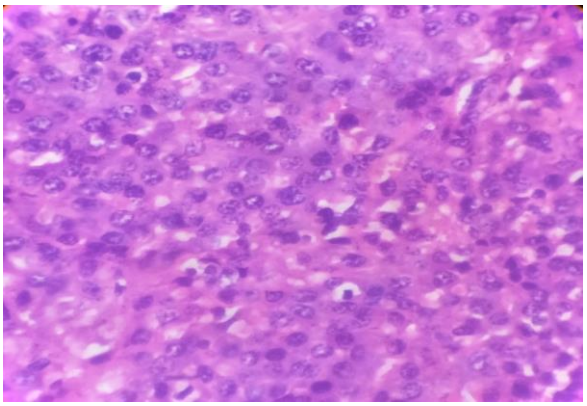

Figure 2b: High power view of solitary plasmacytoma showing sheets of moderately and poorly differentiated plasma cells, coarsely clumped chromatin, prominent nucleoli, and basophilic cytoplasm. H&E X400.

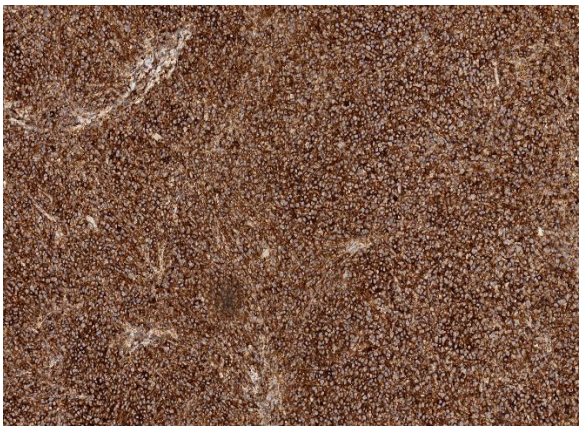

Figure 3: Photomicrograph showing strong positive CD138 reaction. x100

DISCUSSION

Solitary plasmacytoma of bone (SPB) is a lymphoproliferative monoclonal plasma cell disease which rarely affects the orofacial region. This may account for the dearth of literature on their occurrence especially in Africa and Nigeria in particular.[16,19,20] The aetiology of SPB is unknown, however, it has been postulated that chronic irritation, radiation, viral infections and genetic interaction in the reticuloendothelial system may contribute to the development of the lesion.[21]

SPB occurs most commonly among patients in the 5th and 7th decades of life, with the mean age at diagnosis being 55 years.[22,24] It is more prevalent in males than females with a ratio of 2:1.[6] In accord with the studies in the literature above, the present case was reported at the age of 57 years in a male patient.

Though SPB is uncommon in the orofacial region, only 4.4% of the cases occur in bone marrow-rich areas of posterior mandible.[20,23] The present case was reported in the posterior mandible consistent with a study conducted by Pisano *et al*. in which, among 13 cases of SPB, nine lesions were reported in the posterior mandible, as well as other studies.[8,24]

The presenting features of SPB involving the mandible are often underestimated because they are often non-specific. Consequently, this makes clinical suspicion very difficult. However, it may present with pain in jaws, paraesthesia, tooth mobility, spontaneous haemorrhage, swelling in bone with or without soft tissue involvement and pathological fractures.[10,25] Painless left posterior mandibular swelling, spontaneous bleeding and tooth mobility were the principal features observed in our case. This was in agreement with various studies that reported painless swelling as the most common clinical presentation of SPB.[10,15]

Typically, SPB presents variable radiographic features ranging from unilocular to multilocular radiolucent lesions due to bone resorption induced by the cytokines and osteoclast activating factors released by the malignant plasma cells.[13,26] Lae et al.[26] reported three radiographic patterns in SPB, which include multilocular soap-bubble lesions, unilocular radiolucency with cystic appearance and ill-defined destructive bone resorption. In our case, an ill-defined multilocular radiolucency with extensive cortical destruction of the posterior aspect of the mandible was observed. This was characteristic of a malignant lesion of the jaw bone. Based on this, a working clinical diagnosis of osteosarcoma was made since SBP is not a commonly encountered jaw tumour.

Trephine biopsy has also been used in characterizing this disease. A diagnosis of SPB as opposed to systemic myeloma is made in the absence of < 10% clonal plasma cells in marrow aspirate.[19] Microscopically, SPB presents as clusters and sheets of atypical plasma cells with variable degree of differentiation and sparse stroma. Plasma cells are characterized by abundant cytoplasm with eccentric nucleus which often show chromatin clumps typically arranged in a clock-face pattern. Occasionally binucleated cells and inclusion bodies are seen.[8,10,19] The present case was also in harmony with these features. The neoplastic cells may show paranuclear, pale staining area called "hof." Giant cell formation, amyloid deposition and myxoid change are noted in few cases.[8,10]

Most cases of SPB secrete monoclonal light chain immunoglobulins which are identified in serum or urine.[26] In discordant with the literature, the present case showed no elevated serum lambda light chains. Definitive diagnosis of SPB should rule out MM, through extensive clinical investigations which include skeletal radiological survey, bone marrow aspiration, blood cell count, determination of calcium levels and study of renal function.[26] Updated diagnostic criteria for SPB were given by Guidelines Working Group of the UK Myeloma Forum (IMWG), as single area of bone destruction due to clonal plasma cells, absence of M-protein in serum and/or urine, bone marrow not consistent with MM (nucleated plasma cells <10%), normal skeletal survey (and MRI of spine and pelvis if done) and no related

organ or tissue impairment.[27] The present case satisfied all the mentioned criteria. In addition to laboratory investigations, monoclonal restriction to either lambda or kappa light chains is an essential approach to evaluate the suspected case of plasmacytoma.

Urine and serum paraproteins which is very low or absent in SPB was negative in our case. This was similar to most studies in the literature that observed that SPBs neither showed positive Bence Jones proteinuria nor presence of paraproteins in the urine and blood respectively.[16,28]

Apart from histology, ancillary investigations such as immunohistochemical and molecular techniques often aid in the confirmative diagnosis of SPB. Monoclonal proliferation of plasma cells can be confirmed by the presence of kappa/lambda light chain restriction or by polymerase chain reaction-based approach. The presence of paraprotein is assessed by electrophoresis from blood or urine specimen.[29] The application of this tool to determine the diagnosis of SPB is still vague since the presence of paraprotein does not always determine the existence of this lesion. However, it should be emphasized that its diagnostic value is relevant in cases where it is desired to evaluate the presence of M-protein, response to therapy and prognosis.[21,30] Studies have suggested that the persistence of paraprotein after treatment may be indicative of progression to MM and it is the only independent adverse prognostic factor for myeloma free survival.[31,32]

Histopathologically, it is necessary to differentiate SPB from plasmablastic lymphoma (PBL), reactive inflammatory lesions (plasma cell gingivitis), non-Hodgkin's lymphoma and melanoma which presents a diagnostic challenge to clinicians.[1,8] Features such as neoplastic cells exhibiting abundant cytoplasm, vesicular chromatin and central nuclei with prominent nucleoli are commonly observed in PBL. Neoplastic cells of PBL are positive for CD38, MUM1, CD138 and VS38c, while CD20 is not expressed in PBL. In our case, immunohistochemical analysis showed CD138 and Ki-67 positivity, while the tumour was negative for CD20, CD79a, CD56 and cyclin D1 which ruled out lymphoma, leukemia and neuroendocrine metastasis. CD138 was suggestive of terminally differentiated plasma cells. In SPB, Ki-67 labelling index is usually reported as 10%.[24] In our case, it was 5%, which suggested low tumour aggressiveness.

Presence of non-plasmocytic neoplastic component, IgM expression and positivity for pan B-cell surface markers such as CD20 and CD79a favours diagnosis of lymphomas. Immunohistochemical studies are used to rule out malignant melanoma which shows positive expression for S100, Melan-A and HMB-45 markers, whereas plasmacytoma shows negative expression of these markers.[8]

Some lesions of the oral and maxillofacial region including lymphoproliferative disorders and undifferentiated carcinoma may also exhibit similar microscopic features to those seen in plasmacytoma. Thus, the diagnosis of solitary plasmacytoma of bone (SPB) requires a solitary bone lesion, with both confirmatory histopathological and IHC evaluation. IHC markers such as CD138 and CD56 are vital to show abnormal hematopoietic activity.[33] CD138 is a marker for plasma cells, plasmablasts, and some immunoblasts. Its main reactivity in hematolymphoid neoplasms includes plasma cell neoplasms and some large B-cell lymphomas. CD138 in conjunction with κ and λ light chains helps in differentiating reactive from clonal PCs.[34]

Solitary cases of plasmacytoma may be misdiagnosed as benign lesions including plasmacytosis or gingival plasmacytosis which can be clinically ruled out based on the tumour size, bone involvement, evolution, and most importantly, histopathology. Plasmacytosis is plasma cell granuloma which is polyclonal and not neoplastic. In contrast, plasmacytoma is monoclonal and has a single kappa or lambda light chain restriction.

The treatment modalities available for SPB are radiotherapy, surgery and chemotherapy. There are no randomized studies about the best

treatment approach to SPB due to its rarity. However, radiotherapy is considered the standard treatment as these tumours are highly radiosensitive.[35] There is no clear relationship documented between response and radiotherapy dose for SPB. Tumour bulk is the most significant factor influencing prognosis of SPB. It is recommended that a margin of at least 2 cm and a dose of 35-40 Gy in 20 fractions for lesions ≤5cm and those >5 cm, a higher dose of up to 50-67 Gy in 25 fractions should be considered though despite this, local failure have been reported.[17,18] The present case was managed with palliative radiotherapy 30 Gy in 10 fractions over a period of 2 weeks.

The evolution of SPB is relatively benign and the prognosis is better than MM. Progression to MM, local recurrence and development of new bony lesion other than MM are the three patterns which worsen the prognosis of SPB. Dimopoulos MA et al.[36] have reported that SPB has a significantly higher risk of progression to MM at a rate of 65%–84% in 10 years and 65%–100% in 15 years. The patient in this case was reviewed up to 6 months and then briefly lost to follow-up. This case was not demonstrated to progress to MM. The multiple abdominal masses later diagnosed one year post radiation treatment could not be histologically confirmed due to the poor cachectic condition of the patient at presentation. MM was still not diagnosed at this stage. However, the multiple abdominal masses would have contributed to the poor prognostic outcome in our case.

Different imaging techniques should be compared for SPB diagnosis and follow-up. Techniques should be developed to identify prognostic subgroups in SPB. Precise guidelines to establish diagnosis as well as treatment modalities in solitary plasmacytoma of jaw bone are required. RT as valid treatment modality with quantification of their doses to prevent progression of SPB to MM requires an evaluation. Large randomized clinical trials could be carried out to evaluate the use of novel agents and to define the optimal treatment approach for patients presenting with poor prognostic factors.

CONCLUSION

Plasma cell neoplasms of jaw bones in the absence of myelomatosis are rare. Distinguishing one from the other has significant implications for treatment and survival. SPB of jaw manifests as a single osteolytic lesion and has better prognosis compared to MM. As found through our review, SPB of jaw most commonly presents clinically as a painless swelling, and radiographically as multilocular radiolucency with ill-defined borders. Diagnosis of the SPB depends on the microscopic evidence of plasma cell proliferation and absence of any other bone involvement. Ancillary techniques such as immunohistochemistry play an important role in distinguishing SPB from other haematological diseases. The management must be rapid following diagnosis with effective monitoring, because of its higher propensity to transform into multiple myeloma.

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