Plasmacytoma and Amyloidoma Presenting as an Iliac Mass

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CASE PRESENTATION

A 78-year-old woman with ongoing treatment for a longstanding history of IgG kappa multiple myeloma (MM) presented with pelvic pain and left-sided hip pain. The patient was initially diagnosed with monoclonal gammopathy of unknown significance (MGUS) (initially diagnosed in 1999), which evolved into MM in 2016. She had been on combination treatments for multiple myeloma with various agents including lenalidomide (later stopped due to peripheral neuropathy), steroids and cyclophosphamide. About two years prior to presentation, she was started on daratumumab and carflizomib due to progressive disease. Although paraprotein levels had been slowly increasing over the past six months from 0.8 to 1.4 g/dL, she did not have any evidence of anemia, hypercalcemia, renal dysfunction, proteinuria or cardiac dysfunction. Due to left hip pain, she underwent magnetic resonance imaging (MRI) of the left hip which revealed a large mass centered in the medial aspect of the left iliac bone with large soft tissue components (Figure 1). She underwent biopsy of the mass which showed sheets of kappa light chain restricted plasma cells interspersed with amyloid deposit (Figures 2a, 2b, 2c, 2d). Fluorescence in situ hybridization (FISH) for plasma cell neoplasm was negative. Cytogenetics was unrevealing. In addition to the left iliac

Figure 1. T2 fat saturated MRI left hip image showing a large (6 by 10 by 16 cm) heterogeneous mass (black arrow) with high signal intensity centered in the medial aspect of left iliac bone with soft tissue components.



Figure 2a. 40X magnification showing sheets of plasma cells (in purple) interspersed with pink, amorphous amyloid.

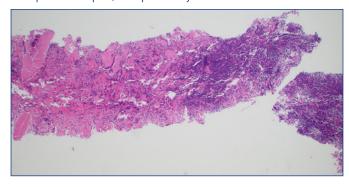


Figure 2b. 200X magnification showing Congo red staining of amyloid deposits (black arrow).

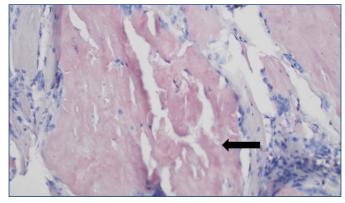


Figure 2c. 200X magnification showing apple green birefringence (red arrow) of amyloid deposits under polarized light.

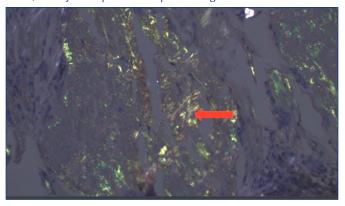


Figure 2d. 400X magnification showing CD138 staining of plasma cells.



lesion, positron emission tomography (PET) scan showed hypermetabolic areas involving the left scapula, right clavicle and C6 vertebra. She underwent palliative radiation to the left pelvic mass. She was subsequently started on bispecific T-cell engager (BiTE) therapy with Teclistamab and zoledronic acid.

Our case is unique for several reasons. First, it illustrates the full spectrum of plasma cell dyscrasia, progressing from MGUS to MM and then to plasmacytoma with localized amyloidosis, all within the treatment course. Second, unlike the more common sequence of plasmacytoma evolving into MM, here plasmacytoma emerged as a progression of MM. Finally, plasmacytoma was associated with localized amyloidosis, a rare finding with only a few reports in the literature.1

Reference

1. Sharma N, Sharma S, Bindra R. Plasmacytoma with amyloidosis masquerding as tuberculosis on cytology. J Cytol. 2009;26(4):161-163. doi:10.4103/0970-9371.62190

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