## **International Journal of Science and Research (IJSR)**

ISSN: 2319-7064

ResearchGate Impact Factor (2018): 0.28 | SJIF (2019): 7.583

## A Case of MGUS with Skeletal Erosion - An Abnormal Presentation

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Abstract: Presented here is a case of 52 years old women who came to emergency with complains of back pain, headache, weight loss and difficulty in walking for 6 months. She had H/O Pulmonary TB for which she took ATT, completed 1 year back. She had no trauma/recent medical or surgical interventions significant. On CNS examination she features of compressive myelopathy. Her CBC showed, Normocytic-Normochromic anemia with lymphocytic leukocytosis TLC-6500, ESR-raised, CRP-raised and normal PBS. Serum calcium raised and RFT was midly deranged, with normal LFT, R/E and M/E urine. An initial x ray of DL spine and MRI Spine revealed multiple punched out erosions in vertebrae, ilium and femur with few pathological fractures. There was collapse of L2 vertebra without and paradiscal changes or IVDP. An X ray skull showed multiple round punched out bony erosions. Serum free light chain assay revealed kappa chain markedly raised and kappa to lambda ratio highly raised with normal beta chain. Beta 2 microglobulin was > 20000 with a normal serum protein electrophoresis. a diagnosis of plasma cell dyscrasia was made. Her immunoglobulin assay was normal, 24 hours Urine protein electrophoresis had band on beta chain and IFE confirmed it kappa paraprotein with normal UPCR and ACR and raised total urinary protein. Bone marrow study was normal. Based on above findings she was diagnosed for monoclonal gammopathy of unknown significance with abnormal bony erosion. Patient was sent to higher center and unfortunately was lost to follow-up.

## 1. Case Report

Presented here is a case of 52 years old women who came to emergency with complains of back pain, headache, 5-6 kg of weight loss and difficulty in walking for 6 months. She had no previous history of any trauma of surgery. She was symptom free about 1.5 years back when she first noticed intractable productive cough with occasional blood tinge in sputum. Was given FDC CAT I DOTS regimen ATT for 9 months which she completed 3 months back. On examination she had stable vitals with mild pallor. Her spino-motor examination revealed raised bilateral lower limb DTR and Babinski positive. No evident spinal deformity or tenderness was found.

Her CBC revealed, hb-8.4gm% (Normocytic-Normochromic) TLC-6500 ( $N_{40\%}L_{52\%}$ ) ESR-150(raised) CRP-raisedand PBS with no abnormal cell. She had normal electrolytes and serum calcium-10.8mg/dl(n-6.8-9.9mg/dl). Urea-120mg/dl(n=15-45mg/dl), creatinine-2.1mg/dl(n=0.9-1.3mg/dl)) with normal LFT and R/E and M/E urine. All virological markers were non reactive.

A provisional diagnosis of compressive myelopathy was made, following background of tuberculosis and having ATT mostly POT'S Spine was made and patient was planned for relevant imaging studies.a serological study of RF factor and Anti CCP was made and was negative.

An initial x ray of dorso-lumbar spine was done along with MRI Spine. It unexpectedly revealed multiple punched out erosions in vertebrae, ilium and femur was seen with few pathological bony fractures. There was collapse of L2 vertebra without and paradiscal changes or IVDP. An X ray skull was also done that showed multiple round punched out bony erosions. The diagnosis got a whole different turn and the above findings was suggesting plasma cell disorder.

Her blood sample sent for free light chain assay and was found her kappa chain >35000(N) kappa to lambda ratio-18.9.Beta 2 microglubulin was > 20000 but her serum protein electrophoresis revealed a normal pattern without any band. Her immunoglobulin assay was done and was found to be within normal levels.24 hours Urine protein electrophoresis had band on beta chain and IFE confirmed it kappa paraprotein with normal UPCR and ACR and raised total urinary protein. A bone marrow study was done that revealed a normal cellular picture with normal plasma cells. Based on above findings she was diagnosed for monoclonal gammopathy of unknown significance with abnormal bony erosion. The patient was sent to higher centre for destined oncological setup. But unfortunately the patient was lost to follow up.

The case above has opened a possible manifestation of MGUS presentation with bony erosions. Due to lack of destined oncological and genetic labs at our center the cellular typing could not be done and patient was sent to other center for further management.

Volume 9 Issue 5, May 2020 www.ijsr.net

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Paper ID: SR20426032408 DOI: 10.21275/SR20426032408 229

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The x ray skull suggestive of multiple punched out bony erosions, a picture more in favor for multiple myeloma but lab investigations and bone marrow is suggesting MGUS. An abnormal but presentation of it. The link or entity needs to be established for the same.

Volume 9 Issue 5, May 2020 www.ijsr.net

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Paper ID: SR20426032408 DOI: 10.21275/SR20426032408