Extensive Amyloid Deposit in Bone Marrow Aspirate

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A 62-year-old female presented in the Hepatology OPD with progressive weakness, loss of appetite and dyspnoea. On examination she had icterus, anasarca and hepatomegaly. The liver was enlarged 8 cm below the right costal margin and hard on palpation with vague nodularity. There was no splenomegaly or lymphadenopathy. She was a known case of hypothyroidism. X-ray chest showed bilateral pleural and pericardial effusion. Echocardiography was suggestive of restrictive cardiomyopathy. MRI showed hyperintensity of liver on T2. Liver function tests were deranged. Kidney function tests were normal. Complete hemogram showed Hb of 11 gm/dl, total leukocyte count $7.8 \times 10^9/L$ and platelets were $178 \times 10^9/L$.

Liver biopsy performed for the workup revealed extensive amyloid deposit in the vessels and the sinusoids near completely replacing hepatic parenchymal cells. Endoscopic biopsies from duodenum and rectum showed amyloid deposition in blood vessels. Serum and urine electrophoresis and immunofixation did not show monoclonal M band. Serum Ig-free light chain showed κ to λ ratio of 4.12 (normal 0.26–1.65) and β_2 microglobulin was raised. Bone marrow aspirate and biopsy was performed to rule out multiple myeloma. Aspirate showed hypocellular marrow particles with extensive deposits of

blue-pink acellular amorphous (Fig. 1A, B) extracellular congophilic material (Fig. 1C) displaying green birefringence under polarised light. There was mild erythroid hyperplasia with adequate representation of myeloid and erythroid series of cells with normal maturation and no dysplastic changes. The plasma cells were approximately 6 % out of 500 cell count. A few isolated small aggregates of 4–6 plasma cells were also seen. Bone marrow biopsy also revealed homogenous pale eosinophilic acellular deposits of amyloid in the interstitium and vessel walls (Fig. 1D). Plasma cell group or nodules were not seen. Immunohistochemistry for kappa and lambda was performed on the bone marrow biopsy which showed plasma cells showing positivity predominantly for kappa and focally for lambda. A final clinico-pathological diagnosis of plasma cell dyscrasia with AL (kappa) systemic amyloidosis was made.

Primary or AL amyloidosis is the commonest cause of systemic amyloidosis worldwide [1,2]. Bone marrow examination in these cases is performed in order to evaluate plasma cell infiltration. Plasma cells may be increased to the level of overt multiple myeloma or the number could be <10 % in which case it is classified as plasma cell dyscrasia or monoclonal gammopathy of unknown significance (MGUS). Amyloid is usually not seen or is very sparse if present in aspirate, however biopsy usually shows amyloid deposition on vessel wall and rarely in interstitium [3]. In a study by Swan et al. the bone marrow core biopsy demonstrate amyloid deposition in 60 % cases out of 100 biopsies of patients with AL (primary) amyloidosis studied [2]. In their series plasma cell dyscrasia was observed in 83 cases with majority of lambda (65 cases) light chain disease. They also observed that interstitial amyloid deposition was significantly more commonly associated with patients with a monoclonal kappa light chain gammopathy,

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Fig. 1 Bone marrow aspirate showing extensive deposits of *blue-pink* acellular amorphous extracellular material (**A**, **B**) congophilic with congo red stain (**C**). Bone marrow biopsy also revealed homogenous pale eosinophilic acellular deposits of amyloid in the interstium and vessel walls (**D**). (Color figure online)

like in our case. Few studies have shown mean plasma cell percentages ranging from 7 to 14 % by analyzing the bone marrow core biopsy specimen in AL amyloidosis using immunohistochemical stains for light chain immunoglobulins [2, 4]. However, the finding of extensive bone marrow amyloid deposition in the aspirate smears is very unusual and rarely reported to the best of our knowledge.

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