Relapsed Myeloma with Hepatic Involvement and Tumour Lysis with Bortezomib

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A 82 year old gentleman was diagnosed with IgG multiple myeloma and had treatment with attenuated cyclophosphamide, thalidomide and dexamethasone (CTDa) chemotherapy. Patient achieved a very good partial response. Fourteen months from the initial diagnosis, he presented with anaemia, renal impairment (creatinine 142 $\mu\text{mol/L}$) and hypercalcemia (2.65 mmol/L). The monoclonal M band and SFLC ratio rose from 2.0 to 22.0 g/L and 11 to 2016 respectively. Trephine biopsy confirmed 95% bone marrow infiltration by CD 138 positive cells (Fig. 1). β_2 microglobulin was 22 mg/L (range 1.2–2.4 mg/L). In addition, patient had deranged liver function tests; bilirubin 39 $\mu\text{mol/L}$, alkaline phosphatase 207 U/L and alanine transaminase 111 U/L. Clinical examination revealed non-tender hepatomegaly 5 cm below right sub-costal margin. A subsequent CT scan demonstrated hepatosplenomegaly (Fig. 2). The liver biopsy showed extensive infiltration by diffuse sheets of discohesive, highly atypical cells with plasmacytoid and blastic morphology (Fig. 3). Immunohistochemistry demonstrated strong reactivity for CD 138 (Fig. 4) and MUM1 with kappa light chain restriction.

He was commenced on dexamethasone and weekly bortezomib chemotherapy with allopurinol. Four days following the second dose of bortezomib chemotherapy, patient developed acute kidney injury with a creatinine 500 $\mu\text{mol/L}$. Blood results met the Cairo–Bishop criteria for tumor lysis syndrome because of the renal impairment, high urate 1158 $\mu\text{mol/L}$ (range 200–430 $\mu\text{mol/L}$) and high phosphate of 2.51 mmol/L (range 0.78–1.65 mmol/L). Biochemical abnormalities improved with haemofiltration. However patient died of rapidly progressive liver failure. Bilirubin was 204 $\mu\text{mol/L}$ prior to death.

In series of autopsied cases, hepatic infiltration by plasma cells has been observed in up to 40% of patients [1]. However, multiple myeloma causing clinically significant liver dysfunction is very uncommon. Macroscopically these can present as either diffusely infiltrative or focally nodular types. These can mimic primary liver condition like cirrhosis or metastatic malignancy on imaging techniques. Indolent haematological conditions like myeloma rarely cause tumor lysis. It has been reported in cases treated with bortezomib. Physicians should be aware of the

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Fig. 1 Bone marrow trephine biopsy

Fig. 2 CT abdomen

risk of tumor lysis with proteasome inhibitor particularly in cases with heavy bone marrow infiltration, extensive liver involvement and high beta-2 microglobulin.

Fig. 3 Liver biopsy

Fig. 4 Plasma cells in liver biopsy by CD138

Reference

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