agent of choice in patients suspected having recurrence of MTC (Clarke *et al* 1988)[4]. The method of uptake of ^{99m}Tc (V) DMSA into tumor is not understood, however, Ohta *et al* (1985)[6] have postulated that pentavalent DMSA resembles the phosphate ion, and suggest that this is the mechanism by which ^{99m}Tc (V) DMSA accumulates in tumours, particularly in MTC where calcification is a well recognise phenomena.

This case illustrates an uptake of ^{99m}Tc (III) DMSA, a known agent for kidney imaging, by a primary MTC. Unlike pentavalent form of DMSA, which needs special preparation, trivalent form is easily prepared and available in all nuclear medicine department. It is not a recognised agent for tumour imaging. To the best of my knowledge, no such case has been reported. Perhaps, this agent can be used to diagnose primary and recurrent MTC.

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A Patient with Two Haematological Malignancies

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Summary

This is a patient with a double diagnosis of Acute Myeloid Leukaemia and Multiple Myeloma. In our patient the plasma cells were clonal and could clearly be distinguished from the myeloblasts.Treatment using the anti-myeloma regimen resulted in rapid clinical deterioration and progression of the acute myeloblastic leukaemia.

Key Words: Acute myeloid leukaemia, Plasma cells, Monoclonal gammopathy, Chemotherapy

Introduction

An excess of plasma cells in the bone marrow of patients with AML is a recognised phenomenon. The plasma cells in the marrow are in a minority of the cells, are not clonal and there is no paraprotein, the

significance of their presence is often uncertain[1]. Earlier reports[2,3] had described the association of myeloma with acute myeloid leukaemia and some Chronic Myelomonocytic Leukaemia and this clinical conditions remains a rare phenomenon.

We believe the coexistence of two populations of malignant cells represent two different disease entities, each possibly with a different chemosensitivity profile. We are uncertain if this case represents a coincidence, where the two diseases occurred simultaneously or arose sequentially.

Case Report

Our patient is a 67-year-old Malay lady, who first presented to another hospital with the symptoms of left ventricular failure, and a two months history of anorexia and weight loss. Clinical diagnosis of ischaemic heart disease aggravated by moderately severe anaemia was made. The initial blood count showed a Hb of 8.5 gms/L, total white count $4.9x10^9$/L and a platelet count of 100 x 10^9/L. The peripheral blood film revealed circulating myeloblasts. Our patient was transfused with red cell concentrates, and treatment with diuretic commenced, and she was then transferred to our hospital.

A bone marrow biopsy was performed, and the May-Grunwald Giemsa stains showed increased cellularity, very little normal haemopoietic tissues are present, the predominant cells being myeloblasts and plasma cells (see Fig.1 and 2). The number of the plasma cells varied between 30 to 40% and the myeloblasts 60-70% of the nucleated cells in different areas; cytochemical stain for myeloperoxidase showed positive reaction only in the myeloblasts.

Immunophenotyping studies done on the marrow by the immunoperoxidase method showed that the myeloblasts were positive for CD13 and CD33 but negative for CD10, and the plasma cells were negative for all these markers. Further staining for the immunoglobulin light chains using similar methods revealed the ratio of the plasma cells staining positive for the lambda light chain to the kappa light chain is greater than 16:1, in favour of the lambda light chain. This indicated that the plasma cells were possibly of a monoclonal origin.

Other tests performed to investigate for the presence of monoclonal protein and the other related abnormalities. In the 24 hours urine collected, the protein measured 1.34 gms/L, and urine electrophoresis and immunofixation identified the protein as Lambda light chain. Immunofixation on the serum detected a monoclonal protein as well which was identified as IgG lambda; and the amount of the M-protein was estimated to be 21.6 gms/L. The biochemical profile of the patient showed that the patient had mild renal

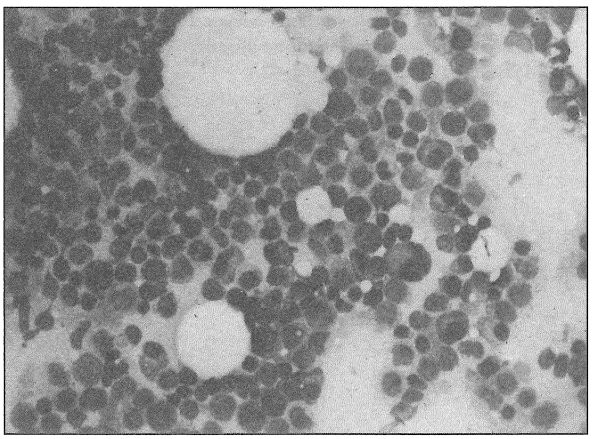

Fig. 1: This is a x10 view of the bone marrow showing the increased number of plasma cells and myeloblasts, normal haemopoietic tissue is severely depleted

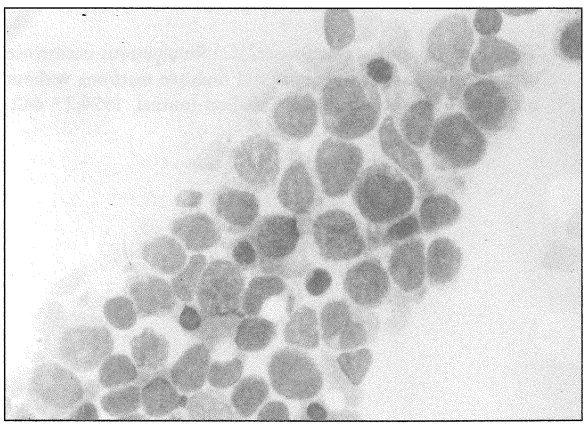

Fig. 2: This is a x40 view showing in more detail the morphology of the myeloblasts

impairment, with a normal calcium level. Skeletal X-ray showed only osteoporosis with no lytic lesions.

The above information led us to conclude that there was sufficient evidence to make the diagnosis of Acute Myeloid Leukaemia and Multiple Myeloma.

We then commenced our patient on chemotherapy using the VAD regimen, which consisted of vincristine, adriamycin both being given as twenty four hours infusion for four days and dexamethaxone given as once daily bolus also for four days. The total protein subsequently declined, but the total white cell count consisted mostly of myeloblasts began to increase in the peripheral blood. This differential response in the laboratory tests were associated with deterioration in the patient's platelet counts, and the emergence of infective complications. The patient remained weak and frail, and requested to be returned home for convalescence and terminal care.

Discussion

Our patient has manifested two haematological malignancies which in the ontogeny of the haemopoiesis suggest that the malignant change would have occurred at a very early pluripotent stage of the stem cells. One way by which this may be proven would be to analyse the G6PD isoenzymes present in the two populations of cells. The presence of different isoenzymes would discount the suggestion. Cytogenetic studies to look for certain non-random chromosomal changes seen simultaneously in the myeloblasts and the plasma cells would have been very useful as well. It is not easy to obtain metaphases in the myeloma plasma cells due to their low proliferative rate. There are no specific cytogenetic changes described in Myeloma. Having said that, a shared, common, abnormal cytogenetic profile between the plasma cells and the myeloblasts would be an important clue to suggest a common origin. It was obvious that this patient's illness can only be treated palliatively, nevertheless her rapid and progressive deterioration after the chemotherapy was unexpected. We elected to treat the myeloma first, we were interested to observe the differential response her disease manifest to the treatment administered, we believe this is further evidence to support the notion that the two diseases exist as two separate entities.

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