

Bone Marrow and Peripheral Blood Changes in Non-Hodgkin's Lymphoma

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ABSTRACT

Background: 47 patients with non-Hodgkin's lymphoma (NHL) were studied retrospectively to determine their marrow and blood changes at diagnosis.

Methods: The blood counts, blood films, marrow smears, trephine and tissue biopsies of patients at diagnosis were reviewed. The scheme proposed by the International Lymphoma Study Group (REAL) was utilised for lymphoma subclassification.

Results and Conclusion: 21.3% had lymphoblastic lymphoma, 21.3% had peripheral T-cell lymphoma (unspecified), 29.8% had diffuse large B-cell NHL, 10.6% had Burkitt's lymphoma and 17% had others. The incidences of anaemia, one or more abnormal counts, lymphocytopenia, increased marrow reticulin and marrow eosinophilia at diagnosis were 66%, 85.1%, 41.3%, 40.9% and 44.7% respectively. Marrow involvement was present in 46.8% of the patients, with diffuse infiltration noted in 71.4% of these cases. Abnormal counts and anaemia were common in all the NHL subtypes. In lymphoblastic lymphoma, the common haematological abnormalities were peripheral atypical lymphocytes and diffuse marrow involvement. In peripheral T-cell lymphoma (unspecified), common features were peripheral lymphocytopenia, increased marrow reticulin and eosinophilia. In diffuse large B-cell NHL, peripheral lymphocytopenia, peripheral myeloid precursors and/or nucleated red cells and marrow involvement were common. In Burkitt's lymphoma, diffuse marrow involvement and eosinophilia were common. No significant differences were noted between most of the haematological parameters of B and T-NHLs. In comparison with other reports, we recorded higher overall incidences of anaemia and diffuse marrow involvement, and a lower incidence of marrow infiltration in peripheral T-cell lymphoma (unspecified).

Keywords: Non-Hodgkin's lymphoma, marrow, peripheral blood

Singapore Med J 2000 Vol 41(6):279-285

INTRODUCTION

Changes in the full blood counts, peripheral blood (PB) differential white cell counts, as well as bone marrow involvement (MI) frequently occur in non-Hodgkin's lymphoma (NHL). Examination of these tissues at the time of diagnosis often provides useful information on the staging of the disease and its effects on the haemopoietic system⁽¹⁻³⁾. Literature search yields limited information on these haematological parameters in Asian NHL patients. The purpose of this study is (1) to ascertain the frequency and pattern of MI, (2) to determine the other accompanied changes in the marrow such as the presence of lymphocytosis, eosinophilia and increase in reticulin, and (3) to determine the changes in the PB cell counts and haematological parameters in the different subtypes of NHL, in a group of Malaysian patients.

METHODS

47 patients who had been diagnosed to have NHL (based on tissue biopsies) between January 1993 and July 1995 at University Hospital Kuala Lumpur, and had available pretreatment tissue biopsies, marrow specimens and full blood counts, were studied retrospectively.

The full blood counts (consisting of haemoglobin level, total white blood cell count and platelet count) of all 47 patients at presentation were assessed for the presence of anaemia, leucopenia or leucocytosis, and thrombocytopenia or thrombocytosis by comparison with the age-specific normal ranges in the 8th edition of Practical Haematology by Dacie and Lewis. These parameters had been measured using a Coulter STKS counter (Coulter Electronics, Miami, Florida).

The PB film at diagnosis (retrieved in 46 patients) had been stained using the Leishman's stain. A manual differential count was performed on each patient's blood film by counting 200 white blood cells per film. Lymphoma cells, atypical lymphocytes and myeloid precursors were included in the differential counts. Examination for the presence of nucleated red cells was also carried out. The absolute counts of neutrophils, lymphocytes, monocytes and eosinophils were calculated using the manually obtained differential counts, and

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the presence of neutropenia or neutrophilia, lymphocytopenia or lymphocytosis, monocytosis and eosinophilia were determined by comparing with the normal ranges outlined in the 8th edition of Practical Haematology by Dacie and Lewis.

Adequate marrow smears (retrieved in 42 patients) had been obtained by unilateral aspiration of marrow from the posterior superior iliac crest using Klima needles. They were fixed using absolute methanol, stained with May-Grunwald-Giemsa's stain and evaluated by light microscopy for the presence of lymphoma cells. A differential count was also performed by counting 500 nucleated cells. Marrow lymphocytosis as detected by examination of the marrow smear was defined as the presence of more than 20% lymphocytes, and eosinophilia as the presence of more than 6% eosinophils and eosinophilic precursors, in patients above 12 years of age⁽⁴⁾. For patients who were 12 years old or younger, the normal ranges used were as outlined by Glaser K et al⁽⁵⁾.

Adequate unilateral trephine biopsies (retrieved in 46 patients) had been obtained using Jamshidi needles from the same anatomical site as the aspirates, fixed in 10% buffered formalin, decalcified using 5% formic acid and embedded in paraffin. 4 micron-thick sections were stained with haematoxylin and eosin as well as silver reticulin stain using the Gordon and Sweet method, and evaluated by both the authors. The presence of lymphoma infiltrates, increase in reticulin fibres as well as other abnormalities were recorded. The pattern of trephine involvement by lymphoma was classified according to guidelines given by Bain BJ⁽⁶⁾. The presence of aggregates of lymphocytic cells were regarded as benign in nature if they were small, with cells which appeared mature, had a well-defined border and were randomly distributed⁽⁷⁾. Immunohistochemical stains

were performed on the trephine biopsies when deemed necessary, to assist in the detection of MI. A standard three-stage immunoperoxidase technique, following antigen retrieval using the microwave was used. A panel of antibodies against CD20 antigen (L26), CD30 antigen (Ber-H2), CD61 (RUU-PL7F12, Becton Dickinson, CA) and CD3 antigen (polyclonal CD3) were utilised.

The diagnostic tissue biopsy material for all the patients studied was retrieved and reviewed by both authors. The lesions were subclassified using the proposed list by the International Lymphoma Study Group (REAL). The slides were stained using haematoxylin and eosin. Immunohistological studies were performed on 45 cases using the same staining technique as mentioned above. A panel of antibodies against the CD15 antigen (Leu-M1, Becton Dickinson, CA), CD20 antigen (L26), CD30 antigen (Ber-H2), CD43 antigen (DFT1), CD45 antigen (DAKO-LC), CD45RO antigen (UCHL1) and CD3 antigen (polyclonal CD3) were used. Unless otherwise stated, all antibodies used for the trephine and tissue biopsies were from Dako, Denmark.

The frequencies of each abnormal feature were tabulated according to the NHL subtype. The data was analysed with non-parametric tests using the SPSS 8.0 programme. Analysis of correlation between the haematological findings and the different subtypes of NHL was not performed due to the small numbers of patients in each subtype.

RESULTS

Of 47 patients studied, 29 had B-NHL, 15 had T-NHL and 1 patient had non-B, non-T NHL. Tissue immunophenotyping was not performed in 2 patients due to the inadequacy of material. 10 (21.3%) had

Table I. The changes in the full blood count in each subtype of NHL.

NHL subtype	Anaemia	TWBC*		Platelet		Abnormal counts	Multiple cytoperna
		Decr [#]	Incr [#]	Decr	Incr		
LB	6/10 (60%)	1/10 (10%)	4/10 (40%)	2/10 (20%)	4/10 (40%)	8/10 (80%)	2/10 (20%)
PTCL (unspecified)	7/10 (70%)	2/10 (20%)	3/10 (30%)	4/10 (40%)	0/10 (0%)	9/10 (90%)	4/10 (40%)
BLCNHL	9/14 (64.3%)	2/14 (14.3%)	3/14 (21.4%)	4/14 (28.6%)	4/14 (28.6%)	12/14 (85.7%)	4/14 (28.6%)
BL	4/5 (80%)	0/5 (0%)	1/5 (20%)	1/5 (20%)	2/5 (40%)	5/5 (100%)	1/5 (20%)
Others	5/8 (62.5%)	0/8 (0%)	1/8 (12.5%)	1/8 (12.5%)	1/8 (12.5%)	6/8 (75%)	0/8 (0%)
Total	31/47 (66%)	5/47 (10.6%)	12/47 (25.5%)	12/47 (25.5%)	11/47 (23.4%)	40/47 (85.1%)	11/47 (23.4%)

*TWBC – total white blood cell count # Incr – increased; Decr - decreased

lymphoblastic lymphoma (LB), 10 (21.3%) had peripheral T-cell lymphoma (unspecified) (PTCL-u), 14 (29.8%) had diffuse large B-cell lymphoma (BLCNHL) and 5 (10.6%) had Burkitt's lymphoma (BL). Of the 10 patients with LB, 3 had B-LB, 5 had T-LB while the remaining 2 cases were the ones without immunohistochemical staining. No significant difference was noted in the frequencies of each of these 4 main subtypes ($p=0.243$, chi square test). Of the remaining 8 (17%), 2 had B mantle cell NHL (BNHLMC), 2 had follicular lymphoma (FL), 1 had T-cell rich, B cell lymphoma (TCRBCL), 1 had non-T, non-B anaplastic large cell lymphoma (null-ALCL) and 2 cases of B-NUL could not be subclassified due to poor morphology.

The patients' ages ranged from 3 to 78 years; the mean age was 34.2 years. 11 patients were 12 years old

or younger, while the remaining 36 were more than 12 years old. A significant difference was noted between the mean ages of patients in the different subtypes of NHL ($p=0.001$, Kruskal Wallis test). BLCNHL affected mainly older patients (mean age: 53.7 years) while BL and LB affected mainly younger patients. The mean ages of BL patients and LB patients were 11.6 years and 19.6 years respectively. LB was diagnosed in 6 of the 36 patients (16.7%) who were above 12 years of age and 4 of the 11 patients (36.4%) who were 12 years old or younger. PTCL-u was seen in all age groups (mean age: 35.1 years). There were 32 male patients (68.1%) and 15 female patients (31.9%). This difference was statistically significant ($p=0.020$, binomial test). The male patients predominated in every subtype seen in this series.

Table II. The changes in the counts of the peripheral white blood cells in each NHL subtype.

NHL subtype	Neutrophils		Lymphocytes		monocytosis	Eosinophilia	lymphoma cells	A-typical lymphocytes	m/nrbc*
	Decr	incr	Decr	incr					
LB	3/9 (33.3%)	1/9 (11.1%)	2/9 (22.2%)	4/9 (44.4%)	1/9 (11.1%)	1/9 (11.1%)	1/9 (11.1%)	6/9 (66.7%)	2/9 (22.2%)
PTCL (unspecified)	2/10 (20%)	3/10 (30%)	5/10 (50%)	0/10 (0%)	0/10 (0%)	1/10 (10%)	0/10 (0%)	0/10 (0%)	2/10 (20%)
BLCNHL	2/14 (14.3%)	4/14 (28.6%)	6/14 (42.9%)	0/14 (0%)	1/14 (7.1%)	0/14 (0%)	0/14 (0%)	2/14 (14.3%)	6/14 (42.9%)
BL	0/5 (0%)	0/5 (0%)	1/5 (20%)	1/5 (20%)	0/5 (0%)	0/5 (0%)	1/5 (20%)	2/5 (40%)	1/5 (20%)
Others	1/8 (12.5%)	1/8 (12.5%)	5/8 (62.5%)	1/8 (12.5%)	0/8 (0%)	0/8 (0%)	1/8 (12.5%)	1/8 (12.5%)	2/8 (25%)
Total	8/46 (17.4%)	9/46 (19.6%)	19/46 (41.3%)	6/46 (13%)	2/46 (4.3%)	2/46 (4.3%)	3/46 (6.5%)	11/46 (23.9%)	13/46 (28.3%)

* myeloid precursors and / or nucleated red blood cells

Table III. The changes in the bone marrow in each subtype of NHL.

NHL subtype	MI*	lymphoma		pattern		lymphocytosis	eosinophilia	Increased reticulin
		diffuse random	focal para* *	focal	Interstitial			
LB	6/10 (60%)	5/5 (100%)	0/5 (0%)	0/5 (0%)	0/5 (0%)	5/10 (50%)	4/10 (40%)	3/9 (33.3%)
PTCL (unspecified)	3/10 (30%)	1/3 (33.3%)	2/3 (66.7%)	0/3 (0%)	0/3 (0%)	3/10 (30%)	5/10 (50%)	4/9 (44.4%)
BLCNHL	6/14 (42.9%)	4/6 (66.7%)	2/6 (33.3%)	0/6 (0%)	0/6 (0%)	2/14 (14.3%)	6/14 (42.9%)	4/13 (30.8%)
BL	2/5 (40%)	2/2 (100%)	0/2 (0%)	0/2 (0%)	0/2 (0%)	0/5 (0%)	4/5 (80%)	2/5 (40%)
Others	5/8 (62.5%)	3/5 (60%)	0/5 (0%)	1/5 (20%)	1/5 (20%)	0/8 (0%)	2/8 (25%)	5/8 (62.5%)
Total	22/47 (46.8%)	15/21 (71.4%)	4/21 (19%)	1/21 (4.8%)	1/21 (4.8%)	10/47 (21.3%)	21/47 (44.7%)	18/44 (40.9%)

* marrow involvement

** focal paratrabecular

Changes in the peripheral blood

The findings are detailed in Tables I and II. There is frequent occurrence of at least one abnormal parameter in the full blood count irrespective of the lymphoma subtype. The frequency of anaemia ranged from 60% to 80%. Lymphocytopenia was generally seen more commonly than lymphocytosis ($p=0.015$, binomial test), with the exception of LB patients. The other peripheral haematological changes occurred less commonly. PB lymphoma cells was only seen in 3 patients: 1 had LB, 1 had BL and the third had BNHLMC. Leucoerythroblastosis (simultaneous presence of circulating red blood cells and myeloid precursors) was noted in the PB films of 6 out of 46 patients (13.0%), and all of them had marrow involvement. Of the 6, 2 had LB, 3 had BLCNHL, and 1 was a case of TCRBCL.

Changes in the bone marrow

The findings are detailed in Table III. Approximately half the total number of cases had MI. Infiltration was questionable in one case of PTCL-u, where pleomorphic medium to large-sized lymphoid cells with prominent nucleoli and high mitotic rate were seen, occurring interstitially and in groups of two or three in one area in the trephine biopsy. These cells did not appear to express either T or B-cell antigens. No lymphoma cells were detected in the marrow smear of this patient but an increase in ruptured cells was seen in some areas. Diffuse marrow infiltration was more commonly seen compared to the other patterns of infiltration. However, this finding was not statistically significant ($p=0.078$, binomial test).

21 of 47 patients had marrow eosinophilia, and of these, only 2 had a spill-over of eosinophils into the blood. The other manifestations were uncommon: increased marrow vascularity in 1 case of PTCL-u with MI, necrosis in another case of PTCL-u with MI, increased histiocytes in one case of BLCNHL without MI and one TCRBCL patient with infiltration, increased plasma cells in one BLCNHL patient without MI.

B-NHL versus T-NHL

29 patients had B-NHL and 15 had T-NHL. No significant difference was found between B and

T-NHL with regards to the frequency of abnormal PB haematological parameters (Fisher's exact test or Continuity correction^a, chi-square tests). Some of the findings are listed in Table IV. 17.2% (5/29) of B-NHL patients and 33.3% (5/15) of T-NHL patients had leucocytosis. 27.6% (8/29) of B-NHL patients and 6.7% (1/15) of T-NHL patients had thrombocytosis. Both peripheral monocytosis and eosinophilia were generally uncommon and were seen only in 3.4% (1/29) of B-NHL patients and 6.7% (1/15) of T-NHL patients respectively. Peripheral lymphoma cells were detected in 10.3% (3/29) of B-NHL patients but was not seen in any of the T-NHL patients. Peripheral atypical lymphocytes were seen in 27.6% (8/29) of B-NHL patients and 20.0% (3/15) of T-NHL patients.

Patients with B and T-NHLs also did not differ significantly with regards to frequency of the following abnormalities in the marrow: involvement by lymphoma ($p=0.384$, Continuity correction^a, chi square tests), eosinophilia ($p=1.000$, Continuity correction^a, chi square tests) and increase in reticulin ($p=0.544$, Continuity correction^a, chi square tests). Marrow lymphocytosis appeared to be more common in T-NHL, occurring in 40.0% (6/15), but was seen only in 13.8% (4/29) of the B-NHL patients. This observation was however not statistically significant ($p=0.067$, Fisher's exact test). 55.2% (16/29) of B-NHL cases and 35.7% (5/14) of T-NHL cases had MI. Increased marrow reticulin was seen in 46.4% (13/28) of B-NHL patients and 4/13 (30.8%) of T-NHL patients. Of the involved B-NHL cases, 75.0% (12/16) had diffuse infiltration, 12.5% (2/16) had focal random infiltration, 6.3% (1/16) had focal paratrabecular infiltration and 6.3% (1/16) had interstitial infiltration. Of the involved T-NHL cases, half had diffuse infiltration and the other half had focal random infiltration.

In the patients with B-NHL, marrow eosinophilia was more commonly found in the 13 patients without marrow infiltration (9/13) than in the 16 with involvement (4/16). This difference was statistically significant ($p=0.045$, Continuity correction^a, chi square tests). In the patients with T-NHL, the opposite was noted: marrow eosinophilia was seen more commonly in the 5 patients with MI (3/5) but less frequently in the 9 without infiltration (3/9). However, the difference in this case was not statistically significant ($p=0.580$, Fisher's exact test).

DISCUSSION

The frequency of anaemia in our patients (66%) was the highest when compared with other reports: 7% (9 out of 121 patients) by Stein et al, 37% by Bloomfield et al, 42% by Conlan et al and 37.5% (high grade NHL) – 53.3% (low grade NHL) by Cheng et al^(2,3,8,9). The

Table IV. Full blood counts and peripheral blood cell counts: B-NHL versus T-NHL

	Anaemia	Leucopenia	Thrombo- cytopenia	Multiple- cytopenias	Neutropenia	Lympho- cytopenia	m/nrbc
B-NHL	19/29 (65.5%)	3/29 (10.3%)	8/29 (27.6%)	7/29 (24.1%)	5/29 (17.2%)	11/29 (37.9%)	11/29 (37.9%)
T-NHL	9/15 (60.0%)	2/15 (13.3%)	4/15 (26.7%)	4/15 (26.7%)	3/15 (20.0%)	7/15 (46.7%)	2/15 (13.3%)
p values	0.976	1.000	1.000	1.000	1.000	0.814	0.162

criteria for anaemia in Stein et al's study was a haemoglobin level of less than 12 g/dl, in Bloomfield's study was haemoglobin level below 12 g/dl for females and below 14 g/dl for males. Anaemia was defined by Conlan et al as a haemoglobin level less than 12.5 g/dl; and by our group as haemoglobin level below 11-13 g/dl (differed with age and sex). Cheng et al did not state the normal range of haemoglobin used by them^(2,3,8,9). These variations in criteria are small and unlikely to bias the results significantly. Hence, the higher incidence of anaemia noted in this series is more real than apparent.

The presence of at least one abnormal parameter in the full blood count was noted in 85.1% of our patients at the time of diagnosis, compared with 57% (54 out of 94 patients) by Lai et al, 57% by Bloomfield et al and 63% by Conlan et al^(2,3,10). Similar criteria were utilised by these 3 groups of authors and our group to define anaemia, leucopenia and leucocytosis^(2,3,10). However, Lai et al and Bloomfield et al used more stringent criteria for thrombocytopenia and thrombocytosis. Thrombocytopenia was defined as platelet counts of less than 100,000/mm³ by Lai et al and less than 140,000/mm³ by Bloomfield et al, while thrombocytosis was defined as counts of more than 450,000/mm³ by the former and 440,000/mm³ by the latter^(3,10). This does not appear to have contributed much to the disparity observed, as the incidence of abnormal counts in our study did not differ much even when these cut-off points were used. Therefore, NHL appears to have a greater degree of adverse effect on the haematological system in our patients.

Another common finding was PB lymphocytopenia, which was seen in almost half of the population studied. A large proportion of patients with this abnormality had either PTCL-u or BLCNHL. Bloomfield et al also noted a similar incidence of peripheral lymphocytopenia in their patients (42% in poorly differentiated nodular lymphocytic lymphoma – 56% in histiocytic lymphoma, diffuse)⁽³⁾. We did not rule out other causes of lymphocytopenia like HIV infection and autoimmune disease and neither did Bloomfield et al⁽³⁾. The questions that remain unanswered are whether the lymphocytopenia preceded and predisposed to the development of NHL, or was it a consequence of this disease. Further studies may be able to clarify this and determine the effect of this abnormality on the patient.

The overall incidence of MI by NHL in this series was 46.8%. This figure is within the range of incidences noted from other studies, that is, 29% to 69%⁽⁸⁻¹⁵⁾. MI is far from rare in NHL. A much higher incidence of diffuse marrow infiltration was seen in this series (71.4%, 15 out of 21 cases), compared to that reported by Foucar K et al (27 out of 90 patients)⁽¹¹⁾. This may be due in part to the larger numbers of LB and BL patients seen

in this study when compared to others^(11,14, 16-18). There also appeared to be a higher incidence of diffuse infiltration in the BLCNHL patients (4 of 6 patients)^(11,19).

We appear to be one of the first groups to utilise the NHL classification scheme proposed by the International Lymphoma Study Group (REAL) for a study on haematological abnormalities in NHL subtypes. On extensive literature search, we found that most, if not all of the reports published so far on this particular area of research utilised other classification Systems like the Working Formulation, Lukes-Collins classification and Rappaport classification. Comparison of our data with data from these authors was not easy in some cases because of this. However, the REAL and other comparable schemes are being used more commonly worldwide in lymphoma classification, and this will simplify comparison with future studies.

In LB patients, common findings at initial diagnosis were anaemia, leucocytosis (usually due to lymphocytosis), thrombocytosis, neutropenia and peripheral atypical lymphocytes. Conlan et al recorded similar findings in patients with high grade NHL, but no data specific for LB cases were described in their paper⁽²⁾. MI was common in LB patients in our series (60%) and all the patients showed a diffuse pattern of infiltration. These concur with findings by others: Stein et al noted MI in 61% of 33 LB patients⁽⁸⁾; Foucar et al recorded infiltration in 60% of 10 LB cases, all of whom had diffuse infiltration⁽¹¹⁾; Lai et al reported a 56% incidence of marrow disease in 18 LB cases, with 5 out of 8 cases who had trephine biopsies showing diffuse involvement⁽¹⁰⁾.

The more common abnormalities of peripheral blood haematological parameters observed in our PTCL-u patients were anaemia, lymphocytopenia, thrombocytopenia and multiple cytopenias. These findings were largely similar to those noted by Hanson et al⁽²⁰⁾. Weisenburger et al reported a similar incidence (64%, 25 of 39 patients) of lymphocytopenia in their series⁽²¹⁾. The reason for the reduction in lymphocytes is not clear.

MI was noted in 30% of our patients with PTCL-u and 43% of patients in a study by Liang et al⁽²²⁾, as compared to 80% (24 out of 30) by Hanson et al⁽²⁰⁾, 73% (27 out of 37) by Gaulard et al⁽²³⁾, and 60% by McKenna et al⁽²⁴⁾. Foucar et al reported a 65% incidence (11 of 17 cases) of infiltration in T-cell lymphoma patients⁽¹¹⁾. It appears that the reported incidences of marrow disease in Asian patients (Liang et al and our study) are lower than the Western figures. The reason is not immediately apparent, but an ethnic difference in the incidence of MI by PTCL remains a possibility and more Asian patients should be studied to investigate this. The infiltration pattern noted in our series was either

diffuse or randomly focal; focal paratrabecular infiltration was not seen. This observation was similar to that noted in other studies, and emphasizes the rarity of the focal paratrabecular pattern of marrow involvement in PTCL^(20,23-26).

Lymphocytopenia, and the presence of peripheral myeloid precursors and/or nucleated red blood cells were the common findings in BLCNHL. The incidence of marrow infiltration (42.9%) noted here was slightly higher compared to that reported by Foucar et al (13 of 41 patients, 31%) in the Lukes-Collins equivalents of BLCNHL⁽¹¹⁾. The diffuse pattern of marrow infiltration predominated in our study (4 of 6 patients, 66%) while Foucar et al noted this pattern of infiltration in only 5 of 11 patients (45.5%), with their remaining 6 cases showing focal infiltration⁽¹¹⁾. McKenna RW also observed a highly variable pattern of marrow disease in the Working Formulation equivalents of BLCNHL⁽¹⁹⁾. However, larger numbers of patients need to be examined to determine if our BLCNHL patients do indeed have a higher tendency to have diffuse marrow infiltration.

Marrow eosinophilia, thrombocytosis, and PB atypical lymphocytes were common features in BL patients. A 40% incidence of MI was noted which was within the range of 0% - 57% recorded by Foucar et al⁽¹¹⁾, Jones et al⁽²⁷⁾, Stein et al⁽⁸⁾ and Brunning et al⁽²⁸⁾. Both the BL patients in our series had diffuse involvement of the marrow, and this was similar to the findings of Foucar et al⁽¹¹⁾ and Brunning et al⁽²⁹⁾.

In our study, no significant difference was noted between B-NHL and T-NHL in most of the parameters examined. Conlan et al also did not find any significant differences between the two groups with regards to PB haematological parameters⁽²⁾. However, an interesting observation was made in that, marrow eosinophilia was more commonly found in B-NHL patients *without* MI by lymphoma (difference is statistically significant), while marrow eosinophilia was seen more frequently *with* marrow infiltration in T-NHL patients. T-lymphocytes and T-lymphoma cells have been found to cause eosinophilia by secreting eosinophil chemotactic factors and excess interleukin 5, while malignant B-lymphoid cells were noted to have the ability to stimulate normal T-lymphocytes and other peripheral blood mononuclear cells to produce eosinophil chemotactic factor⁽³⁰⁻³⁵⁾. Further studies need to be carried out to ascertain if these observations have any correlation with our findings.

ACKNOWLEDGEMENTS

This project was funded by IRPA grant (number: 06-02-03-0576). We would like to thank Mr Lim Meng Yong, of the Department of Pathology, University of Malaya (UM), for performing the immunohistological

stains, and Mr Ong Leong Huat of the Department of Social and Preventive Medicine, UM, for his assistance with the statistical analysis of data.

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