Beautiful Bone Marrows
making sense of all things myeloid

BLPG, Bristol, May 2014

Dr Zbigniew Rudzki
Consultant Histopathologist
Honorary Senior Lecturer,
School of Cancer Sciences

zbigniew.rudzki@heartofengland.nhs.uk
aesthetically pleasing marrows...
not all isolated 5q- cases have the classical megakaryocytic morphology
not everything which looks like 5q- syndrome is 5q- syndrome:

complex karyotype, including 5q-
> 10% of blasts

CD34

RAEB2
digression:

simple immunohistochemistry and 5q- prognosis

**strong nuclear p53**

*35% of cases (n=85)*

Saft L et al. Haematologica 2014, ahead of print
Mimics of MDS and AML

Polish, wz. 1993 under heavily French influence, as usually
Megaloblastic anaemia (B12)

note
'dysplastic'
Mgk!
Megaloblastic anaemia – folate deficiency
MCV = 87

Vit B12 deficiency & Fe deficiency
Toxic myeloid maturation arrest

CD34
M 40’ HIV+, new fever, anaemia and lymphadenopathy
Had Hodgkin’s lymphoma at that time...

HIV “Myelopathy”
Acute myeloid leukaemia in human immunodeficiency virus-infected adults: epidemiology, treatment feasibility and outcome

Laurent Sutton, Pascal Guenel, Marie-Laure Tanguy, Bernard Rio, Nathalie Dhedin, Philippe Casassus and Olivier Lortholary for the French Study Group on Acute Myeloid Leukaemia in HIV-infected Patients

British Journal of Haematology 2001. 112, 900–908

Summary. The epidemiology and clinical outcome of acute myeloid leukaemia in human immunodeficiency virus (HIV)-infected adults is poorly documented. We retrospectively surveyed all French haematology centres for adult acute myeloid leukaemia (AML) cases diagnosed between January 1990 and July 1996 who were found to be HIV-seropositive before or at the time of AML diagnosis. Medical charts were reviewed to determine the stage of HIV infection, the characteristics of AML and the response of AML to chemotherapy. Sixteen cases of AML (13 men, three women) were reported by 12 haematology units. Based on assumptions on the size, age and sex distribution of the HIV-infected population in France, the estimated risk of AML in 1990 to 1996 among HIV-infected adults was twice that of the general population (standardized incidence ratio = 2.05; 95% confidence interval: 1.17–3.34). Two other cases occurring before 1996 were spontaneously notified to the authors and were included in the clinical analysis. At AML diagnosis, the median CD4$^+$ cell count was $275 \times 10^9/l$ and nine patients had acquired immune deficiency syndrome (AIDS). Fifteen patients were scheduled for remission-induction therapy of AML. No deaths were related to AML treatment. Complete remission was obtained in 11 out of 15 patients. Three patients were long-term survivors: two remain alive in complete remission at 8 years and 9 years, respectively, and the third died of AIDS at 8 years. A CD4$^+$ cell count above $200 \times 10^9/l$ at AML diagnosis was predictive of longer survival (log-rank test: $P = 0.004$). Like many other malignancies, the incidence of AML appears to be increased in HIV-infected patients. Our results show a twofold higher incidence, although this needs to be confirmed in a specifically designed prospective epidemiological study. Such patients, especially those with CD4$^+$ cell counts above $200 \times 10^9/l$ at AML diagnosis, should receive remission-induction therapy, which can confer long-term survival.

Keywords: acute myeloid leukaemia, HIV infection, chemotherapy.
Clinical and cytogenetic characteristics of myelodysplastic syndrome in patients with HIV infection

Koichi Takahashi\textsuperscript{a,c,*}, Mariko Yabe\textsuperscript{e}, Ilan Shapira\textsuperscript{d,g}, Sherry Pierce\textsuperscript{b}, Guillermo Garcia-Manero\textsuperscript{b}, Mala Varma\textsuperscript{f,g}

\textsuperscript{a} Division of Cancer Medicine, The University of Texas MD Anderson Cancer Center, Houston, TX, USA
\textsuperscript{b} Department of Leukemia, The University of Texas MD Anderson Cancer Center, Houston, TX, USA
\textsuperscript{c} Department of Medicine, Beth Israel Medical Center, Albert Einstein College of Medicine, New York, NY, USA
\textsuperscript{d} Department of Hematology and Oncology, Beth Israel Medical Center, Albert Einstein College of Medicine, New York, NY, USA
\textsuperscript{e} Department of Pathology, St. Luke's-Roosevelt Hospital Center, Columbia University College of Physicians and Surgeons, New York, NY, USA
\textsuperscript{f} Department of Hematology and Oncology, St. Luke's-Roosevelt Hospital Center, Columbia University College of Physicians and Surgeons, New York, NY, USA
\textsuperscript{g} Continuum Cancer Centers of New York. New York, NY, USA

\section*{ABSTRACT}

We report \textbf{eight patients} of myelodysplastic syndrome (MDS) with HIV infection. Compared to a historical cohort of HIV-uninfected MDS patients, HIV/MDS were younger ($p=0.019$), had more complex cytogenetics ($p=0.015$), and more often had \textbf{7q deletion or monosomy} ($p=0.011$). \textbf{In five patients, HIV/MDS transformed to acute myeloid leukemia} with a median time to transformation of 7 months. Also, the median overall survival was shorter in the HIV/MDS than in their HIV-uninfected counterparts (8 vs. 22 months; $p=0.003$). These results suggest that HIV/MDS is a \textbf{high-risk MDS} necessitating thorough follow-up.

© 2012 Elsevier Ltd. All rights reserved.

median survival 8 vs. 22 months
M40

long history of HIV infection (currently negligible viral load)

2003: B-ALL
2013: sudden pancytopenia

CD34
CD61
M82

Pancytopenia
Transfusion-dependent
Excessive alcohol consumption

<table>
<thead>
<tr>
<th>Hb</th>
<th>9</th>
<th>g/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>plt</td>
<td>60</td>
<td>x 10^9/L</td>
</tr>
<tr>
<td>N</td>
<td>0.7</td>
<td>x 10^9/L</td>
</tr>
<tr>
<td>Ly</td>
<td>0.7</td>
<td>x 10^9/L</td>
</tr>
</tbody>
</table>

no significant increase in blasts on BM aspirate and flow cytometry

no dysplasia on BM aspirate
normal karyotype
...but remains asymptomatic & stable for last 6 years
<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Country</th>
<th>Cases</th>
<th>Controls</th>
<th>OR</th>
<th>95% CI</th>
<th>Type of controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pekmezovic T</td>
<td>2006</td>
<td>Serbia Montenegro</td>
<td>37</td>
<td>43</td>
<td>2.96</td>
<td>1.67–5.27</td>
<td>Hospital</td>
</tr>
<tr>
<td>Strom SS</td>
<td>2005</td>
<td>US, Texas</td>
<td>172</td>
<td>73</td>
<td>0.76</td>
<td>0.51–1.11</td>
<td>Hospital</td>
</tr>
<tr>
<td>Dalamaga M</td>
<td>2002</td>
<td>Greece</td>
<td>83</td>
<td>1</td>
<td>2.02</td>
<td>0.18–22.76</td>
<td>Hospital</td>
</tr>
<tr>
<td>Nagata C</td>
<td>1999</td>
<td>Japan</td>
<td>55</td>
<td>55</td>
<td>1.42</td>
<td>0.84–2.42</td>
<td>Population</td>
</tr>
<tr>
<td>Ido M</td>
<td>1996</td>
<td>Japan</td>
<td>56</td>
<td>60</td>
<td>1.31</td>
<td>0.79–2.18</td>
<td>Hospital</td>
</tr>
</tbody>
</table>

**Review**

**Smoking and alcohol intake as risk factors for myelodysplastic syndromes (MDS)**

Yan Du a, Jon Fryzek b, Mikkael A. Sekeres c, Emanuela Taioli d, *

a Department of Epidemiology, University of Pittsburgh, Graduate School of Public Health, Pittsburgh, PA, USA
b Global Epidemiology, Amgen Inc., Thousand Oaks, CA, USA
c Cleveland Clinic Taussig Cancer Institute, Cleveland, OH, USA
d Department of Epidemiology and Biostatistics, SUNY Downstate, 450 Clarkson Ave., Brooklyn, NY 11203, USA

**ABSTRACT**

The term myelodysplastic syndromes (MDS) include a diverse group of diseases in which the bone marrow production of blood cells is disrupted. In spite of the wealth of information on therapeutic options, little is known about the epidemiology of MDS, including population variations and risk factors. A narrative review of published literature and meta-analyses were conducted, identifying and summarizing key reports that describe the association between smoking, alcohol, and MDS. There were ten case-control studies that looked at the association between smoking and MDS, for a total of 1839 cases and 2831 controls. The meta-estimate for the association between ever smoking and MDS was 1.45 (95% CI: 1.21–1.74), with heterogeneity among studies (p = 0.05), but no evidence of publication bias. The relationship between alcohol consumption and MDS has been examined in five studies, including 745 cases and 1642 controls. The overall association was 1.31 (95% CI: 0.79–2.18), with significant heterogeneity (p = 0.003) and no evidence of publication bias. This re-analysis of published data strongly suggests that smoking is significantly associated with MDS, while alcohol does not seem to play a major role in MDS etiology. Large epidemiological studies incorporating biomarkers of exposure, along with pooled analysis are needed to better address the contribution of lifestyle factors to the development of MDS.

© 2009 Elsevier Ltd. All rights reserved.
Mimics of Myeloproliferative Neoplasms

Germany, 1989 Truppenversuch. The Best.
F53

Summer 2012: liver failure of unclear aetiology

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total white cell count</td>
<td>42.46</td>
<td>Dec 2012</td>
</tr>
<tr>
<td>Red blood cell (RBC) count</td>
<td>3.26</td>
<td>10^{12}/l</td>
</tr>
<tr>
<td>Haemoglobin estimation</td>
<td>10.0</td>
<td>g/dl</td>
</tr>
<tr>
<td>Haematocrit</td>
<td>0.333</td>
<td>l/l</td>
</tr>
<tr>
<td>Mean corpuscular volume (MCV)</td>
<td>102.1</td>
<td>fl</td>
</tr>
<tr>
<td>Mean corpuscular haemoglobin (MCH)</td>
<td>30.7</td>
<td>pg</td>
</tr>
<tr>
<td>Mean corpuscular Hb. conc. (MCHC)</td>
<td>30.0</td>
<td>g/dl</td>
</tr>
<tr>
<td>RBC Distribution Width</td>
<td>14.6</td>
<td>%</td>
</tr>
<tr>
<td>Platelet count</td>
<td>483</td>
<td>10^{9}/l</td>
</tr>
<tr>
<td>Platelet distribution width</td>
<td>11.0</td>
<td></td>
</tr>
<tr>
<td>Neutrophil count (Absolute)</td>
<td>37.24</td>
<td>x10^{9}/l</td>
</tr>
<tr>
<td>Lymphocyte count (Absolute)</td>
<td>1.80</td>
<td>x10^{9}/l</td>
</tr>
<tr>
<td>Monocyte count (Absolute)</td>
<td>1.49</td>
<td>x10^{9}/l</td>
</tr>
<tr>
<td>Eosinophil count (Absolute)</td>
<td>1.64</td>
<td>x10^{9}/l</td>
</tr>
<tr>
<td>Basophil count (Absolute)</td>
<td>0.29</td>
<td>x10^{9}/l</td>
</tr>
<tr>
<td>Nucleated RBC</td>
<td>0.00</td>
<td>x10^{9}/l</td>
</tr>
</tbody>
</table>

BCR/ABL (-)
JAK2 WT
karyotype normal
no dysplastic features on aspirate/film
no excess of blasts


denies excessive alcohol consumption (statement supported by the patient’s husband)
CONCLUSION

TREPHINE BONE MARROW BIOPSY:

- UNRESOLVED DIFFERENTIAL DIAGNOSIS BETWEEN CHRONIC MYELOMONOCYTIC LEUKAEMIA (CMML-1) AND FLORID REACTIVE CHANGES OF UNKNOWN ORIGIN (possibly secondary to the liver disease).

2nd opinion:

Suspicious but not diagnostic of myeloproliferative or overlap myelodysplastic/ myeloproliferative process although not classifiable and reaction cannot be excluded.
liver biopsy
alcoholic hepatitis
(diagnosis: Dr G Langman)
a bottle of wine per day
(in the morning)


...I was pleased to see you looking quite well in yourself, having put on a lot of weight and also pleased to report that your blood tests are also normalising.

I think this has all been achieved following rigorous abstention from alcohol consumption ...
M, late 40'

history of thrombocytosis and polycythaemia splenomegaly at presentation (2005)
JAK2-negative

diagnosis of myeloproliferative disorder in outside institution

put on Hydroxycarbamide, later discontinued - cytopenias

no evidence of paraproteinaemia
A lot of confusion
I have suspected a lymphoma...

POEMS syndrome strongly suggested by a Dermatologist
Bone marrow histopathology in POEMS syndrome: a distinctive combination of plasma cell, lymphoid, and myeloid findings in 87 patients

Linda N. Dao, Curtis A. Hanson, Angela Dispenzieri, William G. Morice, Paul J. Kurtin, and James D. Hoyer

Department of Laboratory Medicine and Pathology and Department of Hematology, Mayo Clinic, Rochester, MN

POEMS is an uncommon syndromic disorder characterized by polyneuropathy, organomegaly, endocrinopathy, monoclonal protein, and skin changes. There are few descriptions of the bone marrow pathology of POEMS; therefore, peripheral blood smears and bone marrow aspirates and biopsies from 87 patients (143 total, bridization. Monotypic plasma cells were detected in 44 pretreatment cases (66%); the majority of plasma cells expressed lambda light chain (91%). The monotypic plasma cells typically were present in a background of increased polytypic plasma cells. Lymphoid aggregates were found in 33 (49%) pretreatment cases and in tested had the JAK2V617F mutation. In summary, we have identified a novel constellation of features that should strongly suggest POEMS syndrome as part of the differential diagnosis. The constellation of lambda-restricted monoclonal gammopathy, plasma cell rimming around lymphoid aggregates, and megakaryocytic hyperplasia in a bone marrow is highly suggestive of this diagnosis, especially in the context of a peripheral neuropathy. (Blood. 2011;117(24):6436-6444)

Table 1. Summary of clinical findings of patients with POEMS syndrome

<table>
<thead>
<tr>
<th>Total patients, n = 87</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y (range, median)</td>
</tr>
<tr>
<td>(20-74, 49)</td>
</tr>
<tr>
<td>Sex</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>57</td>
</tr>
<tr>
<td>Female</td>
</tr>
<tr>
<td>30</td>
</tr>
<tr>
<td>CBC</td>
</tr>
<tr>
<td>Hemoglobin, g/dL (range, median)</td>
</tr>
<tr>
<td>(7.8-17.7, 13.7)</td>
</tr>
<tr>
<td>White blood cells, ×10^9/L (range, median)</td>
</tr>
<tr>
<td>(0.3-18.8, 6.4)</td>
</tr>
<tr>
<td>Platelets, ×10^9/L (range, median)</td>
</tr>
<tr>
<td>(21-1281, 371)</td>
</tr>
<tr>
<td>Serum protein studies</td>
</tr>
<tr>
<td>IgA λ</td>
</tr>
<tr>
<td>39</td>
</tr>
<tr>
<td>IgG λ</td>
</tr>
<tr>
<td>32</td>
</tr>
<tr>
<td>IgM λ</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>IgA λ and IgG λ</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>IgG κ and IgA λ</td>
</tr>
<tr>
<td>5</td>
</tr>
<tr>
<td>IgG κ and IgG λ</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>IgG κ</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>IgA κ and IgG κ</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>None detected</td>
</tr>
<tr>
<td>4</td>
</tr>
</tbody>
</table>

'MPN-like' PB counts possible
28% - megakaryocytic atypia
35% - excess of megakaryocytes
78% - clusters of megakaryocytes
Figure 3. Bone marrow biopsy of lymphoid aggregate rimmed by PCs. (A) The lymphoid aggregate has a regressed germinal center that is Castleman-like (H&E). (B) CD138 positive PCs form a distinctive rim around the lymphoid aggregate. (C) The PCs are monotypic for λ immunoglobulin light chains (D) and negative for κ immunoglobulin light chains by IHC. Photomicrographic images were obtained with an Olympus BX51 microscope equipped with an Olympus DP71 camera and software. Original magnification 20×/0.50 UPlanFL N lens for all panels.
25 cases with PCs rimming lymphoid aggregates:
24 kappa+ PCs
1 lambda+ PCs
7 polytypic PCs
Back to 2005
Mediastinal LNs

(over 30 papers on Castleman-type lymphadenopathy in POEMS, only 1 on sarcoid-like granulomas)
M51

regular blood donor
20cm spleen found

<table>
<thead>
<tr>
<th>FBC</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total white cell count</td>
<td>18.50</td>
<td>$10^9/l$</td>
</tr>
<tr>
<td>Haemoglobin estimation</td>
<td>12.5</td>
<td>g/dl</td>
</tr>
<tr>
<td>Platelet count</td>
<td>153</td>
<td>$10^9/l$</td>
</tr>
<tr>
<td>Red blood cell (RBC)</td>
<td>4.87</td>
<td>$10^{12}/l$</td>
</tr>
<tr>
<td>Mean corpuscular volume (MCV)</td>
<td>80.7</td>
<td>fl</td>
</tr>
<tr>
<td>Mean corpuscular haemoglobin (MCH)</td>
<td>25.6</td>
<td>pg</td>
</tr>
<tr>
<td>Haematocrit</td>
<td>0.39</td>
<td>l/l</td>
</tr>
<tr>
<td>RBC Distribution Width</td>
<td>14.0</td>
<td>%</td>
</tr>
<tr>
<td>% Hypochromic RBC's</td>
<td>1.2</td>
<td>%</td>
</tr>
<tr>
<td>Neutrophil count (Absolute)</td>
<td>16.41</td>
<td>$x 10^9/l$</td>
</tr>
<tr>
<td>Lymphocyte count (Absolute)</td>
<td>1.44</td>
<td>$x 10^9/l$</td>
</tr>
<tr>
<td>Monocyte count (Absolute)</td>
<td>0.40</td>
<td>$x 10^9/l$</td>
</tr>
<tr>
<td>Eosinophil count (Absolute)</td>
<td>0.14</td>
<td>$x 10^9/l$</td>
</tr>
<tr>
<td>Basophil count (Absolute)</td>
<td>0.03</td>
<td>$x 10^9/l$</td>
</tr>
</tbody>
</table>

N $23 \times 10^9/L$ already 5 years earlier!
CD34: no excess of blasts
monoclonal IgA kappa 5.1 g/L
4% of plasma cells on aspirate
FC: kappa/lambda = 2/1
clinical letters:

“MGUS & myeloproliferative disorder”

not treated
Clonality of chronic neutrophilic leukaemia associated with myeloma: Analysis using the X-linked probe M27β

G R Standen, F J Steers, L Jones

Abstract
Aims—To determine whether myeloid proliferation was monoclonal or polyclonal in a woman with chronic neutrophilic leukaemia and myeloma.
Methods—The X-linked probe, M27β was used to determine the clonality of the neutrophil population by analysis of restriction fragment length polymorphisms and X inactivation pattern.
Results—A polyclonal pattern of X inactivation was obtained for the neutrophil population in this patient.
Conclusion—The myeloid expansion in chronic neutrophilic leukaemia associated with myeloma represents a polyclonal reactive response to the plasma cell clone rather than a co-existent myeloproliferative disorder.

(J Clin Pathol 1993;46:297–298)
Granulocyte-colony stimulating factor concentrations in a patient with plasma cell dyscrasia and clinical features of chronic neutrophilic leukaemia

M Nagai, S Oda, M Iwamoto, K Marumoto, M Fujita, J Takahara

Abstract
In order to study the pathogenesis of plasma cell dyscrasias with associated clinical features of chronic neutrophilic leukaemia, the concentration of granulocyte-colony stimulating factor (G-CSF) was measured in a patient, a 73 year old man, who underwent steroid pulse treatments and subsequently. Cultures of bone marrow cells, but large amounts of interleukin-6 were found in the culture supernatant. These observations suggest that the neutrophilia observed in the patient represented a reactive response to G-CSF secreted from abnormal plasma cells or stromal cells rather than the existence of a genuine myeloproliferative disorder.

Keywords: chronic neutrophilic leukaemia, myeloma, G-CSF, steroid, regulation.
Talking about G-CSF...

Sweden, M90
Good on long distance, poor on short distance
may reflect contemporary Swedish military doctrine
M 60, "gastric cancer, chemotherapy, anaemia"
Less known G-CSF effects mimicking myeloid neoplasia

Denmark, M84 – a rarity; anybody heard about a Danish army?
Lymphoplasmacytic Lymphoma/WM on R-CVP, neutropenia
Stem cell factor-induced bone marrow mast cell hyperplasia mimicking systemic mastocytosis (SM): histopathologic and morphologic evaluation with special reference to recently established SM-criteria.


Author information

Abstract

Although systemic mastocytosis (SM) is a well-defined hematologic neoplasm, it is sometimes difficult to discriminate between SM and a reactive mast cell (MC) hyperplasia. We describe a patient with aplastic anemia who was treated with recombinant stem cell factor (SCF). In response to SCF, the patient showed transient hematologic improvement and developed a marked increase in MC as well as a transient increase in serum tryptase. Histologic and immunohistochemical examination revealed a huge increase in MC in the bone marrow with focal infiltrates similar to SM. However, most of the SM-criteria were not met: First, MC showed normal cytomorphological characteristics without significant atypias (no cytoplasmic extensions, no oval nuclei, no hypogranulated cytoplasm). Furthermore, bone marrow MC were CD2- and CD25-negative and did not exhibit the C-KIT 2468 A-->T mutation (Asp-816-Val). After discontinuation of SCF the MC hyperplasia resolved confirming its reactive nature. Based on our case and similar cases mimicking mastocytosis, it seems of importance to apply recently established SM criteria in order to discriminate between reactive MC hyperplasia and true mastocytosis with certainty.
MPO

$N = 0.19 \times 10^9/L$

put on G-CSF

before the diagnosis
2 years later – relapse, **this time no G-CSF**

platelets were always normal or slightly low
And finally, we divide all diseases into those which are acquired and...
F 19, plt 1340, dgn.: ET, put on Hydroxycarbamide
Her 17-year old cousin, plt 840, dgn.: ET (in another hospital)
A de novo splice donor mutation in the thrombopoietin gene causes hereditary thrombocytopenia in a Polish family

Kun Liu, Robert Kralovics,* Zbigniew Rudzki, Barbara Grabowska, Andreas S. Buser, Damia Olcaydu, Heinz Gisslinger, Ralph Tiedt, Patricia Frank, Krzysztof Okoni, Anthonie P.C. van der Maas,* and Radek C. Skoda

1Experimental Hematology, Department of Biomedicine, Basel University Hospital, Basel, Switzerland, 2Department of Pathomorphology, Collegium Medicum, Jagiellonian University, Kraków, Poland, 3Department of Hematology, Ludwik Rydygier Memorial District Hospital, Kraków, Poland, Clinical Hematology, 4Basel University Hospital, Basel, Switzerland, 5Department of Internal Medicine I, Division of Hematology and Blood Coagulation, Medical University of Vienna, and 6Department of Internal Medicine, Medical Centre Haaglanden, The Hague, The Netherlands

Haematologica 2008;93:706-14
Summary

- Think twice before diagnosing 'unusual' MPN, MDS/MPN
- Have a mimic checklist
- CNL mimics are probably much more frequent than genuine CNL
- No excess of blasts – suspect a mimic ...
CD34+ (?) blasts 6 months post alloSCT for AML
CD10 ≥ PAX5 > CD79a > CD20 ≥ TdT > CD34
Summary

- Think twice before diagnosing 'unusual' MPN, MDS/MPN
- Have a mimic checklist
- CNL mimics are probably much more frequent than genuine CNL
- No excess of blasts – suspect a mimic (remember haematogones)
- Very ugly “MDS”, not-that-ugly blood counts - suspect a mimic

- Check the clinical history (referral cases!)

- Never believe self-professed teetotallers