Single Centre Experience in Use of Leukapheresis for Management of Leucostasis in High Count Leukaemia.

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Introduction

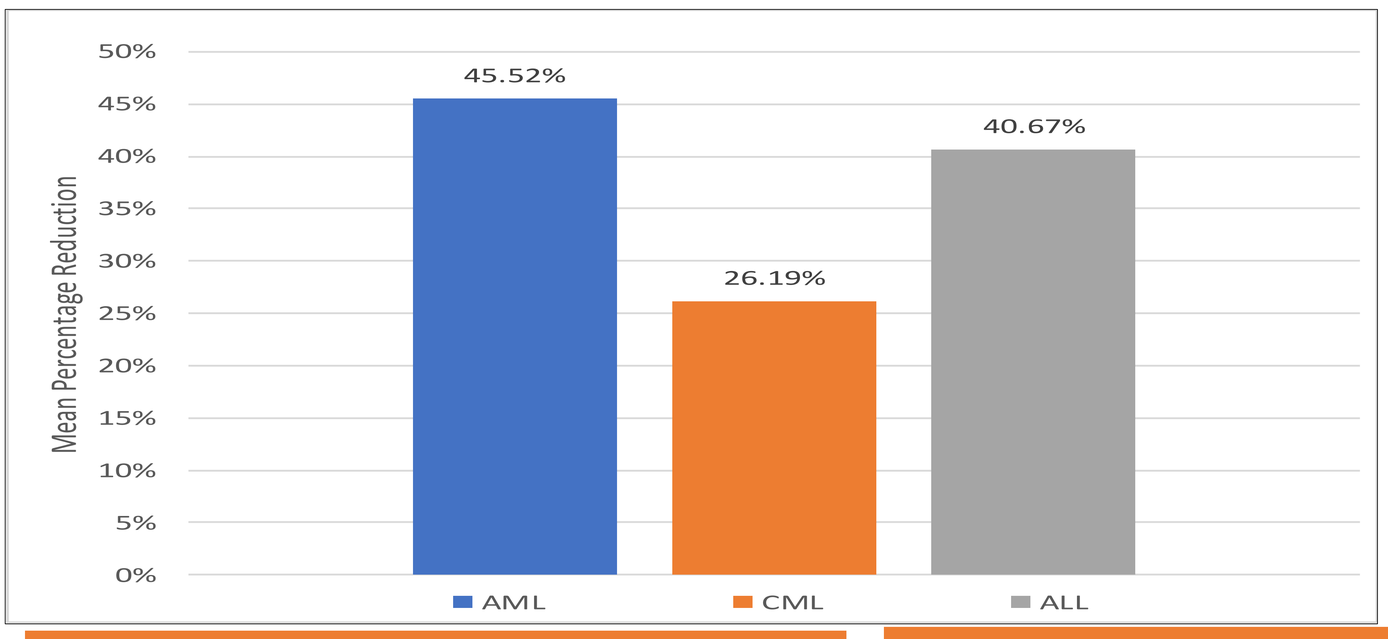
- Leukostasis is an oncological emergency defined by a white blood cell (WBC) count of ≥50-100 10^9/L in the presence of clinical signs and symptoms of decreased tissue perfusion and ischaemic tissue injury[1].
- Rapid depletion of the WBC count via leukapheresis is often the treatment of choice used to prevent a fatal course of the disease in high count leukaemia patients[2].
- Since 2009, St. George's Hospital, London has been using leukapheresis to treat select patients presenting with symptoms of leukostasis. This includes patients with acute myeloid leukaemia (AML), chronic myeloid leukaemia (CML) and acute lymphoblastic leukaemia (ALL).
- This retrospective study reviews the outcomes of this therapeutic approach at this tertiary centre

Patient Characteristics

Characteristic	n=24 n (%)
Age (years)	
Median	42
Range	18-76
Sex	
Male	14 (58)
Female	10 (42)
Diagnosis	
AML	12 (50)
CML	8 (33)
ALL	4 (17)
Number of apheresis procedures (per patient)	
Median	2
Range	1-6

- Data was collected by analysing patient notes from the apheresis database, electronic patient records and PowerChart.
- All adult patients since 2009 treated with leukopheresis were selected for this study.
- Patients with AML were the largest subgroup
- Most patients had one leukopheresis however the range was 1-6. Each procedure processed 1.5-2 blood volumes.

Reduction in WBC count (%)



- The efficacy of leukopheresis can be measured via percentage reduction to WBC count.
- The percentage reduction in WBC count, in all patients, across all diagnoses, was greater than 20%.
- Patients with AML demonstrated the greatest percentage reduction to WBC count. This was significantly different to percentage reduction in patients with CML ($p<0.05\%$).
- The CML group demonstrated the smallest mean percentage reduction in WBC count.

AML (n=12)

Median age 54 years (range 18-75)

Pre-leukopheresis WBC count (mean) 248 x10^9/l (range 122- 480 x10^9/l)

In patients with data available (n=7), 3 (43%) had secondary AML and 4 (57%) had high risk AML (high risk cytogenetics)

10 patients required ITU admission

All tolerated the leukapheresis procedure but 6 (50%) patients died in induction due to complications of advanced disease.

Complications	n
Type 1 respiratory failure (T1RF)	6
Tumour lysis	3
Neurological compromise	4
Acute Kidney injury (AKI)	4

CML (n=8)

Median age 39 years (range 27-76)

Pre-leukopheresis WBC count (mean) 411 x10^9/l (range 244-739 x10^9/l)

One patient required ITU due to multiple complications (AKI, T1RF, Disseminated intravascular coagulation) and died day 2. A second patient died 2 years later, post stem cell transplant of disease progression (blast crisis).

ALL (n=4)

Median age 22 years (range 18-67)

Pre-leukopheresis WBC count (mean) 280 x10^9/l (range 111-408 x10^9/l)

All achieved complete remission following induction chemotherapy, two died more than 1 year later post stem cell transplant (both had high risk disease)

Conclusions

1. These results confirm that in patients with leucostasis due to hyperleucocytosis, the leukapheresis procedure is effective in lowering the WBC count.
2. Leukapheresis provides only a temporary reduction in WBC with the aim of alleviating symptoms of leukostasis. This therapeutic approach needs to combined with cytoreductive treatment in all patients.
3. The efficiency of leukapheresis in CML patients is less compared to the AML patients. This is partly due to buffy coat layer in AML being well defined, in comparison to CML where the buffy coat layer contains both maturing WBCs and blasts and often extends into the red cell layer. In addition to this, CML patients often have a markedly enlarged spleen and during the leukapheresis procedure WBCs are released from the spleen into the peripheral blood.
4. Our results show that patients presenting with hyperleucocytosis and leukostasis have a poor prognosis. In many patients this poor outcome is inevitable due to high risk nature of their disease.

References

1 Porcu P. et al, Leuk Lymphoma 2000, vol.39 1-2 (pg 1-18)
2. De Santis GC et al, J Clin Apher, 2011, vol. 26 4(pg 181-185)

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ALL and AML
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