Blood Academy

Interactive Cases

First edition

Dr Ali Mahdi
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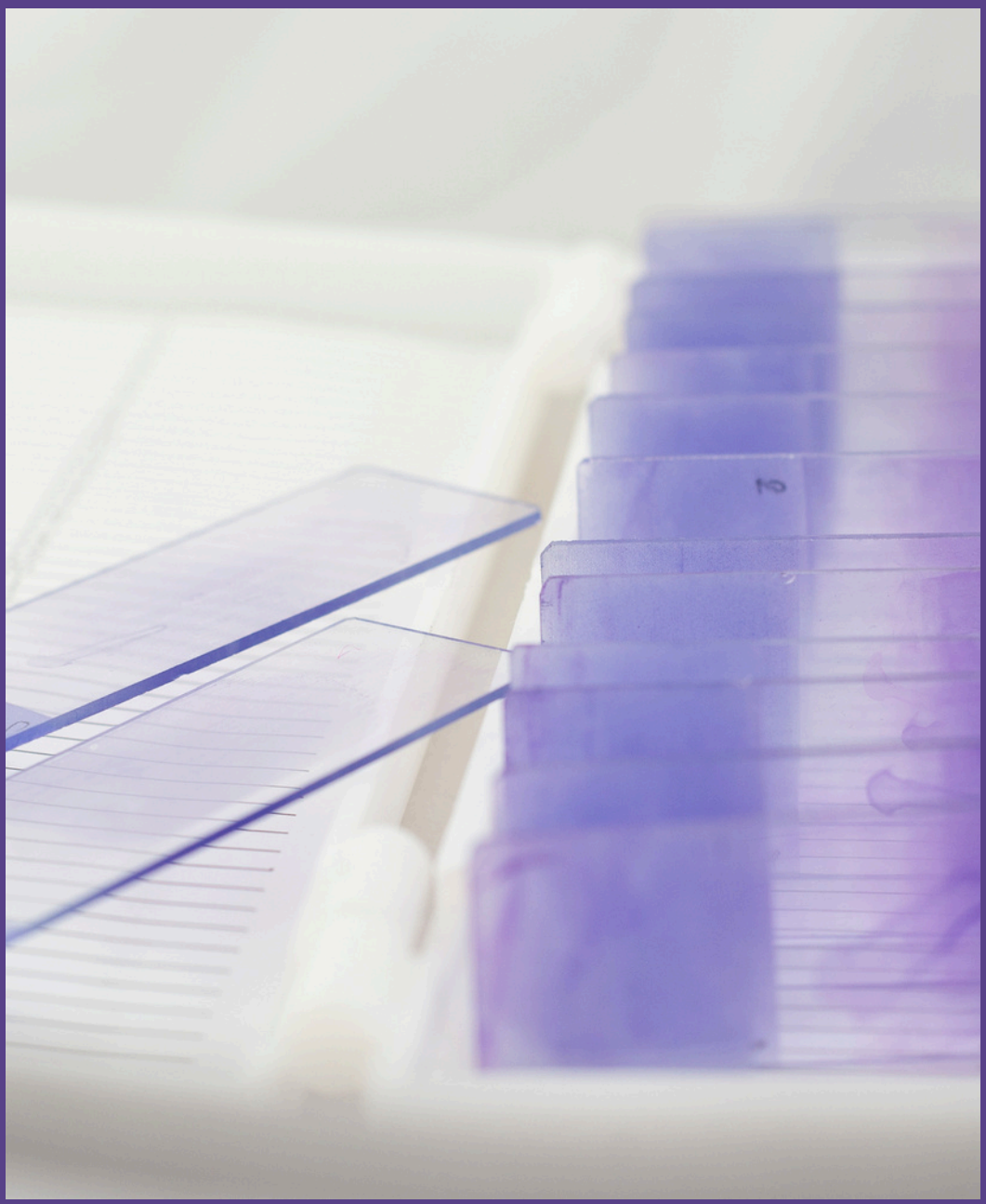

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First edition

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Acknowledgements

I wish to acknowledge the influence and inspiration from my previous mentors, who have shaped my professional journey and guided me in ways beyond measure. Their teachings, encouragement, and unwavering belief in my abilities continue to guide me and have been a driving force behind this book.

Furthermore, I must express my heartfelt appreciation for the unwavering support of my wife and children. The countless hours spent in front of my laptop were made bearable by their love, patience, and understanding. Their encouragement and presence became the comfort and inspiration that sustained me during the arduous process of writing. They have been the silent strength behind this book, and I thank them from the bottom of my heart for being my constant companions in this meaningful journey.

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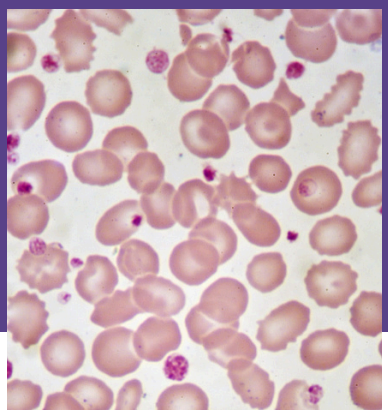

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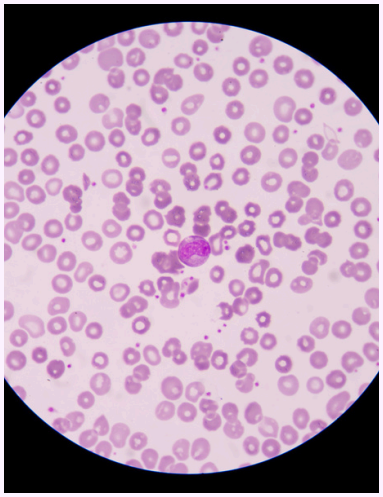
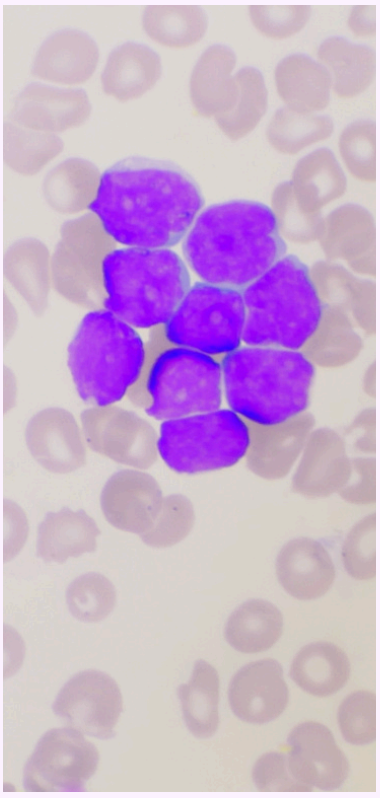
Preface



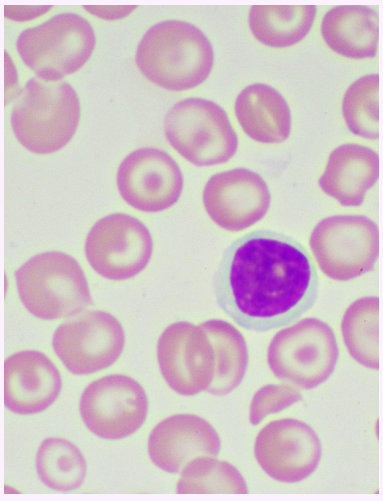
When I wrote the companion to this resource, *Essential Blood Cell Morphology*, my intention was to create a clear and accessible foundation for anyone beginning their journey into the microscopic world of blood films. Over the past two years, I have had the privilege of seeing learners use that book, and the accompanying course, to build confidence in recognising the normal and abnormal cell. Yet one message has repeatedly surfaced in conversations with students and colleagues alike: true mastery comes from practice.



That realisation was always at the heart of this project. From the moment the first book was completed, I knew it needed a companion, something that allowed learners not only to study morphology, but to apply it. Interactive Cases grew out of that vision. My hope is that this book provides the bridge between knowledge and recognition, turning theory into a skill that feels intuitive and familiar.

This collection of 45 cases, supported by digital slides, video walkthroughs, and guided questions, is designed to mirror the way we learn in real clinical practice: by observing, comparing, reflecting, and returning to the microscope again and again. Each case encourages you to pause, to look closely, and to trust your developing eye. Whether you are new to morphology or refreshing existing skills, I hope these cases give you the space to consolidate your understanding and challenge yourself in a supportive, structured way.

As always, I welcome your thoughts and suggestions. Please feel free to reach out to me at **ajm@blood-academy.com** with any feedback. Your input shapes future editions and helps ensure these resources continue to meet the needs of learners across haematology.



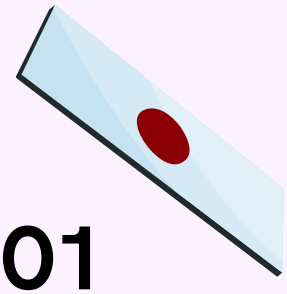
Thank you for allowing this book to be a part of your learning journey.

– Dr Ali Mahdi

Using this Book

This book has been designed to complement the Interactive Cases learning experience by guiding you through real digital blood films supported by clinical scenarios. Each chapter provides an opportunity to interpret morphology in a practical, case-based format. This book is intended to be used alongside the following online resources.

01 Digital Films



Each case begins with a short summary of the patient's presentation, laboratory findings, and relevant background information to frame your interpretation and mirrors how morphology is assessed in real practice.

SCAN ME

Scan the QR codes at the start of each chapter to access the corresponding digital blood film.

02 Video Tutorials

A detailed video tutorial accompanies every case. These tutorials highlight the important diagnostic features on the slide, explain how to interpret them, and outline the clinical considerations associated with the likely diagnosis.

Whether you are consolidating your foundations or refining more advanced skills, these videos offer a structured and supportive walkthrough of each case.

SCAN ME

Scan the QR codes to watch the tutorial for each case.

03 Answer Sheet

Use the answer sheet to record your findings, overall interpretation, suggested investigations, and key learning points for each case.

SCAN ME

Access the answer sheet via the QR code to the left.

Abbreviations

ADP	Adenosine diphosphate
ALAL	Acute leukaemias of ambiguous lineage
ALL	Acute lymphoblastic leukaemia
AMKL	Acute megakaryoblastic leukaemia
AML	Acute myeloid leukaemia
ANA	Antinuclear antibody
APC	Antigen-presenting cell
APML	Acute promyelocytic leukaemia
ATLL	Adult T-cell leukaemia/lymphoma
BFU	Burst-forming unit
BL	Burkitt lymphoma
BPCDN	Blastic plasmacytoid dendritic cell neoplasm
B-ALL	B-cell acute lymphoblastic leukaemia
Ca	Calcium
CD	Cluster of differentiation
CDA	Congenital dyserythropoietic anaemia
CEL	Chronic eosinophilic leukaemia
cfDNA	Cell-free DNA
CFU	Colony-forming unit
CHIP	Clonal haematopoiesis of indeterminate potential
CLL	Chronic lymphocytic leukaemia
CML	Chronic myeloid leukaemia
CMML	Chronic myelomonocytic leukaemia
CNL	Chronic neutrophilic leukaemia
cnLOH	Copy-neutral loss of heterozygosity
CODXM-IVAC	Cyclophosphamide, Vincristine, Doxorubicin, Dexamethasone, Methotrexate, Ifosfamide, Etoposide Cytarabine
CT	Computed tomography
DAT	Direct antiglobulin test
DA-EPOCH	Dose-adjusted Etoposide Prednisone Vincristine Cyclophosphamide Doxorubicin
DIC	Disseminated intravascular coagulation
DLBCL	Diffuse large B-cell lymphoma
DNA	Deoxyribonucleic acid
ELTS	EUTOS long-term survival score
EMA	Eosin-5-maleimide binding test
EPB42	Erythrocyte membrane protein band 4.2
ET	Essential thrombocythaemia
EUTOS	European Treatment and Outcome Study
FGFR1	Fibroblast growth factor receptor 1
FISH	Fluorescence in situ hybridisation
FLT3-ITD	*FLT3* internal tandem duplication
FL	Follicular lymphoma
G-CSF	Granulocyte-colony stimulating factor
GEMM	Granulocyte Erythroid Macrophage Megakaryocyte
GM	Granulocyte Macrophage
Hb	Haemoglobin
HbA	Adult haemoglobin
HbA2	Minor adult haemoglobin
HCL	Hairy cell leukaemia
HDFN	Haemolytic disease of the fetus and newborn
HELLP	Haemolysis, Elevated liver enzymes, Low platelets
HES	Hypereosinophilic syndrome
HIV	Human immunodeficiency virus
HL	Hodgkin lymphoma
HLA-DR	Human leukocyte antigen – DR isotype
HPFH	Hereditary persistence of foetal haemoglobin
HS	Hereditary spherocytosis
HSCT	Haematopoietic stem cell transplantation
HPLC	High-performance liquid chromatography
HUS	Haemolytic uraemic syndrome
ICC	International Consensus Classification
ICSH	International Council for Standardization in Haematology
IL	Interleukin
JMML	Juvenile myelomonocytic leukaemia
KIR	Killer immunoglobulin-like receptor
LCAT	Lecithin–cholesterol acyltransferase

Abbreviations

LDH	Lactate dehydrogenase
LGL	Large granular lymphocyte
LPL	Lymphoplasmacytic lymphoma
MCH	Mean cell haemoglobin
MCHC	Mean cell haemoglobin concentration
MCL	Mantle cell lymphoma
MCV	Mean cell volume
MDS	Myelodysplastic syndrome (neoplasm)
MF	Myelofibrosis
Mg	Magnesium
MHC	Major histocompatibility complex
MRI	Magnetic resonance imaging
MPN	Myeloproliferative neoplasm
MPV	Mean platelet volume
MYH9	Non-muscle myosin heavy chain IIA
N:C	Nuclear:cytoplasmic ratio
NGS	Next-generation sequencing
NHL	Non-Hodgkin lymphoma
NK	Natural killer
NOS	Not otherwise specified
NPM1	Nucleophosmin 1
PCL	Plasma cell leukaemia
PCR	Polymerase chain reaction
PDGF	Platelet-derived growth factor
PET	Positron emission tomography
Ph	Philadelphia chromosome
PNH	Paroxysmal nocturnal haemoglobinuria
PRIMA-PI	PRIMA Prognostic Index
PTP	Post-transfusion purpura
PV	Polycythaemia vera
RA	Rheumatoid arthritis
RDW	Red cell distribution width
RNA	Ribonucleic acid
R-CHOP	Rituximab, Cyclophosphamide, Doxorubicin, Vincristine, Prednisolone
RS	Ring sideroblasts
SLE	Systemic lupus erythematosus
SLVL	Splenic lymphoma with villous lymphocytes
SS	Sézary syndrome
STEC	Shiga toxin-producing Escherichia coli
TAM	Transient abnormal myelopoiesis
TL-DS	Transient leukaemia of Down syndrome
T-PLL	T-cell prolymphocytic leukaemia
TPO	Thrombopoietin
t-MDS	Therapy-related myelodysplastic syndrome (neoplasm)
TTP	Thrombotic thrombocytopenic purpura
UTI	Urinary tract infection
VEGF	Vascular endothelial growth factor
VWF	von Willebrand factor
WAS	Wiskott-Aldrich syndrome
WHO	World Health Organization

Case 1

9-year-old male
Fatigue

Haemoglobin 106 g/L
White cell count 18.1 x 10^9/L
Platelet count 111 x 10^9/L

BLOOD FILM

VIDEO

Morphology

At low power magnification, a clear leucocytosis is evident, dominated by abnormal mononuclear cells. On higher power examination, these abnormal cells show features consistent with blast cells. These cells are pleomorphic in size and shape, ranging from small forms (only slightly larger than a mature red cell) to medium and large cells. The larger cells have more abundant grey-blue cytoplasm and typically have compact chromatin. Some cells have cytoplasmic granules. Smear cells are also present.

In the background, several additional features are noted:

- Red cells show polychromasia, with the occasional late nucleated red cell precursor present.
- Platelets are mildly reduced, reflecting the automated platelet count.
- Mature neutrophils are present but reduced. This is an important finding imparting a risk of significant infection.

The next diagnostic step is to determine whether the blasts are of myeloid or lymphoid origin. This distinction can be challenging on morphology alone, particularly in the absence of Auer rods.

In this case, the relatively small blast size, agranular cytoplasm and condensed chromatin pattern suggests a lymphoid origin to the diagnosis. When considered alongside the patient's age, these features strongly suggest acute lymphoblastic leukaemia (ALL), although morphology alone is insufficient. Definitive classification of blasts requires flow cytometry, which remains the gold standard for lineage determination in acute leukaemia.

Acute Lymphoblastic Leukaemia

ALL is the most common malignancy of childhood. In contrast to adults, where acute myeloid leukaemia (AML) predominates, ALL accounts for the majority of acute leukaemias in the paediatric population.

Immunophenotyping

Accurate lineage assignment in ALL requires flow cytometry or immunohistochemistry, which identify characteristic lymphoid antigens:

- **B-cell markers:** CD19, CD20, CD22, CD79a, PAX5
- **T-cell markers:** CD1a, CD3, CD4, CD5, CD7, CD8
- **Natural killer (NK) cell marker:** CD56
- **Markers of immaturity:** CD10, terminal deoxynucleotidyl transferase (TdT)

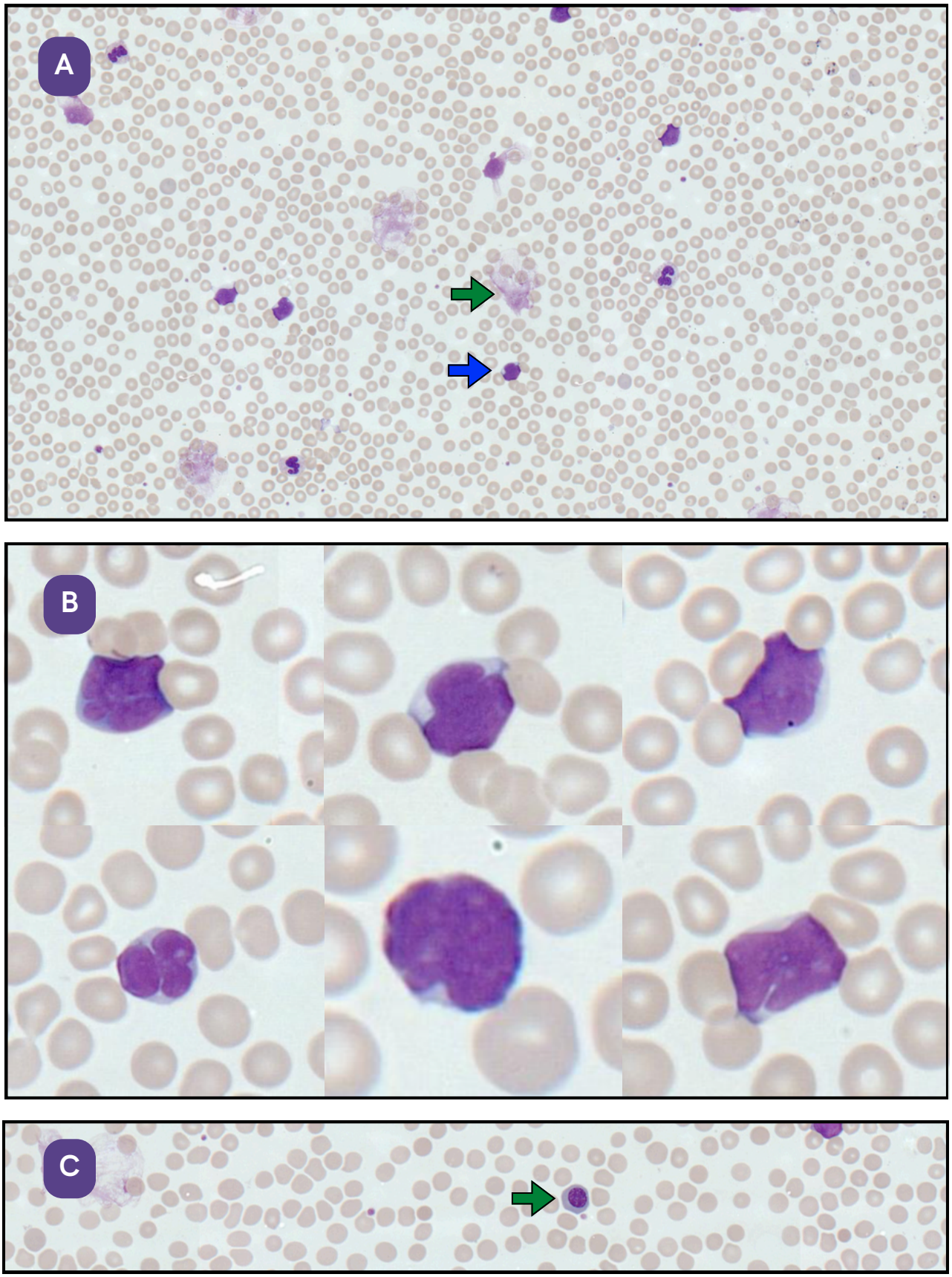


Figure 1.1 - Morphological Features of Case 1
A. Low-power view: smear cell (green arrow) and a blast cell (blue arrow), B. Examples of lymphoid blast morphology, C. Nucleated red cell (green arrow)

Classification

The current World Health Organization (WHO) (1) and International Consensus Classification (ICC) (2) classifications define ALL primarily by the presence of recurrent genetic abnormalities (see Table 1.1). These include specific chromosomal rearrangements or gene fusions that direct both prognosis and management. If no defining rearrangement is identified, the disease is designated ALL, not otherwise specified (NOS). T-ALL has fewer recognised genetic rearrangements compared with B-ALL.

Clinical Presentation

The clinical presentation of ALL is highly variable, reflecting both marrow failure and extramedullary involvement. Of the most important clinical features are:

- Lymphadenopathy.
- Haematological manifestations include:
  - Bleeding due to thrombocytopenia
  - Anaemia-related symptoms such as fatigue, pallor, or in children, poor feeding.
  - White cell count at diagnosis is variable. Leucocytosis is more frequently seen in T-ALL, often associated with a thymic (mediastinal) mass.
- Central nervous system (CNS) involvement may cause headaches, vomiting, or seizures. For this reason, cerebrospinal fluid (CSF) morphological and flow cytometry examination is needed, along with prophylactic intrathecal chemotherapy administration even if no CNS disease is detected.

Outcomes with Modern Therapy

The prognosis of paediatric ALL has improved dramatically over recent decades, reflecting advances in our understanding of disease biology, the development of risk-adapted and targeted therapies, and major improvements in supportive care (3). With contemporary regimens, around 98% of patients achieve complete remission. The five-year overall survival rate is now close to 90%. Mortality during induction therapy remains very low at approximately 1%, and long-term treatment-related mortality is less than 3% over a 10-year period. These outcomes represent an important source of reassurance for families at the time of diagnosis.

Case Outcome

The patient was treated with a paediatric ALL protocol, incorporating asparaginase, intrathecal methotrexate, vincristine, and dexamethasone. He achieved complete remission after one month of therapy and has remained in remission after four years of follow-up.

Final Diagnosis
B-lymphoblastic leukaemia/lymphoma

Key Learning Points

- Recognition of blasts on a blood film is a critical feature and should raise the alarm for acute leukaemia and prompt further investigation.
- Auer rods are the only reliable morphological marker of myeloid lineage; otherwise, lineage requires further investigation with flow cytometry, immunohistochemistry and genetic investigations.
- In children, smaller blasts with agranular cytoplasm and condensed chromatin suggest lymphoid origin.
- ALL is the most common childhood malignancy, with excellent outcomes on modern protocols.

B-lymphoblastic leukaemias/lymphomas with recurrent genetic abnormalities
• B-lymphoblastic leukaemia/lymphoma with high hyperdiploidy • B-lymphoblastic leukaemia/lymphoma with hypodiploidy • B-lymphoblastic leukaemia/lymphoma with *iAMP21* • B-lymphoblastic leukaemia/lymphoma with *BCR ABL1* fusion • B-lymphoblastic leukaemia/lymphoma with *BCR ABL1*-like features • B lymphoblastic leukaemia/lymphoma with *KMT2A* rearrangement • B lymphoblastic leukaemia/lymphoma with *ETV6 RUNX1* fusion • B-lymphoblastic leukaemia/lymphoma with *ETV6 RUNX1*-like features • B lymphoblastic leukaemia/lymphoma with *TCF3 PBX1* fusion • B lymphoblastic leukaemia/lymphoma with *IGH IL3* fusion • B lymphoblastic leukaemia/lymphoma with *TCF3 HLF* fusion • B-lymphoblastic leukaemia/lymphoma with other defined genetic alterations
B-lymphoblastic leukaemia/lymphoma, NOS
T-lymphoblastic leukaemia /lymphoma
• T-lymphoblastic leukaemia / lymphoma, NOS • Early T-precursor lymphoblastic leukaemia / lymphoma
Aggressive Natural killer-cell leukaemia

Table 1.1 - summary of the World Health Organization Classification of acute lymphoblastic leukaemia/lymphoma

ALL-acute lymphoblastic leukaemia/lymphoma, NOS-not otherwise specified

B-lymphoblastic leukaemias/lymphomas with recurrent genetic abnormalities	
• B-ALLwith t(9;22)(q34.1;q11.2)*/BCR::ABL1* ◦ with lymphoid only involvement and multilineage involvement • B-ALLwith t(v;11q23.3)/*KMT2A* rearranged • B-ALL with t(12;21)(p13.2;q22.1)/*ETV6::RUNX1* • B-ALL, hyperdiploid • B-ALL, low hypodiploid • B-ALL, near haploid • B-ALL with t(5;14)(q31.1;q32.3)/*IL3::IGH* • B-ALL with t(1;19)(q23.3;p13.3)/*TCF3 PBX1* • B-ALL, *BCR::ABL1*-like, ABL-1 class rearranged • B-ALL, *BCR::ABL1*-like, JAK-STAT activated • B-ALL, *BCR::ABL1*-like, NOS • B-ALL with *iAMP21* • B-ALL with *MYC* rearrangement	• B-ALL with *DUX4* rearrangement • B-ALL with *MEF2D* rearrangement • B-ALL with *ZNF384(362)* rearrangement • B-ALL with *NUTM1* rearrangement • B-ALL with *HLF* rearrangement • B-ALL with *UBTF ATXN7L3/PAN3,CDX2 ("CDX2/UBTF")* • B-ALL with mutated *IKZF1 N159Y* • B-ALL with mutated *PAX5 P80R* • Provisional entity B-ALL, *ETV6 RUNX1*-like • Provisional entity B-ALL, with *PAX5* alteration • Provisional entity B-ALL, with mutated *ZEB2* (p.H1038R)/*IGH CEBPE* • Provisional entity B-ALL, *ZNF384* rearranged-like • Provisional entity B-ALL, *KMT2A* rearranged-like
B-lymphoblastic leukaemia/lymphoma, NOS	
T-lymphoblastic leukaemia /lymphoma	
• T-lymphoblastic leukaemia /lymphoma, NOS • Early T-cell precursor ALL with *BCL11B* rearrangement	
Natural killer cell ALL	

Table 1.2 - summary of the International Consensus Classification of acute lymphoblastic leukaemia/lymphoma

ALL-acute lymphoblastic leukaemia/lymphoma, NOS-not otherwise specified

Case 2

68-year-old male
Routine hypertensive screen

Haemoglobin 131 g/L
White cell count 14.6 x 10^9/L
Platelet count 311 x 10^9/L

BLOOD FILM

VIDEO

Morphology

At low magnification, there is a clear leucocytosis. The film shows a mixture of normal and abnormal cells. Amongst the normal elements are neutrophils, monocytes, and occasional eosinophils. A moderate lymphocytosis is also apparent. These lymphocytes are identified by their small to medium size, scant cytoplasm, and a condensed chromatin pattern within the nucleus. Occasional cells show nuclear indentation, while others display slight variation in cytoplasmic volume, sometimes with coarse cytoplasmic granules. Although some lymphocytes have an irregular nuclear outline, this is not a predominant feature. Rare examples of lymphocytes with plasmacytoid morphology are present, characterised by an eccentric nucleus, deeply basophilic cytoplasm, and a perinuclear hof (perinuclear clearing).

Overall, the lymphocytes are relatively uniform in appearance or monomorphic. Smear (smudge) cells are also present. These arise due to the mechanical fragility of lymphocytes during slide preparation. There is mild platelet clumping, with red cells largely normal in size and shape. The findings are strongly suggestive of a chronic lymphoid process although morphology alone is insufficient for making a definitive diagnosis, and additional immunophenotypic and genetic studies are required.

Chronic Lymphoid Leukaemias to Consider

A monomorphic, mature lymphocytosis, as demonstrated in this case, raises three principal diagnostic possibilities:

- Chronic lymphocytic leukaemia (CLL)
- Mantle cell lymphoma (MCL)
- Splenic marginal zone lymphoma (SMZL)

Although smear (smudge) cells are classically associated with CLL, they are not diagnostic and may be encountered in other conditions. Other chronic lymphoid leukaemias usually have more distinctive morphology. For instance, hairy cell leukaemia (HCL) typically shows lymphocytes with abundant pale cytoplasm and fine cytoplasmic projections, features not present in this case. Accompanying monocytopenia and leucoerythroblastic features are also commonly seen in HCL.

When faced with a mature lymphocytosis, particularly in older patients, several key steps should guide assessment:

- **Review previous blood counts:** a persistent lymphocytosis, especially in older patients, often signals a chronic lymphoid malignancy.
- **Consider the clinical history**: features such as painless lymphadenopathy,

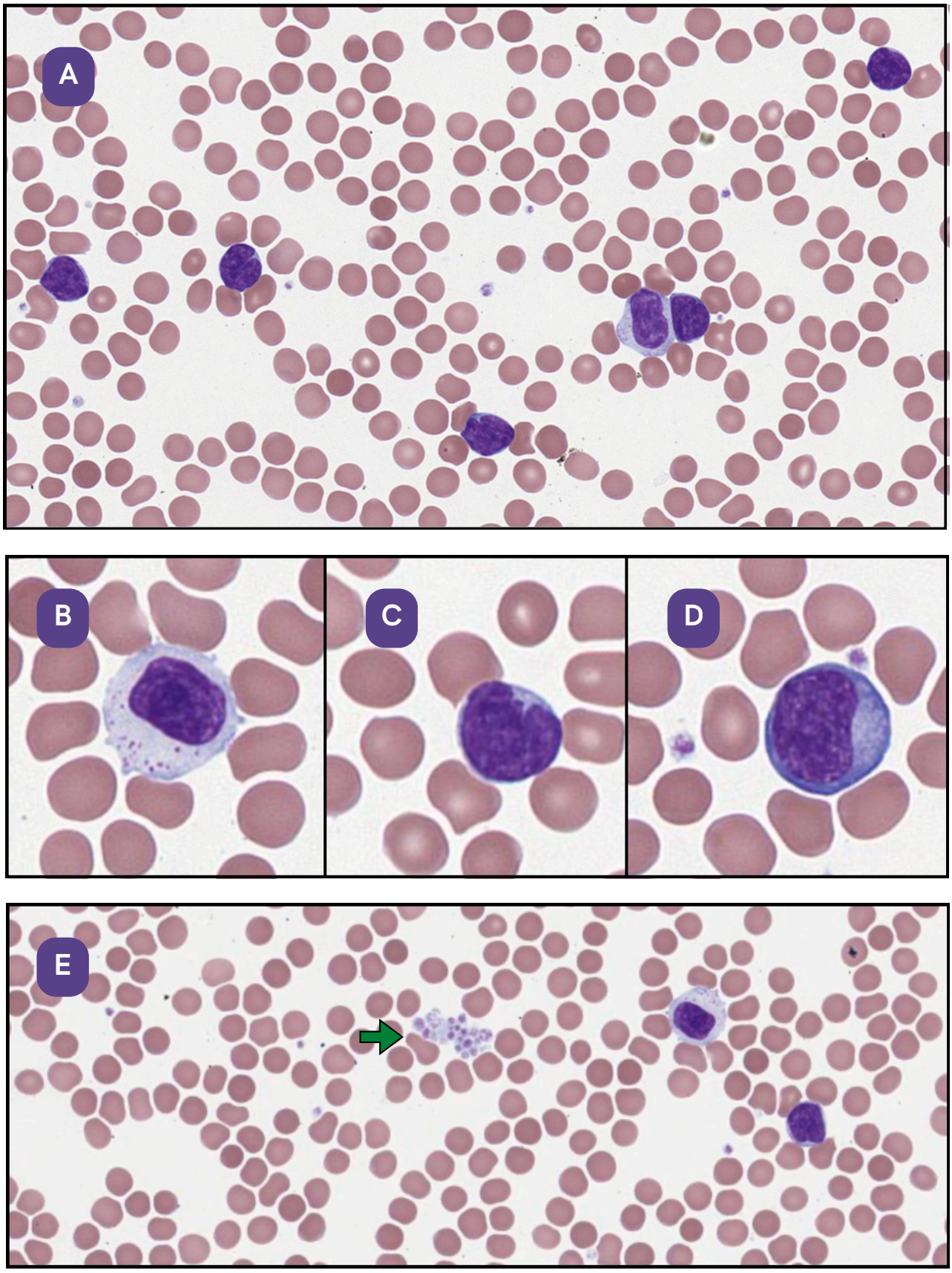


Figure 2.1 - Morphological Features of Case 2
A. Medium-sized lymphoid cells, B. Larger lymphoid cell with cytoplasmic granules, C. lymphoid cell with indented nucleus, D. Plasmacytoid cell, E. Platelet clump (green arrow)

splenomegaly, or systemic B-symptoms (weight loss, night sweats) raise suspicion for a chronic lymphoid disorder.

- **Flow cytometry:** this is the critical investigation to establish lineage, clonality and immunophenotype.
- **Additional investigations:** Fluorescence in situ hybridisation (FISH) for recurrent genetic abnormalities, computed tomography (CT) or positron emission tomography (PET) imaging, and, when required, lymph node biopsy provide further diagnostic clarity.

Flow Cytometry and Cytogenetics

Flow cytometry is the starting point to distinguish between common chronic lymphoid neoplasms. The typical immunophenotypic features are summarised below in Table 2.1.

In this patient, flow cytometry revealed the lymphocytes to be:

- CD5 positive, CD23 negative
- Bright CD20 and CD79b expression
- Bright surface immunoglobulin
- CD200 negative

Mantle Cell Lymphoma

In this case, the mature lymphocytosis, favourable clinical state, and preserved haemoglobin and platelet counts pointed to an indolent subtype of MCL, non-nodal variant (nnMCL). It is characterised by:

- A mostly asymptomatic presentation
- Involvement of blood, bone marrow, and spleen, with little or no lymphadenopathy

Marker	Chronic lymphocytic leukaemia	Mantle Cell Lymphoma	Marginal Zone Lymphoma
CD5	Positive	Positive	Negative
CD23	Positive	Negative (usually)	Negative
FMC7	Negative / dim	Positive	Variable
CD20	Dim (weaker expression)	Bright	Bright
CD79b	Dim / weak	Bright	Bright
Surface Ig	Dim	Bright	Bright
Cyclin D1	Negative	Positive (key marker)	Negative
SOX11	Negative	Usually positive (esp. aggressive disease)	Negative
CD200	Positive	Negative	Negative
CD10	Negative	Negative	Negative

Table 2.1 - Immunophenotypic Profiles of Chronic Lymphocytic Leukaemia, Mantle Cell Lymphoma, and Marginal Zone Lymphoma

- Distinct biological features compared with classic MCL:
  - Lack of SOX11 expression
  - Low Ki-67 proliferative indices
  - Frequent lack of CD5 expression
  - A distinct *IGHV* gene repertoire, often dominated by *IGHV1-8* usage and higher levels of somatic hypermutation (4)
  - Fewer genetic alterations and less genomic complexity, although TP53 aberrations occur at a similar frequency to classic MCL (5)

In contrast, aggressive forms of MCL exist, in which the lymphoma cells acquire a blast-like morphology. These cases can mimic acute lymphoblastic leukaemia (ALL) or even acute monoblastic leukaemia, underlining the importance of accurate immunophenotypic and genetic testing.

Case Outcome

The patient had no significant lymphadenopathy or splenomegaly and was clinically well at the time of assessment. Importantly, his blood count has remained stable after two years of follow-up.

Final Diagnosis
Non-Nodal Mantle Cell Lymphoma

Key Learning Points

- A monomorphic mature lymphocytosis raises suspicion for a chronic lymphoid malignancy.
- Main differentials: CLL, mantle cell lymphoma (MCL), splenic marginal zone lymphoma (SMZL).
- Smear cells are common in CLL but are not diagnostic.
- Flow cytometry and FISH are essential to confirm lineage and genetic abnormalities.
- nnMCL presents with blood, marrow, and spleen involvement, usually without lymphadenopathy, and can follow an indolent course.
- Some patients can be safely managed with observation if counts and clinical status remain stable.