Corrigenda to first print run of WHO Classification of Tumours of Haematopoietic and Lymphoid Tissues, revised 4th edition

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In addition to corrections of minor typographical errors, corrections were made in this print run to improve the text. The chromosomal localization of genes was improved, and Human Genome Variation Society (HGVS) notation was used throughout for translocations, insertions, and other gene alterations. Gene symbols are given in italics as is common usage.

The following acknowledgement of funding bodies was added to page 5:

This volume was produced with support from the following organizations:
American Society of Hematology
Fondazione Italiana Linfomi ONLUS
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University of Chicago Medicine Comprehensive Cancer Center
Leukemia Clinical Research Foundation

Summary of corrections:

Chapter 2: Myeloproliferative neoplasms **p. 54**
Chronic eosinophilic leukaemia, NOS > Table 2.15

1. A referral to p. 57 has been added to the table title, to refer the reader to the *Myeloproliferative neoplasm, unclassifiable* section, to which this table relates.
2. The word "either" has been removed as shown below. All criteria should be met.

Original Text	Corrected Text
Table 2.15 Diagnostic criteria for myeloproliferative neoplasm (MPN), unclassifiable	**Table 2.15** Diagnostic criteria for myeloproliferative neoplasm (MPN), unclassifiable (see p. 57)
The diagnosis of myeloproliferative neoplasm (MPN), unclassifiable, requires that either all 3 criteria are met.	The diagnosis of myeloproliferative neoplasm (MPN), unclassifiable, requires that all 3 criteria are met.

Chapter 6: Myelodysplastic syndromes **p. 108, column 3, ¶ 5**
Myelodysplastic syndrome with single lineage dysplasia > Genetic profile

The spelling of the word "cohesin" has been corrected.

Original Text	Corrected Text
Somatic driver mutations have been identified in 60–70% of cases of MDS-SLD. The underlying mutations affect a haematopoietic stem cell and are present in all lineages despite the limitation of dysplastic findings to one lineage {4354}. *TET2* and *ASXL1* appear to be the most commonly mutated genes in MDS-SLD {1513}. However, mutations in other DNA methylation genes, splicing factors, RAS pathway genes, cohesion complex genes and *RUNX1* are …	Somatic driver mutations have been identified in 60–70% of cases of MDS-SLD. The underlying mutations affect a haematopoietic stem cell and are present in all lineages despite the limitation of dysplastic findings to one lineage {4354}. *TET2* and *ASXL1* appear to be the most commonly mutated genes in MDS-SLD {1513}. However, mutations in other DNA methylation genes, splicing factors, RAS pathway genes, cohesin complex genes and *RUNX1* are …

Chapter 6: Myelodysplastic syndromes **p. 113, column 1, ¶ 1**
Myelodysplastic syndrome with multilineage dysplasia > Genetic profile

The spelling of the word "cohesin" has been corrected.

Original Text	Corrected Text
monosomy 5, del(5q) and del(20q), as well as complex karyotypes, are found in as many as 50% of patients with MDS-MLD {2423}. Whole-genome sequencing has shown that more than half of all cases of MDS-MLD carry mutations in genes that are also mutated in MDS with excess blasts and acute myeloid leukaemia. These include genes from the cohesion family (*STAG2*), chromatin modifiers …	del(5q) or t(5q), and del(20q), as well as complex karyotypes, are found in as many as 50% of patients with MDS-MLD {2423}. Whole-genome sequencing has shown that more than half of all cases of MDS-MLD carry mutations in genes that are also mutated in MDS with excess blasts and acute myeloid leukaemia. These include genes from the cohesin family (*STAG2*), chromatin modifiers …

Chapter 8: Acute myeloid leukaemia and related precursor neoplasms **p. 142, column 2, ¶ 1**
Acute myeloid leukaemia with mutated NPM1 > Genetic profile

The spelling of the term "cohesin complex" has been corrected.

Original Text	Corrected Text
and most frequently involve *FLT3* and *DNMT3A*, but mutations of *IDH1*, *KRAS*, *NRAS*, and cohesion-complex genes are also relatively common {545, 1149}. Although…	and most frequently involve *FLT3* and *DNMT3A*, but mutations of *IDH1*, *KRAS*, *NRAS*, and cohesin complex genes are also relatively common {545, 1149}. Although…

Chapter 12: Precursor lymphoid neoplasms **p. 210, column 2, ¶ 3**
T-lymphoblastic leukaemia/lymphoma > Microscopy

The word "hyperplasia" was changed to "neoplasia".

Original Text	Corrected Text
Cases with histological findings of T-LBL with a significant infiltrate of eosinophils among the lymphoma cells may be associated with eosinophilia, myeloid hyperplasia, and an 8p11.2 cytogenetic …	Cases with histological findings of T-LBL with a significant infiltrate of eosinophils among the lymphoma cells may be associated with eosinophilia, myeloid neoplasia, and an 8p11.2 cytogenetic …

Chapter 13: Mature B-cell neoplasms **p. 231, column 2, ¶ 2**
Hairy cell leukaemia variant > Microscopy

The word “atretic” was changed to “regressed”.

Original Text	Corrected Text
Like in HCL and splenic diffuse red pulp small B-cell lymphoma, the red pulp of the spleen is diffusely involved and expanded in HCL-v, with atretic or absent …	Like in HCL and splenic diffuse red pulp small B-cell lymphoma, the red pulp of the spleen is diffusely involved and expanded in HCL-v, with regressed or absent…

Chapter 13: Mature B-cell neoplasms **p. 236, column 1, ¶ 3**
IgM monoclonal gammopathy of undetermined significance > Localization

The diagnostic criterion was changed as shown below.

Original Text	Corrected Text
There may be as much as 10% bone marrow infiltration by an IgM+ clonal lymphoplasmacytic population. Cases with …	There may be bone marrow infiltration by an IgM+ clonal lymphoplasmacytic population, but it must be < 10%. Cases with…

Chapter 13: Mature B-cell neoplasms **p. 241**
Plasma cell neoplasms > Table 13.04

Table 13.04 has been reformatted as shown below.

Table 13.04 Plasma cell neoplasms

Non-IgM (plasma cell) monoclonal gammopathy of undetermined significance (precursor lesion)
Plasma cell myeloma **Clinical variants:** Smouldering (asymptomatic) plasma cell myeloma Non-secretory myeloma Plasma cell Leukemia
Plasmacytoma Solitary plasmacytoma of bone Extraosseous (extramedullary) plasmacytoma
Monoclonal immunoglobulin deposition diseases Primary amyloidosis Systemic light and heavy chain deposition diseases
Plasma cell neoplasms with associated paraneoplastic syndrome POEMS syndrome TEMPI syndrome (provisional)

Chapter 13: Mature B-cell neoplasms **p. 242**
Non-IgM monoclonal gammopathy of undetermined significance > Table 13.05

One of the diagnostic criteria for light-chain monoclonal gammopathy of undetermined significance (MGUS) was changed as shown below.

Original Text	Corrected Text
Abnormal free light chain ratio (< 0.26 or > 1.65) Increased level of the involved free light chain No immunoglobulin heavy chain expression on immunofixation electrophoresis Urinary M protein < 500 mg/24 hours Clonal plasma cells < 10% Absence of end-organ damage (CRAB) and amyloidosis	Abnormal free light chain ratio (< 0.26 or > 1.65) Increased level of the involved free light chain No abnormal immunoglobulin heavy chain expression on immunofixation electrophoresis Urinary M protein < 500 mg/24 hours Clonal plasma cells < 10% Absence of end-organ damage (CRAB) and amyloidosis

Chapter 13: Mature B-cell neoplasms **p. 246, column 3, ¶ 3**
Plasma cell myeloma > Postulated normal counterpart

A sentence has been deleted as shown below.

Original Text	Corrected Text
The postulated normal counterparts are post–germinal centre long-lived plasma cells in which the IG genes have undergone class switch and somatic hypermutation. The cell of origin has not been established.	The postulated normal counterparts are post–germinal centre long-lived plasma cells in which the IG genes have undergone class switch and somatic hypermutation.

Chapter 13: Mature B-cell neoplasms **p. 247**
Plasma cell myeloma > Table 13.09

Table 13.09 has been reformatted as shown below.

Table 13.09 The International Myeloma Working Group (IMWG) consensus recommendations on genetic testing. Adapted from Fonseca R, et al. {1232}

FISH (on cell-sorted samples or cytoplasmic immunoglobulin FISH)
Minimal panel: t(4;14)(p16;q32), t(14;16)(q32;q23), del(17p13.1)
More comprehensive panel: t(11;14)(q13;q32), del 13, ploidy category, chromosome 1 abnormalities
Clinical trials should incorporate gene expression profiling

Chapter 14: Mature T- and NK-cell neoplasms **p. 386, column 2, ¶ 2**
Mycosis fungoides > Staging

The page number for Table 14.06 and a referral to Table 14.07 have been added.

Original Text	Corrected Text
See Table 14.06.	See Table 14.06 (p. 387) and Table 14.07 (p. 390).

Chapter 14: Mature T- and NK-cell neoplasms **p. 419**
Anaplastic large cell lymphoma, ALK-negative > Fig. 14.171

The caption for panel C of Fig. 14.171 has been modified as shown below.

Original Text	Corrected Text
C Typical morphological and phenotypic features of ALCL, including a hallmark cell (arrow), that arose in the gastrointestinal tract (glandular epithelium is visible in the upper left).	**C** Typical morphological and phenotypic features of ALCL, including a hallmark cell (arrow), that arose in the gastrointestinal tract.

Chapter 15: Hodgkin lymphomas **p. 428**
Introduction > Fig. 15.08

The caption for panel A of Fig. 15.08 has been modified as shown below.

Original Text	Corrected Text
A Radioactive in situ hybridization for Igμ mRNA is negative in the Hodgkin/Reed–Sternberg cells (arrows), whereas the two non-neoplastic plasma cells in the upper edges are strongly positive, and the non-neoplastic bystander small B cells are moderately strongly positive.	**A** Radioactive in situ hybridization for Igμ mRNA is negative in the Hodgkin/Reed–Sternberg cells (arrows), and the non-neoplastic bystander small B cells are moderately strongly positive.

Chapter 15: Hodgkin lymphomas **p. 428**
Introduction > Fig. 15.09

The word “non-radioactive” has been removed from the caption for panel B of Fig. 15.09.

Original Text	Corrected Text
B EBV-infected Hodgkin/Reed–Sternberg cells consistently show a strong expression of EBV-encoded small RNA (EBER) in their nuclei as revealed by non-radioactive in situ hybridization.	**B** EBV-infected Hodgkin/Reed–Sternberg cells consistently show a strong expression of EBV-encoded small RNA (EBER) in their nuclei as revealed by in situ hybridization.

Chapter 16 Immunodeficiency-associated lymphoproliferative disorders — p. 459, column 2, ¶ 3

Monomorphic B-cell PTLD > Clinical features

A cited PMID has been replaced with the corresponding reference number (2832A), and the reference (as follows) has been added to the Reference list at the back of the book (on p. 549):

> **2832A.** Nassif S, Ozdemirli M (2013). EBV-positive low-grade marginal zone lymphoma in the breast with massive amyloid deposition arising in a heart transplant patient: a report of an unusual case. Pediatr Transplant. 17:E141–5. PMID:23773403

Original Text	Corrected Text
The clinical presentation of monomorphic B-PTLDs is not distinctive and is, in general, similar to the presentation of the lymphomas or plasma cell neoplasms that they resemble. The EBV+ MALT lymphoma M-PTLDs are distinctive, with a frequently cutaneous/subcutaneous presentation. They occur late after transplantation and are solitary, and the patients do well {23773403}.	The clinical presentation of monomorphic B-PTLDs is not distinctive and is, in general, similar to the presentation of the lymphomas or plasma cell neoplasms that they resemble. The EBV+ MALT lymphoma M-PTLDs are distinctive, with a frequently cutaneous/subcutaneous presentation. They occur late after transplantation and are solitary, and the patients do well {2832A}.

List of abbreviations — p. 585

The following entries have been added to the list of abbreviations at the back of the book:

HAART	highly active antiretroviral therapy
IG gene	immunoglobulin gene
KSHV	Kaposi sarcoma–associated herpesvirus – an alternative name for human herpesvirus 8 (HHV8)
LMP1	latent membrane protein 1 (of Epstein–Barr virus)
MALT	mucosa-associated lymphoid tissue
R-CHOP	the CHOP chemotherapy regimen plus rituximab
TR gene	T-cell receptor gene

Back cover

The barcode is printed incorrectly on the back cover.

WHO Classification of Tumours of Haematopoietic and Lymphoid Tissues, Revised 4th edition
Correction made after third print run

Summary of corrections:

The definition of B-lymphoblastic leukaemia/lymphoma with iAMP21 has been corrected as detailed below.

Chapter 12: Precursor lymphoid neoplasms p. 208
B-lymphoblastic leukaemia/lymphoma with iAMP21 > Definition

Original text	Corrected text
Definition B-lymphoblastic leukaemia/lymphoma (B-ALL/LBL) with iAMP21 is a neoplasm of lymphoblasts committed to the B-cell lineage characterized by amplification of a portion of chromosome 21, typically detected by FISH with a probe for RUNX1 that reveals ≥ 5 copies of the gene (or ≥ 3 extra copies on a single abnormal chromosome 21) {1561,1597}.	**Definition** B-lymphoblastic leukaemia/lymphoma (B-ALL/LBL) with iAMP21 is a neoplasm of lymphoblasts committed to the B-cell lineage characterized by amplification of a portion of chromosome 21. iAMP21, as its name implies, is defined by intrachromosomal amplification of a particular region of chromosome 21 that reliably involves the RUNX1 gene. It may be recognized in most cases by interphase FISH by identifying ≥ 5 copies of the RUNX1 gene with ≥ 4 copies clustered on a single chromosome 21. However, accurate distinction of iAMP21 from the gain of whole chromosomes 21 in interphase cells may require metaphase FISH, array comparative genomic hybridization, or interphase FISH with two different chromosome probes including one directed against the subtelomeric region of chromosome 21.