The EHA SIOPe Pediatric Extended Syllabus

2024

European Hematology Association

eha

FOREWORD

The European Hematology Association (EHA) and European Society for Paediatric Oncology (SIOPe) Pediatric Extended Syllabus was created in 2023 as addendum to the Adult Hematology Syllabus, and is meant to become part of the European Hematology Curriculum, which has been developed as the backbone of the EHA education activities.

The Pediatric Syllabus comprises all the areas that Pediatric Hematology covers as a medical discipline, from hematological malignancies, already included in the SIOPe Syllabus, to non-malignant hematology, which has been newly created for the purpose of this extended syllabus.

The level of knowledge for each topic expected from a Pediatric Hematologist who has finished the training is also included. The entire content is not required in all countries.

As per Hematology, considering the lack of homogeneity in terms of training for Pediatric Hematology throughout Europe, the aim is to harmonize educational requirements, trying to find the common knowledge that can be demanded at the European level. This is particularly relevant as one of the aims of the Syllabus is to serve as a tool to facilitate mobility.

The present first version is the fruit of the work of several pediatric hematologists, experts in the different fields of this topic, and comprises 8 sections, with each section having been carefully prepared and revised by a group of these experts. Diagnostic tools and specific aspects of Pediatric Hematology, as well as novel treatment modalities, have been included.

The Syllabus also aims to serve as a self-assessment tool for trainees and pediatric hematologists who want to find out their knowledge gaps and help them in their continuous training. A recommendation for the length of training in Hematology and a detailed description of the level of competence are included. We would like to thank the members of EHA's Specialized Working Group on Pediatric Hematology for their contribution to the success of this project. We expect that the Pediatric Hematology Syllabus, as part of the European Hematology Curriculum, will serve as the basis and backbone of the EHA education activities in Pediatrics, as well as a tool for self-assessment.

Joseph Vormoor
Chair, EHA Specialized Working Group on Pediatric Hematology

I.) Recommended length of training

Automatic recognition of professional qualifications across EU Member States, based on enhanced and harmonized minimum training requirements, is of crucial importance for the mobility of hematology professionals and, ultimately, for safeguarding the quality and safety of patient care. Given the wide scope of the discipline of hematology, as described in the Hematology Syllabus, EHA recommends a minimum training requirement for Hematology of five years, or three years when previous training encompassed the equivalent of at least two years in internal medicine.

II.) Structure of the Syllabus

The Syllabus is composed of eight main sections divided into subsections fitting into one of these categories: Clinical skills; Laboratory skills; Competences related to regulations and principles. Each one of these sections is composed of topics in Hematology that are assigned a recommended competence level according to endorsed European standards.

III.) Instructions to undertake the self-assessment

In order to complete the self-assessment, work through each section, select the level that most closely represents your current level and enter your responses. You will be able to see the recommended level of each topic and compare them against your responses, and in doing so identify your strong points of knowledge in Pediatric Hematology as well as learning opportunities in the topics wherein you need to enhance your skills.

Levels descriptor

Level 1

I am confident I can:

Clinical skills (patient management and treatment)

- Describe the clinical features and epidemiology of a condition or indications for a specific treatment/procedure or appropriateness/utility of a test
- Recognize a patient who may have this condition or require this treatment or benefit from this test

Laboratory skills

- Recognize the appropriateness and utility of a specific test for diagnosing and follow-up of specific hematological conditions
- Competences related to regulations and principles
- Identify applicable regulations or principles

Level 2

I am confident I can:

Clinical skills (patient management and treatment)

- Describe the pathogenesis
- Identify clinical features and investigations required to diagnose condition and interpret test results correctly
- Describe prognosis
- Identify correct referral routes or initiate appropriate treatment (according to established protocol)
- Identify the need for and establish urgent consultation with subspecialist (particularly if the condition has potentially life-threatening debut symptoms)

Laboratory skills

- Choose/order appropriate test(s) for a specific patient, taking into account:
    - Indications
    - Accuracy and limitations
    - What is entailed for the patient in performing the test
    - Interpret results for a specific patient

Competences related to regulations and principles

- Apply this regulation/principle relevantly and appropriately within my own clinical work

Level 3

I am confident I can:

Clinical skills (patient management and treatment)

- Decide and manage first-line treatment
- Identify treatment failure and need for second-line management
- Identify when there is a need for, and deliver, genetic counseling
- Seek out and integrate new knowledge and concepts in relation to condition/treatment

Laboratory skills

- Create/issue an interpretative report of test results
- Select/justify tests according to their cost-effectiveness

Competences related to regulations and principles

- Explain regulation/principle in appropriate language to a non-specialist audience (patient or student/trainee)
- Seek out and integrate new knowledge and concepts in relation to regulation/principle
- Recognize and plan how to improve own limitations, and demonstrate improvement

Section 1A: Red Blood Cell Disorders

Pediatric syllabus	Corresponding section in the Adult Syllabus	Learning points	EHA competence level
Classification of anemias		• Reticulocyte response (hypo-/non hyporegenerative): evaluate Coombs test and other hemolysis parameters • Mean corpuscular volume values (normo- micro-, macrocytic) • Involvement of other cells lines (isolated vs non isolated) • Congenital (inherited/*de novo*) vs acquired: investigate family history/past medical history/drugs	**3**

Hemoglobinopathies sickle cell anemia	**1Ae**	• Pathophysiology
		• Genetic basis including variants in combination with thalassemia/other hemoglobin
		• Geographic distribution
		• Clinical manifestation, age-related – multi-organ involvement including hematopoiesis, splenic function, pulmonary, central nervous system (CNS), liver, skeletal, renal
		• Management of disease manifestations – acute events and chronic sequelae
		• Approach to acute sickling events – anemia (hemolysis vs aplastic crisis), pain crisis, acute chest, splenic sequestration, dactylitis, acute stroke, penile erection
		• Approach to chronic sequalae:
		  ○ Indications for chronic transfusions (top-off vs exchange transfusions), management of these treatment protocols-complications (iron overload)
		  ○ Treatment of chronic pain
		  ○ Pulmonary hypertension
		• Therapeutics – indications for hydroxyurea, newer therapies, stem cell transplantation (SCT)
		• Family counseling/planning
			3

Thalassemia syndromes and other hemoglobinopathies	**1Ad**	• Pathophysiology
		• Spectrum of thalassemia syndromes
		• Clinical manifestation
		• Diagnosis
		• Treatment:
		  ○ Blood transfusion: transfusion dependent, non-transfusion dependent
		  ○ Splenectomy
		  ○ Fetal hemoglobin induction
		  ○ SCT
		  ○ Gene therapy
		  ○ Management of disease manifestations and chronic sequelae
		  ○ Iron overload: monitoring, prevention, treatment
		• Prognosis and follow-up
		• Family counseling/planning
		• Psychosocial issues
			3

Hereditary persistence of fetal hemoglobin	**1Af-1**	• Pathophysiology • Clinical Phenotype • Genetic counseling	**2**

Methemoglobinemia	**1Af-2**	• Pathophysiology and classification • Etiology • Clinical presentation • Diagnosis • Treatment • Family counseling	**2**
Other hemoglobinopathies	**1Af-3**	• Pathophysiology • Inheritance • Clinical phenotype • Evaluation and diagnosis • Treatment • Family counseling/planning	**2**
RBC membrane defects	**1Ag**	• Hereditary spherocytosis • Hereditary elliptocytosis • Hereditary pyropoikilocytosis • Hereditary stomatocytosis For each of the listed red blood cell membrane defects: • Pathophysiology • Clinical presentations • Diagnostic testing • Differential diagnosis • Treatment options • Prognosis • Family/genetic counseling	**3**
Enzyme defects	**1Ah**	• Pyruvate kinase deficiency • Glucose-6-phosphate dehydrogenase deficiency • Other rare enzyme disorders For each of the listed enzyme defects: • Pathophysiology • Clinical presentations • Diagnostic testing • Differential diagnosis • Treatment options • Prognosis	**2**
Immune hemolytic anemia	**1Aj**	• Classification: warm/cold agglutinins, paroxysmal cold hemoglobinuria • Etiology • Clinical presentation • Diagnosis • Therapy	**3**
Congenital dyserythropoietic anemias (CDA)	**1Ai-1**	• Classification: Type I-IV, CDA as part of a broader syndrome • Clinical presentation • Chronic sequelae • Diagnosis • Treatment • Family/genetic counseling	**1**

Diamond–Blackfan anemia ***(also see "Bone Marrow Failure Syndromes")***	**1Bd**	• Genetic basis/inheritance/*de novo*, ribosomopathy • Pathophysiology and genetic basis –ribosomopathy • Clinical presentation • Diagnostic testing – typical macrocytic, reticulocytopenic anemia, bone marrow adenosine deaminase (ADA) findings, ADA testing, genetic analysis • Differential diagnosis – transient erythroblastopenia of childhood (TEC), congenital parvovirus infection, vitamin deficiencies, myelodysplastic syndrome (MDS), other bone-marrow failure (BMF) syndromes • Clinical signs/symptoms • Great range in clinical picture/severity (mild anemia-transfusion dependence, genotype–phenotype) • Possibility of trilineage involvement • Physical anomalies – skeletal (triphalangeal thumb), typical facies/upper body anomalies, genitourinary tract • Increased risk for malignancy – acute myeloid leukemia (AML), acute lymphoblastic leukemia (ALL), lymphoma, breast cancer, colorectal carcinoma, osteogenic sarcoma • Treatment options • Steroids (recommended after 1st year, completion of vaccination schedule) • Regular transfusion regimen – treatment of iron overload • Stem cell transplantation, pre transplant iron overload needs to be abated due exquisite sensitivity of Diamond–Blackfan anemia patients to this risk, post-SCT late tumors risk • Prognosis • Genetic counseling/prevention • Age-related issues	**2**
Transient erytroblastopenia of childhood	**1Ai-2**	• Pathophysiology • Clinical manifestation • Diagnostic testing • Differential diagnosis • Treatment options • Prognosis	**3**
Pure red cell aplasia (acquired)	**1B**	• Pathophysiology – autoimmune, T-cell mediated • Clinical manifestation • Diagnosis/differential diagnosis (rule out secondary causes) • Treatment options – steroids, cyclosporin A • Prognosis • Psychosocial issues	**1**
Pearson's syndrome	**1Cb**	• Pathophysiology and genetic basis – mitochondrial DNA disorders • Clinical manifestation – neutropenia, pancreatic failure, delay/loss of developmental milestones • Differential diagnosis • Treatment options – supportive therapy • Prognosis – poor • Family/genetic counseling • Psychosocial issues	**1**

Nutritional anemias	**1Aa**	• Iron deficiency anemia:
		  ○ Iron metabolism and pathophysiology
		  ○ Risk factors: diet, age, population
		  ○ Clinical presentation: microcytic anemia
		  ○ Diagnosis: hemogram abnormalities, iron balance
		  ○ Prevention and treatment
		• Iron-refractory iron deficiency anemia:
		  ○ Pathophysiology and epidemiology
		  ○ Clinical presentation: microcytic anemia, peculiar iron pattern deficiency
		  ○ Diagnostic evaluation: iron balance, response to iron treatment, genetic testing
		  ○ Treatment: i.v. iron and supportive treatment
		  ○ Prognosis
		  ○ Genetic counseling
		• Vitamin B12 and folate deficiency anemia:
		  ○ Pathophysiology
		  ○ Etiology – risk factors
		  ○ Clinical presentation: megaloblastic anemia, neurological symptoms, neuropsychiatric symptoms
		  ○ Diagnosis: hemogram abnormalities, vitamin B12 and folate serum levels
		  ○ Prevention and treatment
			3

Neonatal anemias	**1A**	• Normal complete blood count (CBC) values by gestational and post-natal age
		• Differential diagnosis of neonatal anemia:
		  ○ Reduced red cell production
		  ▪ Parvovirus B19
		  ▪ Diamond–Blackfan anemia
		  ○ Hemolysis
		  ○ Red cell enzymopathies
		  ○ Red cell membrane disorders
		  ○ Hemoglobinopathies presenting in the neonate
		  ○ Alloimmune (hemolytic disease of the newborn)
		  ▪ Blood loss
		  ▪ Feto–maternal
		  ▪ Twin–twin
			3

Erythrocytosis/ polycythemia	**1Al**
	1Am
		• Definition
		• Etiology:
		  ○ Relative (i.e., volume depletion)
		  ○ Secondary acquired (i.e., increased erythropoietin [EPO] production for cardiopulmonary disease or ectopic production, high androgens, etc.)
		  ○ Juvenile/familial erythrocytosis
		  ○ Polycythemia Vera/myeloproliferative neoplasm,
		• Clinical presentation
		• Evaluation: hemogram, pulse oximetry, P50, serum EPO, serum chemistry, radiological evaluation, enzyme deficiency, genetic analysis - *HBB, HBA, BPGM, PKLR, VHL, EGLN1, EPAS1, EPO, EPOR, JAK1 or WGS/WES*)
		• Complications
		• Treatments and follow-up
		• Family/genetic counseling
			2

Section 1B: Bone Marrow Failure Syndromes

Pediatric syllabus	Corresponding section in the Adult Syllabus	Learning points	EHA competence level
Congenital amegakaryocytic thrombocytopenia (CAMT)	**1Bd**	• Pathogenesis/molecular basis(*c-mpl, TPO*) – megakaryocyte signaling • Differential diagnosis – neonatal alloimmune thrombocytopenia (NAIT), thrombocytopenia-absent radius (syndrome, congenital infections • Diagnosis – plasma thrombopoietin (TPO) level, mutation analysis, rule out NAIT, congenital infections, bone marrow analysis • Clinical manifestations – thrombocytopenia (neonatal), pancytopenia (by 2nd decade), MDS/AML • Treatment – TPO agonists, platelet transfusions, platelet component transfusions, granulocyte colony-stimulating factor (G-CSF), SCT – not indicated if the causative mutation is in TPO! (produced in the liver) • MDS/AML screening, surveillance • Family/genetic counseling	**2**

CARD11	**1Bd**	• Pathophysiology and genetic basis
		• Diagnostic testing –next-generation sequencing (NGS) panels vs whole exome sequencing (WES), testing in non-hematopoietic tissue
		• Differential diagnosis – other BMF syndromes, MDS, autoimmune lymphoproliferative syndrome (ALPS), other causes of immune dysregulation
		• Systems involved, varied clinical presentations:
		  ○ Allergy and atopic disease
		  ○ Autoimmunity
		  ○ Immune dysregulation
		  ○ Susceptibility to infections
		  ○ Neutropenia
		  ○ Lymphoproliferative disease
		  ○ B-cell defect and hypogammaglobulinemia
		  ○ BMF
		  ○ Other
		  ○ Cancer predisposition (lymphoma)
		• Treatment options and indications:
		  ○ SCT
		  ○ Side-effects and potential complications
		• Prognosis
		• Secondary malignancies surveillance program
		• Family/genetic counseling
		• Psychosocial issues
			1

DADA2 (adenosine deaminase) deficiency	**1Bd**	• Pathophysiology, genotype–phenotype association • Diagnostic testing – enzymatic, molecular analysis • Differential diagnosis – other BMF, rheumatic diseases, autoinflammatory diseases • Clinical manifestations – hematologic, vasculitis (polyarteritis nodosa, vascular accidents seen, massive palmar erythema nodosum, livedo racemose, arthritis/arthralgia, CNS), immune deficiency • Treatment options – tumor-necrosis factor (TNF) blockade, other immune suppression/modulation, regular transfusions, SCT? • Family/genetic counseling	**1**

Diamond–Blackfan anemia
 	
(also see Red Cell Disorders)
	1Bd	• Genetic basis/inheritance/*de novo*, ribosomopathy
		• Pathophysiology and genetic basis –ribosompathy
		• Clinical presentation
		• Diagnostic testing – typical macrocytic, reticulocytopenic anemia, bone marrow findings, ADA testing, genetic analysis
		• Differential diagnosis –TEC, congenital parvovirus infection, vitamin deficiencies, MDS, other BMF syndromes
		• Clinical signs/symptoms:
		  ○ Great range in clinical picture/severity (mild anemia-transfusion dependence, genotype–phenotype)
		  ○ Possibility of trilineage involvement
		  ○ Physical anomalies – skeletal (triphalangeal thumb), typical facies/upper body anomalies, genitourinary tract
		  ○ Increased risk for malignancy – AML, ALL, lymphoma, breast cancer, colorectal carcinoma, osteogenic sarcoma
		• Treatment options:
		  ○ Steroids (recommended after 1st year, completion of vaccination schedule)
		  ○ Regular transfusion regimen – treatment of iron overload
		  ○ Stem-cell transplantation, pre transplant iron overload needs to be abated due exquisite sensitivity of Diamond–Blackfan anemia patients to this risk, post-SCT late tumors risk
		• Prognosis
		• Genetic counseling/prevention
		• Age-related issues
			2

Dyskeratosis congenita	**1Bd**	• Pathophysiology and genetic basis
		• Diagnostic testing – telomere length flow-FISH vs ELISA, genetic analysis panels vs WES, testing in non-hematopoietic tissue
		• Differential diagnosis – short telomeres in severe aplastic anemia, MDS
		• Systems involved, varied clinical presentations, included age-related manifestations:
		  ○ Bone marrow failure, MDS, AML
		  ○ Lung
		  ○ Liver
		  ○ Gastrointestinal tract
		  ○ Brain
		  ○ Skin
		  ○ Vascular fragility – bleeding
		  ○ Other
		  ○ Cancer predisposition
		• Treatment options – indications, androgens – how they work, G-CSF/TPO receptor agonists (discuss possible clinical use/EPO):
		  ○ SCT – side effects, potential complications (including increased risk for late tumors as in all constitutional BMF)
		• Prognosis
		• Preventative cancer screening/surveillance regimen
		• Family/genetic counseling
		• Psychosocial issues
			2

Fanconi anemia	**1Bc**	• Pathophysiology and genetic basis
		• Diagnostic testing – chromosomal breakage (diepoxybutane and/or mitomycin C test), evaluation of cellular cycle arrest in G2, Western Blot test for Fanconi anemia group D2 ubiquitination, genetic analysis – NGS panels vs WES, testing in non-hematopoietic tissue, somatic mosaicism
		• Differential diagnosis – telomeropathies, other bone marrow failure syndromes, MDS, other syndromes with vertebral defects, anal atresia, cardiac defects, tracheo-esophageal fistula, renal anomalies, and limb abnormalities (VACTERL) anomalies
		• Systems involved, varied clinical presentations, included age-related manifestations:
		  ○ Short stature and failure to thrive
		  ○ Genitourinary apparatus
		  ○ Bone
		  ○ Skin
		  ○ Bone marrow failure, MDS, AML
		  ○ Other
		  ○ Cancer predisposition
		• Treatment options and indications:
		  ○ Androgens and how they work
		  ○ G-CSF/EPO
		  ○ SCT
		  ○ Gene therapy (available on selected experimental trials)
		  ○ Side effects and potential complications (i.e., increased risk/earlier onset of post-SCT tumors as in all constitutional BMFs)
		• Prognosis
		• Secondary malignancies surveillance program
		• Family/genetic counseling
		• Psychosocial issues
			3

***MDS1* and *EVI1* Complex Locus (MECOM) associated syndromes**	**1Bd**	• Pathophysiology and genetic basis
		• Diagnostic testing – NGS panels vs WES, testing in non-hematopoietic tissue, dominant transmission, high percent *de novo*
		• Differential diagnosis – CAMT, other BMF syndromes, MDS
		• Systems involved, varied clinical presentations:
		  ○ Bone (especially radioulnar synostosis)
		  ○ BMF, amegakaryocytic thrombocytopenia
		  ○ Congenital heart defects
		  ○ Renal malformations
		  ○ Deafness
		  ○ Immunological impairment (B-cell deficiency and hypogammaglobulinemia)
		  ○ Endocrine system
		  ○ Other
		• Treatment options and indications:
		  ○ SCT
		  ○ Side-effects and potential complications
		• Prognosis
		• Family/genetic counseling
		• Psychosocial issues
			1

Ohdo syndrome	**1Bd**	• Pathophysiology and genetic basis
		• Diagnostic testing –genetic analysis → gene-target deletion/duplication analysis or NGS/WES panels, testing in non-hematopoietic tissue
		• Differential diagnosis – other bone marrow failure syndromes, MDS
		• Systems involved, varied clinical presentations, included age-related manifestations:
		  ○ Craniofacial alteration
		  ○ Short stature and failure to thrive
		  ○ Development and behavior
		  ○ CNS
		  ○ Musculoskeletal
		  ○ Auditory and ophthalmological alterations
		  ○ Genitourinary system
		  ○ Cardiopulmonary system
		  ○ BMF
		  ○ Other
		• Treatment options and indications:
		  ○ G-CSF/EPO
		  ○ Supportive therapy
		  ○ SCT
		  ○ Side-effects and potential complications
		• Prognosis
		• Family/genetic counseling
		• Psychosocial issues
			1

***SAMD9/L* mutation syllabus**	**1Bd**	• Pathophysiology – relationship to chromosome 7 aberrations (“self-correction”), marrow failure opposed to MDS/AML • Diagnostic testing – new player in the field of severe aplastic anemia (SAA)/MDS analyses, need high clinical suspicion, include in all aplastic anemia work-ups, • Clinical manifestations: cytopenias, neurologic symptoms (e.g., ataxia) • Differential diagnosis – SAA vs MDS, other BMF syndromes • AML/MDS work-up including cytogenetics, somatic analysis is essential • Treatment decision tree – “watch-and-wait” vs growth factors vs SCT • Prognosis (still being discovered) • Family/genetic counseling/psychosocial issues – segregation studies are necessary, asymptomatic mutation “carriers”	**2**

Severe congenital neutropenia	**1Cd**	• Wide variety of genes involved, inheritance, pure neutropenia vs syndromic (Shwachman–Bodian–Diamond syndrome (SBDS) is in a separate section), pathogenesis of the major subtypes (*ELANE, HAX1, SRP54*)
		• Diagnostic testing – autoantibody testing (positive does not rule-out!), typical bone marrow early maturation arrest (not in all cases), genetic analysis – panels, WES
		• Differential diagnosis – post-infectious, alloimmune, autoimmune, other BMF syndromes, MDS
		• Clinical picture:
		  ○ Typical infections (early onset, gingivitis, skin, deep-seated), typical bacteria
		  ○ Cyclic vs chronic neutropenia
		  ○ Severity of neutropenia
		  ○ Syndromic cases (genes involved [e.g., *G6PC3*, *SRP54*, others])
			2

		• Treatment options:
		  ○ G-CSF preventatively vs per infection – recommendation is for continuous G-CSF treatment at the least effective dose, when to initiate G-CSF therapy?
		  ○ Proper antibiotic/antifungal therapy,
		  ○ Need for expert infectious disease consults
		  ○ SCT – when to transplant?
		• MDS/AML surveillance – CBC vs marrow analysis, cytogenetic/chromosomal abnormalities, recognized somatic mutations (*GCSF-R*, *RUNX1*)
		• Prognosis – malignancy rates in various subtypes
		• Family/genetic counseling
		• Psychosocial issues – chronic treatment	

Shwachman–Diamond syndrome	**1Cd**	• Pathophysiology and genetic basis – ribosomopathy
		• Diagnostic testing → genetic analysis – NGS panels vs Sanger evaluation, testing in non-hematopoietic tissue, tests for pancreatic insufficiency (fecal elastase, low serum pancreatic trypsinogen [< 3 years] and low isoamylase [> 3 years])
		• Differential diagnosis – telomeropathies, severe congenital neutropenia, other BMF syndromes, MDS
		• Systems involved, varied clinical presentations, included age-related manifestations:
		  ○ Short stature and failure to thrive
		  ○ Bone
		  ○ Exocrine pancreatic failure and malabsorption
		  ○ High susceptibility to infections
		  ○ Genitourinary apparatus
		  ○ Cardiovascular apparatus
		  ○ Endocrine system
		  ○ Skin
		  ○ Oral and dental alteration
		  ○ BMF, MDS, AML
		  ○ Behavioral disorders and cognitive deficits
		  ○ Cancer predisposition
		  ○ Other
		• Treatment options and indications:
		  ○ G-CSF
		  ○ Endocrine and malabsorption management
		  ○ SCT
		  ○ Side-effects and potential complications (i.e., increased risk of post-SCT tumors as in all constitutional BMFs)
		• Prognosis
		• Secondary malignancies surveillance program
		• Family/genetic counseling
		• Psychosocial issues
			2

Acquired aplastic anemia	**1Ba**	• Recognize and diagnose patients with acquired aplastic anemia • Understand the potential causes (e.g. autoimmune, toxic) • Manage patients with acquired aplastic anemia	**3**

Section 1Cb/1Cc: Isolated Neutropenia

Pediatric syllabus	Corresponding section in the Adult Syllabus	Learning points	EHA competence level
Neutrophil normal ranges	**1Cc-1**	• Awareness that neutrophil normal ranges vary between some populations/ethnicities	**1**
Neutrophil production defects	**1Cb/1Cc-2**	• See “Bone Marrow Failure” section • See also “Primary Immunodeficiencies” (PID) section for discussion around neutrophil function defects • Neutropenia of prematurity: ○ Seen not uncommonly in preterm infants; often of multifactorial cause	**2**
Transient viral neutropenia	**1Cc-3**	• Transient neutropenia: ○ Common following viral infection in children; generally self-resolving	**3**
Drug-induced neutropenia	**1Cc-4**	• Importance of knowledge of medication history • Drugs known to cause neutropenia, including but not limited to carbamazepine, colchicine, some antibiotics	**3**

Immune-mediated neutropenia	**1Cc-5**	• Alloimmune neutropenia:
		  ○ Transplacental antibodies to human neutrophil antigens due to mismatch between maternal and paternal antigens
		• Autoimmune neutropenia as a post-viral phenomenon in children:
		  ○ Diagnosis – anti-neutrophil antibodies
		  ○ Natural history – the majority resolve, but can take many months
		  ○ Clinical management – most children do not need any regular medication and need advice and education around management of fever. Prophylactic antibiotics and/or G-CSF rarely required
			3

Chronic immune neutropenia due to immune dysregulation	**1Cc6**	• Non-remitting neutropenia with and without antibodies against neutrophils • Delayed diagnosis • Sometimes anticipatory sign of immune dysregulation • Underlying variants of immune dysregulation	**2**

Section 1Cd: Primary Immunodeficiencies (PID)

Pediatric syllabus	Corresponding section in the Adult Syllabus	Learning points	EHA competence level

PID Classification	**1Cd-1**	Knowledge of main PID categories:
		• Immunodeficiencies affecting both cellular and humoral immunity
		  ○ T-cell-negative, B-cell-positive severe combined immune deficiency (SCID)
		  ○ T-cell negative, B-cell-negative SCID
		  ○ Combined immunodeficiency (CID), generally less profound than SCID
		• Combined immunodeficiencies with associated or syndromic features:
		  ○ Immunodeficiency with congenital thrombocytopenia
		  ○ DNA repair defects
		  ○ Thymic defects with additional congenital anomalies
		  ○ Immuno-osseous dysplasias
		  ○ Hyper-immunoglobulin (Ig) E syndromes
		  ○ Defects of vitamin B12 and folate metabolism
		  ○ Anhidrotic ectodermal dysplasia with immunodeficiency
		  ○ Calcium channel defects
		  ○ Others
		• Predominantly antibody deficiencies:
		  ○ Severe reduction in <u>all</u> serum Ig isotypes with profoundly decreased or absent B cells, agammaglobulinemia
		  ○ Severe reduction in at least 2 serum Ig isotypes, with normal or low number of B cells, common variable immune deficiency (CVID) phenotype
		  ○ Severe reduction in serum IgG and IgA, with normal/elevated IgM and normal number of B cells, hyper IgM
		  ○ Isotype, light chain, or functional deficiencies with generally normal numbers of B cells
		• Diseases of immune dysregulation:
		  ○ Familial hemophagocytic lymphohistiocytosis (FHL) syndromes
		  ○ FHL with hypopigmentation
		  ○ Regulatory T-cell defects
		  ○ Autoimmunity with or without lymphoproliferation
		  ○ Immune dysregulation with colitis
		  ○ ALPS
		  ○ Susceptibility to Epstein-Barr Virus and lymphoproliferative conditions
		• Congenital defects of phagocyte number and function:
		  ○ Congenital neutropenias
		  ○ Defects of motility
		  ○ Defects of respiratory burst
		  ○ Other non-lymphoid defects (incl. GATA2 deficiency)
		• Defects in intrinsic and innate immunity:
		  ○ Mendelian susceptibility to mycobacterial disease
		  ○ Epidermodysplasia verruciformis (human papillomavirus)
		  ○ Predisposition to severe viral infections
		  ○ Herpes simplex encephalitis
		  ○ Predisposition to invasive fungal disease
			1

		○ Predisposition to mucocutaneous candidiasis ○ Toll-like receptor signaling pathway deficiency with bacterial susceptibility ○ Other inborn errors of immunity related to non-hematopoietic tissues ○ Other inborn errors of immunity related to leukocytes • Autoinflammatory disorders: ○ Type 1 interferonopathies ○ Defects affecting the inflammasome ○ Non-inflammasome related conditions • Complement deficiencies • BMF • Phenocopies of inborn errors of immunity: ○ Associated with somatic mutations ○ Associated with autoantibodies	
PID - diagnosis	**1Cd-2**	• Recognize PID patterns and main warning signs of PID in pediatric patients • Take an accurate personal and family history • If PID is suspected or runs in the family, delay live-attenuated vaccinations and do not postpone immunological investigations • Investigate clinical history, including maternal pregnancy and neonatal history, growth and development, vaccine history, ongoing/previous treatments, concomitant/previous disease, family history, social history • Investigate features of infections (age at onset, length/frequency/severity of infectious episodes, sites of infections, recurrence at particular sites, microbiological etiology, treatment and response to it) • Perform a focused complete clinical examination to assess for nutritional status, dysmorphic features, alterations in skin and annexes/oral cavity/ears, nose, throat/lungs/heart/lymphoid tissue/joints/nervous system, clubbing, hepatosplenomegaly • Set up a clinical presentation-guided diagnostic process, including general screening tests and immunological investigations. • Use age-matched reference values to avoid misinterpretation of immunological test results. • First-step investigations: ○ CBC with leucocyte differential ○ Immunoglobulin isotype levels • Second-step immunological investigations: ○ Lymphocyte subsets analysis ○ Specific antibody response to vaccine antigens ○ IgG subclasses analysis ○ Lymphocyte function testing (with mitogen and antigen stimulation) • In case of hypogammaglobulinemia, exclude causes of secondary forms. • In case of CD4-positive T-cell lymphopenia, exclude HIV infection • Indication to more specific tests according to suspected type of PID (based on clinical presentation): ○ Neutrophil oxidation burst ○ Complement screening ○ Phagocyte studies ○ Enzymatic activity (e.g. ADA, purine nucleoside phosphorylase) ○ Natural killer (NK) cytotoxicity studies ○ Cytokine/cytokine receptor studies, anti-cytokine antibodies	**2**

		• Family/genetic studies (single-gene analysis, NGS panels of selected genes, WES – functional tests for validation needed in selected cases) • Newborn screening for PID available in some countries	
PID - SCID	**1Cd-3**	• Knowledge that SCID is a medical emergency! • Maternal engraftment should be excluded in case of apparently normal T-cell count in high clinical suspicion • Knowledge that “leaky” SCID or Omenn syndrome can be caused by hypomorphic mutations in genes known to cause classical SCID	**1**
PID - non-SCID	**1Cd-4**	• T-cell defects are at risk for infections from opportunistic pathogens → *Pneumocystis Jirovecii* pneumonia (PJP) prophylaxis is needed • Timely recognition of antibody deficiency prevents future organ damage	**2**
PID - CVID	**1Cd-5**	• Knowledge that morbidity is not limited to infections, but also to non-infectious complications: splenomegaly, chronic gastrointestinal disease, chronic pulmonary disease, bronchiectasis, autoimmune cytopenias, granulomas, tumors • Monitoring and early treatment of associated diseases	
PID - treatment	**1Cd-6**	• Antimicrobial prophylaxes (bacterial, fungal, viral, PJP) • Aggressive and timely treatment of infections • Immunoglobulin replacement (s.c./i.v.) • Immune suppressants • Biologic agents in selected diseases (e.g. abatacept in lipopolysaccharide-responsive and beige-like anchor protein deficiency) • Allogeneic hematopoietic SCT (HSCT) • Autologous gene therapy	**2**
PID - Other	**1Cd-7**	• Prognosis and follow-up (including autoimmune manifestations and tumors) • Family counseling/planning • Psychosocial issues	**1**

Section 4: Hematopoietic Stem Cell Transplantation (HSCT) and Gene Therapy (HSC-GT)

Pediatric syllabus	Corresponding section in the Adult Syllabus	Learning points	EHA competence level
Indications to allogeneic HSCT	**4Bb**	• Hematologic malignancies (leukemias, lymphomas, MDS) • Inherited BMF syndromes • Hemoglobinopathies (thalassemia, sickle cell disease) • Inborn errors of immunity (including primary hemophagocytic lymphohistiocytosis [HLH] and autoinflammatory diseases) • Inborn errors of metabolism (metachromatic leukodystrophy [MLD]; X-linked adrenoleukodystrophy; mucopolysaccharidosis type IH, IIIA [MPSIH, MPSIIIA]; others) • Infantile malignant osteopetrosis • Secondary HLH • Autoimmune diseases (selected cases)	**3**
Indications to hematopoietic stem cell gene therapy (HSC-GT)	**4Bj**	• Inborn errors of immunity (ADA-SCID; SCID-X1, WAS, X-CGD and p47 CGD; LAD, RAG1-SCID; Artemis-SCID) • Hemoglobinopathies (ß-thalassemia; sickle cell disease) • Inherited BMF syndromes: Fanconi anemia • Inborn errors of metabolism (MLD; cerebral adrenoleukodystrophy, MPSIH, IIIA, Fabry disease) • Clinical trials vs standard of care	**2**
Indications for CAR T cells	**4Bi**	• Chimeric antigen receptor (CAR) cell therapy for ALLs • Emerging indications for lymphomas and other hematological malignancies • Emerging indications for solid and brain tumors	**3**
Other cellular therapies	**4Bj**	• Donor lymphocyte infusion • Virus-specific T cells • NK cells • Cytokine-induced killer (CIK) cell • Mesenchymal stromal cells • Dendritic cells • CARs: T, NK, and CIK cells	**1**
Mobilization, collection and manipulation of hematopoietic stem cells	**4Bc**	• Identification of target dose • Bone marrow harvesting, leukapheresis and cord blood procurement • Graft manipulation	**1**
Criteria for selection of intensity for the preparative regimens	**4Aa** **4Bd**	• Myeloablative conditioning, reduced toxicity conditioning , reduced intensity conditioning • Chemotherapy, irradiation, serotherapy and biological agents	**1**
Identification and selection of stem cell donor	**4Be**	• Donor type (autologous, human leukocyte antigen [HLA]-identical family donor, unrelated donor, haploidentical family donor) • Hematopoietic stem cell (HSC) source (bone marrow-derived HSC, mobilized peripheral HSC, cord blood) • HLA and other non-HLA compatibility assessment	**1**
Acute and chronic graft-versus-host disease (GvHD)	**4Bf**	• Pathogenesis • Clinical presentation and grading • Therapy	**2**

Other (early) complications	**4D**	• Infectious complications • Bleeding and thrombotic complications • Graft failure • Early complications of endothelial origin • Chemo- and radiotherapy-related acute toxicities • Cytokine release syndrome • Autoimmune cytopenias • Thymic exhaustion	**2**
Late complications	**4Bg**	• Late complications from chemo-/radiotherapy, biologicals, and immunosuppressive agents • Secondary cancer, post-transplant lymphoproliferative disease • Insertional mutagenesis (only HSC-GT) • Growth and development issues	**2**
Post-transplant monitoring	**4Bh**	• Central venous catheter management • Infection control and isolation procedures • Prevention and management of GvHD, graft rejection, relapse of malignancy • Psychological support, schooling, and education program during HSCT • Monitoring and management of the principal advanced cellular therapies toxicities • Monitoring of immune reconstitution and chimerism • Monitoring of chimerism	**1**
Pediatric fertility preservation program	**4Ai**	• Specific fertility preservation strategy and gametes cryopreservation • Protection of gonadal function during chemotherapy	**2**

Section 6: Platelet Disorders, Thrombosis and Hemostasis

Pediatric syllabus	Corresponding section in the Adult Syllabus	Learning points	EHA competence level
Bleeding disorders – General	**6Aa**	• Relevant and accurate personal and family bleeding history • Focused clinical examination to assess for abnormal bleeding symptoms and signs • Comprehensive differential diagnosis, including acquisition of relevant laboratory tests • Management plan for patients with abnormal bleeding, including familial genetic counseling, management in case of bleeding, trauma, or surgery, and multidisciplinary guiding • Coagulation pathways including control mechanisms and fibrinolysis	**3**
Hemophilia A and B	**6Ca**	• Clinical manifestations of hemophilia • Diagnosis of hemophilia A and B by interpretation of laboratory tests, diagnosis of patients with inhibitors to factor (F) VIII and FIX • Genetics of hemophilia patients and carriers, the impact of genetics upon future risk (e.g., inhibitor formation) • Hemophilia treatment in case of bleeds, trauma, or surgery (desmopressin, factor replacement, bypass agents, antifibrinolytics) • Hemophilia prophylaxis - replacement therapy (primary and secondary prophylaxis, use of coagulation concentrates) and non-replacement therapy (e.g., emicizumab) • Joint pathology and long-term outcomes in hemophilia • Hemophilia treatment in the presence of inhibitors including treatment of bleeds, immune tolerance induction therapy. and treatment with prophylaxis, including NRT (e.g., emicizumab). • Current status and studies of gene therapy in hemophilia	**3**
von Willebrand Disease (VWD)	**6Cb**	• Understanding the incidence, inheritance, classification (including molecular and genetic aspects), clinical manifestations, natural history, and clinical complications of patients with VWD • Diagnosis and classification of VWD subtypes (Type 1, 2A, 2B, 2M, 2N and 3) by interpretation of laboratory tests including coagulation factors levels and activity, platelet aggregation studies and interpretation of VW multimers' studies, and molecular diagnostics. • Treatment of bleeds and surgery in patients with VWD, including use of desmopressin acetate, FVIII/von Willebrand factor (VWF) concentrates, antifibrinolytics and supportive care (e.g., oral contraceptives for women).	**3**
Rare bleeding disorders	**6Cc**	• Pathophysiological mechanisms, incidence, clinical manifestations, and treatment of quantitative and qualitative disorders of FII, FV, FVII, FX, FXI, FXIII, fibrinogen, and other isolated and combined rare bleeding disorders, and relate this to clinical management of patients with these disorders • Genetic background of rare bleeding disorders and provide family counseling accordingly • Management of patients with rare bleeding disorders during prophylaxis or interventions (e.g., acute bleeding, surgery)	**2**

Platelet disorders	**1D**
	6Ce
		• Diagnostic pathway for patients with thrombocytopenia and platelet function defects
		• Diagnosis and management of patients with immune thrombocytopenia, indications for treatment and treatment options, including steroids, immunoglobulins, anti-D, anti-CD20, thrombopoietin mimetics, and splenectomy
		• Diagnosis and management of patients with drug-induced platelet disorders
		• Diagnosis of patients with hereditary disorders of platelet function, including Bernard–Soulier syndrome and Glanzmann thrombasthenia:
		  ○ Interpretation of results of light transmission aggregometry and flow cytometry analysis of these disorders
		• Genetics of hereditary platelet disorders
		• Management of patients with hereditary disorders of platelet function:
		  ○ Treatment plans of bleeding episodes, surgical interventions etc., taking into consideration the status of anti-platelet antibodies
		• Diagnosis and management of patients with congenital and acquired thrombotic thrombocytopenic purpura as well as other microangiopathic disorders
		• Diagnosis and management of patients with heparin-induced thrombocytopenia
			3

Hemostasis in the newborn	**6A**	• Relating knowledge of developmental hemostasis to the interpretation of laboratory coagulation tests (coagulation factors activity, natural coagulation inhibitors and global hemostatic assays) for clinical management of neonates and children • Diagnosis and management of hemorrhagic disease of the newborn, including vitamin K deficiency • Diagnosis and management of thrombocytopenia in neonates including applying and interpreting tests for diagnosis of fetal and neonatal alloimmune thrombocytopenia	**2**
Bleeding diathesis without diagnosis	**6B** **6C**	• Differential diagnosis in case no coagulation disorder is found, with laboratory testing and when and to whom to refer (i.e., non-accidental injuries (child abuse, auto-mutilation), hormonal causes of menorrhagia, hereditary hemorrhagic telangiectasia, connective tissue disease like Ehlers–Danlos syndrome)	**1**
Thrombotic disorders	**6D**	• Epidemiology and molecular basis of thrombotic disorders in children affected by these conditions • Relevant personal and family history • Understanding the normal hemostatic parameters in neonates, children, and adolescents, particularly in relation to inhibitors of coagulation and the fibrinolytic system • Diagnosis of hypercoagulable states (inherited and acquired) by interpreting laboratory tests, and the use of age adjusted normal ranges during childhood • Indications for thrombophilia testing • Recognizing the presentation of homozygous protein C and S deficiency and treatment plan • Interpretation of the clinical relevance of heritable thrombophilia to venous and arterial thrombosis in pediatric patients and provide family counseling in case of thrombophilia and/or positive family history for thrombosis	**3**

		• Diagnosis and management of thrombosis during childhood (including treatment options, treatment duration, supportive care and follow-up)	
Clinical aspects of venous thromboembolism (VTE)	**6D**	• Understanding the types and locations of thrombosis observed in neonates, children and adolescents • Risk factors for thrombosis in neonates, children, and adolescents • Diagnosis of patients with suspected VTE by proper imaging studies (Doppler ultrasound, computed tomography angiography/venography [CTA/V], magnetic resonance venography) • Treatment of patients with acute VTE • Risk of recurrence in patients with VTE and risk-based treatment plans • Recognition and management of patients with post-thrombotic syndrome	**3**
Clinical aspects of arterial thromboembolism	**6D**	• Types and locations of arterial thrombosis observed in neonates, children and adolescents • Risk factors for arterial thrombosis in neonates, children and adolescents • Diagnosis of patients with suspected arterial thrombosis by proper imaging studies (CT, CTA, magnetic resonance imaging/angiography • Treatment of patients with acute arterial thrombosis (including indications for thrombolysis/thrombectomy) • Relating the principles of the epidemiology of arterial thrombosis to clinical care of children affected by these disorders • Age-related therapy considerations relevant to perinatal stroke, perinatal arterial thrombosis, and arterial thrombosis in children/adolescents • Evaluation and management of patients with arterial thrombosis, including cerebrovascular risk factors and anatomic malformations (e.g., Moyamoya syndrome, Kawasaki disease)	**3**
Antithrombotic therapy	**6Da**	• Indications and methods of anticoagulation, thrombolysis, thrombectomy in neonates, children and adolescents • Indications and methods of prophylactic anticoagulation in children and adolescents (primary and secondary) • Applying the understanding of the mechanisms of action and therapeutic indications of anticoagulant agents in patients' management • Management of children receiving anticoagulants, including advice on duration and intensity and interactions with other medications • Interpretation of tests for anticoagulant control (e.g. international normalized ratio, activated partial thromboplastin time, anti-Xa levels, thrombin clotting time, specific trough/levels) • Management of anticoagulation and antiplatelet therapy in association with invasive procedures • Management of patients with anticoagulant associated bleeding • Management of patients on antiplatelet agents • Management of patients on fibrinolytic drugs, including streptokinase, urokinase, tissue plasminogen activator	**2**

Section 7: Special Aspects of Pediatric Transfusion Management

Pediatric syllabus	Corresponding section in the Adult Syllabus	Learning points	EHA competence level
Neonatal and pediatric compatibility testing	**7A**	• Maternal antibody testing for neonates • Importance of maternal transfusion history and baby's transfusion history (including transfusions given in utero) • Compatibility requirements for neonates and infants	**2**
Component specifications	**7B**	• Fetal/neonatal/infant specification components (donor specifications, antibody testing, cytomegalovirus [CMV] testing, age of red cell components): o Use of neonatal split red cell packs for top-up transfusion (minimizing donor exposure) o Component specifications for small and large volume transfusions in neonates (including neonatal exchange) o Knowledge of specification and volumes available (country-specific in terms of exact unit specifications) • Components for cardiac surgery: o Age of product (theoretical risks of hyperkalemia)	**1**
Neonatal and pediatric transfusion thresholds and indications	**7C**	• Red cells: o Transfusion thresholds in neonate and children o Formula to calculate volume required (in ml) • Platelets: o Transfusion thresholds in neonates and children o Volume for transfusion • Plasma/cryoprecipitate: o Inductions for transfusion o Volumes required • Granulocyte transfusions: o Indication – refractory infections in severe neutropenia	**3**
Special requirements relevant to neonatal and pediatric practice	**7D**	Knowledge of appropriate use of: • CMV-negative components • Irradiated components • Rhesus phenotype matched (sickle, thalassemia, and other chronically transfused patients) • Age of red cell components	**3**

Special transfusion situations in neonates and children	**7E**	• Intrauterine transfusions:
		  ○ Product specifications and special requirements
		• Fetal NAIT (FNAIT)/NAIT:
		  ○ Laboratory investigation of FNAIT
		  ○ Transfusion and clinical management of NAIT (for example HPA1a5b negative platelets)
		• Neonatal exchange transfusion:
		  ○ Management of hemolytic disease of the newborn
		  ○ Provision of red cells
		• Exchange transfusion in red cell disorders (in older children):
		  ○ Red cell requirements
		  ○ Complications of exchange transfusion
		• Massive hemorrhage in infants and children:
		  ○ Evidence for and use of tranexamic acid
		  ○ Blood product management
		  ○ Management of coagulopathy
			3

Transfusion complications/reactions and hemovigilance	**7F**	• As per adult practice but to include more pediatric complications of transfusion such as transfusion associated necrotizing enterocolitis, issues with patient identification and inappropriate volumes transfused	**2**

Pediatric aspects of patient blood management	**7G**	• As per adult practice but also:
		  ○ Minimizing blood sampling/sample volume
		  ○ Use of near patient testing
		  ○ Delayed cord clamping
		  ○ Appropriate management of iron deficiency/hematinic deficiency
		  ○ If appropriate use of cell salvage
			2

Appendices

APPENDIX I

The EHA SIOPe Pediatric Extended Syllabus was reviewed and endorsed by Tomás Navarro Ferrando (chair) on behalf of the EHA Curriculum Committee.

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