



Kingdom of Saudi Arabia  
Ministry of National Guard - Health Affairs  
Prince Mohammed bin Abdulaziz Hospital



مستشفى الملك فيصل التخصصي ومركز الأبحاث  
King Faisal Specialist Hospital & Research Centre  
Gen. Org. مؤسسة عامة



# Middle East And North Africa Hematology Congress 2019

## 8th PAN ARAB HEMATOLOGY ASSOCIATION CONGRESS

### 17<sup>th</sup> Annual Meeting OF SAUDI SOCIETY OF HEMATOLOGY

# CONGRESS BOOK



[www.paha2019.com](http://www.paha2019.com)





تحت رعاية صاحب السمو الملكي

الأمير فيصل بن عبدالعزيز آل سعود

أمير منطقة المدينة المنورة



# Welcome message

On behalf of organizing, scientific and abstract review committees' members, we cordially welcome and invite you to the....

Middle East and North Africa Hematology Congress 2019, & 8th Pan Arab Hematology Association (PAHA) Congress and 17th Annual Meeting of Saudi Society of Hematology (SSH). The scientific committee has come up with a very interesting scientific program that represents the interests of Hematology professionals' and promotes the multidisciplinary of the field. All multimodality approaches in the management of hematological disorders are addressed.

We deeply thank the abstracts submitters. Where we have reached a new record this year with sum of 122 abstracts submitted, 93 abstracts have been accepted. All abstracts have gone over multi-steps review and scoring. We hope this year hematology knowledge take the up-coming congresses to a new era of research-oriented congresses.

We wish everyone a fruitful and fulfilling time during this conference. Your active participation will give an important boost to the success of this event.

## Dr. Turki Alwasaidi, MD

President, Middle East and North Africa Hematology Congress 2019, 8th Pan Arab Hematology Association Congress, 17th Annual Meeting of Saudi Society of Hematology

## Dr. Ahmad Tarwah, MD

Co-President, Middle East and North Africa Hematology League congress 2019

## Dr. Abdulhadi Tashkandi, MD

Chairman of Organizing Committee

## Dr. Abdulqader AlHebshi

Chairman of Scientific Committee



## GENERAL INFORMATIONS

**QIBLA DIRECTION** at Madinah:  
**Toward south**

## PRAYER TIME AT PROPHET MOSQUE:

Isha	Maqreb	Aser	Zoher	Sunrise	Fajer	February
07:37 PM	06:07 PM	03:45 PM	12:36 PM	07:03 AM	05:43 AM	01
07:38 PM	06:08 PM	03:45 PM	12:36 PM	07:03 AM	05:43 AM	02
07:39 PM	06:09 PM	03:46 PM	12:36 PM	07:02 AM	05:43 AM	03
07:39 PM	06:09 PM	03:47 PM	12:36 PM	07:02 AM	05:42 AM	04
07:40 PM	06:10 PM	03:47 PM	12:36 PM	07:01 AM	05:42 AM	05

## WEATHER:

Day	Description	High / low	Precipitation	Wind	Humidity
FRI FEB 1	AM Showers	29° 17°	40%	WSW 9 mph	43%
SAT FEB 2	Sunny	28° 16°	0%	SSE 8 mph	34%
SUN FEB 3	Sunny	28° 16°	0%	SE 8 mph	31%
MON FEB 4	Sunny	28° 16°	0%	ESE 9 mph	28%
TUE FEB 5	Sunny	29° 17°	0%	SE 8 mph	25%

## SHUTTLE BUS

Please follow schedule

## VENUE

Convention Center, Taibah University



## CONFERENCE COMMITTEE:

### HIGHER ORGANIZING COMMITTEE:

Dr. Walid Yafi  
Executive Director Medical Services, MNGHA-PMBAH

Mr. Mansour Al-Askar  
Executive Director of Operation, MNGHA-PMBAH

Dr. Turki Alwasaidi  
President of the Conference

Dr. Ahmed Tarawah  
Co President

~~Prof. Soad Aljaoni  
President of Saudi Society of Hematology~~

~~Prof. Mohammad Qari  
President of Pan Arab Hematology Association~~

## ORGANIZING COMMITTEE:

Dr. Abdulhadi Tashkandi  
Chairman of Organizing Committee

Dr. Mohammed Adnan Zolaly  
Co-Chairman of Organizing Committee

### Members:

- Dr. Abdulhadi Habeb
- Dr. Amal Albayhani
- Dr. Abdulqader AlHebshi
- Mr. Anas Jan
- Mr. Amer Tajaldeem
- Mr. Hussam Suliman
- Mr. Mutas Badwais
- Mr. Saees Abdullah
- Mrs. Dania Alayoubi
- Ms. Randa Ramadan
- Ms. Rasha Ramadan



## **SCIENTIFIC COMMITTEE:**

Dr. Abdulqader AlHebshi  
Chairman of Scientific Committee

Dr. Ahmed Tarawah  
Co-Chairman of Scientific Committee

### **Members:**

- Dr. Ali Alqraqri, KSA
- Dr. Ali Mullah Ali, Kuwait
- Dr. Amal Al sabahi, Oman
- Dr. Ahmed Al Askar, KSA
- Dr. Azza Kamel, Egypt
- Dr. Adnan Bakarman, Yemen
- Dr. Ahraf Warsi, KSA
- Dr. Amal Alabdulwahab, KSA
- Dr. Ahmad Alsaeed, KSA
- Dr. Arwa Omar Alyamani, KSA
- Dr. Ahmad Al Rustamani, UAE
- Dr. Eman Khattab, Jordan
- Dr. Eman Taryam, UAE
- Dr. Fatema Shams, Bahrain
- Dr. Fathia Alqurashi, Bahrain
- Dr. Faiha Bazzeh, Jordan
- Prof. Gamal Abdulhamid Alalawi , Yemen
- Prof. Gailal Mokhtar , Egypt
- Dr. Hayder Al Momen, Iraq
- Dr. Hani AlHashmi, KSA
- Dr. Kamel Aldosari, KSA

## **Abstracts Review Committee :**

Dr. Ahmad Tarawah ( Coordinator )

### **Members:**

- Prof. Azza Kamel
- Prof. Galila Mokhtar
- Prof. Mervat Mattar
- Prof. Azza Tantawy
- Dr. Abeer Abdelmoneim



## **SOCIAL COMMITTEE:**

Dr. Mohammed Adnan Zolaly  
Chairman of Social Committee

Dr. Ahmed Tarawah  
Co-Chairman of Social Committee

### **Members:**

- Dr. Amal Albayhani, KSA
- Dr. Saud Balilah, KSA
- Mr. Yousef Shahani, KSA



# PROGRAM AT GLANCE

Time	Track A	Track B	Track C
DAY 1, Friday, February 1 <sup>st</sup> , 2019			
16:00		SCA Patients And Families Workshop	Hemophilia Patients & Families Workshop
DAY 2, Saturday, February 2 <sup>nd</sup> , 2019			
08:00	KEYNOTE LECTURE 1: Medicine in AlMadinah, Historical Overview		
08:40	KEYNOTE LECTURE 2: Multi-Modal Mechanisms of Novel Sulfated Non-Anticoagulant LMWH in Sickle Cell Disease		
09:20	WFH / MENA Joint Session: Hemophilia and Emerging Therapies: Global Perspective		
10:00	Break / AlMadinah Traditions		
10:30	ISH / MENA / PAHA Joint Session	Acute Myeloid Leukemia	Sickle Cell Anemia
11:30			Sickle Cell Anemia Oral Abstracts
12:00	Highlights From The Pan-Arab Congress For Bleeding Disorders I	MPN	
12:40	Prayer and Lunch		
13:30	Highlights From The Pan-Arab Congress For Bleeding DisordersII	Plasma Cell Dyscrasias	Acute Myeloid Leukemia Oral Abstracts
14:20	Medical Student and Residents Educational Program (I)	CML	Bleeding Disorders Oral Abstracts
16:00		Hematology Lab Workshop	
DAY 3, Sunday, February 3 <sup>rd</sup> , 2019			
08:00	KEYNOTE LECTURE 3: ICUS/CHIP/CCUS: What determines a correct diagnosis? When to treat and when not treat, older patients with AML		
08:40	PLENARY SESSION 1: Phoenix Dactylifera Palm Fruit (AlMadinah Ajwa)		
09:20	EHA / SSH / PAHA Joint session: High Throughput Flowcytometry for Combined MRD Measurements & Immune Monitoring		
10:00	Break / AlMadinah Traditions		
10:30	Thrombosis And Anticoagulants	Lymphoma	Acute Lymphoblastic Leukemia Oral Abstracts
12:20	Lunch and Prayer		
13:00	Transfusion Medicine	Acute Lymphoblastic Leukemia	Thalassemia Disorders
14:30	Transfusion Medicine Oral Abstracts	Miscellaneous	
15:15	Lymphoma Oral Abstracts	Thalassemia Disorders Oral Abstracts	Thalassemia Disorders Oral Abstracts
DAY 4, Monday, February 4 <sup>th</sup> , 2019			
08:00	Medical Student And Residents Educational Program II	Plenary Session 2: Cutaneous Lymphoma	Plenary Session 3: Iron Deficiency Syndromes
08:40		CLL / SCT	Iron Deficiency Syndromes Oral

ما زالت المشكلة قائمة

تحتاج تصغير


عشان الصفحة تكون كاملة



# DAY 1- FEBRUARY 1, 2019 (FRIDAY)

## PRE-CONGRESS WORKSHOPS


### WORKSHOP 1

 16:00 – 18:00

**TRACK ( B ) :- MENA / MHBDCS  
JOINT WORKSHOP SCA PATIENTS AND FAMILIES WORKSHOP**

 ZAKARIA ALHAWSAWI (KSA)

### WORKSHOP 2


 16:00 – 18:00

**TRACK ( C ) :- MENA / MHBDCS  
JOINT WORKSHOP HEMOPHILIA PATIENTS AND FAMILIES WORKSHOP**

 SAOUD BALILLAH (KSA)

## DAY 2- FEBRUARY 2, 2019 (SATURDAY)

### “ MERGE TRACK ”


 07:30 - 08:00

**WELCOMING AND INTRODUCTION**

 08:00 - 08:40


**KEYNOTE LECTURE 1 MEDICINE IN ALMADINAH, HISTORICAL OVERVIEW**

 SAEED TOLAH (KSA)

 08:40 - 09:20

**KEYNOTE LECTURE 2 MULTI-MODAL MECHANISMS OF NOVEL SULFATED  
NON-ANTICOAGULANT LOW MOLECULAR WEIGHT HEPARIN IN SICKLE CELL DISEASE**

 SHAKER MUSA (USA)

 09:20 - 10:00

**WFH / MENA JOINT SESSION HEMOPHILIA AND EMERGING  
THERAPIES: GLOBAL PERSPECTIVE**

 GLENN PEIRCE (USA)

**BREAK / ALMADINAH TRADITIONS**






# DAY 2- FEBRUARY 2, 2019 (SATURDAY)

## TRACK A


### TRACK A : ISH / MENA / PAHA JOINT SESSION

**MODERATORS: SABRI KEMAHLI (TURKEY) , MERVET MATTER (EGYPT)**

 10:30 - 10:50


#### **TRANSFUSION MEDICINE EDUCATION**

 SABRI KEMAHLI (TURKEY)

 10:50 - 11:10


#### **HEMOPHILIA INHIBITORS**

 KAAAN KAVAKLI (TURKEY)

 11:10 - 11:30


#### **HEMOPHILIA IN MOROCCO**

 MOHAMED ELKHORASSANI (MOROCCO)

 11:30 - 11:50

#### **ROLE OF IMAGING IN THE ASSESSMENT & MANAGEMENT OF HEMOPHILIC ARTHROPATHY**

 MUSA HAWSAWI (KSA)


 11:50 - 12:10

#### **FI & FVII DEFICIENCY**

 ROULA FARAH (LEBANON)


### **SESSION 1A: HIGHLIGHTS FROM THE PAN-ARAB CONGRESS FOR BLEEDING DISORDERS (I)**

**MODERATORS: ASAAD HAFFAR (CANADA) TAREK OWIDAH (KSA)**

 12:10 - 12:30

#### **OVERVIEW OF CLINICAL ASSESSMENT DIAGNOSIS AND MANAGEMENT OF VWD**

 MAGDY ELEKAYBI (EGYPT)

 12:30 - 12:50

#### **UPDATE IN REPLACEMENT THERAPY IN VWD**

 KAAAN KAVAKLI (TURKEY)




**PRAYER AND LUNCH**






## SESSION 2A: HIGHLIGHTS FROM THE PAN-ARAB CONGRESS FOR BLEEDING DISORDERS (II)

**MODERATORS: MAGDY ELEKAYABI (EGYPT) , YASER WALI (OMAN)**

 **13:30 - 13:50**

### **RARE BLEEDING DISORDERS, DIAGNOSIS AND MANAGERMENTS**

 **FLORA PEYVANDI (ITALY)**


 **13:50 - 14:10**

### **WOMEN AND BLEEDING DISORDERS**

 **CLAIRE MCLINTOCK (AUSTRALIA)**

 **14:10 - 14:30**


### **GAUCHER DISEASE**

 **AYMAN HIJAZI (KSA)**

## DISCUSSION


## WORKSHOP 3

### **MEDICAL STUDENT AND RESIDENTS EDUCATIONAL PROGRAM (I) HEMATOLOGY MENTORSHIP, MEET THE PIONEER MODERATOR : DR. ABEER ABDULSALAM ALBOSHI**

 **14:40 - 15:00**

### **APPROACH TO BLEEDING TENDENCY**

 **ROULA FARAH (LEBANON)**

 **11:30 - 11:50**

### **MENTORSHIP**

**OPEN DISCUSSION WITH PROF. SHAKER MUSA**



# DAY 2- FEBRUARY 2, 2019 (SATURDAY)

## “ TRACK B ”


### SESSION 1B: ACUTE MYELOID LEUKEMIA

**MODERATORS: NAWAL AL MASHAYKHI (OMAN) - AHMAD ALSAEED (KSA)**

 10:30 - 10:50


#### ACUTE MYELOID LEUKEMIA: MOVING BEYOND 7+3

 NAIF ALJOHANI (KSA)

 10:50 - 11:10


#### DISEASE BIOLOGY IN AML AT DIAGNOSIS AND RELAPSE

 SAYED OSMAN (KSA)

 11:10 - 11:30


#### TREATMENT OF PEDIATRIC PATIENTS WITH AML

 IBRAHIM ABUSADAH (KSA)

 11:30 - 11:50

#### APPROACH TO INHERITED BM FAILURE


 SAAD ALDAMAAH (KSA)

 11:50 - 12:00

#### DISCUSSION


### SESSION 2B: MPN

**MODERATORS: AHMAD AL RUSTAMANI (UAE) - AMAL ALBIHANI (KSA)**

 12:00 - 12:20

#### MOLECULAR GENETICS OF MPN

 OSAMA KHOJA (KSA)

 12:20 - 12:40

#### MANAGEMENT OF ADVANCED PHASE MPN

 AHMED ALSAGHEIR (KSA)




**PRAYER AND LUNCH**






## SESSION 3B: PLASMA CELL DYSCRASIAS

**MODERATORS: AZZA KAMEL (EGYPT) - AYMAN MAASHI (KSA)**

 13:30 - 13:50


### **PROGNOSTIC FACTORS IN MULTIPLE MYELOMA**

 MAJED ALAHMADI (KSA)

 13:50 - 14: 10


### **APPROACH TO THE TREATMENT OF THE YOUNG, FIT PATIENT WITH MYELOMA: FROM DIAGNOSIS TO RELAPSE**

 FAHAD ALSHARIF (KSA)

 14:10 - 14: 30

### **APPROACH TO THE TREATMENT OF THE OLDER, UNFIT PATIENT WITH MYELOMA: FROM DIAGNOSIS TO RELAPSE**

 SAUD ALHAILI (KSA)

 14:30 - 14:40

### **DISCUSSION**

## SESSION 4B: CML

**MODERATORS: AMEERA RADHI (BAHRAIN) - MOHAMMAD ALBLAWI (KSA)**

 14:40 - 15:00


### **OUTCOME OF NEWLY DIAGNOSED CML PATIENTS NEWLY TREATED WITH FIRST OR SECOND GENERATION TKI**

 HANI ALHASHMI (KSA)

 15:00 - 15: 20

### **MOLECULAR MONITORING OF CML PATIENTS ON TKI AND APPROPRIATE TIME-DEPENDENT MOLECULAR TARGETS**

 AHMED ALSAGHEIR (KSA)

 15:20 - 15:40

### **CML: BETWEEN CYTOGENETICS AND MOLECULAR DIAGNOSTICS**


 AMEERA RADHI (BAHRAIN)

 15:40 - 15:50


### **DISCUSSION**



## WORKSHOP 4

 16:00 – 18:00

### HEMATOLOGY LAB WORKSHOP HEMATOLOGY LAB: ACCREDITATION ROADMAP FOR HEMATOLOGY LAB STAFF

 16:00 - 16:30

#### LABORATORY ACCREDITATION AS A PROJECT.

 OMAR QASSAS (KSA)


 16:30 - 17:00


#### CHALLENGES IN HEMATOLOGY CLINICAL LABORATORY.

 ASMA ALFETAYEH (KSA)

 17:00 – 17:30

#### QUALITY INDICATOR IN HEMATOLOGY LABORATORY.

 ABDULRAHEEM ALSHAREEF (KSA)

 17:30 – 18:00

#### PRACTICAL VALIDATION EXERCISE.

 ASMA ALFETAYEH (KSA)




# DAY 2- FEBRUARY 2, 2019 (SATURDAY)

## TRACK C


### SESSION 1C: SICKLE CELL ANEMIA

**MODERATORS: FATHIA ALQURASHI (BAHRAIN) - MUNEER ALBAGSHI (KSA)**


 10:30 - 10:50

#### **STEM CELL TRANSPLANT IN SCD**

 MOHSEN ALZAHIRANI (KSA)

 10:50 - 11:10

#### **NEWS THERAPIES IN SCD**

 SULTAN ALMOTAIRY (KSA)


 11:10 - 11:30

#### **HYDROXYUREA AND INFERTILITY**


 MOHSEN SALEH ELALFY (EGYPT)


### SESSION 2C: SICKLE CELL ANEMIA ORAL ABSTRACTS

**MODERATORS: HAFEZ MULHAN (KSA) - MURTADHA ALSULTAN (KSA)**

 11:30 - 11:45

#### **PREDICTORS AND RISK FACTORS OF CHRONIC PULMONARY DISEASE IN PEDIATRICS SICKLE CELL DISEASE**

 AZZA TANTAWY (EGYPT)

 11:45 - 12:00

#### **OXIDANT-ANTIOXIDANT STATUS IN SICKLE CELL DISEASE PATIENTS IN RELATION TO TRANS-CRANIAL DOPPLER (TCD) VELOCITIES**

 MONA EL-GHAMRAWY (EGYPT)

 12:00 - 12:15

#### **MALIGNANCY AND PRIMARY IMMUNODEFICIENCY DISEASES**

 SOHILLA LOTFY (EGYPT)



**PRAYER AND LUNCH**





## SESSION 3C: ACUTE MYELOID LEUKEMIA ORAL ABSTRACTS

**MODERATORS:** ~~FAIHA BAZZEH (JORDAN)~~ AMAL ABDULWAHAB (KSA)

⌚ 13:30 - 13:45

### NOVEL THYROINTEGRIN AVB3 ANTAGONIST FOR THE TREATMENT OF AML

🎤 SHAKER MUSA (USA)

⌚ 13:45 - 14:00

### HIGHER MTOR EXPRESSION IN AML: BAD NEWS

🎤 NAHLA OSMAN (EGYPT)

⌚ 14:00 - 14:15

### POLYMORPHISMS OF XERODERMA PIGMENTOSUM GENES ( XPC, XPD AND XPG) AND SUSCEPTIBILITY TO ACUTE LEUKEMIA AMONG A SAMPLE OF EGYPTIAN PATIENTS.

🎤 IMAN ABDELMOHSEN SHAHEEN (EGYPT)

## SESSION 4C: BLEEDING DISORDERS ORAL ABSTRACTS

**MODERATORS:** OHOUD KOSHARI (KSA) - NOUF ALNUMAIR (KSA)

⌚ 14:15 - 14:30

### IL-4RA GENE POLYMORPHISM IN CHRONIC IMMUNE THROMBOCYTOPENIA PATIENTS AND ITS RELATION TO DISEASE SUSUBILTY, SEVERITY AND RESPONSE TO TREATMENT

🎤 MOHAMED ABDELKADER MORAD (EGYPT)

⌚ 14:30 - 14:45

### SCREENING FOR INHIBITORS DEVELOPMENT AND IT'S RISK FACTORS IN PATIENTS WITH SEVERE HAEMOPHILIA A IN OMAN.

🎤 LAILA AL KHANBASHI (OMAN)

⌚ 14:45 - 15:00

### IMPACT OF THROMBOPHILIA ON THE RISK OF HYPOXIC ISCHEMIC ENCEPHALOPATHY IN TERM NEONATES

🎤 HANAN G. ABDELAZEEM (EGYPT)

⌚ 15:00 - 15:15

### HEMOPHILIA AND CHILDHOOD ACUTE LYMPHOBLASTIC LEUKEMIA

🎤 NESREEN ALI (EGYPT)



## DAY 3: FEBRUARY 3, 2019 (SUNDAY)

### " MERGE TRACK "

⌚ 07:30 - 08:00

#### WELCOMING AND INTRODUCTION

⌚ 08:00 - 08:40

**KEYNOTE LECTURE 3**  
**ICUS/CHIP/CCUS: WHAT DETERMINES A CORRECT DIAGNOSIS?**  
**RED**  
**WHEN TO TREAT AND WHEN NOT TREAT, OLDER PATIENTS WITH AML**  
**GULAM MOFTI (UK)** **OK**

### PLENARY SESSION 1 PHOENIX DACTYLIFERA PALM FRUIT (ALMADINAH AJWA)

⌚ 08:40 - 08:45

#### INTRODUCTION

🎤 MOHAMAD ZOLALI (KSA)

⌚ 08:45 - 09:05

#### AJWA IN HEMATOLOGY MEDICINE

🎤 SHAKER MUSA (USA)

⌚ 09:05 - 09:20

**EFFECTS OF PHOENIX DACTYLIFERA AS ANTIOXIDANT ON DISEASE COMPLICATIONS,  
SAFETY AND SURVIVAL AMONG PEDIATRIC CANCER PATIENTS IN KAUH:  
CONTROLLED STUDY OVER NINE YEARS**

🎤 SOUAD ALJAOUNI (KSA)

⌚ 09:20 - 10:00

**EHA / SSH / PAHA  
JOINT SESSION**

**HIGH THROUGHPUT FLOWCYTOMETRY FOR COMBINED MRD  
MEASUREMENTS & IMMUNE MONITORING**  
**ALBERTO ORFAO (SPAIN)**

**BREAK / ALMADINAH TRADITIONS**





# DAY 3: FEBRUARY 3, 2019 (SUNDAY)

## TRACK A

### SESSION 3A: THROMBOSIS AND ANTICOAGULANTS

**MODERATORS: GALILA ZAHER (KSA) - AZZA TANTAWI (EGYPT)**


 10:30 - 10:50


#### DOAC

 TURKEY ALSHOAIBY (KSA)

 10:50 - 11:10


#### ANTIPHOSPHOLIPID SYNDROMES

 BASEM BAIROTI (KSA)

 11:10 - 11:30


#### ANTI COAGULATION FOR PEDIATRIC

 MAHASSEN SALEH (KSA)

 11:30 - 11:50


#### MANAGEMENT OF ANTICOAGULANT DURING SPECIAL SITUATIONS COMPREHENSIVE THROMBOSIS CLINIC

 TAREK OWIDAH (KSA)

 11:50 - 12:10

#### THROMBOTIC THROMBOCYTOPENIC PURPURA (TTP)

 GALILA ZAHER (KSA)

 12:10 - 12:20

#### DISCUSSION




**PRAYER AND LUNCH**






## SESSION 4A: TRANSFUSION MEDICINE

**MODERATORS: MAHA BADAWI (KSA) - OSAMA FAIOMI (KSA)**

 13:00 - 13:20


### RESPIRATORY TRANSFUSION COMPLICATIONS

 AFRAA ALDAYEL (KSA)


 13:20 - 13:40


### TRANSFUSION MANAGEMENT OF THE PATIENT WITH AIHA

 MOHAMMED ALMOHAMMADI (KSA)


 13:40 - 14:00


### TRANSFUSION MANAGEMENT OF PATIENTS WITH HEMOGLOBINOPATHIES

 SANAA ABDULSHAFI (EGYPT)

 14:00 - 14:20

### PREVENTION AND MANAGEMENT OF PLATELET REFRACTORINESS

 NIVEEN ABDULLAH (JORDAN)

 14:20 - 14:30

### DISCUSSION




## PRAYER AND LUNCH




## SESSION 5A: TRANSFUSION MEDICINE ORAL ABSTRACTS

**MODERATORS: AHMED AL BAHRANI (KSA) - MOHAMED ALLAM (KSA)**

 14:30 - 14:45

### HEMATOLOGICAL CELLULAR ALTERATIONS IN PLATELETPHERESIS DONORS

 SANAA SHAKER ALI (EGYPT)


 14:45 - 15:00

### COMPATIBILITY STUDIES WITH PHENOTYPING TEST AT BLOOD BANK OF MATERNITY AND CHILDREN HOSPITAL, ALMADINAH, SAUDI ARABIA

 SANAA SHAKER ALI (EGYPT)

 15:00 - 15:15


### PEDIATRIC NON-MALIGNANT BLOOD DISORDERS REGISTRY: A ROBUST MODEL TO REPORT REGIONAL INCIDENCE AND OUTCOMES

 MAHASSEN ALSALEH (KSA)





## SESSION 6A: LYMPHOMA ORAL ABSTRACTS

**MODERATORS: HASSAN MASMALI (KSA) - AHLAM QATARI (KSA)**

 **15:15 - 15:30**

**BEAM VERSUS SINGLE AGENT HIGH DOSE MELPHALAN (HDM) CONDITIONING REGIMEN FOR AUTOLOGOUS HEMATOPOIETIC STEM CELL TRANSPLANT (ASCT): A RETROSPECTIVE MATCHED ANALYSIS IN RELAPSE/REFRACTORY HODGKIN LYMPHOMA.**

 **HANI AL HASHMI (KSA)**

 **15:30 - 15:45**

**GENETIC VARIATIONS IN TUMOR NECROSIS FACTOR-RELATED APOPTOSIS-INDUCING LIGAND-1 (TRAIL1) AND THE SUSCEPTIBILITY TO B CELL NON-HODGKIN LYMPHOMA IN EGYPT**

 **MERVAT KHORSHIED (EGYPT)**

 **15:45 - 16:00**

**EXCELLENT OUTCOME OF NODULAR LYMPHOCYTE PREDOMINANT HODGKIN LYMPHOMA IN THE EASTERN PROVINCE OF SAUDI ARABIA. A REAL-WORLD CASE SERIES OF 49 CONSECUTIVE PATIENTS TREATED AT A REFERRAL CENTER FROM 2006 TO 2017**

 **JOHN APOSTOLIDIS (KSA)**

 **16:00 - 16:15**

**PEDIATRIC ADVANCED STAGE HODGKIN LYMPHOMA TREATED IN DEVELOPING COUNTRIES; DOES RADIOTHERAPY IMPACTS SURVIVAL?**


 **HANAA RASHAD MAHMOUD (EGYPT)**



## TRACK B


### SESSION 5B: LYMPHOMA

**MODERATORS: ADNAN BAKARMAN (YAMEN) - SAUD BALILA (KSA)**

 10:30 - 10:50


#### **PRECISION THERAPY OF LYMPHOMA VIA GENOMICS**

 **ALI BAZERBASHI (LEBANON)**


 10:50 - 11:10


#### **LATEST ADVANCES IN IN DIAGNOSTIC AND TREATMENT OF DLBCL**

 **MUBARAK ALMANSOUR (KSA)**

 11:10 - 11:30


#### **PEDIATRIC APPROACH OF MANAGING HODGKIN LYMPHOMA**

 **ALI ALQRAQRI (KSA)**

 11:30 - 11:50

#### **CAN WE IMPROVE THE OUTCOME OF PATIENTS WITH PTCL**

 **ALI BAZERBASHI (LEBANON)**

 11:50 - 12:10

#### **THE TSUNAMI OF TARGETED THERAPY: ARE LABORATORIES READY FOR THE STORM**

 **SALEM KHALIL (KSA)**

 12:10 - 12:20

#### **DISCUSSION**



**LUNCH AND PRAYER**






## TRACK B


### SESSION 6B: ACUTE LYMPHOBLASTIC LEUKEMIA

**MODERATORS:** AZZA KAMEL (EGYPT) - ARWA ALYAMANI (KSA)

 13:00 - 13:20


#### UPDATE IN MANAGEMENT OF PEDIATRIC PATIENTS WITH PRE-B CELL ALL

 MOHAMMED ALESSA (KSA)

 13:20 - 13:40


#### UPDATE IN MANAGEMENT OF ADOLESCENTS AND YOUNG ADULTS (AYA) PATIENTS WITH ALL.

 AHMED ALABSSI (USA)

 13:40 – 14:00


#### RECENT ADVANCES IN THE BIOLOGY AND TREATMENT OF T CELL ALL

 MOSAB DAMLAJ (CANADA)

 14:00 – 14:20

#### FEVER NEUTROPENIA


 FAISAL KORDY (KSA)

 14:20 – 14: 30

#### DISCUSSION


### SESSION 7B: MISCELLANEOUS

**MODERATORS:** HADEEL SALEH (KUWAIT) - ABEER ABDULMONEM (KSA)

 14:30 – 14:50


#### PRIMARY FAMILIAL AND CONGENITAL POLYCYTHEMIA; THE FORGOTTEN ENTITY

 MANSOUR ALJABRY (KSA)

 14:50 – 15:10

#### HEMATOLOGICAL MANIFESTATIONS OF PRIMARY IMMUNODEFICIENCY DISORDERS

 AZZA TANTAWY (EGYPT)


 15:10 – 15:20

#### DISCUSSION



## SESSION 7B: THALASSEMIA DISORDERS ORAL ABSTRACTS


**MODERATORS: MORTHADA HUSSAIN (IRAQ) - ~~AMAL ABDULWAHAB (KSA)~~**

 **15:20 – 15:35**

**WAHEED TURKOSTANI (KSA)**


### **CLINICAL AND LABORATORY WORKUP OF A PATIENT WITH WHIM SYNDROME**

 **RABAB ELHAWARY (EGYPT)**

 **15:35 – 15:50**

### **ASSOCIATION BETWEEN GENOTYPE AND DISEASE COMPLICATIONS IN EGYPTIAN PATIENTS WITH BETA THALASSEMIA**

 **TAMER HASSAN (EGYPT)**

 **15:50 – 16:05**

### **EXPRESSION LEVEL OF PRO-APOPTOTIC GENES DETERMINE DISEASE SEVERITY OF HBE/BETA THALASSEMIA**

 **ROSLINE HASSAN (MALAYSIA)**



# DAY 3: FEBRUARY 3, 2019 (SUNDAY)

## TRACK C


### SESSION 5C: ACUTE LYMPHOBLASTIC LEUKEMIA ORAL ABSTRACTS

**MODERATORS: MOHAMED ALSHAHRANI (KSA) - MOHAMED SALEM (KSA)**

 **10:30 - 10:45**

**MINIMAL RESIDUAL DISEASE DETECTION IN ACUTE LEUKEMIA PATIENTS: COMPARATIVE**

 **EMAN KANDEEL**

 **10:45 - 11:00**


**LANDSCAPE OF CONVENTIONAL AND MOLECULAR CYTOGENETIC ABNORMALITIES AMONG NEWLY DIAGNOSED SAUDI ACUTE LYMPHOBLASTIC LEUKEMIA PATIENTS**

 **OSAMAH KHOJAH**


 **11:00 - 11:15**


**IMPACT OF THYMOQUINONE ON GENOMIC AND METABOLOMIC IN ACUTE LEUKEMIA CELLS**

 **ASMA ALGHAMDI**


 **11:15 - 11:30**


**CD27 AND CD44 EXPRESSION PATTERN IN PEDIATRIC PRECURSOR B-ALL: CLINICAL AND PROGNOSTIC IMPLICATIONS**

 **RANDA A.OSMAN**

 **11:30 - 11:45**

**OSTEONECROSIS IN CHILDREN WITH ACUTE LYMPHOBLASTIC LEUKAEMIA: A REPORT FROM CHILDREN'S CANCER HOSPITAL EGYPT (CCHE).**

 **NESREEN ALI**

 **11:45 - 12:00**

**MANAGEMENT OF ADULT ACUTE LYMPHOBLASTIC LEUKEMIA WITH A PEDIATRIC-BASED REGIMEN: A SINGLE CENTER EXPERIENCED**

 **NIHAD MOKHTAR (KSA)**

 **12:00 - 12:15**

**NEUROCOGNITIVE EFFECTS OF CENTRAL NERVOUS SYSTEM-DIRECTED CHEMOTHERAPY IN NON-HODGKIN LYMPHOMA DISEASED CHILDREN**

 **ABEER ABDELMONEIM (KSA)**



**PRAYER AND LUNCH**





# DAY 3: FEBRUARY 3, 2019 (SUNDAY)

## TRACK C

### SESSION 6C: THALASSEMIA DISORDERS

**MODERATORS: GALILAH MOKTAR (EGYPT) - ABDULHAKEEM ALRAWAS (OMAN)**

 13:00 - 13:20


#### STEM CELL TRANSPLANT IN THALASSEMIA VS GENE THERAPY

 ABDULLAH ALJEFRY (KSA)

 13:20 - 13:40

#### THALASSEMIA INTERMEDIA

 ALI ALMADHANI (OMAN)

 13:40 - 14:00


#### OXIDATIVE STRESS AND ANTIOXIDANTS IN B THALASSEMIA

 GALILAH MOKTAR (EGYPT)

 14:00 - 14:20


#### ENDOCRINOPATHIES IN THALASSEMIA

 ABDULHADI HABIB (UK)


 14:20 - 14:40


#### PREIMPLANTATION GENETIC DIAGNOSIS

 AMMAR ALHASSANI (KSA)

 14:40 - 15:00

#### THE NEW HORIZON IN THE MANAGEMENT OF THALASSEMIA

 AMAL EL-BESHLAWY (EGYPT)

 12:00 - 12:15

#### NEUROCOGNITIVE EFFECTS OF CENTRAL NERVOUS SYSTEM-DIRECTED CHEMOTHERAPY IN NON-HODGKIN LYMPHOMA DISEASED CHILDREN

 ABEER ABDELMONEIM (KSA)




**PRAYER AND LUNCH**






## SESSION 7C: THALASSEMIA DISORDERS ORAL ABSTRACTS

**MODERATORS: MOHSEN ELALFY - HUDA ALFARAIDI (KSA)**


 **15:00 – 15: 15**


**NEUROCOGNITIVE DYSFUNCTION IN CHILDREN WITH B THALASSEMIA MAJOR: PSYCHOMETRIC, NEUROPHYSIOLOGIC AND RADIOLOGIC EVALUATION.**

 **MOHSEN ELALFY (EGYPT)**

 **15:15 – 15: 30**

**LEARN THE SECRETS OF VITAMIN D IN B THALASSEMIA**

 **GALILA MOKHTAR(EGYPT)**

 **15:30 – 15: 45**

**SAFETY AND EFFICACY OF EARLY START OF IRON CHELATION THERAPY WITH DEFERIPRONE IN YOUNG CHILDREN NEWLY DIAGNOSED WITH TRANSFUSION-DEPENDENT THALASSEMIA**

 **GALILAH MOKTAR (EGYPT)**



**DAY 4- FEBRUARY 4, 2019 (MONDAY)**

**TRACK A**

**WORKSHOP 5**


**MEDICAL STUDENT AND RESIDENTS EDUCATIONAL PROGRAM (II)  
TOWARD BETTER UNDERSTANDING OF HEMATOLOGY CHAPTER**

**MDERETOR: BASHAYR SAAD ALHUBAYSHI**

 **08:00 - 08:30**


**APPROACH TO THROMBOCYTOPENIA**

 **ARWA ALYAMANI (KSA)**

 **08:30 - 09:00**

**ABCS IN BLOOD TRANSFUSION**

 **MAHA BADAWI (KSA)**

 **09:00 - 09:30**

**HOW TO READ BLOOD SMEAR/CBC INTERPRETATION**

 **MAHER ALJEHANI (KSA)**


 **09:30 - 10:00**

**DISCUSSION**

**BREAK / ALMADINAH TRADITIONS**




**MERGE TRACK**

 **10:30 - 11:00**

**KEYNOTE LECTURE 4**

**IMPACT OF NANOBIO TECHNOLOGY ON THE FUTURE OF MEDICINE (NANOMEDICINE):  
THE ROAD TOWARD PRECISION MEDICINE**


**SHAKER MUSA (USA)**

 **10:30 - 11:00**

**KEYNOTE LECTURE 5**

**ALMADINAH: HISTORICAL AND GEOGRAPHICAL PROSPECTIVE**

**ABDULLAH KABER (KSA)**

 **10:30 - 11:00**

**CONGRESS CEREMONY**



**PRAYER AND LUNCH**






**DAY 4- FEBRUARY 4, 2019 (MONDAY)**

**PLENARY SESSION 4**

**BEST OF MENA**


**MODERATORS:** AZZA KAMEL (EGYPT) - GALILAH MOKTAR (EGYPT)  
AZZA TANTAWY (EGYPT) - ABEER ABDULMONEM (KSA)

**FACILITATOR:** AHMAD TARAWAH (KSA)

 13:30 – 13:15

**INTRODUCTION**

 AHMAD TARAWAH (KSA)

 13:15 – 13:30


**CHILDHOOD CANCER EPIDEMIOLOGY AND OUTCOME WITH HIGHLIGHT ON  
ACUTE LYMPHOBLASTIC LEUKEMIA**

 TAHA KHATTAB (KSA)

 13:30 - 13: 45

**EVOLUTION OF ACCELERATED AND BLASTIC PHASES OF CHRONIC MYELOID LEUKEMIA:  
MOLECULAR, CYTOGENETIC, FLOWCYTOMETRIC AND ELECTRONMICROSCOPIC STUDIES**

 FAYEK GHALEB (EGYPT)

 13:45 - 14: 00

**OUTCOME OF AGE-ADAPTED APPROACH TO HLA-IDENTICAL RELATED  
HEMATOPOIETIC STEM CELL TRANSPLANTATION IN SEVERE SICKLE CELL DISEASE:  
SAUDI EXPERIENCE**

 MOHSEN ALZAHRANI (KSA)

**WORKSHOP 6**

**MEDICAL STUDENT AND RESIDENTS EDUCATIONAL PROGRAM (III)  
TOWARD BETTER UNDERSTANDING OF HEMATOLOGY CHAPTER**

**MODERATOR: ISRAA ISMAIL AL-TURKESTANY**

 14:00 - 14: 30

**APPROACH TO THROMBOSIS**

 ASHRAF WARSY (KSA)

 14:30 - 15: 00


**USE AND CHOICE OF BLOOD AND BLOOD COMPONENTS**

 SABRI KEMAHLI (TURKEY)

 15:00 - 15:30

**BLOOD COMPONENT THERAPY IN COAGULATION DISORDERS**

 SABRI KEMAHLI (TURKEY)

 15:30

**DISCUSSION**



**DAY 4- FEBRUARY 4, 2019 (MONDAY)**

**TRACK B**


**PLENARY SESSION 2  
CUTANEOUS LYMPHOMA**

**MODERATORS: HATEM ALWASIEDY (KSA) - OSAMA ALSHARIF (KSA)**

 **08:00 - 08:20**

**CUTANEOUS LYMPHOMAS: CLINICAL PICTURE, DIAGNOSTIC AND TREATMENT**

 **PERCI LEHMANN (GERMANY)**


 **08:20 - 08:40**

**PHOTOTHERAPY: PHOTOCHEMOTHERAPY (PUVA) IN THE TREATMENT OF CUTANEOUS LYMPHOMAS**

 **PERCI LEHMANN (GERMANY)**


**SESSION 8B: CLL / SCT**

**MODERATORS: TALAL ALHARBI (KSA) - RANDA ALNOUNOU (KSA)**

 **08:40 - 09:00**


**HOW TO MANAGE RELAPSED CLL**

 **AHMED ALASKER (KSA)**

 **09:00 - 09:20**


**CURRENT STRATEGIES TO PREVENT AND TREAT GVHD IN ALLO HCT**

 **SHAROKH HASHMI (KSA)**

 **09:20 - 09:40**

**LOCAL EXPERIENCE FOR SC DONATION REGISTRY**

 **MOHSEN ALZAHRANI(KSA)**

 **09:40 - 10:00**

**BMT UPDATE IN MDS**

 **GULAM MOFTI (UK)**

 **10:00 - 10:05**

**DISCUSSION**

**BREAK / ALMADINAH TRADITIONS**





## MERGE SESSIONS


 11:30 – 12:30

### CONGRESS CEREMONY




#### PRAYER AND LUNCH



 13:30 – 16:00


### HEMATOLOGY NURSES WORKSHOP

**MAHMUD ABURAIASH / KHALED HABAIBAH**

 13:30 – 14:00


#### VON WILLBRAND DISEASE

 KHALID HABAIBAH

 14:00 – 14:30


#### ASSESSMENT AND MANAGEMENT OF BLEEDING IN HEMOPHILIA

 MAHMOUD ABURAIASH

 14:30 – 15:00

#### CHALLENGES IN HEMOPHILIA TREATMENT

 KHALID HABAIBAH

 15:00 – 15:30

#### MANAGEMENT OF ARTHROPATHY IN BLEEDING DISORDERS

 MAHMOUD ABURAIASH

## DISCUSSION



**DAY 4- FEBRUARY 4, 2019 (MONDAY)**

**TRACK C**


**PLENARY SESSION 3  
IRON DEFICIENCY SYNDROMES**

**MODERATORS: ZAKARIA ALHAWSAWI (KSA) - WAHEED TURKOSTANI (KSA)**

 **08:00 - 08:05**


**INTRODUCTION**

 **ZAKARIA ALHAWSAWI (KSA)**

 **08:05 - 08:25**


**IRON DEFICIENCY SYNDROMES**

 **TAREK OWIEDAH**

 **08:25 - 08:45**

**GENETIC MANIPULATION OF IRON METABOLISM**

 **HANAN HAMED (EGYPT)**

 **08:45 - 09:00**

**IRON DEFICIENCY ANEMIA IN POST BARIATRIC SURGERIES PATIENTS**

 **SAAD ALMOTIRI (KSA)**


**IRON DEFICIENCY SYNDROMES ORAL ABSTRACTS**

**MODERATORS: AMAL EL-BESHLAWY (EGYPT) - MOHAMMAD DARWISH (KSA)**


 **09:00 - 09:15**

**IRON DEFICIENCY ANEMIA: MADINAH SCREENING PROGRAM**

 **ZAKARIA ALHAWSAWI (KSA)**

 **09:15 - 09:30**


**PREVALENCE OF ANEMIA AMONG CHILDREN AGED 6 MONTHS - 12 YEARS ATTENDING  
EMERGENCY ROOM IN PRINCESS RAHMA TEACHING HOSPITAL FOR CHILDREN,  
NORTH OF JORDAN**

 **KHALED SHALBY**

 **09:30 - 09:45**

**ROLE OF SERUM HEPCIDIN LEVELS IN THE DIAGNOSIS OF IRON DEFICIENCY ANEMIA IN  
CHILDREN IN SAUDI ARABIA**

 **ZAKARIA ALHAWSAWI (KSA)**

 **09:45 - 10:00**


**DISCUSSION**

**BREAK / ALMADINAH TRADITIONS**





## MERGE SESSIONS

 11:30 – 12:30

### CONGRESS CEREMONY




#### PRAYER AND LUNCH




### SESSION 8C: CHRONIC MYELOID LEUKEMIA / MYELOMA ORAL ABSTRACTS

**MODERATORS: EMAN KHATTAB (JORDAN) - FATMA ALBATNIJI (KSA)**

 13:30 – 13:45

**CLINICAL DIFFERENCES IN CHRONIC MYELOID LEUKEMIA PRESENTATION IN PATIENTS OF KHYBER PUKHTOON KHWA PROVINCE, PAKISTAN**

 **SHAHTAJ KHAN (PAKISTAN)**

 13:45 – 14:00

**GENETIC CHARACTERISTIC OF 123 NEWLY DIAGNOSED PLASMA CELL MYELOMA PATIENTS: A FIVE YEARS' EXPERIENCE OF A SINGLE ONCOLOGY CENTER**

 **OSAMA KHOJAH (KSA)**


 14:00 – 14:15

**CONCORDANCE BETWEEN CONVENTIONAL AND MOLECULAR CYTOGENETIC TECHNIQUES IN IDENTIFICATION OF GENETIC ABNORMALITIES AMONG NEWLY DIAGNOSED MULTIPLE MYELOMA PATIENTS**

 **OSAMA KHOJAH (KSA)**

### SESSION 9C: MISCELLANEOUS ORAL ABSTRACTS

**MODERATORS: NABILA AL-BAZ (KSA) - SAMI ALBATTAT (KSA)**

 14:15 – 14:30


**PRESEPSIN AS A DIAGNOSTIC MARKER OF BACTERIAL INFECTIONS IN FEBRILE NEUTROPENIC PEDIATRIC PATIENTS WITH HEMATOLOGICAL MALIGNANCIES**

 **MARWA ZAKARIA (EGYPT)**

 14:30 – 14:45

**IMPACT OF IRON DEFICIENCY ANEMIA ON THE FUNCTION OF THE IMMUNE SYSTEM IN CHILDREN**

 **TAMER HASSAN (EGYPT)**

 14:45 – 15:00

**HEMATOGONES AS A PROGNOSTIC INDICATOR IN ALLOGENEIC HEMATOPOIETIC STEM CELL TRANSPLANTATION IN SEVERE APLASTIC ANEMIA, SINGLE CENTER EXPERIENCE**

 **DOUAA SAYED (EGYPT)**





# **ABSTRACTS**



## Multi-Modal Mechanisms of Novel Sulfated Non-Anticoagulant Low Molecular Weight Heparin in Sickle Cell Disease

Dr. Shaker A. Mousa, PHD, MBA, FACC, FACB



*Professor of Pharmacology, Endowed Chair Executive Vice President and Chairman, The Pharmaceutical Research Institute (PRI) & President of R & D at Vascular Vision Pharmaceuticals Co.*

*Dr. Mousa held a senior principal research scientist and fellow at DuPont and DuPont Merck Pharmaceuticals for over 17 years. He is also a Visiting Scholar at the Johns Hopkins University and holds academic appointments of Adjunct Professor in the State University of New York, and Rensselaer*

*Polytechnic Institute. He is tenured and Endowed Professor of Pharmacology, Vice Provost at Albany College of Pharmacy and Health Sciences, Executive Vice President and Chairman of the Pharmaceutical Research Institute (PRI), President of R & D at vascular Vision Pharmaceuticals Co. and founder of a number of startup biotechnology and Pharmaceutical companies. His work has been reported in over 1,000 peer reviewed publications and holds over 350 US and International Patents*

The pathogenesis of Sickle Cell Disease (SCD) comprises a complex interplay of factors associated with vascular endothelial activation, intense inflammation, and increased sickle cell adhesion. Microvascular occlusion in SCD is initiated by adhesion of sickle red blood cells (RBCs) to the endothelium, leading to acute painful vasoocclusive crisis (VOC) and clinical morbidity. Current treatment strategies remain suboptimal and are limited by significant side effects. The inherent complexity of SCD makes it unlikely that a single therapeutic strategy will be universally beneficial. We have previously shown that the low molecular weight heparin (LMWH) tinzaparin significantly shortened both duration of VOC crisis and hospitalizations by ~40%, and resulted in statistically significant and rapid reduction of pain). However, safety concerns associated with the narrow therapeutic index (bleeding risks) of LMWH are a major barrier to dose escalation/optimization of treatments.

We have developed a novel sulfated non-anticoagulant LMWH, named S-NACH, with an extensive range of bioactivities that would constitute a multi-modal approach to management of SCD. We generated and significantly optimized S-NACH for VOC to: 1) exert its beneficial activities without causing hemostatic (bleeding) side effects that are associated with the clinical use of LMWHs; and 2) incorporate an additional, potent direct anti-sickling property besides its anti-selectin and anti-inflammatory activities.

We conducted *in vitro* and *in vivo* investigations on the efficacy of S-NACH on the biophysical properties of RBCs. For the *in vitro* study, 21 subjects comprising 12 SCD patients

37°C for 1.5 h, in the absence (control) or presence of 1, 5, or 10 ug/mL of S-NACH or LMWH. For the *in vivo* study, we obtained pre-treatment samples from Townes' SCD mice (n=6 mice/treatment group) and treated the mice subcutaneously with PBS or 30-100 mg/kg S-NACH. Two hours after treatment, blood samples were evaluated for the percentage of sickled cells in pre- and post-administration samples using Leishman's stain and wet smears.

Incubation with S-NACH *in vitro* under hypoxia showed a dose-dependent, significant inhibition of sickling (up to 80%) in samples from all subjects while LMWH showed no anti-sickling effect. S-NACH had no effect on the osmotic fragility of both AA and SS RBCs. Importantly; we observed a 40-50% decrease in levels of circulating sickled cells in treated SCD mice, an effect that persisted for up to 6 h. Our *in vitro* studies show that the direct anti-sickling effect is partly due to dose-dependent modification of Hb S to the high-affinity adduct form and the corresponding increase in oxygen affinity, as demonstrated with cation HPLC and oxygen equilibrium analyses. Summarily, our previous findings showed the efficacy of S-NACH as anti-adhesive and anti-inflammatory in SCD, and our current results demonstrate the direct anti-polymerization action of S-NACH on sickle RBCs. Our data document for the first time the supplemental direct anti-sickling effects of a novel S-NACH derivative, suggesting a rational mode of action for these effects and make a compelling case for future studies. Planned detailed structural studies of our S-NACH derivatives complexed with Hb are expected to further illuminate the anti-sickling properties. Our novel Nanoformulated S-NACH



## Emerging therapies for hemophilia : global prospective

Glenn F. Pierce, MD PhD



*World federation of hemophilia*

*Montreal, Canada*

Progress in hemophilia pharmacotherapy has been unprecedented the past 60 years, since the development of cryoprecipitate and clotting factor concentrates. Improving viral safety emerged as the top priority in the 1980s-90s. More recently, breakthroughs in protein engineering have enabled the development of "improved" clotting factors, biobetters. These include higher specific activity variants and use of chemical conjugation or gene fusion to create extended half-life (EHL) Factors VIII and IX. While Factor IX has achieved half-life extensions that permit dosing every 1-3 weeks, the best FVIII half-life extension (1.5x) has been limited by dependence on von Willebrand factor (VWF) in the circulation. When VWF is metabolized ( $t_{1/2} \sim 19$  hours), Factor VIII, included the EHL Factor VIII, is also removed from the circulation. This limitation has been overcome with the creation of a fusion protein containing the D'D3 region of VWF to prevent binding of the EHL-modified FVIII to endogenous VWF, increasing its half-life 3-4x in a small Phase 1/2 clinical trial.

A bispecific antibody has recently been approved for use in all persons with Hemophilia A in some countries, including those with inhibitors. The two arms of this antibody, emicizumab, bind Factors IX and X, thus replacing the function of Factor VIII to create a Tenase complex and the required thrombin burst. A recent Phase 3 study demonstrated superiority of emicizumab over prophylaxis with FVIII, with regard to bleeding events. It may dose subcutaneously every 1-4 weeks, a further advantage. Other "non-factor" replacement therapies that are administered subcutaneously are in clinical trials, including 3 anti-TFPI antibodies to neutralize TFPI and its role in dampening the initiation of coagulation. Fitusiran, an inhibitory RNA for antithrombin is also in Phase 3 clinical testing to assess whether decreased levels of antithrombin, which inactivates thrombin and other proteases, will compensate for Factor VIII or IX deficiency. Finally, gene therapy to replace the defective or missing Factor VIII or IX gene is well underway, with one Phase 3 trial for Hemophilia A and two Phase 3 trials for Hemophilia B ongoing. These trials are now achieving sufficient levels of clotting factor to convert severe phenotypes to mild (5-40%) or normal (>50%).



## Women and Bleeding Disorders

Claire McLintock, MD



*Obstetric Physician and Haematologist, National Womens' Health. Auckland City Hospital, President of the International Society on Thrombosis and Haemostasis*

*Auckland, New Zealand*

While it is recognized that males are affected by bleeding disorders such as haemophilia, less attention is directed towards manifestation of bleeding disorders in girls and women. Girls and women face unique haemostatic challenges in life, including menstruation, pregnancy and childbirth that may unmask clinically significant bleeding disorders. Careful clinical assessment is required so that bleeding disorders are recognized and treated appropriately during pregnancy and at birth to ensure safety for mother and child.

### Educational Objectives of Talk

- Summarize clinical presentation and genetics of bleeding disorders and their manifestation in girls and women
- Develop clinical approach to identify women who may have bleeding disorders and appropriate diagnostic testing
- Identify clear management pathway for care during pregnancy and during labour and birth for women with bleeding disorders including:
  - approaches to management to minimize women's risk of bleeding, especially of postpartum haemorrhage
  - development of management plan to optimize birth outcomes for infants with inherited bleeding disorders





## **Transfusion Medicine Education**

Sabri Kemahli, MD

*Professor of Pediatrics and Pediatric Hematology, Faculty of Medicine, Yeditepe University, Istanbul, Turkey*

*Professor of Pediatrics, College of Medicine, Al Faisal University, Saudi Arabia*

*Secretary General, European-African Division, International Society of Hematology*

*National Councillor, International Society of Hematology (representing Turkish Society of Hematology)*



# Hemophilia Inhibitors

Kaan Kavakli, MD



Professor of Pediatric and Pediatric Hematology, Ege University Children's Hospital, Turkey

*Dr. Kaan has held the position of Chairman of the Department of Paediatric Haematology at Ege University Children's Hospital in Izmir, Turkey. He was also appointed as Director of the Ege Haemophilia Centre and Ege Haemophilia Association in 2000, and General Director of the Haemophilia Federation (of Turkey). Dr. Kavakli had also held the posts of Chairman of both the Ethics Committee of Ege University Medical Faculty and the Haemostasis and Thrombosis Subcommittee of the Turkish Paediatric Haematology Society, for over 10 years. Now he is responsible for chairman of Hemophilia Subcommittee for Turkish Hematology Association.*

*Dr. Kavakli has more than 100 publications and he is a member of numerous international societies and a reviewer for the journals; Journal of Thrombosis and Haemostasis. He is also an Editorial Board member of the Haemophilia Journal (2010–2018).*

Inhibitor development is the most important treatment complication of Hemophilia-A (HA) therapy. Incidence of inhibitor in Previously Untreated patients (PUP) for HA is approximately 30% whereas Hemophilia-B (HB) patients have 3% inhibitor risk. Inhibitor concept means a neutralizing anti-FVIII antibody against for FVIII protein in therapy. It is an allo-antibody and not auto-immune disorder in hemophilia patient. 1 Bethesda Unit of inhibitor activity is defined as 100% FVIII activity decreased to 50% FVIII activity in patients' plasma.

In severe HA, inhibitor screening tests must be done in every 6 months or annually at least. Other HA patients and in HB patients, annual tests are enough. However, in PUP period in childhood; inhibitor test should be done 5 times at least before completing 50 exposure days with FVIII.

More than 5 BU/ml of inhibitor activity and anamnesis after FVIII therapy denote High Responder (HR) inhibitors. HR inhibitors are together with clinically bleeds and hemophiliac arthropathic complications.

The most gold-standard inhibitor test is Nijmegen-Bethesda Test (NBT). In classic Bethesda test, patients' plasma is diluted with imidazol buffer and normal plasma pool is not buffered. In NBT test, immune depleted FVIII is used for plasma dilutions and bufferization is started with normal pool plasma together with control-plasma. If patient recently had to be used FVIII therapy before inhibitor test, heating procedure is recommended with 56 C degrees for 30 minutes.

Treatment of bleeding and surgery are provided by by-passing agents (recombinant FVIIa and activated PCC). Secondary prophylaxis is also possible for preventing bleedings and arthropathy.



# Hemophilia in Morocco

Mohamed ElKhorassani, MD



*Dr Mohamed El Khorassani*

*Professor of medecine at Mohamed V University*

*Pediatrician, Hematology and Oncology specialist*

*Dr. El Khorassani is a professor at Mohamed V University, specialist pediatrician in hematology and oncology, and referring physician in haemophilia and bleeding deasorders, in public practice at the hematology and oncology center, Children hospital, unversity hopsital. Rabat.*

*Dr El Khorassani is the author of “Guide de prise en charge de l’hémophilie au Maroc”, a manual that describes the guidelines of haemophilia treatment in Morocco and he is the principal investigator of several trial studies*

## Role of Imaging in the assessment & management of hemophilic Arthropathy

*Alhaosawi , Mousa Mohammad Thalth ,MD*



*Dr.Alhaosawi is a Clinical Assistant professor of orthopaedic ( Taiba university, Al-madinah ),he is General Director of almadinah specialty hospital, Head of examination committee of Saudi orthopedic board , A.O International Lecturer Faculty of Trauma surgery*

*Dr Alhaosawi is an Examiner in orthopedic Saudi Board ,External examiner in orthopedic surgery*

*He isHead of committee for Saudi Casting Certificate , Head of committee for critical Courses for Nursing in Ministry of Health , Supervisor of critical Courses for Nursing in Al-madinah region and a Member of Saudi Doping committee of Saudi football federation*

Hemophilic arthropathy (HA) contributes the greatest morbidity and cost in the hemophilic population. Imaging plays a crucial role in accurately monitoring the disease process in all phases and evaluating treatment. It has been shown that prophylactic factor VIII replacement therapy, if given early in life, can prevent or delay the progression of

HA and reduce the frequency of hemarthroses. The purpose of this talk is to evaluate the role of symptomatology and conventional radiographic scoring in predicting synovial hypertrophy, which could affect the clinical management of hemophilic patients.



## Factor VII Deficiency in Lebanon, from Mild to Severe: Clinical Phenotype and Molecular Studies

Roula A. Farah, M.D, FAAP



*Dr Farah is an Associate Professor of Pediatrics and Pediatric Hematology/Oncology at the University of Balamand/Saint-George Hospital University Medical Center in Beirut.*

*She is Board certified by the American Boards of Pediatrics and Pediatric Hematology/Oncology and is a Fellow of the American Academy of Pediatrics.*

*Dr. Farah obtained her medical degree from St Joseph University of Beirut. She completed her pediatric internship at Columbia University in New York, and her residency at Albert Einstein College of Medicine in New York. Dr. Farah has several publications in the field of pediatric hematology/oncology and is an active member of several professional organizations such as the American Society of Hematology (ASH), the International Society of Pediatric Oncology (SIOP), the International Society of Thrombosis and Hemostasis (ISTH) and the International Histiocyte Society.*

*She served as a president of the Lebanese Pediatric Hematology/Oncology Group and is currently on the executive committees of the Lebanese Cancer Society and the Lebanese Society of Pediatrics.*

**Background:** Congenital deficiency of Factor VII is rare but is the most common autosomal recessive hemorrhagic disorder. The clinical phenotype varies from asymptomatic forms to lethal hemorrhagic diathesis characterized by central nervous system (CNS) and gastro-intestinal (GI) hemorrhages occurring commonly during the neonatal period. Severe phenotypes account for 10-15% of symptomatic FVII deficient patients. Some mutations are consistently associated to the severe clinical phenotype, mostly specific missense and invariant AG or GT splice site mutations. Mild and moderate cases can also have bleeding, while some patients with very low factor levels do not bleed. There is little data on the spectrum of mutations and clinical phenotype in Lebanese patients.

**Methods and Aims:** We reviewed medical records of several Lebanese children with factor VII deficiency, from mild to severe, looking at the bleeding patterns observed, the family history,

F7:c.291+1G>C splice site mutation known to be associated with life-threatening bleeding phenotype, and others had milder phenotypes and various other mutations. These five children were originating from four different families and presented as a first symptom with GI bleeds (n=4) or CNS bleed (n=1) within the first two months of life (range Day 1 to Day 40 of life). Prophylaxis with Recombinant factor VII two or three times per week was implemented in these families with positive impact on morbidity and mortality.

Two other children had Factor VII levels below 3% but had other mutations and a milder phenotype and were only treated with recombinant Factor VII on demand.

The other patients had various phenotypes and higher levels of factor VII.

**Conclusions:** A spectrum of clinical phenotypes of factor VII deficient patients was observed in Lebanon. Severe variants carrying the same



# Overview of clinical assessment diagnosis and management of von Willebrand Disease (VWD)

Magdy El Ekiaby, MD



*Dr El Ekiaby has served the haemophilia community in Egypt since 1985, both through his profession as a haematologist and blood transfusion specialist, and as a member of the board and Vice President of the Egyptian Society of Hemophilia. He is also a Board of Directors of World Federation of Hemophilia, Montreal. Dr El Ekiaby is currently the Head of Blood Transfusion & Hemophilia Centre, Shabrawishi Hospital, a position held since 1983. Dr Magdy received World Federation of Hemophilia Inga-Marie Nielson Award for Scientific Research achievements in Melbourne 2014. He was also awarded Life Time Achievement Award from Arab Transfusion Medicine Forum in Cairo 2015.*

**Von Willebrand Factor (VWF):** It is a large multimeric protein which is encoded by a gene on the short arm of chromosome 12 and is expressed by two types of cells namely endothelial cells and megakaryocytes. The main functions of VWF is facilitation of platelet adhesion to sub-endothelial sites of vascular injury, platelet aggregation and binding FVIII which protects the later from premature clearance from circulation.

**Von Willebrand Disease:** It can be caused by either quantitative deficiency or qualitative dysfunction of VWF. It is classified as quantitative deficiency (type 1 & 3) or dysfunctional type 2 which is further sub-classified to types 2A, 2B, 2M and 2N. The mode of inheritance is different according to the type of VWD where it is autosomal dominant for all types except for types 2N and 3 which are inherited as autosomal recessive type[1]. Prevalence of VWD with bleeding symptoms is estimated to be 1/10,000 population. Bleeding symptoms are mainly mucocutaneous bleeds, epistaxis, oral bleeding as well as post-operative and postpartum bleeds. Musculoskeletal bleeding can happen with type 3 when FVIII levels are very low [2].

The severity of bleeding symptoms are variable and do not correlate consistently with the quantitative or qualitative VWF levels and were rather found correlating with bleeding scores; the higher the score the more frequent bleeding can happen

**Diagnosis of VWD:** Family history and objective evaluation of bleeding symptoms and their severity using bleeding assessment tools are important elements for the diagnosis. Laboratory diagnosis is complex, expensive and mainly rely on determination of VWF antigen and activity as well as FVIII level. Many factors can lead to variation in the levels of VWF such as blood groups and others. Other tests may be necessary for sub-types. DDAVP infusion test can be a rapid clinical test to identify VWD

**Management of VWD:** VWF levels below 30% are diagnostic of bleeding VWD. Types 1 and 2 (except 2B) may benefit from DDAVP infusion, unless contraindicated or found unresponsive. FVIII concentrates containing VWF, purified VWF concentrates and recently available recombinant VWF are mainly used to treat bleeding episodes and may also be used for prophylactic therapy where indicated



## UPDATE IN REPLACEMENT THERAPY IN VON WILLEBRAND DISEASE

Kaan Kavakli, MD



Professor of Pediatric and Pediatric Hematology, Ege University Children's Hospital, Turkey

von Willebrand disease (vWD) is the most frequent congenital bleeding disorder. Unlike hemophilia, vWD is together with males and females. Type-1 vWD has mild character and rarely needs replacement therapy as most frequent subtype in vWD. DDAVP is mostly used for treatment of bleedings together with Tranexamic acid pills in patients.

vWF products generally used for Type-2 and Type-3 patients. However pure vWF products are not available in most countries. Wilfactin (LFB), Wilate (Octafarma) and Haemate-P (Behring), Fandhi (Grifols),

Factor 8Y (BPL) are the most preferred vWF products in Western countries. These products contain FVIII protein together with different amounts of vWF except Wilfactin.

The latest treatment modality is recombinant vWF product. Vonvendi (Shire) has been recently approved by FDA and then EMA for episodic treatment and surgery for adult patients with vWD. This product contains enough amount of HMW-Multimers. Recombinant products are now in phase-3 clinical trials for prophylaxis in adults' patients and episodic treatments for children and babies. Up to day, efficacy and safety reports related recombinant product were very good.



## Rare Bleeding Disorders, Diagnosis and Managements

Flora Peyvandi



*Dr. Peyvandi is Professor of Internal Medicine at the University of Milan and the Director of the Angelo Bianchi Bonomi Hemophilia and Thrombosis Centre, Fondazione IRCCS Ca' Granda, Ospedale Maggiore Policlinico, Milan, Italy. Dr Peyvandi's basic and medical science research has focussed on the investigation of coagulation disorders. Dr. Peyvandi has contributed extensively to the study of*

*RBDs which were rarely diagnosed and poorly characterised due to their low frequency. She has published extensively on this group of disorders and developed international registries with the aim of harmonising internationally the diagnosis and classification of RBDs. Dr. Peyvandi has authored and co-authored more than 400 scientific publications that have been published. Since 1999, she has been invited as an expert speaker at more than 140 national and international meetings and congresses. She has been the successful recipient of more than 40 project grants funded by Italian and International organisations. she was Chair of the International Society of Thrombosis and Haemostasis (ISTH) Scientific and Standardization Committee on Factor VIII, Factor IX and Rare Coagulation Disorders. She is member of the ISTH council, member of the Executive Committee of the World Federation of Hemophilia (WFH), member of the executive committee of the European Association for Haemophilia and Allied Disorders (EAHAD) and member of the Medical Advisory Group of the European Hemophilia Consortium (EHC). In 2014 she was awarded the "Great Hippocrates" that is delivered to Italian medical researcher of the year.*

## Gaucher Disease

Ayman Hijazi, MD



*Dr. Ayman Alhejazi is a Consultant and currently the Section Head of Adult Hematology, Department of Oncology at King Abdulaziz Medical City-Riyadh, Saudi Arabia. He is also an Assistant Professor of Hematology at King Saud bin Abdulaziz University for Health Sciences in Riyadh.*

*Dr Alhejazi obtained his Internal Medicine Board in 2002 and subsequently attained Fellowships in oncology, haematology, and blood and marrow transplantation from the University of Ottawa, Ontario, Canada. Dr Alhejazi has published numerous publications in peer-reviewed journals and abstracts at international meetings.*



## Inherited aplastic anemia in children and adolescents

Saad Ahmed Al Daama, MD



*Dr. Saad is Consultant of Pediatric Hematology/Oncology & Stem Cell Transplant at King Fahad Specialist Hospital-Dammam.*

*Dr Saad has Diploma in health leadership from George Washington University*

*He did his Fellowship in Pediatric Hematology, Oncology and Stem Cell Transplantation from*

*University of Toronto*

*KFSH&RC and University of Toronto.*

*Published more than 30 publication in pediatric Hematology, oncology and HSCT*

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Aplastic anemia (AA) is a rare disorder characterized by pancytopenia and a hypocellular bone marrow. AA can result from either inherited or acquired causes. The incidence is triphasic, with one peak in childhood at two to five years (due to inherited causes), and two peaks in adulthood, 20 to 25 years and the majority of patients presenting beyond 55 to 60 years of age (typically due to acquired causes). The associated neutropenia and thrombocytopenia can lead to potentially life-threatening infections and bleeding. Chronic red cell transfusion therapy for the associated anemia can lead iron overload, which, if not treated, can lead to significant morbidity and mortality.

The majority of children (75%) with inherited bone marrow failure have an identifiable etiology. The major inherited causes of AA in children are:

1 Fanconi anemia (FA)

2 Dyskeratosis congenita (DC)

3 Shwachman-Diamond syndrome (SDS)

Congenital amegakaryocytic thrombocytopenia (CAMT)

5 Diamond-Blackfan anemia (DBA)

6 Congenital neutropenia such as Kostmann Syndrome (KS)

These syndromes may present with or without physical anomalies and do not necessarily demonstrate complete pancytopenia, particularly during the early phases of disease. Acquired AA, which accounts for most cases of AA in children, is a disorder that often responds to immunosuppressive therapy. In contrast, immunosuppressive therapy is not indicated in the inherited syndromes.

The four major causes of inherited BMF are FA, DC, SDS and CAMT. DBA and KS are single line bone marrow failure syndrome. Having said that, a few cases of aplastic anemia in patients with Diamond-Blackfan anemia have also been reported.

An overview of inherited aplastic anemia in children including CAMT, FA, DC, SDS, DBF, KS and others are going to be summarized in this lecture.



# GENETIC TESTING IN MPNS: THE CONVENTIONAL & BYOUND

Osamah T. Khojah, M.D., M.Sc., IFCAP



*Dr. Osama is Consultant of Hematopathology & Blood Transfusion, KSUMC*

*Dr Osama is the Director of the Clinical Pathology program, KSU as well as the Medical Lab Director, Sultan Bin Abdulaziz Humanitarian City.*

*Dr. Osama got Master in Medicine "Infectious Disease and Immunity", the University of Sydney: 2010, Master in Science "Immunogenic", Oxford University: 2012, Fellowship in Hematopathology and Blood Transfusion, KSU: 2016, International Fellowship of College of American Pathologists (IFCAP), 2016 and Advanced certificate in Cellular Therapy by*

*AABB, May 2018.*

*He published around 15 researches in Immunology, Hematology and Genetic fields with 3 scientific awards*

Since Dr. William Dameshek introduced in the Blood journal in 1951 the concept of myeloproliferative disorders as conditions characterized by excessive proliferation of hematopoietic precursors in the bone marrow and excessive production of mature blood cells as an editorial article entitled "Some speculations on the myeloproliferative syndromes", these conditions have been evolved and categorized more with the plethora of scientific observations and proper utilization of laboratory advancement, genetics in particular.

In 2008, to underscore the clonal nature of myeloproliferative disorders, the authors of the *WHO Classification of Tumours of Haematopoietic and Lymphoid Tissues* introduced the name "myeloproliferative neoplasms" (MPNs). The revised version of this classification includes the following MPNs: chronic myeloid leukemia (CML), *BCR-ABL1*<sup>+</sup>; chronic neutrophilic leukemia (CNL); polycythemia vera (PV); primary myelofibrosis (PMF); essential thrombocythemia (ET); chronic eosinophilic leukemia, not otherwise specified; and MPN, unclassifiable.

Over recent years, there have been tremendous advances in our understanding of the genetic basis of MPNs and related myeloid neoplasms. Milestones in this field include the following discoveries: the *FIP1L1-PDGFR* fusion gene in 2003; the unique *JAK2* (V617F) mutation in 2005; oncogenic *CSF3R* mutations in 2013; and somatic mutations of *CALR* in classical MPNs in 2013. Therefore, the current criteria of any MPNs must include; evidence of proliferation (cytosis), morphological features for peripheral blood and/or bone marrow and genetic testing to identify clonal evidence in one of the key genes implicate in the MPN pathogenicity.

In this "Hematopathology Review" meeting, it is wise to address the critical and latest updates in the genetic testing related to MPNs arena, diagnosis, prognosis and overall management. The talk will address three questions;

- What is the current best practice towards an MPN-suspected case in term of genetic testing?
  - What are the main pitfalls of depending solely on genetic testing when a diagnosis of MPNs is investigated.
  - Is there a clear role of genetic testing in the MPNs' prognosis and general management?
- Finally, some excellent local published and uniquely unpublished experiences related to molecular findings in our MPN patients will likely be presented.



## Prognostic factors in multiple myeloma

Majed Alahmadi, MD

*Dr. Majed Alahmadi, received his medical degree from King Abdulaziz University. Then, completed residency training at Western University, Canada in Internal Medicine and Hematology. That followed by a clinical fellowship in lymphoma and multiple myeloma at Princess Margaret Hospital, Toronto, Canada. He is a Fellow of the Royal College of Physicians of Canada and American Board of Internal Medicine Certified. He has multiple publications in multiple journals including Blood Journal. Currently, he is a consultant in adult hematology/BMT at Princess Norah Cancer Centre at King Abdulaziz Medical City, Jeddah and assistant professor at King Saud Bin Abdulaziz University for Health Sciences.*

## Approach to the Treatment of the Older, Unfit Patient With Myeloma: From Diagnosis to Relapse

Saud Alhaili, MD



*Dr. Saud currently is Consultant Hematology and BMT at KFSH&RC, He has completed Fellow Medical Oncology on 2012 KFSH&RC, Fellow Hematology on 2014 KFSH&RC, Leukemia fellowship on 2015, Princess Margaret Cancer Center, Toronto, Canada, Bone Marrow Transplant Fellow on 2016 Princess Margaret Cancer Center, Toronto, Canada and finally Myeloma fellowship on 2017 Princess Margaret Cancer Center, Toronto, Canada.*

## Outcome Of Newly Diagnosed CML Patients Newly Treated With First Or Second Generation TKI

Hani Hassan Al-Hashmi, MD



*Dr. Hani Al Hashmi is currently the Director of Oncology Center and a Consultant Hematologist Oncologist. He joined King Fahad Specialist Hospital Dammam in April 2011 and was appointed as Chairman, Department of Adult Hematology and SCT in March 2013. Dr. Al Hashmi finished his Bone Marrow Transplantation Fellowship Program in the University of Calgary. Experience wise, from Aug 1999 to May 2000, Dr. Al Hashmi was the Medical Laboratory Scientific Officer in Hematology & Hemostasis Laboratory at King Fahd Armed Force Hospital, Jeddah, KSA. From July 2003 to June 2006 did his Internal Medicine Residency Program at University of Western Ontario, London, Ontario. From July 2006 to June 2008 he did his Adult Hematology Residency program, McMaster University, Hamilton, Ontario. From July 2008 to December 2009 he finished his Medical Oncology Residency Program, University of Calgary, Calgary, AB. From Jan 2010 to December 2010 Dr. Al Hashmi finished his Bone Marrow Transplantation fellowship program in University of Calgary, Calgary, AB. From Jan 2011 to March 31<sup>st</sup> he finished the Bone Marrow Transplantation, visiting physician program / Elective, Fred Hutchinson Cancer Research Center / Seattle Cancer Care Alliance, Seattle, Washington, USA*



## Molecular Monitoring Of Chronic Myeloid Leukemia Patients Newly Treated With First Or Second Generation TKI

Gamal Abdul Hamid



*Prof Dr Gamal Abdul Hamid has received his German board in internal medicine and PhD in hematology-oncology from faculty of medicine (Caral Gustav Carus), University of Dresden during the period of 1987- 1993. Currently, he is working as Director of National program of cancer control and Head of Hematology and Clinical Laboratory in Faculty of Medicine, University of Aden, Yemen and general secretary of Yemen Cancer Society and founder of Aden cancer registry. He is serving as an editorial member of several national and global journals. He has authored or co-authored many articles in a great variety of journals and has delivered lectures at many conferences and institutions in Yemen and internationally and referee for national and international journals. He is member of ESMO, ASCO, INCTR, Pan Arab Oncology and WAMS. Prof Gamal has many publication papers and books in hematology oncology field.*

Treatment of chronic myeloid leukemia (CML) with target the rapy approved 5 different tyrosine kinase inhibitors (TKIs; imatinib, nilotinib, dasatinib, bosutinib and ponatinib) according to the disease risk, disease stage, comorbidities and BCR-ABL genotype. Imatinib was the most common drug of choice for treatment of newly diagnosed CML patients in the last 15 years. In the last 5 years nilotinib, dasatinib and bosutinib , also new drugs with higher potency approved against BCR-ABL and against imatinib-resistant BCR-ABL mutations . Ponatinib is the newest TKI indicated for chronic phase , accelerated phase, or blast phase chronic myeloid leukemia (CML) who are resistant or intolerant to dasatinib , nilotinib or bosutinib .

Allogenic stem cell transplantation is a curative treatment for patients with CML, but the excellent curative remission with TKI therapy have challenged the role of allogeneic stem cell transplantation as a first line therapy.

When treatment begins, monitoring the response to Tyrosine Kinase Inhibitor (TKI) using standardized techniques by either metaphase cytogenetics or reverse transcriptase polymerase chain reaction that also determines type of mRNA transcript and guidelines is important to check for failure of response and thus, plan timely intervention by increasing the dose of TKI or opting for second line TKIs.

The evaluation of hematologic and cytogenetic responses was sufficient to gauge treatment efficacy in patients with chronic myeloid leukemia. However, with more potent TKI therapies, the majority of patients achieve complete cytogenetic response. Furthermore, deeper molecular responses are now commonly achieved, necessitating a reliance on molecular monitoring to assess minimal residual leukemic disease.



## **CML: between Cytogenetics and Molecular Diagnostics CML: between Cytogenetics and Molecular Diagnostics**

Ameera Radhi,MD,FRCPA,KFSU

Chronic Myeloid leukemia (CML) is the most common myeloproliferative disorder among chronic neoplasms. The history of CML joins with the development of cytogenetic analysis techniques in human. CML was the first neoplasm to be associated with a recurrent chromosomal alteration, reciprocal translocation between the long arms of chromosome 9 and 22 – Philadelphia chromosome which was discovered in 1960. Later the cloning of the BCR and ABL1 gene that fuse which result on the Chimeric BCR-ABL1 gene that encodes for an abnormal fusion protein with tyrosine kinase active domain that causes uncontrolled cell proliferation, ultimately leading to the clinicopathological features of CML.

Because the BCR-ABL1 gene fusion is the sine qua non of CML, cytogenetic and molecular testing are invaluable tools in the evaluation and monitoring of CML. These techniques include conventional cytogenetic analysis using chromosome banding, molecular cytogenetic techniques (e.g., fluorescence in situ hybridization [FISH]), and the polymerase chain reaction (PCR). Conventional cytogenetic analysis characterizes the type of translocation (simple or complex) and documents the presence or absence of

additional chromosomal abnormalities. Molecular cytogenetic techniques can confirm the presence of a BCR-ABL1 fusion in interphase (nondividing) cells, and additionally can document submicroscopic deletions on the derivative chromosome 9, which occur in approximately 15% of cases of t(9;22). Conventional cytogenetic analysis can also identify clonal abnormalities arising in Philadelphia chromosome-negative cells in patients receiving tyrosine kinase inhibitor (TKI) therapy. The widespread use and success of TKI therapy necessitated the development of testing methods sufficiently sensitive to assess continually decreasing levels of disease. Quantitative real-time reverse transcriptase-PCR (qRT-PCR) technology provides the means to define specific therapeutic goals and to follow BCR-ABL1 transcript levels over time. Such monitoring is critical for early recognition of TKI resistance, as well as for identification of the mutations underlying such resistance. Cytogenetic and molecular diagnostic laboratories thus play an integral role in the care of patients with CML, from initial diagnosis to minimal residual disease (MRD) testing and assessment of therapeutic resistance. Advances such as array-based comparative genomic hybridization and next-generation sequencing promise to elucidate further the pathophysiology of this disease.



## Stem Cell Transplant in SCD

Mohsen Alzahrani, MD



*Dr. Mohsen Alzahrani obtained his medical degree (M.B.,B.Ch) in 2000 from King Abdulaziz University in Jeddah. He obtained the Saudi Board of internal medicine in 2005. He completed the Hematology residency training in 2009 from Dalhousie University, Halifax, Canada and he did 2 years of fellowship in hematopoietic stem cell transplant and lymphoma at Dalhousie university, Halifax and University of Toronto, Canada.*

*Dr. Alzahrani currently is consultant of hematology and Hematopoietic stem cell transplant, head division of Stem Cell Transplant and Cellular Therapy at King Abdulaziz Medical City, Riyadh.*

Sickle cell disease (SCD) is an autosomal recessive disorder characterized by production of abnormal hemoglobin S and is associated with high morbidity and mortality. Disease phenotype can be variable depending on the coinheritance of other genetic disorders. Patients with severe phenotype manifesting with complications such as Stroke, recurrent acute chest syndrome, recurrent painful vaso-occlusive crisis not responding to hydroxyurea should be counselled about the option of Allogeneic Stem Cell Transplant (AlloSCT).

Pediatric studies

of AlloSCT from match related donors using myeloablative regimen showed excellent outcome with OS and EFS more than 90%. Risk of graft failure and TRM is low but the main drawback is high infertility rate. Transplantation using other options such as RIC or NMA conditioning or Haploidentical donors has shown promising results and can be used in carefully selected patients.

Studies of AlloSCT in adults' patients are limited and associated with higher rate of graft failure and TRM. However, recent studies using RIC and NMA regimens and match related donors has shown acceptable rate of success similar to what is reported in pediatric studies.

## News therapies in SCD

Sultan AlMotairy, MD



*Dr Sultan is Consultant, Hematology, KAMC- GH, Jeddah , Saudi Arabia,.He finished his Fellowship degree in hemoglobinopathies, McMaster University September 2017 and got FRCPC, Hematology, McMaster University, Canada , November 2016, ABTht, Hematology, October 2016 Master degree in Medical Education , Acadia niversity, Canada, May 2015 FRCPC, He has completed Internal Medicine, Dalhousie University, Canada , June 2015. Dr Sultan has presented many talks in the field of hematology oncology.*



# Hazardous Influence of Hydroxyurea on Spermatogenesis in Thalassemia Intermedia Patients: An Egyptian Cohort

Mohsen Saleh Elalfy, MD



*Professor Mohsen is Chairman of Department of Paediatrics, University of Ain Shams 2013-2016, Honorary Consultant Paediatric hematology / Oncology Ain Shams University Hospitals. He has over 200 scientific publications ( NJM , Blood , Hematologica , Seminars in Hematology,... ) and book chapters. He is the Reviewer for 12 scientific Peer review International and Regional journals*

*and board member & co-editor / board member for another 6 international peer review journals; 2200 citations , H-index 26. Dr Mohsen is the president of Egyptian Pediatric- Oncology Forum,,President Egyptian Society of Pediatric. Hematology/Oncology (ESPHO).Board Member of Inter-continental ITP group (ICIS).President of Ain Sham Pediatrics' Association 2013- Now.Project Leader in 5 clinical Trials, PI for 25 more clinical trials since 2002, 3 running clinical trials*

**Background:** Hydroxyurea (HU), frequently used in thalassemia intermedia (TI), might have adverse effects on spermatogenesis.

**Aim:** To assess the effects of HU treatment on sperm parameters and potential reversibility on its discontinuation in TI patients.

**Methods:** Twenty fully-pubertal TI males regularly followed-up at the Ain Shams University Thalassemia Center were classified according to previous HU treatment; first group had received HU for  $\geq$  one year, while the second had never received HU. All recruited patients were subjected to full clinical assessments. Sperm parameters (number, abnormal forms, motility and forward progression) were assessed at enrollment and reassessed six months after stopping HU treatment.

**Result:** Eleven thalassemics on HU therapy had statistically significant lower sperm count in comparison to those who had never received HU. At six months off HU therapy, there was statistically significant improvement of all sperm parameters. Nevertheless, such parameters were still lower than those of patients who had never received HU. Statistically significant relationships were noted between total sperm count and HU dose, compliance and duration of therapy.

**Conclusion:** HU appears to have a hazardous yet reversible effect on sperm health in pubertal TI patients. Counseling should be offered with close follow-up of its effect on fertility.





## **ICUS/CHIP/CCUS: What determines a correct diagnosis?**

## **When to treat and when not treat, older patients with AML**

Gulam Mofti, MD

Professor Ghulam J Mufti is head of the department of haematological medicine at Guy's, King's and St Thomas's hospitals, King's College London, with a team of 13 professors, 6 senior lecturers/lecturers and approximately 100+ research staff. In addition Professor Mufti is the Clinical Director of Laboratories Sciences and a non-executive director of King's College Hospital NHS Foundation Trust.

Professor Ghulam Mufti has extensive clinical and research expertise in leukaemias, lymphomas and in particular myelodysplastic syndromes, for which he is internationally renowned. His particular area of research has focused on molecular aberrations in MDS/AML and the identification of novel therapies that include gene and cell based therapies. He has published 400+ papers and chapters in scientific journals and textbooks on leukaemias and MDS.

He heads the research groups at King's working on the molecular genetics of MDS/aplastic anaemia/AML, and is a member of the working group that produced national and international guidelines on the treatment and prognosis of MDS. He is a founding member and chair of the UK MDS Forum. He is also a member of the European Bone Marrow Transplantation Group and a founding member of the Board of the International Myelodysplastic foundation, for which his department at King's College Hospital is a recognised Centre of Excellence. The department is also a centre of excellence for Leukaemia Lymphoma Research and is the largest allogeneic bone marrow transplant centre in the UK, and the only gene and cell based therapies centre for myeloid leukaemia and allied diseases.





## **High Throughput Flowcytometry for Combined MRD Measurements & Immune Monitoring**

Alberto Orfao, M.D., Ph.D

Alberto Orfao is currently Full Professor of Immunology at the Department of Medicine of the University of Salamanca. He is also subdirector of the Cancer Research Center, Director of the General Cytometry Service and the Cell sorting Service of the University of Salamanca, and Scientific Director of the Spanish National DNA Bank Carlos III and the Network of Tumor Biobanks of Castilla y Leon (Spain). His research activities are based on his position as one of the Principal Investigators of the Cancer Research Center and The Institute for Biomedical Research of Salamanca (IBSAL), and they are mainly focused on the field of hematological malignancies and the relationship between the immune system and cancer development and progression. He has contributed to >800 publications and book chapters, being co-author of >650 scientific papers in international journals, with an overall h-index of 76 and more than 29,000 accumulated citations; in addition, he is inventor of 45 (granted/pending) patents. He has received over 40 awards and recognitions including the Berend Howen and the Wallace Coulter awards of the International Society for Laboratory Hematology and the International Society for Clinical Cytometry, respectively, and the 2012 Castilla and Leon Prize for Scientific and Technical Research as well as the 3rd Prize in Applied Biomedical Research of the Valdes-Salas Foundation. He is currently member of the external scientific committees of several research institutions in Spain and other European countries, and he is member of multiple national and international scientific evaluation panels and groups, including the EuroFlow Consortium (chair), European Scientific Foundation for Laboratory Hemato-Oncology (ESLHO) and the European Leukemia Net (ELN).



## Direct-Acting Oral Anticoagulants (DOACs)

Turkey Alshoaiby



*Dr. Turki Is Consultant in Internal Medicine and Hematology & deputy head of department of Medicine at King Fahd Hospital Jeddah (KFHJ) since October 2016. Dr. Turki is Head , hematology division since October 2017as well as Chairman of Venous thromboembolism (VTE) & Blood components utilization committees at KFHJ . Dr is Member , scientific committee for VTE awareness and prevention , directorate of health affairs Jeddah and Clinical associate professor and external examiner at King Abdul-Aziz University , Rabigh branch and Albatrjee medical college.*

Venous thromboembolism (VTE), consisting of deep vein thrombosis and pulmonary embolism, is a prevalent, potentially fatal health problem. a major clinical concern associated with significant morbidity and mortality. The cornerstone of management of VTE is anticoagulation, and traditional anticoagulants include parenteral heparins and oral vitamin K antagonists. it has disadvantages. Heparin and its derivatives must be administered parenterally, whereas use of oral vitamin K antagonists is complicated by unpredictable pharmacokinetics and pharmacodynamics, drug-food and drug-drug interactions and the requirement for frequent laboratory monitoring. Recently, new oral anticoagulant drugs have been developed and licensed, including direct factor Xa inhibitors (e.g.rivaroxaban, apixaban and edoxaban) and thrombin

inhibitors(e.g. dabigatran etexilate). Randomized phase 3 trials have demonstrated that patients receive similarly effective anticoagulation with the DOACs when compared with warfarin, with similar or reduced risk of bleeding. Extended therapy trials have consistently demonstrated superior effectiveness for DOAC treatment when compared with placebo in preventing VTE recurrence.

This presentation presents a concise review of the pharmacokinetics, pharmacodynamics and accumulated clinical trial evidence for each DOAC for short-term, long-term and extended VTE therapy, and it considers the potential implications these agents have for the clinical management of VTE.



# Antiphospholipid Syndrome (APS)

Bassim T. Albeirouti, MD, MSC, CIP, FRCPC, FACP



*Consultant of Adult Hematology & BMT at KFSH&RC Jeddah*

*Dr. Bassim is a Full time consultant of Adult Hematology & BMT, section of Adult Hematology & BMT, Department of Oncology, King Faisal Specialist Hospital & Research Center – Jeddah Branch, Saudi Arabia .*

*He is Early Retired Assistant Professor in the department of hematology, faculty of medicine, King AbdulAziz University (KAU), & head of adult hematology & BMT division and consultant in the department of hematology at King AbdulAziz University Hospital (KAUH), Jeddah, Saudi Arabia.*

Antiphospholipid Syndrome (APS) is a very common disease in our area. Unfortunately, very few publications are coming from our area.

APS as it was described by Dr. Graham Hughes who described the syndrome back in 1983 in UK is a multifaceted disease.

The clinico-pathological features of APS was established initially as Sapporo Diagnostic criteria through ISTH in 1999 that were updated later in 2000, and finally updated in Sydney in 2006. However, more and more features has been described in the literature adding more and more clinical features of this fascinating syndrome.

Through this presentation we will learn the most updated clinico-pathological features of APS, along with the discussion of the local experience of the author of APS in KSA at different institutions and cities in KSA.

The summary and practical tips of the disease summarized form the most recent edition of APS book by Dr. Munther Khamashta will be presented also.

This syndrome should be kept in the mind of all practicing physicians as an important differential diagnosis of any patient presenting with problematic case scenario.

## Management of anticoagulant during special situations Comprehensive thrombosis clinic

Tarek Owidah, MD



Professor of Hematology at Alfaisal University

Consultant Hematology at King Faisal Specialist Hospital



## Thrombotic Thrombocytopenic Purpura(TTP)

Galila Zaher, MBBS, Dip RCPATH, MRCPATH, FRCPATH .UK

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*Dr. Galila is Consultant Haematologist in Haematology Department in KAUH, Jeddah*

*Dr. Galila has Membership in Haematology, The Royal College of Pathologist London, UK and Diploma in Haematology The Royal College of Pathologist London, UK M.B., B.Ch. 1988. She was actively involved in all areas of clinical and laboratory Haematology with special emphasis on bench work training in blood bank*

Thrombotic Thrombocytopenic Purpura(TTP) is part of Thrombotic microangiopathic syndromes (TMA). Types of TTP include :Congenital TTP, Acquired idiopathic TTP, and Secondary TTP

TTP has an estimated annual incidence of 2 -10 cases /106. Both sexes may be affected but females are more commonly affected than males.

VWF multimers under high shear stress bind sub-endothelial collagen to platelets causing platelets adhesion and aggregation. Normally A disintegrin and metalloprotease with eight thrombospondin -1-like domain (ADAMTS13) cleave ULVWF at A2 subunit as they are secreted. Severely ADAMTS 13 deficiency prevents timely cleavage of ULVWF. Gene encoding for ADAMTS has been identified to be encoded on chromosome 9q34 and the metalloprotease has Plasma activity of 50-178% and a half-life of 2-3 days.

The pathogenesis in TTP is characterized by a wide spread of intravascular thrombosis leading to an organ ischemia in brain, kidney or cardiac muscle. Blood flows through partially occluded vessels by platelet aggregates causes red blood cell fragmentation (schistocytes). The clinical presentation include thrombocytopenia, microangiopathic hemolytic anemia (MAHA), renal failure, neurologic abnormality and fever, however the presence of thrombocytopenia and MAHA should be sufficient to initiate treatment.

TTP could be Congenital, acquired idiopathic or secondary to drugs, pregnancy, post transplant or secondary to autoimmune diseases

Upshaw-Schulman syndrome also known as congenital or familial TTP is characterized by Chronic relapsing course. it is a rare disease usually presenting at infancy or childhood. the disease has an autosomal recessive mode of inheritance and both parents have 50% of activity of ADAMTS13. The patients typically has ADAMTS13 level <5% of normal plasma and

The classical pentad of thrombocytopenia, microangiopathic hemolytic anemia (MAHA), renal failure, neurologic abnormality and fever needed initially to diagnose TTP has been appreciated to thrombocytopenia, MAHA.

Patients may present with nonspecific symptoms such as weakness, abdominal pain, nausea, vomiting, and diarrhea.

Neurological deficit are present in 90% of patients. Confusion, seizures, motor deficits, blurred vision or headache are common symptoms, however

TIA and stroke are less frequently seen. Cardiac manifestations such as arrhythmia and cardiogenic shock can occur. Heart failure can present in 10%, and acute myocardial infarction in up to 18% in some series. Cardiac involvement are associated with high mortality rate and Renal symptoms in the form of Oliguria, anuria, acute renal failure can occur.

TTP has to be differentiated from other TMA, preeclampsia-HELLP syndrome, disseminated intravascular coagulation, Catastrophic antiphospholipid syndrome(APS), Evan Syndrome, heparin-induced Thrombocytopenia, paroxysmal nocturnal hemoglobinuria, immune thrombocytopenia purpura and autoimmune hemolytic anemia.

No "gold standard" for diagnosis of TTP, however it is considered as a medical emergency.

ADAMTS13 activity, auto-antibodies against ADAMTS13 molecule and genotypic characterization of ADAMTS13 gene can help establish the diagnosis of TTP and differentiate congenital from acquired TTP. However, these assays are not always available, results may take a long time and not all assays are completely standardized. Furthermore detectable ADAMTS13 activity in acute phase does not exclude TTP. In addition Low levels : of ADAMTS13 could occur in liver disease, DIC, chronic inflammatory conditions, uremia, HIT, late pregnancy, newborn and even some healthy controls have levels between 20-50%.





## **Respiratory Transfusion Complications**

Afraa AlDayel, MD

## **Transfusion Management of The Patient With AIHA**

Mohammed Almohammadi, MBBS, FRCPC



Consultant Hematopathologist and Transfusion Medicine,  
*Dr Mohammed is Chairman of Pathology and Lab Medicine at King Abdulaziz Medical City, WR, Ministry of National Guard, Saudi Arabia since May 2018.*

*He is Assistant Professor (joint appointment) at King Saud Bin Abdulaziz University for Health Sciences since 2015 and an attending Consultant Hematopathologist and Transfusion Medicine. In-charge of stem cell collection and stem cell Laboratory.*

*Dr Mohammed is Associate Director of Saudi Stem Cell Donor Registry, WR*

*Graduated from King Abdulaziz University, Faculty of Medicine. Completed hematological pathology residency and transfusion medicine Royal College Fellowship at Dalhousie University, Halifax, Canada in 2013 Followed by Fellowship in cellular therapy and stem cell lab processing.*



## **Transfusion Management of Patients with Hemoglobinopathies**

Sanaa Sayed Abdelshafy, MD

Dr. Sanaa is a professor of Clinical and Chemical Pathology, and former Deputy Dean of Faculty of Medicine Beni Suef University. She is the founder and CEO of Trust Labs in Egypt, and director of Misr University for Science and Technology (MUST) Blood Bank.

Dr. Sanaa received her MD in Immunology from Cairo University. She won the National Award for her research in Advanced Medical Technology in 2003. She also received several awards for her distinguished research, from esteemed foundations such as The International Federation of Clinical Chemistry (IFCC), The Egyptian Medical Syndicate, The Egyptian Society of Cardiology. Dr. Sanaa is currently a recognized speaker and reviewer for several international journals, the most prominent of which being The Egyptian Heart Journal, and The Critical Care Medicine Journal. She co-founded and directed several blood banks in Egypt including Beni Suef University blood bank, Nasser Institute Blood Bank, Specialized Zayed Hospital Blood Bank, Haram Hospital Blood Bank.



## Prevention and Management of Platelet Refractoriness

Niveen ABDULLAH, MB BS, M.D., FRCPath, CCST

*Dr. Niveen is Consultant of Histopathology & Molecular Pathology, Deputy, Medical Director of Blood Bank King Hussein cancer center, Jordan. She has completed her Fellowship doctor in Department of Pathology and Laboratory Medicine from King Hussein cancer Center in Jordan then she Joined Leeds Teaching Hospitals NHS Trust, Leeds-UK as an International Training Fellow Doctor in Molecular Pathology and Transfusion Medicine ,Royal College of Pathologists-UK.( FRCPath)*

Platelet refractoriness is considered when the platelet count response is significantly less than expected to **two or more** platelet transfusions. An average adult given one unit of single donor apheresis platelets is expected to have a rise in platelet count of approximately 30,000/microL in 10 to 60 minutes, with a return to baseline at two to three days. An immediate (within one hour) post-transfusion platelet count increment of  $>10,000/\text{microL}$  is an acceptable response.

Failure to achieve an immediate post-transfusion platelet count increment of  $10,000/\text{microL}$  on more than one occasion defines alloimmune refractoriness. This pattern is seen in patients with antibodies to antigens of the human leukocyte antigen (HLA) or human platelet antigen (HPA) system and may be seen in patients with massive splenomegaly. In these cases, there are several strategies to consider when selecting more "compatible" platelets for transfusion. These include HLA-matched platelets, HLA "compatible" (antigen-negative) platelets, platelets selected by crossmatch tests, and HPA compatible platelets when

HPA antibodies are present. Other strategies to consider while awaiting the laboratory results includes transfusing the most fresh, ABO compatible platelets and to recruit family members as directed donors. A normal platelet count increment at one hour, with a shorter time to return to the baseline count is typical of the pattern seen with reduced platelet survival. This can be due to sepsis, hematopoietic cell transplantation, disseminated intravascular coagulation, and medications that interfere with platelet survival. In these cases treating the underlying condition will improve platelet survival.

Methods for reducing platelet alloimmunization can include utilizing leukocyte-reduced blood components for transfusion and providing ABO-identical (preferred) or ABO-compatible platelets whenever possible.



## Latest Advances in Diagnostic and Treatment of DLBCL

Mubarak Almansour, MD





## **Pediatric approach of managing Hodgkin Lymphoma**

Ali Alqraqri, MD

Pediatric Hodgkin lymphoma treatment approach continues to evolve as new means of assessing response to treatment, appreciation of therapy related long-term risk and complication, and more effective therapeutic agents become available.

Treatment algorithms integrating functional imaging now provide the opportunity to adopt a

response-based approach, allowing adjustment of type, duration, and intensity of chemotherapy and rationale identification of children who may benefit from the addition of radiotherapy.

This interactive case-based session will equip participants with up-to-date, evidence-based knowledge on the how to treat pediatric Hodgkin lymphoma.



## **Can we improve the outcome of patients with PTCL**

Ali Bazarbachi, MD, PhD

Dr.Ali is a Professor of Medicine (Hematology and Oncology), Professor of Anatomy, Cell Biology and Physiological Sciences, Associate Dean for basic research, and Director of the bone marrow transplantation program at the American University of Beirut-Medical Center. Dr. Ali Bazarbachi's research focuses on developing targeted therapies for human leukemia and lymphoma and bone marrow transplantation. He has co-authored more than 220 articles in leading scientific and serves as a reviewer for many of these journals. He is the Chairman of the EMBMT Leukemia Working Party, Chairman of the NCCN Lymphoma Group for Middle East and North Africa, past President of the Lebanese Society of Hematology, and Associate Editor of Bone Marrow Transplantation.

## **Update in management of pediatric patients with Pre-B cell ALL**

Mohammed Fuad M Essa.MD



*Dr.Mohammed is Consultant , Pediatric Hematology/Oncology/SCT department Pediatric hematology Oncology. He is Pediatric hematology Oncology fellowship program director and Assistant professor at King Saud bin Abdulaziz University for Health Sciences*

*Dr Mohammed is Section head , Pediatric Hematology Division and Pediatric block medical students course coordinator at King Saud bin Abdulaziz University for Health Sciences He has many Projects/Publications as well as a lot of presentations in the field of pediatric*

hematology oncololy



## The tsunami of Targeted Therapy: Are laboratories ready for the storm

Salem H. Khalil, MD, FRCPA, FCAP



*Dr. Salem Khalil is Currently the Section Head of Cytogenetic and Molecular Genetics, and Consultant Hematopathologist, Department of Pathology and Laboratory Medicine at King Faisal Specialist Hospital and Research Centre. He is Assistant Professor at Al Faisal University Riyadh and Clinical Assistant Professor at King Saud University Riyadh. Dr. Khalil is a former Member of the Executive Board for Saudi Scientific Society of Blood and Marrow Transplantation (SSBMT) and one of the Founding Members of the Saudi Society of Hematology in October 2007. He is an Author and co-author of more than 45 articles published in local and international journals, with more than 120 abstracts presented at local and international meetings and more than 60 invited lectures were presented at different institutions. He is involved with several on-going research projects in collaboration with other Oncologists and Scientists.*

Precision medicine is an emerging approach for disease treatment and prevention that takes into account individual variability in genes, environment, and lifestyle to develop an individualized treatment plan. Clinical applications that will benefit from precision medicine include improving patient diagnosis and prognosis, predicting treatment response, and determining predisposition to certain cancers. This information will be incorporated into an individualized patient treatment plan that will provide maximum benefit while reducing the use of drugs that have serious side effects and are unlikely to benefit the patient.

Targeted therapy provides the foundation of precision medicine. Even in individuals with similar clinical cancer phenotypes, drug therapy is only effective in a subset of patients. Owing to recent advances in molecular biology, genomics, and bioinformatics, research has shown that different drug response is often a result of differences in genetic alterations. An in-depth understanding of the biology of the tumor, including molecular changes and altered signaling pathways will allow for the identification

of patients who are likely to benefit from such treatments. With the discovery of additional targeted agents pathologists and laboratory staff are required to identify the “target” population or the subset of patients who have cancers with druggable molecular abnormalities. The challenge relies on identifying the most appropriate molecular biomarker that will triage the patient to a “personalized” therapeutic regimen with the highest probability of eradicating the tumor. This task becomes increasingly complex because allocation for molecular testing becomes a crucial decision in managing patients with cancer. Furthermore, because many targets can be evaluated by multiple laboratory methods, like sequence analysis, fluorescence in situ hybridization, PCR, or Next Generation Sequencing (NGS), it is critical that pathology organizations develop standardizing methodologies for marker testing in the clinical laboratory. In this presentation further strategic planes and previous tertiary care hospital laboratory experience will be discussed.



## Update in management of Adolescents and Young Adults (AYA) patients with ALL

Ahmed Absi, MD



*Consultant Hematology –Medical Oncology –Bone Marrow Transplantation Assistant Professor of Hematology / Oncology Princess Norah Oncology Center King Abdulaziz Medical City- Ministry of National Guard Health Affairs, Assistant Professor of Hematology / Oncology - King Saud bin Abdulaziz University for Health Sciences (Resolution dated April –2016)*

*Dr.Ahmed participate in teaching medical students at King Saud bin Abdulaziz University for Health Sciences (KSAU-Jeddah). My teaching activities are delivering lectures during the Hematology Block, supervise students during their In-Hospital electives and mentor students in their research projects.He has a lot of publications ,books in the that field*

## Recent Advances in the Biology and Treatment of T Cell ALL

.Mosab Damlaj, MD



*Dr. Damlaj is a consultant in the department of Oncology / division of Stem Cell Transplant and Cellular Therapy at King Abdul Aziz Medical City – Riyadh. He obtained a Bachelors of Science in Microbiology & Immunology with Honours followed by a Doctor of Medicine degree at McGill University in Montreal Canada. Subsequently, he completed post graduate training in Internal Medicine, where he also served as chief medical resident at the McGill University Health Center followed by Hematology fellowship at the same institution. He then moved to Mayo Clinic in Minnesota to undertake a fellowship program in Blood & Marrow Transplant under the supervision of Drs. Mrinal Patnaik& Mark Litzow. Research interests include outcome research post transplantation and quality improvement and he has a number of publications in this field. He is a member of several organizations including the SSBMT, ASH, EHA and ASBMT.*

Acute lymphoblastic leukemia of T-cell lineage disease (T-ALL) is an aggressive neoplasm that can manifest as leukemic and / or lymphomatous presentation. Recently, our understanding of this complex disease has improved with the discovery of NOTCH1 and FBXW7 activating mutations among others. These were found to be of prognostic and potential therapeutic significance. Pediatric inspired regimens have been increasingly used in eligible adolescents and

young adults with improvement in outcome compared to historical controls. Allogeneic hematopoietic stem cell transplantation remains a viable option for long term disease control in those with high risk features and relapsed or refractory disease. In this talk, overview of the biology and treatment of T-ALL will be presented with particular emphasis on newly described mutations and the potential for targeted therapy.



## Fever Neutropenia

Faisal Kordy, MD

*Dr. Faisal is Consultant Pediatric Infectious Diseases and Head of Pediatric Infectious Diseases division, Madinah Maternity and Children Hospital. He has completed the following: Clinical and research fellowship in infections in immunocompromised children 2016-2018. Clinical fellow focusing in pediatric TB and HIV at Sick Kids 2012-2016. Fellow Pediatric Infectious Diseases at Sick Kids 2010-2012. Fellow in pediatric infectious diseases and infection control at King Faisal Specialist Hospital & Research Center, Riyadh, KSA 2001-2004. He has PEER REVIEWED PUBLICATIONS and other projects and presentations in the same field.*

Infection is a major cause of morbidity and mortality in cancer patients. Fever may be the first manifestation of a life-threatening infection, particularly during periods of neutropenia. Febrile episodes occur in about 30% neutropenic episodes in children with chemotherapy-induced neutropenia or after hematopoietic stem cell transplantation.

The demonstration of markedly reduced infection-related morbidity and mortality with the empiric use of broadspectrum antibiotics during periods of febrile neutropenia was a major advance in the field of oncology in the 1970s. Subsequent studies identified factors associated with a higher risk of bacterial infection and facilitated a more tailored approach to empiric

antimicrobial therapy. Because of important differences between oncology and hematology patients with neutropenia, fever in the pediatric cancer patient during periods of therapy-induced neutropenia are reviewed here.

### Objectives:

- Define fever and neutropenia 1)
- Categorize patients according to infection risk. 2)
- Identify the important aspects that are usually overlooked in history and physical exam. 3)
- Select proper antimicrobials based on infection risk according to the guidelines. 4)



# Primary familial and congenital polycythemia; The forgotten entity

Mansour Aljabry



*Dr. Mansour is Senior Consultant of Haematopathology and Blood Transfusion Specialty at King Khalid University Hospital, King Saud University Medical City. Program Director of KSU Fellowship program in Hematopathology and blood transfusion. Head of Flowcytometry unit - King Khalid University Hospital and Medical College, King Saud University, Riyadh, Kingdom of Saudi Arabia.*

*Consultant in Hematopathology and blood bank – Pathology Department at King Fahad Medical City – (Part-time 2013-2017). Dr Mansour is Assistant Professor & Consultant Hematology Unit-King Khalid University Hospital & King Saud University Medical City and Medical College, King Saud University, Riyadh, Kingdom of Saudi Arabia. Member of: The Saudi Scientific Hematology Society and Member of editorial board of Journal of Applied Hematology. Has a lot of researches, publications and teaching activities in his field.*

Primary familial and congenital polycythemia (PFCP) is a familial disorder characterized by isolated erythrocytosis due to the inheritance of mutated hypersensitive erythropoietin receptor (EPOR). The data regarding the true prevalence of PFCP are scarce and insufficient. However, it is likely to be underdiagnosed and underreported as definitive diagnosis requires molecular characterization of the implicated gene mutation, as well as exclusion of wide variety of closely related differential diagnoses. Moreover, some patients may undergo regular phlebotomies as symptomatic therapy for hyperviscosity related

to polycythemia without further investigations. PFCP is a rare autosomal dominant disorder caused by hypersensitivity of erythropoietin receptor of erythroid progenitors leading to increased rate of erythropoiesis at any given serum erythropoietin level. The hallmark of this disorder is isolated erythrocytosis with the absence of splenomegaly and lack of secondary causes of polycythemia. In this review, we will shed light on various aspects of PFCP with special focus on molecular pathogenesis, diagnostic approach as well as the most common differential diagnoses and the modalities of management.





## Stem Cell Transplant In Thalassemia Vs Gene Therapy

Abdullah Aljefri, MD

### Hematological Manifestations Of Primary Immunodeficiency Disorders

Azza Abdel Gawad Tantawy

*Professor Of Pediatrics , Pediatric Hematology/Oncology Unit , Children's Hospital, Faculty Of Medicine , Ain Shams University, Cairo, Egypt*

Primary immunodeficiency disease (PID) is an inborn error of the immune system, and is characterized by not only susceptibility to infection but also frequent combination with autoimmune diseases and malignancies<sup>(1)</sup>. Autoimmunity and immune dysregulation may lead to cytopenia that may be the initial presenting symptom of patients with PID, irrespective of a previous history of severe infections, autoimmunity, or a familial predisposition. Because hematologic diagnostic procedures rarely include the differential diagnosis of PIDs and because clinical immunologists often have little experience in the management of newly diagnosed cytopenias, awareness of this challenging and growing field is critical<sup>(2)</sup>. The pathogenesis of cytopenia in PIDs is heterogenous and comprise cellular or humoral autoimmunity, immune dysregulation in form of hemophagocytosis or lymphoproliferation with or without splenic sequestration, bone marrow failure and myelodysplasia, or secondary myelosuppression. In some patients, cytopenia may be detected as an incidental finding, whereas other patients may be severely ill<sup>(1,2)</sup>. Especially when cytopenia is the initial symptom of a PID, the order and depth of diagnostic steps have to be performed in accordance with both an immunologic and a hematologic

approach and will help exclude disorders such as systemic lupus erythematosus, common variable immunodeficiency, and autoimmune lymphoproliferative syndromes, hemophagocytic disorders, lymphoproliferative diseases, and novel PID disorders.<sup>(3)</sup>

Recognition of PID associated cytopenia is important as immunosuppressive treatment often needs to be initiated urgently, which impedes further relevant immunologic laboratory analyses aimed at defining the underlying PID. Awareness of potentially involved disease spectra ranging from hematologic to rheumatologic and immunologic disorders is crucial for identifying a certain proportion of PID phenotypes and genotypes among descriptive diagnoses such as autoimmune hemolytic anemia, chronic immune thrombocytopenia, Evans syndrome, severe aplastic anemia/refractory cytopenia, and others<sup>(4)</sup>. Also, recent advances in genetic analysis revealed that defects of the same gene might cause PID and hematological disease. Therefore, PID and hematological diseases are not exclusive but rather complementary, and clinicians should remain open to investigating these diseases<sup>(1)</sup>. The main intention of this presentation is to increase awareness of PID among haematologists and to identify clinical clues to the diagnosis and to classify the types of cytopenia that occur in the context of PID to facilitate correct timely management.



## Thalassemia Intermedia

Ali Ahmed Almadhani, MD



*Dr Ali is consultant Hematologist and Executive director of Sohar Hospital 2012 till date*

*He is Head of training and staff development department Sohar hospital 2007- 2012*

*He got Diploma of Fellowship FRCP (Glasg) Sept-2016. And MSc in Haemoglobinopathy (UCL-UK) Nov,2012.He Has MRCP Royal College of Physician (London-Uk). 2005-Diploma in Tropical Medicine & Hygiene (DTM&H) from Royal College of Physician (London-UK). Middle East Leadership Programme. (INSEAD), March, 2012.*

*His interest in Hemoglobinopathies and hemophilias and he has many publications and wide activities in this field*

Thalassemia intermedia is a term used to define a group of patients with [β thalassemia](#) in whom the clinical severity of the disease is somewhere between the mild symptoms of the β thalassemia trait and the severe manifestations of β thalassemia major.

Phenotypically β<sup>0</sup>thalassemias are classified as β<sup>0</sup>thalassemia major, β<sup>0</sup>thalassemia minor and β<sup>0</sup>thalassemia intermedia. Hereby, we would like to share our experience on thalassemia intermedia phenotype,

Occasionally patients with thalassemia intermedia are completely asymptomatic until adult life with only mild anemia. The major and intermedia forms of the disease are the two extremes of a wide range of clinical variability. Each group includes a continuous scale of severity, as demonstrated by the variability in age at which thalassemia major patients need transfusion; from months to years of life.



## Endocrinopathies in Thalassemia

Abdulhadi Habib, MD



## Oxidative stress and antioxidants in B thalassemia

Galila M. Mokhtar Kamal, MD



*Professor (Emeritus) of Pediatrics, and Pediatric Hematology /Oncology, Former Head of the Pediatric Department 2010-2012 & Head of Neonatology Unit Ain Shams University, 2006-2008 Head of Pediatric Hematology/Oncology Unit, Ain Shams University 2008-2012. Head of Genetic Research center Ain Shams University 2010-2012 Member of the Egyptian Board of Pediatrics, Ministry of Health & Population Moderator of the Egyptian Board of Pediatric Oncology.*

*Prof Jalila has Post Doctorate training in Edward Herriot Hospitals, Lyon, France, 1984 ,European school of Oncology, Athens, Greece, 1992 and Training courses of International Society of Hematology & International society of Pediatric Oncology.*

*She is Editorial board of Egyptian Journal of Hematology and other medical journals ,has many publications in pediatric hematology oncology field .She has many memberships like International Society of Hematology, International Society of Pediatric oncology ,World Federation of Hemophilia, American Society of Hematology ,European Hematology Association and others.*

In B thalassemia many factors contribute to increased oxidative stress : namely excess alpha globin protein on the expense of beta globin protein with the result of formation of hemin and free iron that promote the formation of free oxygen species ,this induces oxidation of membrane lipids and subsequent hemolysis as well as increased apoptosis and ineffective erythropoiesis .On the other hand frequent blood transfusions add to the danger of the oxidative stress with the generation of redox-reactive labile iron, which promotes the production of other reactive oxygen species (ROS). If not neutralized, uncontrolled production of ROS leads to damage of various intra- and extracellular components such as DNA, proteins, lipids, and small antioxidant molecules .Damage includes cellular damage, red blood cell hemolysis ,

inflammation ,vascular damage and platelet activation ending with thrombosis Endogenous antioxidants as 1- Superoxide dismutase, catalase, glutathione peroxidase, &ferroxidase, terminate the activities of ROS. 2- Non enzymatic endogenous defense mechanisms include metal binding proteins (ceruloplasmin, haptoglobin, albumin) 3- Endogenously produced free radical scavengers (glutathione GSH ubiquinol, &uric acid). In B thalassemia defense mechanism may be insufficient and extra needs for external antioxidants as vitamins A C E as well as zinc, selenium and folic acids are usually required to restore the balance.



## Pre-implantation Genetic Diagnosis (PGD)

*Ammar Alhasani, MD*



*Dr Ammar is Maternal fetal medicine consultant and OB/Gyn Residency training program Director, NGHHA Madinah*

*He has completed Medical school in King Abdulaziz University, Residency training in OB/Gyn in NGHHA – Jeddah and obtained Saudi Board in OB/Gyn*

PGD provides an early genetic diagnosis for an embryo before it is implanted into the uterus. It is an effective method to prevent the transmission of hereditary diseases to the next generations in the families with single gene disorders or chromosomal abnormalities. PGD requires combined expertise in the field of reproductive medicine together with molecular genetics and/or cytogenetic. It involves two stages: Firstly; IVF for ovarian stimulation, monitoring and timing of oocyte retrieval, fertilization and embryo biopsy. Secondly; a genetic diagnosis that is reliable and efficient is necessary to report the result in early enough time for embryo transfer to take place in the same cycle.

Couples referred for PGD are at risk of having a pregnancy affected with a known genetic disease, the majority of them at least have one affected child of their own or in the close family. The available reproductive option for them was prenatal diagnosis, which remains an unsatisfactory solution for some couples at high genetic risk, e.g. couples who had undergone repeated termination of affected pregnancies often ask for diagnosis prior to pregnancy. Furthermore, the couples that find termination of pregnancy unacceptable for religious, emotional, and social reasons also ask for PGD. Such couples feel that starting a healthy pregnancy using PGD is the only option for them since pregnancy termination is particularly distressing.

There is also a group of patients who are sub-fertile, or with previous pregnancy with chromosomal abnormality, or had repeated miscarriages, recurrent implantation failure following IVF cycles, or for those mothers with advanced maternal age, PGD and Preimplantation Genetic Screening (PGS) allows the screening for chromosomal abnormalities.

Another group of patients suffers from cancer predisposition such hereditary breast and/or ovarian cancer, or had inherited some genes that develops cancers such as the Familial adenomatous polyposis A genes (colon cancer). Most of the cancer patients receive radiotherapy/chemotherapy as part of their treatments which may permanently affect their fertility. Therefore, IVF/ PGD cycles help them to diagnose the embryos, and those healthy embryos which do not have the cancer causing mutations can be transferred to the mother in order to initiate disease free pregnancy. For those patients underwent radiotherapy/chemotherapy, fertility preservation is highly recommended as a part of the treatment supported with PGD following fertilization, where healthy embryos can be frozen and consider for transfer after completing the radiotherapy/chemotherapy treatments.



## New Horizon In The Management of Thalassemia

Amal El-Beshlawy, MD



*Dr. El-Beshlawy was graduated from Cairo University (with the degree of honor) in 1967 where she did all her academic career. She is the founder and president of the Pediatric Hematology Department of Cairo University from 1984 till 2005, Emirate professor and senior consultant till present. She received her training in pediatric hematology and bone marrow transplantation in Necker Hospital Paris, France in 1978-1979. She is the founder and the president of the " Egyptian Thalassemia Association (ETA)" since 1990. ETA is a member of the Thalassemia International Federation (TIF) since 1992. Prof. El-Beshlawy has been awarded the "Prize of the state for Excellence in Medical Sciences" (Mainly Pediatric Hematology) in 2002, The Prize of The World Federation of Hemophilia in 2003, the Prize of "Cairo University in Medicine" in 2005 and the Prize of " Distinction in Medicine in 2008". Sultan Bin Khalifa International Thalassemia Award from UAE for Best Research in 2015, The Prize of the state in Health and Medicine in 2017. She is a member of the National Committees for Blood Diseases in Pediatrics, by A Ministerial Decree in 2007 . She is the principal supervisor for more than 114 MD & MSc. Thesis mostly on Thalassemia&SCD , Hemophilia and &Gaucher Disease. The Principal Investigator (PI) of 36 International Research Projects mostly on Thalassemia, SCD , Hemophilia and Gaucher Disease. Prof. El-Beshlawy authored and co-authored more than 120 International publications on thalassemia, SCD, hemophilia and Gaucher disease. EDITOR: for the "Journal of The American Medical Association (JAMA) Pediatrics Middle East" from 1998,*

Significant advances have been achieved over the last few years in better investigating the pathophysiology of thalassemia. This led to the development of new therapeutic options for the management of this disease. Understanding the pathophysiology of ineffective erythropoiesis the main cause of the disease outcome promising treatment for thalassemia the activin receptor ligand traps Luspatercept (ACE-536) has been investigated. In a preclinical studies Luspatercept has been shown to act as a ligand trap for GDF11 and other members of the transforming growth factor (TGF)-B superfamily which inhibits the differentiation and maturation of red blood cell (RBC's) precursors. In clinical trials Luspatercept increases the hemoglobin level in patients with thalassemia with reduction of their transfusion burden in the majority of the transfusion dependent thalassemia patients (TDT) and increase of 1-2g/dl or more in non transfusion dependent patients (NTDT).

The role of JAK-2 inhibitors in attenuating ineffective erythropoiesis have been evaluated . Ruxolitinib a potent and selective oral JAK-2 inhibitor used in a clinical trial in thalassemia patients a noticeable reduction in spleen volume was observed with a slight improvement in the pre-transfusion hemoglobin level. Ruxolitinib treatment can serve as an alternative option for patients with TDT who are potential candidates for splenectomy.

Hepcidin the iron hormone regulator, molecules that either increase its production or stimulate its action have also been studied (TMPRSS6 and Mini – Hepcidin) with promising results.

Finally studies on Gene therapy are currently ongoing with very encouraging possible results.



## Approach to Thrombocytopenia

Arwa Alyamani, MD



*Dr. Arwa is Consultant Pediatric hematology&Oncology /BMT specialist, department of pediatric Oncology at King Khalid National Guard Hospital, Jeddah, KSA*

*She has completed Hematology and Oncology Fellowship at Hospital of Sick Kids, Toronto, Canada with Full BMT Fellowship at the Department of BMT at The Hospital for Sick Children, Toronto on 2007*

*Dr Arwa is the Deputy Chairman– Oncology department western region for Quality management and patient safety as well as the Secretary general of Saudi Arabia Pediatric Hematology and Oncology Society SAPHOS. She has a lot of educational activities including oral presentations, workshops as well as researches activities in Pediatric Hematology Oncology field.*

## ABCs In Blood Transfusion

Maha Badawi, MD



*-Dr. Maha Obtained MBBS degree from King Abdulaziz University in Jeddah, Saudi Arabia*

*-Completed postgraduate training in internal medicine, hematology, and transfusion medicine at University of British Columbia*

*- Currently working as an assistant professor in the faculty of medicine, King Abdulaziz University in Jeddah, Saudi Arabia, and the director of Blood Transfusion Services in King Abdulaziz University Hospital.*

Transfusion of blood and blood components is a common intervention for many medical and surgical patients. Health care professionals from various disciplines must be aware of basic prescribing information of blood and components, and should be able to handle transfusion complications and adverse reactions. Physicians must be

aware of the importance of ABO and Rh groups, and need to be knowledgeable of essentials of compatibility testing. Each transfusion must be preceded by obtaining an informed consent from patients. This requires knowledge of benefits, risks, and alternatives to blood transfusion.



# How To Interpret And Pursue An Abnormal Complete Blood Cell Count (Cbc) And Peripheral Blood Film (Pbf)

MAHER M. AL JOHANI, MD



*Dr. Maher is Consultant and Assistant Professor of Hematopathology and Blood Bank*

*He is Head of Pathology Department, Collage of Medicine, Taibah University in Madinah Saudi Arabia*

complete blood cell count (CBC) is one of the most common laboratory tests in medicine. CBC and peripheral blood film (PBF) remain the mainstay of hematologic diagnosis. CBC and peripheral blood film (PBF) can be truly diagnostic for a disease condition such as a blood film diagnosis of sickle cell disease or Malaria. In other cases, it is a best indicative for further laboratory work-ups or more advanced investigations such as cytochemistry, flow cytometry,

Acytogenetics or molecular techniques especially when dealing with malignancies.

Clinicians should be abreast with its clinical utility and proper application of the reports in the management of patients. This lecture will highlight how to interpret and pursue an abnormal complete blood cell count (CBC) and peripheral blood film (PBF) and help you to achieve a successful clinical practice



# **Impact of Nanobiotechnology on the Future of Medicine:**

## **The Road from Nanomedicine to Precision Medicine – Case Studies**

Shaker A. Mousa, PhD, MBA, FACC, FACB

Endowed Chair, Tenure Professor of Pharmacology, Executive Vice President, and Chairman

The Pharmaceutical research Institute, ACPHS, Albany, NY USA



Dr. Mousa finished PhD from Ohio State University, College of Medicine, Columbus, OH and Post-doctoral Fellowship, University of Kentucky, Lexington KY. He also received his MBA from Widener University, Chester, PA. Dr. Mousa is currently an endowed tenure Professor and Executive Vice President and Chairman of the Pharmaceutical Research Institute and Vice Provost for Research at ACPHS. Prior to his academic career, Dr. Mousa was a senior Scientist

and fellow at The DuPont Pharmaceutical Company for 17 years where he contributed to the discovery and development of several FDA approved and globally marketed diagnostics and Therapeutics.

He holds over 350 US and International Patents discovering novel anti-angiogenesis strategies, antithrombotics, anti-integrins, anti-cancer, and non-invasive diagnostic imaging approaches employing various Nanotechnology platforms. He has published more than 1,000 journal articles, book chapters, published patents, and books as editor and author. He is a member of several NIH study sections, and the editorial board of several high impact Journals. His research has focused on diagnostics and therapeutics of angiogenesis-related disorders, thrombosis, vascular and cardiovascular diseases.

Over the past few years, evidence from the scientific and medical communities has demonstrated that nanobiotechnology and nanomedicine have tremendous potential to profoundly impact numerous aspects of cancer and other disorders in term of early diagnosis and targeted therapy. The utilization of nanotechnology for the development of new nano-carrier systems has the potential to offer improved chemotherapeutic delivery through increased solubility and sustained retention. One of the major advantages of this cutting edge technology is its unique multifunctional characteristics. Targeted delivery of drug incorporated nanoparticles, through conjugation of tumor-specific cell surface markers, such as tumor-specific antibodies or ligands, which can enhance the

efficacy of the anticancer drug and reduce the side effects. Additionally, multifunctional characteristics of the nano-carrier system would allow for simultaneous imaging of tumor mass, targeted drug delivery and monitoring (**Theranostics**). A summary of recent progress in nanotechnology as it relates specifically to nanoparticles and anticancer drug delivery will be reviewed. Nano Nutraceuticals using combination of various natural products provide a great potential in diseases prevention. Additionally, various Nanomedicine approaches for the detection and treatment of various types of organ specific delivery, vascular targeting, and vaccine will be briefly discussed.



## **Blood Component Therapy in Coagulation Disorders**

Layla Bashawri, MD



*Prof. Layla is Professor of CLS, Consultant Hematopathologist University of Dammam) Fahd Hospital of the University (KFHU) Al-Khobar, Saudi Arabia*

*Currently she is Vice Dean College of Applied Medical Sciences Female Sector, Program Director Hematology Fellowship Program 2012 , Chairperson CLS 2007-2015 and Director of Diagnostic Laboratories 2000-2002.*

*She is speak in international conferences and has many Published Refereed Scientific Researches in Hematology field*

## **Cutaneous Lymphomas: Clinical Picture, Diagnostic and Treatment**

Perci Lehmann, MD



## **How to manage relapsed CLL**

Ahmed Sulaiman Alaskar, MD, FRCP(C), FACP



*Consultant, Adult Hematology & HSCT*

*Department of Oncology - King Abdulaziz Medical City -  
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*Associate Professor of Medicine, College of Medicine, King Saud bin Abdulaziz  
University for Health Sciences. December 2004 to present.*

*Executive Director*

*King Abdullah International Medical Research Center,*

*Riyadh, Saudi Arabia*

*President, Saudi Scientific Society of Blood & Marrow Transplantation. March 2010 to  
present.*

*Medical Director, Cord Blood Bank, King Abdullah International Medical Research Center,  
Riyadh, Saudi Arabia*



## **Current strategies to prevent and treat GVHD in Allo HCT**

Shahrukh Hashmi, MD



*Dr.Hashmi is Associate Professor of Medicine - Alfaisal University – Riyadh, Saudi Arabia*

*2017-present*

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*07/2016 - Present*

*Director, BMT Long Term Follow Up Clinic, KFSHRC, Riyadh, Saudi Arabia*

*10/2016 - Present*

*Consultant (Supp): Blood and Marrow Transplant Division, Department of Internal Medicine, Mayo Clinic, Rochester, Minnesota*

*Dr.Sharukh has has a lot of Leadership Positions and Active Memberships like Center for International Blood and Marrow Transplant Research (CIBMTR) – Milwaukee, Wisconsin, US*

*Member, Advisory Committee at Large (Non-North America): 03/2018, Center for International Blood and Marrow Transplant Research (CIBMTR) – Milwaukee, Wisconsin, US*

*Co-Chair, Health Policy and International Health Working Party: 02/2018 ,American Society for Blood and Marrow Transplantation Survivorship SIG – Arlington Heights, Illinois, US*

*Chair 10/2016 – present and others .He has more than 100 Peer-Reviewedd Articlcs*

## **Genetic Manipulation Of Iron Metabolism**

HANAN HAMED, MD



*Prof Hanan is Professor of Internal Medicine and Clinical Hematology Faculty of Medicine Ain Shams University from October 2004 - till now.*

*She is Member of Hematology Board at Faculty of Medicine Ain Shams University, Member of Bone Marrow Transplantation Board at Faculty of Medicine Ain Shams University.*

*Prof.Hanan is Head of Internal Medicine and Clinical Hematology Unit Ain Shams University Specializea Hospital ASUSH Cairo – Egypt.*



## Local experience for SC donation registry

*Mohsen Alzahrani, MD*



*Dr. Mohsen Alzahrani obtained his medical degree (M.B.,B.Ch) in 2000 from King Abdulaziz University in Jeddah. He obtained the Saudi Board of internal medicine in 2005. He completed the Hematology residency training in 2009 from Dalhousie University, Halifax, Canada and he did 2 years of fellowship in hematopoietic stem cell transplant and lymphoma at Dalhousie university, Halifax and University of Toronto, Canada.*

*Dr. Alzahrani currently is consultant of hematology and Hematopoietic stem cell transplant, head division of Stem Cell Transplant and Cellular Therapy at King Abdulaziz Medical City, Riyadh.*

In Saudi Arabia, 30% of adult patients and 60% of pediatric patients cannot find a matching family donor. Therefore, the establishment of Saudi Stem Cell Donor Registry for unrelated donors is of utmost need. Stem cell transplantation is used to treat many life-threatening diseases such as Leukemia and Non-Hodgkin's Lymphoma.

This registry is a national project launched in line with international standards, and is currently in the World Marrow Donor Association (WMDA) and is already part of the World Wide Bone Marrow Registry.

SSCDR offers another chance for patients who cannot find a family donor, by providing a rich database of potential unrelated donors. Our life-saving registry plans to recruit 100,000 donors in its first five years through nationwide public awareness campaigns, an ambassador program, marathons, and educational campaigns. Today, the Kingdom of Saudi Arabia is the first in the Arab world with over 70,000 stem cell donors in our registry.



# Prevalence of Iron Deficiency Anemia In Infants age 6 months to 2 years attending well-baby clinic at Al-Madina City in Saudi Arabia

ZAKARIA MOHAMMED HAMZA AL HAWSAWI, MD



*Consultant Paediatric Haematology/ Oncology and Assistant Professor , Pediatric Department Medical College – Taiba University . He was Chairman of Paediatric Department, Madina Maternity & Children's Hospital, 1993-1995, Medical Director, Madina Maternity & Children's Hospital 1995-2000. ,Acting Hospital Director on many occasions and Chairman of many Hospital Committees. Dr Zakaria was President of Madina Hereditary blood disease society for 7 years and Board member of many charity societies. Dr Zakaria Present and attended many local and international conferences ,Has 37 publications in local and international previewers Journals as well as books publications .*

## Objective:

The objective of this study is to determine the prevalence of iron deficiency anemia (IDA) in infants aged 6 – 24 months attending a well baby clinics at primary health care centers at Al-Madina City in Saudi Arabia.

## Methods:

The study was conducted in 5 primary health care centers, selected randomly from four sectors. The sample size was 500 infants, and 100 infants were screened from each centers. Iron deficiency anemia was defined as infants with either hemoglobin level less than 11 gm/dl, or serum ferritin level less than 10 µg/L. Blood samples were obtained for estimation of hemoglobin and serum

ferritin for all infants aged 6 – 24 months, attending the well baby clinics during study period. The data were analyzed by excel computer program, and all infants diagnosed as IDA, received iron therapy for 3 months, and were followed at PHC.

## Result:

The result showed that, out of 500 infants, 246 (49%) had IDA, with the mean age of 13 months, and 130 (53%) were males, and 116 (47%) were females.

Out of 274 Saudi infants 126 (51%), had IDA.

## BACK COVER

MIDDLE EAST & NORTH AFRICA HEMATOLOGY CONGRESS  
8TH PAN ARAB HEMATOLOGY ASSOCIATION CONGRESS  
17TH ANNUAL MEETING OF SAUDI SOCIETY OF HEMATOLOGY  
KING SALMAN CONVENTION CENTER  
ALMADINAH, SAUDI ARABIA  
1-4 February 2019

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