

Main Topics:

- Thrombosis & Haemostasis
- Regenerative Medicine
- SCT
- Anaemia & Haemoglobinopathies
- Case Presentations and Hematomorphology
- Hematopoietic Malignancies
- Free Papers

Call for Abstract:

Abstract should be written on computer font 14, Size A4 should be submitted to conference secretariat (info@pioneer-events.org)
The Dead line to receive abstracts 15th September 2012 & we will reply 28th September 2012

Registration Fees:

L.E 250 Registration for the conference & Registration for the Egyptian society of Hematology
L.E 200 per nonmember delegates
L.E 150 per member delegates
L.E 100 Junior

Contact Information

Conference Secretariat
Pioneer Events
30 Dr. Anwar El Mofti St. Apt. 61 Nasr City, Cairo, Egypt
Tel.: (+202) 24053575 Fax: (+202) 24020609
e-mail: info@pioneer-events.org

The Egyptian Society of Hematology & Research (ESHR)
National Cancer Institute, Fom El Khalig, Cairo, Egypt
Tel: (+202) 23635083



9th International Conference of the Egyptian Society of Haematology & Research (ESHR) Update in Hematology

17-18 October 2012
Grand Nile Tower, Hotel
Cairo - Egypt

**President of the
conference & Society
Prof. Faiza Hammouda**

**Vice President
Prof. Amal El Beshlawy**

**Secretary General
Prof. Azza Kamel**



Welcome Message:

Once again we meet in the 9th International Conference of the Egyptian Society of Haematology & Research (ESHR) that will be held at Grand Nile Tower Hotel, Cairo, on 17-18 October, 2012.

On behalf of the scientific and organizing committees, we would like to invite you to attend the most enlightening experience in Haematology.

The Conference will cover the different aspects of both clinical and laboratory haematology including Thrombosis and haemostasis, Anaemia, Oncologic Haematology, BMT and Haematomorphology. National as well as International figures in haematology will address the conference with state of art lectures in the various topics.

Finally we hope this conference will help to enhance the clinical and laboratory skills and knowledge of participants and enable them to discuss with speakers all aspects of Haematology.

President of the Conference
Prof. Faiza Hammouda

Organizing Committee:

President : Prof. Faiza Hammouda
Vice President : Prof. Amal El Beshlawy
Secretary General : Prof. Azza Kamel
Moderator : Prof. Magdy El Ekiaby
Moderator : Prof. Somaya El Gawhary

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Prof: Dalal Hindawy
Prof: Hussein Khaled
Prof: Mervat Matter
Prof: Nevine Kassem
Prof: Azza Moustafa
Prof: Hossam Kamel
Prof: Magda Assem
Prof: Mohamed Raafat Khalaf

Scientific Committee:

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Prof. Magda Sultan
Prof. Mervat Matter
Prof. Mohamed Raafat Khalaf
Prof. Mona El Tagui
Prof. Normine Kaddah
Prof. Ossama El Safi
Prof. Sheble Said Sheble
Prof. Somaya El Gawhary



**9th International Conference of
the Egyptian Society of
Haematology & Research (ESHR)
Update in Hematology**

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Prof. Azza Kamel

**Under The Patronage Of
Prof.Dr. Mohamed Mostafa Hamed
Minister of Health and Population**

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WELCOME MESSAGES

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Thank you all and welcome again to the 9th International Conference of the Egyptian Society of Hematology & Research (ESHR).

President of the Conference
Prof. Faiza Hammouda

COMMITTEES

Organizing Committee:

| | |
|-------------------|---------------------------|
| President | : Prof. Faiza Hammouda |
| Vice President | : Prof. Amal El Beshlawy |
| Secretary General | : Prof. Azza Kamel |
| Moderator | : Prof. Somaya El Gawhary |

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| Prof. Ahmed Selim | Prof. Alaa Haddad |
| Prof. Azza Moustafa | Prof. Hamdy Abdel Azim |
| Prof. Heba El Zawahry | Prof. Hossam Kamel |
| Prof. Hussein Khaled | Prof. Magda Assem |
| Prof. Magdy El Ekiaby | Prof. Mervat Mattar |
| Prof. Nevine Kassem | Prof. Somaya El Gawhary |

Scientific Committee:

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| Prof. Mohamed Badr | Prof. Mohamed Raafat Khalaf |
| Prof. Mona El Kasas | Prof. Mona El Tagui |
| Prof. Nevine Kassim | Prof. Normine Kaddah |
| Prof. Omar Fahmy | Prof. Ossama El Safi |
| Prof. Salwa Youssef | Prof. Sheble Said Sheble |
| Prof. Sherif Abo El Naga | Prof. Somaya El Gawhary |
| Prof. Youssef El Tonbary | |

GENERAL INFORMATION

Official Language:

The official language of the congress is English.

Time Difference:

Egypt time is 2 hours ahead of Greenwich Mean Time (GMT+2).

Climate:

Egypt has a warm and sunny climate all year round, although on the whole it can be best described as mild. While the mid summer months can get quite hot, the heat is less taxing than else-where because of low humidity.

For the rest of the year the weather is ideal, and sunny. Rainy days are few and far between in Cairo, and nearly unknown in Upper Egypt.

Therefore, it would be wise to pack both lightweight and warm clothing.

Electricity:

Electricity Outlets for 220 volts are dominant in Egypt. Always check the power supply before using your equipment.

Liability and Insurance:

The Organizing Committee will take no liability for personal injuries sustained by or for loss or damage to property, belongings of congress participants or accompanying persons, either during or as a result of the congress or during their stay in Egypt. It is, therefore, advised that participants arrange their own personal health, accident and travel insurance.

Business Hours:

Friday is the official weekend. Most embassies are closed Friday and Saturdays, but few close on Saturdays and Sunday. Shops are generally open from 9:00 to 21:00 hours and most of them close on Sunday.

Tipping:

Whilst tipping is not essential, people who provide a service, for example, hotel porters, waiters, drivers and guides generally expect some tipping. There is no set amount of tip given, it is left to the individual as appreciation of service provided.

Badges:

You will receive your name badge on registration. For security and administrative reasons you should wear your name badge throughout the conference, breaks, and exhibition hall.

Certificate of Attendance:

Certificate of Attendance will be delivered on the second day at the registration desk .

Coffee Breaks:

It will be served in the foyer in front of Conference rooms.

Exhibition Hall:

Medical Industry and Pharmaceutical companies will be present in the foyer in front of the Conference rooms. Please feel free to visit the medical exhibition during the breaks.

Information Desk:

For any inquiries please contact the organizers .

Mobile Phones:

Mobile Phones must be switched off inside the meeting rooms.

Lost and Found:

For your missing or lost items contact the Conference Information Desk.

Medical Emergencies:

Please contact the emergency phone numbers or Conference Information Desk.

Preview Room :

All Speaker are kindly requested to deliver their presentation at least 2 hours before their talk to the slide room which will be beside the meeting room.

Conference Secretariat:**Pioneer Events**

30 Anwar El Mofti St., Nasr City

Cairo ,Egypt

Tel : 202 24046672 – Fax :202 24020609

E-mail:info@pioneer-events.org

PROGRAM AT A GLANCE**Wednesday 17/10/2012**

| | |
|---------------|---|
| 09:00 –10:00 | Opening Ceremony |
| 10:00 –11:15 | Anemia and BM Failure Syndromes |
| 11:15 – 11:45 | Coffee Break |
| 11:45 –12:30 | Plenary I |
| 12:30 –13:30 | Novartis Symposium |
| 13:30 – 14:30 | Hemato-oncology I (State of Art Lectures) |
| 14:30 – 15:00 | Coffee Break |
| 15:00 – 16:30 | Thrombosis and Hemostasis |
| 16:30 – 18:00 | Egyptian Society of Hematology and Research (ESHR) & Egyptian Society of Hemophilia (ESH) Spotlight on Problems of patients with Hemophilia |
| 18:00 – 18:30 | Lunch |

Thursday 18/10/2012

| | |
|---------------|-------------------------------------|
| 09:00 – 10:30 | Stem Cell Therapy |
| 10:30 – 11:00 | Coffee Break |
| 11:00 – 12:30 | Hemato-oncology II |
| 12:30 – 13:30 | Free Papers I |
| 13:30 – 14:00 | Coffee Break |
| 14:00 – 15:00 | Free Papers II |
| 15:00 – 17:00 | Case Presentation &Hematomorphology |
| 17:00 – 17:30 | Lunch |

PROGRAM DETAILS

Wednesday 17/10/2012

Opening Ceremony (09:00 – 10:00)

Anemia and BM Failure Syndromes (10:00 – 11:15)

Chairpersons:

Prof. Normine Kaddah
Prof. Galila Mokhtar
Prof. Amina Hassab
Prof. Hoda Hassab

10:00 – 10:20 Dyserythropoietic Anemias
(Amal El-Beshlawy)

10:20 – 10:45 Refractory Anemias
(Mona El-Tagy / Azza Mostafa)

10:45 – 11:05 Bone Marrow failure
(Mona Wagdy)

11:05 – 11:15 Discussion

11:15 – 11:45 Coffee Break

**Plenary I: (11:45-12:30)
(In the honor of Prof. Dr. Faiza Hammouda)**

Chairpersons:

Prof. Faiza Hammouda
Prof. Azza Kamel
Prof. Mervat El-Ansary

11:45 – 12:30 Induced Pluripotent Stem Cells (iPS Cells) and Future of Regenerative Medicine
(Emin Kansu: Turkey)

Wednesday 17/10/2012

Novartis Symposium

(12:30 – 13:30)

Chairpersons:

Prof. Dr. Lamis Ragab
Prof. Dr. Mona El-Tagy
Prof. Dr. Hamdi El-Zawam
Prof. Dr. Mervat Mattar

12:30 – 13:00 Impact of iron Chelation on the management and prevention of thalassemia complications
(Amal El-Beshlawy)

13:00 – 13:15 Second generation TKIs as first line treatment in CML
(Raafat Abdel-Fattah)

13:15 – 13:30 When to shift to second line treatment
(Mervat Mattar)

**Hemato-oncology I
State of Art Lectures**

(13:30-14:30)

Chairpersons:

Prof. Hossam M Kamel
Prof. Ashraf El-Ghandour
Prof. Sameh Shamaa

13:30–14:00 Clinical Advances in Malignant Lymphoma in the last decade
(Hussein Khaled)

14:00 – 14:30 AML: Update
(Alaa El-Haddad)

14:30-15:00 Coffee Break

Wednesday 17/10/2012

Thrombosis and Hemostasis (15:00 – 16:30)

Chairpersons:

Prof. Amal El-Beshlawy
Prof. Nevine Kassem
Prof. Youssria Abdel-Rahman
Prof. Shebl S. Shebl
Prof. Mona El-Kassas

- 15:00-15:20 What is new in ITP
(Alia Abdel-Aziz)
- 15:20-15:40 Antiphospholipid Syndrome:Update
(Nevine Kassem)
- 15: 40-16:00 Malignancy and thrombosis: A complex interacting scenario
(Manal Ghozlan)
- 16:00-16:20 Outreach for Von-Willebrand Disease: Update
(Magdy El Ekiaby)
- 16:20-16:30 Discussion**

Wednesday 17/10/2012

**Egyptian Society of Hematology and Research (ESHR)
& Egyptian Society of Hemophilia (ESH) Conjoint Session
(16:30-18:00)**

Spotlight on problems of patients with Hemophilia

Chairpersons:

Prof. Amal El-Beshlawy
Prof. Nadia Moharram
Prof. Mohammed Badr
Prof. Magdy El-Ekiaby

- 16:30-16:45 Hemophilia in Egypt
(Nadia Moharram)
- 16:45-17:00 Hemophilia Care in an Egyptian Health Insurance
Hospital (Children of Egypt Hospital)
(Naglaa Shaheen)
- 17:00-17:15 Daily challenges of a patient with Hemophilia
(Ossama El Tahan)
- 17:15-17:45 Together delivering life-changing
(Susanne Brunner)
- 17:45-18:00 Open discussion
- 18:00 – 18:30 Lunch**

Thursday 18/10/2012

Stem Cell Therapy (09:00-10:30)

Chairpersons:

Prof. Mohammed Khalaf
Prof. Mohamed Awad
Prof. Youssef El Tonbary
Prof. Osama El-Safy

- 09:00-09:25 The Allo Dilemma...
A recipient with no donor! Is Haplo-identical transplant
the answer in Egypt ?
(Mohammed Khalaf)
- 09:25-09:50 Graft Vs. Host Disease
(Mohammed Abdel-Moeti)
- 09:50-10:15 The Empty Marrow
(Omar Fahmy)
- 10:15-10:30 Spinal Cord regeneration following MSC injection:
Evidence-based study
(Hala Gabr)
- 10:30-11:00 Coffee Break**

Thursday 18/10/2012

Hemato-oncology II (11:00- 12:30)

Chairpersons:

Prof. Aida Nazeer
Prof. Mostafa Nassar
Prof. Mahmoud Salah
Prof. Enas Asfour

- 11:00-11:25 Autophagy: Role of self-eating
(Hanan Hamed)
- 11:25-11:50 Myeloproliferative Neoplasms (MPN):
A single center experience
(Mervat Mattar)
- 11:50-12:15 AML: The ever growing complexity of molecular genetics
(Magda Assem)
- 12:15-12:30 Discussion**

Free Papers I (12:30- 13:30)

Chairpersons:

Prof. Dr. Hoda Seoud
Prof. Dr. Nabih Fadali
Prof. Dr. Taghreed Gaafar
Prof. Dr. Maha Akl

- 12:30 -12:40 Increased circulating red cell microparticles (RMP) and platelet
microparticles (PMP) in immune thrombocytopenic purpura
(Eman Sewify)
- 12:40-12:50 Platelets antibodies and serum leptin in childhood
Immune thrombocytopenic purpura
(Hosny Badrawy)
- 12:50-13:00 DNA diagnosis of low HbA2
(Mohamed Samir A Khalil)

- 13:00-13:10 The Effect of Hydroxyurea on adhesion receptor Integrin-associated protein (CD47) expression in patients with Sickle Cell Disease
(Mohammed A Soliman)
- 13:10-13:20 Hypoxia, oxidative stress biomarkers and circulating microparticles in pediatric Upper Egyptian thalassemic patients
(Khaled EI-Sayeh)
- 13:20-13:30 Alloimmunization in patients with transfusion-dependent β -thalassemia major and acute leukemia in Upper Egypt
(Asmaa Zahran)
- 13:30-14:00 Coffee Break**

Free Papers II (14:00- 15:00)

Chairperson:

Prof. Dr. Magda Assem
Prof. Dr. Adel Abdel-Rehim
Prof. Dr. Nabila Thabet
Prof. Dr. Saad EI-Esh

- 14:00-14:10 Snake (*Walterinnesia aegyptia*) venom combined with silica nanoparticles induces apoptosis and growth arrest of human multiple myeloma cells through its direct effects on the DNA
(Douaa EI-Sayed)
- 14:10 -14:20 Dendritic cell based-therapy in Chronic Myeloid Leukemia
(Manal EI-Wahsh)
- 14:20-14:30 Flowcytometric immunophenotyping for diagnosis of myelodysplastic syndrome Patients
(Amany H. Mansour)
- 14:30- 14:40 The reliability of endothelial progenitor cells (CD34+, KDR+ and CD34+/KDR+) in diagnosis of coronary artery disease and its severity ?
(Eman Sewify)
- 14:40-14:50 Anti-HBc and HBV-DNA detection in blood donors negative for hepatitis B virus surface antigen
(Rania Bakry)

Thursday 18/10/2012

Case presentation & Hematomorphology (15:00-17:00)

Chairperson:

Prof. Nadia Mowafi
Prof. Azza Mostafa
Prof. Tayseer Eiada
Prof. Somaya El Gawahary
Prof. Ahmed Selim

Presenters: in alphabet order

Alaa- El-Haddad
Douaa sayed
Eman Kamal
Eman Zaghloul
Hala Farawela
Hend Nabil
Homam Sharshera
Magda Sultan

17:00 Lunch

ABSTRACT

Congenital Dyserythropoietic Anemias

Prof. Amal El-Beshlawy

Pediatric Hematology Department, Cairo University

Dyserythropoietic anemias are highly heterogeneous set of anemias that result from various kinds of abnormalities during late erythropoiesis. This types of anemias can be primary e.g. B-thalassemia sideroblastic anemia, thiamin responsive anemia and the most important congenital Dyserythropoietic anemias. Other variable etiological causes e.g. B12 and Folate deficiencies, benzene intoxication and others can cause secondary dyserythropoietic anemias.

Congenital Dyserythropoietic Anemias (CDA), are characterized by congenital anemia with suboptimal reticulocyte response and abnormal bone marrow red blood cell precursors. Ineffective erythropoiesis, Iron overload which can cause organ dysfunction, cholelithiasis and hepatosplenomegaly.

There are three types: CDA-I which can start any time between the neonatal period and late adulthood. Bone marrow shows erythroid hyperplasia with megaloblastoid appearance and characteristically intranuclear bridges between erythroblasts with figures ranging from 0.5% to 8% requiring prolonged searching. By electron microscopy 40% to 60% of the intermediate and late erythroblasts show a characteristic heterochromatin pattern (Swiss cheese like) which is the gold standard for diagnosis of CDA-I. In some cases of CDA-I there may be increased Hb A2 and increased α /non α globin chain synthesis. This thalassemia - like features are most likely secondary to dyserythropoiesis.

CDA-II: which presents late in life with mild symptoms, Jaundice in 80% of cases and splenomegaly in 70%. Iron overload with liver cirrhosis, cardiac or endocrine dysfunction can be present. Bone marrow is hypercellular with normoblast appears with large number of binuclear Erythroblasts (10 %- 35%) of similar size. CDA-II can be confused with hereditary spherocytosis but the inadequacy of the reticulocyte count for the degree of anemia can differentiate the two conditions.

CDA – III : Very rare and may be associated with angiod streaks, gammopathies and myeloma.

Conclusion

Congenital Dyserythropoietic Anemias should be considered when we are facing a patient with thalassemia like syndrome not proved to be thalassemia. It should also be considered in un diagnosed congenital spherocytosis anemia.

iPS Cells and Regenerative Medicine

Emin Kansu, M.D, FACP

Hacettepe University Cancer Institute, Ankara, Turkey

Embryonic stem cells are derived from the inner cell mass of blastocyte having high potential of self-renewal capacity and differentiate into three germ layers. In recent years, Yamanaka and co-workers demonstrated that mouse and human somatic cells could be re-programmed into pluripotent embryonic stem cell stage by adding Oct-4, Nanog, Sox-2 and Klf-1 (Yamanaka cocktail) transfer factors. These newly formed cells are called “Inducible Pluripotent (iPS) Cells” and believed to be a major advance in the stem cell research and promising breakthrough in regenerative medicine for the future. These iPS Cells can be produced from any adult cell by transduction with re-programming of Yamanaka factors.

Shinya Yamanaka (Japan) and John B. Gurdon (U.K.) have received **2012 Nobel Prize in Medicine or Physiology, on October 8, 2012**, for their novel discoveries. iPSCs have highly similar features to embryonic stem cells, namely ability to differentiate into different cell types and to produce viable mice with mouse iPSCs. The generation of iPS Cells represents a state of “*turning cell to anti-clockwise*”. iPSCs generation involves several molecules and “re-programming” of adult cell nuclei. Adult stem cells and well-differentiated cells can be re-programmed but efficiency may show wider range of viability. Adipose-derived stem cells can be induced into osteogenic, chondrogenic, hepatic, neurogenic, cardiogenic and adipogenic lineages.

iPS Cells can also be generated from human somatic cells, including patients with diseases. Mature cells including nerve, heart and liver cells can be derived from these iPS cells, thereby allowing scientists to study

disease mechanisms in novel ways. Induced pluripotent stem cells (iPSCs) constitute an alternative source of autologous cells that are amenable to ex-vivo expansion, genetic correction and molecular characterization. The unlimited proliferative capacity of iPSCs is particularly attractive with regard to regenerative therapies and disease models. The inability of human iPSCs to yield transplantable hematopoietic progenitors, in combination with concerns about cell numbers, viral integration sites in iPSCs and teratoma formation in vivo, currently precludes the clinical translation of this technology.

iPS cells have an attractive and unique capacity to differentiate into all cell types in the living organism. If we can capture the genomic basis of a disease it may be possible to duplicate the pathogenesis of the patient's condition in iPS cells. iPS cells can be generated in vitro and can be potentially useful in pathogenesis and diagnosis of diseases such as in neuropathies and cardiomyopathies, drug – screening, pharmacologic testing, tissue engineering, cell transplantation and in the future for customized therapies.

References:

1. Rawalho-Santos M. iPSC cells: Insights into Basic Biology. *Cell* 138;616-618, 2009.
2. Dey D, Evans GRD. Generation of Induced Pluripotent Stem (iPS) Cells by Nuclear Re-programming. *Stem Cells International* 2011:1-11, 2011.

Chelation Therapy : The way of Prevention and Management of Thalassemia Complications.

Prof. Amal El- Beshlawy, Professor of Pediatric Hematology, Cairo University

Combination of transfusion and chelation therapy has dramatically extended the life expectancy of thalassemia patients. Without chelation a significant clinical manifestations of iron toxicity can be expected; impaired growth, cardiomyopathy, hepatic cirrhosis, diabetes mellitus and hypogonadism.

Deferoxamine subcutaneous infusion prolonged the life expectancy of patients remarkably but the compliance of the patients is very poor . With the discovery of the oral iron chelators deferriprone and deferasirox (EXJADE) the compliance of the patients to chelation therapy is remarkably improved .Deferasirox (EXJADE) as an oral dispersible tablets to be taken once per day from very young age improves the compliance of the iron overloaded patients in Egypt and worldwide to chelation therapy.

By the use of deferasirox reduction of the liver and cardiac iron burden and complications is remarkable by the use of the most recent technology of MRI techniques (T2* & R2). Prevention of iron overload toxicity is achieved by the regular monitoring of iron burden in the liver and heart of patients under chelation by deferasirox (EXJADE).

Proper management of iron overload is the main issue in prevention of iron toxicity and complications in thalassemics. Regular monitoring of the patients different organs function is needed for better outcome of patients management.

Second Generation TKI in newly diagnosed CML-CP

Prof.Raafat Abdel Fattah,MD

With first-line imatinib 400 mg/day treatment of CP CMI31-43%fail to achieve a CCyR by 12 month, 60-88% fail to achieve a MMR by 12 months, Patients with ECP CML who fail to achieve an early CCyR or MMR are at increased risk of disease progression.High-risk patients have significantly worse long-term outcomes on imatinib.Poor adherence to imatinib treatment reduces response rates.Discontinuation rate ~45%.

Both Nilotinib and Dasatinib are more potent than imatinib.Early signs of decreased transformation andmore rapid CCyR means fewer BM aspirations procedures will be necessary to monitor patients.Higher likelihood of CMR achievement may provide more patients with a possibility of discontinuing therapy.Lack of serious irreversible toxicity is important in CP-CML patients .So the use of 2nd generation TKI in newly diagnosed CML-CP isencouraging.

What The New in ITP

Prof. Alia Abdel Aziz

Immune thrombocytopenia (ITP) is commonly encountered in clinical practice. Our approach to ITP has undergone a number of changes over the past several years. There is a plethora of novel agents and new informations. The most fundamental area of change has been in our understanding of pathophysiology. The “old” concept was that thrombocytopenia resulted from antibody-mediated platelet destruction. There are now “new” concepts the most developed is that the same antibodies that mediate platelet destruction also mediate impaired platelet production by damaging megakaryocytes and/or blocking their ability to release proplatelets. Therapy also has been affected by these new advances. The most obvious has been the development and licensure of two thrombopoietic agents.

Other new therapeutic developments include use of more aggressive treatment upfront in newly diagnosed adults fatigue and thrombophilia has now been shown in a number of patients.

VWD Outreach

Magdy El Ekiaby, MD

Head of Shabrawishi Hemophilia Treatment Center

VWD is a hereditary bleeding disorder caused by inherited dominant or recessive defects of VWF. These defects may be quantitative or qualitative. The defect causes increased tendency of bleeding in the affected person. Muco-cutaneous bleeding is particularly a prominent feature of the bleeding tendency, although other forms of bleeding may occur. The prevalence of the disease is mentioned to be up to 1% of the population, but most of the affected individuals have mild bleeding symptoms and in most of the cases may not be diagnosed.

Outreach for patients with VWD requires to target populations that may be at risk of having the disease. Since the disease affects both males and females, menstruation is of particular importance as many of VWD female patients present mainly by menorrhagia. On the other hand, menorrhagia is a common problem in females during their fertile period. Due to these last two facts, outreach for patients with VWD may best be approached among women with menorrhagia. Other target groups include screening relatives of diagnosed patients with VWD. Increasing awareness among health professionals about VWD is an important way to refer suspect cases, and consequently diagnosis of patients with VWD.

Aim of the study: The study is an epidemiological surveillance to identify prevalence of VWD among women with menorrhagia due to dysfunctional uterine bleeding (DUB), relatives of cases with VWD and referred patients due objective bleeding history.

Patients and methods:

Patients: Females with Dysfunctional uterine bleeding, relatives of cases with VWD and referred cases with objective bleeding history

Methods

1-Bleeding scoring sheet for subjective evaluation of patient bleeding history

2-Menorrhagia scoring sheet

3-CBC & Ferritin

4-Prothrombin time, aPTT& TT

5-FVIII assay, VWFRiCof& antigen

6-VWF collagen binding & FVIII binding assays for classification of the type of VWD in diagnosed cases

6-Other clotting factor assays, when there is a need as well as platelet function test

Results and conclusion

75 patients complaining from menorrhagia 46 women with DUB were diagnosed. Out of 46 women, 7 were diagnosed as having VWD with a prevalence rate of 15%. These data suggest that females with menorrhagia due to DUB should be thoroughly investigate for the possibility of suffering from VWD or other hereditary bleeding disorders.

The Allo Dilemma...

A recipient With No Donor .. Is Haplo-identical transplant the Answer in Egypt ?

Mohamed Khalaf, MD

Head, Department of Hematology and Stem Cell Transplantation

Maadi Armed Forces Medical Compound,

Cairo, Egypt

Allogeneic Stem cell transplant confers a genuine curative modality for many benign and malignant conditions. It ranks in the front for many and is the only hope for a lot.

Unfortunately only less than one third of eligible and fit patients will have this option possible by having a suitable MHC-matched sibling. For the remaining majority, however, an allograft can still be done in most if a suitable matched unrelated donor or a reasonable cord is found and accessed in a timely fashion. The search procedure, the slow logistics are cumbersome and the cost is formidable to make these alternate stem cell sources unrealistic answer in Egypt.

Haplo-identical graft is an alternative, readily available source that is feasible for most if not all such patients. Results of such procedures that violate the hematopoietic stem cell transplantation rules had been very disappointing in the past. The resilient efforts worldwide from Germany, Italy, China and USA had made great astride in improving the outcome.

Results of pilot works performed at our center in the last decade will be discussed and the hurdles and promise of implementing such transplants in Egypt will be addressed.

Increased Circulating Red Cell Microparticles (RMP) and Platelet Microparticles (PMP) in Immune Thrombocytopenic Purpura

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It has been suggested that patients with ITP have an increased thrombotic risk compared to the general population and compared to those with other causes of acquired thrombocytopenia. The procoagulant role of microparticles in some clinical situations has been reported, yet, very few data is available about microparticles in ITP and their effect.

Aim of the work: To assess the levels of red cell microparticles (RMP), platelet microparticles (PMP) and their possible relation to some haemostatic parameters in ITP patients

Patients and methods : The levels of RMP and, PMP in addition to FVIII, FIX, FXI, PC and aPTT were assessed in 29 patients with chronic ITP (8 of them had splenectomy). Ten apparently healthy volunteers served as control. We compared the levels of the studied parameters in ITP patients with that in controls. Correlations of these parameters with each other and with the platelet count were studied.

Results: RMP ($p = 0.0001$), PMP ($p = 0.0001$), FVIII ($p = 0.049$), FIX ($p = 0.0001$) and FXI ($p = 0.0001$) were significantly higher in ITP patients compared to controls. PTT was significantly longer in ITP patients ($p = 0.0001$) but PC showed no significant difference, however RMP was associated with shorter PTT. Generally, the coagulation factors were

negatively correlated with platelet count in ITP patients. Compared to control, ITP patients preserved higher levels of RMP and PMP even in those with near-normal platelet count. Splenectomy was associated with lower FIX ($p = 0.0001$) and FXI ($p = 0.028$) and higher RMP ($p = 0.0001$).

In conclusion: Chronic ITP is associated with increased levels of RMP and PMP. FVIII, FIX and FXI increased in ITP patients but showed a negative correlation with platelet count. Splenectomy is associated with increased levels of RMP and lower levels of FIX and F XI. The high level of microparticles in ITP might point towards a prothrombotic tendency.

Platelets Antibodies and Serum Leptin in Childhood Immuno-thrombocytopenic Purpura

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Background and aim: Platelets antigens, Anti-platelets antibodies, serum leptin measurement may be important in defining the pathogenesis of thrombocytopenic states.

Methods: In this study we measured the platelets CD41, CD61, CD62P, Platelets IgG, IgM by flow cytometry and serum leptin by ELISA of 20 children diagnosed as ITP and 20 normal children as control.

Results: We observed that there were no significant difference in white blood cells count, hemoglobin concentration between ITP patients and controls. Platelets count was significantly decreased, and mean platelet volume (MPV) was significantly increased in patients than controls $P=0.000$. The percentage of CD41-expressing platelets was significantly lower in ITP children compared to controls ($P=0.001$) but the percentage of CD61-expressing platelets was not significantly different between ITP patients and controls. Platelet activation marker CD62P was significantly expressed in patients than controls (0.000). Furthermore, the amount of CD62P per cell, represented by the MFI was significantly higher in patients than controls (0.000). The percentage of platelets associated IgM and IgG were significantly increased in patients than controls ($P=0.000$). Also the MFI of IgM and IgG were significantly higher in patients than controls. Finally the concentration of serum leptin was increased in patients than controls ($P=0.000$) (table 2). There was a negative correlation between the platelets count and Platelets IgG ($P=0.000$ and $r=-0.88$).

Conclusion: We concluded that the demonstration of antiplatelet antibodies (PAIgG, PAIgM), decreased detection of platelet surface antigens (CD41, CD61) and increase the serum leptin level in children with immune thrombocytopenic purpura (ITP) have a diagnostic and pathogenetic role.

Dna Diagnosis of Low Hb α_2

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Background: Mutations in the δ -gene are clinically silent, thus of no clinical significance, yet δ -thalassaemia/variant can mask the presence of β -thalassaemia trait.

Patients and Methods: One hundred twenty cases with a suspected HbA α_2 variant or δ -thalassaemia were collected for DNA analysis. Cases were selected by HPLC showing a shifted HbA α_2 peak or a reduced amount of HbA α_2 .

Results: Ninety two cases (76.7%) were diagnosed as HbA α_2 ' (HbB α_2). Ten different variants were identified, including three novel ones. The novel mutations were HBD:c.157G>C, HBD:c.244C>T and HBD:c.7C>A. The other results were: one case carried HbA α_2 -Indonesia (cd 69 GGT@CGT); 2 cases carried the HbA α_2 -Coburg (cd 116 CGC@CAC); 4 patients carried the HbA α_2 -Babinga (cd 136 GGT@GAT); 2 cases carried the δ -thalassaemic mutation (cd 4 (ACT@ATT) delta+); 10 cases carried the HbA α_2 -Yialousa (cd 27 GCC@TCC); 1 case carried the HbA α_2 -Troodos (cd 116 CGC@TGC) and 1 case was homozygous for the thalassaemic mutation -68 (C→T).

Conclusions: We are reporting 3 novel δ -globin gene mutations (HBD:c.157G>C, HBD:c.244C>T and HBD:c.7C>A). Also, we can conclude that 4 δ -globin gene mutations (HbA α_2), HbA α_2 -Indonesia, HBD:c.157G>C and HBD:c.244C>T) had a characteristic shifted retention time on HPLC that can be used for a probable diagnosis.

The Effect of Hydroxyurea on Adhesion Receptor (CD47) Expression in Patients With Sickle Cell Disease

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Background: Patients with sickle cell disorders (SCD) are prone to episodes of micro vascular obstruction. CD47 is one of the important participating adhesion molecules in this process.

Aim of this work: To study the role of RBCs CD47 expression as a predictor for severity of sickle cell disease and its relation to Hb F % and hydroxyurea therapy.

Subject and methods: The study involved 48 sickle cell disease children, divided into three groups: G1; patients not on hydroxyurea (HU) therapy in steady state, G2; patients on HU in steady state, and G3; patients in painful crisis at the time of evaluation. Twenty normal children were involved as a control group. For all individuals RBCs CD47 expression by flowcytometry expressed as % of positive cells was performed.

Results: The mean RBCs CD47 expression was significantly higher in the patient group compared to the control group. The mean CD47 expression in G1 (87.25 ± 8.46) was lower than in G3 (89.04 ± 5.14) but higher than in G2 (85.74 ± 18.37) yet the difference was not significant ($p > 0.05$). CD47 expression was significantly positively correlated with both WBCs count and ANC with non significant negative correlation with HbF%.

Conclusion: The adhesion molecule CD47 expression could be a contributing factor to acute and chronic vaso-occlusion characteristic of SCD.

Key words: Sickle Cell Disease, Adhesion molecules, Integrin-associated protein CD47, Hydroxyurea.

Oxidative stress, Hypoxia biomarkers and circulating microparticles in pediatric Upper Egyptian thalassemic patients

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This study aimed to investigate the oxidative stress, hypoxia biomarkers and circulating microparticles in β thalassemia major. The study included 56 thalassemic children and 46 healthy controls. Hypoxia biomarkers, oxidative stress biomarkers and total plasma fragmented DNA (fDNA) were detected by the standard methods. Microparticles were assessed by flow cytometry. Hypoxia and oxidative stress biomarkers, fDNA and microparticles were higher and total antioxidant (TAC) was lower in thalassemic patients than the controls. In splenectomized patients, vascular endothelial growth factor, malondialdehyde, fDNA, endothelial, platelet and activated platelet microparticles were higher and TAC was lower than in non splenectomized patients. In conclusion; the increased tissue hypoxia, oxidative stress in β thalassemia and its relationship with DNA damage and microparticles release could explain many complications of thalassemia and may have therapeutic implications. Drugs with antioxidant properties, or diets with high antioxidant content, could reduce severity of thalassemia.

Key word: β thalassemia major, fragmented DNA, circulating microparticles, oxidative stress, hypoxia

Alloimmunization in Patients With Transfusion-Dependent B-Thalassemia Major and Acute Leukemia in Upper Egypt

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Background: Repeated blood transfusions, is associated with the development of alloantibodies against red blood cells (RBC) antigens.

Objective: We evaluated the frequency of alloimmunization and factors influencing it in children with β -thalassemia major and those with acute leukemia in our locality.

Patients and methods: The study was carried out on 85 multiply transfused pediatric patients with β -thalassemia major and 75 pediatric patients with acute leukemia. Informed consents were obtained from patients' parents. Clinical and transfusion records of all studied patients were reviewed. Indirect antiglobulin test was performed to detect the presence of alloantibodies by standard blood bank procedure.

Results: Red cell alloantibodies were detected in 7.1% β -thalassemia major patients and in 2.7% acute leukemia patients. 11.8% of acute myeloid leukemia (AML) patients and none of acute lymphoblastic leukemia (ALL) patients had alloantibodies. In thalassemic patients, anti-K antibodies were the commonest (66.7%), followed by anti-C (50%), anti-E (33.3%), anti-cw (16.7%) and anti-D (16.7%). In acute leukemia, anti-E and anti-K antibodies were detected in the 2 patients, and anti D were detected in one patient

Conclusion: Red cell alloimmunization is an important problem in patients with transfusion dependent β thalassemia and patients with AML. Red cell alloantibody formation was influenced by age at first transfusion, number of blood transfusions and splenectomy. In our patients, transfusion of phenotypically-matched blood for the Rh (D, C, c, E, e) and Kell systems in addition to ABO systems seems to be effective in preventing alloimmunization.

Increased Circulating Red Cell Microparticles (RMP) and Platelet Microparticles (PMP) In Immune Thrombocytopenic Purpura

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It has been suggested that patients with ITP have an increased thrombotic risk compared to the general population and compared to those with other causes of acquired thrombocytopenia. The procoagulant role of microparticles in some clinical situations has been reported, yet, very few data is available about microparticles in ITP and their effect.

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Patients and methods: The levels of RMP and, PMP in addition to FVIII, FIX, FXI, PC and aPTT were assessed in 29 patients with chronic ITP (8 of them had splenectomy). Ten apparently healthy volunteers served as control. We compared the levels of the studied parameters in ITP patients with that in controls. Correlations of these parameters with each other and with the platelet count were studied.

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conclusion: Chronic ITP is associated with increased levels of RMP and PMP. FVIII, FIX and FXI increased in ITP patients but showed a negative correlation with platelet count. Splenectomy is associated with increased levels of RMP and lower levels of FIX and F XI. The high level of microparticles in ITP might point towards a prothrombotic tendency.

Dendritic Cell Based-Therapy in Chronic Myeloid Leukemia

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Persistence of residual leukemia cells in chronic myeloid leukemia (CML) will eventually lead to a relapse of the disease. Dendritic cell-based vaccines might constitute a therapeutic option for leukemia patients to control or eradicate minimal residual disease. This study aimed at generating an autologous DC vaccine expressing leukemia associated antigens differentiated from peripheral blood mononuclear cells to boost the immune system and improve the clinical outcome of CML patients, so this vaccine can be used as an adjuvant therapy for CML patients alongside their chemotherapy. This study included (40) patients diagnosed as having chronic myeloid leukemia in the chronic phase. Only 28 of them attended regularly for our study. They were divided into two groups. Group I included 14 CML patients that were vaccinated with dendritic cells. Group II included fourteen age and sex matched CML patients that were not vaccinated with dendritic cells but were injected with physiological saline under complete aseptic conditions. Twenty ml of venous blood were obtained from each patient to generate DCs by suspending them in liquid culture medium containing granulocyte monocyte colony stimulating factor (GM-CSF) and interleukin 4 (IL-4) and activated by adding tumor necrosis factor alpha (TNF- α). The DCs were identified morphologically and their maturity was assessed by using monoclonal antibodies against CD83. The clinical response was evaluated by monitoring any clinical changes in the patients after vaccination and comparing it with those

before vaccination. The immune response was monitored by measuring serum INF- γ level before and after vaccination. There was some clinical improvement and some immunological response so we conclude that, DC vaccine may be used as an effective adjuvant therapy in CML patients alongside with their chemotherapeutic regimen.

Key words: Chronic myeloid leukemia, dendritic cells, CD83, INF- γ .

Flowcytometric Immunophenotyping for Diagnosis of Myelodysplastic Syndrome Patients

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Background: Myelodysplastic syndromes (MDSs), are a group of malignant myeloid hematopoietic disorders. Diagnosis of myelodysplastic syndrome can be difficult especially in cases with a low blast count and a normal karyotype. Flow cytometry has been used to distinguish myelodysplastic syndrome from non-clonal cytopenias. No one single simple flow cytometric parameter has been proposed to be diagnostic of myelodysplastic syndrome. Methods: Marrow aspirates from 29 patients, including 13 with MDS, 19 with leukemia, who were diagnosed in the Hematological Department in mansoura oncology center and 15 with non-clonal disorders(normal controls) were enrolled in this study. (control group), were analyzed using FCM. Blasts, non-blast myeloid cells, and monocytes were gated based on CD45 expression and side scatter (SSC). The phenotypes were then compared among the 3 groups .

Results: Compared to non-clonal disorders, the granulocytic lineages of MDS showed decreased SSC ($P=0.0001$), increased CD45 intensity ($P=0.004$), decreased CD10-positive granulocytes ($P= 0.030$), and a higher CD56-positive rate ($P=0.002$). Also similar results were obtained in the leukemia group, and these findings were not related to the phenotypes of the leukemic cells. Using blast and monocytic gating, useful parameters for generating a differential diagnosis were not found.). The expression rate of CD123+ was significantly higher in MDS patients than that in normal controls $P <0.01$).

Conclusions: Gating the granulocytic region is a relatively easy method for MDS immunophenotyping. Among the parameters studied, SSC, CD10, CD123 and CD56 were the most useful for differentiating MDS from non-clonal disorders. While immunophenotypic changes in MDS appear to be useful for differentiating MDS from non-clonal disorders.

Keywords: myelodysplastic syndromes·transformation·monocyte ,blast and leukemia

The Reliability of Endothelial Progenitor Cells (CD34+, KDR+ And CD34+/KDR+) in Diagnosis of Coronary Artery Disease and Its Severity

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Bone-marrow-derived endothelial progenitor cells (EPCs) play an integral role in the regulation and protection of the endothelium, as well as new vessel formation. Changes in EPC number and function during coronary heart disease (CHD) allow their use as a biomarker. However, so far, there is no suggested definite level at which we can diagnose CAD or determine the severity of the disease.

Aim of the work: is to assess the sensitivity, specificity and accuracy of the circulating EPCs count to diagnose CHD and to predict the severity of the disease.

Patients and Methods: The percentage and count of circulating EPCs (CD34+KDR+, CD34+ and KDR+) were measured by flow cytometry in 52 patients who underwent diagnostic angiography. The correlation between the level of the EPCs on one hand and the presence or absence of CAD and the Gensini score calculated for each patient on the other hand was done.

Results: 38 patients were found to have CAD and 14 had normal coronaries. For those with CAD, 22 had single vessel disease and 16 had multiple vessel disease. It was found that EPCs (CD34+KDR+% and count and CD34+%) were significantly lower in CAD compared to those with normal coronaries. CD34+KDR+% had AUROC of 0.846 to diagnose CAD with a sensitivity of 45.5%, specificity of 100%, Positive Predictive Value (PPV) of 100%, Negative Predictive Value (NPV) of 64.3% and accuracy of 72.75%. CD34+% and count and CD34+KDR+ count were negatively correlated with Gensini score. CD34+% has an AUROC of

0.802 to diagnose Gensini score of more than 6 with a sensitivity of 100%, specificity of 55.2%, PPV of 69%, NPV of 100% and accuracy of 77.6%

Conclusion: EPCs decrease in patients with CAD compared to those without. CD34+KDR+% can diagnose CAD with sensitivity, specificity, PPV, NPV and accuracy of 45.55, 100%, 100%, 64.3% and 72.75%. CD34% can help to exclude the presence of severe CAD with sensitivity, specificity, PPV, NPV and accuracy of 100%, 55.2%, 69%, 100%, and 77.6% respectively.

Anti-HBc and Hbv-Dna Detection in Blood Donors Negative for Hepatitis B Virus Surface Antigen

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Background: Occult HBV infection is defined as the presence of hepatitis B virus (HBV) DNA in blood or liver tissues in patients negative for Hepatitis B surface Antigen (HBsAg). Those patients may or may not be positive for HBV antibodies. The objective of this study is to determine the presence or absence of HBV DNA in the serum samples from HBsAg negative blood donors. In addition we aimed to assess the magnitude of occult HBV infection and to reduce the risk of HBV infection.

Patients & Methods: Over a period of one year a total of 7340 blood units were collected at blood transfusion center in our locality for the prevalence of HBV infection and 180 HBsAg negative blood specimens were randomly selected for anti- HBcIgM, anti-HBs antibody and HBV DNA.

Results: Ninety seven out of 7340 collected blood units were positive for HbsAg (1.3%). The randomly selected 180 tested donors revealed 7(3.8%) positive for antiHBc IgM and 34 (18.8%) were positive for anti-HBs antibodies. Four out of 7 positive for anti-HBc IgM were also positive for anti-HBs and 2 /180 (1.1%) specimens were positive for HBV DNA by PCR.

Conclusion: Anti-HBc antibody should be tested routinely at any blood transfusion center and if they were positive regardless of anti-HBs titer, the blood should be discarded. Also HBV DNA is preferable to be performed to all blood donors to present completely safe blood transfusion.

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