Main Topics:

- Thrombosis & Haemostasis
- Regenerative Medicine
- SCT
- Anaemia & Haemoglobinopathies
- Gene Therapy
- Hematomorphlogy
- Hematopoietic Malignancy I
- Hematopoietic Malignancy II
- Free Papers

Call For Abstract:

Abstract should be written on computer font 14,Size A4 should be submitted to conference secretariat
The Dead line to receive abstracts 1st September 2010 & we will reply 15th September 2010

Registration Fees:

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

Registration for workshop: (10-12 October 2010) (BMA, BMB & immunhistochenisty)

L.E 600 Workshop
L.E 650 Workshop + Conference per member delegates
L.E 700 Workshop + Conference per non member delegates
L.E 750 Workshop + Conference + Registration for the Egyptian society of Hematology

Contact Information

Conference Secretariat
Pioneer Events
30 Dr. Anwar El Mofti St. Apt. 61 Nasr City, Cairo, Egypt
Tel.: (+202) 24053575 - 24046672
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

7th International Conference

Egyptian Society Of Hematology and Research

Update In Hematology

13 - 14 October, 2010
Ramses Hilton
Cairo-Egypt

President of the Conference & Society
Prof. Faiza Hammouda

Vice President
Prof. Amal El Beshlawy

Secretary General
Prof. Azza Kamel

FIRST ANNOUNCEMENT AND CALL FOR ABSTRACT

Welcome Letter :

Once again we meet in the 7th International Conference of the Egyptian Society of Haematology & Research (ESHR) that will be held at Ramses Hilton Hotel, Cairo, on 13-14 October , 2010.

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 & 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

Members:

Prof. Alaa Haddad
Prof. Dalal Hindawy
Prof. Hussein Khaled
Prof. Mervat Matter
Prof. Nevine Kassim
Prof. Azza Moustafa
Prof. Hossam Kamel
Prof. Magda Assem
Prof. Mohamed Raafat Khalaf

Scientific Committee:

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Prof. Faiza Hammouda
Prof. Hany Hussein
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Prof. Ilham Abdel Karim
Prof. Magda Assem
Prof. MagdyEl Ekiaby
Prof. Mohamed Badr
Prof. Mona El Kasas
Prof. Nevine Kassim
Prof. Omar Fahmy
Prof. Salwa Youssef
Prof. Sherif Abo El Naga
Prof. Youssef El Tonbary

7th International Conference
Egyptian Society Of Hematology and Research
Update In Hematology

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FINAL BOOK

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Welcome Messages

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Thank you all and welcome again to the 7th International Conference of the Egyptian Society of Hematology & Research (ESHR).

President of the Conference

Prof. Faiza Hammouda

Committees

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Prof. Sheble Said Sheble
Prof. Somaya El Gawhary
Prof. Faten Moftah

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 worm 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 on 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 provide.

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 13/10/2010

09:00 - 10:00	Opening Ceremony
10:00 - 11:30	Hemato-oncology I (Lymphoid Malignancies)
11:30 - 12:00	Coffee Break
12:00 - 12:45	Plenary I
12:45 - 13:15	Egyptian Society of Hematology and research (ESHR)&Egyptian Society of Hemophilia (ESH) Conjoint Lecture
13:15 - 14:45	Anemia: Update
14:45 - 15:15	Coffee Break
15:15 - 16:45	Case presentation & Hematomorphology
16:45 - 17:30	Lunch

Thursday 14/10/2010

09:00 - 10:30	Thrombosis and Hemostasis
10:30 - 11:00	Coffee Break
11:00 - 12:30	Hemato-oncology II (Myeloid malignancies)
12:30 - 14:00	Free Papers
14:00 - 14:30	Coffee Break
14:30 - 15:15	Plenary II
15:15 - 16:45	Regenerative Medicine
16:45 - 17:30	Lunch

Program Details

Wednesday 13/10/2010

Opening Ceremony
(09:00 - 10:00)

Hemato-oncologyI (Lymphoid Malignancies)
(10:00 - 11:30)

Chairperson:
Prof. Mostafa Nassar
Prof. Aida Nazeer
Prof. Mohammed Khalaf
Prof. Enas Asfour
Prof. Mohamed Rafaat Khalaf

10:00 - 10:25	The role of PET in lymphoma *(Mohamed Khalaf)*
10:25 - 10:50	GIT Lymphoma *(Ahmed Selim)*
10:50 - 11:15	Immune modulation in MM. *(Hanan Hamed)*
11:15 - 11:30	Discussion
11:30 - 12:00	***Coffee Break***

Wednesday 13/10/2010

Plenary I: (12:00 - 12:45)

Chairperson:

Prof. Faiza Hammouda
Prof. Azza Kamel
Prof. Farha El-Chennawy

12:00 - 12:45	Natural killer cell-based therapy of high risk leukemias *(Lorenzo Moretta: Italy)*

Wednesday 13/10/2010

Egyptian Society of Hematology and research (ESHR) & Egyptian Society of Hemophilia (ESH)

Conjoint Lecture (12:45 - 13:15)

Chairperson:

Prof. Nadia Moharram
Prof. Nabila Thabet
Prof. Alya Abdel Aziz

12:45-13:15	New protocol for prophylactic treatment of patients with Hemophilia A in Egypt *(Magdy El Ektiaby)*

Wednesday 13/10/2010

Anemia: Update (13:15 - 14:45)

Chairperson:

Prof. Lamis Ragab
Prof. Somaya El-Gawhary
Prof. Normine El Kaddah
Prof. Galila Mokhtar
Prof. Mona El Kassas

13:15 - 13:35	Update of management and prevention of thalassemia Complications *(Amal El-Beshlawy)*
13:35 - 13:55	Recent advances in transfusion medicine in anemias *(Nermine El-Desouky)*
13:55 - 14:15	Diagnosis of anemia: From basic to sequencing *(Somaya El-Gawhary)*
14:15 - 14:35	Paroxysmal nocturnal hemoglobinuria (PNH) *(Azza Abo El-Enein)*
14:35 - 14:45	Discussion
14:45 - 15:15	***Coffee Break***

Wednesday 13/10/2010

Case presentation & Hematomorphology
(15:15 - 16:45)

Chairperson:

Prof. Nadia Mowafi
Prof. Tayseer Eiada
Prof. Amina Hassab
Prof. Ahmed Selim

Presenters:

Hannan Hamed
Magda Assem
Nahla El Sharkawy
Magda Sultan
Hommam Sharshera
Doaa sayed

16:45 - 17:30 ***Lunch***

Thursday 14/10/2010

Thrombosis and Hemostasis(09:00-10:30)

Chairperson:

Prof. Nevine Kassem
Prof. Youssria
Prof. Mohammed Badr
Prof. Magdy El Ekiaby

09:00 - 09:20	International consensus on the investigation and management of primary immune thrombocytopenia *(Abdel Rahman Soliman)*
09:20 - 09:40	Criteria for diagnosis of Anti-Phospholipid syndrome: An update *(Nevine Kassem)*
09:40 - 10:00	Rare hereditary bleeding disorders *(Magdy El Ekiaby)*
10:00 - 10:20	Genetic challenge in warfarin therapy *(Manal Fawzy Gabr)*
10:20 - 10:30	Discussion
10:30 - 11:00	***Coffee Break***

Thursday 14/10/2010

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

Chairperson:

Prof. Saad El-Esh
Prof. Nabih Fadali
Prof. Mervat Matter
Prof. Mahmoud Salah
Prof. Hassan Abd El-Ghaffar

11:00 -11:20	Molecular design of targeted therapy in CML *(Mervat Matter)*
11:20 - 11:40	Role of hypomethylating agents in MDS *(Ashraf El-Ghandour)*
11:40 - 12:00	Novel therapies In AML *(Mohemed Abdel Mooty)*
12:00 - 12:20	More than a decade in an Egyptian transplant center: The older the better…! *(Omar Fahmy)*
12.20 - 12.30	Discussion

Thursday 14/10/2010

Free Papers (12:30- 14:00)

Chairperson:

Prof. Dr. Azza Ahmed
Prof. Dr Taghreed Gaafar
Prof. Dr Azza Mostafa
Prof. Dr Hoda Seoud
Prof. Dr Maha Akl
Prof. Dr Mona El Tagy

12:30 - 12:45	D-dimer assay in Egyptian patients with Gaucher disease: Correlation with bone and lung involvement *(Amira A Adly)*
12:45 - 13:00	T-cell CD38 expression in B-chronic lymphocytic leukemia *(Nashwa K Abousamra)*
13:00-13:15	Prognostic significance of cd44v6 in CLL patients: Relation to standard prognostic factors *(Dina Fouad)*
13:15 - 13:30	Procalcitonin compared to other inflammatory markers as a diagnostic tool of infection in patients with hematological malignancy *(Eman El-Sewaify)*
13:30 - 13:45	Bcl2 and Bcl6 genes rearrangements in patients with non-Hodgkin lymphoma: Impact on disease outcome *(Eman Isameal)*
13:45 - 14:00	Serum pleiotrophin (ptn) and ptn gene expression in patients with multiple myeloma: Relation to disease status *(Eman Isameal)*
14:00 - 14:30	***Coffee Break***

Thursday 14/10/2010

Plenary II (14:30-15:15)

Chairperson:

Prof. Amal El-Beshlawy
Prof. Magda Assem
Prof. Sameh Shamaa

14:30 - 15:15	Current Perspectives on Stem Cells and Potential Use in Clinical Medicine *(Emin Kansu, Turkey)*

Thursday 14/10/2010

Regenerative Medicine (15:15 – 16:45)

Chairperson:

Prof. Youssef El Tonbary
Prof. Mohamed Awad
Prof. Osama El-Safy
Prof. Mervat El Ansary

15:15 - 15:45	Mesenchymal stem cells in regenerative medicine: the six year experience of the unit of biochemistry and molecular biology, Faculty of Medicine, Cairo University *(Hazem Atta)*
15:45 - 16:15	Safety controls of in vitro manipulated products for cellular Therapy in humans *(Giovanni Melioli: Italy)*
16:15 - 16:45	Mesenchymal stem cells for clinical usage *(Heba Abdel-Razik)*
16:45 - 17:30	***Lunch***

ABSTRACTS

GASTROINTESTINAL LYMPHOMA

Ahmed Selim

Prof. of Medical Oncology

Faculty of Medicine, Cairo University

GIT lymphoma is typically primary or secondary lymphoma infiltration of the GIT from oropharynx to rectum. Non Hodgkin's lymphoma represents the majority while Hodgkin's lymphoma may very rarely affect the GIT. Gastric lymphoma represents about 70% of the cases and pathologically is divided between MALT lymphoma and diffuse B large cell lymphoma. Lymphoma of the small intestine is the second most common site and the most common histologic types include IPSID-related lymphoma, enteropathy associated T cell lymphoma (EATL), diffuse large B-cell lymphoma, mantle cell lymphoma, Burkitt lymphoma, and follicular lymphoma. Colonic lymphoma is less common and is usually of mantle cell or Burkitt lymphoma type.

Management is primarily depending on identification histopathological type. For gastric MALT lymphoma, treatment is directed to associated pylori infection along with use of surgery, radiation and/or chemotherapy and the prognosis is relatively good. For enteropathy-associated T-cell intestinal lymphoma (EATL), which is most often a sequela of celiac disease; the prognosis is poor and is worse than that of other intestinal B-cell lymphomas. Treatment of diffuse B large cell, Mantle cell, Burkitt's or follicular lymphoma is largely similar to the treatment of same histopathology affecting other sites.

Multidisciplinary approach for management for GIT lymphoma is extremely important because of the peculiar complication that may take place during and after treatment particularly perforation, bleeding and fistulae. These complication may be life threatening and may outweigh the achievements fulfilled by specific anticancer modalities.

Update In Treatment And Prevention Of Thalassemia Complications

By Amal El Beshlawy

Prof. Of Hematology, Cairo University

Combination of transfusion and chelation therapy has dramatically increased the life expectancy of thalassemia patients. Transfusion corrects anemia and enable growth, normal activity and prevents enlargement of the spleen and inhibit bone marrow expansion. Iron overload and transfusion related viral infections are the main complications which can be prevented by proper and adequate chelation therapy together with the updated viral screening of the donated blood. Iron overload can cause cardiac, endocrine and hepatic function impairment. Cardiac disease remain the commonest cause of death in thalassemia especially in developing countries. Improved chelation regimens and medications with the use of T2* MRI scanning enabling the detection of early cardiac siderosis leads to the decline in incidence of cardiac failure in developed countries. Regular assessment of cardiac iron from the age of 10 years. Endocrine deficiencies are common and potentially preventable. Regular assessment of growth every six months, of puberty annually from the age of 10 years and for biochemical evidence of glucose intolerance from the age of 10 years are mandatory for proper management of thalassemia patients. Liver complication is common in thalassemics contributed to many factors mainly hepatic iron toxicity and viral infections. Annual screening for HBV & HCV together with the liver function tests are important for early detection and management of theses viruses. Vaccination against HBV and HAV are important for thalassemia patients. Evaluation of the liver iron consuntration (LIC) by MRI (R2 or T2*) to maintain it below 7 mg/gm dry weight is important for the life long of these patients. In conclusion regular monitoring of the patients different organ functions is a must for the better outcome of the patients management. Cooperation of the hematologist with physicians in specific specialties can give the patient health and life.

Diagnosis Of Anaemia: From Basic To Sequencing

By Prof.Dr.Şomaya El Gawhary

Diagnosis of anaemia should start by peripheral blood .Some nutrional deficienes stem cell disorders, and bone marrow abnormalities will also affect production, function and or morphology of platelets and / or granulocytes .

Finding abnormalities in the leukocytes and or platelets may provide clues as to the cause of the anemia

A. bone Marrow smear & biopsy used when other tests are not conclusive Hemoglobin electrophoresis can be used to identify the presence of hemoglobinopathies.

The most important haemalytic anaemia in our medetrainian area is thalassemia

The Clinical severity of thalassemia major and sickle cell syndromes make them priority genetic diseases of prevention programs through prenatal diagnosis for carrier couples. In corporation of automated DNA sequencing

That enable the characterization of mutation not detected by other mutation specific detection procedures was a prime concern of this work

The use of PCR amplification and direct sequencing have permitted the accurate characterization for unidentified alleles and successfully solved 100% of the examined samples.

Paroxysmal Nocturnal Hemoglobinuria(Pnh)

Prof. Azza Abo El-Enein

Paroxysmal nocturnal hemoglobinuria is an acquired disease due to non malignant clonal expansion of one or several hemopoietic stem cells that have acquired a somatic mutation of the phosphatidylinositol glycan complementation class A gene (PIG-A).

Progeny of the affected stem cells are deficient in glycosyl phosphatidylinositol-anchored protein (GPI-APs). This results in the deficiency of GPI-AP on hematopoietic cells (CD16, CD24, CD52, CD55, CD59, CD58, CD66b/CD67, CD73, CD87, CD90 and CD108). Deficiency of GPI-AP CD55 (decay accelerating factor) and CD59 (membrane inhibitor of reactive lysis) accounts for the complement-mediated intravascular hemolysis that results in intermittent hemoglobinuria, the main manifestation of the disease.

PNH has an average estimated incidence of 2 to 5 new cases per million every year. This, coupled with its protean manifestations, make diagnosing PNH a challenge for even the most astute diagnostician. Classical PNH is usually more conspicuous than hypoplastic PNH.. Patients with PNH classically present with hemolytic anemia, venous thrombosis, and deficient hematopoiesis associated either with marrow aplasia or with a cellular marrow. Some patients show evolution into MDS or acute leukemia. PNH clones may also be present in patients who present with the classic features of aplastic anemia or MDS.] Classical PNH is usually more conspicuous than hypoplastic PNH. Patients typically present with a direct antiglobulin negative hemolytic anemia, hemoglobinuria, and mild to moderate cytopenias

The aim of this presentation is to highlight the pathogenetic mechanisms involved in PNH, as well as the presenting signs and symptoms , possible complications and malignant evolution of the disease. Diagnostic methods will be briefly reviewed, with special emphasis on the flow cytometric assessment of GPI anchor-based assays including using anti CD-55 and CD-59 and the more recent aerolysin assays(FLAER).

International Consensus On The Investigation And Management Of Primary Immune Thrombocytopenia

By

Abdel Rahman Soliman, M.D.
Professor Of Medicine
Hematology And Oncology Unit
Ain Shams University

Primary immune thrombocytopenia (ITP) is an acquired immune-mediated disorder characterized by isolated thrombocytopenia, defined as a peripheral blood platelet count less than 100× 109/L, and the absence of any obvious initiating and/or underlying cause of the thrombocytopenia.

Until recently, the abbreviation ITP stood for idiopathic thrombocytopenic purpura, but current awareness relating to the immune-mediated nature of the disease, and the absence or minimal signs of bleeding in a large proportion of cases have led to a revision of the terminology.

Concepts surrounding the mechanisms of thrombocytopenia in ITP have shifted from the traditional view of increased platelet destruction mediated by autoantibodies to more complex mechanisms in which both impaired platelet production and T cell–mediated effects play a role.

Previously published guidelines for the diagnosis and management of ITP require updating largely due to the introduction of new classes of therapeutic agents, and a greater understanding of the disease pathophysiology. However, treatment related decisions still remain principally dependent on clinical expertise or patient preference rather than high-quality clinical trial evidence. This consensus aims to report on new data and provide consensus-based recommendations relating to diagnosis and treatment of ITP in adults.

Criteria For Diagnosis Of Antiphospholipid Syndrome: An Update

Prof. Nevine Kassim, Clinical Pathology Dept, Ain Shams University

One of the conclusions of the subcommittee meeting on Lupus Anticoagulant/ Phospholipid dependent antibodies, held in Geneva on 2007, was the need to update the guidelines on serological tests for diagnosis of AntiPhospholipid syndrome. Emphasis was given to several aspects as patient selection, time of testing, choice of test, pre analytical variables, stress on the importance of establishing cut-off limit for every laboratory, result interpretation, mixing studies, confirmatory tests, transmission of results & its interpretation.

A full laboratory profile comprising Lupus Anticoagulant, Anti cardiolipin Abs (aCL) & antiβ2glycoprotein (aβ2GPI) antibodies ELISAs should be done and accordingly, patients are classified into different risk groups which are correlates with thrombosis or fetal loss.

Genetic Challenge In Warfarin Therapy

Prof.Manal Fawzy Gabr

Warfarin(coumarin) is currently the most widely used vitamin K antagonist (VKA) wordwide. VKAs are prescibed to millions of people each year for primary and secondary prevention of various arterial and venous thromboembolic diseases.However VKAs are challenging to use in clinical practice.

Improvement in the safety and effectiveness of oral anticoagulant treatment resulted from the standerdization of the prothrombin time test by the adoption of International Normalization Ratio (INR) Optimal therapeutic ranges with target INRs have been defined for each indication.

However, It is well recognized that the effectiveness of oral anticoagulant in clinical practice is limited ,a substantial percentage of patients cannot be maintained within the recommended therapeutic range ,despite major efforts from healthcare providers to meticulously adjust drug dosage and educate patients. Identifying amenable factors that predict overanticoagulant or underanticoagulant has been the focus of intense basic and clinical research.

Since decades,it is well known that the maitenance dose of VKAs is influenced by different acquired factors, including demographic data, dietary vitamin K intake,comorbid conditions,acute illnesses,and comedication.

Warfarin pharmacokinetics are affected by funtional polymorphisms in cytochrome P450 2C9(CYP2C9).in addition, warfarin effects are modulated by polymorphisms in vitamin K epoxide reductase complex1(VKORC1) enzyme, acritical component of thevitamin K cycle.

Both VKORC1 and CYP2C9 polymorphisms independently correlate with warfarin dose and other clinical outcomes such as time tostabilized dose,bleeding events, and time withen the target therapeutic range.

Combined polymorphisms in VKORC1 and CYP2C9 explain approximately 30%(20%-25%)for VKORC1 and 5%-10% for CYP2C9 of the variance in the stabilized warfarin dose distribution.

D-DIMER ASSAY IN EGYPTIAN PATIENTS WITH GAUCHER DISEASE: CORRELATION WITH BONE AND LUNG INVOLVEMENT

Eman M Sherif[1], Azza A Tantawy[1], Amira A Adly[1], Hossam Abdel Kader[2], Eman A Ismail[3]

Pediatric[1], Radiology[2], and Clinical Pathology[3] Departments, Faculty of Medicine, Ain Shams University

Background: Gaucher disease (GD) is the most frequent lysosomal storage disorder. Bone and lung involvement are two major causes of morbidity in this disease. D-dimer is a reliable indicator of active microvascular thrombosis, even in patients without overt hypercoagulation**.**

Aim: This study aimed to assess D-dimer levels in GD correlating this marker to clinical characteristics and radiological parameters to investigate its role as a potential predictor for the occurrence and severity of skeletal and pulmonary manifestations.

Materials and Methods: The study population consisted of 56 Egyptian patients with GD; 36 had type 1 (64.3%) and 20 had type 3 (35.7%). Thirty healthy individuals were enrolled as a control. Immunoturbidimetric assay of serum D-dimer was performed.

Results: D-dimer levels were significantly higher in all patients with GD compared to controls ($p<0.001$). Patients with type 3 showed significantly higher D-dimer concentrations compared to type 1 ($p<0.001$). Pulmonary involvement was present in a significant proportion among type 3 GD ($p<0.05$) while bone changes were present in a higher percentage in type 1 compared to type 3 GD. D-dimers were significantly higher in patients with abnormal magnetic resonance imaging (MRI) findings of long bones and in those with ground glass appearance on high-resolution computerized tomography (HRCT) chest compared to patients with normal radiology ($p<0.001$). Splenectomized patients displayed significantly higher D-dimer levels than others ($p<0.001$).

Conclusion: Our results suggest that D-dimer is significantly elevated in GD particularly type 3 and may be considered as a potential marker of risk prediction of bone and lung involvement that could be used to monitor treatment response.

T-CELL CD38 EXPRESSION IN B-CHRONIC LYMPHOCYTIC LEUKEMIA

Nashwa K Abousamra[1], Manal Salah EL-Din[2], Emad Azmy [3]*

[1]Department of Clinical Pathology, Hematology Unit, Faculty of Medicine, Mansoura University, Egypt

[2]Department of Medical Oncology, Oncology Center, Mansoura University, Egypt

[3]Department of Clinical Hematology, Faculty of Medicine, Mansoura University, Egypt.

Background: B-cell chronic lymphocytic leukemia (B-CLL) is a heterogeneous disease with some patients having an indolent course never needing treatment, while others having rapidly progressive one requiring intensive treatment. In recent decades, numerous prognostic markers, such as IgVH mutational status, ZAP-70 and the expression of CD38 on leukemic cells were introduced to screen for patients likely to have progressive course of B-CLL bearing the potential to facilitate risk-adapted treatment strategies.

In B-CLL, T cell function is shown to be dysregulated. CD38 has been demonstrated to be an important transmembrane signaling molecule of T cell with a direct effect on its function.

Aim: The present study was conducted to analyze CD38 expression on T cells to evaluate its impact on the clinical course and correlate it with other risk factors.

Material and Methods: The study was conducted on 88 unselected B-CLL patients. CD38 expression level was evaluated by flow cytometry.

Results: CD38 expression level on T cells was shown to predict the clinical course of B-CLL in male patients but not in female patients. Male patients showed CD38 expression on T cells in a stage-dependent manner, in contrast to female patients who showed higher expression irrespective to clinical staging. CD38 expression on T cells negatively interacted with treatment-free survival in male patients. Multivariate analysis revealed that CD38 expression level on T cells is an independent prognostic factor in B-CLL male patients. A simultaneous evaluation of CD38 expression on both B-CLL cells and T cells allowed predicting male patient groups with the most favorable prognosis as well as those with the worst.

Conclusion: CD38 expression level on T cells is an independent prognostic factor in B-CLL male but not female patients.

PROGNOSTIC SIGNIFICANCE OF CD44v6 IN CLL PATIENTS: RELATION TO STANDARD PROGNOSTIC FACTORS

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Background: Increased expression of the adhesion molecule CD44v6 was shown to be responsible for an increased apoptotic resistance in malignant cells, tumor progression and metastasis.

Aim: The current work aimed to analyze CD44v6 expression in CLL patients, assess its prognostic significance and its association with other standard prognostic factor.

Material and Methods: Fifty seven B-CLL patients were enrolled into this prospective study. Flow cytometry using the routine panel for lymphoproliferative disorder in addition to Phycoerythrin (PE)-conjugated monoclonal anti-human CD44v6 was performed. In addition, fluorescence in situ hybridization (FISH) technique using combination of routine locus-specific identifier (LSI) probes for detection of 13q14 deletion, 17p (p53) deletion, 11q22 and 11q23 rearrangement and CEP probe for detection of trisomy 12, were applied to peripheral blood samples of all patients.

Results: Out of the studied, patients CD44v6 was positive in 34 patients (59.6%) with mean florescent intensity (MFI) >2.5 in 28/57 (49.1%). Significant higher expression of CD44v6 was detected in patient group compared to control group ($p<0.05$). According to FISH analysis, chromosomal aberrations were detected in 47 of 57 cases (82.4%). The most frequent aberrations were a deletion in 13q (45.6%), a trisomy of 12q (15.8%), deletion/rearrangement in 11q (14%), and the least frequent aberration was deletion in 17p (7%). CD44v6 positive patients showed significantly older age , advanced RAI staging , lower Hb, higher TLC, positive CD38, lymphocyte doubling time (LDT)<12 months, unfavorable cytogenetic profile and poor therapeutic response ($p<0.05$). Survival curves analysis showed shorter overall survival of CD44v6 positive patients in comparison to negative patients. As regards response to therapy, only age, LDT and unfavorable cytogenetic profile proved to be independent prognostic factors.

Conclusion: CD44v6 positive expression identifies a group of high risk CLL patients with poor response to standard therapeutic regimen. It is significantly associated with standard poor prognostic markers as high TLC, advanced Rai staging, positive CD38, reduced LDT<12months and unfavorable cytogenetic profile. Multivariate analysis identified Age, lymphocyte doubling time and unfavorable cytogenetic profile as reliable independent prognostic markers in B-CLL, valuable as first-line screening for assignment of efficient therapeutic protocol. Trials of modified therapeutic protocol include monoclonal antibody against CD44v6 would be recommended for patients with positive CD44v6 expression associated with del 17p (p53), trisomy 12 and 11q del/rearrangement; as these factors anticipate therapeutic resistance.

PROCALCITONIN COMPARED TO OTHER INFLAMMATORY MARKERS AS A DIAGNOSTIC TOOL OF INFECTION IN PATIENTS WITH HAEMATOLOGICAL MALIGNANCY

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Background: Accurate and early diagnosis of fever in patients with hematological malignancy is a very important aspect of management. Inflammatory biomarkers have emerged as potentially useful in diagnosis of bacterial and fungal infection. Procalcitonin has been increasingly used as an inflammatory marker to identify patients with systemic infection.

Aim: To assess the diagnostic accuracy of serum levels Procalcitonin (PCT) compared to other inflammatory markers in diagnosis of infection in febrile patients with haematological malignancy.

Patients and Methods: Eighty febrile patients with recently diagnosed haematological malignancy were enrolled in this study. Twenty nine were neutropenic (neutrophil< 1500/ ml) and 51 were non-neutropenic. Blood samples were taken to assess the levels of Procalcitonin, CRP, TNFα and IL6. Samples of blood, urine, sputum, throat swab, stool in addition to any apparent site of infection and intravenous cannula were cultured and identification of the isolates was done. The significance of serum level of PCT, CRP, TNFα and IL-6 for detection of infection, the presence of multiple organisms and the presence of fungal infection were studied

Results: Nine of the studied patients had negative culture (2 neutropenic and 7 non-neutropenic). Serum levels of PCT, TNFα and IL6 but not CRP were significantly higher in febrile neutropenic compared to non-neutropenic patients with Procalcitonin showing the highest significance (p< 0.0001, p=0.026 and p=0.001 for PCT, TNFα and IL-6 respectively)**.** PCT, CRP, TNFα and IL-6 levels were significantly associated with fever due to infection than fever due to other etiology (P=0.003, 0.011, 0.042 and 0.023 for PCT, CRP, TNFα and IL-6 respectively). Procalcitonin, CRP, TNFα and Il-6 have AUROC (Area under Receiver Operating Characteristics) for detection of infection of 0.962, 0.885, 0.873 and 0.819 successively in febrile patients. Levels of ≥ 1.5 ng/ml, ≥ 9mg/dl, ≥ 23.5 ng/ml and ≥ 83.5ng/ml of PCT, CRP, TNFα and IL-6 respectively can diagnose infection as an underlying cause of fever with PPV as high as 100%. Procalcitonin was the best among others in this aspect having an accuracy of 96.2% an NPV of 92.9%. Only IL-6 showed a significant increase ((p=0.035) in its level in Gram positive compared to Gram negative organisms (AUROC: 0.61, at 27 pg/ml, Sensitivity: 60%, specificity: 58.3% and accuracy: 59.15%). The levels of PCT (p=0.029), TNFα (p=0.005) and IL6 (p=0.031) but not CRP showed a significant increase in patients who had multiple compared with those who had single organism cultured. The highest significance was shown by TNF-α (AUROC: 0.691, at a level of 9 pg/ml with a sensitivity of 94.6% and NPV of 88%). Only CRP (p=0.042) showed a significant increase in fungal infection compared to pure bacterial infection.

Conclusion: Procalcitonin appears to be the best of the 4 markers for diagnosing infection as a cause of fever in hematological malignancy. However, it does not seem to have diagnostic role in fungal infection or discriminating role between Gram positive and Gram negative organism or between unimicrobial and polymicrobial infection.

BCL2 AND BCL6 GENES REARRANGEMENTS IN PATIENTS WITH NON-HODGKIN LYMPHOMA: IMPACT ON DISEASE OUTCOME

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Background: The discovery of specific cytogenetic and molecular abnormalities involved in the pathogenesis of non-Hodgkin lymphoma (NHL) allowed more accurate definition of clinical subtypes with distinct prognosis and treatment response rates. The BCL-family members are proto-oncogenes that seem to be important regulators of lymphocyte function, differentiation and survival.

Aim: This study aimed to detect rearrangements of BCL2 and BCL6 genes in adults with newly diagnosed NHL and their relations with the clinical and laboratory features, to evaluate their prognostic value and impact on treatment response as well as clinical outcome.

Materials and Methods: Forty-six adults with newly diagnosed NHL were enrolled into this prospective study and divided into 2 groups; 25 patients with diffuse large B-cell lymphoma (DLBCL) and 21 patients with follicular lymphoma (FL). Detection of BCL2 and BCL6 genes rearrangements was performed using conventional cytogenetic analysis and fluorescent in situ hybridization (FISH).

Results: Using FISH, BCL2 gene rearrangements were detected in 24% of patients with DLBCL and in 81% of FL patients whereas BCL6 gene rearrangements were detected in 36% of DLBCL patients and in 10% of FL patients. No significant association was found between these molecular markers and the clinicopathological features or the standard prognostic factors in both groups of patients ($p>0.05$) except for staging in DLBCL patients with BCL6 gene rearrangements ($p<0.05$). BCL2 positive patients displayed a high tendency for resistance to therapy, poor clinical outcome and a significantly shorter overall and disease-free survival times in DLBCL ($p<0.05$). A clear trend for a better response to therapy, favorable clinical outcome and a longer survival was found in BCL6 positive patients with DLBCL ($p<0.05$). Similar results were observed in patients with FL but did not reach statistical significance ($p>0.05$).

Conclusion: BCL2 and BCL6 gene rearrangements occur frequently in NHL and could be considered as independent prognostic factors that carry different impacts on treatment response and clinical outcome of patients. Thereafter, the use of these molecular tools in NHL would be recommended to guide therapeutic regimens and to provide a basis for developing novel therapies.

SERUM PLEIOTROPHIN (PTN) AND *PTN* GENE EXPRESSION IN PATIENTS WITH MULTIPLE MYELOMA: RELATION TO DISEASE STATUS

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Background: Pleiotrophin (PTN) is a heparin-binding protein involved in the differentiation and proliferation of neuronal tissue during embryogenesis. PTN is produced by many different solid tumors, and recently, it was found to be secreted by the malignant plasma cells from multiple myeloma (MM). Expression of *Ptn* gene contributes to malignant proliferation and disease progression.

Aim: This study aimed to investigate the potential merit of measuring PTN serum levels and analyze the expression of *Ptn* gene in patients with MM in relation to disease status and response to therapy.

Materials and Methods: Fifty patients with MM and 30 age- and sex-matched healthy controls were enrolled into this study for assessment of serum PTN levels by enzyme linked immunosorbent assay (ELISA) and expression of PTN mRNA by reverse transcription- polymerase chain reaction (RT-PCR). Patients were defined as showing responsive, refractory, stable, progressive or relapsing disease.

Results: PTN serum levels were significantly elevated in MM patients compared to healthy control group ($p<0.001$). Patients with progressive or relapsing disease at the time of evaluation had significantly higher serum PTN concentrations than patients with responsive or stable disease ($p<0.05$). Patients with responsive disease had lower PTN levels compared to refractory cases or those with stable disease ($p<0.05$). This was further confirmed by the results of RT-PCR. The expression of PTN mRNA was not detected in 15 normal bone marrow (BM) samples. *Ptn* gene expression was easily detected in BM of patients with refractory or progressive disease and in relapsed patients but the gene was minimally expressed in patients with responsive or stable disease.

Conclusion: Our results suggest that PTN may be a reliable indicator of MM that is highly expressed by malignant BM plasma cells and closely related to disease status. Serum levels could be considered as a potential biomarker for monitoring disease progression and the efficacy of therapy.

CURRENT PERSPECTIVES ON BIOLOGY AND CLINICAL IMPLICATIONS OF STEM CELLS

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Stem cells are defined as group of cells with self-renewing capacity,ability to multi-lineage differentiation, having homing and clonality features. Stem cells require homing sites defined as "niche" in the tissues. In recent years, studies revealed at least four major group of human stem cells including **embryonic (hESC)**, **adult (ASC), mesenchymal (MSC)** and **cord blood stem cells (CBSC)**. The ability of tissue – specific stem cells to differentiate to cell types different from the tissue of origin has been defined as ***"stem cell plasticity"*** or "***transdifferentiation of stem cells***".

Multipotent stem cells are found in mature tissues and are formed by the body to replace worn out cells in tissues and organs. Hematopoietic stem cells (HSCs) are present in peripheral blood, bone marrow and cord-blood and are capable to give rise to blood and immune system cells. The potential uses of hematopoietic stem cells (HSCs) in the therapy of human diseases continue to progress very rapidly.

Many laboratories are doing studies to establish stem-cell based therapies and stem cell research has a very important potential for future regenerative medicine. Embryonic stem cells (ESCs) are pluripotent cells derived from blastocysts that can be propagated indefinitely undifferentiated in vitro, can differentiate to all cell lineages in vivo and can be induced to differentiate to all cell lineages in vivo, and can be induced to differentiate to most cell types in vitro. ESCs are unrestricted in their pattern of differentiation mainly to the somatic and germ cell lineages. ESCs can also be manipulated to differentiate into a diversity of cell types. To date, ESCs have only been utilized in basic in vitro and in vivo animal research to invesigate their potential source of stem cells for the amelioration and treatment of human diseases which can not be treated by conventional techniques and modalities. The formation of teratoma in SCID/beige mouse after ESC injection still is a major issue and needs to be resolved. Since 2006, Shinya Yamanaka, Jamie Thomson, Kevin Eggan, Douglas Melton and several other teams were able to generate induced pluripotent stem cells (iPS), embryonic –like stem cells made without use of embryos. These researchers introduced "pluripotency genes" into skin cells and were able to produce iPS –cells.

Eggan and co-workers were able to generate first patient –specific iPS-cells from two ALS patients. These new technology called "re-programming of stem cells" will be a very important and promising technology for future use of embryonic stem cells in regenerative medicine.

The MSCs are being used in limited number of clinical trials to treat GVHD after stem cell transplantation and they have a great potential for future use in orthopaedic and storage disorders. The best use of HSCs as adult stem cells is in stem cell transplantation to treat non-malignant and malignant hematological disorders as well as some auto-immune diseases such as multiple sclerosis.

The notion of using adult stem cells in some therapies is very attractive. Methods for transplanting stem cells need to be developed. It will be crucial to ensure that the transplanted cells are located in the tissues and function properly in heart, neural tissues and other organs. The use of embryonic stem cells in regenerative medicine raises ethical, legal and social concerns among the public. These new approaches may also offer an another advantage of overcoming the problem of tissue rejection in transplantation. But it is highly critical to perform intensive pre-clinical studies in animal models before making rational proposals for clinical research. Still, many unforeseen side effects or complications may develop after applications of stem cell products, so special precautions must be exercised for potential local and systemic adverse reactions of stem cells from various sources.

The recent experiments by Yamanaka and Thomson showed production of newly defined induced *pluripotent stem cells (iPS) having biologic features of embryonic stem cells. These scientists reprogrammed mature human skin cells into induced pluripotent stem cells by expressing critical transcription factors expressed in embryonic stem cells. These findings are very promising for future basic and clinical embryonic stem cell research.

The current best practice for translational research of stem cells can be achieved by following the **"International Society of Stem Cell Research (ISSCR) Guidelines for the Clinical Translation of Stem Cells"** published on December 3 ,2008 (*www.isscr.org*) :

a. "Pre-clinical studies should demonstrate proof-of-principle for a desired therapeutic effect in a relevant animal model whenever possible for the clinical condition and the tissue physiology to be studied.

b. All studies involving clinical applications of stem cells ,whether publicly or privately sponsored, must be subject to independent review, approval , and ongoing monitoring by human subjects research oversight bodies with supplemental appropriate expertise to evaluate the unique aspects of stem cell research and its application in a variety of clinical disciplines.

c. A stem cell-based approach must aim at being clinically competitive or superior to existing therapies. Care must also be taken to not take advantage of the hopes of patients with poor short-term prognoses.

d. Peer-review should also judge whether the proposed stem cell –based clinical study is likely to lead to improvement in health or may generate important new knowledge.

e. There should be persuasive preclinical evidence of safety and benefit for the stem cell-based intervention to justify proceeding to clinical trials in humans.

f. ISSCR condemns the administration of unproven uses of stem cells or their direct derivatives to a large series of patients outside of a clinical trial, particularly when patients are charged for such services. Scientists and clinicians should not participate in such activities as a matter of professional ethics. Health care institutions and research institutions should not participate in such activities. Regulators in countries where such illegitimate therapies are offered have a responsibility to prevent exploitation of patients and, if necessary, to close fraudulent clinics and to take disciplinary action against the clinicians involved."

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http://www.isscr.org/clinical_trans/app2.cfm

http://www.isscr.org

MESENCHYMAL STEM CELLS IN REGENERATIVE MEDICINE:

The six year experience of the Unit of Biochemistry and Molecular Biology, Faculty of Medicine, Cairo University

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Mesenchymal stem cells (MSCs) have the capacity to differentiate into a variety of connective tissue cells including bone, cartilage, tendon, muscle, and adipose tissue. These cells may be isolated from bone marrow with ease and expanded in culture through many generations, while retaining their capacity to differentiate when exposed to appropriate signals. The isolation of these cells from adult tissues raises opportunities for the development of novel cellular therapies without the ethical considerations associated with the use of embryonic stem cells. Multipotent cells have been isolated from various mesenchymal tissues in adults, including skeletal muscle, fat, and synovial membrane as well as hematopoietic, neural and hepatic tissues. Because of their multipotentiality and capacity for self-renewal, adult stem cells may represent units of active regeneration of tissues damaged as a result of trauma or disease. In certain degenerative diseases such as osteoarthritis (OA), stem cells are depleted and have reduced proliferative capacity and reduced ability to differentiate. The systemic or local delivery of stem cells to these individuals may therefore enhance repair or inhibit the progressive loss of joint tissue.

The research group of the unit investigated the role of MSCs in ameliorating liver fibrosis, myocardial infarction, articular cartilage defects, endometriosis, and ovarian failure. Summary of the already published work and the current research will be presented.

MESENCHYMAL STEM CELLS FOR CLINICAL USAGE

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Recently Human mesenchymal stromal cells (MSC's) have attracted a lot of attention for their clinical use in immunmodulation especially for the prophylaxis and treatment of acute GvHD and promoting engraftment after HSCT. However, little is known about the effect of different culture conditions on the functional properties and hence the clinical application or use of these cells. They have been clinically used without standardized culture conditions and release criteria. We hereby explain the effect of different cultural conditions on the proliferative and functional characteristics of these cells

Mesenchymal stem cells derived from discarded boney tissues were cultured in 3 different conditions. Two conditions using commercially available media used for MSC expansion which are supplemented with serum (condition1: Mesencult and condition2: MesenPro). 10% Human Platelet Lysate (HPL) in RPMI was used as a growth supplement for the third condition. After 2 or 3 passages in each culture condition; time of expansion, number of cells obtained, proliferation, morphology and cell surface markers were evaluated. No growth factors were added to determine the effect of the constitutively produced factors from MSC's.

MSC's were efficiently expanded from all culture conditions and met their basic criteria; plastic adherence, spindle-shaped morphology, positive for CD73, CD90, CD105, CD166 and HLA-ABC while being negative for CD34, CD14,CD45 and surface HLA-DR. Although they displayed comparable morphology HPL expanded MSC were thinner and shorter (had both lower forward and side scatters). Proliferation rates showed statistically significant differences ($P<0.5$) between the 3 conditions, the highest of which was condition 3 (HPL expanded MSCs). Immunophenotypically, the 3 conditions were similar for MSC markers but HPL expanded MSC's had significante ($P<0.5$) lower expression of DNAM ligands (nectin-2 and PVR). Cell culture supernatants revealed that HPL expanded MSCs had a lower production of PGE2.

MSCs can be generated and expanded by different culture conditions and they can be classified as MSCs. However effector functions and secretary products are changed according to the conditioned media which may affect their invivo functions. Thus these data must be taken in consideration before the clinical use of MSCs. Proper standardization of the culture condition used to expand MSC's according to the clinical usage is still needed to addressed. More standardization of the cultutre conditions are needed before clinical application.