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President's Column


Professor Khalid Hassan

As I write these lines, we are drawing close to the end of the year 2010, and also to the end of the tenure of the current office bearers of PSH. The **forthcoming annual conference is less than a month away**, and I am sure the organizing committee must be finalizing its programme by the end of this month. We all wish the conference a grand success.

I am glad that during the year 2010, **multiple academic activities** were carried out across the country, in collaboration with PSH. These included multiple workshops on hemostasis organized by Dr Tahir Shamsi and his team in almost all the large cities; morphology workshops held at Foundation Medical College, Rawalpindi by Brig Zahur-ur-Rehman and Col Lubna Zafar; Morphology Workshop held at the University of Health Sciences, Lahore by Dr Shahida Mohsin and Prof Khalid Hassan; Seminar on Hematologic malignancies and bone marrow transplantation held at NIBD, Karachi, and a postgraduate course held at the Children Hospital, Lahore by Dr Nisar Ahmed. In addition, multiple seminars and workshops on thalassemia and hemophilia were arranged at Rawalpindi-Islamabad by Prof Tahira Zafar and her team.

PSH Haematology Updates 2010 is near its completion, and hopefully, the copies will be dispatched to you before the conference. In addition to the Haematology Updates, PSH is introducing the first volume of a new education series, which will be named as **"PSH Haemimages".** This book series will highlight images of various haematological lesions, along with the relevant description for the benefit of postgraduate residents. I would recommend that a volume of this series is published at least every two years. I am very optimistic that PSH will not only make these publications a regular features, but will also open many more academic avenues, like publication of various monographs and fascicles; guideline on various aspects of haematology designed for our own requirements, and above all, an official journal of PSH.

This is my last "President's Column", and I take this opportunity to extend my gratitude to all my colleagues for extending an out-right support on all fronts. I particularly thank the energetic secretary- Dr Nadeem Ikram- who was instrumental in all what we did in our tenure. I am deeply indebted to Brig Parvez Ahmed for his continued support and for supervising the publication of the newsletter. I am indeed happy that I am handing over my office to a better person, General Suhaib Ahmed. Let all of us give him all our support!

With regards,
Professor Khalid Hassan

Academics

Molecular Therapy in Haematology

Dr Nadeem Ikram
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Last two decades have witnessed a surge in the knowledge related to genetics. The role of chromosomes, genes, and DNA and protein bases in the disease process is delineating. The journey from cell to nucleus, from nucleus to chromosomes, from chromosomes to genes, from genes to DNA and from DNA to protein bases is spell bound and breathe holding. Down, downward they went, and yet further down- their descent at each step seeming to out measure their advance. But, it's for the sure, that time has arrived to taste the fruit of success, as opportunity follows struggle. It follows efforts. It follows hard work. It does nor come before. The medicine is trying to have a position near to this target. Now, the knowledge of genetics is heading from the diagnostic realms to the treatment corridors or from laboratory bench to the bed side of the patient.

On the basis of the accumulated genetic information the efforts are underway to have a check on the molecules either gene or DNA or nucleotide or protein bases. This molecular therapy is acclaimed to be very well target oriented, personalized, with minimal side effects, without any ill effects on innocent bystanders ,an ease of administration and a curative intent in many circumstances. Out of different fields of medicine, haematology is on the forefronts to grab the benefits of molecular therapy. The drugs having a specific molecular target, DNA vaccines and gene therapy for different haematological problems are showing promising prospects.

Chronic Myeloid Leukaemia (CML) was the first human malignancy to be associated with a specific genetic lesion, the Philadelphia chromosome, harboring the bcr-abl oncogene. The essential role in the genesis of CML is played by a region on bcr-abl oncoprotein which encodes abnormal tyrosine kinase activity. Tyrosine kinases are enzymes that phosphorylate proteins on tyrosine residues and typically function in signal transduction cascades. Through this abnormal tyrosine activity the CML cells then escape apoptosis and adherence, and attain a capability of uncontrolled proliferation. Molecular therapy to control this abnormal tyrosine kinase activity is a major breakthrough in the field of molecular medicine. A tyrosine kinase inhibitor, imitinab mesylate (STI571; Gleevec) has a remarkable degree of specificity and its effects no other tyrosine kinases are negligible, but its role in advanced stage disease and issue of resistance are presently under observation

Designing of new analogues of imitinab and development of other small molecular inhibitors of bcr-abl are in development, and appear to partake in slightly different molecular interaction. Out of these compounds Adaphostin exerts its anti-tyrosine kinase effects by a mechanism distinct from imitinab and is also effective in imitinab resistant CML cases. Therapy with the aim of destabilizing or breaking bcr-abl fusion or inhibiting its expression, like Geldanamycin, are also in the stream line. Bcr- abl transduces its oncogenic signals through several downstream pathways which are then constitutively activated in a leukaemic cell. It is thus conceivable that inhibition of these pathways as an alternative or in addition to bcr-abl inhibition may be a viable mean of controlling leukaemic cell proliferation. Many drugs are under trials to explore this possibility. Then, in theory, the bcr-abl mRNA should be an ideal molecular target for therapy by designing antisense oligonucelotides. Ribozymes and Deoxyribozymes (DNAzymes) are under experimentation with an approach to target bcr-abl mRNA.[1-4]

More than 200 different chromosome translocations and other mutation events have been described in acute myeloid leukaemia (AML). The novel gene mutations in AML can be divided into two categories, mutations that confer a proliferation advantage and those which inhibit myeloid differentiation. Besides chromosomal rearrangements and gene mutations, epigenetic regulation of genes-meaning changes in gene expression by mechanisms other than changes in the underlying DNA sequence also represents an important mechanisms of leukemogenesis.[5] Screening for some of these mutations is now part of the initial diagnostic workup in newly diagnosed AML patients. Information about the mutation status of specific genes is useful for risk stratification, minimal residual disease (MRD) monitoring and increasingly also for targeted therapy.

In AML the best example of oncogene targeted therapy is all-trans-retinoic acid (ATRA), which specifically inhibit the transforming activities of PML- RAR- α oncogene in acute promyelocytic leukaemia fusion oncogene formed by the balanced translocation t(15;17). Therapies containing ATRA are associated with better remission rates, long term disease free survival and high percentage of cure. ATRA is found to modulate 169 genes, among these 100 were up regulated and 69 down regulated. These genes involve transcription proteins, signal transduction modulations, cell cycle regulation, apoptosis-related proteins, cell structure mobility proteins and cell adhesion proteins and others.[6-7] Regulators of apoptosis in AML have been extensively studied and are considered excellent therapeutic targets. Expression levels of these antiapoptotic proteins, like Apoptosis Repressor with Caspase recruitment domain (ARC), are found over expressed in AML and can be a potential therapeutic target in AML.[8]

There are a growing number of tyrosine kinases now known to be activated by mutation in blast cells from patients with acute myeloid leukaemia.These mutational activators of tyrosine kinases then result in continuous ligand-independent proliferation and viability signals . Selection inhibition of these mutated tyrosine kinase by small molecule inhibitors represent a strategy to disrupt those signaling pathways which promote neoplastic growth and survival. At least four compounds (CEP-701, CT 53518, PKC 412, SU 5416) are underdevelopment with an aim to inhibit a tyrosine kinase, FLT3, in acute myeloid leukaemia.[7] Defects in programmed cell death (apoptosis) mechanisms play important role in the pathogenesis and progression of haematological malignancies. In neoplastic cells many oncogenes, which are endogenous antagonists of apoptosis, are identified, e.g., bcl-2, bcl-x_L, mcl-1. Over expression of bcl-2 probably occurs in more than half of the haematological malignancies, rendering neoplastic cells resistant to many types of apoptotic stimuli including most cytotoxic anticancer drugs. Attempts to overcome the antiapoptotic effects of these oncogenes include three strategies (1) directly attacking these proteins with small molecular drugs; (2) shutting of gene transcription; (3) inducing mRNA degradation through antisense oligonucelotides, like Genasense and G3- 139. Clinical trials targeting bcl- 2 mRNA have advanced to phase III for multiple myeloma, chronic lymphocytic leukaemia, acute myeloid leukaemia and melanoma. In multiple myeloma mcl-1 is an important survival factor. Antisense oligonucelotides to specifically inhibit this oncogene expression lead to the rapid induction of Caspase activity and apoptosis (within three hours in some cases) . Dysregulation of Cyclin D gene appears to be a nearly universal event in the pathogenesis of multiple myeloma. There may be a therapeutic window in targeting this pathway for all molecular subtypes of multiple myeloma.[9,10]

Recently, the description of the gain of function mutation of JAK-2 (JAK2V617F) has been identified in classical Philadelphia- negative myeloproliferative disorders. It provides a rational target for novel innovative treatment strategies. Currently, clinical studies testing various JAK2- inhibitors in polycythemia vera, essential thrombocythemia and idiopathic myelofibrosis are under way. [11]

DNA vaccines have now moved from an exotic possibility to practical testing in the clinic. The genetic vaccines delivered through naked DNA alone or through viral vectors are showing promise in clinical trials in lymphoma and myeloma patients. The concept of gene-based vaccines is to take a tumour- associated gene sequence and deliver it directly to the patient so that the gene is transcribed and translated, with subsequent presentation of the protein to the immune system in situ. The ability of the immune system to attack and maintain immune vigilance is then utilized against tumour cells, by the same principle as combination chemotherapy, but without the collateral damage. In a setting of DNA vaccination, the idiotypic (id) immunoglobulin (Ig) of B cell malignancies is selected as the initial antigen . As it is tumour specific so there are no chances of inducing autoimmunity. [12]

Gene therapy of haemophilia is a model system for developing a basic understanding of how gene therapy will be achieved. The infusion of factor VIII or IX gene through a viral or non-viral vector ensures sustained long term (for years) production of the coagulation factors, without evidence of inhibitors production . By using new viral modes like adeno and lentivirus and non-viral vectors like naked plasmid DNA the problem of immune response to the transgene product or the viral vectors is also circumvented. The use of circulating endothelial progenitor cells to carry factor VIII gene is showing an added advantage of the presence of von Willebrand factor, a carrier protein necessary for factor VIII stability in plasma. Different studies show that, by gene therapy in haemophilia A or B, the factor levels are maintained at higher levels for a longer period and even relatively low levels of factor dramatically reduce spontaneous bleeding. [13,14]

Haemoglobinopathies like sickle cell anaemia and beta thalassaemia major are also a target for gene therapy. Gene therapy with lentivirus vectors has been successful using sickle transgenic mice. An anti-sickling beta globin gene was introduced into the sickle transgenic mice . Long term expression (upto 10 months) was achieved. In addition haematological parameters correction, inhibition of cellular dehydration and loss of sickling tendency were also demonstrated . [15]

The transgenic mice models for beta thalassaemia major received human beta globin gene through lentivirus vectors. The mice exhibited durably increased haemoglobin levels for more than forty weeks. Ineffective erythropoiesis and extramedullary haemopoiesis regressed as reflected by normalization of spleen size. The induced transduction was sustained for more than seven months. These models provide a solid foundation for the human clinical trials in sickle cell and beta thalassaemia major. [16]Haemopoietic stem cell is under trial to be evaluated as a primary target for gene therapy, owing to its capacity for differentiation and self-renewal, whereby multiple cell lineages can potentially be corrected for the life time of an individual. A one such model is studied in Diamond Blackfan Anaemia (DBA). Patients with DBA usually show a mutation in ribosomal protein RPS19. Mobilized peripheral blood $CD34^{+}$ cells were transduced by oncoretroviral vector particles. Vectors contain different RPS19 expression levels. It was demonstrated that corrected DBA patients cells gain a proliferative advantage over control cells in vivo, thereby demonstrating improved engraftment following gene therapy . In patients of beta thalassaemia major, lacking a suitable marrow donor, gene therapy as transplantation of autologous genetically corrected haemopoietic progenitor/ stem cells, represents an attractive alternative to bone marrow transplantation, since is not limited by the histocomaptibility barrier and does not required immunosuppression. Use of LV-GLOBE, harbouring the human β-globin gene is under trial in beta thalassaemia major patients. The challenges which gene therapy still faces are of high-energy gene transfer efficiency, safety,feasibility,transgene expression for sufficient therapeutic benefits and safe vectors with minimal risks for insertional mutagenesis. [17-20]

Novel approaches often move from wild enthusiasm through pessimistic cynicism to eventual useful application. New molecular techniques are an attempt to over view thousands of genes and proteins simultaneously, analyze the individual protein signaling pathways which are being utilized by the diseased cells, characterize the disease biologically, and select specific targeted treatment modalities with optimum therapeutic efficacy and minimal toxicity, known as "personalized" molecular medicine. Much remain to be learned about how best to exploit these new potential therapies but improved clinical outcomes may not be far from realization. [21]

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PSH News

PSH Executive Committee Meeting

Pakistan Society of Haematology , Executive committee meeting was held on 06th November ,2010, at the office of Professor Samina Naeem, King Edward Medical College, Lahore. Following attended the meeting. Meeting was attended by,Prof Dr Khalid Zafar Hashmi (Karachi), Prof Dr Khalid Hassan (Islamabad),Prof Samina Naeem (Lahore),Prof Dr Tahir JAmil (Lahore),Dr Nisar Ahmad (Lahore),Dr Muhammad Nadeem (Karachi),Dr Nadeem Ikram (Rawalpindi)

Prof Khalid Hassan appraised the meeting about correspondence with Pakistan Medical and Dental Council, about the seats of Pathology in Medical Colleges. In principle PMDC agreed for 4 posts of assistant professors and four posts of associate/professors. But PSH reaffirmed its stand to revive the old structure of seats.

Prof Khalid Hassan give a brief summary of upcoming issue of "Haematology Updates, 2010". Prof Khalid Zafar Hashmi appreciated "Haematology Updates- 2009".Update was given about the publication of "Haematology Images".The election process of PSH next elections was discussed. It was decided that election committee will sent all the relevant material to members. It was decided that in case of more than one candidate for one seat voting will be done at the time of elections.House discussed the issues of the age limit of the president elect. It was decided that the matter will be put on the agenda of PSH Executive Committee meeting , at the time of National Conference at Karachi.

Prof Khalid Zafar Hashmi and Dr Muhammad Nadeem updated about the progress of Next PSH conference at Karachi . They informed that after giving due consideration to the input from financers it is decided to shift the venue from Liaqat National Hospital to Expo Centre.Dr Muhammad Nadeem floated the suggestion to have corporate/ associate members and to include the laboratory technologists in the membership of PSH. It was decided to put the matter on the agenda of next general body meeting. Dr Nisar informed the house that he will submit the detail report of Lahore PSH Annual conference to the PSH Centre .The executive committee approved membership of new members.Prof Khalid Zafar Hashmi put the proposal to financially assist the local chapters, after approval from executive council, if any local chapter request for it. It was decided to put it one the agenda of next general body meeting.

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Haematology Oncology and Transplantation update at NIBD Karachi

National Institute of Blood Diseases and Bone Marrow Transplantation had organized "Haematology Oncology and Transplant Update 2010" on 1st and 2nd Oct 2010. Originally this event was planned for the month of August but due to law and order situation in Karachi, and later the devastating country wide floods, the workshops and conference were postponed. By the Grace of Almighty Allah the response during rescheduled two days of the workshops and conference was tremendous.

On 1st Oct two workshops on "Stem Cell Transplantation" and "Lung Cancer were organized in NIBD and JPMC Karachi respectively. Dr. Tahir Shamsi in NIBD and Dr. Ahmed Nadeem in JPMC conducted the full day workshops. The attendance was more than expected and more than eighty participants attended both the workshops, though these workshops were planned for only 50 post graduates, 25 in each center.

During the day of conference the plenary session was attended by prominent oncologists and haematologists of the country. Among them were Gen Masood Anwar, Prof Khalid Zafar Hashmi, Dr. Nisar Ahmed, Dr. Tahir Shamsi, Brigadier Mohammad Ayyub, Brigadier Pervaz Ahmed, Dr. Salman Adil, Brigadier Tariq Satti, Brigadier Jaleel Anwar, Prof Waseem Iqbal, Dr. Sarfaraz Jaffery, Dr. Bushra Moiz, Dr. Atifa Suhaib, Dr. Samina Amanat, Dr. Shahida Mohsin and Dr. Muhammad Nadeem. The session was chaired by "Major General ® Abdul Qadir Usmani" Chairman Human Organ Transplant Authority. During the session audience proactively participated in the discussion, past, present and future of bone marrow transplantation in Pakistan was comprehensively dealt with by both physician's point of view and regulating authority's point of view.

The symposia on various hamatology and oncology topics were conducted as four parallel sessions in two seminar halls. The prominent speakers among the oncologists were Dr. Azmina Taj, Dr. Adnan Zaidi, Dr. Naila Zahid, Dr. Najeeb Niamtullah, Dr. Nehal Masood, Dr. Ahmed Fawad and Prof Aijaz Masood. On the other hand General Masood Anwar, Dr. Javeria Manan, General Suhaib Ahmed, Brig Pervaz Ahmed, Dr. Samina Amanat, Dr. Munira Borhany, Dr. Tahir Shamsi and Dr. Saqib Ansari spoke on updates and recent advances of various haematological disorders. The high point of the conference was interactive teleconference session with Dr. James Bussel from UK who presented the data on the latest therapeutic modality for patients of chronic ITP who have exhausted all other options of cure.Haematology Oncology & Transplantation update has not only disseminated thought provoking knowledge to audience but also created a sense of awareness among the participants about the latest research going on in the world, for some well known but difficult to treat, problems of haematology and oncology.

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Northern Areas Thalassaemia Summit

On 4th December, 2010, northern areas thalassaemia summit was held at Pearl Continental hotel Rawalpindi. The inaugural session was chaired by Mr Zmurad Khan, chairman Pakistan Bait- ul mal. He high lighted the efforts done by bait-ul-mal for the treatment of patients suffering from thalassaemia major and haemophilia. He briefed about the contribution of bait-ul-mal for iron chelation therapy and factor provision. The topics regarding diagnosis, prevention, management, blood transfusion , iron chelation therapy and bone marrow transplantation were discussed by Prof Khalid Hassan(Pakistan Institute of Medical Sciences), Prof Tahira Zafar(Blood Disease Centre), Brig Pervaiz Ahmad (Armed Forces Bone Marrow Trasnplantation Centre), Dr Naila (Pakistan Institute of Medical Sciences), Dr Atifa Shoaib(Rawalpindi Medical College), Prof Tahir Ch (Islamic International Medical College),Dr Nadeem Ikram (Rawalpindi Medical College)and Dr Naghmi (Islamabad Medcial College) . The children with thalassaemia performed at the occasion by speeches, songs, variety/dress shows.

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13th National PSH Conference at Karachi From 12th to 15th January 2011

Thirteenth PSH conference is scheduled from 12th to 15th January 2011, at Expo Centre Karachi. Prof Dr Khalid Zafar Hashmi is the chairman of the conference and Dr Muhammad Nadeem is the secretary of the conference. Programme of the workshops, scientific sessions and other events is as follow.

12th January 2011

Workshop 1: Flow Cytometery & Cytogenetics;(0800-1300 hrs) at Agha Khan University Hospital.

Workshop 2:Morphology:(1400-1700 hrs) at Liaquat National Hospital.

13th January,2011

Workshop 3: Blood Banking ;(0900-1300 hrs) at Ziauddin University. .

Workshop 4: Haemostasis;(1400 1700 hrs) National Institute of Blood Diseases.

PSH Executive Council Meeting : (1700 -1800 hrs) , at Expo Centre

Inaugural Ceremony: (1830 -2100 hrs) , at Expo Centre.

14th January,2011

Benign Haematology:(0900-1230hrs);Invited talks and free papers.
Leukaemia,Lymphoma & Myeloma: 0900-1230 hrs Invited talks and free papers.
Haemostasis: (1500-1700 hrs). Invited talks and free papers
Paediatric Haematology: (1500 1700 hrs). Invited talks and free papers

15th January,2011

Transfusion Medicine:(0900-1300 hrs). Invited talks and free papers
Stem Cell Transplantation: (1500 1700 hrs). Invited talks and free papers
Closing Ceremony & Award Distribution: 1700-1730 hrs

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34th National Conference Pakistan Association of Pathologists, at KEMU, Lahore

In the 34th National Conference of Pakistan Association of Pathologists, held at King Edward Medical University Lahore, haematologists from Pakistan and abroad participated. There were two workshops for haematology residents. Maj Gen Suhaib Ahmad(Armed Forces Institute of Pathology, Rawalpindi) conducted workshop on PCR application in Haematology. The salient features of this workshop were the genome extraction of nucleic acids, polymerase chain reaction, RT-PCR, genomic sequencing and quality control in PCR diagnostics. Another workshop on Molecular Biology/Genetics was conducted by Dr Saqib Ahmad from University of Health Sciences, Lahore. Plenary lectures were given by Maj Gen Suhaib Ahmad (AFIP, Rawalpindi), Dr Tahir Shamsi (NIBD, Karachi) and Dr Zia Uddin Zaidi(Kingdom of Saudi Arabia), on the topics of Molecular diagnosis of haematological malignancies, Stem cell: beyond bone marrow transplantation and New insights in Acute Myeloid Leukaemia. There were two scientific sessions devoted for haematology research papers.

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Your views and news

Dear Colleagues : Your contributions to PSH newsletter are backbone to its success. Please send short communications, case reports, scientific

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