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EAST DELHI PHYSICIANS' ASSOCIATION

17th Annual Conference of East Delhi Physicians' Association (EDPA)

EDPACON 2016

Sunday, 11th December, 2016
11.00 am

Venue

Le Meridien

New Delhi

E- Souvenir

www.edpadelhi.com

Contents

All correspondence to :
Dr. Pankaj Nand Choudhry, Editor
35 X, East Delhi Physicians Association Office, First Floor, IMA EDB Bhawan,
Karkardooma Institutional Area, Opposite Kendriya Vidyalaya, Delhi-110 092
Mob. : 9350290527, 8744013613
E-mail : pankajnchoudhry@yahoo.com, edpa_physician@rediffmail.com

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Editor's Column

Greetings from East Delhi Physicians Association!

It gives me a immense pleasure on bringing out the first ever e-souvrnir to be released during annual meeting of East Delhi Physicians Association annual meeting "EDPACON 2016" being scheduled on 11th December at Hotel Le Meridien, New Delhi.

We took the late decision to release it so we could not be able to encorporate write ups of the deleberations to be presented during the CME. May be next year we will do the justice.

For the past few years we have started PG program in our annual meetings which was immensely successful. This year also we have which has been are doing Post graduate short case presentation in the morning session, abstracts of these cases have been encorporated in the souvenir.

I take this opportunity to impart best wishes to the members of the organising committee of EDPACON 2016 who have not let any stone unturned to make this event a mega success.

"Whatever you vividly imagine, ardently desire, sincerely beleive and enthusistically act upon will come to pass."

- Paul J Meyer

Dr Pankaj Choudhary
Chief Editor
E- Souvenir
EDPACON 2016

Co-editor's Message

Dear senior, colleagues and EDPA members!

We are proud to state that EDPA is again fulfilling its commitment by delivering purely academic deliberations through its annual CME. We wish EDPA to grow day by day and set new heigjts and standrads and achieve glory of shining height in coming future.

We request all members to participate in academic and social activities which are always on going throughout the years. In fact we encourage all of you to come forward and share your experience through CME so that each one of us can learn from you.

We wish all the best to EDPA and wish for its everlasting growth

Warm regards

Dr Naresh Agarwal
Co-editor
E- Souvenir
EDPACON 2016

Presidential Address

Dear Friends,

It gives me an immense pleasure to Welcome you all on the occasion of 16th annual conference of EDPA , EDPACON 2016.This scientific meeting is the most prestigious event of EDPA and most eagerly awaited too. Over the years EDPA has really emerged as one of the most cohesive and Scientific National body. The annual conference of EDPA is considered as a bench mark and reference for all the practicing physicians . The popularity of EDPACON is increasing year by year and internists from Delhi & NCR are actively participating in the event.

The aim of this scientific extravaganza is to upgrade ourselves with the latest developments in the field of Internal Medicine. The organising team under the dynamic leadership of Dr Ajay Kumar Gupta has done a commendable job to make it a mega success. The scientific content and the speakers have been selected very meticulously by series of meetings & discussions.

The Editorial team under the guidance of Dr Pankaj Chaudhary & Dr Naresh Aggarwal needs lot of appreciation in bringing out first ever E-souvenir of EDPA.

Every year we honour one of our Senior Memmber with Life Time Achievement Award and this year we are extremely pleased to confer this award to our Respected Dr A S Dave Sir.

This year's Prestigious Bela Devi Oration has been conferred to the renowned Hematologist& HOD Dept of Hematology AIIMS , Dr RenuSaxena for her contribution to the Medical Field and Society.

In the end I would say that it's a Mega Scientific Event & I am sure that everyone will enjoy & benefit from it. I am again thankful to the Executive and the Organising committee for their hard and relentless efforts to make this event a huge success.

I look forward to meet you at Hotel Le Meridien on 11h Dec 2016. Your valuable feedback and suggestions are most desired for future and keeping the scientific standards of the conference at highest levels.

Dr. Rajeev Bansal
President, EDPA

Secretary's Notes

Greetings and regards to all my Seniors!!

It's that time of the year when the weather is cold, but the activity levels in EDPA heat up.

It's been my privilege to witness the unending efforts of the trio of Dr Ajay Gupta, Dr Parul Khurana and Dr Kapil Khanna in making sure that EDPACON 2016 is a huge success.

Demonitisation has had an effect on the private practice of most of us, but then again, all treatments have their side effects. And most side effects are self limiting. Nonetheless, 2016 has been a rather eventful year for our association.

On the academic front, the monthly clinical meets have continued. The pristine quality of the presentations notwithstanding, the falling attendance still remains a cause for concern.

We have had a lot of Evening CME's, and plan on continuing with them in the future as well.

On the Sports front, the cricket club has been regularly organising cricket matches for our members. One of those matches was a Day night match, the players were accompanied by their families, and was an event that was enjoyed by all.

I wish you all the very best for the coming year.

Thanking you

Yours' Sincerely

Dr. Anirudh Lochan
Secretary
EDPA

Chairman's Message

Dear Colleagues,

I welcome you all to EDPACON 2016.

I thank the executive body of East Delhi Physicians Association for giving me the opportunity to organise this prestigious yearly conference.

The venue for the conference is kept to make delegates all over Delhi NCR comfortable and convenient.

I thank all the Faculty and Chairpersons for taking their valuable time to be with us for sharing the scientific knowledge.

This year we have kept the theme "Recent Advances in the field of Medicine" and the topics selected are according to the theme.

I am thankful to the souvenir committee Dr Pankaj Choudhary and Dr Naresh Agarwal for releasing the first ever e-souvenir in the history of EDPACON.

The organising secretaries Dr Kapil Khanna, Dr Parul Khanna along with the entire organising team have not left any stone unturned to make the conference a successful one.

I hope you will enjoy our hospitality and the academic feast.

Dr. Ajay Kumar Gupta
Chairman
EDPACON 2016

Message From Organizing Secretaries

We welcome all our friends from different parts of world who have come here to make this academic event a great success, the carefully selected topics by scientific committee, and world renowned speakers , is highlight of this conference. EDPACON Is considered to be one of the premier conference of Delhi NCR ,THIS REPUTATION WOULD NOT HAVE BEEN POSSIBLE WITHOUT ACTIVE PARTICIPATION OF OUR Members as well as Pharma industry.May the coming years we grow further in knowledge and wisdom.

Dr Parul Khurana
Organizing Secretary
EDPACON 2016

Dr Kapil Khanna
Organizing Secretary
EDPACON 2016

Life time achivement award

Dr Achal Shankar Dave

Born on the 8th of March 1950

MBBS : Batch of 1968 from Sardar Patel Medical College Bikaner

MD(Medicine): Batch of 1977 from Sardar Patel Medical College Bikaner

Practiced Medicine in the United Kingdom from April 1978 till June 1982

Consultant physician in the Department of Medicine at Walia Nursing home since July 1982.

Smt. Bela Devi Oration

Smt. Bela Devi
(1907-1995)

Smt. Bela Devi (1907-1995) was a great visionary, a lady of the future, who had long ago realized the value of higher education. Inspire of being educated only upto third standard, it did not deter her to think ahead of her time.

She was born in 1907 in a middle class family of Khurja, then a small town of district Bulandshahr. UP married at a very young age of 15 years, she shifted to Shahdara, Delhi. She was blessed with eight daughters and one son.

Educating her children was always a prime concern and objective of her life. This she achieved against all odds. She was an ardent advocate of "Girls Education" and was of the firm belief that girls should be independent.

Shahdara was a small satellite town of Delhi. It was separated from it by the River Yamuna and a thick forest. Only a Primary School existed for girls on this side of Yamuna. The only mode of transportation available to further pursue their education (Middle and High School and College) was a tonga or a train. One could only imaging the obstacles and hardships forced by her children in those difficult times of pre-independence and immediately after freedom.

Delhi was not her limit. She was also willing to encourage her children to go abroad if they so desired. One can only imagine the odds against which she must have carried on. Her life exemplifies that female foeticide is a big mistake which is depriving the society of potentially great women. Thanks to her, that we have Dr. Saroj Prakash, one of her very able daughters who rose to such heights that will make any mother proud. She is a renowned physician and a great academician. She is also a founder member of our association.

Rightly so, that her daughter has dedicated this oration in the memory of a legendary lady, a loving and caring mother.

Bela Devi Oration consists of a talk on a specified, unrivalled and unparalleled topic in International Medicine of 30 minutes duration. The members of the association express their reverence in the form of a medallion and a taken money of rupees five thousand.

This is the 12th successive year in which this Oration is being delivered. Previously we have been honoured with eminent dignitaries of great repute in their respective field.

Speaker	Year	Speciality	Topic
Dr. Savitri Srivastava	2000	Paediatric Cardiologist	Congenital Heart Diseases
Dr. Harbans S. Wasir	2001	Cardiologist	Preventive Cardiology
Dr. P.S. Gupta	2002	Physician & Gastroenterologist	PUO
Dr. G.K. Ahuja	2003	Neurologist	Single CT Lesion
Dr. J.S. Guleria	2004	Cardiologist & Chest Physician	Sarcoidosis
Dr. J.N. Pande	2005	Physician and Chest Specialist	Interstitial Lung Disease
Dr. V.S. Sukhija	2006	Nephrologist	Management of Renal Failure
Dr. B.N. Tondon	2007	Gastro Enterologist	Issues in Management Hepatitis B
Dr. A.N. Malviya	2008	Rheumatologist	Distinction Between inflammatory & Mechanical Arthritis
Dr. K.K. Malhotra	2009	Nephrologist	Approach to CRF Diagnostic Considerations and Principles of Management
Dr. S.N. Chugh	2010	Professor of Medicine	Medicosocial implications of Pesticide (Aluminium Phosphide) Poisoning
Dr. M. Khallilulla	2011	Senior Interventional Cardiologist	Journey of a Cardiologist - Goals Achieved & What's New on Horizon
Dr. Neena Valecha	2012	Director, National Institute of Malaria Research (ICMR), New Delhi	Current Perspective on Diagnosis & Treatment of Malaria
Dr. P.D. Gulati	2013	Sr. Consultant, Nephrologist, Tirath Ram Shah Hospital, New Delhi	Cardiovascular Morbidity in CKD
Dr. S.K. Sarin	2014	Dirctor, ILBS Hospital, New Delhi	Overview of Portal Hypertension
Dr. Naresh Trehan	2015	CMD Medanta, The Medicity, Gurgaon, Delhi NCR	Heart to Hearts

This year's Bela Devi Oration is being conferred on Dr Renu Saxena, Professor & Head Deptt of Hematology, AIIMS, New Delhi. She will be speaking on " A Heamatologist Journey in Thrombotic Disorders".

Curriculum Vitae

Dr. R Saxena

Professor & Head
Dept. of Hematology, AIIMS, New Delhi &
Member Secretary Institute ethics committee, AIIMS from 2008 to 2013

Dr. Saxena has done her MBBS and MD from AIIMS and has been faculty in Dept of Hematology, AIIMS since 1986. She is currently Prof. & Head Dept. of Hematology, AIIMS. Her major interest has been in Hemostasis, including coagulation disorders, platelet disorders, like glanzman's thromboesthenia, vWD, storage pool disease etc. She is currently coordinator of ISHTM-AIIMS EQAP Programme.

Special Interest:

Hemostasis, Thrombosis, Leukemia, Molecular Genetics, platelet function defects, quality assurance, anemias, hemoglobinopahties.

Publications:

She has a large number of publications in National and International Journals:

PUBLICATIONS	**Nos.**
Publications in Journals (Full original articles+ short papers+ Reviews)	183
Abstracts in Journals	45
Chapter in books	85
Books/Monogram edited	8
Total	**321**

ACHIEVEMENTS

a. Honors/Awards:

- **BC Roy award** for eminent medical teacher by MCI 2016
- **Felicitated by West Bengal Society of Hematology** for outstanding work in hematology in 2016
- **Oration award by IAPM Chhattisgarh Chapter** held in CAPCON Bhilai in November 2015
- **Lifetime achievement award for hemophilia care** by Hemophilia Society of Delhi.2012
- **J.B. CHATTERJEA** memorial oration on 28th February by the CALCUTTA SCHOOL OF TROPICAL MEDICINE? 2011
- **Kshanika oration award by ICMR** for outstanding woman scientist in biomedical sciences **2009**
- **Life time achievement award** in Hematology by Mumbai Hematology Group **2009**
- **Distinguished career award** by South Asian Society of Atherosclerosis and thrombosis **(SASAT 2008)**
- **Malti Sathe oration** awarded by ISHTM 2006
- **Fellow of National Academy of Medical Sciences (FNAMS) 2006**
- Dr R Saxena was awarded **BGRC Silver Jubilee Oration Award for outstanding research in the field of Haematology and Immunohaematology** by **ICMR** at New Delhi for 2002 in **July 2005**
- Dr R Saxena was awarded **Prof. Dinkar Chandra Memorial Oration Award**: by UP Chapter IAPM at KGMU Centerary celebrations, Lucknow, **March 2005**
- **Manorama Sapre Oration award**: **1999** awarded by Indian Society of Haematology and Transfusion Medicine for out standing contributions to Haematology
- Member round table discussions on Dengue haemarrhagic fever, **Ranbaxy** research foundation, Delhi **1997**
- Elected as **fellow of International medical sciences academy**, associate member of the council for International Organisation of Medical Sciences (CIOMS) Geneva, in recognition of outstanding contribution for advancement of medical science **1996.**
- Member - Molecular Haematology " sub-committee in Molecular Pathology Association of India

Best paper award to 20 papers submitted under my direct supervision in various conferences

b. Membership of Editorial Boards of Journals

Indexed International	:-	Medicolegal Update
Indexed National	:-	Indian J Pathology and Microbiology Indian J Hematology and Transfusion Medicine
Non Indexed International	:-	Journal of Basic and Applied Biomedicine American J Immunology

Non Indexed National :- Aorta
Medical Journal Armed Forces India
Physician's Digest
Newsletter of the Delhi Society of Hematology1997

c. Reviewer for **Indexed Journals** **:-** European Journal of Cancer
British Journal of hematology
Blood Coagulation and Fibrinolysis
British J of Obstetrics and Gyenacology
J Paediatrics and Infectious Diseases
Yonsei Medical Journal
Clinica Chemica Acta
Thrombosis Research
Indian Journal of Pediatrics
Ind. Pediatrics
Indian J Medical Oncology
Journal Association Physicians of India
Indian J. Experimental Biology
Indian J. Pathology and Microbiology
Indian Journal of Medical Research
Indian Journal of Experimental Biology Indian Journal of Hematology and
Transfusion Medicine

d. Reviewer for projects;

1. Indian Council for Medical Research **(ICMR)**
2. Department of Science and Technology **(DST)**
3. Council of Scientific and Industrial Research **(CSIR)**
4. Department of Biotechnology **(DBT)**

e. Visiting Professor:

Department of Pathology, **BPKIHS, Dharan, Nepal. 1996**

f. Member of National Scientific Bodies

1. Founder member, Pulmonary Pathology Society of India 2007 onwards
2. SAC, Pre SAC committee for IIH Mumbai ICMR 2006 onwards.
3. Member,**ICMR** project review committee for Genetics,hematology, anatomy**2005 onwards**
4. Member: Subject expert committee for technical evaluation of proposals under women scientist scheme, **Department of Science and Technology, Government of India. June 2005 onwards**
5. Board of Research Studies. National Institute of Communicable Diseases (**NICD**), New Delhi. Since 2005 onwards
6. **ICMR** committee for Developing capacity building of young scientists on North-East Region for carrying out Basic, Clinical and Operational Research in the field of Nutrition. Since 2004 onwards

7. NABL Accreditation committee, **DST**. 2003 to 2006
8. Expert at **UPSC** for Pathology Since 2000
9. Resource person for **WHO** strategies for prevention and control of iron deficiency anemia amongst under three children. USAID. Since 2000

6. Ethics:

Participated in Good Clincal Practice Workshop for Ethics Review Boards January 2009 organized by Clinsys Clinical Research

Ethics committees:

1. Member Secretary Ethics Committee AIIMS **2008-2013**
2. Chairperson, ICMR Ethics committee (Nutrition) – 2012 to till date
3. Member, Ethics committee, JNU – 2012 to till date
4. Member, Animal Ethics committee, AIIMS 2008 to 2013
5. Member, ICSCRT, AIIMS 2009 till date 2016
6. Member Ethics committee,IIT Delhi 2013 till date

7. RESEARCH PROJECTS COMPLETED/IN PROGRESS WITH HELP OF RESEARCH GRANT:26

Sr No	Research Projects Principal Investigator	Source
1	A Comparison of the Clinical and Molecular Features of Chronic Lymphocytic Leukemia (CLL) in Indian and Canadian Populations" (2014-16).	Indo – Shastri Grant
2	Molecular profiling of Acute Myeloid Leukemia in North East India (2013-16)	DBT
3	Role of modulating factors on the phenotype of sickle cell disease (2014-16)	DST
4	Molecular characterization of FV in Indian Patients with APCR and FV deficiency (2010-13)	ICMR
5	Evaluation of Platelet Receptor Polymorphism In Patients With Coronary Artery Disease. (2007-2010)	ICMR
6	Role of Nitric oxide in the development of stroke (2008-2011)	DST
7	Validation of Haemoglobin Colour Scale for Hemoglobin estimation: a multicentric study(2008 – 2009)	WHO
8	Evaluation of molecular parameters affecting biology of chronic lymphocytic leukemia in India 2009-2013	ICMR
9	Modulation of genes in response Imatanib therapy in Philadelphia chromosome positive chronic myeloid leukemia patients 2008-2012	ICMR
10	Development of Hemoglobin color scale for use in field 2007-2008	WHO

11	Hemoglobin color scale for Hb determination 2006-07	WHO
12	Elucidation of mutations in hemophilia A and B 2004-2007	DBT
13	Elucidation of molecular genetics of leukemia 2005-08	ICMR
14	Molecular Characterization and treatment of AcuteLymphoid Leukemia on protocol MCP 841.International Network for Cancer Treatment and Research 2001-2012	INCTR
15	Role of TAFI in Pathogenesis of young Indian stroke 2004-07	ICMR-INSERM
16	Quality assessment in Hemogram 2003-2005	NABL-DST
17	Prevalence of alpha thalassemia in patients with thalassemia intermedia. 2002 – 2005	AIIMS
18	Sub characterization of vWD in Indians. (2003-2006).	DST Project
19	Molecular Genetics of Glanzmann Thrombasthenia. 2002-2004.	Indo-US project.
20	Identification of genetic defects underlying Indian hemophilia A patients and their role in carrier detection and prenatal diagnosis..1999 to 2004.	Department of Biotechnology, Ministry of Health, Government of India
21	To evaluate the role of functional antithrombin III assay in detecting subclinical disseminated intravascular coagulation. 1998-2000.	All India Institute of Medical Sciences, India
22	FV Leiden defect in hereditary Deep Vein Thrombosis. 1996 to 1999 . Research. India.	Council for scientific and Industrial
23	Role of Activated protein C in Deep Vein Thrombosis. 1995-1997.	All India Institute of Medical Sciences, India .
24	Role of glycoprotein in etiopathogenesis of thrombopathic thrombasthenia. 1992-1994. Sciences, India	All India Institute of Medical
25	Role of prostaglandin metabolism in pathogenesis of Hemolytic Uremic Syndrome. 1990.	AIIMS
26	Role of Platelet factor 4 in pathogenesis of CAD. 1987.	AIIMS

Co-Investigator

1.	Phase II clinical study to evaluate the efficacy and safety of NRC-AN-019 in patients with Chronic Myeloid Leukemia who have failed prior standard therapy and are not eligible for alternative therapy	NATCO
2.	Detection of molecular changes in the disease progression in chronic myeloid leukemia patients by Microarray (2012-14)	AIIMS
	"Elucidation of cytokine gene polymorphisms and its correlation with various disease phenotypes in patients of Aplastic anemia"	AIIMS

3.	Community based planned intervention for the change in perception and health seeking behavior of cancers in Delhi. A pilot study (2009-12)	ICMR
4.	Study No ABB-09-001. Inhibitor Development in Previously Untreated Patients (PUPs) Or Minimally Blood Component –Treated Patients (MBCTPs) When Exposed to Plasma –Derived Von Willerbrand Factor –Concentrates and to Recombinant Factor VIII (rFVIII) Concentrates: An Independent International, Multicentre, Prospective, Controlled, Randomised, Open Label, Clinical TrialMulti centric study (60 centres in 25 countries), (2010-2013)	Fondazione Angelo Bianchi Bonomi
5.	To study the molecular biology fo multiple myeloma (2008-2010)	DBT
6.	An open label, phase 1 dose escalation clinical trial of nrc-an-019 in patients with all phases of chronic myeloid leukemia, who are resistant or intolerant to imatinib mesylate".(2008-2010)	NATCO
7.	Evaluation of molecular genetic in HbE syndrome 2008-10	AIIMS
8.	Molecular cytogenetics study in acute leukemia and myelodysplastic syndrome: Diagnosis and prognostic implications 2005 to 2008 (Haemet)	AIIMS
9.	Incidence of deep venous thrombosis (DVT) among patients admitted in medical wards and intensive care unit (ICU) of a tertiary care hospital (A study protocol) 2006 onwards (Mediceine)	Clexane
10.	Homocysteine: A marker of male infertility 2006 (Urology)	AIIMS
11.	Efficacy of Intramuscular Vitamin K1 versus Intramuscular Vitamin K3 in prevention of sub-clinical classic Vitamin K deficiency; a randomized controlled trial 2004 to present (Pediatric)	AIIMS
12.	A comparative study of Efficacy and Saftey of Ferric Hydroxide Polymaltose Complex And Ferrous glycine sulphate in patients with iron deficiency anemia. 2002-2003. (Haemet)	Emcure
13.	To evaluate the role of Alpha-2b Interferon in CML . 2000 to present. (Haemet)	Fulford.
14.	Scientific evaluation of the impact of changes in life style along the lines of yogic discipline on cardiorespiratory health. 1990 (Physiology)	ICMR

EDPACON 2016

PROGRAMME

Registration 08.00-08.30am

Post Graduate Case Abstract Presentation 08:30-09:20am

Chairpersons Dr. N.P. Singh, Dr. Sandeep Garg, Dr. Pankaj Choudhry

Time allotted per case is 6 min followed by 1 or 2 questions
Best 3 cases will be given awards during inaugural function
Sequence of PG presentation:
Post graduate Case presentation sequence

Welcome Address by Chairman 09:20-09:30am

Dr Ajay Kumar Gupta

Management of Non Ulcer Non Variceal GI Bleed 09:30-10:00am

Speaker Dr. S P Mishra
Chairpersons Dr. Lalit, Dr. Naresh Agarwal
Dr. Saroj Dubey, Dr. Kunal Dass

Managment of Advanced Heart Failure-Ventricular assisted Devices 10:00-10:15am

Speaker Dr. Z.S. Meharwal

Management of Advanced Heart Failure-ECMO 10:15-10:30am

Speaker Dr. Vishal Rastogi
Chairpersons Dr. Ajay Mittal, Dr. Anand Pandey
Dr. Sunil Bhardwaj, Dr. Gaurav Minocha

Maximizing with the minimum: Managing diabetes with comorbidites 10:30-10:50am

Speaker Dr. A H Zargar

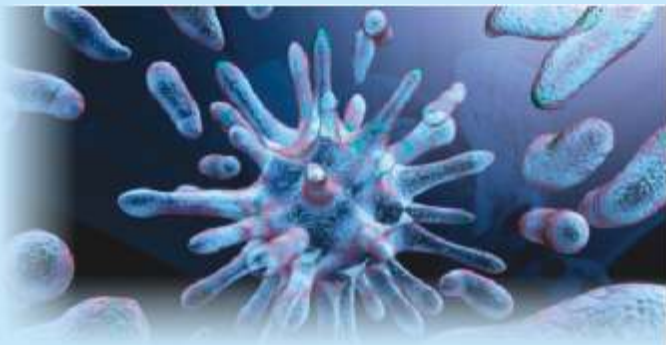
Diabetes Session Coordinator - Dr. Aman Rohtagi

Newer Dpp4's -The Indian perspective 10:50-11:10am

Speaker Dr. (CoL) Surender Kumar
Chairpersons Dr. S V. Madhu, Dr. Anil Motta
Dr. Rajiv Chawla, Dr. Vijay Arora

Indian Phenotypes: Breaking the Nexus With SGLT 2 Inhibitors 11:10-11:30am

Speaker Dr.Kalyan Kumar Gangopadhyay
Chairpersons Dr. Naresh Dang, Dr. Navin Atal
Dr. Rajesh Rajput, Dr. Amitesh Aggarwal



Insulin Intensification Strategies — 11:30-11:50am
Dr. Abhay Alluwalia

Advancements in GLP1 receptor agonist Therapy : Benefits & Limitations — 11:50-12:10pm
Speaker — Dr. Sunil Mishra
Chairpersons — Dr. S.K Wangnoo, Dr. Rajiv Gupta, Dr. Saibal Chakraborty, Dr. Vimal Narka

Meet the Experts of Diabetes — 12:10 -12:209m
Speaker — Q&A

Inauguration of EDPACON 2016 — 12:20-12:50pm

Late Mrs. Bela Devi Oration — 12:50-13:40 pm
Speaker — Prof. (Dr.) Renu Saxena, HOD Haematology, AIIMS

Lunch — 13:40-14:30 pm

Update on Infectious Diseases — 14:30-15:00pm
Moderator — Dr. Rajendra Kapila
Chairpersons — Dr. Ashok Grover, Dr. Micky Malhotra, Dr. Mukesh Mehra, Dr. Sanjay Joshi

OSA : What a Physician should know? — 15:00-15:30 pm
Speaker — Dr. Randeep Guleria
Chairpersons — Dr. N. K . Govil, Dr. B. K. Tiwari, Dr. Parveen Pandey, Dr. Arjun Khanna

Recent Advances in COPD — 15:30-16:00pm
Speaker — Dr. Ashok Mahasur
Chairpersons — Dr. Randeep Guleria, Dr. Saurabh Shrivastav, Dr. M. K. Seth, Dr. K K Pandey

Recent Advances on ART — 16:00-16:30 pm
Speaker — Dr. B B Rewari
Chairpersons — Dr. Rajiv Lochan , Dr. KJS Narula, Dr. MPS Chawla, Dr. S K Gupta

CPC — 16:30-17:00
Moderator — Dr. Paras Gangwal
Presentor — Prof. Dr. N.P. Singh
Discussant — Dr. Nitin Sinha

Valedictory Function

Acute Febrile Illness An Uncommon Etiology

***Atul Chopra**
****P N Choudhry**
*****Ashok Kumar Grover**

Introduction :

Here we report a case of febrile illness due to brucellosis an uncommon etiology in urban set up.

Case :

Mrs R , 35year old female, resident of UP, was admitted with complaints of fever since 14 days, breathing difficulty since 4 days and vomiting since 2 days. On examination patient was febrile, Pulse- 110/min, RR- 24/min, SpO2- 92%, icterus was present. Pallor, pedal edema, lymphadenopathy, cyanosis, clubbing absent. Abdomen distention was present, tenderness present in right upper abdomen region, shifting dullness present. R/S- B/L breath sounds decreased in basal area, and B/L crepts. Patient was put on ventilator support.

Investigation revealed Hb 13, TLC- 4700, Platelet- 35000, SGOT-328, SGPT- 108, Billirubin- 7.3/ 7.1, Albumin- 2, urea -101, creatinine 1.1. INR- 1.6 CXR- B/L lower zone homogeneous opacities.

Patient was evaluated for dengue fever, chikungunya, malaria, enteric, and rickettsial , leptospira, connective tissue disorder. In view of hematological, hepatorenal and pulmonary dysfunction, she was started on broad spectrum antibiotics and antimalarial. Doxycycline also started prophylactically in view of history of contact with animals. Patient developed coagulopathy on day 3 . So FFP , SDP and PRBC were transfused.

In view of the history of drinking unpasteurized milk the diagnosis of was considered and brucellosis serology was sent which came positive (Brucellosis IgM -4.36). Patient was initiated on IV Gentamycin after which patient showed significant improvement. Brucellosis agglutination assay also sent which also came reactive. After 10 days of gentamycin and doxycycline patient liver function test and coagulation profile improved. Patient also investigated for tuberculosis and malignancy. Pleural fluid examination done which showed exudative picture but ADA level was normal, AFB stain and culture sterile. Bone marrow aspiration and biopsy was also done which was normal. ANA ,dsDNA , cANCA, pANCA, Anti cardiolipin and phospholipid antibody all came negative. Patient initiated on rifampicin and discharge.

On follow up patient was showen significant recovery. Her thrombocytopenia recoverd. LFT, KFT, PT-INR normal and CXR have became normal.

Patient was given gentamycin for 14 days then discontinued. Rifampicin and doxycycline continue for 6 weeks.

* *DNB Medicine 3rd year, Deptt of Medicine, Max Hospital, Vaishali*
** *Senior Consultant, Max Superspeciality Hospital,Vaishali*
*** *Senior Consultant, DNB Medicine 3rd year, Deptt of Medicine, Max Hospital, Vaishali*

Discussion

Brucellosis is one of the world's major zoonoses. The presence of brucellosis in India was first established early in the previous century and since then has been reported from almost all states. It remains an uncontrolled problem in regions of high endemicity such as the Mediterranean, Middle East, Africa, Latin America and parts of Asia1. Brucella abortus, B. melitensis and B. suis are pathogenic for man. Of main concern in India are B. melitensis and B. abortus. Brucellosis is almost invariably transmitted to man from infected domestic animals . Transmission from human to human, mainly mother to child, has been reported but is very rare2. B. melitensis is present in and transmitted by goats and sheep and related animals and is most virulent for man.

Brucellosis is caused by members of the bacterial genus Brucella. These are facultative intracellular Gram-negative pathogens. The ability of Brucella to replicate and persist in host cells is directly linked with its capacity to cause persistent disease and to circumvent innate and adaptive immunity.

Fresh milk and dairy products prepared from unpasteurized milk such as soft cheeses, yoghurts and ice creams may contain high amounts of the bacteria and consumption of these is an important cause of human brucellosis3. Infection also may occur through cuts and abrasions of the skin, via the conjunctiva and by inhalation. These routes of infection are important for farmers, veterinarians and butchers who all have an increased risk of infection through their contact with animals and animal products. The changing and fast growing dairy industry in India has resulted in intensified trade and animal movements and provide a new and increased risk in spreading the infection.

Clinical manifestations :

Fever, chills, sweats, aches, lack of energy, joint and back pain, headache and loss of appetite are observed in majority of the patients4. Commonly patients feel better in the morning, with symptoms worsening as the day progresses. Patients have a strong desire to rest and may be depressed.

Chronic brucellosis refers to those patients in whom symptoms persist for 12 months or more from the time of diagnosis and treatment.

Complications :

Bone and joint involvement are the most frequent complications of brucellosis, occurring in up to 40% of cases.

Infective endocarditis is the most common cardiovascular manifestation, and is said to be the most common cause of death from brucellosis.

Meningitis or meningoencephalitis are the most common manifestations.

Pulmonary complications include hilar and paratracheal lymphadenopathy, interstitial pneumonitis, bronchopneumonia, lung nodules, pleural effusions, and empyema.

Orchitis and epididymitis are the most frequent genitourinary complications of brucellosis in men.

Brucellosis during the course of pregnancy carries the risk of spontaneous abortion or intrauterine transmission to the infant.

Laboratory diagnosis:

Culture from the blood of a patient provides definite proof of brucellosis.

The classical Rose Bengal test (RB) is often used as a rapid screening test.

The sensitivity of RB is very high (>99%) but the specificity can be disappointingly low.

For confirmation of RB the Wright or serum agglutination test (SAT) or in more sophisticated equipped laboratories enzyme linked immunosorbent assay (ELISA) may be used5.

A simple and rapid diagnostic test, the Brucella IgM/IgG flow assay very useful and has sensitivity and specificity > 95 %.

Treatment :

For acute brucellosis in adults and children older than 8 years, the World Health Organization (WHO) guidelines recommend the following:

Doxycycline 100 mg PO twice daily plus rifampin 600-900 mg/day PO Both drugs are to be given for 6 weeks; this regimen is more convenient but probably increases the risk of relapse

Doxycycline 100 mg PO twice daily for 6 weeks and streptomycin 1 g/day IM for 2-3 weeks This regimen is believed to be more effective, mainly in preventing relapse; gentamycin can be used as a substitute for streptomycin and has shown equal efficacy.

Georgia Vrioni at al published study in 2014 which showed that doxycycline-streptomycin-rifampin regimen eliminates brucella DNA more efficiently than doxycycline-streptomycin, which may result in superior long-term clearance of brucella6.

Preventions :

1. All dairy products should be prepared from heat-treated milk.
2. Consumption of raw milk or products made from raw milk should be avoided.
3. Meat should be adequately cooked.
4. Special precautions should be taken by laboratory workers.
5. Physicians and health workers should be aware of the possibility of brucellosis.
6. Public health education should emphasize food hygiene and occupational hygiene.

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CASE REPORT

***Dr. Sanket Mathphukia**
****Dr. Atul Chopra**
*****Dr. Pankaj Chaudhary**
******Dr. Ashok Grover**

Introduction

Histoplasmosis is a granulomatous fungal disease caused by the intracellular dimorphic fungus Histoplasma capsulatum.Also known as "Darling's disease," "Ohio valley disease", "Caver's disease".[1][2]The organism is more prevalent in certain parts of North and central America and has been documented in the soil of the Gangetic plains of Eastern India.[7]Sporadic cases have been reported in southern parts of India. It exists in two forms, the infective mycelial or mould form in the soil and the yeast form in human macrophages. Histoplasmosis occurs most commonly in immunosuppressed patients, there are a few case reports of this disease in immunocompetent hosts. We hereby report a case of pulmonary histoplasmosis in an immunocompetent individual.

Case Report

A 53 year old femalehousewife, resident of shahadra (delhi), presented with complaints ofFeveracute in onset, moderate to high grade, continuous and not associated with chills or rigors, no diurnal variation, FEVER relieved with paracetamol tablets, Cough which was initially dry for 10-14 days, later produced yellowish expectoration scanty associated with chest pain on lower side bilaterally and pain in abdomen in right upper side , acute onset, dull aching, non colicky, non radiating, not associated with nausea or vomiting. No history of headache, dyspnoea or palpitation, weight loss or anorexia, urinary symptoms, loose stools or any rash over body. She had No significant past history.

Prior to admission patient was investigated in other premises where routine blood invstigations revealed

TABLE: 1 Investigations

Haemoglobin	8.6
Total leukocyte count	9.8
Platelet count	80
PCV	27.3
UREA	13.4
Creatinine	0.8
Serum sodium	132
Serum potassium	3

* *DNB Medicine 2nd year, Deptt of Medicine, Max Hospital, Vaishali*
** *DNB Medicine 3rd year, Deptt of Medicine, Max Hospital, Vaishali*
*** *Senior Consultant, Max Superspeciality Hospital,Vaishali*
**** *Senior Consultant, DNB Medicine 3rd year, Deptt of Medicine, Max Hospital, Vaishali*

TABLE: 2 Investigations

SGOT	267.3
SGPT	60.7
ALP	144
GGT	145
TOTAL BILIRUBIN	1.3
DIRECT BILIRUBIN	0.8
Albumin	2.8
ESR	40
CRP	3.2

Urine R/M was not significant while USG (W/A) suggested moderate hepatomegaly with mild splenomegaly. Chest x ray was normal. Further investigations like Quantiferon TB GOLD test which was positive but Mx test was negative. Sputum examination was done which denoted AFB Negative, and no any fungal elements seen. Both blood and urine cultures were sterile. As routine investigations not more conclusive, CECT chest was performed suggestive of mediastinal lymphadenopathy, small tiny subpleural nodule in right upper lobe, diffuse ground glass opacity in bilateral lungs predominantly involving lower lobes. Intravenous antibiotics were prescribed to her for 14 days and treated as bacterial pneumonia. But fever persists even after antibiotic course.

On admission patient was Conscious, oriented,Temperature – 100.2 degree F, PR- 82/ minute, BP- 130/80, RR- 18/min, RBS- 110 mg/dl, SpO2- 98% on room airpallor +No icterus, clubbing, cyanosis, edema, lymph node enlargement, JVP not raised, No neck rigidity, on systemic examination chest auscultation suggestive of Bilateral vesicular breath sounds and basal crepts, per abdomen, was Soft, No splenomegaly, no tenderness, no free fluid was noted, liver was just palpable.

Based on CT report and prior history of patient, she was advised for endoscopic ultrasound and FNAC which finally revealed Fungal infection morphologically compatible with Histoplasmosis. Patient started on itraconazole 200mg twice a day for 12 weeks and intravenous amphotericin B 3 mg/kg once a day 14 days. Patient has shown improvement, fever subsided in two weeks of starting therapy. Amphotericin B Stopped with continuing itraconazole to complete the course.

DISCUSSION

Histoplasmosis is rare in the immunocompetent host. Histoplasmosis is an uncommon

disease caused by Histoplasma capsulatumvarcapsulatum and Histoplasma capsulatumvarduboisii. [1][2] In India,as the disease can mimic tuberculosis clinically and histopathologically, many cases may potentially be missed or misdiagnosed as tuberculosis. In India histoplasmosis is rare, as it is thought to be endemic only in West Bengal in western India. [3][4] However, sporadic cases from southern India are frequently reported. Our patient was a resident of a nonendemic region of north India, [5] but area near her resident was quite damping, open gutter nearby. We believe that our patient obtained the disease via inhalation of conidia of Histoplasma fungus present in the soil contaminated by excreta of birds. [6] Inhalation of either fungal conidia or mycelial fragments is the primary mode of infection for histoplasmosis. Once inhaled, these fungal elements transform into yeast forms and may disseminate systemically.

Primary pulmonary histoplasmosis in the vast majority (approximately 90%) of cases is asymptomatic or presents with subclinical disease. Symptomatic hosts with primary pulmonary histoplasmosis often present with nonspecific symptoms of fever, chest pain and cough that are self limited. Immunocompetent hosts are able to control and limit infections of Histoplasma; however, hosts with defective cell-mediated immunity, including patients with hematolymphoid malignancies, solid-organ transplants, and those exposed to chemotherapeutic and immunosuppressive agents, are at risk of developing progressive disseminated histoplasmosis involving the reticuloendothelial system, including the liver, spleen, kidney, lymph nodes, bone marrow and mucocutaneous tissues.Recognition of varied clinical manifestations of histoplasmosis, improved laboratory facilities and extensive population based studies to know the endemicity of histoplasmosis in various regions is essential for early diagnosis and effective treatment.

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SLE with active nephritis and concomitant tuberculosis: A therapeutic challenge

***Jyotsana**
****Nitin Sinha**

Abstract :

A 23 years old married lady presented with complaints of fever, malaise, polyarthralgia, undocumented weight loss, oral ulcers and rash over the face occuring over last two months. She had maculo-papular rash over malar area, oral ulcers, dry dangrene over tip of left ring finger and non-tender cervical lymphadenopathy. Her investigations revealed normocytic normochromic anemia, elevated ESR and significant proteinuria. ANA and antidsDNA were positive and there was hypocomplementemia. Chest X-ray and Ultrasound abdomen were normal. HIV was non-reactive. FNAC from cervical lymph node showed caseous necrosis with acid fast bacilli suggestive of Mycobacterium tuberculosis infection. Renal biopsy showed Class III Lupus Nephritis.

Infections and nephritis are the leading cause of mortality in SLE patients in the first decade of the disease. Tubercular infections in SLE patients have been proposed to occur as a consequence of SLE treatment. In our patient, SLE with Lupus Nephritis and Tubercular Lymphadenitis were diagnosed concomitantly. Due to active tuberculosis, immunosuppressant therapy could not be initiated despite presence of organ threatening SLE nephritis. Only pulse methylprednisolone was given and later, she has been continued on oral steroids. She has also been initiated on anti tubercular therapy. She is under active follow up.

* *Post Graduate (2nd year), Department of Medicine, PGIMER and Dr. RML Hospital, New Delhi*
** *Associate Professor, Department of Medicine, PGIMER and Dr. RML Hospital, New Delhi*

Thrombotic Thrombocytopenic Purpura in a patient with Brucella infection

***Dr. Jai Khullar**
****Dr. R K Singhal**

Abstract :

Thrombotic thrombocytopenic purpura (TTP) is characterized by disseminated thrombotic occlusions located in the microcirculation and a syndrome of microangiopathic hemolytic anemia (MAHA), thrombocytopenia, fever, and renal and neurologic abnormalities. Although several factors such as viral and bacterial pathogens, pancreatitis, drugs, collagen-vascular diseases, cancers, and pregnancy have been reported to be associated with TTP, brucellosis is an exceptional cause of this disorder. We present a case of a 33 year old male who was initially investigated at a government hospital for PUO and started on empirical ATT after Whole body PET-CT showed generalized, non-necrotic lymphadenopathy. Following unsatisfactory response, he presented to our hospital 2 weeks later and during the course of admission he was found to have Brucella antigen (IgG) positivity. He responded well to antibiotic therapy directed to Brucella infection and was discharged, however, after an initial good symptomatic response for 15 days, he reported B/L diminution of vision, and became febrile yet again. He was found to have severe thrombocytopenia. Ophthalmology opinion was taken and patient was diagnosed to have retinal haemorrhages, in view of which he was readmitted. He was found to have severe thrombocytopenia with a normal coagulogram and raised LDH with renal azotemia. Peripheral blood smear showed fragmented RBCs, laboratory findings being consistent with Thrombotic thrombocytopenic purpura. He was taken up immediately for Plasma exchange under guidance of Hematology and Transfusion Medicine departments, however, he relapsed after initial five cycles. He underwent further four plasma exchanges with unsatisfactory response and was eventually started on Rituximab to which he responded well.

* *DNB Resident (Medicine), Department of Internal Medicine*
BLK Super Speciality Hospital, New Delhi
** *Senior Consultant, Department of Internal Medicine*
BLK Super Speciality Hospital, New Delhi

Disseminated Histoplasmosis (DH) is a granulomatous disease of varied presentations.

***Dr Roli Bansal**
****Dr Anil Yadav**

Immunocompromised individuals are more susceptible for the disease. We present a case of a 60 year old diabetic and hypertensive male presenting with features of anorexia, weight loss and skin lesions of 3 months duration. On examination he had hypotension and papular lesions on the chest. Evaluation revealed presence of bilateral adrenal enlargement on MRI and adrenal insufficiency on ACTH stimulation test. Biopsy of the skin and the adrenal tissue was done. It was suggestive of histoplasmosis. Culture of the adrenal tissue also revealed histoplasmosis. Patient was treated with antifungals and showed marked improvement. DH is a relatively rare entity in the Indian subcontinent with very few case reports of DH with adrenal and skin involvement. As its clinical presentation shares common symptomatology with disseminated tuberculosis, a high index of suspicion should be kept to pick up such treatable cases.

* *Senior Resident, Deptt of Medicine, UCMS and GTB Hospital, Delhi*
** *Medical Superintendent, GTB Hospital, Delhi*

A case of Abnormal Head Shaking

***Dr Neha Agarwal**
****Dr Suman Kushwaha**

An interesting case of abnormal continuous involuntary head shaking (no no type) movements in a 13 yr old boy.

The movements were acute in onset, non jerky, stereotyped, and continuous for last 1 wk and only suppressed during sleep. Also, associated with abnormal vertiginous sensation in the head.

He was being treated as psychogenic movement disorder by various physicians. Psychogenic movement disorders are the diagnostic challenge and should be thoroughly investigated and managed accordingly.

* *DM Neurology, Institute of Human Behavior & Allied Sciences, Delhi*
** *Prof. Neurology, Institute of Human Behavior & Allied Sciences, Delhi*

SIMULTANEOUS OCCURRENCE OF CEREBRAL BLEED AND INFARCT

***Dr. Ankur Chikara**
****Dr. Amitesh Aggarwal**

Abstract:

A female patient aged 80 year old, came with chief complaints of altered sensorium since last 4 days. Patient was apparently normal 4 days back when she developed altered Sensorium which was sudden in onset. Patient was bedridden, was not speaking to anyone, was not accepting meals. There was no h/o preceding fever or seizures.

On examination patient was conscious but disoriented, pallor was present, BP-92/64 mm hg in right arm supine position, PR-112 with regular rhythm, good volume and normal vessel wall character.

Further detailed investigations were done which showed hb-10.1 gm/dl, platelet count-92000, Na-126, KFT and LFT were normal, RBS-124 mg/dl and lipid profile was normal. Peripheral blood smear revealed microcytic hypochromicanaemia.USG whole abdomen was s/o cholecystitis. NCCT head revealed hyperdense lesion in left parieto-occipital lobe with peri-lesionalhypodensity, s/o parenchymal bleed with peri-lesional edema and wedge shaped hypodense area in the right parieto-occipito watershed zone s/o infarct with no midline shift.

During the next 4 days of hospital stay platelet count remained consistently low, serum electrolytes and serum blood urea were normal and TLC increased to 15500 cumm on day 4th.

Management:

Target for blood pressure control

Use of antiplatelets

Use of procoagulants for haemostasis

To maintain cerebral perfusion pressure, which may require inotropes

Discussions:

Simultaneous occurrence of intracerebral bleed and infarct in a patient is a rare event which possess challenge in management

Both type of strokes share the same common vascular pathology but management is different

In intracranial haemorrhage-the aim is to limit haemorrhage growth, hemostasis with procoagulant agents and surgical evacuation

Recommendation-To decrease event, reduce MAP<130 mm hg during acute phase

Ischaemic infarct-If in window period, use of antiplatelets and statins for thrombolysis

Stroke prone period exist after any stroke, so to prevent maintain CPP>60 mm hg

During the pro inflammatory stroke prone period, using of antiplatelets and statins is beneficial to prevent secondary neuronal injury

Check for the other risk factors like atrial fibrillation, coronary artery stenosis and hypercoagulable states

Sugar levels should be maintained strictly between 140-180 mg/dl

* *DNB Resident (MEdicine), UCMS & GTB and Hospital*
** *Associate Professor UCMS & GTB Hospital*

RATIONALE USAGE OF HUMAN ALBUMIN SOLUTION

***Dr N P Singh**

Introduction:

Human albumin is a physiological plasma expander.Its limited availability and high cost make it essential to define recommendations for its appropriate use, as an alternative to other therapeutic strategies including solutions of crystalloids and non protein colloids. Total body albumin pool measures about 250300 g for a healthy 70 kg adult and about 50% of the overall content of plasma protein which is responsible for about 80% of the intravascular oncotic pressure. It creates 75% of colloid osmotic pressure. Liver produces about 10-12 g of albumin each day, which is immediately secreted into the intravascular space by the cells without being stored. The entire process of synthesis and secretion is quite rapid, taking about 30 minutes. About 7 g/h of albumin passes into the interstitial space.In regions of endothelium with large gaps, the filtration of albumin is passive. In regions with non-fenestrated endothelium, its filtration is under the particular action of a specific receptor, i.e., albondin. The passage of albumin between the intravascular and the interstitial spaces is a continuous process, with a return to the blood-stream through the action of lymph drainage. At the end of the entire process, albumin is usually degraded ubiquitously, in an amount comparable to that synthesized by the liver (10-12 g/ 24 h).

Normal albumin shift

In normal subjects, about 6 g of albumin move from the intravascular to the interstitial space every hour, returning to the lymphatic system. Since each g of albumin binds about 18 grams of water, this results, theoretically, in a fluid circulation of about every 24 hours.

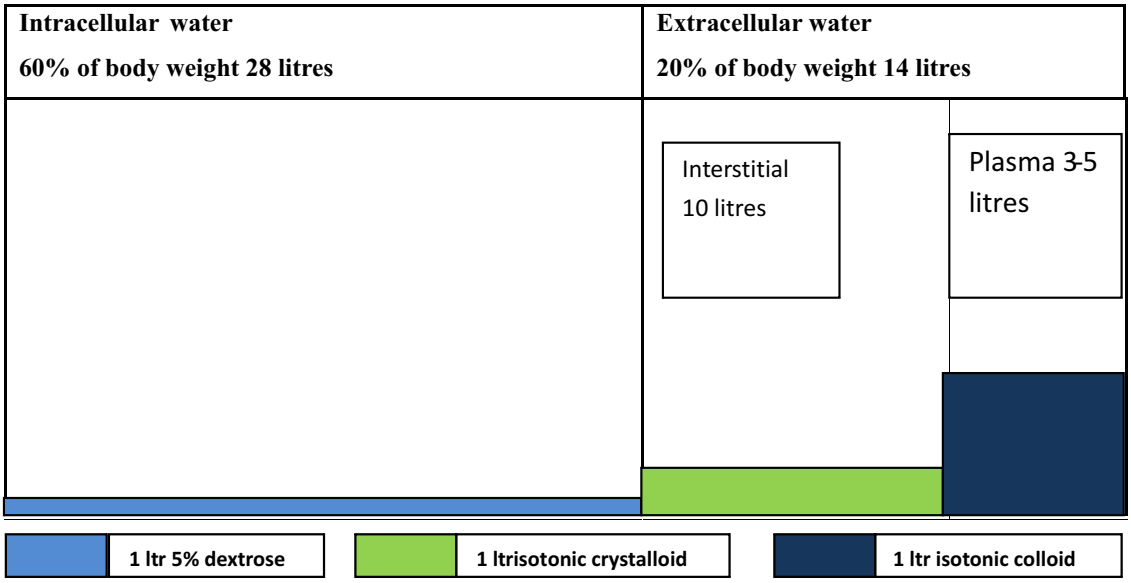

Fig. 1.Distribution of body water within the different compartments at equilibrium after the infusion with the 3 specified solutions.

* *Director Dept. of Internal Medicien & Medical Advisor, Max Super Speciality Hospital, Vaishali*

Preparations of albumin

Preparations of albumin Solutions of albumin are prepared from the plasma of healthy donors. The albumin is pasteurised at 60 °C for 10 hours. It can be infused independently of the recipient's blood group. Preparations of 5%, 20% and 25% are available. The solutions of 5% human albumin have an osmotic pressure almost identical to that of normal plasma; the 20% and 25% solutions are hyperosmotic. All the preparations contain 130 - 160 mEq of sodium per litre. 20% albumin is also available in low salt version (containing 125 mEq of sodium per litre).

Advantage of albumin

Albumin offers several advantages compared with artificial colloids, including less restrictive dose limitations, lower risk of impaired haemostasis, absence of tissue deposition, reduced incidence of anaphylactoid reactions, and ease of monitoring to prevent fluid overload.

How to choose?

Albumin (Human) 5%

- Osmotically equivalent to an equal volume of normal human plasma
- increase the circulating plasma volume by an amount approximately equal to the volume infused

Albumin (Human) 25%

- colloid osmotic effect is approximately 5 times its volume of human plasma
- increase the circulating plasma volume by an amount of about 3.5 times the volume infused

Albumin as drug

Albumin clearly has important physiological functions, and hypoalbuminemia is strongly associated with poor clinical outcomes. This does not translate into evidence that albumin infusion results in improvements, as assessed through different meta-analysis.Despite this, albumin's therapeutic potential based on its physiological functions has led to several **proposed clinical roles in a number of indications as-**

Hypovolemia: While most internal organs can lose more than 50% of their function before organ failure is apparent, loss of only 30% to 40% of the blood volume can result in life-threatening circulatory failure, and even minor degrees of hypovolemia can cause ischemia and organ dysfunction. In hypovolemic shock albumin is used as a **second choice,** when **solutions of crystalloids or non-protein colloids (first choice treatment)** have already been used at maximum doses without having produced a clinically adequate response and in cases in which non-protein colloids are contraindicated. Crystalloid and colloid solutions must not be considered as blood replacements when oxygentransporting capacity is reduced. Albumin 5% must be used.

Major surgery: The use of albumin may be indicated in subjects undergoing major surgery (> 40% resection of the liver, extensive intestinal resection) when, after normalisation of circulatory volume, the serum albumin is < 2 g/dL is recommended. The use of albumin in the immediate post-operative period is never advised for any other type of operation.

Burns: There is no indication to use albumin in the resuscitation phase in the first 24 hours after burn injuries, that is, in the period of increased capillary permeability. Subsequently, albumin 5% is indicated, using different doses according to the amount of body surface area (BSA) involved:

- BSA 30 - 50%: 0.3 mL x kg x % of burnt BSA, in 24 hours;
- BSA 50 - 70%: 0.4 mL x kg x % of burnt BSA, in 24 hours;
- BSA 70 - 100%: 0.5 mL x kg x % of burnt BSA, in 24 hours.

In the post-resuscitation phase, once the problems of circulatory volume caused by the marked capillary permeability have been overcome, albumin 5% or 20% is infused at a dose of 1 - 2 g/kg/die if: - albumin < 1 g/dL (end-point 2 g/dL) OR albumin 1-2 g/dL and the patient cannot tolerate an enteral diet or has massive tissue oedema or pulmonary dysfunction, which could be aggravated by a low oncotic pressure (end-point 2 g/dL).

Heart surgery: Albumin can be used as a post-operative volume expander, as a last choice of treatment after crystalloids or non-protein colloids, following heart surgery.Crystalloids are the first choice for priming the circuitry in the case of extracorporeal circulation; the association with non-protein colloids can be preferable to avoid the accumulation of fluid in the pulmonary interstitium.

Volume replacement/expansion in CABG: The use of albumin for volume replacement/expansion in coronary artery bypass graft (CABG) surgery, both as a pump priming fluid and postoperatively, is well reflected in current usage. Albumin maintains a less positive fluid balance then crystalloids when used as a priming agent. Albumin levels are a strong predictor of morbidity and mortality and albumin administration maintains hemodynamic stability and decreases mortality, through mechanisms which may include albumin's role in suppressing inflammation and endothelial activation, which are both pathological outcomes in CABG.

Organ transplantation: Albumin can be useful in the post-operative period following liver transplantation, in order to control the ascites and peripheral oedema and to replace the loss of ascitic fluid through the drainage tubes. It is administered in the following circumstances: albumin < 2.5 g/dL, pulmonary capillary pressure < 12 mmHg, haematocrit> 30% (Grade of recommendation: 1C). There is not definitive evidence that albumin and/ or non-protein colloids are effective during or after kidney transplants.

Therapeutic plasmapheresis: The use of albumin is appropriate only for the exchange of large volumes of plasma: more than 20 mL/ kg in a single session or 20 mL/kg/week in successive sessions.In the case of exchange of small volumes of plasma, it is worth considering, for cost-benefit reasons, crystalloid solutions or the association of albumin/ crystalloids.

Chronic states of low albuminaemia

Liver cirrhosis with refractory ascites

There is a lack of consensus on the use of albumin in advanced liver disease, but there is some evidence to support its use in the following circumstances:

1) Ascites not responsive to diuretics

This is the most controversial indication. Albumin is usually ineffective, except in patients with

serum albumin < 2 g/dL. Subjects with ascites are at risk of diuretic-induced hyponatraemia and deteriorating renal function (prerenaluraemia); the risk is highest in subjects with hypoalbuminaemia and advanced disease. Albumin can improve the response to diuretics and prevent complications. The patients who can gain most benefit from this treatment are those in the most precarious clinical condition, with hypovolaemia and ascites that responds poorly to diuretics: in these cases albumin can be administered even when the concentration of albumin is > 2.5 g/dL.

2) Large volume paracentesis

A paracentesis volume > 5L can, in some cases, lead to hypovolaemia and particularly unfavourablehaemodynamic changes, with the possible risk of deterioration of renal function; dilutionalhyponatraemia;rapidly recurrent ascites; shortened survival.In order to reduce the risks in such cases, albumin is used at a dose of 5 g/L of fluid removed, in a single administration at the end of the paracentesis. The 20% - 25% preparations are preferable.

3) Hepatorenal syndrome (HRS)

HRS consists of deterioration in renal function, which occurs in 10% of subjects with advanced cirrhosis and ascites. It is considered the extreme outcome of the haemodynamic dysfunction of cirrhosis, associated with impaired cardiac function due to the reduced venous return. The deterioration in renal function can be rapidly progressive (type 1 HRS) or stable-slowly progressive (type 2 HRS); the mortality rate of patients with type 1 HRS is very high, with a median survival (without therapy) of less than 1 month. The treatment of choice is liver transplantation. Medical treatment consists of a combination of vascoconstrictors and high doses of albumin (Grade of recommendation: 2B). Administration of HAS and vasoconstrictors are effective therapy in 60% of patients with HRS and is associated with improved survival. Terlipressin: 0.5 2mg IV every 4 hours, plusDay 1: 1g / kg HAS

Day 2: 16: 20 40 g HAS / day. Recommend continued until serum creatinine falls below 130 mol/lNB. Where creatinine is rising despite Rx, 60g HAS /day is clinically indicated.

4) Spontaneous bacterial peritonitis

Spontaneous bacterial peritonitis is a common and severe complication of ascitic cirrhosis and occurs in about 20 - 30% of patients; it is characterised by spontaneous infection of the ascitic fluid, in the absence of abdominal sources of infection, and can evolve, in about 30% of the cases, into HRS. Albumin 20% - 25%, in association with antibiotics, can be used in the treatment of spontaneous bacterial peritonitis and reduces the probability of the onset of HRS and mortality.

Nephrotic syndrome

Short-term infusion of albumin 20% - 25%, in association with diuretics, is appropriate in patients with serum albumin < 2 g/dL, with marked hypovolaemia and/or acute pulmonary oedema and/or acute renal failure.

Malnutrition syndromes

Albumin must not be used for nutritional purposes; the correct treatment is enteral nutrition, using peptide-based formulas, or total parenteral nutrition. However, the administration of albumin can be useful in patients with diarrhoea who cannot tolerate enteral nutrition in the following circumstances:

- Volume of diarrhoea> 2 L/die;
- Serum albumin < 2 g/dL;
- Continuing diarrhoea despite the administration of short-chain peptides and mineral formulas;
- No other cause to explain the diarrhea.

Hypoalbuminemia

Hypoalbuminemia is well established as a prognostic indicator in many disease states and, despite a paucity of evidence that albumin administration improves survival, analysis of dose-dependency in controlled trials of albumin therapy suggested that complication rates may be reduced when the serum albumin level attained during albumin administration exceeds 30 g/l. This observation may explain the continuing use of albumin in this condition, including a substantial usage in correcting hypoalbuminemia-related hypotension in hemodialysis patients.

Characteristics for critically ill

The molecular structure of albumin has three main characteristics which may be considered important for critically ill patients: (i) cysteine residues, (ii) domains I and II, and (iii) imidazole residues. Cysteine residues expose a SH radical group (thiol), which is one of the main extracellular antioxidants. SH residues bind nitric oxide to form S-nitrous thiols, thereby neutralising one of the most important mediators of pathological conditions such as sepsis. Albumin domains I and II are responsible for the transport of the numerous molecules. Finally, albumin has 16 histidine imidazole residues with pH 6.75, which are responsible for the buffer function of albumin.

Critically ill traumatic brain injury

As mentioned above, the SAFE study suggested that trauma patients, especially those with traumatic brain injury, treated with albumin infusion had a higher 28-day mortality rate than that of patients treated with normal saline. In fact, based on the findings summarized above, we may clearly state that in this specific category of critically ill patient, i.e., patients with an active brain injury due to cerebral trauma, albumin administration should be avoided, preferring other types of fluids, such as normal saline, for acute volume resuscitation.

Peripheral odema in resuscitation phase of critically ill

Along the same line of reasoning, the role of albumin, especially because of its oncotic properties, may gain even greater importance in the clinical phase that usually follows the acute phase of volume replacement and resuscitation. The clinical priority is normally elimination of the excessive fluid previously accumulated in the interstitial space during the resuscitation phase. The acute florid phase during volume replacement tends to normalise. The albumin infused in this situation is, therefore, more likely to "remain" within the intravascular space than usually occurs during the acute phase.

Sepsis

Sepsis is a very dramatic syndrome commonly affecting most patients admitted to ICUs. It involves many inflammatory mediators which have been considered responsible for the haemodynamic alterations and energy failure, as well the multi-organ dysfunction. Besides

its oncotic properties, albumin may play a critical role in aiding the normalisation of many of the inflammatory pathways through its secondary functions, such as the modulating action on nitric oxide metabolism and free radical production, its buffer effect in the acid-base equilibrium, and its action as a transporter of many different substances and drugs. In a subgroup analysis of the SAFE study and a subsequent meta-analysis a survival benefit was apparent when treating septic patients with albumin. This may initiate cascades of thiol oxidative-reductive reactions and influence cellular signaling processes.Despite the increased capillary permeability seen in sepsis, albumin administered to septic patients expands the plasma volume and exerts a hemodynamic effect. Preliminary results from a major trial confirm the benefits of albumin in septic shock. These findings suggest cumulatively that the Surviving Sepsis Campaign's recommendation of albumin as the fluid of choice when colloid resuscitation is needed is based on evidence.

Maintenance of serum albumin?

The normal serum concentration of albumin in healthy adults is approximately 35 to 50 gL-1. Hypoalbuminaemia is common in seriously ill patients. Frequency of hypoalbuminaemia (serum albumin concentration of less than 34 gL-1) as 21% at the time of admission in adult hospitalized patients. Because of its importance as an outcome predictor, serum albumin level has been added as one of the component parameters in the APACHE III score. However, it is to be remembered that changes in its values are the result of pathological events, and not the cause of them.

Low serum albumin in acutely ill

Each 10 gL-1 decline in serum albumin concentration significantly raised the odds of mortality by 137%, morbidity by 89%, prolonged the ICU and hospital stay by 28% and 71% respectively. A serum albumin level of <2.0 gdL-1 in critically ill patients has been shown to be associated with a mortality of nearly 100%.

Conclusion in critically ill

At the moment, based on the evidence currently available, we can state that albumin is not necessary for normal volume replacement in moderate critically ill patients and, furthermore, that it should be avoided in patients with traumatic brain injury. In contrast, in patients with severe hypoalbuminaemia and peripheral oedema during the recovery phase after acute volume replacement, albumin administration may have a beneficial impact, especially on the elimination of the excessive accumulated volume.Finally, one of the most important categories of patients for which preliminary results suggest a potential beneficial role of albumin on outcome is that of patients with severe sepsis.

Dosage calculation

The dose needed to obtain a serum albumin 2.5 g/dL is calculated using the following formula:

Dose (g) = [desired albumin concentration (2.5 g/dL) actual albumin concentration (g/dL)] x plasma volume (0.8 x kg)

Hemodynamic performance monitored regularly -

- Arterial blood pressure and pulse rate
- Central venous pressure

- Pulmonary artery wedge pressure
- Urine output
- Electrolyte
- Hematocrit/ hemoglobin

Safety of albumin infusion

Albumin is usually well tolerated. However, immediate allergic-type reactions are possible with fever, shivers, nausea, vomiting, urticaria, hypotension, increased salivation, and effects on respiration and heart rate. Very fast infusions (20 - 50 mL/minute) can cause a brusque fall in systemic blood pressure and, in elderly subjects and those at risk of congestive heartfailure, it can induce manifest congestive heart failure, particularly when the more concentrated solutions of albumin are used.

What can go wrong ?

There are several reasons why albumin supplementation might make things worse for critically ill patients. Cardiac decompensation may occur after rapid volume replacement with 20% albumin since this leads to a four fold increase in volume retention.In patients with capillary leak syndrome, albumin may become detrimental when albumin and water cross the capillary membrane and cause or worsen pulmonary edema, thus compromising tissue oxygenation and finally leading to multi-organ failure.The antihaemostatic and platelet lowering properties of albumin may increase blood loss in post surgical or trauma patients.Albumin administration in the resuscitation of hypovolaemic shock may impair sodium and water excretion and worsen renal failure.Certain commercially available preparations of albumin contain remarkable quantities of ions generated during the preparation process. In patients with acute renal failure, potentially toxic concentrations of aluminium may accumulate after massive albumin administration. Hypotension has been reported to occur after albumin administration and is most likely caused by vasoactive peptides.

Recommended readings:

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DOPING: WHAT A PHYSICIAN SHOULD KNOW?

***Dr Nitin Sinha**

Introduction

Doping is a sporting menace and has been prevalent from the time competitive sports has existed. Eminent athletes from different sports being caught for doping is not unheard of. Doping not only is detrimental to the career of the athlete but also brings disgrace to the country for which athlete is playing. Doping is any of the following:-

1. Presence of prohibited substance or method
2. Use or attempted use of a prohibited substance or method
3. Evading or refusing sample collection after being notified
4. Failure to file athletes whereabouts information
5. Tampering with any part of doping control process
6. Possession of a prohibited substance or method
7. Trafficking a prohibited substance or method
8. Administering/attempt to administer any prohibited substance or method to an athlete
9. Prohibited association
10. Complicity- Assisting, encouraging or any other type of Intentional Complicity

Certain substances and methods give athletes an undue advantage over their competitors. A physician must be aware about these substances and methods and be cautious while prescribing them to athletes.

Agencies Looking after Doping:-

World Anti Doping Agency (WADA) is the International body while National Anti Doping Agency (NADA) is the body in India. These conduct dope tests, conduct enquiry and proceedings in doping cases, issue Therapeutic Use Exemption certificates (TUE) and educate athletes, physiotherapists, coaches and doctors regarding doping. Over last couple of years, NADA has been trying to educate doctors regarding this menace as they noticed that many of the times doctors claim unawareness regarding drugs that can be used in athletes.

List of Prohibited Substances:-

It is imperative to know that certain substance/method use is prohibited whether athlete is in-competition or out of competition. It is noteworthy here that an athlete can be tested anytime, anywhere by the authorities and hence, these substances/methods should never be used or used with proper certification, if required. There are certain substances which are prohibited only in-competition and further, there are certain substances which are prohibited only in particular sports. Following is a brief list of these substances as per 2017. Detailed list can be viewed on NADA website (www.nada.nic.in).

* *Associate Professor PGIMER & RML Hospital, Delhi*

SUBSTANCES AND METHODS PROHIBITED AT ALL TIMES (in-competition and out of competition):-

Substances:-

S0: Non-approved substances--- Any of the pharmacological substances which is not addressed by any of the subsequent sections of the List and with no current approval by any governmental regulatory health authority for human therapeutic use is prohibited at all times

S1: Anabolic Agents---- Eg-- Anabolic androgenic steroids (AAS)

S2: Peptide Hormones, Growth Factors, related substances and Mimetics---- Eg-- Erythropoietin receptor agonists, Growth Hormone

S3: Beta-2 agonists

S4: Hormones and Metabolic Modulators--- Eg-- Insulin

S5: Diuretics and Other Masking Agents-- Eg-- Furosemide

Methods:-

M1: Manipulation of Blood and Blood Components--- Autologus, homologus or allogenic red blood cell products transfusion

M2: Physical and Chemical Manipulation--- Tampering of dope samples, intravenous injections or infusions of more than 50 mL per 6 hour period except for those legitimately received in the course of hospital admissions, surgical procedures or clinical investigations.

M3: Gene Doping-- Transfer of normal or genetically modified cells, transfer of polymers of nucleic acids or nucleic acid analogues.

SUBSTANCES PROHIBITED IN-COMPETITION:-

S6: Stimulants--- Eg-- Cocaine, Modafinil, Pseudoephedrine, Ephedrine, Epinephrine (approved for topical use only)

S7: Narcotics

S8: Cannabinoids

S9: Glucocorticoids--- Prohibited use only by intravenous, intramuscular, oral or rectal route

SUBSTANCES PROHIBITED IN CERTAIN SPORTS:-

P1: Alcohol-- Cut-off blood value is 0.1g/L. Prohibited in Air sports, automobile sports, archery and power boating.

P2: Beta blockers-- Prohibited in power boating, archery, billiards, darts, golf, shooting and under water sports.

Therapeutic Use Exemption (TUE):-

As noticed above, certain substances are used as drugs to treat medical disorders (Eg:- Beta blockers for hypertension, insulin for diabetes, beta 2 agonists for asthma). In athletes who are suffering from a particular disease, a TUE is issued to use a particular banned substance but only in approved doses and approved route. A TUE is issued when all the following are met:-

- Athlete health will be significantly impaired if they do not take the substance
- The substance does not enhance the athlete performance to beyond what brings athlete to normal level
- There is no alternative treatment available

Certain drugs which are otherwise prohibited but are often required in management of certain medical conditions along with the maximum permitted dosages are listed in the table below (Table 1):-

Table 1: Prohibited substances but often used for medical conditions along with their maximum permitted dosages

SUBSTANCE	PERMISSIBLE DOSE/ROUTE
Inhaled Salbutamol	1600 mcg in 24 hours, never give whole dose as single dose, Urinary level to be less than 1000 ng/mL
Inhaled Formoterol	54 mcg over 24 hours, never to give entire dose as single dose, urinary level to be less than 40 ng/mL
Inhaled Salmeterol	200 mcg in 24 hours
Epinephrine	Not prohibited for local administration
Pseudoephedrine	Urine concentration less than 150 mcg/mL

Conclusion:-

A physician must be aware of substances which are not to be used in athletes or which can be used with proper reasoning and in permitted doses. Always history of type of sport athlete plays and whether he/she is in or out of competition is important before prescribing a prohibited substance. Let us all contribute to "FAIR PLAY".

GOOD NEBULIZATION PRACTICE

***Dr. Rajeev Bansal**

Inhalation therapy in COPD

- **Inhalation therapy is a cornerstone of treatment for COPD, offering targeted drug delivery, thus producing higher local concentrations, less systemic exposure, and potential for improved efficacy .**
- A variety of inhaler devices is available for bronchodilator treatment in patients with COPD, including metered-dose inhalers (MDIs), dry powder inhalers (DPIs), and nebulizers .
- All 3 devices are equally effective when used correctly; however, technique-related errors are common, particularly with MDIs and DPIs. Such factors should be taken into consideration when selecting an appropriate device for the individual patient .
  - The main cause of poor inhalation technique seen with MDIs is that patients find it difficult to coordinate MDI actuation with inspiration.
  - One of the most important factors in the correct use of DPIs is the generation of a forceful and deep inhalation, because most DPIs require a minimum inspiratory capacity to generate adequate drug delivery .
    - This is especially common in elderly patients (peak inspiratory flow rate declines with age) and in those with increasing disease severity who have substantial airflow limitation and/or cognitive impairment.
- **Characteristics Of An Ideal Delivery Device**
  1. Easy to use
  2. Robust
  3. Linked by patients
  4. Range of therapies
  5. Accurate and correct dose delivery.

1. Problems Associated With Hand Held Inhalers

1. Require hand-breath coordination.
2. More training and/or counseling required.
3. More technique related errors .
4. Acceptance of patients….myths
5. In patients with low inspiratory flow.

2. Nebulization helps to overcome these problems associated with hand-held inhalers.

* *Senior Consultant, Goel Hospital, Krishna Nagar, Delhi*

NEBULIZATION

A process by which liquid medication is converted into a fine mist that can be inhaled by the patient.

1. Advatages Of Nebulization

1. High dose medication can be administered at once in a way that 1 dose of nebulizer=8-10 puffs of MDI with spacer.
2. Can be given in patients with low inspiratory flow.
3. A wide range of drugs can be given via nebulizers ex. Bronchodilators, antibiotics, corticosteroids, mucolytics .
4. No additional training is required.
5. No hand breath co-ordination is required.
6. Patient's efforts are not required.

2. Patient's Views On Nebulization

1. Approx. 56-91% reported improved symptom control, well-being and self-confidence with nebulisation.
2. Approx. 70% people felt that with nebulization emergency visits culd be avoided,55% patients could avoid hospitalization and 59% people could avoid unscheduled doctor visits.

3. Current Problems With Nebulizers

1. No proper use.
2. Used where patients can be managed with inhalers

   'Nebulizer epidemic!'
3. Higher chances of side-effects
4. Regular cleaning .... maintenance?
5. In a study where home visits were made to 50 patients aged 1 to 88 years and who were using jet nebulizers at home for diseases such as asthma, chronic obstructive bronchitis and cystic fibrosis and bacterial samples were obtained from facemasks or mouthpiece. Nearly 23% were infected with Staphylococcus albus, nearly 11%were infected with Diphtherioids.
6. In children,Lung deposition increased with tight fitted mask but, there was facial drug deposition, higher chances of side effects .

4. Nebulizers should not be used between patients without disinfection. Nebulizers should be changed or sterilized at conclusion of the dose administration or at least every 24 hours.

5. *Good Nebulization Practice (GNP) is the judicious and correct use of nebulization by both healthcare professionals and patients, thereby enhancing the efficacy and safety leading to optimum treatment outcomes."*

- **Indications For Correct Nebulizer Use**
  1. Patient too sick or incapable of managing hand-held inhalers
  2. Need for large drug dose
  3. Drug not available in hand-held inhalers
  4. Practical convenience
  5. Patient preference
- **GNP Includes:**
  1. Indications for correct nebulizer use
  2. Key aspects to remember before and during nebulization
  3. Using nebulizer correctly
  4. Situations where face mask or mouthpiece should be used
  5. Continuous vs. intermittent nebulisation.
  6. Drug compatibility.
  7. Cleaning and maintenance of nebulizer.
- **Mouthpiece Should Be Preferred If Delivering-**
  1. Nebulized steroids and antibiotics to avoid administration on facial skin and eyes.
  2. Anticholinergics in patients with glaucoma.
- **Face mask should be used in**
  1. Acutely ill patients.
  2. Babies and young adults who find it difficult to coordinate with the mouthpiece.

INTERPRETATION OF LIVER FUNCTION TEST (LFT)

***Dr Naresh Agarwal**

In this article, we intend to understand how to analyze liver function test (LFT) in our routine clinical practice. Interpretation must be made after taking into consideration the patient's age, sex, risk factors, clinical signs and symptoms. Although assessment of liver function test rarely provides a specific diagnosis but it helps in categorizing the patient into different etiologies of liver diseases. It forms the integral part of many scores used commonly worldwide to classify patients into different risk strata particularly those who may require liver transplantation.

LFT is very sensitive, noninvasive and easy method of knowing the gross changes in liver synthetic function. These values lack sensitivity and specificity. It should always be correlated with history and physical examination of the patient.

Liver function test is usually classified in 3 groups:

1. Synthetic functions - Includes albumin and prothrombin time (INR). Few Clinicians also include bilirubin as part of liver synthetic function.
2. Indicators of liver cell injury - Aspartate aminotransferase (AST) and Alanine aminotransferase (ALT)
3. Markers of cholestasis - Includes billirubin, alkaline phosphatase and gamma glutamyl transferase (GGT)

Albumin

It is a non specific Marker for liver disease with a half life of around 12 to 14 days. Therefore it is not a reliable marker of acute liver disease. A low albumin suggests chronic liver disease. Sometimes albumin is quite helpful marker when other parameters are not grossly deranged.

Level of serum albumin indicates —

1. Volume status of the patient
2. Vascular integrity
3. Third space loss
4. Nutritional status of the patient

Hypoalbuminemia happens because of commonly known reasons as follows –

1. Decreased synthesis — Chronic liver disease, massive hepatocecllular necrosis
2. Protein malnutrition
3. Chronic inflammatory conditions.

* *MD, DM (Gastroenterology), Senior Consultant Gastroenterologist, Hepatologist and Therapeutic Endoscopist, Rajdhani Gastro and Liver Clinic*
Yashoda Superspeciality Hospital / Max Superspeciality Hospital

4. Increase loss (protein losing enteropathy)
5. Malabsorption
6. Increased intravascular volume— leading to dilutional hypoalbuminemia (eg: massive ascites, overhydration, cirrhosis and renal failure)
7. Increased turnover like catabolic states

Globulins

These are produced by stimulated B lymphocytes. They are increased in most of the common liver diseases particularly in chronic liver disease, hepatic malignancies and chronic inflammatory conditions.

Prothrombin time:

Prothrombin time is a coagulation factor (factor II) produced by liver. Deficiency of prothrombin leads to marked impairment in intrinsic pathway of coagulation. Formation of prothrombin is dependent on factor 1, 5, 7 and 9. Its half life is very short (around 6 hours). Prothrombin time has a prognostic value in liver diseases. Prolonged PT is associated with poor prognostic in both acute and chronic liver diseases. It forms the integral parts of many scoring systems used worldwide like Child Pugh score and MELD score. List below indicates the main causes of prolonged PT—

1. Vitamin K deficiency as can happen in malabsorption
2. Prolonged use of antibiotics
3. Nutritional deficiencies
4. Massive transfusions particularly in congenital diseases
5. Use of warfarin and
6. Disseminated intravascular coagulation

In cases of obstructive jaundice, vitamin K absorption gets impaired. Therefore prothrombin time increases. Replacing vit K parenterally rectifies prothrombin and INR is restored to a large extent. In contrast, in hepatocellular diseases (like CLD, ALF) parenteral use of Vitamin K does not rectify the prolongation of PT because in such cases synthetic function of liver cells is impaired.

Aminotransferases

Aminotransferases (alanine and aspartate Aminotransferases) are one of the most frequently use markers of liver disease and they indicate hepatocellular necrosis. Initial values of these markers correlate with initial severity of liver disease but they do not have prognostic significance. Their improvement indicates either resolving disease or is associated with poor prognosis because liver cells are depleted in severe necrotic stages. This is true for few advanced liver diseases

Table indicates the profile of AST and ALT. When analyzing AST and ALT, a number of factors should be taken into consideration, like level of rise, rate of acceleration and distinction

between AST and ALT, and lastly rate of decrease when resolving. AST and ALT more than 30-50 times of upper limit show acute or subacute inflammation, upto 5 times of normal limit shows chronic liver injury and massive increase (upto 200-300 times of upper limit) indicates either ischemic liver injury or drug related liver injury.

The ratio of AST to ALT is also important and has diagnostic value. When it is more than 1, it suggests alcoholic liver disease, cirrhosis and Wilson's disease. The ratio of less than 2 happens in majority of liver diseases. The ration of more than 4 suggests fulminant Wilson's disease. Rapid rise in AST and ALT happens most commonly patients with ischemic liver disease which is also associated with a very high level of enzymes. The levels may be as high as 10000 or more. It may be because of drug related liver injury too. This table flight number 23 conditions causing predominant ALT or AST

Table 1 shows common medicines leading to abnormal AST and ALT

• Acetaminophen overdose	• Antituberculosis drugs
• Statins	• Herbal remedies,
• NSAIDs	• Alternative medications
• Antibiotics	• Substance abuse
• Antiepileptics	

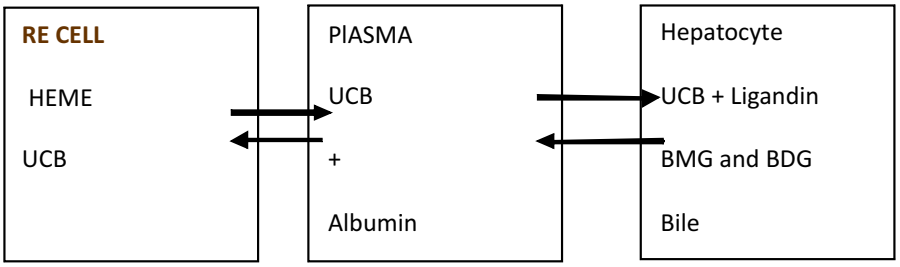
Table: Common drugs causing high aminotransferases

Lactate dehydrogenase

This (LDH) is a very non specific marker of LFT and apart from liver is also synthesized by myocardium. It is elevated in myocardial infarction, ischemic stroke and acute or acute-on-chronic liver disease. Its use is limited in clinical practice except in cases of ischemic hepatitis where it increases along with AST and ALT.

Bilirubin

Most commonly used component of LFT is bilirubin. Most of the bilirubin is formed by destruction of cells by reticulo-endothelial system, which leads to formation of unconjugated bilirubin. It gets conjugated by liver and excreted as stercobilinogen and urobilinogen. Figure 1 suggests the cycle of bilirubin metabolism.



Bilirubin is of extreme importance in assessment of liver disease. Direct bilirubin reacts directly with the reagent and indirect bilirubin requires addition of alcohol for colour development during measurement in the lab.

Unconjugated bilirubin is indirect form and the conjugated bilirubin is mono or di-glucuronide form of bilirubin.

Tablet slide 44 indicates the approach in a case of predominant indirect hyperbilirubinemia. Predominant indirect hyperbilirubinemia is said to be there when indirect fraction of bilirubin is more than 85%. It can happen in cases of hemolysis, Gilbert syndrome and Criglar-Nazzar Syndrome.

In both Gilberst syndrome and CN syndrome, glucuronodation of bilirubin is impaired. In first, it is partial, therefore this syndrome is fairly compatible with life, whereas CN syndrome is not.

Gilbert syndrome is a common syndrome found in 5-7% of healthy Indian population and does not have any hazardous effect on lifespan. It is commonly confused with the presence of significant liver disease because fractionation of billirubin requires a standardized lab. Careful assessment of direct and indirect bilirubin should be done if Gilbert syndrome is suspected. It happens because of a polymorphism in CATA box of UDP gene related to bilirubin glucuronidation. In Gilbert's syndrome, bilirubin rises during fasting state, systemic illness and hemolysis. Many young subjects have marked anxiety related to jaundice of Gilberts syndrome. Treatment of Gilbert syndrome not required apart from symptomatic treatment of associated illness.

Table 2 indicates the causes of conjugated hyperbilirubinemia and it is called so when the fraction of direct bilirubin is more than 50% of total bilirubin.

• Bile duct obstruction	• Sepsis
• Hepatitis	• Total parenteral nutrition Intrahepatic cholestasis of pregnancy
• Cirrhosis	• Benign recurrent cholestasis
• Medications/Toxins	• Vanishing bile duct syndromes
• Primary biliary cirrhosis	• Dubin-Johnson syndrome
• Primary sclerosing cholangitis	• Rotor syndrome

Alkaline phosphatase

Per se, this is the marker of biliary obstruction, either intrahepatic or extrahepatic. Isolated abnormality of alkaline phosphatase is of minor importance and should be correlated with clinical features and other LFT parameters. Level of ALP does not correlate the degree of biliary obstruction Alkaline phosphatase is also formed by placenta, bone and some connective tissues. Isolated hepatic Alkaline phosphatase elevation can happen in bile duct obstruction, use of medications, infiltrative liver diseases, hepatic metastasis, primary biliary cirrhosis, primary sclerosing cholangitis, cirrhosis, vanishing bile duct syndrome and benign recurrent cholestasis (BRIC) .

Fungal infections, hepatocellular carcinoma, metastatic malignancies, amyloidosis and sarcoidosis and other drug rheumatic diseases

In malignant biliary obstruction, Alkaline phosphatase is usually more than 1000 and in benign diseases like structure of common bile duct and stones, it is between 400 to 1000. There are few medications which can lead to a abnormal Alkaline phosphatase and table 4 indicates these medicines

- Anabolic steroid
- Allopurinol
- Amoxicillin-clavuronic acid
- Captopril
- Carbamazepine
- Chlorpropamide
- Cyproheptadine
- Diltiazem
- Erythromycin
- Estrogens
- Floxuridine
- Flucloxacillin
- Fluphenazine
- Gold salts
- Imipramine
- Indinavir
- Iprindole
- Nevirapine
- Methytestosterone
- Methylenedioxymethamphetamine
- Oxaprozin
- Pizotyline
- Quinidine
- Tolbutamide
- TPN
- Trimethoprim-sulfamethoxazole

Gamma glutamyl transferase (GGT)

This enzyme belongs to a group of particular peptides. GGT is abundant in liver, kidney, pancreas and GI tract. It not found in bone and in routine clinical practice, high GGT usually indicates isolated liver disease.

A common reason for high GGT is alcohol use which induces this enzyme and can be associated with high GGT as only abnormality of LFT. It should not be confused with the presence of biliary obstruction. It can be increased upto 2 to 3 months after the last use of alcohol. GGT with Alkaline phosphatase are hallmarks of biliary obstruction. Which are deleted in slide 65 which can lead to high GGT slight 66 indicates the summary of abnormal liver functions and there in preservation

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