INSTITUTE OF PULMOCARE & RESEARCH

DG-8, Near Rabindra Tirtha, Action Area-I,
(On the way to New Town DPS School), New Town, Kolkata-700 156

WELCOME ALL FACULTY MEMBERS and DELEGATES to PULMOCON - '22

20th All India Update On Pulmonary Medicine

: CONTENT :

Pulmocon '22

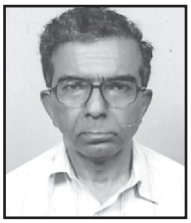
Organized by Institute of Pulmocare & Research

Highlights of the Programme

Topics		
Day 1 (24/9/22)	**Hall**	**Time**
➢ The Dr. S N De Memorial Oration	A	12.15pm – 1 pm
➢ Workshop: preparing for final PG exam (oral / practical) – with dummy examination (including long cases and short cases, OSCE & tips).	A	3 pm- 7 pm
➢ Symposium on career option for young pulmonologists	B	4.30 pm – 6.30 pm
➢ Medical education - some perspectives from home and abroad.	B	3 pm – 4.30 pm
➢ Setting up and running a pulmonary rehab program: the lessons learnt.	B	11 am – 11.30 am
Day 2 (25/9/22)		
➢ Workshop on IOS + FOT.	B	12.15 pm - 1.30 pm

➢ Multiple Case Based Discussions covering – difficult asthma, COPD, DPLD, Pleural Diseases, Sleep disorder breathing, Pulmonary Eosinophilia, Antibiotic selection in NDR infections. (Please see the program schedule for details) (We have left blank pages to note the lessons after each discussion in the souvenir)		
➢ A few topic base lectures		
➢ Talk: Igniting innovations in young mind		

FROM THE PRESIDENT'S DESK



"I am happy to welcome you at Pulmocon 2022. This annual All India update has really become a chosen and admired academic event of our fraternity from different corners of our country.

The organising committee has tried its best to make it a successful event. I would pledge you all to excuse us for shortcomings, if any and enrich us with your suggestions and positive criticisms."

Dr. Dhiman Ganguly
President
Pulmocon - '22

FROM THE SECRETARY'S DESK

"I am happy and honoured to welcome you at Pulmocon '22. This happens to be the 20th Pulmocon in a row – and it gives me pleasure to see that the popularity of the conference has been scaling high and higher over the years.

I welcome all the delegates from across the country and hope that our effort will let you carry home messages to cater better to your ailing patients.

I wish you all continue to bestow with love and affection upon us so that this institute can grow further and the future pulmocons turn better and more meaningful.

With regards and thanks."

Dr. Parthasarathi Bhattacharyya

Dr. Parthasarathi Bhattacharyya
Organising Secretary
Pulmocon - '22

PULMOCON - '22

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Dr. Dhiman Ganguly

Organizing Secretary :
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ডাঃ শম্ভুনাথ দে

জন্ম ঃ ১লা ফ্রেব্রুয়ারী, ১৯১৫ মৃত্যু ঃ ১৫ই এপ্রিল, ১৯৮৫

বাঙালি চিকিৎসক ও বৈজ্ঞানিক, যাঁর পরিচয় জাতির ইতিহাসে প্রায় বিলুপ্ত

স্যার আলেকজাণ্ডার ফ্লেমিং যেমন পেনিসিলিনের আবিষ্কর্তা, স্যার রোনাণ্ড রস্ যেমন ম্যালেরিয়ায় মশার ভূমিকার আবিষ্কারের জনক, তেমনি **ডাঃ শম্ভু নাথ দে কলেরার বিষ (toxin) এর আবিষ্কর্তা**।

সারা পৃথিবী যখন ফ্লেমিং, রস্ কে শ্রদ্ধা জানায় তখন নীরবে বৈপ্লবিক গবেষণা করা এই বাঙালী তথা ভারতীয় চিকিৎসক লোকচক্ষুর অন্তরালেই থেকে যান।

বিজ্ঞান গবেষণায় প্রেরণার অফুরন্ত উৎস হিসেবে আমরা আজ তাঁর মৃত্যুর ৩৭ তম বছরে তাঁকে নিবিড় ভাবে স্মরণ করি।

তাই ২০১৯ সালে অনুষ্ঠিত আমাদের **পালমোকন - ইনস্টিটিউট অব্ পালমোকেয়ার অ্যাণ্ড রিসার্চ** এর বাৎসরিক বক্ষরোগ সংক্রান্ত সর্বভারতীয় সম্মেলনে আমরা প্রয়াত ডাঃ দে - কে আমাদের আন্তরিক শ্রদ্ধা জানানোর চেষ্টা করেছি। তাঁর নামে আমরা ২০০৯ সাল থেকে একটি বাৎসরিক স্মারক বক্তৃতার সূত্রপাত করেছি। এ বছর ঐ বক্তৃতা দিতে আসছেন ভারত বিখ্যাত বৈজ্ঞানিক ও চিকিৎসক অধ্যাপক ডাঃ কঞ্জাক্ষ ঘোষ ।

এক আত্মবিস্মৃত জাতির বর্তমান গ্লানিময় পরিস্থিতির প্রেক্ষাপটে ডাঃ শম্ভুনাথ দে-র উজ্জ্বল জীবনালেখ্য আমাদের চলার প্রেরণা ও পাথেয় হোক। চিকিৎসা বিজ্ঞানে বাঙালীর অবদান আমাদের ও পরবর্ত্তী প্রজন্মকে হারিয়ে যাওয়া সম্মান পুনঃ উদ্ধারে ব্রতী করুক। হীনমন্যতার মলিনতা থেকে বাংলার ও এদেশের চিকিৎসা ব্যবস্থা ঘুরে দাঁড়াক এক নতুন প্রত্যয়ের এবং একনিষ্ঠ প্রচেষ্টার আলোয়।

–ঃ জীবনী ঃ–

১৯১৫ সালে শম্ভুনাথ দে'র জন্ম হুগলীর এক সাধারণ ব্যবসায়ী পরিবারে। ছোটবেলা থেকেই তিনি মেধাবী ছাত্র হিসাবে চিহ্নিত হন - এবং ম্যাট্রিকুলেশনের পর প্রথমে হুগলীর মহসীন কলেজ ও পরে কোলকাতা মেডিকেল কলেজ থেকে ১৯৩৯ সালে MB এবং ১৯৪২ সালে DTM (Diploma in Tropical Medicine) পাস করেন। ১৯৪৭ সাল অবধি কোলকাতা মেডিকেল কলেজে প্যাথোলজি বিভাগে ডেমনস্ট্রেটর হিসাবে কাজ করার পর, তিনি লণ্ডনে PhD করার উদ্দেশ্যে যান। ফিরে আসার পর তিনি কোলকাতা মেডিকেল কলেজের প্যাথোলজি বিভাগে যোগ দেন এবং মূলত কলেরা সংক্রান্ত গবেষণায় মনোনিবেশ করেন।

সে সময়ে কলেরা ছিল একটি মহামারী বিস্তার করা রোগ এবং কলেরা সম্মন্ধে মানুষের জ্ঞান ছিল সীমিত। ১৮৮২-৮৩ সালে রবাট কচ্ (Robert Koch) কলেরার জীবানু আবিষ্কার করেন কিন্তু ঠিক কিভাবে কলেরা হয় তা একটা বিরাট প্রশ্নচিহ্নের মত থেকে যায় – কারন, রবার্ট কক্ এবং তৎপরবর্তী অন্যান্য বৈজ্ঞানিকরা এই রহস্য উদ্‌ঘাটনে ব্যর্থ হন। সাধারণ ব্যাকটেরিয়ারা যে ভাবে মানুষকে আক্রান্ত করে, ঠিক সেই হিসাবে কলেরার আক্রমণ ও রোগ সৃষ্টিকে তারা বুঝতে ও বোঝাতে পারেননি। আসলে কলেরার জীবাণু (vibrio cholerae) রোগ সৃষ্টি করে সম্পূর্ন অন্য উপায়ে - জীবানু নিঃসৃত Toxin বা বিষ অন্ত্রের উপর কাজ করে দাহ্য ঘটায়। অন্ত্রের (ইলিয়াম) এর লুপ মডেলের ব্যবহার করে ডাঃ দে দেখান যে কলেরা হয় ঐ বিষ বা Toxin এর জন্য। জীবানু ও তার দেহ নিঃসৃত বিষ যে এভাবে অসুখ সৃষ্টি করতে পারে এ তথ্য প্রতিষ্ঠার জনক হন **ডাঃ শম্ভুনাথ দে** । ১৯৫৯ সালে এই আবিষ্কারের পর, ১৯৬১ সালে তিনি London বিশ্ববিদ্যালয়ের D.Sc. (শরীর বিদ্যা) হন।

পরবর্তী কালে ডাঃ দে নীলরতন সরকার মেডিকেল কলেজ ও বসু বিজ্ঞান মন্দিরেও কাজ করেন। প্রভূত প্রতিকূলতার মধ্যে তাকে কাজ করতে হয়। তাঁর কাজে সাহায্য করা দূরে থাক, পদে পদে তাঁকে হেনস্থা হতে হয়। এক সময় হতাশ হয়ে এবং একরকম বাধ্য হয়ে তিনি গবেষণার কাজ বন্ধ করে দেন। একটা ছোট প্যাথলজি ল্যাবরেটরী করে এই মহান বৈজ্ঞানিককে দিন যাপন করতে হত। **জীবদ্দশায় তাঁর ভাগ্যে কোন সম্মান জোটেনি; 'মহান' এই দেশ তাঁকে কোথাও কোন রকম স্বীকৃতি দেয় নি।**

ডাঃ দের গবেষণার ফলশ্রুতিতে আরও অনেক আবিষ্কার যেমন দাস্থ তে ORS চিকিৎসা, কলেরা-টীকা সমস্তই সম্ভব হয়েছে তার আবিষ্কারের সৌজন্যে। **একাধিক বার তিনি বিদেশ থেকে নোবেল পুরষ্কারের জন্য মনোনীত হন, ১৯৭৮ সালে - তিনি নোবেল কমিটিতে বক্তব্য রাখেন।**

মহান এই বৈজ্ঞানিক **১৯৮৫ সালে লোকান্তরিত হন**। যদিও জীবদ্দশায় তিনি তার নিজের দেশে কোন সম্মান ও স্বীকৃতি পাননি, আমেরিকার ও দেশ বিদেশের গবেষকরা কিন্তু ডাঃ শম্ভুনাথ দে কে দুহাতে সম্মান সূচক অর্ঘ সাজিয়ে দিয়েছেন। তার নামে লেখা অনেক প্রবন্ধ, ছাপা হয়েছে বিদেশী বৈজ্ঞানিক পত্র-পত্রিকায়। যদিও তার মৃত্যুর দশ বছর পর **১৯৯৪ সালে কোলকাতা বিশ্ববিদ্যালয় তাঁকে সম্মানীয় D.Sc. উপাধি দেন।**

ডাঃ দের মৃত্যুর ৩৭ বছর পরে তাঁকে সম্মান জানাতে পেরে আমরা নিজেদের কৃতার্থ বলে মনে করছি। আমাদের এই ছোট্ট প্রতিষ্ঠান এই সম্মান প্রদর্শনের মধ্য দিয়ে গভীর ভাবে প্রেরণা অনুভব করছে।

হয়ত বহুশ্রুত নাটকের এই উক্তিই সত্যি - ''সত্য সেলুকাস্, কি বিচিত্র এই দেশ !''। জাত-পাত, ধর্ম-গোষ্ঠী, রাজনীতির সদা সংকীর্নতা, মানসিক ও আত্মিক দীনতার, আত্মবিশ্বাস হীনতার অন্ধকারে এমনি হারিয়ে যান কত জ্যোতিষ্ক। মৃত্যুর ৩৭ বছর পরও আমরা তাকে কোন মরনোত্তর সম্মান দিতে পারিনি – রাজপথ দূরে থাক, একটা কানা গলি, কি একটা শিক্ষা প্রতিষ্ঠানে কোথাও তাঁর নাম নেই।

আমরা আশা করবো ডাঃ শম্ভুনাথ দে কে আমাদের সমাজ ও সরকার উপযুক্ত মূল্যায়ন করবেন এবং তার মাধ্যমে বাংলা তথা ভারতবর্ষের চিকিৎসা জগৎ - এ গভীর ও সুস্থায়ী পরিবর্তনের সূচনা হবে।

Dr. Sambhu Nath De

Born : 1st February, 1915 **Passed away : 15th April, 1985**

A great doctor and scientist from Bengal – who is forgotten in our national history.

Dr. Sambhu Nath De can be placed in the same row with Dr. Alexzander Flamings (the inventor of penicillin) and Sir Ronald Ross (the inventor of the role of mosquito in malaria). Dr. De discovered that toxins secreted from the cholera germ causes loose motion and this fact changed the face of treatment of diarrhoeal diseases. But Dr. De remains in oblivion when the world pays tribute to the other two and many more.

We wished to bring his name to light in his birth Centenary and after 37 years of his passing away. So, we initiated a memorial oration in his name in our annual update from 2009 onward. This year, Prof. Dr. Randeep Guleria will deliver the oration.

Let the life and work of Dr. Sambhu Nath De inspire us and make us stride to bring glory and confidence to our efforts in healthcare and research.

ABOUT DR. SAMBHUNATH DE

EARLY CAREER

Sambhu Nath De was born in Hooghly District, West Bengal, India. His father Mr Dasarathi De was a small businessman. Supported by his uncle Asutosh De, De completed the Matriculation examination with distinction that helped him to get the District scholarship as well as to pursue further education in Hooghly Mohsin College. De passed his M.B. examination in 1939 from Calcutta Medical College and completed a Diploma in Tropical Medicine (DTM) in 1942. Soon after graduation he joined Calcutta Medical College as a Demonstrator of Pathology. In 1947, De joined as a Ph.D. student under Sir Roy Cameron at the Department of Morbid Anatomy, University College Hospital Medical School, London, and obtained his Ph.D. degree in Pathology in 1949. After his return, De worked on pathogenesis of cholera and started publishing his findings. In 1955, De became the Head of Pathology and Bacteriology Division of the Calcutta Medical College, which he continued until his retirement.

CONTRIBUTIONS

De made significant contributions to our recent understanding of cholera and related diarrheal diseases. Followed by the discovery of Vibrio cholerae in 1884 by Robert Koch, many works have been carried out all over the world to answer many questions related with its pathogenesis and mode of transmission in causing outbreaks. Three of his works viz., ligated intestinal loop method for studying cholera in rabbit model; ileal loop model to demonstrate the association of some strains of E. coli with diarrhea and lastly but most importantly is his discovery of cholera toxin in 1959 in the cell-free culture filtrate of V. cholerae that stimulated a specific cellular response.

In 1959 De was the first to demonstrate that cholera bacteria secrete enterotoxin. This discovery eventually promoted research to find a treatment aimed directly at neutralizing the cholera enterotoxin. De's paper "Enterotoxicity of bacteria-free culture-filtrate of Vibrio cholerae," while initially unrecognized, today is considered a milestone in the history of cholera research. Biochemist W.E. van Heyningen, professor emeritus, University of Oxford, UK, and John R. Seal, former scientific director, National Institute of Allergy and Infectious Diseases, Bethesda, note that De's paper "deserves to go down as a classic in the history of cholera, and, indeed, as later developments have shown, in the history of cellular physiology and biochemistry."

"An experimental study of the mechanism of action of Vibrio cholerae on the intestinal mucous membrane" is De's most-cited paper, cited 340 times until August 1986. De's most-cited paper has been core to cholera research fronts for many years, especially research fronts on "E. coli and Vibrio cholerae enterotoxin: detection, characterization, and role of adherence" and "Characterization of cholera enterotoxin and other enterotoxins". As noted by John Craig, State University of New York Health Science Center at Brooklyn, De's work was truly creative and novel, and it "forever altered our concepts surrounding the pathogenesis of secretory diarrhoea."

These famous findings came out from the work he carried out at the Nilratan Sircar Medical College, Calcutta Medical College and Bose Institute, Kolkata in extremely modest laboratory settings. Using research methodology that was very simple, easy to perform and inexpensive, he set the highest standards of excellence in novel experimental design and execution.

The oral rehydration therapy (ORT) for replenishing the massive fluid loss in cholera patients, has saved innumerable lives, should be considered as a direct outcome of De's discovery of cholera toxin. His findings on exotoxins set the stage for the modern views of diseases caused by toxin producing bacteria, helped in the purification of cholera and heat-labile (LT) enterotoxins produced by V. cholerae and E. coli, respectively, and in the development of series of cholera and enterotoxigenic E. coli (in short ETEC strains) vaccines.

POST-RETIREMENT

De retired in 1973 from the Calcutta Medical College at the age of 58. After his retirement, he showed no interest in higher positions but continued his research at the Bose Institute, Calcutta. De's desire to purify the cholera toxin did not progress any further as the protein purification technology was not well established in his research settings. In 1978, the Nobel Foundation invited De to participate in the 43rd Nobel Symposium on Cholera and Related Diarrhoeas.

De died on April 15, 1985 at the age of 70. His life's ambition was to make the world a better place to live in through his dedicated selfless services in medical science.

Nobel laureate Prof. Joshua Lederberg had nominated De for the Nobel Prize more than once. Said Lederberg, **"our appreciation of De must then extend beyond the humanitarian consequences of his discovery. . . he is also an examplar and inspiration for a boldness of challenge to the established wisdom, a style of thought that should be more aggressively taught by example as well as precept."**

And yet De was never elected a fellow of any Indian academy and never received any major award. Indeed as Professor Padmanabhan Balaram pointed out in an editorial in Current Science, "De died in 1985 unhonoured and unsung in India's scientific circles. That De received no major award in India during his lifetime and our Academies did not see it fit to elect him to their Fellowships must rank as one of the most glaring omissions of our time. De's heroic story of persistence, dedication and achievement should serve as an inspiration to the many who are increasingly bewildered by the current fashion of mega projects, surrounded by fanfare and publicity and most often surprisingly little discernible scientific output."

Retrieved from "http://en.wikipedia.org/wiki/Sambhu_Nath_De"

A TRIBUTE TO THE MEMORY OF DR. SAMBHUNATH DE

Prof. A. K. Nandy

Dr. Sambhunath De worked and died in this city of Calcutta (now Kolkata), and it was in this city that he discovered the Cholera Enterotoxin - fifty years back in 1959, which as Prof. Van Heyningan of the University of Oxford, noted "deserves to go down as a classic in the history of cholera and, indeed as later developments have shown, in the history of cellular physiology and biochemistry".

This great scientist, though recognised internationally, and by the Nobel Commitee itself for his great work, remained almost unknown and neglected in his own country, in his own State, and even the city he worked in. No wonder then, when Professor P. Balaram in an Editorial in the 'Current Science' journal, wrote "De died in 1985 unhonoured and unsung in India's scientific circles", ... must rank as one of the most glaring omissions of our time. De emerges in retrospect as a moment self-effacing scientist driven by inner compulsions to grapple with a major scientific problem of the time. "De's heroic story of persistence, dedication and achievements should serve as an inspiration to many who are increasingly bewildered by the current fashion of megaprojects - surrounded by fanfare and publicity and most often, surprisingly little discernible scientific output".

Research work of Dr. S. N. De

His work on cholera started when he was the Professor of Pathology in N.R.S. Medical College. His paper published during this period in 1953 has been his most cited paper, cited 340 times until August 1986 and has been designated as a 'Citation Classic' in 1987, indeed a very rare recognition. He continued his work in Calcutta Medical College where he joined as the Director-Professor of Pathology in 1956 and also as a 'Honorary Worker' in the Bose Institute. His work started getting keen attention from the international scientific community, as was exemplified from the words of Dr. John Craig of the State University of New York : "De's work was truly creative and novel, and forever altered our concepts surrounding the pathogenesis of secretory diarrhoea".

By 1960, De established the existence of "CHOLERA EXOTOXIN". In his own words "Vibrio Cholerae has now been promoted to the rank of few exotoxin-producing bacteria. Diphtheria exotoxin was discovered within 4 years of discovery of the bacillus, Tetanus exotoxin within 6 years. It has taken 75 years for cholera exotoxin to be discovered in 1959, after organism was discovered by Robert Koch in 1884" (B.C.Roy Oration).

De wanted to continue with his research for further purification of the toxin ultimately develop that he discovered, and ultimately develop a vaccine against cholera. But, unfortunately various constrains and lack of support for necessary facilities forced him to limit himself. He stated - "by 1963-64, I was forced to discontinue my work and lost all interest in cholera" (B.C.Roy Oration).

But his unfinished work was picked up by scientists abroad in few years' time, when they noted its implications. Extensive work was started on the foundation laid by De; its wider applications in a variety of investigations were also worked out in the proceding years. Applause started reaching him from all corners of the world except his own country. Noble Laureate Professor Joshua Lederberg noted, " ... his findings on exotoxins set the stage for modern views of diseases caused by the toxin-producing bacteria, ... helped in purification of the cholera and E.Coli enterotoxins, ... and in the development of cholera and E.Coli vaccines". He had nominated De for the Nobel Prize more than once.

The Nobel Foundation invited De in 1978 to participate in the 43rd Noble Symposium on "Cholera and Related Diarrhoes". He was highly applauded there by all present there (see letter). Noble Prize seened to be a possibility. But Lady Luck disappointed hid this time also.

He died a brooken-hearted man on April 15, 1985 from Hepatitis-B infection followed by hepatic coma.

The Indian scientific community woke up from its 'Kumbha-Karna Sleep' at long last "Current Science", the pioneer Indian scientific journal brought out a "Special issue on S.N.De and cholera Enterotoxin" in 1990 with contributors from all over the world. This was for the first time the prestigeous journal brought out a special issue on a particular scientist.

PULMOCON '22

24th and 25th September 2022

Venue : CII-Suresh Neotia Centre, City Centre, Sector-I, Salt Lake, Kolkata-64

Organised by : **Institute of Pulmocare & Research,** Kolkata.

Acharya Prafulla Chandra Roy Memorial Award, 2019

Bengal has given birth to many great people – Acharya Prafulla Chandra Roy had been one amongst them.

Acharya P C Roy dreamt of self reliance and promotion of self sufficiency in all fields through gaining excellence. The great scientist of his days was a great entrepreneur too. He established Bengal Chemical, the 1st Indian entrepreneurship in Chemical and Pharmaceutical industry over 103 years ago. That was, infact, the beginning of pharmaceuticals industry in India.

The beauty of his personality was in simple living but in extraordinarily thinking, in noble ambitions and the ability to withstand odds. They were admixed with a generosity and extreme love and affection for his students and the countrymen. A story goes as that once he was very angry with a student who spent one penny extra for his professors tiffin but on the same day of the incidence, the apparently miger professor donated ` 50,000/- to the National Congress Party for draught-relief in North India.

To commemorate him and to instill the sprit of innovations amongst ourselves we have incorporated an award in our annual Pulmocon from the year, 2009. The 1st Acharya P C Roy memorial award was offered to Dr. Sujay Guha and the next was confered to famous scientist and innovator Dr. Tinku Acharyya who innovated the digital photography technology with over 150 US patents to his credit. In 2011 we had felicitated Prof. Indu Bhusan Chatterjee for his distinguished contribution in research and innovations in 2012, similarly, in subsequent years doyens in research and innovations have been conferred with this award. In Pulmocon 2022 we are happy to award Prof. Sivaji Chakravorti.

THE ORATOR OF PROF. S. N. DE MEMORIAL ORATION

Dr. Randeep Guleria

Dr. Randeep Guleria is Professor and Head, Department of Pulmonary Medicine and Sleep Disorders at All India Institute of Medical Sciences, New Delhi, where he has been working since the last 23 years. Dr. Guleria is the first Indian to get a Doctorate of Medicine (DM) in Pulmonary and Critical Care Medicine. He joined AIIMS in 1992 as Assistant Professor in the Department of Medicine and in the subsequent years he has been promoted to the post of Associate Professor, Additional Professor and Professor. He has done pioneering work in respiratory medicine. The Department of Pulmonary Medicine & Sleep Disorders has been created under his dynamic leadership in 2011 and has been ranked as the best department in Pulmonary Medicine in 2014, 2015 and 2016 by the NIELSEN survey published in WEEK. He was instrumental in starting DM course in the discipline of Pulmonary, Critical Care & Sleep Medicine was started at AIIMS in 2011.

Dr. Guleria was conferred with the prestigious "Padma Shri" Award in 2015 by the President of India and recently with "Dr. B.C. Roy Award in the category Eminent Medical Person" for the year 2014. In addition to these, he has several awards and honours to his credit, notably, Himachal Gaurav Award in 2011, Zee TV and LIC "Swasth Bharat Award" in 2010, "Lung India Award" for the best original article in Lung India by the Indian Chest Society in 2014, Dr. Reddy's Chest Oration by the National College of Chest Physicians (India) 2013, Association of Physicians EMerck award for research in Chest Diseases in 2008, Raj Nanda and Royal College of Physicians Joint fellowship in Pulmonary Diseases in 2007-08, Dr. Mahinder Narain Oration in 2005, Indian Chest Society, Dr CV Ramakrishnan Oration award for his outstanding contribution in Respiratory Diseases in 2011 by the Academy of Respiratory Medicine, Dr P S Shankar Oration in 2011, Humanity (India) Oration at 6th UKAPICON 2015 by the Association of Physicians of India and the CRS Oration award 2015 at TAPPCON 2015 by the Coimbatore Respiratory Society.

He has been invited as an expert in several National and International forums including the Center for Disease control (CDC), USA to their influenza division study tour, Advisor to WHO, Scientific Advisory Group of Experts (SAGE), Geneva, on "Influenza Vaccination and Immunization" from 2010 to 2013. He has been a consultant to International Atomic

Energy Agency (IAEA), Vienna, on Radiation Protection in Medicine since 2007. He has been invited by the European Commission and the IAEA to their International Workshop in Brussels in 2009. He has been invited by the Government of Germany, WHO and IAEA to attend their International Conference on Radiation Protection in Medicine: "Setting the stage for the next decade" in 2012. He has also worked at the prestigious Royal Brompton Hospital, London as a Clinical Research Fellow.

He has been Personal Physician to the Prime Minister of India Shri Atal Bihari Vajpayee from 1998 to 2004 and has since continued to be his Personal Physician. He has been asked by the Government of India to see various national and international dignitaries including Mr. Koirala, the then Prime Minister of Nepal.

He has done pioneering work on respiratory muscle functions, lung cancer, asthma and COPD and has more than 400 publications in International and National journals and 49 chapters in various National and International books. He has delivered more than 300 lectures in International and National conferences. He has been invited by the American College of Chest Physicians as a faculty to their Annual International Conference in Chicago 2014. He has been invited by the National Associations in Sri Lanka and Nepal to deliver talks in their conferences.

He is a member of many key committees in the Government of India including the Joint Monitoring Committee to monitor outbreaks of new diseases including Pandemics, bird flu, Ebola etc. to formulate National policy to control antibiotic resistance in India, member of Program Advisory Committee on Health Science of the Department of Science and Technology, member Project Review Committee of the Indian Council of Medical Research, member of the project advisory committee of the National Institute of Virology and a member of the Project Advisory Committee on Nutritional Profile of Assam, Manipur and Meghalaya of the ICMR. He has been an advisor to several organizations like UPSC and a member of the Selection Committee for the International Commonwealth fellowship. He is a fellow and member of several National and International bodies including National Academy of Medical Sciences, International Medical Science Academy, Indian Association of Sarcoidosis and other Granulomatous Disorders, Association of Physicians of India, National College of Chest Physicians of India, Indian Chest Society and AIDS Society of India. Dr. Guleria is on the editorial board of a number of journals including the Indian Journal of Chest Disease, Lung India, Journal of American Medical Association (India), Chest (India), etc.

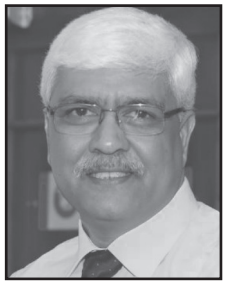
Dr. Guleria has been an external examiner to various universities including Delhi University, PGI, Chandigarh, VP Chest Institute, KGMC, Lucknow, BHU Varanasi, Amrita Institute of Medical Sciences, Sher-e Kashmir Institute, Srinagar and Universities in Nepal.

He has been organizing secretary for Medicine CME program for the Department of Medicine, A.I.I.M.S. in 1995 and 1996. He has convened the International and National conference on HIV, AIDS and

Tuberculosis: Past Present and Future in 2006. He has been the organizing chairman of PULMOCRIT AIIMS & American College of Chest Physicians workshops and updates in 2012, 2014, 2015 and 2016. He has been chairman to various committees at AIIMS including drug purchase committee and medical boards.

He has given a large number of talks and participated in programs for AIR, Doordarshan, Lok Sabha TV, Zee TV, NDTV, Star News, India TV and Aaj Tak. He has also participated in interactive TV shows for youth regarding problems of smoking and how to quit smoking. He has been actively participating in various environmental causes and has been an expert at various environmental forums concerning air pollution. He regularly participates in environmental awareness programs and delivers talks on effect of air pollution to school and college students as part of awareness campaign and green school initiative on environment.

Dr. Guleria has been a dedicated teacher for undergraduate, postgraduate and DM fellows for the last 30 years. He has been actively involved in taking classes for undergraduate students in the area of respiratory medicine. He has supervised more than 30 students in MD (Medicine) and 06 students in DM in Pulmonary, Critical Care & Sleep Medicine. Inherence teaching for students he has organized PG meetings for MD and DNB students which is included bedside case discussions and topic discussion on weekend for students to upgrade their knowledge and skills in case presentation.



Nomination for IPCR Award

Prof. Sivaji Chakravorti

FNAE, FNASc, FAST, FIE, FIETE, AvHumboldt Fellow
Professor of Electrical Engineering,
Jadavpur University, Kolkata 700032
& Former Director, National Institute of Technology Calicut
Ph: 98300 92189 (Mob)
Email: s_chakrav@yahoo.com / schakravorti.electrical@jadavpuruniversity.in
Date of Birth: 18 Sept 1962

1) Contributions as Teacher :

The nominee is a passionate teacher since 1985, when he joined Jadavpur University as a Lecturer in Electrical Engineering.

As a teacher, he has steadfastly emphasized on the laboratory practices of highest standard. Since 2004, he was the Prof-in-Charge of the High Voltage laboratory of Jadavpur University, which is one of its kind in India. To maintain the highest standard, he converted this HV lab into an ISO 9000 certified laboratory and subsequently into a NABL accredited laboratory, which is a notable achievement among the laboratories of academic institutions.

For proper dissemination of his vast knowledge in the teaching domain, he has published several books and developed online courses as detailed below:

A. Book Published/Edited:

I) Book titled "Electric Field Analysis" by S.Chakravorti published by CRC Press, Taylor & Francis, USA, 2015.

ii) Book titled "Recent Trends in the Condition Monitoring of Transformers" by S.Chakravorti, D.Dey and B.Chatterjee published by Springer-Verlag, London, 2013.

iii) Book titled "Electrical Machines" by P.K.Mukherjee & S.Chakravorti published by M/s Dhanpat Rai & Sons, New Delhi, 1993.

iv) Proceedings of "EPIC-IEEE ACE 2002" Edited by N.Chatterjee, D.K.Basu, S.K.Choudhury & S.Chakravorti published by IEEE Calcutta Section, 2002.

v) Proceedings of "Northeastern Himalayan Biodiversity" Edited by S.Chakravorti published by Humboldt Club Calcutta, 2003.

vi) Proceedings of "CalCon 2011" Edited by B.Chatterjee & S.Chakravorti published by IEEE Calcutta Section, 2011.

B. Courses Developed:

Following on-line courses have been web hosted by DLNET-NSDL that is sponsored by US- National Science Foundation.

a) "Electric Field Calculation by Charge Simulation Method" by S.Chakravorti.

b) "Electric Field Calculation by Finite Difference Method" by S.Chakravorti.

c) "Electric Field Computation by Indirect Boundary Element Method" by A.Lahiri and S.Chakravorti.

The nominee believes in closer interaction between academia and industry for value addition in teaching. He has worked in the research and development group 'HGIL' of Power Transmission and Distribution wing of Siemens AG in Berlin in 1998. He has successfully incorporated his developmental works on optimization of Gas Insulated Lines in UG and PG teaching. His collaborative works with NTPC Ltd has resulted in the introduction of a course on 'Condition Monitoring of High Voltage Equipment' at the UG level in Jadavpur University.

2) Contributions as Researcher:

The nominee has impeccable research credentials. He has more than 36 years of teaching and research experience in India and abroad, and has been associated with German as well as North American Universities, Institutes and Industries as an active collaborator.

His research activities have resulted in more than 114 international journal papers including 71 papers in IEEE Transactions, one US patent, three Indian patents and two software copyrights.

He is the recipient of several awards from prestigious academies/institutions of India and other countries as detailed below:

1. Best Researcher Award by IEEE Kerala Section in 2019.
2. Technical University-Munich Ambassador Award in 2013.
3. The "Technology Day Award" from All India Council for Technical Education (AICTE) for the best project in the Research & Development Category in 2004.
4. The Pandit Madan Mohan Malaviya Memorial Medal of the Institution of Engineers (India) in 1995-96.
5. The Union Ministry of Energy: Department of Power Prize of the Institution of Engineers (India) in 1994-95.
6. Certificate of Merit of the Institution of Engineers (India) in 1996-97.
7. Certificate of Merit of the Institution of Engineers (India) in 1992-93.
8. Certificate of Merit of the Institution of Engineers (India) in 1991-92.
9. Certificate of Merit of the Institution of Engineers (India) in 1989-90.

The nominee has successfully guided 22 PhD Theses. The nominee has carried out 15 sponsored research projects, out of which 10 are as Principal Investigator.

In recognition of the high quality of his research works, the nominee received the following research fellowships:

I) Alexander von Humboldt Foundation Fellowship in the Institute of High Voltage Engineering of Technical University, Munich, Germany, during 1995-96, 1999 and 2007 and at the ABB Corporate Research, Ladenburg, Germany in 2002.

ii) Indian National Science Academy Visiting Fellowship at Indian Institute of Science, Bangalore, India in 1994.

iii US National Science Foundation (US-NSF) Visiting Scientist at the Advanced Research Institute of Virginia Tech, USA in 2003.

Two most innovative research contributions of the nominee are: i) accurate estimation of moisture in solid insulation, which is the most significant factor causing reduction of life of expensive high voltage equipment in practice and ii) accurate identification and localization of faults in winding insulation of power transformers.

The nominee has made outstanding contributions to realistic estimation of moisture content in cellulosic insulation of high voltage transformers. The nominee practically implemented his ideas through the development of a completely new integrated condition monitoring instrument, which is now extensively used by one of the large power utilities in India for efficient asset management.

The nominee has worked with great distinction in the area of diagnosis of faults in transformer insulation under impulse excitation incorporating innovative methods that are capable of accurate and unambiguous localization of faults, which is of immense help to power utilities that are seeking intelligent and automated fault indicators.

Through his innovative research works, the nominee has brought in ideas having far-reaching impact on future research.

3) Contributions as Institution Builder :

The nominee has held number of positions as Institutional Head as detailed below:

I) Director of National Institute of Technology Calicut, Kerala, since 19 Aug 2015.

ii) Chairman (I/c) of the Board of Governors of National Institute of Technology Calicut, Kerala, since Nov 2017.

iii) Director (Addl Charge) of National Institute of Technology Puducherry, Karaikal, from June 2016 to April 2017.

iv) Mentor Director of Indian Institute of Information Technology (IIIT) Kottayam, Kerala, from Aug 2015 to Dec 2016.

During his tenure as the Director of NIT Calicut, the rank of NIT Calicut in Engineering in NIRF has improved significantly from 50 to 28, the rank in architecture became 3rd in NIRF 2019, the number of sponsored research projects applied from NIT Calicut has increased by more than 80% and the number of PhD produced in a year increased from 36 to 102. During his tenure of less than one year as the Director (Addl Charge) of NIT Puducherry, he has successfully shifted the entire operation of NIT Puducherry to its own campus, the first of the new NITs to become fully functional in its own campus. During his tenure as Mentor Director of IIIT Kottayam for about one and half years, he completed the approval process and initiated the construction processes of the new campus. As a result of which IIIT Kottayam has shifted to its new campus in 2019, within less than 5 years of its inception, which is a feat in itself.

4) Demonstrated Abilities as Team Leader/Organizer :

The nominee has held number of leading positions in Professional Bodies as detailed below:

I) Vice-President of Indian National Academy of Engineering – 2021-2022

ii) Chairman of IEEE India Council – 2017-2018

iii) Chairman-Elect of IEEE India Council – 2015-2016

iv) Chairman of IEEE Calcutta Section – 2011-2012

v) Vice-Chairman of IEEE India Council – 2001

vi) Member of the Governing Council of Indian National Academy of Engineering – 2017- 2020

vii) Convener of the Electrical Engineering Sectional Committee of Indian National Academy of Engineering – 2017-2018

viii) Member of the Electrical Engineering Sectional Committee of Indian National Academy of Engineering – 2011-2013

ix) Convener of Engineering & Technology Sectional Committee of West Bengal Academy of Science & Technology – 2013-2015.

In 2012, he started the Dielectrics and Electrical Insulation Society Chapter of IEEE in Kolkata as the founder Chairman, which is the first such IEEE Chapter formed in India. When he was the Chairman of IEEE Kolkata Section in 2012, he was successful in unveiling two IEEE Milestone Plaques in Kolkata by IEEE President in honour of the works done by Prof. J.C.Bose and Prof. C.V.Raman. These are the only two IEEE Milestones in India till date, which put India on the globally visible IEEE Milestone map available on IEEE website.

The nominee has also taken several global leadership positions as detailed below:

a) Member of the Global Administrative Committee of IEEE Dielectrics & Electrical Insulation Society – 2016-2017,

b) Secretary of the Global Chapters' Committee of IEEE Power & Energy Society – 2013-2015

c) Member of the Meetings and Conferences Steering Committee of IEEE Power & Energy Society for Region 10 (Asia-Pacific) – 2011-2012

d) IEEE Power Engineering Society Region 10 (Asia Pacific) West Chapter Representative – 2008.

He is also the recipient of following prestigious awards :

1. 2022 IEEE region 10 (ASIA - PACIFIC) outstanding volunteer award.
2. Life Time Achievement Award by IEEE India Council in 2019.
3. Eminent Engineer Award by the Electrical Engineering Division of the Institution of Engineers (India) in 2018.
4. The "Outstanding Small Chapter Award" from IEEE Power & Energy Society as the Chairman of Kolkata Power & Energy Chapter of IEEE in 2010.
5. The "Best Performance Award for Small Chapter" from IEEE Power & Energy Society as the Chairman of Kolkata Power & Energy Chapter of IEEE in 2009.
6. The "Outstanding Chapter Engineer Award" from IEEE Power Engineering Society in 2007.
7. The Third Millennium Medal of IEEE Calcutta Section in 2000.

5) Demonstrated Abilities as a National Leader in Higher Education:

By recognizing his leadership qualities, MHRD-GOI has given him the following national level leadership positions:

I) Chairman of the Anomaly Committee in relation to Recruitment Rules for Faculties in NITs – 2017 (final report submitted and notified by MHRD)

ii) Chairman of the Oversight Committee in relation to Recruitment Rules for Faculties as well as Non-Faculty Staff in NITs – 2018 (final report submitted and notified by MHRD)

iii) Chairman of the NITs Think Tank – 2019-2020 (report presented and accepted in NITSER Council Meeting)

6) Demonstrated Abilities as an Educationist:

In recognition of his standing as an educationist per excellence, the nominee has been bestowed with many prestigious Fellowships:

1) Fellow of the National Academy of Sciences India.
2) Fellow of the Indian National Academy of Engineering.
3) Fellow of West Bengal Academy of Science & Technology.
4) Fellow of The Institution of Engineers (India).
5) Fellow of The Institute of Electronics & Tele-Communications Engineers (India).

IEEE recognized the nominee as an outstanding educationist by nominating him as IEEE Power & Energy Society Distinguished Lecturer since 2005. He has delivered IEEE PES DL talks in Australia, New Zealand, Thailand, Malaysia, Philippines, Hong Kong, Jordan & India.

The nominee has delivered several special invited talks as enumerated below:

1) Faraday Memorial Lecture of IEEE Hyderabad Section in 2018.
2) MS Thacker Memorial Lecture of the Institution of Engineers (India) in 2018.
3) Dr. M.A.Pai Distinguished Lecture of IIT Kanpur in 2014.
4) Special address in the Open Session of 77th General Meeting of International Electro-Technical Commission in New Delhi in 2013.
5) Prof. J.K.Chowdhury Memorial Lecture of the Institution of Engineers (India) in 2010.
6) National Science Day Lecture of Indian National Academy of Engineering Kolkata Chapter in 2009.
7) Prof. P.N.Ghosh Memorial Lecture of the Institution of Engineers (India) in 2008.
8) Thomas Alva Edison Memorial Lecture of IIT Delhi in 2006.

The nominee has

i) Served as Associate Editor of IEEE Transactions of Dielectrics & Electrical Insulation.
ii) Served as Editor of Electrical Engineering Section of INAE Letters.
iii) Served as Member of the Editorial Board of Journal of The Institution of Engineers (India), Series B, published by Springer (India) Pvt Ltd.

7) Demonstrated Abilities in Planning for Developing Higher Education in the Country:

The nominee has taken part in several major committees over the years in planning for higher education in the country:

i) Member of Program Advisory Committee (PAC) on Electrical, Electronics and Computer Engineering, DST, GOI – 2016-2020.
ii) Member of Subject Expert Committee on Engineering Sciences for Fund for Infrastructure in Science & Technology (FIST) program of Department of Science & Technology (DST), Govt. of India - 2011-2020.
iii) Member of AICTE South-Western Regional Committee – 2019-2020.
iv) Member of the Board of Governors of the APJ Abdul Kalam Technical University, Thiruvananthapuram, Kerala – 2015-2020.
v) Member of the Academic Senate, IISER Kolkata – 2015-2016.
vi) Member of the Faculty Council for Post-Graduate & Undergraduate Studies in Engineering & Technology of Bengal Engineering and Science University, Shibpur – 2008-2012.
vii) Member of the PhD committee of West Bengal University of Technology - 2007-2008.
viii) Member of 'The Court' of Jadavpur University - 2006-2010.
ix) Member of the Executive Council of Jadavpur University - 1990-94.
x) Member of the Advisory Board of Assam Don Bosco University - 2011-2012.
xi) Member of the Board of Studies of Electrical Engineering Department of North Eastern Regional Institute of Science & Technology (DU), Nirjuli, Arunachal Pradesh -2006-2008.

In a nutshell, Prof. Chakravorti's accomplishments place him at the top of any group in engineering science and technology.

PULMOCON - 2022

20th All India Update in Pulmonary Medicine,

24th and 25th of September 2022.

Venue : CII Suresh Neotia Centre, Saltlake, Kolkata.

Organised by : Institute of Pulmocare & Research, Kolkata.

PROGRAM SCHEDULE

DAY 1 : 24th September, 2022 (Hall - A)

Time	Topic	Speaker/faculty	Chairperson
10:00 am - 10:01 am	Welcome address	Dr. Parthasarathi Bhattacharyya	
10:01 am - 10:30 am	**Difficult and Severe asthma - case based discussion**	Dr Raja Dhar Dr. Beauty Biswas	Dr. Hindol Dasgupta
10:30 am - 11:30 am	**Sleep disordered breathing**	Dr. Arup Halder Dr. Abhishek Kar	Dr. D J Roy
11:30 am - 11:45 am	***Tea Break***		
11:45 am - 12:15 pm	**Inauguration** **The Award giving ceremony: The Acharya P C Roy award**	Prof. Dr. Randeep Guleria Prof. Sivaji Chakravorti Dr. Debasish Bhattacharyya Dr. Samiran Panda	Dr. Dhiman Ganguly Dr. Parthasarathi Bhattacharyya
12:15 pm - 01:05 pm	**The Dr. S N De Memorial Oration:**	Prof. Dr. Randeep Guleria	Dr. Parthasarathi Bhattacharyya
01: 05 pm – 02:05 pm	***Lunch break***		
02: 05 pm – 03:00 pm	**Pleural diseases: case based review** *(2 cases)*	Dr. Laxmikant Baburao Yenge Dr. Sourabh Maji	Dr. Saibal Ghosh Dr. Arunava Dutta Chowdhury
03:00 pm - 07:00 pm	**workshop: preparing for final PG exam (oral / practical) – with dummy examination**		
	Individual examination: about syllabus, pattern, theory, and oral practical: How to prepare well ***(20 minutes)*** a) MD : Dr Supriyo Sarkar *(4 mins)* b) DNBE: Dr M L Gupta *(4 mins)* c) MRCP: Dr Subhasish Ghosh *(4 mins)* d) USMLE to MD to DNB (USA) - Dr Mridul Gupta *(8 mins)* **Clinical exam – how to do it: a talk (where students flaw) : Dr Arup Kr Kundu (30 minutes)** **Mock examination:** a) **long cases and short cases– real time or video:** Steered by Dr. M L Gupta **(40 minutes)** One / two long cases and two short cases will be discussed	**Faculty members:** Dr Arup Kumar Kundu Dr Supriyo Sarkar Dr M L Gupta Dr Radha Munje Dr. Animesh Ray Dr Subhasish Ghosh Dr Mridul Gupta Dr. Amitava Sengupta Dr. Somnath Kundu	Dr. Ankan Banerjee
	The cases will be video recorded before hand and following the presentation a student will face questions from the panel. **b) Short cases: a few spot cases: Dr. Arup Kundu: (5 minutes)** **Review of scoring and answers by the examiners: discussion and opinion exchange between examiners** **c) Oral tables:** (each 5 mins) **(40 minutes)** o Instruments o Drug o X-rays o HRCT o ABG o Clinical problems o Emergency o Flying *(Each area will start with a photo or video projection, projecting the drug / instrument etc. The students will be asked questions and the answers will be scored by the experts and the best possible reply will be discussed)* ***High tea at 05:15 pm to 05:30 pm: (15 minutes)*** OSCE: Dr Radha Munje, Dr. Animesh Ray **(80 minutes)** **Review of scoring and answers by the examiners: discussion and opinion exchange between examiners (10 minutes)** a) MD : Dr Supriya Sarkar b) DNBE: Dr M L Gupta c) MRCP: Dr Subhasish Ghosh d) Dr. Arup Kumar Kundu e) Dr Radha Munje f) Dr. Animesh Ray g) Dr Ankan Banerjee		

DAY 1 : 24th September, 2022 (Hall - B)

Time	Topic	Panel discussion	
10:00 am – 11:00 am	**Poster presentation**	**Judges:** Dr. Rupak Ghosh, Dr. Ranjan Das, Dr. A N Mondal, Dr. Anirban Sarkar, Dr. Saikat Nag	
11:00 am – 11:30 am	Setting up and running a pulmonary rehab program: the lessons learnt.	Institute of Pulmocare & Research	Dr. Kranti Raymane
11:30 am – 11:45 am	***Tea Break***		
11:45 am – 12:15 pm ***(Hall A)***	*Inauguration* ***(at Hall A)*** *The Award giving ceremony: The Acharya P C Roy award*	*Prof. Dr. Randeep Guleria, Prof. Sivaji Chakravorti Dr. Debasish Bhattacharyya, Dr. Samiran Panda Dr. Dhiman Ganguly, Dr. Parthasarathi Bhattacharyya*	
12:15 pm – 01:05 pm	*The Dr. S N De Memorial Oration:* ***(at Hall A)***	*Prof. Dr. Randeep Guleria*	*Dr. Parthasarathi Bhattacharyya*
01: 05 pm – 02:05 pm	***Lunch break***		
02: 05 pm – 02:45 pm	**National Pulmo Quiz** *(Elimination round)*	Quiz Master: Dr. Sushmita Roychowdhury, Dr. Arindam Mukherjee, Dr. Shivaresmi Unnithan	
02: 45 pm – 03:00 pm			
03:00 pm to 04:30 pm	"Medical education - some perspectives from home and abroad". Medical education: changing methods and practice The scenic route to medicine Reflections of Medical Teacher without borders	Dr. Dhiman Ganguly Dr. Shampa Sinha Dr. Sankar Sinha	Dr. Dhiman Ganguly
	Tea will be served at hall		
Symposium on career option for young pulmonologists:			Dr. A G Ghoshal
04:30 pm to 06:30 pm	1) Private practice in rural areas: my lessons and suggestions	Dr Monotosh Khanra	
	2) Practicing pulmonology over a wide spectrum: my take	Dr. A G Ghoshal	
	3) Joining a corporate hospital: my suggestions	Dr Debraj Jash	
	4) Making research as a career base: how did I make it?	Dr Sundeep Salvi	
	5) Choosing further education: getting in DM	Dr Puneet Saxena	
	6) Choosing a career abroad - a) UK b) USA	Dr Suman Das Dr Parijat Sen	
	7) Want to be a teacher – the way forward	Dr. Randeep Guleria	
	8) Opting in a sub-sub speciality (intervention/ intensive care/ sleep/etc.) – the way out	Dr. Dhruva Chaudhry	
	9) Opting something different: online consultation/medical writing/ working for Pharma/ tele-reporting/others	Dr. Laxmikant Baburao Yenge	
	10) Medicolegal issues for a budding specialist	Dr. Krishnendu Mukherjee, Dr. Biswajit Sukul	
	11) Free discussion between the faculties and interaction with the audience		

DAY 2 : 25th September, 2022 (Hall - A)

Time	Topic	Faculty	
09:30 am – 10:00 am	**Case based discussion: Tuberculosis**	Dr. Supriyo Sarkar	Dr. Shelly Shamim Dr. Kranti Garg
10:00 am - 10:30 am	**Critical care - A case discussion**	Dr. S Todi	Dr. Ajoy Sarkar
10:30 am – 10:45 am	***Tea break***		
10:45 am – 11:45 am	**ILD – case based discussion** *(2 cases)*	Dr. Puneet Saxena Dr. Suranjan Mukherjee	Dr. Ansuman Mukherjee Dr. Debasish Behera
11:45 am – 12:15 pm	Igniting innovations in young mind	Prof. Sivaji Chakravorti	Dr. Parthasarathi Bhattacharyya
12:15 pm – 12:45 pm	**COPD – case-based approach**	Dr. Parthasarathi Bhattacharyya	Dr. Pawan Agarwal
12:45 pm – 01:15 pm	**Lung Cancer**	Dr. Pawan Kumar Singh	Dr. Madhuchhanda Kar
01:15 pm – 02:05 pm	***Lunch Break***		
02:05 pm – 02:55 pm	National Pulmo Quiz ***(Final round)***	Quiz Master: Dr. Sushmita Roychowdhury, Dr. Arindam Mukherjee, Dr. Shivaresmi Unnithan	
02:55 pm – 03:55 pm	**Pulmonary eosinophilia – case based discussion** *(2 cases)*	Dr. Angira Dasgupta Dr. Ajmal Khan	Dr. Joydeep Roy Dr. Sumit Sengupta
03:55 pm – 04:10 pm	***Tea break***		
04:10 pm – 04:40 pm	Management of massive hemoptysis Bi-PAP in critical care: my understanding	Dr. Kranti Garg Dr. Leelavati Thakur	Dr. Sudip Ghosh
04:40 pm – 05:00 pm	Quiz, Research *(Poster presentation)* **AWARD, and Valedictory session**		

DAY 2 : 25th September, 2022 (Hall - B)

10:00 am – 11:30 am	**Poster and platform presentation**	**Judges:** Dr. Rupak Ghosh, Dr. Arunava Dutta Chowdhury, Dr. Shivaresmi Unnithan, Dr. Tanveer Reja, Dr. Anirbn Biswas	
11:30 am – 11:45 am	**Phenotyping of obstructive air-way diseases:** - COPD its applied importance	Dr. Debasish Behera	Dr. Sujan Bardhan
11:45 am – 12:00 noon			
12:00 noon – 12:15 pm	***Tea break***		
12:15 pm - 01:30 pm	**Workshop: IOS + FOT**	Dr. Sundeep Salvi, Dr. Deesha Ghorpade Dr. Saibal Moitra	Dr. Indranil Halder Dr. Parthasarathi Bhattacharyya
01:30 pm - 02:20 pm	***Lunch break at the hall***		
02:20 pm - 04:00 pm	***Continuation of Workshop*: IOS + FOT**	Dr. Sundeep Salvi, Dr. Deesha Ghorpade Dr. Saibal Moitra	Dr. Indranil Halder Dr. Parthasarathi Bhattacharyya

Details of Case-based Discussion

Day-1: 24th September 2022

Sl.no	Areas	Speaker/Presenter	Time
1	Difficult and Severe Asthma	Dr. Raja Dhar	10:01am-10:30am
2	Sleep-disordered breathing	Dr. Arup Halder	10:30am-11:30am
3	Sleep-disordered breathing	Dr. Avishek Kar	
4	Pleural Diseases	Dr. Laxmikant Baburao Yenge	2:05pm-3pm
5.	Pleural Diseases	Dr. Sourabh Maji	

Day-2: 25th September 2022

Sl.no.	Areas	Speaker/Presenter	Time
6.	Tuberculosis	Dr. Supriyo Sarkar	9:30am-10:00am
7.	Critical Care	Dr. S. Todi	10:00am-10:30am
8.	DPLD	Dr. Puneet Saxena	10:45am-11:45 am
9.	DPLD	Dr. Suranjan Mukherjee	
10.	COPD	Dr. Parthasarathi Bhattacharyya	12:15pm-12:45pm
11	Lung Cancer	Dr. Pawan Kumar Singh	12:45pm-1:15pm
12.	Pulmonary Eosinophilia	Dr. Angira Dasgupta	2:55pm-3:55pm
13.	Pulmonary Eosinophilia	Dr. Ajmal Khan	

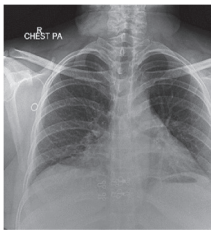
Presenter : **Dr. Raja Dhar**

Severe Asthma Case Discussion

Clinical details..

- 36yrs female , house wife
- Resident of Karnataka
- Diagnosed case of long standing asthma
- No addictions, passive smoking at home
- No biomass exposure
- Weight 77kg, height 156 cm, BMI 27 Kg/m^2.
- No comorbidities.

Clinical details



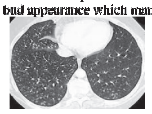
ge of onset of symptoms : 24 years
mptomatic since 12 years
ough, expectoration, wheeze, runny nose and
ezing

gular use of inhaled medicines
ICS/LABA
Initially was advised DPI for 2-3 years (no records), later MDI.
Optimal usage.

story of exacerbations 1-2 times per year initially, as given on/off steroids.

Initial presentation – 2016

- Patient had exacerbation in 2016 required admission in ICU, but no mechanical ventilation required. HRCT thorax showed presence of random nodules and tree in bud appearance which mandate bronchoscopy to rule out infection.



- Her BAL culture showed no growth, BAL galactomannan was 2.48, fungal culture showed growth of aspergillus.
- She was treated with antifungal, iv steroid and iv antibiotics, got better.

Clinical details...

- Family history of asthma- Mother , Maternal grand mother

Asthma comorbidities:

- Allergic rhinitis without polyps
- Allergic dermatitis
- Overweight
- GERD
- No history of anxiety.

Triggers for symptoms:

- Dust
- Cigarette smoke
- Apple
- Cold
- Strong smells.

Treatment received -

- Inhalation therapy
  - High dose ICS (Fluticasone 1000 ug/day) and LABA (Salmeterol 100 ug/day)
  - LAMA (Tiotropium 18 ug/day)
- Montelukast (10 mg/day)
- Oral corticosteroids (for recent exacerbation)

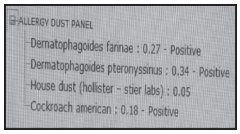
Next steps....

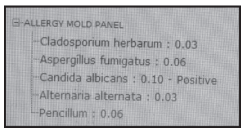
- Inhaler compliance ensured (to the best possible)
- Inhaler technique checked (MDI with spacer)
- Environmental triggers – ruled out
- OSA : Not present on sleep study

Phenotyping asthma - 2016

- **Serum Ig E level** : 635 KIU/L
- **Absolute eosinophil count** : 417 cells/mm3
- **FeNO** : 88
- **PFT** : Moderate obstructive physiology with reversibility

Allergen sensitization testing...





Data base...

- Young lady with asthma
- Allergic rhinitis, GERD and Obesity
- Frequent exacerbations despite good inhaler compliance/technique
- Poor QOL and symptom control
- Now on OCS for symptom control (1 month)

Phenotype of this patient??

T2 high atopic , eosinophilic basically overlap category.

Next steps to optimize are

A. Biologics – anti Ig E

B. Biologics – anti IL 5 biologics

C. Bronchial thermoplasty

D. Macrolides

Treatment given in 2016...

- Exacerbation controlled with OCS
- Switched to SMART therapy with Formoterol/Budesonide
- Continued Tiotropium
- Initiated on Omalizumab 300 mg/month (suboptimal dose) – financial reasons

2016 – 2017 : Omalizumab therapy

- Took Omalizumab 300 mg /month for 1 year

Response:
- Weaned off OCS
- Wheeze resolved and symptoms became better
- Exacerbations decreased (not stopped)
- QOL improved

Stopped Omalizumab after 1 ye
(Financial reasons)

Exacerbations (2016-2020)

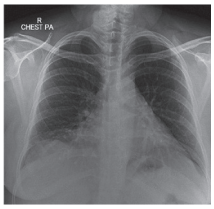
Year	Admissions requiring hospitalisation	Severe exacerbations (requiring OCS)
2016	2 (one in ICU)	3
2017	1	2
2018	0	0
2019	0	3
2020	1 (COVID positive)	2

2021 : Disease control

- Significant worsening of control
- Monthly exacerbations necessitating OCS bursts despite being on ICS/LABA/LTRA
- Restarted on maintenance low dose OCS (10mg/day prednisolone)
- Severe exacerbation necessitating admission following viral URTI

Re evaluated in 2021...

- **Serum Ig E level** : 541 IU/mL
- **AEC** : 350 cells/mm3
- **FeNO** : 40 ppb
- **PFT** : Moderate obstructive physiology with reversibility



Practical problems faced...

1. Financial issues
2. Lack of medical insurance coverage
3. Place of residence > 6 hrs from hospital

In addition to medical issues....!!

Next options are

A. Restart Omalizumab

B. Try anti IL 5 biologic Mepolizumab

C. Try anti IL5 biologic Benralizumab

D. Bronchial thermoplasty

Our case...

Enrolled her in a ongoing trial on Mepolizumab for OCS dependant severe asthma

Take home messages...

1. Effect of biologic will wane after stopping the biologic in a subset of responders..
2. Patients with Overlap phenotype are candidates for both anti Ig E and anti IL 5 biologics.. (Biologic class switch may be considered)

Thank you

1. Indications of bronchoscopy in severe asthma? Do you think it was appropriate for this patient to have a bronchoscopy?
2. What is severe asthma with fungal sensitization?
   Correlation of BAL galactomanan and fungal culture? How would you categorise this patient who has a raised Galactomannan and no Aspergillus on fungal stain and culture?
3. Positioning BAL galactomanan in infective exacerbations of asthma?
4. Significance of FENO in asthma today? How does it help if at all?
5. SPT versus total IgE which one is more sensitive for predicting atopic status?' Specific IgE versus SPT- is there anything to choose between the 2 techniques?
6. What is overlap category?
7. Parameters to assess biologics response?
8. This patient was given a biologic at a sub-optimal dose. Is there any data to suggest that this can be done?
9. Is there any data to show that biologics can be 'weaned off' or stopped? Are there any markers to guide this? When would you restart?
10. Role of biologics in ABPA?

Notes:

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THE CASES FOR DISCUSSION IN PULMOCON - '22

Time : 10.30 AM. Date : 24/09/22

Topic- Sleep disordered breathing

Presenter : **Dr. Arup Halder**

Obesity hypoventilation syndrome

Case 1.

65 years old lady was admitted in ICU with history of acute confusion followed by drowsiness for 1 day. She was obese (BMI=34), diabetic and hypertensive. Family members gave history of low grade fever for last 2 days and dry cough of same duration. They also complaint about the excessive daytime sleepiness and loud snoring with chocking at night. On examination she was hypoxic in room air. Vitals as: Pulse= 94/ min, BP= 150/ 86, Respiratory Rate: 18/min, SpO2= 80% in room air, Work of Breathing: Not increased, No pedal edema, Neck Vein = not engorged. Chest auscultation revealed crepitations in both bases but more on right side. Capillary blood glucose was 254 mg/dl, ABG : Acute on chronic type 2 Respiratory failure with hypoxemia.

Case 2.

A 60 year old gentleman with BMI >40, came for OPD consultation with history of breathlessness on exertion, dizziness while walking and excessive daytime sleepiness, fatigue and snoring during sleep. He had a pre-syncope a day before consultation. He was non diabetic, but had Dyslipidemia and Hypertension . On examination, his vitals were: Pulse 102/ min, Regular, BP 160/100, SpO2= 94% in room air, Respiratory Rate = 20/ min, bilateral pedal edema. Chest auscultation revealed diminished breath sounds bilaterally. Cardiac auscultation revealed loud S2.

Questions?

1. What is the definition of OHS?
2. What is the prevalence of OHS?
3. What are the predisposing and accompanying factors?
4. What are the pathophysiology of OHS?
5. What is the approach to diagnosis of OHS? What are the important clues in history, clinical examinations, and investigations?
6. What is the treatment of OHS?

Notes:

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Presenter: **Dr. Abhishek Kar**

A patient of OSA whose symptoms and apnea-hypopnea index deteriorated with CPAP !!!

A 46-year-old man with a history of snoring , daytime sleepiness and h/o transient episodes of falling asleep while driving presented to our sleep clinic. On subjective and objective evaluation of sleepiness , score was high on both the scales indicative of sleep apnea. After clinical and biochemical evaluation he underwent a split-night sleep study (Tables-1 and 2). During the diagnostic portion, the apnea–hypopnea index (AHI) was severely increased, but the arterial oxygen desaturation was mild. During the titration portion of the study, the AHI remained elevated because of central apneas that did not have a Cheyne-Stokes morphology. Patients ECHO was done which was not s/o CHF. No history of congestive heart failure (CHF) or opioid use was present. The only medications the patient was taking were levothyroxine and telmisartan. The patient was started on continuous positive airway pressure (CPAP) of 8 cm H2O as this was the lowest pressure on which most obstructive events resolved. It was presumed that the central apneas would resolve on chronic treatment. The patient returned to the clinic, and the machine download is shown in Table-3. The patient reported still having many awakenings, and his daytime sleepiness had not improved. He also reported difficulty sleeping on CPAP.

Table-1 : SPLIT-NIGHT STUDY RESULTS

	Diagnostic	Treatment
TST	120	220
REM (minutes)	24	44
AHI	36	32.2
AHI NREM	30	39
AHI REM	60	7
OA	30	7
MA	0	0
CA	5	81
Hypopnea	37	30
Desaturations	72	118
Low SaO_2	85	88

AHI, Apnea–hypopnea index; *CA*, central apnea; *OA*, obstructive apnea; *MA*, mixed apnea; *NREM*, non– rapid eye movement; *REM*, rapid eye movement; SaO_2, saturation of arterial oxygen; *TST*, total sleep time.

Table-2 : PAP treatment table

PAP	TST	REM	AHI	AHI REM	OA	CA	MA	Hypopnea
5	20	0	36	0	8	0	0	4
6	30	0	34	0	5	0	0	12
7	20	6	32	40	5	3	0	3
8	60	18	10	7	0	9	0	1
9	20	0	60	0	0	24	0	6
10	30	10	38	0	0	15	0	4
12	30	10	40	0	0	15	0	5

AHI, Apnea–hypopnea index; *CA*, central apnea; *OA*, obstructive apnea; *MA*, mixed apnea; *PAP*, positive airway pressure; *REM*, rapid eye movement; *TST*, total sleep time.

Table-3 : Download report from CPAP after 1 month of use.

Used 20/30 days	
Average use (days used)	4 hours
Apnea–hypopnea index (#/hr)	20
Clear airway apnea index (#/hr)	18
Obstructive airway apnea index (#/hr)	2
Hypopnea index (#/hr)	0
Large leak (min)	3 minutes

CPAP, Continuous positive airway pressure.

Questions?

1. What is your diagnosis and what intervention do you recommend?
2. Why do you recommend such treatment? Please discuss.
3. What are the risk factors of emergent CSA
4. What is the mechanism of TE-CSA?
5. What is complex sleep apnoea?
6. How would one diagnose TE-CSA?
7. What is the treatment of the condition and how does one follow them up?
8. Is there any specific intervention of TE-CSA?
9. What is the role of carbon-di-oxide supplementation in TE-CSA?

Notes:

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A CASE OF PLEURAL DISEASES

Summary of case

19 yr male was evaluated for right sided pleural effusion in January 2022. Pleural flid analysis was exudate, lymphocyte predominant ADA 102. Baseline AFB Culture was not done.

Q: What is level of confidence in diagnosis of tuberculosis? Can we start ATT?

Q: Should we go beyond this in all cases? If yes what are options?

Q: Sensitivity of various tests for diagnosis of TB?

He was started on ATT, weight based 4 drug which he tolerated well. Serial CXR showed improving trend of pleural effusion. June 2022- he continued to have mild chest discomfort & CXR showed persistent mild to moderate right pleural effusion with suggestion of loculations. (Image)

Q: How do we proceed?

Diagnostic +/- therapeutic tapping planned & USG guided pleural tapping done. Frank pus.

Q: What next? Drain. Q: Small bore vs large bore?

12 F pleural drain inserted. Fluid drained. Check CXR (Image) Residual HPTx. Pus gene Xpert- MTB Low, Rif resistance not detected. Aerobic culture no growth, AFB Culture awaited.

Q: Why was Gene Xpert done in this case? Does it help?

ATT CP continued. HPTx persistent.

Q: What can be done? IPFT/Negative suction?

Put on negative suction -10□20□30□40.

No expansion of lungs.

Q: what next?

I was inclined to remove drain and accept sterile cavity.

Patient being young, family was keen on anatomical improvement.

Surgery opinion & CT Chest. (Image)

Thoracoscopic decortication done. Surgeon noted non expansion of lung at end of decortication with mild air leak. I felt, let's not do more harm and avoid aggressive handling of lung.

POD 2- CXR- (Image), worse than baseline, expiratory air leak.

Q: What next?

Restarted on negative suction

Gradually partial expansion of lung achieved. Air leak decreased but did not stop over 2 weeks.

ICD removed. Moderate loculated pneumothorax- not worsening on serial CXR.

Kept under observation.

AFB culture no growth at 2 months, patient asymptomatic- ATT stopped.

Hoping that nature will heal and close that cavity.

Q: At least retrospectively, did we have any clues for possible failure of surgical options

Review CT Images again.

Notes:

Topic-Pleural diseases: case based review

Unusual cause of pleural effusion

Presenter : **Dr. Sourabh Maji**
Consultant pulmonologist and intensivist
Rtiics ,kolkata

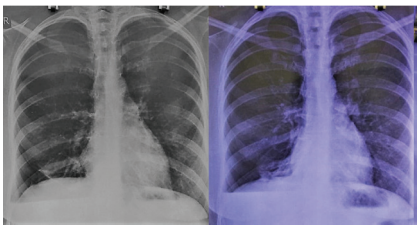
In 2018

- 26 year
- Female
- Software engineer
- No previous comorbidities
- Non diabetic
- Non hypertensive
- Non smoker

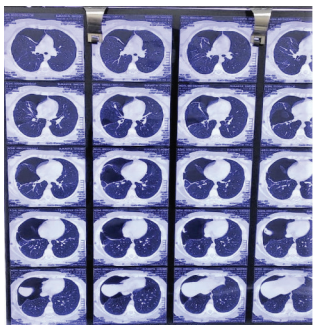
In 2018

- Incidentally detected right sided pneumothorax in regular health check-up
- Thought to be primary spontaneous pneumothorax
- Treated conservatively
- No response
- ICTD was given was

23/02/2018 28/03/2018



Right sided Pneumothorax



Question

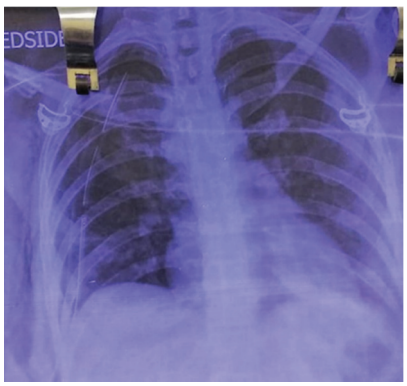
- Diagnosis of pneumothorax?
- Diagnosis of pneumothorax in ventilated patient?
- Classification of pneumothorax?
- Treatment?

- Negative suction applied
- Persisting air leak
- VATS done and biopsy was taken
- Report suggestive of granulomatous inflammation
- Put on ATT non DOTS
- ATT induced hepatitis
- Stopped after 15 days
- There is good radiological response

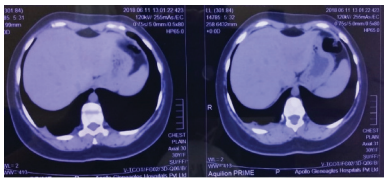
Question

- Role of negative suction in pneumothorax?
- Indication of VATS in pneumothorax?

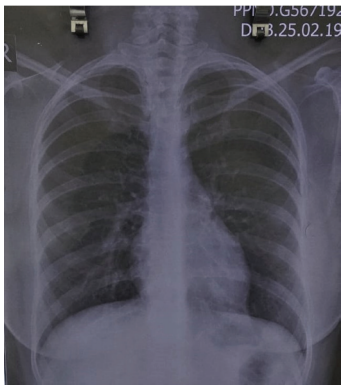
2/5/2018 (post VATS)



06/11/2018



25/02/2019



- Slide and block reviewed in all renowned laboratories in Kolkata
- Granulomatous inflammation the possibility of mycobacterial infection to be considered
- ATT was stopped and patient was under follow up of local physician
- She was doing fine except some pain in the right side

In 2020 January

- C/o right sided heaviness of chest and mild cough
- No fever,cough,expectoration,constitutional symptoms
- X-ray showed right sided pleural effusion
- Reviewed by different doctors
- Opinion to restart ATT
- Went to Bangalore and was advised for either open Biopsy or VATS guided biopsy
- Presented to OPD

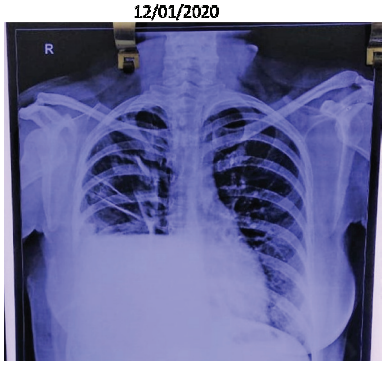
On examination

- Patient is well preserved
- Only mild cough
- No SOB
- No constitutional symptoms
- Right sided mild volume loss and dull note on percussion
- No other organomegaly was noted

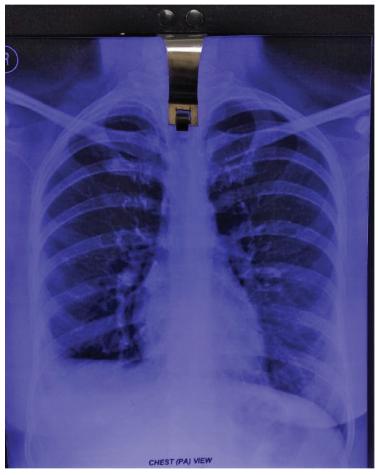
Question

- Opinion regarding restart ATT in this patient?
- What is next investigation biopsy or ATT?
- Mode of biopsy?

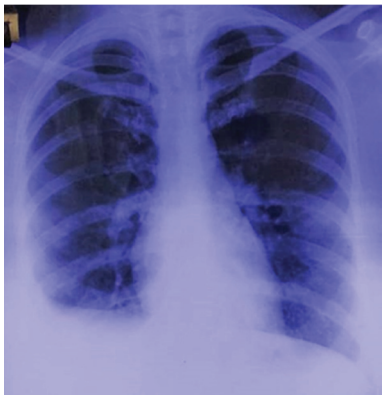
12/01/2020



22/05/2019



12/06/2019



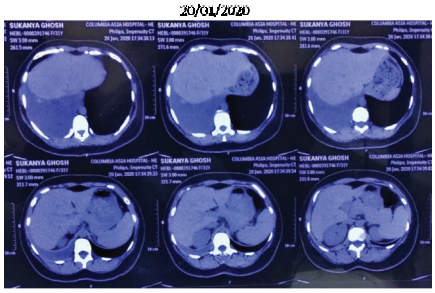
Pleural fluid report

- Haemorrhagic
- Cell-2000/cmm
- $N_{55}L_{20}E_{10}Macrophage_{15}$
- Protien-4.2gm/dl(serum protien-6.4gm/dl)
- Sugar-76mg%
- ADA-24
- No malignant cell seen

Question ?

- Differential diagnosis?
- Next investigation?

20/01/2020



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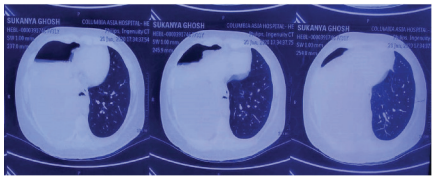
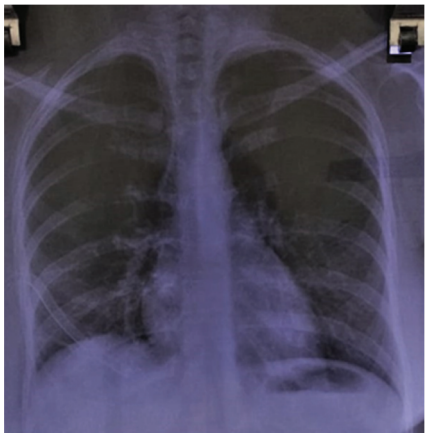


- Medical thoracoscopy and biopsy was done under sedation
- Dull looking pleura
- Adhesion noted
- Biopsy taken and adhesinolysis done as much as possible
- ICTD placed
- Patient was discharged with ICTD situ with proper ICTD care

Indication of medical thoracoscopy?

Post medical thoracoscopy



- **Biopsy report:**
- Evidence of chronic inflammation
- Haemosiderin laden macrophage
- No granuloma and no malignancy

• What next?

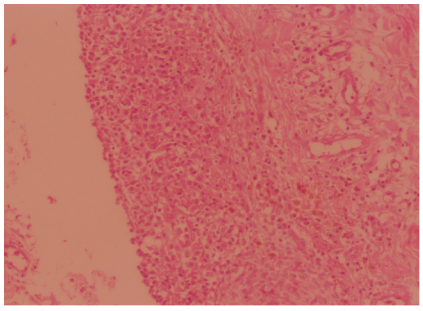
2-3 days after ....

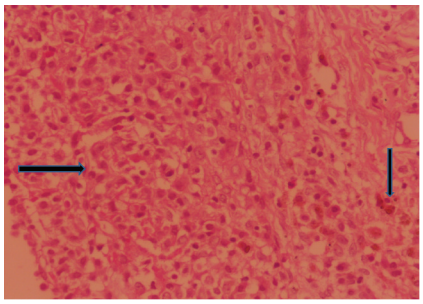
At evening patient presented to emergency with

- Gush of blood coming out through ICTD
- Air leak was there
- No sob
- No other bleeding manifestation
- Her menstruation cycle started two day back
- She was managed on that day conservatively

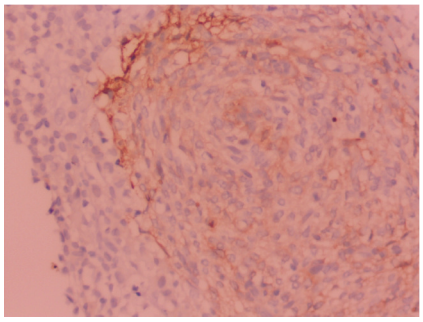
- Change in differential diagnosis?

- This information was discussed with the pathologist
- Asked for old slides and block
- All old slides and blocked was reviewed
- Lucky enough to get it

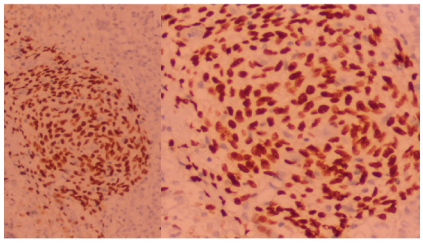




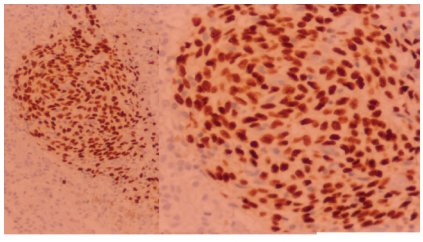
CD10



ER POSITIVE



PR positive



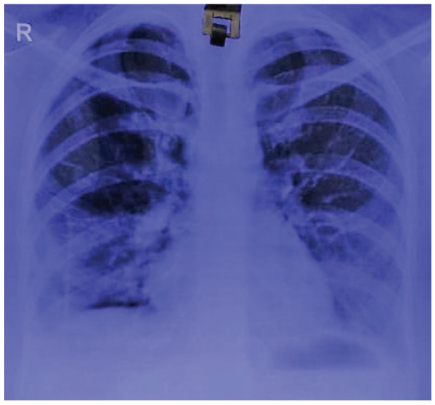
Diagnosis: Pleural endometriosis

After few days....

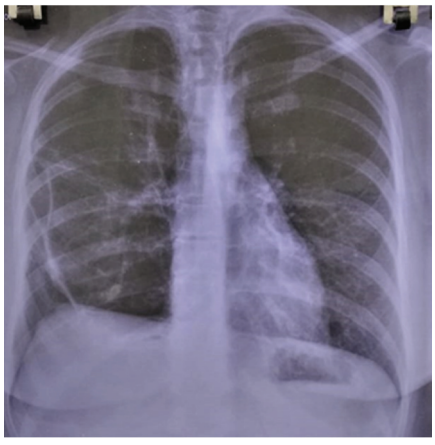
- Repeat x-ray shows lung expanded and no air leak and ICTD drain output less than 50 ml/day
- After waiting for few days Pleurodesis with povidone iodine was done
- ICTD removed
- Referred to gynaecologist
- Having pelvic endometriosis also
- Treated with hormonal therapy
- She conceived with IVF and given birth of a girl child by caesarean section with no complications

- Indication of pleurodesis?
- Materials used in pleurodesis?

30/09/2020



6/12/2020



Literature review

- Presence of endometrial-like glands and stroma outside the uterine cavity
- Affect 6%–10% of reproductive-age women
- Among this population,12% are estimated to experience endometriosis of non reproductive organs
- Most common site outside of the abdomino pelvic cavity within the thoracic cavity
- Lung parenchyma , on the diaphragm, pleural surfaces produces a range of clinical and radiological manifestations

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- Catamenial pneumothorax, hemothorax, hemoptysis, and pulmonary nodules, resulting in an entity known as thoracic endometriosis syndrome (TES)
- Typically develop pelvic endometriosis 5-7 yrs prior to diagnosis of TES
- Among patients diagnosed with TES, 50%–84% have concomitant pelvic endometriosis

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Pathophysiology

- **Retrograde menstruation Theory**
- **Coelomic metaplasia Theory:** Metaplasia of mesothelial cells lining the pleura and peritoneal surfaces into endometrial glands and stroma
- **Lymphatic and Hematogenous dissemination**
- **Prostaglandin Theory:** circulating prostaglandin F2 increases with menstruation, leading to the constriction of bronchioles and blood vessels

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Sign and symptoms

- Depends on location
- Many patients are asymptomatic
- Experience a constellation of temporal symptoms and radiologic findings with menstruation
- Catamenial pneumothorax (80%), catamenial hemothorax (14%), catamenial hemoptysis (5%), and rarely pulmonary nodules
- Catamenial pneumothorax -recurrent pneumothorax occurring within 72 h of the onset of menstruation

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- Right sided hemothorax up to 92% of cases, with 5% of cases involving the left hemothorax and 3% experiencing bilateral involvement
- Bronchopulmonary TES presents as mild to moderate catamenial hemoptysis
- Lung nodules identified on imaging is rare finding
- Massive life-threatening hemoptysis is rare
- isolated diaphragmatic endometriosis typically asymptomatic but can result in irritation of the phrenic nerve

Diagnosis

- Variable presentation
- High level of clinical suspicion is needed
- Diagnostic modalities depends on clinical presentation and site of involvement
- Chest x-ray or ct thorax for pneumothorax or pleural effusion,MRI chest for diaphragmatic involvement
- Atypical presentation: pneumomediastinum, pneumoperitoneum, ground glass opacities, bronchial wall thickening, thin-walled cavities within the lung parenchyma, or bullous formation

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Diagnosis

- Gold standard -video laparoscopy (VL), and video- assisted thoracoscopic surgery (VATS)
- Intraoperative findings: diaphragmatic lesions (38.8%); endometriosis of the visceral pleura (29.6%); discrete lesions such as bullae, blebs, or scarring (23.1%); and no findings (8.5%)
- Diagnostic imaging and tissue sampling have inconsistent results
- Timing of diagnostic imaging and sampling improved the outcome:time of menses vs midcycle

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Management

- Medical management: first line treatment
- Goal to suppress ovarian steroid hormone production
- Gonadotropin-releasing hormone (GnRH) analogs are used first-line
- Surgical treatment:laser,cryo diathermy
- Chemical Pleurodesis

THANK YOU

Notes:

Topic- Case based discussion: Tuberculosis

A middle aged lady presented with insidious onset of cough and dyspnoea

Presenter : **Dr. Supriyo Sarkar**

History

- Name – M D; Age – 44 years; Sex – F; House wife/ cook of Bally, Howrah
- Date of admission – 20.07. 2022 Date of examination – 01.08.2022
- Chief Complain –
  1. Cough for 4-5 years
  2. Dyspnoea for 2 years
- History of present illness –
  - Patient had insidious onset of cough for many years, exact onset patient could not tell. Cough had no seasonal no diurnal variation. Cough was sometimes associated with scanty mucoid to purulent expectoration and blood streak expectoration.
  - Shortness of breath was insidious onset and progressive. At the date of examination, dyspnoea was mMRC grade III. It was not associated with wheeze and variability.
  - She came to our OPD with dyspnoea at rest and hypoxemia (SpO_2 – 76%)

History cont^n—

- Past history –
  - She had pulmonary TB about 40 years back (details she cannot tell and no records are available)
  - She had type II DM detected 2 years back, irregularly treated with OHA
  - She had abdominal hysterectomy with bilateral salphyingo-oophorectomy 4 years back
- Personal history—no addiction, allergic to egg and prawn, she has achieved menopause, she is working as cook and has exposure to biomass fuel.
- Family history—not significant.

General survey

- GCS, Built, Nutrition, - normal; Decubitus – of choice
- Pallor, Cyanosis, Jaundice, Edema, - absent; Clubbing – grade II
- PR – 96/min; BP – 128/76 mm of Hg; RR – 23/ min; Temp – 97.8°F
- Neck veins – not engorged; neck glands – not palpable
- SpO_2 – 98% with 5L/min O_2 by face mask

Examination of respiratory system

- Upper respiratory tract including oral cavity - normal
- Examination of thorax—
- **Inspection—**
  - Shape – elliptical; Symmetry – flattening of left infraclavicular area
  - Signs of respiratory distress – absent
  - Movement – diminished left side
  - Overlying skin – no abnormality detected
  - Pulsation – not visible
  - Sternomastoid sign – positive on left side
  - Examination from back – dropping of left shoulder, winging of left scapula and scoliosis with concavity towards left

Examination of respiratory system cont^n—

- **Palpation—**
  - Temp/ tenderness – NA
  - Movement – decreased in left hemithorax
  - Trachea – shifted to left
  - Apical impulse – at 5th intercostal space in left anterior axillary line
  - Vocal fremitus – decreased on left hemithorax but increased in left infrascapular area
  - Spinoscapular distance –
  - Spine –
  - Chest wall expansion -

Examination of respiratory system cont^n—

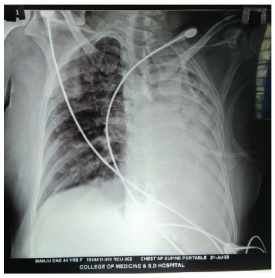
- **Percussion:**
  - Right hemithorax—normal resonance
  - Left hemithorax—
    - Middavicular line—resonance up to 4th intercostal space and impaired downwards
    - Midaxillary line—impaired
    - Suprascapular area—resonant
    - Interscapular area—impaired
    - Infrascapular area—dull

Examination of respiratory system cont^n—

- **Auscultation:**
- Right side — normal vesicular breath sound with normal vocal resonance, no added sound
- Left side—
  - Breath sounds:
    - Tubular breath sound in left infraclavicular area
    - Cavernous breath sound in left infrascapular area
    - Normal to diminished vesicular breath sound in other areas of left hemithorax
  - Vocal resonance — increased in left infraclavicular area and left infrascapular area, other areas normal to decreased
  - Biphasic crackles— scattered over whole left hemithorax, more prominent left infrascapular area

Examination of other systems: Normal

Her chest x-ray dated 21st July 2022



- What are the abnormalities in chest x-ray?
- How will you exclude active TB?
- Explain following sputum for AFB and the CBNAAT findings in this patient

i. AFB (-) & CBNAAT (-)
ii. AFB (+) & CBNAAT (+)
iii. AFB (-) & CBNAAT (+)
iv. AFB (+) & CBNAAT (-)

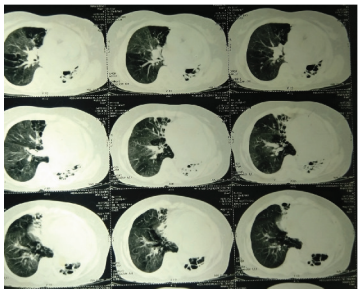
Her ABG showed on 20. 07. 22

With 5L/min O_2 by face mask

PH—	7.33
$PaCO_2$—	57 mm of Hg
PaO_2—	231 mm of Hg
HCO^3—	29.1 mmol/L

- What was the ABG abnormality?
- How will you explain dyspnoea and hypoxemia?
- What other investigations should be done in this case?

Her HRCT-Thorax on 15th July 2022



- What are the CT abnormalities?
- What are the causes of mosaic attenuation?
- What will be the possible causes of mosaic attenuation in this case?
- What is you final diagnosis?

Notes:

Presenter : **Dr. S Todi**

Antibiotic Choices for MDR/XDR Gram negative infections Dr Subhas Todi

Antibiotic choice is a critical step in the management of MDR/XDR Gram negative infection. Dr Subhas Todi, a renowned critical care expert, will deal with the issue with presentation of multiple cases.

Case 1.

60 year male
Admitted with acute pyelonephritis
Hemodynamically stable
Urine c/s : E.Coli >105
Ceftriaxone : R,
Piperacillin / Tazo : S :MIC 8
Meropenem : S : MIC 0.5

Case 2.

70 year Male, diabetic , renal calculi
Admitted with acute Pyelonephritis
Blood /Urine culture Klebsiella
Pip/Taz : R >16
Ertapenem : R >2, Meropenem : S . MIC 0.5
Colistin : I

Case 3.

50 year female , diabetes , impaired renal function, previous hospital admissions
Admitted with lobar pneumonia
Sputum/Blood culture : Klebsiella
Pip/Taz : R, Ertapenem : R, Meropenem : R : MIC 4
Tigecycline : S
Colistin: I

Case 4.

50 year female , diabetes , impaired renal function, previous hospital admissions
Admitted with lobar pneumonia
Sputum/Blood culture : Klebsiella
Pip/Taz : R. Ertapenem : R
Meropenem : R : MIC 4
Tigecycline : S , Colistin: I, Ceftaz Avi : S

Case 5.

40 year female
Admitted with alveolar hemorrhage secondary to vasculitis
Treated with high dose steroid and Rituximab
Developed severe hypoxia and new lung infiltrate
ET suction and blood culture grew Acinetobacter
R to all except Colistin : I MIC : 0.5
Meropenem MIC: 16, Amp/Sulb : Not Done

Notes:

Approach to Interstitial lung diseases: Two case studies

Puneet Saxena, Itishree Singh

CASE 1

A 50-year-old female initially presented with dyspnoea and dry cough with a duration of 3 months. On further questioning, she also complained of muscle weakness and aches involving both upper and lower limbs. She had no other constitutional symptoms suggestive of CTD with no definite exposure and family history. Her clinical examination was normal with few inspiratory squeaks and a chest radiograph was normal. Spirometry showed restrictive pattern with a forced vital capacity (FVC) 1.86L (62% predicted). High Resolution Computed Tomography (HRCT) was done that was suggestive of Fibrotic interstitial lung disease- probable usual interstitial pneumonia (UIP) pattern

What are the essential components of history taking in ILD?

What is the minimum laboratory work up that is important towards the diagnosis of ILD?

When are the domain to classify interstitial pneumonia with autoimmune features?

What is IPAF and how does it differ from UCTD?

What are the various HRCT patterns of UIP?

What are the radiological clues to differentiate CTD-UIP from Non-CTD UIP?

How must the ILD patients be followed up?

What is the differential diagnosis of mosaic attenuation?

What is the radiological (HRCT) approach to mosaic attention?

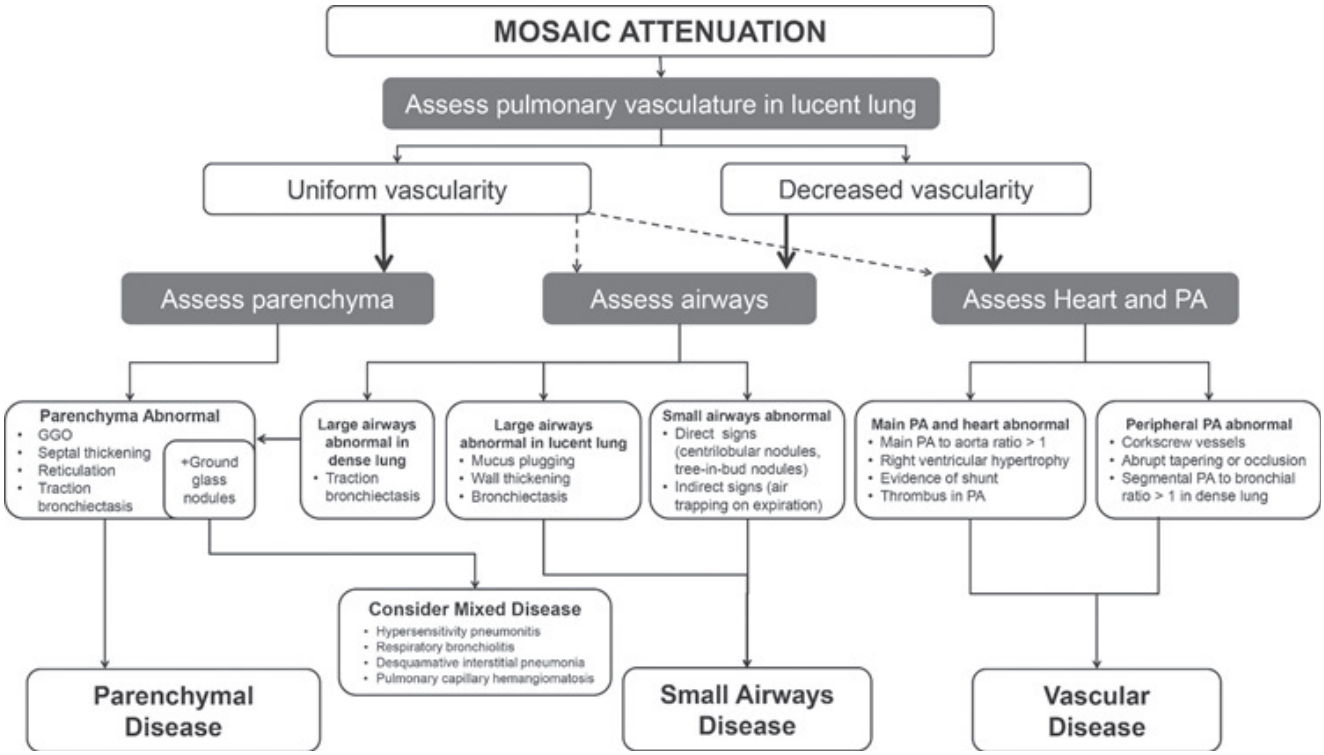


Figure 1:Flowchart for diagnosis of mosaic attenuation taken from Kligerman et al. 2015.(15)

What is Multi-Disciplinary Team discussion and why is it important towards the diagnosis and management?

What is the role of BAL in ILD?

What is the Management of Hypersensitivity Pneumonitis

CASE 2

A 30 year old male presented with symptoms of breathlessness for a duration of two months and wheezing of duration one month. As a routine assessment, exposure history was obtained that involved substantial amount of exposure to farm and hay dust along with non-tobacco smoke emissions. The patient also had a family history of asthma.

What is Unclassifiable ILD?

What is the role of Cryobiopsy for Interstitial Lung Diseases?

How to define Progressive Pulmonary Fibrosis?

Notes:

Dr. Suranjan Mukherjee

A 51 years old Indian lady with background of hypertension, was admitted through emergency, 3 years back, with few episodes of cough and hemoptysis. Further investigations revealed she had diffuse alveolar haemorrhage on the right side. Investigations ruled out any systemic vasculitis, however, she developed clinically and physiologically relevant predominantly right sided diffuse parenchymal lung disease (DPLD), also established on imaging. Further workup for a unilateral DPLD revealed severe stenosis of Right pulmonary artery which is an established cause for unilateral DPLD

Questions :

Differential diagnosis of unilateral ILD

CT scan features of ILD due to vascular occlusion

Mechanism for unilateral ILD and pulmonary artery stenosis

Treatment options

Notes:

COPD: varieties in real life

Dr. Parthasarathi Bhattacharyya

Case 1:

62 years non-smoker male presented with h/o cough+ progressive SOB for 10 years. There were episodes of exacerbation in the preceding 2- 3 years year. Historically, there is allergic (seasonal) rhinitis for over 40 years.

Clinically : the general condition was stable. There were occasional wheeze in both the lung fields (+), Investigations: routine hematology was normal and the routine blood biochemistry (sugar, creatinine, and uric acid) was showing no abnormality.

The spirometry report goes as:

	Pre BD	Post BD
FEV1/FVC	66%	65%
FVC	2.05	2.32 (59%)
FEV1	1.36	1.50 (48%)
FEF25-75	0.79	0.79 (20%)

FENO =34 ppb, CxR (PA)-WNL

The patient's old record revealed FEV1 reversibility 1.09-1.68 lit in 2017 with serum IgE 1390 kU/L, Aspergillus-specific IgE as 4.78 IU/ml..

Case 2:

A video play

68 years old ex-smoker is severely short of breath with occasional cough and no expectoration. The accessory muscles are prominent, there is sure intercostal suction. The spirometry revealed:

	Pre BD	Post BD
FEV1/FVC	21.2%	20.10%
FVC	1.75	1.81
FEV1	0.37	0.36 (19%)
FEF25-75	0.13	0.14

CXR (MA) and HRCT: COPD with centrilobular + paraseptal emphysema

Case 3:

48 years old male presented with SOB for 8 years (presently MRC-III for 1 year). He has history of treatment for tuberculosis (irregular and inadequate treatment) in 2013. Looked dyspnoic and malnourished. In 2 chair test, the Pulse rate was 93 → 104 and SpO2 was 98 →97.

HRCT chest (2022) and 2013 are available.

Notes:

Presenter : **Dr. Pawan Kumar Singh**

A case of Lung Cancer

Dr Pawan Singh
Associate Professor, In-Charge Thoracic Oncology Clinic
D.M. (Pulmonary & Critical Care Medicine)
Pt BDS PGIMS, Rohtak

Case

- Mrs VK
- R/o Bengaluru, Karnataka
- 72 Yr Old Lady
- Homemaker
- Never Smoker
- Known case of Type II Diabetes Mellitus
- Presentation:
  - Progressive Dyspnea for 2 months
  - Chest Pain of left side for 2 months

History of Presenting Illness

- Progressive Dyspnea
  - Insidious in Onset
  - Progressed from mMRCI to IV of one and half months
  - Increases on lying on right lateral position
  - Not associated with any seasonal or diurnal variation
  - No Wheeze or cough
- Left Side Chest pain
  - Insidius onset
  - Dull aching
  - Largely non progressive
  - Increases on deep inspiration and coughing

Course of illness

- Underwent chest radiography
- Told to have fluid on left side of chest
- Underwent drainage
  - Straw coloured
  - Drained 500 ml on two occasions
  - Had partial relief in dyspnoea after drainage
- Started on Antitubercular therapy for 15 days
- After initiation of ATT she has increased dyspnoea with frequent Vomiting

Additional clinical data

History

- History of past illness: Nothing contributory
- Family History: Nothing contributory
- Personal History: Loss of appetite and weight, Sleep disturbed for last 15 days

Clinical Examination

- Vitals-
  - PR: 104/min, RR- 24/min, BP- 110/70mmHg, Temp-100°F
- General Physical Examination-
  - Cachexic, Pale, Icterus present, No clubbing, Pedal oedema present
- Respiratory System-
  - Decreased movement and air entry on left side
- Other systems-
  - No hepatosplenomegaly or any other focal sign

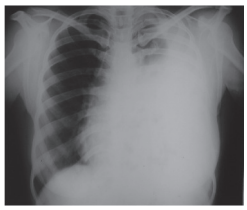
Discussion

- Provisional Diagnosis?
  - Left Side Pleural Effusion
- Aetiology?
  - Tubercular?
  - Malignant?
  - Heart Failure?
- Anything else?
  - ATT induced Hepatitis
- Next Plan of Action?
  - Chest Radiography
  - Pleural fluid Tap
  - Stop ATT?
  - Modify ATT?
  - Computed Tomography of thorax

Investigations

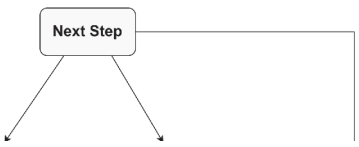
Routine Investigations

Haematology		LFT		RFT		Viral Markers	
Hb	9.8	Bilirubin	3.5	Urea	54	HIV	Neg.
TLC	12000	OT/PT	984 / 754	Creat.	1.0	HBsAg	Positive
DLC	70/28	ALP	350	Uric Acid	2.8	Anti-HCV	Neg.
Platelets	2.9L/mm³	S.Prot / Albumin	5.9/ 2.8				



Next Step	Minimum Investigations	Reports-
• Pleural Fluid Tap (Diagnostic + Therapeutic)	• Pleural fluid TC/DC • Protein Sugar • ADA • Malignant Cytology • AFB (?) • CBNAAT (?)	• TC/DC- 100/L90P10 • Prot/Sugar- 5.1/90 • ADA- 27 • Malig. Cytology- Positive for Adenocarcinoma

Final diagnosis:
Malignant left pleural effusion



In Modern Oncology- Tissue Holds All the Power

Localization

- Malignant pleural effusion has 4 main origins
  - Breast
  - Cervical Cancer
  - Ovarian Cancer
  - Lung Cancer
- IHC Needs to precede PET/CT

Testing of Genetic Alterations

- Which Ones are the bare minimum
  - EGFR
  - ALK
  - ROS1
  - *Any thing else?*

When do you Push Harder?

- Females
- Never Smokers
- Young patients
- Adenocarcinoma Histology
- Asian or South Indian Residence
- NGS Done on histopathology

Why so aggressiv

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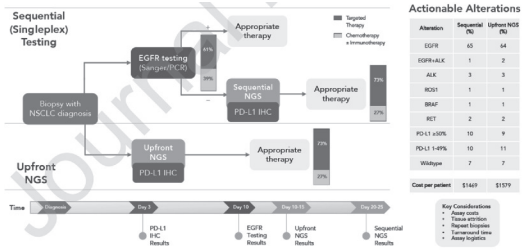
CASE REPORTS

Unearthing *EGFR* Mutations and the Rewards of Persistence in Precision Oncology: Breaching the 10-Year Survival Barrier in Metastatic NSCLC With Active Disease

Check for updates

Pawan Kumar Singh, MD, DM[1]; Rajender Kumar, MD[2]; Amanjit Bal, MD[3]; Nalini Gupta, MD[4]; Rakesh Kapoor, MD[5]; Kuruswamy Thurai Prasad, MD, DM[1]; and ...

Show More



Actionable Alterations

Alteration	Sequential (%)	Upfront NGS (%)
EGFR	65	64
EGFR+ALK	1	2
ALK	3	3
ROS1	1	1
BRAF	1	1
RET	2	2
PD-L1 ≥50%	10	9
PD-L1 1-49%	10	11
Wildtype	7	7
Cost per patient	$1469	$1579

Key Considerations
- Assay costs
- Tissue attrition
- Repeat biopsies
- Turnaround time
- Assay logistics

Key Question 1: Which new genes should be tested for lung cancer patients?

1. *ROS1* testing must be performed on all lung adenocarcinoma patients, irrespective of clinical characteristics.	Strong recommendation
2. ROS1 IHC may be used as a screening test in lung adenocarcinoma patients; however, positive ROS1 IHC results should be confirmed by a molecular or cytogenetic method.	Expert consensus opinion
3. *BRAF* molecular testing is currently not indicated as a routine stand-alone assay outside the context of a clinical trial. It is appropriate to include *BRAF* as part of larger testing panels performed either initially or when routine *EGFR*, *ALK*, and *ROS1* testing are negative.	Expert consensus opinion
4. *RET* molecular testing is not recommended as a routine stand-alone assay outside the context of a clinical trial. It is appropriate to include *RET* as part of larger testing panels performed either initially or when routine *EGFR*, *ALK*, and *ROS1* testing are negative.	Expert consensus opinion
5. *ERBB2* (*HER2*) molecular testing is not indicated as a routine stand-alone assay outside the context of a clinical trial. It is appropriate to include *ERBB2* (*HER2*) mutation analysis as part of a larger testing panel performed either initially or when routine *EGFR*, *ALK*, and *ROS1* testing are negative.	Expert consensus opinion
6. *KRAS* molecular testing is not indicated as a routine stand-alone assay as a sole determinant of targeted therapy. It is appropriate to include *KRAS* as part of larger testing panels performed either initially or when routine *EGFR*, *ALK*, and *ROS1* testing are negative.	Expert consensus opinion
7. *MET* molecular testing is not indicated as a routine stand-alone assay outside the context of a clinical trial. It is appropriate to include *MET* as part of larger testing panels performed either initially or when routine *EGFR*, *ALK*, and *ROS1* testing are negative.	Expert consensus opinion

Key Question 2: What methods should be used to perform molecular testing?

8. IHC is an equivalent alternative to FISH for ALK testing.	Recommendation
9. Multiplexed genetic sequencing panels are preferred over multiple single-gene tests to identify other treatment options beyond *EGFR*, *ALK*, and *ROS1*.	Expert consensus opinion
10. Laboratories should ensure test results that are unexpected, discordant, equivocal, or otherwise of low confidence are confirmed or resolved using an alternative method or sample.	Expert consensus opinion

Course of disease.

- Medical Thoracoscopy with pleural biopsy and Talc pleurodesis in the same sitting
- TTF-1 Positive Adenocarcinoma
- Upfront NGS on tissue
- BRAF v600 Positive

Course of illness

- Dabrefenib + Trematinib Combination was not available in India
- Applied for compassionate access program
- Meanwhile started on Carboplatin + Pemetrexed + Bevacizumab

Response

- Images

Disease is treated with PubMed… Not the Patient

ITR filing 2022: Don't have Form 16? Know how to file Income Tax Return without it

HOME / HEALTHCARE / PHARMA HEALTHCARE / Novartis Tafinlar Mekinist Receives Fda Approval For

Novartis Tafinlar + Mekinist receives FDA approval for treating unresectable solid tumours

According to the company's press statement, Tafinlar + Mekinist is the first and only BRAF/MEK inhibitor to be approved with a tumor-agnostic indication for solid tumors carrying the BRAF V600E mutation, which drives tumor growth in more than 20 different tumor types.

Written by Health Desk

June 23, 2022 8:45:32 pm

Novartis Mekinist Trametinib 2mg Film Coated Tablets
₹ 30,000 / Get Latest Price

Brand	Novartis
Unit	Pack
Prescription required	Prescription
Dosage	2 mg
Basic Name	Trametinib

Tafinlar (Dabrafenib)
₹ 18,000/ Get Latest Price

Total Monthly Cost for therapy- Rs. 3,60,000

Therapy is more than the cost of life…

- Restarted on Chemotherapy (Carb + Pem + Beva)
- After total 6 cycles and multiple Aes- Achieved PR
- Continued on Maintenance Pem + Beva
- Sustained PR

Take Home Points

- Never assume a diagnosis… Always Prove.
- Lung cancer is common even in non-smokers.
- Diagnosis has multiple stage-
  Provisional -> Image based -> Histopathological -> Molecular
- Treat the person not the illness
- Optimize referrals.

Notes:

A typical case of severe asthma

Angira Dasgupta

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BR Singh Hospital, Eastern Railways

KG, a 57 year old gentleman presented to our severe asthma clinic with breathlessness on minimal exertion. He also complained of having frequent exacerbations and at least one hospitalisation every year. According to his family doctor, his exacerbations were mostly related to infections being associated with a high total cell count. His CT scan of thorax showed cystic bronchiectasis which possibly was a result of long standing severe airway disease.

He worked in one of the diesel sheds of a railway workshop which led him to get daily exposure to toxic fumes. He was on treatment with combination inhalers as per GINA guidelines for the past 10 years with short bursts of oral corticosteroids and antibiotics during the exacerbations.

His lung function was extremely poor with a FEV1 and FVC of 0.64 (23%) and 1.74 (50%) respectively (Table 1). Blood reports showed eosinophilia (Absolute Eosinophil Count >300) on 2 occasions in past 3 months (Table 2). He had to be on oral corticosteroids for >2 short courses on an average per year.

Thus, he had already reached the maximum dose of drugs (GINA Step5) while he continued to have frequent exacerbations. The logical next step was phenotyping his disease accurately. It is evident from the blood reports that he had blood eosinophilia which is a nonspecific indirect tool for estimation of the airway inflammation. He would thus require sputum quantitative assay to measure the nature of inflammation in his airways. The test was performed and showed eosinophils to be 3% of total cell count of 6.7 million calls/g of sputum. He was thus given Benralizumab which is an anti-IL5R and an anti-eosinophilic agent. His FEV1 did not improve much; but his FVC improved as the eosinophil count in blood came down.

QUESTIONS FOR DISCUSSION

1. What is the interpretation of the spirometry values?
2. How do we define eosinophilia in various diseases?
3. Name some diseases where we find blood eosinophilia along with asthma
4. What are the different phenotypes of severe asthma? Why is it important to phenotype severe asthma patients?
5. Which tests are done to identify an accurate endo-phenotype in a particular patient?

Table 1 : Spirometry reports; Fonts in red shows reports of post-Benralizumab period

Date	FEV1 in litres	FEV1 %	FVC in litres	FVC%	FEV1/FVC
18-08-2021	0.64	23	1.74	50	0.37
05-10-2021	0.64	23	1.74	50	0.37
22-12-2021	0.78	29	2.26	66	0.35
24-01-2022	0.67	25	2.06	60	0.33
28-03-2022	0.6	22	1.8	53	0.33
22-08-2022	0.65	24	2.18	64	0.29

Table 2 : Shows blood cell counts and total serum immunoglobulin

Date	Total cell count/cu mm	Eosinophil%	Neutrophil%	Immunoglobulin E IU/l
26-12-2004	9600	12	50	
09-02-2011	11700	4	85	
05-12-2013	6,800	3	80	427
30-08-2021	9000	4	82	
12-07-2022	6700	3	56	
01-08-2022	11100	1	87	398

Notes:

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Notes:

Scientific Abstracts and Case Reports

Is Non-smoker COPD similar to Smoker COPD based on their oxidative damage?

Authors : Debkanya Dey1, Sayoni Sengupta1, Sikta Mukherjee1, Debraj Roy2, Dipanjan Saha1, Shuvam Ghosh1, Sayanti Karmakar1, Srijita Sen1, Rajat Banerjee3, Parthasarathi Bhattacharyya4

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Background : COPD, till recently known to be a smoking induced disease, is found near equally prevalent in non-smokers in India. The disease ensues from oxidative damage with other mechanisms and smoking has been a known mechanism of to cause oxidative damage of lungs in COPD. There is very little information available as regards the same pathophysiology in non-smokers.

Materials and methods : Consecutive COPD and 'healthy' subjects (normal spirometry and chest x-ray) were recruited on consent from our out-patient-department. Their serum been isolated following phlebotomy was used to measure the total oxidant status and total antioxidant statusby Rel Assay kits observing the manufacturer's protocol to determine the oxidative stress index. GraphPad prism ver. 8 was used for statistical analysis.

Results : We had 60 patients (30 smoker/ex-smoker and 30 non-smoker) along with 29 normal subjects (12 smokers and 17 non-smokers) for comparison. The results revealed that non-smoker COPD has equally severe oxidative damage as compared to smoker COPD. The COPD (smoker and non-smoker) subjects have significantlyhigher ($p < 0.01$) total oxidant status compared to the 'healthy' subgroup. Non-smoker COPD has significantly lower ($p<0.05$)antioxidant status as compared to other groups. Oxidative stress index for smoker and non-smoker COPD were similar and they were significantly higher ($p<0.01$) than 'healthy' subjects.

Conclusion : It appears that non-smoker COPD subjects are exposed to similar noxious agents as smoking for development of COPD and excessive oxidative stress leadscontributes to the pathogenesis of the disease in them.

Childhood factors important for predicting COPD and asthma in adulthood

Authors : Debkanya Dey1, Dipanjan Saha1, Sayoni Sengupta1, Sahidul Islam2, Mintu Paul2, Ratna Dey2, Malobika Ghosh2, Madan Sharma2, Iti Dutta2, Rana Dey2, Rajat Banerjee3, Parthasarathi Bhattacharyya4

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Background and Aim : Childhood exposures and diseases play an important role in determination of lung diseases in adulthood. Thus, it may be important to look into the childhood events in order to predict the possibility of obstructive lung diseases. The aim of the study is to find out specific childhood events that is associated with presence of asthma or COPD selectively.

Methods : In a detailed questionnaire on history of childhood(age below 8 years), eventssuch as various exposures and diseaseswere recorded at the point of performing spirometry following presentation to our out-patient department. The diagnosis of obstructive lung diseaseswas made; COPD: FEV1/FVC<0.7, and asthma with post-bronchodialator reversibility of FEV1 ≥200ml and ≥12%). The odds ratio of each question was evaluated in both diseases in univariate and multivariate analysis.

Results : The odds ratio for asthma is found to be higher with history of childhood asthma ($p<0.0001$), history of hospitalisation ($p<0.0001$), history of recurrent antibiotic usage ($p<0.0001$), eczema ($p<0.05$), long term wheezing ($p<0.001$), long term sneezing and running nose ($p<0.01$), recurrent purulent expectoration ($p<0.05$), recurrent emergency visit to doctors ($p<0.05$) in childhood. Chances of COPD were higher if a person had a history of tuberculosis as a child ($p<0.05$). There was a considerable overlap region between the two diseases in multivariate analysis.

Conclusion : The identification of the childhood risk factors associated with COPD and asthma needs to be noted in clinical practice. Further research is needed in this field.

Extending the scope of identification of obstructive airway disease in Diffuse Parenchymal Lung Disease: Role of FEF25-75

Authors : Sayanti Karmakar 1, Debkanya Dey1 , Sayoni Sengupta1, Srijita Sen1 , Shuvam Ghosh1 , Dipanjan Saha 2, Avishek Kar3 , Parthasarathi Bhattacharyya3.

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Background : Concomitant OAD (obstructive airway disease) in Diffuse Parenchymal Lung Disease often goes unnoticed on using the GOLD recommended criteria of airflow limitations (FEV1/FVC<0.7). A FEF25-75 (%-predicted) cut-off of 45.5 is seen to differentiate OAD in DPLD with high accuracy. Hence, identification of OLD in DPLD with FEF25-75 is worthwhile.

Methodology : We considered OAD in a) normal FEV1/FVC ratio (absolute value) with FEV1 reversibility >100ml or with disproportionate reduction of FEF25-75 (>25 in % predicted value) or b) the absolute value of FEV1/FVC ratio

being less than its predicted lower limit of normal (LLN) to identify OLD missed by GOLD defined criteria. Such spirometric subsets were tested for presence of OAD applying the cut-off value of FEF25-75 as 45.5.

Results : Out of 165 DPLD patients, OAD was obvious in 14.54% [(n=24) with FEV1/FVC ratio <0.7]. Out of the rest, (n=140), 26 patients (18.57%) had normal FEV1/FVC satisfying other OAD criteria [5(3.57%) having FEV1 reversibility >100ml and 21(15%) having disproportionate reduction FEF25-75]. 4 patients (2.42%) had their FEV1/FVC ratio more than 0.7 but less than their LLN values. All these 30 patients [26+4] eventually had their FEF25-75 (%-predicted) values lower than that of the cut-off. Thus, using both GOLD and FEF25-75 criteria together could identify 54 subjects (32.72%) with OAD while GOLD criteria could only diagnose only 24 of them (14.54%).

Conclusion : Incorporation of FEF25-75 criteria can help to unearth significant proportion of OAD admixed with DPLD, which could have stayed undetected with GOLD criteria alone. The observation demands further research.

Evaluation of spirometric parameters for identification of concomitant obstruction in patients with diffuse parenchymal lung diseases.

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Background: Obstructive airway disease(OAD) and diffuse parenchymal lung diseases(DPLD) often co-exist. It is important to identify the situation to offer the best possible treatment.

Method : The study aims at envisaging the role of FEF25-75 in identifying airflow obstruction in patients with DPLD diagnosed on High Resolution Computed Tomography(HRCT) features. The selected subjects were subjected to spirometry and the GOLD recommendation of FEV1/FVC: <0.7 was used to identify concomitant airflow obstruction. Thus, diagnosed unmixed DPLD and DPLD with OAD were compared based on the other available spirometric variables as FVC (forced vital capacity), FEV1 (forced expiratory volume at first 1sec), FEV1/FVC and FEF25-75(forced expiratory flow at 25-75% of vital capacity). ROC curves were drawn using FEF25-75 and the best cut-off value for differentiation between the two groups were identified.

Results : The GOLD criteria for OAD could identify 23 (14.11%) [male: female - 19:4] out of 163 subjects keeping unmixed DPLD as 85.89% (n=140; male: female as 69:70). The mean %-predicted FEF25-75 was found to be significantly higher in pure DPLD than those with DPLD plus OAD (68.45 ± 36.5 vs. 28.26 ± 29.2, p<0.0001). The ROC curve generated showed a cut-off value of 45.5 to differentiate the entities with a sensitivity and specificity of 91.3% and 90% and negative and positive predictive value as 98.4 and 61.1 % respectively.

Conclusion: A male predominance is obvious in DPLD with OAD overlap patients. FEF25-75 alone can effectively predict airflow obstruction in DPLD patient population. Thus, FEF25-75 may be treated as a marker of airflow limitation.

Studying Forced Vital Capacity in Diffuse Parenchymal Lung Disease patients based on High Resolution Computed Tomography (HRCT) derived underlying features

Authors: Sayanti Karmakar1, Mintu Paul 2, Srijita Sen13 , Shuvam Ghosh 1, Sayoni Sengupta1 , Debkanya Dey 1, Dipanjan Saha 3, Pallav Bhattacharyya 4, Parthasarathi Bhattacharyya5

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Background: It will be interesting to see the base-line lung function of patients suffering from Diffuse Parenchymal Lung Disease (DPLD) on a classification done based on their High Resolution Computed Tomography (HRCT) features.

Methodology: A cohort of DPLD subjects chosen serially from our-outpatient department and the HRCT changes were scored in 0 to 5 scale (0: none and 5 meaning maximum possible). The honeycombing, reticulation with traction bronchiectasis were selected as 'fibrotic' changes, while the ground glass opacities(GGO), and mosaic changes were marked 'non-fibrotic' and the presence of both were considered as "mixed" changes. The scoring were done by a pulmonologist and a radiologist independently and the average was considered for statistical calculation The spirometric variable as FVC (forced vital capacity)values (both absolute and % predicted) were compared between the aforesaid study groups.

Results: A total of 114 (fibrotic=19, mixed=65 and non-fibrotic=30) subjects were recruited. The absolute value of FVC(mean±SD) of fibrotic, non-fibrotic and mixed groups at presentation were 1.37±0.5, 1.6±0.7, and 1.89±0.6 liters while their corresponding percentage predicted values were 51.69±13.75%, 56.46±15.91% and 66.05±16.65% respectively. The non-fibrotic group had significantly higher FVC (p=0.002) compared to the mixed group at presentation in absolute values. As regards the percentage predicted values the mixed group had higher FVC compared to non-fibrotics(p=0.02) and fibrotics(p=0.01).

Conclusion: DPLD patients present late at our OPD services. The Forced vital capacity of fibrotic group is lowest at presentation followed by non-fibrotic and the mixed group.

Specific sputum microbiome signatures were identifiedin airway diseases

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Background : Metagenomics is implemented to understand microbial taxonomic and functional elements of different microbiomes.Multiple metagenomic studies have showndisruption or shift of gut microbiomeas a key player in pulmonarydisease pathobiology. Few human airway metagenomic studies were performed specific to single disease.Theiroutcomes are not comparable across different diseases because of different sample types, sequencing platforms and bioinformatics analysis.

Method : Therefore, we have re-analysedwhole metagenome sequencing dataof sputum samples from5 different airway diseases namely;asthma, bronchiectasis, cystic fibrosis, chronic obstructive pulmonary diseases (COPD), tuberculosis and healthy smokers as control using a common pipeline. We have used Kraken2 for taxonomic assignments and HUMAnN2 for profiling the microbial gene families and functional pathways.

Results : The taxonomic analyses showed 5 bacterial phyla: Proteobacteria, Fusobacterium, Firmicutes, Bacteroidetes and Actinobacteria, present at different ratios across the groups. Bacterial pathogens,Haemophilus influenzae and Streptococcus pneumoniae were found as residents of the airway microbiome.The alpha and beta diversityshowed distinct sputum microbiome traits in asthma, cystic fibrosis, and COPD. The differential analyses of the microbial composition indicated species markers for each group such as Achromobacterxylosoxidans as marker for cystic fibrosis.The functional analyses revealed disease associated microbial pathway(s) and metabolites. Along with bacteria, fungi such as Aspergillus fumigatus, and viruses such as Roseolovirus human betaherpesvirus 7 were detected in the sputum microbiome of COPD samples.

Conclusion : In summary, microbial species, along with the microbial gene families, pathways and metabolites were identified as markers for diseases which can be explored for diagnosis and therapy.

PulmoPred : A web application for spirometry-based classification of obstructive and non-obstructive pulmonary diseases using machine learning

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Background : The majority of pulmonary diseases fall into two categories: obstructive and non-obstructive. There is a significant overlap among the symptoms of these categories that makes the early diagnosis difficult. Further investigation, such as spirometry, is often required for the diagnosis. So, we developed an automated, web-based tool with features from spirometry using machine learning (ML) to perform this class-specific diagnosis.

Methods : ML models were developed with a supervised learning approach using four algorithms - Support Vector Machine (SVM), Random Forest (RF), Naive Bayes (NB) and Multi Layer Perceptron (MLP). The spirometry data used for the training and testing was obtained from IPCR, Kolkata. The models were trained on the data of 1163 patients using 5-fold cross-validation (CV) and further validated with a blind dataset of 151 patients. A web application was developed that uses the optimal model for the prediction (http://dibresources.jcbose.ac.in/ssaha4/pulmopred).

Results: The MLP model achieved optimal performance with an accuracy of 83.7% with a 5-fold CV. The models also showed good performance on the blind dataset. The disease-specific prediction of COPD as obstructive, and DPLD as non-obstructive achieved ~90% accuracy. The optimal MLP model was stored in a web server for use in the web application.

Conclusion : We developed ML based models to efficiently classify major lung diseases into obstructive and non-obstructive based on spirometry features. The web application can serve as a user-friendly and efficient tool for clinicians and patients for the early prediction of lung diseases.

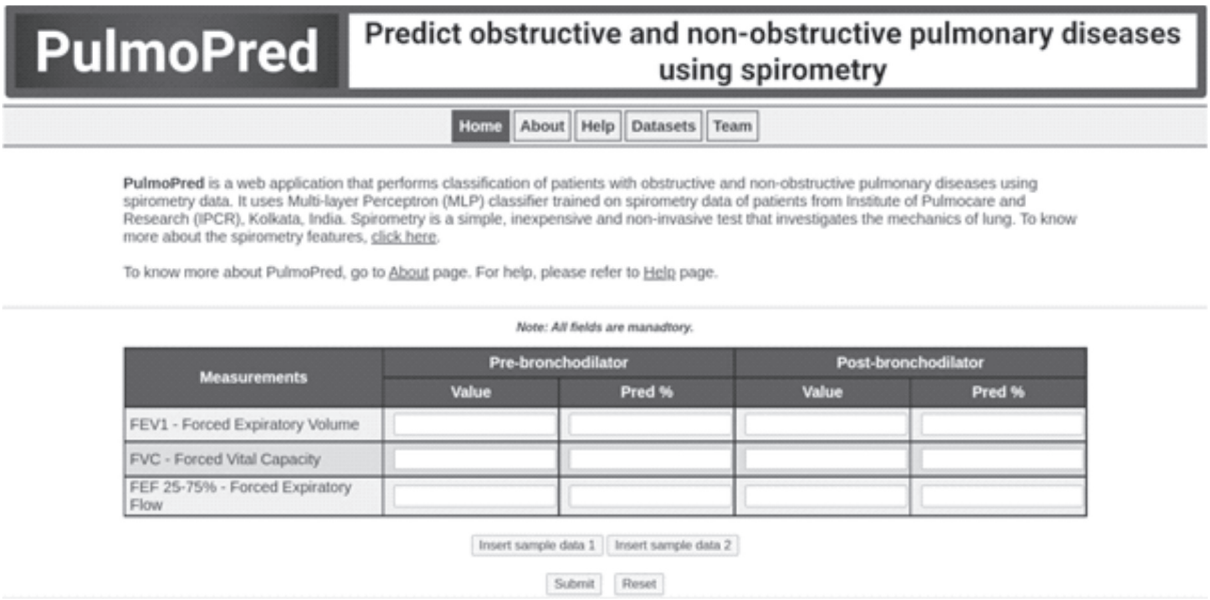

Fig. 1 - Screenshot of the homepage of PulmoPred

Reversibility profile of OAD: a new look

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Background : Bronchodilator responsiveness is an important assessment issue for OAD (Obstructive Airway Disease) patients. Salbutamol inhalation (β2-agonist) is used conventionally. It acts as a measure to differentiate between the common OADs into asthma, COPD and ACOS. Of late, another novel bronchodilator, glycopyrronium reversibility (AMA) is proposed by us.

Methods : We have looked for the serial reversibility of salbutamol and glycopyrronium in successive patients who gave us consent. The responsiveness to these agents were assessed in both absolute value and % change for asthma (FEV1/FVC ≥ 0.7, reversibility ≥ 200ml and 12%), COPD (FEV1/FVC <0.7, reversibility < 200ml and 12%) and ACOS (FEV1/FVC < 0.7 but reversibility ≥200ml and 12%).

Results : Age of the patients were highest in COPD (61.10 ± 11.12) and lowest in asthma patients (41.02 ± 17.54) with ACOS (52.01 ± 14.80)in between. There is a borderline female preponderance in asthma (M:F=24:27) while increasing male preponderance in COPD (M:F=88:32) and ACOS (M:F=48:21).The salbutamol reversibility was lowest for COPD (37.67 ± 66.29 ml), highest in asthma (380.8 ± 148.8 ml) on absolute change but highest in ACOS (29.77 ± 15.14 %)on % change of FEV1. The glycopyrronium reversibility was highest in ACOS (103.4 ± 155.6 ml) followed by COPD (98.23 ± 97.34 ml), asthma (73.85 ± 148.7 ml) in terms of absolute values. In terms of % predicted values, ACOS (37.43 ± 17.60) is highest followed by asthma (26.08 ± 14.60)and COPD (15.57 ± 19.83).

Conclusion : Glycopyrronium reversibility is far better in COPD than asthma but it seems best for ACOS.

Impact of smoking on the lung function variables and reversibility trend to β2-agonists and AMA in Obstructive Airway Disease (OAD)

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Background : Learning of spirometric lung function difference between S-OAD (smoker Obstructive Airway Disease) and NS-OAD (non-smoker OAD) is important. Here, we have tried to look at the difference in spirometric variables and reversibility of all OADs patients based on their smoking status.

Methods : OAD patients selected randomly from out-patient-department were subjected to spirometry and sequential reversibility to salbutamol and glycopyrronium (GP) according to the institutional protocol of IPCR. Statistical analysis were done to see the difference.

Results : Out of 158 S-OAD and 217NS-OAD, the distribution of Asthma, ACOS and COPD were 15 (9.49%), 29 (18.35%) and 62 (39.24%) respectively for smokers and 34 (15.66%), 35 (16.12%) and 56 (25.80%) respectively for non-smokers. The smokerOADwere significantly elder(60.78 ± 12.78 years vs 48.53 ± 16.74 years, p value:<0.0001) with low BMI (23.68 ± 4.398 vs 25.19 ± 4.127, p value: 0.0010) than non-smokers. They had significantly lowerFEV1% (54.70 ± 21.04 vs 63.30 ± 19.89, p value: 0.0002), FEV1/FVC (0.5581 ± 0.1543 vs 0.6633 ± 0.1386, p value:<0.0001) and FEF25-75% (21.82 ± 18.40 vs 33.10 ± 23.18, p value: <0.0001) than NS-OAD.The absolute reversibility to salbutamolwas lower in S-OAD compared to NS-OAD [156.9 ±163.2 Vs 188.0 ± 170.1, p value: 0.0431]. Similarly,reversibility to GP in %-predicted values were significantly lower [18.63 ± 17.22 Vs 24.02 ± 20.66, p value: 0.0134] in S-OADs.

Conclusion : It appears that the overall reversibility [non-specific to clinical entities] to both salbutamol and glycopyrronium is worse in S-OAD than NS-OAD.

Bioinformatics analysis for the screening of gene biomarkers in hypoxic pulmonary hypertension

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Background : Group III pulmonary hypertension (PH) encompasses PH associated with hypoxic lung diseasesincluding chronic obstructive pulmonary disease (COPD).COPD associated PH (COPD-PH), one of the most prevalent forms of PH,is a major burden on the healthcare system. Identification of transcriptomic signatures using bioinformatic analysis opens up the possibility of identifying potential diagnostic molecular markers and understanding disease pathophysiology. The present study aims to explore the changes in the transcriptome and determine the differentially expressed genes (DEGs) in hypoxic vs. normoxic conditions for identification of potential genetic signatures in hypoxic PH. Furthermore, the identified genes are validated in blood samples of COPD-PH patients using qRT-PCR.

Methods : The NCBI GEO database was screened using the keywords 'hypoxic pulmonary hypertension'.The dataset GSE168159containing expression profiles of lncRNAs and mRNAs in hypoxic vs. normoxic adult male C57/BL6 mice(n=24) was selected. DEGs were screened to identify hub genes associated with hypoxic PH. The significantly dysregulated genes were further validated in blood samples obtained from COPD-PH patients in comparison to healthy controls.

Results : Bioinformatic analysis identified 172 DEGs, with 73 upregulated and 99 downregulated genes in hypoxic mice in comparison to normoxic controls. Downstream analysis by cytoHubbaidentified 14 hub genes; CXCL9, CXCL12 and TTN were identified as the most significantly dysregulated genes.The expression levels of threealtered genes in blood samples showed significant dysregulation in COPD-PH patients.

Conclusions : The present study provides an insight intothe disease pathophysiology atthe genetic level and the possible pathways involved.

Comparison of Active Symptoms in COVID-19 patients in the 3 waves: an OPD based observation

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Background : A comparative analysis of the active symptoms experienced by COVID patients in the 1st, 2nd and Omicron waves of the pandemic remains important.

Methodology : Post-COVID patients were enrolled from the out-patient department and they were enquired about their symptoms during the acute phase. The severity index for each symptom were calculated, [i.e., in severity (0-5 scale) multiplied by duration of the symptoms].

Results : We collected response for 78, 222 and 138 post-COVID patients for the 1st, 2nd and omicron waves. It was observed that the severity of cough, throat pain and diarrhoea were similar between the 3 waves.

The 1st wave witnessed the highest score for SOB (27.40±69.42) compared to the 2nd (23.52±62.77) and Omicron wave (8.47±27.14). Body ache score was high in 1st wave (11.17±34.65) when compared to 2nd wave (10.26±28.51) and Omicron wave (9.66±24.80). Expectoration score was worse (10.55±11.66) for the 1st wave when compared to the other waves.

The 2nd wave witnessed the worse score for weakness (83.70±112.37) compared to the 1st wave (63.04±108.04) and Omicron wave (38.29±63.06). Fever, anosmia and loss of taste score were also reportedly worse in 2nd wave (18.95±68.71, 29.72±62.34 and 29.92±65.17) compared to the 1st wave (8.90±13.63, 7.74±21.73 and 10.38±24.08) and Omicron wave (3.34±8.68, 1.81±12.99 and 2.46±13.72).

Inference : SOB, headache, anosmia and loss of taste were reported as prominent active symptoms observed during COVID-19. There was a wide symptomatic variation between the 3 waves of COVID-19. Overall, the 2nd wave was the worst reported.

Comparison of post-COVID-19 patients of different waves: An OPD based appraisal

Authors: Sayoni Sengupta1, Dipanjan Saha1, Debkanya Dey1, Mintu Paul2, Srijita Sen1, Shuvam Ghosh1, Sayanti Karmakar1, Sudip Ghosh 3, Parthasarthi Bhattacharyya4

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Background : A comparative analysis between the 1st, 2nd, and Omicron waves of post covid-19 illness remains important.

Methodology : Post-COVID patients were interviewed regarding the demographic profile, BORG's scale, composite VAS (visual analogue scale: cough, shortness of breath, functional disability, and overall status: the average of all in 0 to 10 scale), and post exercise recovery response (2-Chair Test).

Results : We included 78, 222, and 138 post-covidpatients the 1st,2ndand the Omicron waves. The mean age was 56.17±13.63, 51.31±15.58 and 53.90±16.37 years respectively and the mean gap following the acute illness at presentation were 109.42±88.17, 535.93±4250.43 and 59.92±23.81 days. Hypertension, DM and Hypothyroid were the common co-morbidities and hospitalization rate was 49.27%, 45.94% and 9.42% in the three waves. The BORG and the composite- VAS at the time of presentation were 13.39±9.78, 14±88.17, 16.70±6.21 and 7.35±4.99, 6.19±3.70, 6.75±3.79 for all the groups. Thespirometric lung function status was similar in the three groups. The echocardiography done for the post-covid patients after the 1st and 2nd wave showed low LVGLS(-14.06±1.35 and -15.32±2.86) and low RVGLS (-18.50±8.66) following the 2nd wave. The post-exercise recovery response was compromised in the 1st and 2nd wave (desat max:-2.62±5.84 and -2.22±6.82) but not in the Omicron wave (-1.4±2.29). Cough, SOB and weakness were the prominent post-covid symptoms.

Inference : The 2nd wave remained the worst with high rate of hospitalization and worse overall quality of life in the post-covid period.

Desat-Max in 2-CT based treatment of pulmonary hypertension: an appraisal

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Background : A protocol for treating COPD-PH based on the WHO functional status and desat-max on 2-CT has evolved. It is important to see the clinical responses to treatment on such a protocol.
Methods:The subjects of COPD-PH were diagnosed clinico-radio (HRCT)-echocardiographically. They were screened and selected with desat max≥3% and WHO Class III or IV symptoms, despite providing best possible treatment of COPD including Rehabilitation and Long-Term Oxygen Therapy (LTOT). Further, on consent, they underwent Right Heart Catheterization (RHC) and a fresh echocardiography. The patients were then subjected to vasodilator treatment with Tadalafil. A follow-up was done in a real-world protocol with 2-CT and CAT score.

Results : 17 patients were included in the protocol, where 11 received anti-PH therapy and 6 did not qualify. There was a significant difference in desat max (p-value= 0.02), systolic PAP (p-value= 0.04) and PVR (p-value= 0.014) at the beginning of treatment between the two groups. After a mean follow-up duration of 117 days (117.84±119.30), the treatment group had significant improvement in Desat. Max (-7.1±2.60 to -3.8±3.19, p-value= 0.02) and CAT score (10.09±2.73 to 6.25±2.65, p-value= 0.007). However, the non-treatment group had significant worsening of desat max (-3.37±1.50 to -4.4±2.14, p-value= 0.28) and overall, CAT score (11.5±4.69 to 12±1.85, p-value= 0.78) after a duration of 184 days (184.87±154.43) of follow-up.

Inference : Desat-max based treatment of COPD-PH shows natural selection of sick patients with worse PVR and the treatment response appears to be rewarding in them with vasodilators.

Risk factors associated with mortality in hypersensitivity pneumonitis : a meta-analysis

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Background: Hypersensitivity pneumonitis (HP) related deaths have increased considerably in recent years. Identification of mortality-related risk factors of HP is important to assess disease progression and ensure close patient monitoring.

Methods: Extensive literature search was performed in accordance with the PRISMA guidelines. Literature search of PUBMED, Cochrane Library and EMBASE database between January 2009 and April 2021 using the keywords

"hypersensitivity pneumonitis", "hazard ratio", and "mortality" identified 325 articles. Data of 3152 HP subjects from 22 independent original studies were extracted and assessed.

Results: This systematic review and meta-analysis suggests that advanced age, male sex, honeycombing and traction bronchiectasis patterns on high-resolution computed tomography (HRCT) images are the major mortality-related risk factors of patients with HP. In case of chronic HP, exposure to triggering antigen appeared to be an additional risk factor.

Conclusions: The clinico-radiological risk factors of mortality identified for HP are expected to enable effective prognostication and guide towards precise management decisions. Our observation indicating antigen exposure to be a major mortality-related risk factor for chronic HP suggests that identifying and minimizing/avoiding exposure to triggering antigen is critical to reduce the occurrence of the disease, paving the path for precise diagnosis and management.

Spirometric classification of fibrotic vs non fibrotic DPLD

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Background : The diagnosis of fibrotic and non-fibrotic DPLD can be tried via HRCT chest; but HRCT is unaffordable at times and is not widely available in rural India. Therefore a surrogate for HRCT to understand fibrotic and non-fibrotic DPLD will be worthwhile.

M**ethod :** We selected serial DPLD patients from our outpatient department. Honeycombing and traction bronchiectasis, reticulation with volume retraction were noted as marker of fibrotic DPLD while ground glass opacity, organization and mosaic appearance were noted as non fibrotic DPLD. The two groups were subsequently compared based on FEF25-75 and other derived variables [FVC-FEF25-75, FVC/ FEF25-75 (%) and FVC/FEF25-75 (absolute)] using statistical GraphPAD prism 8.0 and Metaboanalyst5.0 software.

Results : We recruited 182 [fibrotic (n=72) and non-fibrotic (n=111)] ILD patients on HRCT interpretation. On comparison, there is significant difference ($p<0.05$) in FEF25-75 % predicted between the fibrotic [83.50(2.34-111.30)] and non-fibrotic [75.00(51.00-99.00)] DPLD. The variations in the derived parameters of FEF25-75 as FVC-FEF25-75, FVC/ FEF25-75 (%) and FVC/FEF25-75(absolute) are also significantly different (<0.05) between the two groups. The PLSDA analysis also revealed considerable different between the two groups with an overlap region.

Conclusion : The result suggests spirometry can potentially be discriminatory for differentiating fibrotic and non-fibrotic ILD. Further research is needed.

Spirometric comparison of the Th2 and Non-Th2 subtypes in OAD

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Background : Obstructive Airway Disease (OAD) population can be classified into two phenotypes: Th2 and non-Th2 based on the inflammatory markers FENO, absolute eosinophil count (AEC) and IgE. It is important to see the frequency of distribution and the spirometric characteristics of these two important subtypes.

Methods : Serial OAD patientsfrom Outpatient-Department and were subjected to spirometry and classified as COPD or asthma as per salbutamol reversibility of FEV1 (≥200ml and 12% for asthma and <200ml and 12% for COPD). Further, these patients were subjected to FENO, and their serum were used for the measurement of blodd eosinophil count and IgE. Based on the elevation of all three or any two of the three markers, we divided the subjects as "Absolute Th2-high" and "Possible Th2-high" subtypes. Similarly, without elevation of all three or two of the three markers, we had "Absolute Non-Th2-high" and "Possible non-Th2-high" subtypes. Statistical difference between the subtypes has been done.

Results : Theabsolute Th2-high patients were the youngest (45.04 ± 15.30 years) and non-Th2-high patients were the eldest (61.29 ± 14.42 years).The Th2-high subtype (absolute+possible) is more common in asthma (73.12%) than COPD (50.93%) while the non-Th2-high subtype (absolute+possible) is more common in COPD (49.05%) than asthma (26.86%). The Th2-high subtypes had higher BMI (24.82±4.130 vs 23.76±4.000, p=0.07), FEV1% (59.76±18.77 vs 56.90±22.12, p=0.2940), FEF25-75% (26.83±17.86 vs 22.93±19.54, p=0.0173), %reversibility (14.48±13.08 vs 11.12±12.54, p=0.0156) and absolute reversibility (203.3±184.9 vs 124.2±129.3, p=0.0012). For possible-Th2-high and non-Th2-high phenotypes, asthma showed significantly higher BMI, FVC%, FEV1%, FEF25-75%, reversibility (absolute+%) [for both salbutamol and glycopyrronium]than COPD (p<0.05). For absolute-Th2-high and non-Th2-high phenotypes, asthma showed significantly higher values only forsalbutamol reversibility (absolute+%) than COPD (p<0.05).

Conclusion : Th2 based classification of OAD as a whole may help to understand the etiopathological evelopment and can guide the therapeutic approach.

Factors affecting the feasibility of subjects to attend the COPD-rehabilitation program in real world: an appraisal

Authors: Wrick Chakraborty*, Anannya Batabyal, T.D. Viswanathan*, Sneha Biswas**, Dr. Parthasarathi Bhattacharyya*#**

*# consultant, * research assistants, ** Physiotherapist at the department of pulmonary rehabilitation, Institute of Pulmocare and Research, Kolkata

Introduction: Pulmonary rehabilitation (PR) for COPD is not practiced frequently although it is accepted and recommended globally. The standard pulmonary rehabilitation of COPD demands 2-3 visits a week for at least six weeks. We looked for the feasibility of the COPD patients to attend to an out-patient based PR program at the institute.

Materials and methods : The feasibility for attending a COPD rehab was tested on a Google survey through a single research assistant acting face to face to fill up the online response. The questions formed on consensus were on patient's degree of interest in PR, feasibility of comfortable visit frequency, and the major perceived obstacles faced by them to visit the PR program regularly. The responses were recorded in 0-5 scale meaning absence (=0) and maximum possible (=5). Statistical presentation has been done with percent values.

Results : There was a male predominance (67.6%) in the surveyed patients(n=200) with mean age as 60.83. the most feasible options appeared to be monthly visit (34.5%) followed by visits once every 12 weeks (32.5%), and then weekly visits (18-19%). The main obstacles revealed were the time issue and the distance of the patients'residence from the centre followed by lack of time and scarcity of attendants to support the travel to attend the PR program.

Inference: An out-patient based COPD-PR program needs to be individualized as per the number and frequency of visit is concerned. The distance of the residence from the centre is an important obstacle. More operational research is needed in this domain.

Relation of Peak Cough Flow with Spirometry variables

Authors: Wrick Chakraborty*, Anannya Batabyal, T.D. Viswanathan*, Sneha Biswas**, Dr. Parthasarathi Bhattacharyya*#**

*# Consultant, *Research assistants, ** Physiotherapist at the department of pulmonary rehabilitation, Institute of Pulmocare and Research, Kolkata

Introduction: Neuromuscular dysfunction in COPD is common but remains undetected although the impact is deleterious. We looked for Peak Cough Flow (PCF) and compared it with MVV (maximum voluntary ventilation) in spirometry with an idea to see whether the former can act as an surrogate of the other to indicate neuromuscular weakness.

Materials and methods : A cohort of COPD subjects underwent spirometry with MVV and PCF measurements simultaneously by a single trained technician. We compared the PCF with spirometric variables as FVC, FEV1, FEV1/FVC, FEF25-75, 6MWD and CAT score.

Results: We have collected the values of PCF of 38 patients with spirometry. The Peak Cough Flow correlates highly significantly ($p<0.0001$) with post bronchodilator FEV1,FVC, FEF25-75, MVV and also with 6MWD having 'r' values as 0.6777, 0.4625, , 0.4241, 0.6799,0.6932 and R2 value as 4593, 0.2139, 0.1798, 0.4623,0.4805 respectively. PCF showed no significant correlation with CAT score.

Inference: PCF can be a workable surrogate of MVV in resource poor situation. Its role in indicating neuromuscular function or dysfunction needs evaluation.

THE CASES FOR DISCUSSION IN PULMOCON - '22

Case-1

"DERMATOMYOSITIS PRESENTING AS FAMILIAL INTERSTITIAL LUNG DISEASE; AN UNUSUAL CASE REPORT"

Authors **Dr. Soumya Biswas**; 1st year PGT. **Dr. Prashant Kumar**; Associate Professor.
Corresponding Author: **Dr. SOUMYA BISWAS**

Interstitial Lung Doiseases are heterogenous group of progressive disorder characterized by chronic inflammation &/or fibrosis in the lung. While some ILDs can be linked to specific environmental causes,in many individuals no culprit exposure can be identified, these patients are deemed to have Idiopathic Interstitial pneumonia(IIP). Family history is now recognized as the strongest risk factor of IIP. So IIP cases that run in families comprises a syndrome termed as Familial Interstitial Pneumonia.

CASE STUDY : 67 years old gentleman presented to our OPD with-

- Bilateral progressive symmetrical weakness of lower limb and myalgia for 3 years.He was having difficulty in rising from sitting position and climbing stairs.
- Progressive SOB and Dry cough for last 1 year.

Family History- His mother,Elder sister,Younger sister also had the similar presentation and all of them died at the age of 62,54 & 45 years respectively.

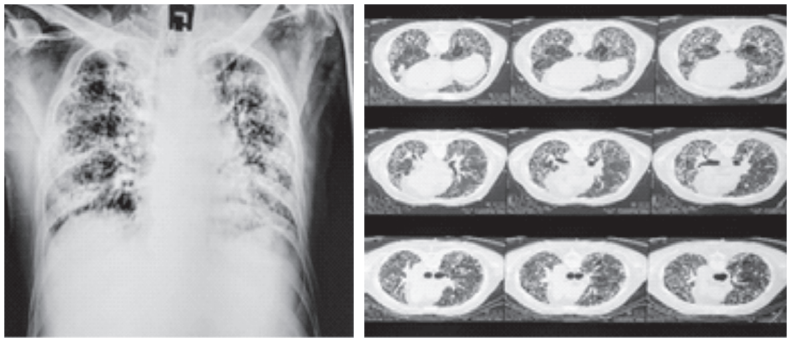
General survey and Systemic examination was unremarkable except

- Skin examination showed Red,scaly lesion over MCP and PIP joints & Reddish-brown discoloration around Eyelids.
- End-inspiratory Crepitations in bilateral Infra-Axillary & Infra-Scapular region which increased on leaning forward.
- Power in B/L lower limb was 3/5.

INVESTIGATIONS :

- Routine investigations were within Normal limit.
- Serum Creatinine Phosphokinase(CPK),CRP was raised.
- Anti MI-2 antibody was Positive but Anti JO-1 antibody was negative.
- RA factor,Anti CCP Antibody and ANA was negative.

- Then Myositis profile was done which showed Anti PL7,PL12 & anti SRP antibody was Positive which confirmed the Diagnosis of Dermatomyositis.
- CXR PA view showed Bilateral Reticular Opacities from apex to base.
- HRCT showed B/L symmetrical fine Reticular opacities,Honey coombing,Traction Bronchiectasis with lower lobe predominance s/o UIP pattern.



DIAGNOSIS : This is a c/o Familial Interstitial Pneumonia associated with Dermatomyositis.

TREATMENT : Now patient is on Nintedanib 150mg BD,Microfenolate Mofetil 500 mg BD and Nebulization Foracort 0.5mg BD.

CONCLUSION :

- Incidence of familial occurrence in IPF is around 25%.
- Incidence of ILD in Inflammatory Myopathy is 5-30%.
- Previously UIP pattern but now NSIP pattern is more common; More common in Females (M:F-3:1).
- Anti MI-2 antibody- Dermatomyositis>>Polymyositis.
- Anti J0-1 antibody- Polymyositis>>Dermatomyositis.
- Prevalance of Anti PL7,PL12 & anti SRP antibodies are 6%,1% & 5% respectively.

THE CASES FOR DISCUSSION IN PULMOCON - '22

Time : 00AM. Date : 00/09/22

Case-2

Concurrent ABPA and Farmer's lung in a 35 year male with previous pulmonary Tuberculosis

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1st Year Resident, IQ City Medical College, Department of TB and Respiratory Medicine
Dr. Prashant Kumar
Associate Proffessor, IQ City Medical College, Department of TB and Respiratory Medicine
Corresponding Author : **Dr. Prashant Kumar**

INTRODUCTION : ABPA occurs as a result of a hypersensivity reaction to germinating fungal spores in the airway wall of Asthma / Cystic Fibrosis patient . Caused by Aspergillus Fumigatus. Farmer's lung is an allergic disease usually caused by breathing in the dust from Moldy hay. Farmer's Lung is a type of Hypersensitivity pneumonitis which is also known as extrinsic allergic alveolitis. It is caused due to results from inhalation exposure to Thermophilic Actinomyces species, M. faeni and occasionally from exposure to various Aspergillus species.

CASE STUDY : 35Y/M presented with shortness of breath , chronic cough and expectoration for last 2 month , cough increasing during night time, with no aggrevating or relieving factor , expectoration was watery in nature with 1 epsisodes of Hemoptysis, he is k/c/o Bronchial asthma since last 5 years and had history of Pulmonary TB 6 month back and was put on ATT . By Occupation he is a farmer.

On general examination all were normal .

On chest examination – B/L Wheeze present in all over the areas.

PFT- Moderate Restrictive Pattern with severe obstructive pattern with reversibility airway disease.

CBC, LFT, RFT, Serum Electrolytes , ECG were Normal.

Serum IgE -18316 KU/L(very high)

Aspergillus Antibodies Panel –

Aspergillus IgG ->200U/ml (High)

Aspergillus IgM- 1.12 U/ml

Aspergillus IgE - 23.00 KUA/L (high)

Farmer's Lung Pannel-

Thermoactinomyces Vulgaris Ig G -31.1mgA/L (HIGH)

Aspergillus Fumigatus Specific IgG- 266 (HIGH)

CXR -Right paracardiac opacity.

HRCT THORAX- 1.Central distribution of bronchietasis

2. Finger in glove sign

3. Signet ring sign

4. Tram –track sign

CONCLUSION : Concurrent ABPA and Farmer's lung. It is very common . But get misdiagnosed due to common presentation as other obstructive airway disease .With all the investigation and serological testing the diagnoses have been made . Plan of action have been emplyoyed to manage the problem . Patient is adviced to follow up .

DISCUSSION :

1. ABPA complicate the course of asthma and cystic fibrosis.
2. Farmer's lung is very common in India but rearely gets diagnosed.
3. Most likely present as acute exacerbation of COPD , Chronic Bronchial Asthma.
4. As CT Thorax and Serology tests are costly and not easily available in lower scioeconomic group of population,so we should increase the awarness and resources for the diagnosis .

THE CASES FOR DISCUSSION IN PULMOCON - '22

Time : 00AM. Date : 00/09/22

Case-3

MYASTHENIA GRAVIS COMPLICATED BY MYASTHENIC CRISIS TREATED WITH ELECTIVE INVASIVEVENTILATION AND PLASMAPHERESIS

Authors **Dr. Promoshi Podder**
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Corresponding Author : **Dr. Prashant Kumar**

Intoduction : ClinicalMyasthenia Gravis is an auto-immune disorder which affects neuromuscular transmission leading to generalized or localized weakness .It is the most common neuromuscular disorder associated with antibody against Ach Receptors in Post synaptic motor end plate. Myasthenic crisis is a complication of Myasthenia Gravis , characterized by muscle weakness which leads to respiratory failure that requires intubation and mechanical ventilation.Investigations36Y, Female who is a k/c/o Myasthenia Gravis since last 2 years, presented at emergency with shortness of breath since morning and productive cough since 1 week along with proximal muscle weakness with drooping of eyelids, jaw weakness , difficulty in chewing and swallowing, loss of finger grip all these features were increasing with exertion and gradually progressive. There was H/O discontinuation of pyridostigmine , prednisolone , Azathioprine since 1 week.

ON EXAMINATION

On General examination- Pallor present, Facial puffiness present, B/L Ptosis of eyelids .

Respiratory system-

Left sided decreased in breath sound in all of the area. Right side crepitation present infra axillary area, infra scapular area. Weakness of accessory group of muscles. Difficulty in counting 20 in single breath.

Neurological examination-1. Tongue fall, 2. Jaw closer weakness, 3. Neck Flexion strength 4/5, 4. Muscle power – RUL, LUL -3/5, RLL, LLL-3/5, 5. Deep and Superficial Reflex were preserved, 6. Poor muscle tone of proximal group muscles, 7. No Balance.

Investigations : Raised TLC with neutrophil leucocytosis. LRT , RFT, ECG, serum electrolytes were normal . Ach R Antibodies present.

Diagnosis36 year, Female K/C/O Myasthenia Gravis since last 2 years with severe respiratory distress precipitated asMyasthenic Crisis due to non compliance to pharmacological therapy .

1. Almost 15-20% Myasthenic patients are affected by myasthenic crisis .

2. Women are twice likely to be affected than men .
3. It has bimodal age distribution .
4. Loss of adherence to pharmacological treatment often leads to myasthenic crisis, which is life threatening .
5. With the availability of better ventilator support , the mortality of myasthenia crisis can be reduced but cost ofmechanical ventilation and plasmapheresis is much higher in tier 2-3 cities.
6. Mortality is around 4%.

THE CASES FOR DISCUSSION IN PULMOCON - '22

Time : 00AM. Date : 00/09/22

Case-4

Unusual aetiology for Breathlessness- Sjogren's Syndrome

Authors **Dr. Vikrant solanki, Dr. Mazher Maqusood,**
Dr. Pradeep nirala, Dr. Abhishek Kumar
Corresponding Author : **Dr. Vikrant solanki**

Intoduction :
Clinical : Sjogren's syndrome (SS) is an autoimmune disease which characterized by lymphocytic infiltration of exocrine glandsresulting in xerostomia and keratoconjunctivitis sicca. Interstitial lung disease (ILD) may be life-threateningcomplications of primary Sjogren's syndrome (Primary Sjogren Syndrome), and has a poor prognosis in terms ofsurvival and quality of life. We present a case of 56 years old Female presented to us with complain graduallyprogressive, multiple joint pain without significant morning stiffness,proximal muscle weakness of hand, low gradeintermittent fever, exertional dyspnea and generalized weakness for past 4 year. She had mild hepato-splenomegalyand bilateral basal end-inspiratory fine crepitations.

Investigations : Blood for rheumatoid factor was positive (22 IU/ml) by ELISA method and also other autoantibodies like antinuclearantibodies, SS-A, SS-B & anti Ro/SSA but Scl-70, ribonucleoprotein, double-stranded deoxyribonucleic acid, JO-1were negative. Schirmer's test and unstimulated saliva secretion were normal as patient is on systemic steroids. RoseBengal test was positive in right eye which denotes breach in the corneal epithelium due to xerophthalmia. Parotidultrasonography showed multiple hypoechoic areas (average 1 mm) in both parotid and submandibularglands.Pulmonary function test revealed restrictive type of lung disease and high-resolution computed tomographythorax showed fine intralobular fibrosis with interstitial thickening of lower zone of both hemithorax without evidence ofcysts suggesting a reticulonodular pattern.

Diagnosis : case of primary Sjogren's syndrome (SS) with interstitial lung disease (ILD) and myositis.

THE CASES FOR DISCUSSION IN PULMOCON - '22

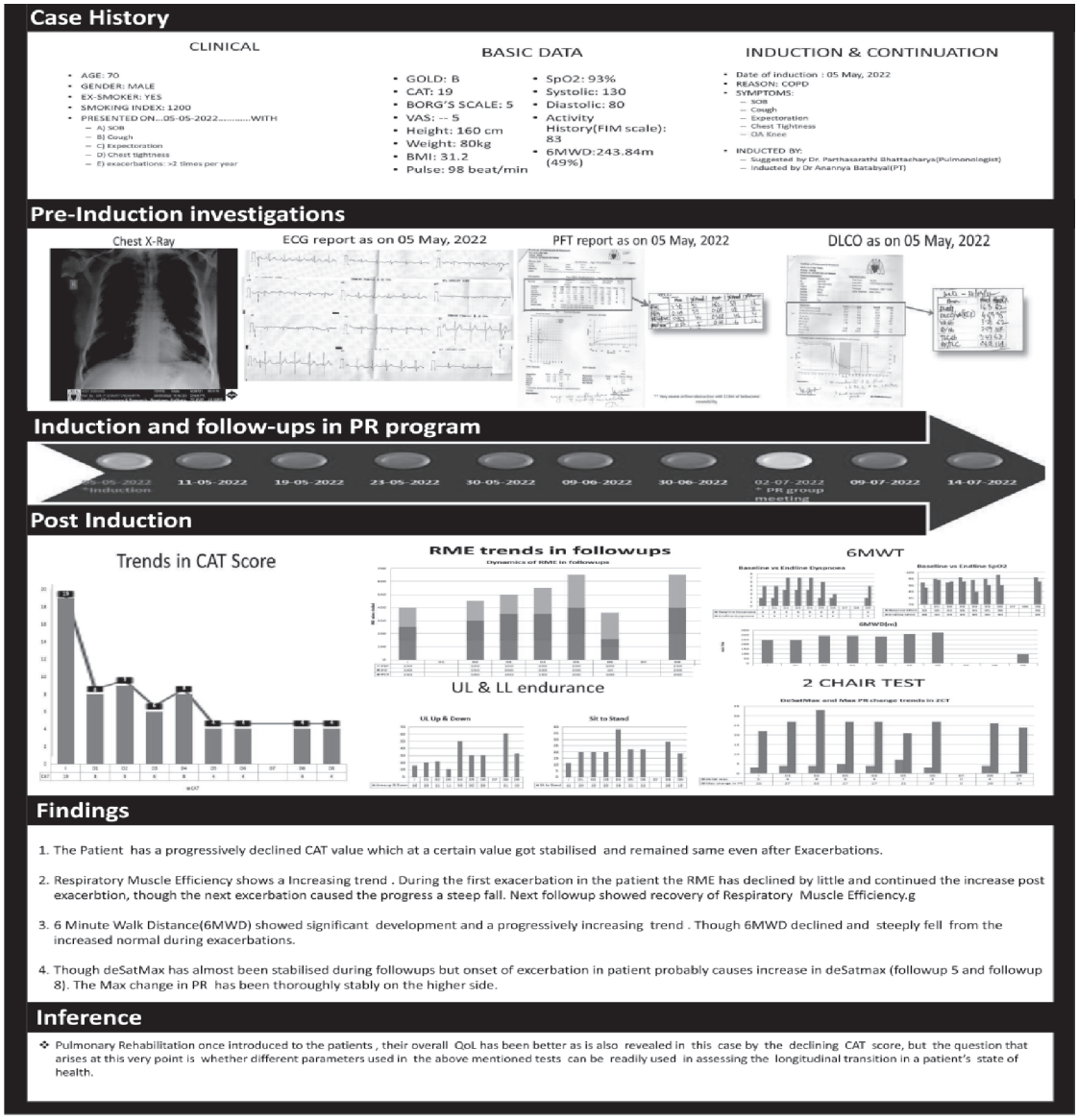
Time : 00AM. Date : 00/09/22

Case-5

Impact of Pulmonary Rehabilitation of COPD with ongoing recurrent exacerbations

Anannya Batabyal, Wrick Chakraborty*, T.D. Viswanathan*, Sneha Biswas**, Dr. Parthasarathi Bhattacharyya***

consultant, * research assistants, ** Physiotherapist at the department of pulmonary rehabilitation, Institute of Pulmocare and Research, Kolkata



A Thoughtful Write-up

Of doctors and the society

Dr Dhiman Ganguly

Quality of health service is the summation effect of efforts (or lack of it) of umpteen number of workers. Doctors are only a cog in the wheels that run the service. A brilliant, conscientious, hard working doctor can hardly make a difference if rest of the system is not optimally functional. Be that as it may, the fact remains that society considers the doctors to be the central figure in the service, and expects him to deliver irrespective of circumstances.

Before we proceed any further, it is important to make two basic facts about life and healthcare, very clear. Firstly, life is inherently uncertain and so is everything else connected with life including medicine, and therefore successfully treating an illness is different from solving a mathematical problem. Julian Huxley (1887-1975)) said "you can predict the Sun, the Moon and the Galaxies, but you cannot predict where the housefly is going to sit next". Medicine, unfortunately for the physician community is a science of probabilities. Secondly, perceived quality of a manufactured product or a service is inversely proportional to expectations. In case of a product (cars, air conditioners etc.) there are some defined expectations, but when it comes to a service like healthcare, expectations are often unrealistic. Buildings, bridges and flyovers are designed and built by engineers – yet when one of these collapse, we need to form an enquiry committee to investigate the cause and at times even after months of meetings and expert opinions a definite cause is not available. A human body is not designed by doctors. neither it is the result of their workmanship, yet they are supposed to do all the maintenance and repair work throughout a lifetime and succeed all the time!!

Doctors would often argue, that there are important causes of poor health service in our country. Government spending on health (and education) is abysmally low, compared to even some of the other developing countries, our hospitals are over-crowded , the basic requirement of public health is lacking in many respects, and huge number of citizens living below the poverty line coupled with lack of education creates the stage for communicable diseases to the whole society. One can go on and on citing deficiencies in the system. The falling rate of infant mortality and rising life expectancy since independence are also cited as example of success of health service.

Leaving aside these arguments and counter arguments – the fact remains that there is ample scope of improvements in the service, and quality of human resources involved is a primary determinant of

quality of service available, irrespective of socio economic constraints and notwithstanding the deficient infrastructure.

The million dollar question is, how to improve the human resources available? More specifically, how to produce "ideal" doctors to start with. Hopefully, a team of ideal doctors will, with the passage of time inspire the full team of health workers to dish out good quality healthcare some time in future. Of all the methods available to the present day society to make people perform, methods like giving them more money (bribing) and scaring them (fear of punishment), might work for sometime, but to make a fundamental difference we need inspiring leadership. There lies the importance of looking at doctors, and if the society is unhappy with the present set of doctors, then there must be something wrong in the way we produce doctors. Doctors are not self-made, they are products of a system. They are allowed to begin their professional life after fulfilling all the conditions laid down by relevant authorities.

The process of producing doctors essentially consists of four steps – eligibility criteria, selection process, education and training.

In our country students appearing at their school leaving examination with physics, chemistry and biology are eligible for selection. In many advanced countries of the world medicine is post graduate degree, consequently students are an older lot, when they begin their medical career. Students from humanities streams are also allowed to pursue medicine. Dr Harold E Varmus, a Nobel Laureate in Medicine who visited Kolkata recently, on the occasion of 125th birth anniversary of Prof. Meghnad Saha, is an example on this point. His graduation in 1962 from Harvard was on English literature. He worked as an apprentice at a mission hospital at Bareilly, Uttar Pradesh in early 60's and decided to study medicine. He completed his MD in 1966 and went on to win the Nobel Prize in medicine in 1989. There are plenty of similar examples elsewhere and the rigid eligibility criteria that we follow is possibly excluding students, who had the potential to become worthy doctors. Some premier medical institutions have experimented with reserving a few seats for graduates in arts subjects. The results have been encouraging, although most of these medical graduates eventually opted to specialize in Psychiatry. Teaching basic physics, chemistry and biology to fresh entrants to a medicine curriculum should not be difficult in these days of animation films and computers.

The next step is the actual selection process. Possible selection tools include school results, admission tests, interviews, multiple mini interviews and personality tests. While selecting candidates for a medical degree, two different attributes need to be looked at. Firstly, the cognitive ability (or potential) to tackle the intellectual burden of the course. This is relatively simple. Secondly, the non-cognitive traits (or potential) to understand and endure the emotional challenges of the professional life of a doctor. This is the more difficult part. Selection tools currently in use, identify who should be selected, rather

than who is more likely to deliver. The problem is that, ability to succeed in a medical course does not necessarily ensure the probability of the individual to become the ideal doctor, society is looking for. In our country, the only selection tool that we are using is the entrance examination called NEET (National Eligibility Cum Entrance Test). This is a multiple choice question test that demands a long and meticulous preparation. India is the only country in the world that admits students in Medicine based only on an examination result. Prospective candidates spend at least a year attending coaching classes, appearing in mock tests and cramming to memory MCQs 16 hours a day, to score a high enough rank, for a seat in a good quality institution. With the increase in the number of seats in recent years, getting a seat in MBBS course is a little easier, but competition remains very stiff for seats in premier institutions. Students failing to secure a seat will often make a second or a third attempt at the selection test. The probability of success in a such a competition depends on (almost robotic) efficiency of memorizing informations. Candidates who are more humane are less likely to do well in this kind of competition. The same procedure is followed for entrance to post graduate medical education too. Hence it is hardly surprising that, there is so much of resentment in the society about doctors' lack of communication skills, their insensitivity to life situations and emotions of patients and their families. While it is also true that emotion driven people are difficult to communicate with, and they have problems to follow logical thinking, nevertheless doctors ideally should have the necessary skill to overcome such problems, and logically one cannot demand humanity from robots !!

Multiple mini interviews (MMI) at different stations have been found to be more effective in judging the cognitive and non-cognitive traits we are looking for in the candidates, rather than increasing the number of interviewers.

MMI was first developed in Michale G DE Groote school of medicine in McMaster University in Canada. It applies the principal of OSCE (Objective Structured Clinical Examination). Like OSCE the MMI overcomes the problem of poor test-retest reliability and context specificity where measurement of an attribute in one context does not necessarily transfer to another.

It has been suggested that increasing number of complaints against doctors is secondary to the medical school selection process, which by emphasising academic rigor overlooks personality flawed individuals. There is growing interest in application of personality testing, used in business and commerce careers, for selection in medical education too. Those who are opposed to this kind of testing argue that homogenization of personality traits of doctors is unnecessary. Choice of speciality can be more relevant with different personality traits. Secondly, at the age of 17 years personality has only started developing and it is likely to change, and thirdly, through training candidates can exhibit desired traits, paradoxically selecting more manipulative individuals.

Somewhat similar to personality testing is the thought of widening access to medical education. The stiff competition for entry, the expenses needed to complete the rather prolonged course, the uncertainties associated with the mandatory training period and delay in starting to earn money to help the family, makes this course a difficult choice for certain sections of the society. Ideally, some people would argue that the medical student community should be structurally similar to that of the society they are supposed to serve. One way of ensuring wider access, that is commonly practiced is to institute a quota. This is familiar to all of us, and pros and cons of quota syetem are being debated for more than half a century. The other approach to widening access that is probably more logical, is organisation of special preparation programs through which underrepresented groups in the medical student community can gain skills to compete at the entry process. Widening access should not be seen as solely the domain of technical experts in the selection committee. It requires the medical school to define its values positions, particularly with respect to communities it purports to serve. Where the communities are underserved or disadvantaged there are compelling reasons for schools to select and retain students from those communities. Adopting a programmatic approach to admission for selection may assist in one of the dilemmas in widening access – how to make decision on the complex combinations of merit, equity, fairness and social accountability issues involved in health professional selection plans. Some institutes in USA and Australia have adopted a community outreach and special program approach to recruit candidates from underserved communities, with some conditions. It has some demonstrated success and deserves more attention as a part of selection process.

After eligibility and selection the third aspects that demands a through introspection is the medical education itself, if we like our doctors to be endowed with ample humane qualities. In practice we need much more than scientific informations in the curriculum. It should be made obvious to students from day one that they are not being prepared to be technocrats, they are different from mechanics repairing and maintaining machines. Each individual is different, they are exposed to different situational difficulties in life, and they react differently even to similar circumstances. It is highly likely that a particular student or a trainee doctor may not have any idea of the life circumstances of a patient and his struggle. Leo Tolstoy began his epic novel Anna K Kerenina with this sentence – "All happy families resemble each other, but all unhappy families are unhappy in their own special way". Doctors ideally, should be able to combine scientific knowledge with the understanding of individual patients to arrive at a clinical judgement, as to what might be of benefit with this patient with this particular problem at this point of his /her life. This is a tall order. The emphasis through the last century has been on giving students scientific knowledge and skills. Medicine is looked upon as a "vocational qualification". But this is certainly not enough. The Geneal Medical Council (GMC) in UK in a recent document Tomorrow's Doctors recommended a greater focus on education, as opposed to training. We talk of being "trained"

to be doctors rather than being educated in medicine. Education has a broader perspective and the process of being educated should be valuable as an end in itself, not because it enables someone to do some specific job. Training has a narrow focus. Education, it is important to point out here touches a student more intimately at a personal level, than the training process. If one wants to develop a mind set (rather than the professional skill), study of literature has a far reaching influence. There is a growing interest now on teaching humanities in undergraduate medical education. The medical council of India also, has recently published a document to revise the undergraduate course. It has been called "competency based curriculum for Indian medical graduate", and includes a course called "Attitude, Ethics and Communication (AETCOM) which will run across years". This is supposed to be effective from the year 2019 itself. What could be the possible role of humanities subjects in a medical course? Firstly, works of literature is likely to expose students to difficult life situation which may be completely unknown to them. Art, literature , drama and music in their many forms, are expressions of human creativity, therefore participating in some form of artistic activity even as a spectator or a reader should contribute to complete education. This must be true for all educational activities, but is specially important for budding doctors. John Stuart Mill (1806-1873) said "it is really of importance, not only what men can do, but also manner of men they are, that do it Among the works of men which human life is rightly involved in perfecting and beautifying, the first of importance is man himself". It may be added here that physicians traditionally has been kabiraj in our country, and kabi in this context in not a poet, he is a philosopher – giving the meaning to the term as "king" of philosophers. Education, as Swami Vivekananda said is supposed to manifest our inherent perfection. Secondly, study of humanities exposes students to a kind of "counter culture" to medicine. Medical students suffer from a great deal of complex, they often have the impression of intellectual and moral superiority over other students and people in other profession. Entrance requirements for medicine undoubtedly are amongst the most difficult in the Unversity system, and once in a medical school they get insulated from students in other disciplines , these factors contribute to strengthening their complex. Introduction of humanities subjects will help reduce this isolation, and eventually foster better relationship between doctors and the outside world.

The fourth and the last step in the making of a doctor is the training period. This period is meant to hone their professional skill, which is much more than making a clinical judgement writing out a prescription or performing a surgery. This is the time they are supposed to learn to communicate. Communication is much more than history taking, its about knowing the person, assessing his personality, learning about his anxiety, his phobia, his mistrust of doctors and his expectations. No two individuals on earth are identical (even uniovular twins have different personalities), and therefore past experience does not really help, one has to explore every new patient with a fresh, unbiased mind and keep exploring them

all the time. There is no fixed methodology of communication skill, what really helps is to watch teachers and colleagues handling patients and their family members and develop one's own style of approach. Professional help might give a broad outline, but ultimately it depends on the concerned individual.

It will be very naïve on our part, if we think that the problems that exist between the society and its doctors will disappear by changing the way we are educating and training the undergraduate and post graduate students in medicine, as this is a multidimensional problem, that is unlikely to disappear completely in near future. But there is a definite necessity for fresh ideas and thoughts. Sooner we begin to do that, the better.

I cannot say whether things will get better if we change; what I can say is,

they must change if they are to get better

G C Lichtemburg (1742-1799)

Management of massive hemoptysis

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Massive hemoptysis is life-threatening hemoptysis that causes significant hemodynamic decompensation or respiratory distress, and if left untreated, may turn fatal, due to airway obstruction, respiratory failure, and/or hypotension. There is no consensus on the exact amount of blood loss while defining massive hemoptysis. Blood losses from anything between 100-1000 ml per 24 hours find mention in literature.

Patient presenting with massive hemoptysis is resuscitated immediately along with prompt and concomitant control of airways. This is followed by identification of the source of bleeding and control of haemorrhage by temporizing methods or otherwise as the patient awaits definitive treatment for the underlying etiology. Multidisciplinary team comprising of pulmonologist, anaesthesiologist, interventional radiologist and thoracic surgeon is preferred for assessment of massive hemoptysis in totality and decision-making regarding best treatment practices.

Use of bronchoscopy as a diagnostic and therapeutic modality and interventional radiological techniques has revolutionized the immediate control of bleeding in massive hemoptysis. Radiology and bronchoscopy are complementary approaches and if utilized together, may help in localization of the site of bleeding, identifying the cause of hemoptysis and subsequent treatment. Past few decades have seen major developments in minimally invasive procedures which are much more precise than ever before, are associated with lesser complications and lesser chances of recurrence. Various therapeutic bronchoscopic interventions like direct/ balloon tamponade, endobronchial airway blockade, endobronchial stent tamponade, laser/argon plasma photocoagulation and interventional radiology practices, like bronchial artery embolization have faired much better than surgical interventions for control of haemorrhage in emergencies.

Use of emergency surgery is now limited to only a sub-group of patients who cannot be shifted to bronchoscopy suite because of cardiopulmonary compromise and hemodynamic instability arising out of life threatening haemorrhage. Definitive surgical resections are reserved for patients with recurrence following bronchial artery embolization, necrotizing lung infections such as mycetomas, aspergillomas, diffuse and complex AV malformations, iatrogenic PA rupture, chest trauma etc.

In summary, the key to guarantee the best chances of patient survival with current approach and practices lies in an early activation and coordinated expedient response from a multidisciplinary team.

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