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- MAHATMA GANDHI
On Cover - The famous photo of Mahatma Gandhi seeing Mycobacterium leprae through the microscope for the first time.
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Dear Friends,

Dr. (Mrs) Dalal after serving as chief editor of Hinduja Newsletter has recently relinquished this post and I have been entrusted the responsibility of editing the newsletter. I have formed a new team. The committee deliberated and opined that a change of format was in order. Accordingly the contents have been changed.

Apart from reviews and articles, we have introduced new sections like: My Journey - Hinduja Grand Rounds, Experience -The Best Teacher, Snapshot, Radio-logic, Legal eye, Viewpoint, Academic update etc.

I am honored to have been made the editor and will try my best to do justice. I thank all the editors thus far who have done a wonderful job. I also thank all my editorial team members for their valuable inputs and effort.

I trust the readers will enjoy this new edition.

We also will be happy to have your feedback.

DR. C. BALAKRISHNAN
Consultant Rheumatologist
When I look back to my early years I recognize they were different to those of my colleagues. I was born in London in 1935 and remember starting junior school with a satchel and a gas mask on my back for it was the beginning of the 2nd World War and there was a serious fear of nerve gas attacks.

**DR. FARAM D. DASTUR**
Director, Medical Education & Hospital Quality

My parents had migrated from Bombay some years earlier, my father was a general practitioner in a singly run practice, it was a hard life, with little off time, I remember the bombing of London and later in 1942-43 the ‘Doodle-bugs’ or pilotless planes programmed to ‘cut out’ over their target and cause maximum damage, a forerunner of today’s drones.

Post 1945 I remember my school years as a happy time. I made some good friends, played cricket for the school and became a mentor. Academically I did not show any special aptitude, but chose medicine as it was a respected profession.

The next event in my journey came some two or three years later. I went with a friend to the last day of the yearly Promenade Concerts (Proms) held in July - August at the Albert Hall. The last night is typically attended mainly by students, the mood is lighthearted, somewhat irreverent, and ends with the singing of patriotic songs such as Land of Hope and Glory and Rule Britannia. Suddenly I felt uncomfortable and a shame to be joining in the singing when India had just gained its independence from the yolk of British, colonial rule. Of course I was Anglicized, and an admirer of certain British qualities, but was I one of them? No.

I made two trips to India before coming to stay permanently. The first was on a troop ship post war to ‘meet family’. The second was after passing MBBS when I came overland (except for the English Channel) in a journey lasting seven weeks with a cosmopolitan group, realizing this was a chance I would not get again once I started post-graduation. It was fascinating to see the gradual change in architecture, dress, customs, manners, eating habits, etc as we made our way from West to East. Three sites left an indelible impression on the mind. First, the Dome on the Rock mosque in Jerusalem for its exquisite carvings, Second, The Lost City of Petra in Jordan, now one of the seven wonders of the world. Third of course was the Taj Mahal at Agra. I found it is true when they tell you “travel broadens the mind”, but what they forgot to tell you is that it also loosens the bowels! 

We all got a bout of traveler’s diarrhoea at some stage or other. After arriving in India in 1967 I landed a job in Pune at Jehangir Nursing Home as resident physician. It was a gentle way to get adjusted to Indian medical practice. I marveled at the ease with which consultants shifted from one language to another to interact with patients, quite the opposite to my ‘kicheri’, but people were very understanding.

Another feature was on a matter of etiquette. When the consultant arrived to see the patient relatives would crowd into the room to witness everything. There was no privacy for doctor patient interview unless the consultant insisted on it. And lastly the obnoxious practice we have of carrying out CPR on all patients when the heart stops. It is not appropriate for incurable and frail patients waiting to die, - but our law makers don’t seem to see it that way.

I wanted to be in a teaching hospital and late 1969 I joined the KEM Hospital in Mumbai as a lecturer in Medicine. I spent a total of 17 years at the institution slowly climbing the ladder to become Professor and head of department of Medicine in 1986. In addition to my medicine duties I looked after the Tetanus Unit which was the subject of a number of investigations and publications regarding the disease. I also saw firsthand the power of immunization as the number of tetanus cases fell from 60 to 10 per month over 15 years. Over the years I taught a number of brilliant students who later distinguished themselves in India and abroad. I witnessed regrettably the “ splintering effect” of super specialization where loyalty to institution was slowly transferred to loyalty to one’s specialty only. I witnessed the major exodus of newly qualified doctors to the USA and UK each year. I was caught up in the strikes by junior doctors as they fought for better pay and working conditions, and recently for more security and protection from irate relatives. The Government only saw fit to react to crises, and unable to financially support medical institutions sufficiently to retain staff particularly as a growing private healthcare sector was offering better remuneration and working conditions. I was caught up in this web myself and in 1987 at the age of 51 years earning only Rs. 5500/month and feeling financially insecure, I resigned from KEM Hospital to join the new Hinduja Hospital.

My education has continued during my years at HNH. We realized early the importance of a postgraduate degree to motivate and retain essential JMS and are presently recognized in 24 specialties by the National Board of Examinations in Delhi for the DNB degree. The consultants have also distinguished themselves and today we are certainly one of the top academic institutions in the private sector. I have also learnt how complex the running of a hospital is especially with our ‘not for profit’ commitment. NABH has guided us along the road to accreditation, -the basic certificate of proficiency for an institution. In all of this the Hinduja family and hospital administration has been kind enough to let me participate and contribute at a time when I thought I was fit only for the rocking chair. And so I end with a thank you to all who helped me along ‘My Journey’ and with the following quotation:

*Age is an opportunity no less than youth itself though in another dress,
And as the evening twilight fades away
The sky is filled with stars invisible by day.*

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Hinduja Hospital and Medical Research Centre, Mumbai Status epilepticus is a serious medical emergency in paediatric practice with a potential for significant morbidity and mortality. Almost 30-40 % of status epilepticus is refractory to first and second line treatment and needs coma producing therapy for controlling seizures. A subset of these patients does not respond and are considered to be super-refractory. Treatment of this super-refractory status epilepticus is extremely challenging with very poor literature on effective therapies. In selected cases and at experienced centers, epilepsy surgery could be considered as a therapeutic option once medical management has failed.

Here we describe two children with medically refractory status epilepticus due to lesional pathology who responded well to emergency epilepsy surgery.

**CASE 1**

PP was first referred to the Pediatric Neurology and Epilepsy centre at the P.D. Hinduja National Hospital at 4.5 months of age for neonatal onset drug-resistant epilepsy. The child had a normal birth history. Seizures started from 15 days of life in the form of flexor spasms. Initially these events were infrequent and gradually increased to daily episodes. The jerks responded to ACTH and topiramate. At this time his developmental milestones and examination was normal. An MRI brain showed a definite right, virtually hemispheric cortical dysplasia. (Fig 1) An EEG showed predominantly right hemispheric epileptiform discharges. The patient was subsequently lost to follow up.

At one year of age he returned with progressively increasing spasms since 7 months of age. He was also noted to have significant left hemiparesis. An option of hemispheric surgery was declined by the parents. He responded to the ketogenic diet and was also continued on clobazam, valproic acid and topiramate. However, relentless seizures and worsening drowsiness with inability to feed and subsequent status epilepticus brought him back to P. D. Hinduja hospital for further management. He was in super-refractory SE despite high level com producing therapy. The EEG showed predominantly right sided ictal as well as interictal epileptic discharges and right sided burst suppression pattern (Fig 1).

**Fig. 1** Preoperative EEG showing right hemispheric runs of epileptiform activity - subclinical seizure (a) and predominant right sided burst suppression pattern in sleep (b)
An emergency right hemispherotomy was done after 10 days of uncontrolled status epilepticus. The consensus to perform surgery was arrived at after careful consideration of clinical details during a multi-disciplinary epilepsy surgery meeting and informed parental consent. All fibre tracts including the corpus callosum were disconnected over the right hemisphere. Right temporal polectomy was done and histopathology confirmed the diagnosis of cortical dysplasia. Post-operative course was uneventful. Seizures stopped completely and the child remained seizure free for next 6 months.

Seizure recurred after 6 months due to presence of incomplete disconnection at the temporal stem; hence a further complete disconnection was done. He remains seizure free at 11 months post operative follow up. He is gaining milestones and can stand independently though his left hemiparesis expectedly persists. A recent EEG continues to show right hemispheric epileptic discharges, which is expected as the underlying cortex has been disconnected and not excised.

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**Fig 2.** MRI Brain T2 weighted axial (a) & coronal image (b) showing the right hemispheric dysplasia seeing as fronto - temporal hypermyelination (hypointense white matter - hallmark of dysplasia in infancy

---

**Fig 3.** Pre-operative Brain PET scan showing increased metabolism on the right hemisphere predominantly fronto-temporal regions suggesting ongoing ictal epilepti activity.
AB, a 7 year old boy presented to the Pediatric Neurology clinic at PDHNH with a history of right sided brief, focal motor seizures from 4 years of age following an possible encephalitis like illness in his village 6 months earlier. Recent increase in seizure activity was associated with a Todd’s paralysis. His MRI brain revealed ill-defined patchy T2 hyperintensities in left frontal and peri-insular cortex. He had failed trials of multiple antiepileptic drugs (phenobarbitone, sodium valproate, levetiracetam and clobazam, oxcarbazepine) with no benefit. In March 2015 he presented with worsening seizures which later evolved to a status epilepticus. His VEEG showed clinical as well as subclinical seizures arising from the left temporal region. Repeat MRI brain showed progressive left cerebral atrophy with thinning of the left putamen and ill-defined patchy T2 hyperintensities in the left frontal and peri-insular cortex. This was suggestive of Rasmussen’s encephalitis. He was tried on multiple AEDs with no response. In view of refractory status epilepticus with a left hemispheric onset and possible Rasmussens’ encephalitis, an emergency hemispherotomy was performed after 7 day. Histopathology of resected specimen showed inflammatory changes confirming the diagnosis of Rasmussen’s encephalitis. Seizures stopped completely after surgery and the child remains seizure free at 3 months follow up. He has started speaking and is ambulant.

DISCUSSION
Refractory status epilepticus is a medical emergency with a substantial mortality rate. Despite the fact that it remains an important clinical problem in all neurology centres worldwide, there is a remarkable lack of published data concerning effectiveness of treatment. Concerns with treatment safety and outcome remain.
CONCLUSIONS
Refractory status epilepticus is associated with a high mortality rate and often does not respond to optimal medical treatment. In selected cases and at experienced centres, epilepsy surgery should be considered as a therapeutic option once medical management has failed. Hemispherotomy is a surgical procedure of hemispheric disconnection that has a low rate of complications and seems to be as safe even in infants as demonstrated in our case. A larger number with a longer follow-up will prove to highlight the long-term efficacy in the Indian scenario.

Our experience with hemispheric procedures consisting of 20 cases (1-22 yrs of age) including 9 hemispherotomies and 11 functional hemispherectomies. Complications included one death (our first case who was a 1 year old boy); three cases developed hydrocephalus needing shunting; 1 developed post-operative venous infarction in the unoperated hemisphere. Sixteen patients are seizure free.

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Fig 6. MRI brain showed progressive left cerebral atrophy and ill-defined patchy T2 hyperintensities in left frontal and per i-insular cortex.
A 13 yr old girl presented with cough, shortness of breath, throat pain and intermittent high-grade fever of 2 months duration. Since 20 days the throat pain had worsened and she has also noted flitting joint pains and transient rash. At admission she was unable to open the mouth, speak or swallow. On examination vital signs were normal. There was severe trismus. A necrotic mass was evident in the right tonsillar area with extensive ulceration of the right soft palate and anterior pillar. Vasculitic lesions were present in the hands and feet. Systemic examination was unremarkable.

Investigatory reports were: Hb 7.6 gm/dl, wbc: 8290 / cumm, platelets: 525, ESR: 106 mm/hr, CRP: 192 mg/l, throat swab: negative, urine routine: normal, creatinine: 0.6 mg/dl, LDH: 323 mg/dl, ANA: negative, ANCA: c-ANCA lin20++ + x-ray paranasal sinuses: normal, chest radiograph: normal and high resolution CT chest: trachea normal, few subcentimeter nodules superior segment of left lower lobe.

HPE of the necrotic mass was consistent with Wegener’s granulomatosis (figure 1).

**DISCUSSION**

Wegener’s granulomatosis is a small/medium vessel vasculitis with protean manifestations (1) Head and neck manifestations, particularly in the sino-nasal tract are common and can affect as many as 90% of patients at presentation (2) In contrast oral lesions are less common and include oral ulceration, perforation of the palate, swelling and destruction of the lips (3-6) Although ischemic and necrotic tissue injury is common, tumorous lesions have been described.
Wegener’s granulomatosi presenting as a tonsillar mass

Goulart et al reported 6 patients wherein the presenting manifestations were tissue swellings in the retroperitoneum, mediastinum (two) breast, retro orbital tissue (two) and gingiva (7). The patient described presented with not only necrosis of the soft palate but, also had a necrotic mass in the right faucial area. Although there was possible lung involvement, there was no kidney involvement. The consistent histopathology and the strongly positive ANCA pointed towards the diagnosis.

REFERENCES


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HINDUJA GRAND ROUNDS
PULMONOLOGY DEPARTMENT

CASE 1
A 33 years old male, non-smoker, known case of bronchial asthma and recurrent sinusitis presented to us with complaints of chronic cough with mucoid expectoration and dyspnea on exertion. His serial chest radiographs showed fleeting opacities and blood tests revealed elevated levels of Total IgE (5200U/L: Normal : 0-300 U/L) and Aspergillus specific IgE (5.2 mU/L Normal : 0-0.35 mU/L). A HRCT Chest was done which is displayed below.

WHAT IS THIS RADIOGRAPHIC SIGN CALLED?
This radiographic sign is known as Finger in glove sign. It is the characteristic finger-like appearance of mucous plugs within dilated bronchiectatic central bronchi. It can be observed on CXR and CT.

WHAT IS THE FINAL DIAGNOSIS?
Final diagnosis is Allergic Bronchopulmonary Aspergillosis (ABPA). It is an idiopathic inflammatory lung disease, occurring commonly in patients suffering from asthma or cystic fibrosis, characterized by an allergic inflammatory response to the colonization of aspergillus species in the lung. Repeated episodes of inflammation, mucoid impaction and bronchial obstruction can lead to bronchiectasis, fibrosis, and respiratory compromise. The clinical picture of ABPA is dominated by asthma complicated by recurrent episodes of fever, expectoration of brownish mucus plugs, and hemoptysis. Investigations usually reveal peripheral eosinophilia, raised serum Total IgE and Aspergillus specific IgE and IgG, skin test reactivity to Aspergillus antigens. Treatment is with inhaled and oral corticosteroids combined with a course of antifungals such as itraconazole. We treated the patients with steroids and itraconazole with good clinical and radiological improvement.

CASE 2
A 40 years old housewife presented to us with 8 month history of dyspnea on exertion and occasional dry cough. There was no history of fever, weight loss or loss of appetite. She was treated with empiric Anti-tuberculous medication for 3 months without any improvement. On examination she had a room air saturation of 90% and bilateral basal crackles were present. A HRCT was done which is shown as below.

WHAT IS THIS CT PATTERN KNOWN AS?
This CT pattern is known as Crazy-paving pattern which refers to the appearance of ground-glass opacity with superimposed interlobular septal thickening and intralobular reticular thickening.

WHAT IS THE LIKELY DIAGNOSIS?
The likely diagnosis is Pulmonary alveolar proteinosis (PAP). It is a diffuse lung disease characterized by the accumulation of surfactant phospholipids and proteins in the distal air spaces. It is not associated with inflammation and lung architecture is preserved. Major symptoms of PAP are progressive dyspnea on exertion, cough, weight loss, and low-grade fever. Bronchoalveolar lavage in PAP has an opaque or milky appearance due to abundant lipoproteinaceous material and cytological examination reveals alveolar macrophages engorged with PAS-positive material. Treatment options include whole lung lavage or lung transplantation and other experimental options like exogenous GM-CSF (Granulocyte Monocyte-Colony Stimulating Factor) and plasmapheresis. We did a whole lung lavage for this patient and there was clinical improvement and clearance of radiographic shadows.
CASE 3

A 26 year old female presented to us with six-month history of cough with salty expectoration and chest pain. There was no history of fever, weight loss or loss of appetite. She was started on empiric anti tuberculous therapy but there was no improvement at the end of three months. She had two pet dogs at home. A CT Scan was done which is displayed below.

**WHAT DOES THE CT SCAN SHOW ?**

CT Scan of thorax shows a left side hydatid cyst of lung. The water-lily sign is seen in hydatid disease when there is detachment of the endocyst membrane which results in floating membranes within the pericyst that mimic the appearance of a water lily.

**WHAT IS THE LIKELY DIAGNOSIS?**

Human cystic echinococcosis (CE) / hydatidosis is a dog-borne zoonoses caused by infection with the larval stage of the dwarf tapeworm of the genus *Echinococcus*. It occurs when humans act as an accidental intermediate host and ingest viable eggs, which have been shed in the faeces of the definitive host. The most common symptoms of pulmonary cystic echinococcosis (CE) include cough, chest pain, dyspnea and hemoptysis. Approximately 60% of pulmonary hydatid disease affects the right lung, multiple cysts are common. Approximately 20% of patients with lung cysts also have liver cysts. Complications include cyst rupture, secondary bacterial infections of the cyst.

The current treatment of hydatid disease of the lung is complete excision of the cyst, including the germinative membrane, with the maximum preservation of lung tissue. Adjunctive Albendazole chemotherapy 10 mg/kg/day is prescribed for 4-6 weeks before surgery to sterilise the cyst and for 2 months post-operatively to reduce the recurrence rate. The patient underwent excision of the cyst and also received Albendazole therapy.

CASE 4

A 34 year old female presented with complains of shortness of breath on exertion and cough with occasional hemoptysis since 2 years. She gave history of Generalised tonic clonic seizures three years ago. On examination she had bilateral basal crackles and a soft fluctuating mass in right lumbar region.

**WHAT IS SEEN IN THIS CT CHEST ?**

Multiple thin wall cysts scattered throughout the lung parenchyma.

**WHAT IS THE LIKELY DIAGNOSIS?**

The likely diagnosis is Lymphangioleiomyomatosis (LAM) LAM is a rare lung disease characterized by Cystic destruction and multifocal, nodular proliferation of immature smooth muscle and perivascular epithelioid cells. There is a strong association with the neurocutaneous syndrome known as tuberous sclerosis complex (TSC). Marked female gender predominance is seen. Nearly all patients are symptomatic at presentation, and approximately 70 percent report dyspnea. Potential complications of LAM include pneumothorax, hemoptysis, chylothorax, chylolymphomegaly, and development of lymphangioleiomyomas. Treatment is symptomatic. New drugs like Sirolimus are being tried. Prevention and management of complication is utmost important. We counselled the patient regarding complications, avoidance of pregnancy and also started her on Sirolimus.
**CASE 5**

A 42-year-old diabetic man presented with recurrent episodes of hemoptysis since the past five years. Sputum examination on multiple occasions was negative for tuberculosis. He had a past history of pulmonary tuberculosis eight years back for which he was treated with anti-tuberculous drugs. Chest CT was done which is shown below.

**WHAT IS THIS RADIOGRAPHIC SIGN CALLED?**

The radiographic sign is known as Crescent Sign reflecting the presence of a fungus ball in a parenchymal cavity.

**WHAT IS THE FINAL DIAGNOSIS?**

Aspergillomas are mass-like fungus balls that are typically composed of Aspergillus fumigatus, and are a non-invasive form of pulmonary aspergillosis. Aspergillomas occur in patients with normal immunity but structurally abnormal lungs, with pre-existing cavities, most commonly due to tuberculosis. Most patients of pulmonary aspergilloma are asymptomatic. Symptomatic patient commonly presents with hemoptysis (in 50 to 80% cases, which may be life-threatening). Aspergillomas typically appear as rounded or ovoid soft tissue attenuating masses located in a surrounding cavity and outlined by a crescent of air. Altering the position of the patient usually demonstrates that the mass is mobile, thus confirming the diagnosis. An asymptomatic aspergilloma does not necessarily require treatment, and the cavity is essentially isolated from any systemic administration of antifungals. In the setting of brisk haemoptysis, angiography may be performed on an emergency basis and selective bronchial artery embolisation can be life-saving. Failing this, or in cases of repeated haemoptysis, surgical excision with a lobectomy remains the gold standard. We subjected the patient to left upper lobectomy in view of recurrent hemoptysis. The patient tolerated the procedure well and is free of his symptoms.

---

**CASE 6**

A 40-year-old male presented with symptoms of recurrent episodes of fever and cough with purulent expectoration (half a cup everyday) since the past ten years. He had no past history of tuberculosis or pneumonia. His personal and family history was non-contributory. He had undergone a bronchoscopy which revealed multiple tracheal diverticuli and excessive collapsibility of lumen of trachea, main bronchi and segmental bronchi during expiration and coughing. Tracheal diverticuli biopsy showed loss of elastin fibres with Verhoff’s stain. His CT scan is shown below.

**WHAT IS THIS RADIOGRAPHIC SIGN CALLED?**

The radiographic sign is known as Crescent Sign reflecting the presence of a fungus ball in a parenchymal cavity.

**WHAT IS THE FINAL DIAGNOSIS?**

Aspergillomas are mass-like fungus balls that are typically composed of Aspergillus fumigatus, and are a non-invasive form of pulmonary aspergillosis. Aspergillomas occur in patients with normal immunity but structurally abnormal lungs, with pre-existing cavities, most commonly due to tuberculosis.
Most patients of pulmonary aspergilloma are asymptomatic. Symptomatic patient commonly presents with hemoptysis (in 50 to 80% cases, which may be life-threatening). Aspergillomas typically appear as rounded or ovoid soft tissue attenuating masses located in a surrounding cavity and outlined by a crescent of air. Altering the position of the patient usually demonstrates that the mass is mobile, thus confirming the diagnosis. An asymptomatic aspergilloma does not necessarily require treatment, and the cavity is essentially isolated from any systemic administration of anti-fungals. In the setting of brisk haemoptysis, angiography may be performed on an emergency basis and selective bronchial artery embolisation can be life saving. Failing this, or in cases of repeated haemoptysis surgical excision with a lobectomy remains the gold standard. We subjected the patient to left upper lobectomy in view of recurrent hemoptysis. The patient tolerated the procedure well and is free of his symptoms.

WHAT IS THE DIAGNOSIS?

The diagnosis is Idiopathic Pulmonary Fibrosis (IPF). IPF is a chronic and ultimately fatal disease characterized by a progressive decline in lung function. IPF belongs to a large group of more than 200 lung diseases known as interstitial lung diseases (ILD), characterized by the involvement of lung interstitium. Lung tissue from people with IPF shows a characteristic histopathologic pattern known as usual interstitial pneumonia (UIP). The diagnosis of IPF requires exclusion of other known causes of ILD and the presence of a typical radiological pattern identified through high resolution computed tomography (HRCT). It is generally seen above the age of 60 years and presents with dyspnea on exertion and dry cough. Prognosis is dismal with the 5-year survival for IPF ranges between 20-40%, a mortality rate higher than that of a number of malignancies, including colon cancer, multiple myeloma and bladder cancer. Treatment includes supportive management such as oxygen therapy. Newer drugs such as pirfenidone and nintedanib have been recently approved for use in IPF. The patient was started on long term oxygen therapy and pirfenidone.
Vikas Agashe, consultant orthopaedic surgeon at P.D. Hinduja hospital in Mumbai has been doing dedicated work on “Bone and Joint TB” for well over 2 decades. Since 2009 under the auspices of the Bombay Orthopaedic Society, he has embarked on a long term study of “Non-responders in Bone and Joint TB” which, is funded by them (unpublished data). Till date 89 patients have been enrolled.

Has the behavior of bone and joint TB changed?

The behavior of B&J seems to have changed over the past decade. The most important fact is the emergence of MDR and XDR TB (see below for the definition). The increased prevalence of HIV has also lead to an increase in the number of patients with resistant B&J TB. A large study of HIV patients published in 2004 by Maniar et al noted that 43.5% of HIV patients who developed TB did so in the extra-pulmonary sites. In this study 40% of patients who had cultures done had resistance to at least one drug. There is a fear that over the last decade, children have been exposed to drug resistant strains of mycobacterium tuberculosis at a very early age. This may result in development of primary complex with a multi drug resistant strain. If that is true, drug resistant TB will continue to be a major problem in the future.

What are the clinical features of Bone and Joint TB?

The most common area involved is the spine. The other areas involved are hip, knee and shoulder. Soft tissue lesions are not uncommon. B&J TB can present with local signs, constitutional symptoms or both.

Patients with spinal tb generally present with gradually increasing backache with stiffness.

Collapse of vertebrae may result in gibbus or kyphus deformity. Occasionally some patients present with para paresis. Local pain & swelling of the involved joint is the most common presentation of extra spinal TB. The joint movements get restricted gradually over weeks and months. Generally only one joint is involved. However in non respondent cases the lesion is often multifocal. 34/89 patients in our study had multifocal disease.

The incidence in our study is high as only those who are not responding to anti-tubercular therapy were included.

What are the radiological features of Bone and Joint TB?

PLAIN RADIOLOGY

X-rays although non-specific do help us in the diagnosis of B&J TB. In the spine 5 types of lesions are identified.

1. Para-discal: This is the most common type. Disc space is diminished & irregular. Most often, it is associated with para-spinal soft tissue shadow, signifying soft tissue abscess. Multiple spinal involvements are common. Collapse of vertebrae is well known

2. Central: The body of vertebra is involved. The patient may not have much pain for a long time as there is no involvement of the joint. Occasionally the abscess can burst leading to collapsed vertebra. Neurological involvement is common after the collapse

3. Anterior: The disease spreads under anterior longitudinal ligament and can have extensive spread. In the dorsal or lumbar spine, can lead to smooth scalloping of the anterior aspect of the vertebrae due to pulsations of the aorta

4. Posterior elements: Involvement of only the posterior elements occurs occasionally.

5. True Arthritis in atlanto-axial joint: Seen as either dislocation or increased pre vertebral soft tissue

Tuberculosis of other sites is characterized on plain radiographs by soft tissue swelling, diminished and irregular joint space and erosions.

MRI

MRI changes by themselves are also non-specific but along with the clinical scenario are very helpful. Though MRI is extensively used to monitor the disease in clinical practice, the findings often lag behind and may not represent the true status of the disease.

MRI is very useful to detect early lesions, define the extent of bony involvement, delineate soft tissue abscesses and detect skip lesions. It also helps in planning the tissue diagnosis especially of deep-seated lesions
Important MRI changes that help in the diagnosis include, effusion, signal intensity of the fluid, synovial thickening, enhancement after gadolinium and bone erosions. Signal abnormalities that are typical of infection are hypo-intensity on T1 weighted images and hyper intensity on T2 images and STIR sequences.

Although it is difficult to distinguish between pyogenic and tubercular lesions on MRI, there are a few useful hints that could help. Marginal erosions more common in TB in early stages and joint space preservation is more common in TB. Although soft tissue extension and abscesses are common in both, it is more common with pyogenic than TB (92% with pyogenic and 72% TB). The abscesses are thin walled and regular in 70% of TB infections and thick and irregular in 82% of pyogenic infections.

How does one make a diagnosis of Bone & Joint TB?

The diagnosis of B&J TB is challenging. It is diagnosed on the basis of clinical impression, radiological features and tissue diagnosis. Of these, clinical features and radiological parameters are non-specific and not diagnostic of Tuberculosis. Due to this tissue diagnosis becomes extremely important. Further since there is increased prevalence of drug-resistant cases, it becomes imperative to obtain tissue biopsy before starting therapy.

OBTAINING TISSUE

Obtaining a tissue is challenging in B&J TB patients. Often invasive means are required. The sample is often obtained by a wide bore needle under USG or CT scan guidance. It is a challenge at times, to identify the representative tissue from the USG/ MRI/ CT images when doing a CT/ USG guided biopsy.

Histopathology/ culture reports are often inconclusive when the sample is obtained by FNAC.

Specimens which often yield proper diagnosis include:

Specimen properly collected and selected by the surgeon (deep tissue biopsy), Granulation tissue or pus in adequate quantity preferably 2/3cc, Promptly transported, When the transport or the processing is delayed, specimens should be stored at 4°C until transported.

SPECIMEN COLLECTION TO BE DONE:

In sterile, leak-proof containers without any additive or preservatives (NOT EVEN NORMAL SALINE) Labeled with the patient’s name and/or identification number before anti-tuberculosis chemotherapy is started.

HISTOPATHOLOGY

A definitive diagnosis of tuberculosis is based on the presence of AFB and/or presence of necrotic epitheloid granulomas with multinuclear giant cells. However at times the number of granulomas, is so few, that a positive diagnosis depends on histopathologist’s patience and perseverance.

At times the histopathological picture of RA synovium could mimic TB. Atypical mycobacteria produce a similar picture non chronic granulomatous inflammation with or without caseation. They need to differentiated on the basis of culture characteristics. Resistant lesions cannot be differentiated from those caused by non resistant strains.
MICROBIOLOGY

Bone and joint TB is a pauci-bacillary disease. The culture positivity is reported to be between 10-60%. The classic culture medium is Lowenstein Jensen or solid medium culture and has a low sensitivity and takes 6 to 8 weeks for growth. The recent cultures methods like Bactec and MGIT (Mycobacterium Growth Indicator Tube) are more sensitive. While Bactec is a semi-automatic method and exposes the personnel to radiation, MGIT, is a very sensitive test and yields a report in 2 to 3 weeks. However it is advisable to do both do LJ as well as MGIT cultures and accept a negative report only when both are negative.

What is the current management of Bone and Joint TB?

MEDICAL TREATMENT

The medical treatment of B&J TB is similar to pulmonary TB. However there are some issues which make medical management of OA TB difficult. Most often in our country as the tissue diagnosis is not done in every case, the diagnosis and treatment is empirical. The change from intensive phase to maintenance phase is again empirical. The end point is not clearly defined (unlike pulmonary TB where 4 months after sputum turns negative is considered as the end point). Various studies have recommended giving AKT for a total of 9 months to 24 months. However in drug-sensitive cases, the results of appropriately instituted and completed therapy are good.

The protocol most commonly followed is

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isoniazide</td>
<td>5mg/Kg</td>
</tr>
<tr>
<td>Rifampicin</td>
<td>10 m/kg</td>
</tr>
<tr>
<td>Ethambutal</td>
<td>15 to 20mg/kg</td>
</tr>
<tr>
<td>Pyrazinamide</td>
<td>25mg/kg</td>
</tr>
</tbody>
</table>

This is followed by

<table>
<thead>
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</tbody>
</table>

The treatment of MDR is generally initiated and managed by infection disease specialists or chest physicians and are on similar basis to resistant pulmonary TB

SURGICAL TREATMENT

Surgical treatment is needed only in selected cases. The common indications for surgical intervention in Pott’s spine are spinal instability, late onset paraplegia, progressive paraplegia and very large abscesses.

In other sites, surgical intervention is done for tuberculous osteomyelitis with abscesses with large sequestrate and sclerotic bones. Best to do surgery before putting the patient on ATT as the chances of obtaining culture in sensitive cases reduces significantly with therapy. Discharging sinuses not responding to medical line of management also need surgical excision.

Totally destroyed joints, causing significant pain or disability, need surgery. Excision, fusion and at times joint replacement are needed. Generally replacement is done a few years after the disease is quiescent however some surgeons prefer to do so even earlier.

SUMMARY

Bone and joint Tuberculosis, although not as prevalent and infectious as its pulmonary counterpart, is increasing in incidence and severity. It poses various diagnostic and therapeutic challenges to a rheumatologist.

The symptoms and signs are often non-specific. In the early stages it may difficult to get a tissue diagnosis. Imaging though very helpful does not differentiate pyogenic from tubercular lesions. Since it is a pauci-articular disease cultures from B&J TB patients do not have a high yield. Along with the traditional culture medium, the newer microbiological methods have improved diagnostic capabilities. There still is no definite consensus as to how long to treat.

REFERENCES


In contemporary ICUs, pulse oximetry is ubiquitous and there is really no reason to routinely use oxygen in all patients. Conventionally, we target a SO2 of > 85-90% in stable awake patients. It is reasonable to give hypoxic patients some supplemental oxygen to keep the SO2 above the desired target. It is much more important to find & correct the cause of the hypoxemia. Though it is widely assumed that we actually help patients by reversing hypoxemia, the impact on clinical outcomes is not well documented.

A few recent trials allow us to get some perspective as to whether giving oxygen to most of our patients is really necessary or even safe. In fact, two recent Randomized Control Trials (RCT) & one very large observational trial suggest that liberal use of oxygen may be causing significant harm.

One RCT in acute exacerbation of COPD found that the risk of death in all patients was significantly reduced by 58-78% with titrated oxygen treatment compared to liberal oxygen. Another RCT compared Air versus Oxygen non-hypoxic patients with ST elevation myocardial infarction. The use of oxygen in these patients was associated with higher rates of arrhythmias (40.4 vs 31.4%) and recurrent myocardial infarctions (5.5 vs 0.9%). It was also associated with larger initial infarct size as judged by CK-MB & Troponin peak, and also larger infarct size at 6 months as evaluated by cardiac MR.

A large observational study looked at clinical outcomes of exposure to high oxygen concentrations following CPR. They defined Hyperoxia as being > 300mmHg, and hypoxemia as being < 60 mmHg. They noted that arterial hyperoxia was independently associated with increased in-hospital mortality compared to either hypoxia or normoxia. In other words, not only did the patients who had a PaO2 > 300mmHg do worse than those with a normal oxygen level; they also did worse than patients who were hypoxic.

What messages can we take home from these three large trials. Mainly that oxygen must be used judiciously as it can cause more harm that is superficially apparent.

**REFERENCES**


3. Association Between Arterial Hyperoxia Following Resuscitation From Cardiac Arrest and In-Hospital Mortality. JAMA. 2010;303(21):2165-2171

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## IN A NUTSHELL – ILD PATTERNS

**DR. JAI MULLERPATTAN**  
Associate Consultant

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Associated Condition</th>
<th>HRCT findings</th>
<th>Histology</th>
<th>Clinical features</th>
<th>Treatment</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>UIP</td>
<td>IPF, Asbestosis, RA-ILD, Chronic Hypersensitivity Pneumonitis.</td>
<td>Peripheral basal subpleural reticular opacities, honeycombing, traction bronchiectasis, no nodules or ground glass opacities.</td>
<td>Usual Interstitial Pneumonia pattern - Temporal heterogeneity, fibroblastic foci,</td>
<td>Male, &gt;50 yrs., Velcro crepts, clubbing, chronic course.</td>
<td>Poor response to steroid and cytotoxic drugs, newer drugs in the pipeline.</td>
<td>Median survival 3-years (for IPF).</td>
</tr>
<tr>
<td>NSIP</td>
<td>Idiopathic NSIP, Collagen Vascular Diseases, Drug induced</td>
<td>Peripheral, subpleural, basal symmetric ground glass opacities with septal thickening, traction bronchiectasis.</td>
<td>honeycombing, Non-Specific Interstitial Pneumonia pattern - cellular, fibrotic or mixed.</td>
<td>Age 30-70, more in females, maybe associated with collagen vascular disease, subacutec to</td>
<td>Cellular: Good response to steroids may need additional immunosuppression Fibrotic: Similar to UIP.</td>
<td>Generally good, better than UIP.</td>
</tr>
<tr>
<td>BOOP</td>
<td>Idiopathic COP, Drug induced, Connective Tissue Diseases, Post transplant (Lung, Heart, Bone marrow), Viral</td>
<td>Subpleural or peribronchial patch consolidation with nodule.</td>
<td>Organising pneumonia, Masson’s bodies.</td>
<td>Age 50-70 years, non smokers: smokers 2:1, many associated conditions.</td>
<td>Corticosteroid responsive, may need additional immunosuppression.</td>
<td>Good.</td>
</tr>
<tr>
<td>AIP</td>
<td>Idiopathic, Toxic exposure induced, Hamman Rich Syndrome</td>
<td>Diffuse bilateral ground glass often with lobular sparing.</td>
<td>Diffuse Alveolar Damage.</td>
<td>Acute, usually &gt; 40 years, Fever, cough, dyspnea, may have family history.</td>
<td>Corticosteroid responsiveness unknown but high dose pulses often used.</td>
<td>Poor.</td>
</tr>
<tr>
<td>DIP/RB</td>
<td>Smoking related ILD.</td>
<td>DIP: Diffuse ground glass opacity in mid lower lung zones RB-ILD: Bronchial wall thickening, centrilobular nodules, patchy ground glass opacity.</td>
<td>Deaquamative Interstitial Pneumonitis pattern, Respiratory bronchiolitis.</td>
<td>&gt;90% smokers subacute presentation, age 3040 years.</td>
<td>Smoking cessation. May respond to steroids.</td>
<td>Good.</td>
</tr>
</tbody>
</table>

**UIP:** Usual interstitial pneumonia  | **NSIP:** Non-specific interstitial pneumonia  
**BOOP:** Bronchiolitis obliterans with obstructive pneumonia  | **AIP:** Acute interstitial pneumonia  
**LIP:** Lymphoid interstitial pneumonia  | **DIP:** Desquamative interstitial pneumonia
**THROMBOCYTOPENIA IN SLE PATIENTS**

**PLEASE PERFORM**
- Blood film for clumps, red cell fragments, spherocytic changes.
- Drug History (Methotrexate / Azathioprine etc.) and history suggestive of infections
- Look at other CBC parameters for other cytopenias and haemolytic features (reticulocyte count, LDH)
- Clotting and mixing study, if prolonged PTT and inhibitor screen, LA
- Haematinics level (B12/Folate/Iron studies)

---

**(A) LOW PLATELET ASSOCIATED WITH OTHER CYTOPENIAS**

**NORMAL OR REDUCED RETICULOCYTES**
- MDS (Myelodysplastic Syndrome)
- B12 And Folate Deficiency (macrocytosis)
- Drug Effect
- Acquired Bone Marrow Failure

**HAEMOLYTIC FEATURES**
- Evans syndrome
- DIC (Microspherocytes)
- MAHA (Microangiopathic Haemolytic Anaemia)

**LOOK FOR OTHER FEATURES OF MAS (HIGH FERRITIN ETC)**
- MAS (Macrophage Activation Syndrome)

---

**(B) ISOLATED THROMBOCYTOPENIA**

**PROLONGED PTT WITH INHIBITOR**
- Lupus anticoagulant*

---

**NO CLOTTING ABNORMALITY**
- Normal haematinics
- No splenomegaly
- No offending drug
- BM: Adequate megakaryocytes
- BM: No MDS
- No viral infections

---

**ITP (IMMUNE THROMBOCYTOPENIA)**

---

_For more information_

(*If the thrombocytopenia is more severe than expected for inhibitor, it is likely to be ITP
** Anaemia because of other reasons like anaemia of chronic disease can be associated with thrombocytopenia, but the degree of thrombocytopenia in acute ITP is much more severe.)
INFECTED PROSTHESIS

Dr Vivek Shetty, Consultant Orthopaedic Surgeon, Hinduja Hospital, Mumbai

<4 weeks
Pain +/- Fever

- Debridement/
washout
- Tissue/Fluid Culture
- Antibiotics
- 6 Weeks
- Retainment of implant

No improvement

4 weeks - 3 months
Painfree join becomes painful.
+/- Fever

- Aspirate the Joint

Culture Positive

- Debridement +
Antibiotics

Culture Negative

- Raised
ESR/CRP/neutrophilia
in the synovial fluid

- ESR/CRP normal
No neutophilia in the
synovial fluid

NSAIDS
Close follow-up

Implant removal

STAGE 1
Implant removal - C/S
Antibiotic impregnated cement
spacer OR Articulating device
spacer with loosely packed
cement

3 months of antibiotics.
Monitor ESR/CRP and when
normal

STAGE 2
Revision or fusion

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A thirteen year old female complained of localised pain in the right shoulder since 4 years. Clinically there was focal nodular tenderness over the deltoid muscle. Shoulder USG revealed a well circumscribed oval lesion within the deltoid muscle. It had a heterogeneous echotexture with lobular echogenic areas within and was tender on applying probe pressure. These findings favour a solid soft tissue intramuscular tumor. Further evaluation with MRI revealed an enhancing well-defined posterior intradeltoid tumor - nerve sheath tumor.

Targeted pre-operative ultrasound localisation of the lesion was performed as the lesion was small and changed position with movement. Specific position of the arm was maintained that was conveyed to the surgeon. This also helped in reducing the size of the incison. Patient underwent an excision biopsy and histopathology report suggested vascular leiomyoma (SMA was strongly positive and S 100 was negative).

This case highlights the multipurpose role of ultrasound in soft tissue lesions such determining the presence of an abnormality and then in localising it, assessing internal morphology (solid, cystic, fat, etc.), making a provisional diagnosis, and when needed, localising it either for biopsy or surgical removal. It is a quick and simple way to assess the abnormality preliminary to a dedicated MRI, especially in small / non-palpable / mobile / deep lesions as in this case.

---

ACUTE ABDOMEN - EPIPILOC APPENDAGITIS

A thirty-eight year old male came with an acute non-radiating right flank pain. Severe point tenderness was noted on palpation. There was no obvious evidence of acute appendicitis on ultrasound. CT with oral and intravenous contrast revealed a focal oval fat density area (about 3.5 cms in size) with surrounding inflammation in the right flank between the ascending colon and redundant sigmoid colon. Central linear area was noted possibly representing a thrombosed vein. Constant relation was maintained between the lesion and sigmoid colon. Findings represent inflamed fat, likely epiploic appendagitis.

Torsion of the appendage (which is attached to the serosal surface of the colon by a vascular stalk) leads to vascular occlusion, ischemia and resultant acute pain. It is more common in males in the 4th and 5th decade. Usual presentation is pain in the left iliac fossa with no fever and leukocytosis.

In our case, there was right sided pain due to a redundant sigmoid colon. Epiploic appendagitis is a self-limiting condition and resolves with anti-inflammatory drugs. In recent times, it is diagnosed more often because of the increasing use of CT in acute abdomen. Knowledge of this entity avoids unnecessary hospitalization and surgery. Differentials include an omental infarct.

---

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In all civil cases (including consumer), the onus of proof is on the complainant to prove his complaint. This means that the complainant must convince the court that the version of events is more than 50% to be correct which, can be difficult. However there are times when the complainant need not do this. He could resort to a legal maxim termed “res ipsa loquitur”

Literally translated, res ipsa loquitur means “The situation speaks for itself”. This means the accident could happen only if someone (for eg. nurse or doctor) was negligent. On application of this maxim, it is for the doctor to satisfy the court that there was some other cause for the damage to the complainant.

The complainant has to show that:

1) That the damage caused to the complainant was due to an accident.
2) The event which caused the accident was solely under the control or management of the opposite party.
3) That the accident was such that, in ordinary course of things, the accident would not have happened if the opposite party had taken proper care.

Some examples of res ipsa loquitur applicable are:

- Development of meningitis after spinal anaesthesia
- Rupture of ear drum after ear syringing
- Permanent brain damage after appendicectomy
- Amputation of wrong limb or wrong digit or operation
- Burning of skin after strong antiseptic solution
- Injury, damage or death at a place where the attendants of patients have no access (Labor room, ICU, OT)
- Leaving swabs or surgical instruments after an operation

The principle of application of res ipsa loquitur is clearly illustrated in the following cases:

A.II(2014)CPJ 5(SC)SUPREME COURT OF INDIA

{Mazumdar vs "B" Hospital}

Mazumdar was admitted on the third floor in the “B” Hospital. He was running high fever and was in a delirious state. After four days, in the night around 2.20 am, the sister of the patient who was staying with him in the room noticed the absence of the patient from the room. She promptly informed the hospital staff and a search was conducted to trace out the patient. The security found him lying on the ground floor in the oncology gallery of the hospital below the window of the room which the patient was occupying. The patient had sustained multiple fractures of lumbar vertebrae with complete dislocation of the spinal cord and became a paraplegic.

JUDGMENT:

The judge held that duty of a hospital is not limited to diagnosis and treatment but extends to looking after the safety and security of the patients. At the time of the incident, the patient was an inpatient of the hospital. It was the duty of the hospital authorities to take care of the patient who was suffering from high fever and was in a delirious state. Due to absence of due and reasonable care of the hospital authorities the incident occurred, disabling the patient for the rest of his life and affecting his employment and career prospects. Applying the principle of res ipsa loquitur, the judge concluded that:

- Patient had sustained multiple injuries while he was an inpatient in the hospital.
- The hospital has a duty towards the safety and security of its patients.
- Had the hospital exercised due care, such accident would not have occurred.
In this case, Dr. G had applied a plaster cast and Dr. S had inserted a pin. But they had no records to prove that they advised antibiotics or had taken any other steps to prevent gas gangrene. Both these doctors committed egregious mistake by not cleaning the wound properly. They were aware of the fact that gas gangrene could be a complication under such circumstances. Thus applying the principle of *res ipsa loquitur*, the judge concluded that there was negligence, inaction and passivity on their part which led to amputation of the leg. Prompt action on part of Drs. G and S would have prevented the gas gangrene and saved the leg of the patient.

Two and half lacs with 10% interest was awarded payable to the complainant (30% by Dr. G and 70% by Dr. S).

Hence the hospital is held liable and negligent for not maintaining the necessary vigil in the hospital premises to ensure safety of its patients.

**DAMAGES OF 7 LACS WITH 12% INTEREST WAS AWARDED .**

**B.II(2014)CPJ 499 (NC): Yogesh Vs DR. S & Ors.**

Yogesh, who sustained multiple injuries and fracture in his leg was taken initially to Dr. G who plastered his right leg. After two days, patient approached Dr. S who operated him and inserted a pin. Later on, Dr. S advised the patient to go to a government hospital. Meanwhile, the foot emanated foul smell since pus was present. The patient went to Safdarjung Hospital who diagnosed that gas gangrene had set in due to ill-treatment. AK amputation was done immediately to save the life of the patient.

**JUDGEMENT:**

The court relied upon literature which clearly mentioned that the organism Clostridia Botulinum of the gas gangrene group multiply in anaerobic condition. Therefore as per standard line of treatment while managing such a wound in the leg, gas gangrene is better prevented by debridement of all necrotic tissue, evacuation of pus, wound irrigation, prophylactic antibiotics, gentle but effective application of plaster to avoid compression of blood vessel. Above all, the wound should not be tightly closed.

In this case, Dr. G, had applied a plaster cast and Dr. S had inserted a pin. But they had no records to prove that they advised antibiotics or had taken any other steps to prevent gas gangrene. Both these doctors committed egregious mistake by not cleaning the wound properly. They were aware of the fact that gas gangrene could be a complication under such circumstances. Thus applying the principle of *res ipsa loquitur*, the judge concluded that, that there was negligence, inaction and passivity on their part which led to amputation of the leg. Prompt action on part of Drs. G and S would have prevented the gas gangrene and saved the leg of the patient.

Two and half lacs with 10% interest was awarded payable to the complainant (30% by Dr. G and 70% by Dr. S).

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What Doctor’s Day Means To Me?

Each year when a new calendar arrives I check out the national, holidays, - do they fall on Sat / Sun or not, then the dates of important religious holidays, - how do the dates vary from the previous year, and finally the dates of our select hospital holidays, and then that’s it.

For other celebrity days such as Mother’s day, Father’s day, Teachers day and Doctors day I hardly give them a thought as I regard them as gimmicks put in place by a commercialized society. But now I find I am wrong.

Doctors Day is only commemorated by a handful of countries worldwide of which India is one. We observe 1st July every year as it happens to be the birth anniversary of Dr. B. C. Roy, an outstanding medical personality a freedom fighter, a women’s rights advocate and a former chief minister of West Bengal.

The practice was started in 1991 and has been followed yearly thereafter. Seen from this sober perspective I feel we at HNH should use the occasion for some healthy introspection.

Today, doctors have fallen from grace and are seen by society as a profession steeped in corrupt practices, commercialism and greed. Alas this view is partly true and lies behind the several violent attacks on doctors reported from different parts of Mumbai by an enraged public seeking quick justice for fatalities they perceived as acts of medical negligence.

As leaders of the medical community what can we at HNH do to dispel this ‘radicalized’ view of our profession? We must be the flag bearers to restore again a strong doctor patient relationship which is at the heart of a noble profession.

But all is not gloom by any means. Great strides have been made in each and every specialty of medicine year on year. Technology is the driver behind many of these; - what was new yesterday risks being obsolete by next year. Only human nature remains largely unchanged, seeking succor in times of sickness and suffering. To those that combine compassion with science - Happy Doctors Day!

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1. Dr. Camilla Rodrigues: TB diagnosis - standard of care in 2015

Dr. Rodrigues explained the various molecular genotypic tests available for TB diagnostics and also stressed the need to identify the type of mutation which can predict high level or level resistance for the various drugs.

2. Dr. Zarir F Udwadia: Management of MDR TB - a case based approach

Dr. Udwadia presented three difficult cases of drug resistance TB each requiring varying approaches ranging from new drugs such as bedaquiline, meropenem and clavulanate and surgery or a combination of these.

3. Dr. Bhavin Jankharia: HRCT in the diagnosis of ILD’s

Dr. Jankharia elaborated on the various patterns of ILD as seen on an HRCT and clues to differentiate between them.

4. Dr. Randeep Guleria: Newer drugs in COPD

Dr. Guleria gave an overview on the newer drugs for COPD both in the market and in the pipeline for various stages in the pathogenesis right from smoking cessation, anti-inflammatories, bronchodilators and drugs to prevent exacerbation.

5. Dr. Lancelot Pinto: Role of Pulmonary Rehabilitation in COPD

Dr. Pinto gave an excellent lecture on the need for pulmonary rehabilitation and the various components for the same and the benefit they had in the quality of life and mortality of patients with COPD.

6. Dr. R Vijai Kumar: Asthma - COPD overlap syndrome

Dr. Vijai Kumar provided insights on this newly recognised entity along with clues to diagnosis and treatment and differentiation from pure asthma or COPD.

7. Dr. Sujeet Rajan: Difficult asthma

Dr. Rajan expounded on difficult asthma on the basis of difficult disease, difficult patient and difficult doctors. The need to educate patients and check the proper use of devices was emphasized as well as the fact that truly difficult disease was a rare entity. Dr. Raja Dhar: Asthma phenotypes

8. Dr. Dhar provided an insight into various asthma phenotypes based on a case based approach along with a need to individualise treatment in each of the cases such as obesity related asthma, neutrophilic asthma, eosinophilic asthma etc.


Dr. Mullerpattan educated the audience on the various severity scoring systems for community acquired pneumonia as well as other devices to do the same such as biomarkers etc. An overview of the management including antibiotic use and adjunctive therapy was also provided.
10. Dr. C Balakrishnan: Lung in Connective Tissue Diseases
   Dr. Balakrishnan lectured on the differing pulmonary presentations in patients with connective tissue
disease and diagnosis/management of the same. Lung involvement in Rheumatoid arthritis, Systemic
sclerosis, Sjogrens syndrome, SLE and inflammatory myopathies were covered.

11. Dr. J C Suri: Sleep in respiratory diseases
   Dr. Suri covered the change in patterns of breathing in sleep and the effect of the same in respiratory
diseases along with need for sleep study followed by nocturnal NIV or oxygen.

12. Dr. B K Smruti: Advances in the management of NSCLC
   Dr. Smruti provided new insights in the advances of management of NSCLC while stressing the need to
obtain a histopathology diagnosis including type of NSCLC and presence of EGFR/ALK/K-Ras mutation so
that treatment can be tailored according to the same.

13. Dr. PP Prabhudesai: Management of the undiagnosed/difficult pleural effusion
   Dr. Prabhudesai dwelled on length on the various causes of difficult pleural effusion along with clues to
diagnose them including biochemical tests and thoracoscopy.
1) She qualified as a doctor from Grant Medical College during her reign as Miss World. Who is she?

2) Name any Doctor who has also played test cricket.

3) What is the abnormality seen on this X Ray?

4) Why is July first celebrated as “Doctor’s Day”?

5) Which Hollywood Actor who passed away recently, first achieved international fame for his role as a Russian doctor.

6) This patient was depressed because of her disease and consumed a poison one night. The next morning she was found smiling (instead of being found dead). Which disease? Which poison?

7) Siddhartha Mukherjee won the Pulitzer prize for his book “The Emperor of all Maladies”. What according to him is the “Emperor of all maladies”?

8) Who is the first Indian Doctor to scale Mount Everest?

9) Which is India’s (and probably Asia’s) biggest Hospital (by bed strength)?
10) In the movie “Anand” what was the disease that Anand was suffering from?

11) In the film “Trimurti”, the role of Kokha Singh was played by a doctor. What is his speciality?

12) Which was the first hormone to be discovered?

13) The Second nobel prize in medicine was awarded to a scientist for his work done in India. Who was this scientist?

14) Both of these patients are suffering from Herpes zoster, which of them would you be more worried about and why?

15) Who performed the first successful heart transplant and where?
NEW CONSULTANTS

DR. RUCHA KAUSHIK
MBBS, M.S.
Consultant Breast Cancer Surgeon

DR. SHIVKUMAR V. DALVI
M.S.(General Surgery, K.E.M. Hospital, Bombay)
Part Time Consultant in Health Check-up (Surgery)

DR. AMRESH SUDARSHAN BALIARSING
MCh, DNB
Consultant Plastic Surgeon

DR. HEMANTH KUMAR PANDHARPURKAR
M.S. (General Surgery), M.R.C.S. (Edin.), Fellowship – Vascular Surgery (Sree Chitra)
Consultant Vascular Surgeon

DR. KEDAR DEOGAONKAR
MS (Orth), DNB, MRCS, MSc, FRCS (Tr & Orth), CCT (UK)
Consultant Spinal Surgeon

DR. NISHANT KUMAR
MRCOphth (London), FRCOphth (London)
Consultant Ophthalmologist & Vitreo - Retinal Surgeon

DR. SATYAKAM KRISHNA SAWAIMOON
Consultant MD - Pathology & Surgical Pathology

DR. BHoomika THakore
MBBS, DNB Anaesthesiology
Post Doctoral Fellowship in Neuroanaesthesiology
Consultant Anaesthesia

DR. PRANJALI ADVANT
MBBS, MD (Radiology), DNB, MNAMS, PDCC,
European Diploma in Radiology, EULAR Certified
Consultant Radiology
ICICI Lombard and CNBC TV18 Best Multi Speciality Hospital in India-Megapolis Award 2014

Best Multi Speciality Hospital-Mumbai at Healthcare Achievers Awards 2014

"Best Multi Specialty Hospital" at e-HEALTH Maharashtra by Department of Public Health, Government of Maharashtra in 2014

Qimpro - Best Prax Benchmark, Recognition 2013 & 2010, for Excellence in Managerial Practices

"TB Champions" award by Global Health Strategies, 2013

Operational Excellence Award at FICCI HEAL, 2013

Excellence Award for CSR project at The Asia Healthcare Management Awards, 2012

International Diamond Prize for Customer Satisfaction, by ESQR, 2012

IMC Ramkrishna Bajaj National Quality Award, 2007

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Excellence in Hospital at the Medscape India National Awards 2013

Best ICT enabled hospital in Maharashtra at e-Maharashtra Awards, 2013


'Excellence in Community Engagement' by Association of Healthcare Providers of India (AHPI) 2014.

Doc N Doc Awards Best Multi-speciality Hospital (Metro) 2014

MC Ramkrishna Bajaj National Quality Performance Excellence Award 2014 in Healthcare Category

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