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Indian cancer congress 2011

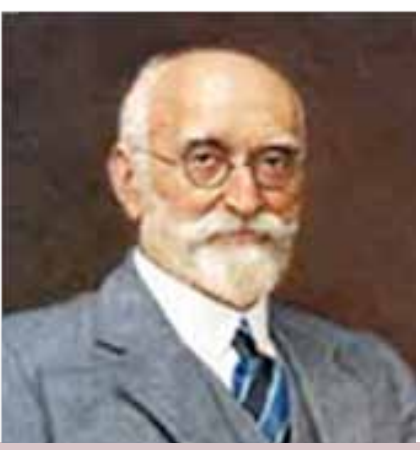
Bhubaneshwar

12th February 2011

Biennial joint conference of ISMPO (Indian Society of Medical & Paediatric Oncology) and ISO (Indian Society of Oncology)

Theme: Targeting Cancer with Humility

Inauguration



Sir Dorabji Tata - In March 1932, sir Dorabji TATA put all his wealth, estimated at Rs. 10 million in to the trust for use without any distinction of place, nationality or creed, for the advancement of learning and research, the relief of distress and other charitable purposes. This was the beginning of the Sir Dorabji TATA trust - a trust that has gone on to build institutions of national importance, uplift the nation and its people and change lives.

Sir Dorabji Tata Oration (ISO): By Dr. T. Rajkumar

Molecular Oncology: Bench to bed-side

Dr. T. Rajkumar is Professor and Head, Dept. of Molecular Oncology at Adyar Cancer Institute, Chennai.

Dr. Rajkumar is the first qualified Medical Oncologist with a Ph.D. in Basic Science (Molecular Oncology) in India, Visiting Professor, Dept. of Clinical Oncology, RPMS and Hammersmith hospital, London, UK and elected as Fellow of the National Academy of Medical Sciences (FAMS).

Dr. Rajkumar has been bestowed with various prizes, Medal and rewards throughout his academic as well as professional career. He has been involved with various research program and projects and holds prestigious membership to Technical Expert Group and Consultancy. Dr. Rajkumar has various national and international publications and is a journal reviewer to British Journal of Cancer, Asia Pacific Journal of Cancer Prevention, BMC Cancer, Hereditary cancer in clinical practice, Indian Journal of Biochemistry and Biophysics, Indian Journal of Medical and Pediatric Oncology, Indian Journal of Medical Research and Indian Journal of Gastroenterology.

ISO Past President Oration

Dr. Ramakant K. Deshpande

MUMBAI

Cancer is unbridled proliferation of functionally useless and potentially lethal cells. Cancer is known in to occur from the Egyptian mummies, animals, birds and fishes. Cancer reference is noted even in CharakSamhita, Vedas, early Scriptures. Large mutiliating surgeries were the norm in 19th and early 20th century. The year 1957 witnessed a major change in the cancer management.

The understanding of cancer biology has changed from the Halstedian concepts (1894) to the Fisher (alternative) hypothesis of tumor biology (1968).

As the technology advanced, newer radiological tests like CT scan, MRI scan, PET scan, nuclear medicine scan and endoscopies came into existence .

The newer technologies aims at improving accuracy, reducing side effects, maintain function and prolonging life with useful values and quality of life.

The current technology used in cancer surgery are:
1. Laser surgery
2. Cryosurgery/electrosurgery
3. Endoscopic surgery Laproscope Thoracoscope Mediastinoscope
4. High-intensity focused ultra sound Micro waves, Radiowaves
5. Stereotactic radiation
6. Gamma knife
7. Imaging robatic surgery
8. AESOP
9. Da Vinci Zeus systems
10. Navigator Nanorobots

Prognostic/predictive markers are used for breast cancer where low risk in optimised local and hormonal therapy, High risk is involved in systematic therapy based on prediction.

The recent technology is using of robotics in oncology, Mount Sinai Robotics program is one among the successful robatic program.

Nanoparticles (NP) has led to targeted drug delivery and hormonal therapy. The common nanoparticle based Chemotherapy (Conjugated Antracycles/Epirubicin), Immunotherapy (Trastuzumab) , thermotherapy (Conjugated with tumor targeted ligand, magnetic NP can ablate tumors), anti-angiogenic therapy and gene therapy .

"CHANGES IS THE ONLY CONSTANT ".

Symposium on CML

CML – Indian Scenario

Dr. Kumar Prabhash

MUMBAI

CML refers to the Chronic Myeloid Leukemia. Mutations in CML have single lab data from India. Epidemiology of CML in India has data mainly derived from the Mumbai Cancer registry. Age specific incidence rates (ASR) per 100,000 of CML for pediatric, young, adult and geriatric population in males and females was stratified by six calendar (years from 1976-2005) in Mumbai. Number of patient analyzed (n=1074). Interruption in patient not achieving CCyR-34.7% (n=242). Interruption in patient achieving CCyR-21.4% (n=832). Overall interruption 24.1%, Non compliance with Generic: 15%, Non compliance with Glivec: 26%. Imatinib was first line therapy or was initiated within 6 months of diagnosis & treatment with hydroxyurea - when Imatinib became available in India (20 patients up to 6 months of HU). Complete Hematological response was achieved by all patients. Both the patients in AP achieved and continued to remain in CCR. Bosutinib is used in first line therapy for CML in chronic phase. Dasatinib is used as first line therapy for CML patients in chronic phase. The main ongoing trials are the PHASE 2 multicentre study of IY5511 HCL in Ph positive CML patients without optimal response or tolerance to Bcr-Abl TKI inhibitors. Nilotinib is a suboptimal responder in patients with CML in chronic phase (LASOR study). Nilotinib is a first line therapy for sokal high risk patients in chronic phase.

Complete hematological response was achieved by all patients in a median time to CHR being 4 months. The GIPAP program run by the MAX foundation has been a big help for pts to take Rx with Glivec (80%). Generic brand of Imatinib (Veenat) seems to be feasible alternative. Status of mutations in the Indian patient with CML results from a single large lab.

Imatinib Resistance Diagnosis and Management

Dr. Shyam Aggarwal

NEW DELHI

Resistance can be defined on the basis of its time of onset. Primary resistance is the failure to achieve a significant hematological or cytogenetic response. Secondary or acquired resistance is the progressive reappearance of the leukemic clone after an initial response to the drug. The defects which come in picture with respect to drug transporters are the influx the transporters OCT1 inhibition mediates active transport of Imatinib into cells. OCT1 inhibition decreases intracellular concentration of Imatinib. OCT1 expression is higher in patients who achieve a CCyR. Mutation screening is important in patient monitoring. Mutations have varying degrees of resistance characterizing the mutation will have an impact on therapeutic intervention. Detection before onset of resistance may allow intervention, clinical implications of resistance to first line Imatinib in chronic phase. Suboptimal response had outcomes similar to failure at earlier time points of 6 months and 12 months. ELN recommendations for failure or suboptimal response failure is patient has to be moved to other available treatments. Suboptimal responses may still have benefit from continuing with Imatinib, but long term outcome is not likely optimal. Management of resistance to Imatinib in chronic phase CML dose escalation is by IRIS. The second generation tyrosine kinase inhibitors (phase2) are Dasatinib, Nilotinib, Bosutinib. There are treatment options of CML with T315I. The major multi-kinase inhibitors are AP24534 (Ariad), PHA-739358 (NERVIANO), XL228 (Exelixis), DCC-2036, DCC-2157 (Deciphera). The mechanism of action (Omacetaxine for CML with T315I) acts as inhibitor of elongation protein first in class Cetaxine. It acts as a potent inhibitor against leukemic cells, including T315I+ cells1. Its activity is independent of Bcr-Abl binding; it induces apoptosis by inhibiting the anti-apoptotic oncoprotein Mcl-12. It inhibits cell growth and induces apoptosis in CD34+cells3.

Nilotinib

Dr. Devendra Hiwase

INDIA

The major three conceptual models of CML progression are Chronic, Accelerated, and Blast. Chronic phase is the one which mainly deals with the increase in inability to proliferate. The point mutational chromosomes have decreased rate of differentiations, increased cell cycle, and decreased rates of apoptosis. The accelerated phase is also known as the advanced phase. A total number of 553 patients' were taken and a study was conducted during a period of 7 years. About 40% of the patients' stopped their medication procedures and discontinued from the experiments, whereas another 60% continued and their datas were updated in CCyR the number of patient's were 317, roughly 57% of them and in not CCyR the number of patient were 15, roughly about 3% About 71 patients' (71/221, 32%). One progression to AP/BC and 2 non-CML related deaths occurred in year 8. Estimated rate of freedom from progression to AP/BC at 8 years=92%. There is a need to improve frontline therapy in CML. Excellent results with frontline Imatinib. However 18% do not achieve CCyR, 4-8% intolerant, 11% lost CCyR, most patients' have residual disease, and there is need for treatment indefinitely. In addition approximately about 50% of patients' do not achieve CCyR with 2nd generation TKI and approximately about 10% have lost CCyR after 2 years. In the predictions of the long term out-come intrinsic CML biology are sokal score and microarray. The possibility of identifying patients at risk of an inadequate response to Imatinib is by intrinsic sensitivity to TKI, OCT-1 activity, TKI trough level, drug adherence and compliance, CCyR at 12 months, MMR at 18 months. 31-43% of patients' with newly diagnosed CML-CP fail to achieve a CCyR by 12 months with Imatinib treatment compared with patients who achieved CCyR by 12 months of Imatinib treatment, patients who failed to achieve CCyR by 12 months had 6.5-fold higher rate of progression or death at 60 months. 13 fold higher rate of death at 60 months. Second generation TKI used as front line therapy is Nilotinib, Dasatinib, Bosutinib. Nilotinib has no significant effect on other kinase evaluated including Src, FLT3, VEGFR, EGFR, InsR, RET, MET, IGFR at concentrations, 3000 nM. Front line therapy for newly diagnosed CML-CP patients, standard dose Imatinib is 35 to 40% failure rates, long term safety.

Case Discussed

Case 1

A 46 years old man was diagnosed with CML-CP where BM shows 100% Ph +ve. The treatment was started on Imatinib 600 mg/day. At 6 months, the CBC was within normal limits, BM 75%, Ph positive. At 12 months - In CHR, BM 60% Ph positive and at 18 months - In CHR, BM 40% Ph positive.

Question

Based on the degree of response in this patient, what would you suggest?

1. No change, continue same Rx
2. Increase the dose of Imatinib to 1200 mg
3. Add low dose Ara-C to Imatinib
4. Add alpha-IFN to Imatinib
5. Change to Dasatinib or Nilotinib and initiate HLA typing of patient and family members

Correct Answer: 5. Failure to achieve major CyR (<35% Ph) by 12 months and complete CyR at 18 months indicates Imatinib resistance. The dose of 1200 mg Imatinib found to be intolerable. No data supports adding low dose Ara-C or IFN can reverse Imatinib resistance. Dasatinib and Nilotinib are highly active TKIs with high activity in Imatinib resistant CML. Considering the young age of the patient, Allo BMT should be considered.

Case 2

A 52 years old male was diagnosed with CML-CP, treated with Imatinib 400 mg/day. At 12 months: in CHR, partial CyR with 80% Ph negative. At 24 months: in CHR, BM Ph negative (complete CyR) but 3/20 metaphases showed trisomy 8.

Question

What would the appropriate subsequent treatment for this patient?

1. Continue Imatinib 400 mg/day
2. Increase Imatinib to 800 mg/day
3. Change to Dasatinib or Nilotinib
4. Switch to alpha-IFN
5. Consider AlloBMT

Correct Answer: 1. Approximately 5% of patients of CML-CP on Imatinib will develop other chromosomal abnormalities (OCA) in Ph negative cells. About 50% of these OCA are trisomy 8, 10% trisomy 8 with OCA and 15% involve chromosome 7. Some cases, especially with OCA involving Chromosome 7, may progress to myelodysplasia. In most cases, the patient remains clinically stable and the OCA clone disappears. OCA, especially OCA not involving Chromosome 7, do not signal a change in treatment.

Case 3

A 22 year old woman with Ph positive, BCR-ABL-positive (Sokal good risk) CML, started treatment with Imatinib at 400 mg daily. She achieves a CyR at 3 months and subsequently MMR at 9 months. At 12 months her transcripts are 0.008% (international scale) and at 24 months undetectable - CHR & CMR. She now tells you would like to have child.

Question

What would you advise her?

1. Tell her to forget it?
2. Stop Imatinib & tell her to get pregnant with close monitoring of disease?
3. Stop Imatinib, start IFN and tell her to get pregnant?
4. Something else?

Correct Answer: 2. It is possible for patients who have been on Imatinib & in prolonged MCR to stop drug with close monitoring (STIM Trial). About 40 to 50% continue to remain in CMR. The other 50 to 60% respond well to Imatinib once it is re-started. IFN does not cross placental barrier and is safe in pregnancy, could be considered if the patient has a molecular relapse.

Lung Cancer

Gene signature vs Individual Biomarkers in Lung Cancer

Dr. Ramaswamy Govindan

USA

BIOMARKER, it is a characteristic that is objectively measured and evaluated as an indicator of normal biologic process, pathogenic process or pharmacologic responses to a therapeutic intervention. Few of the examples of Biomarkers in clinical medicine are electrocardiogram, PET brain image, serum chemistries, auto-antigen in blood, bone densitometric measurement, pulmonary function test, neonatal Apgar score. Bioimarkers in clinical medicine is used for diagnosis, used as a tool for staging the disease, and also to predict and monitor clinical responses to an intervention. The eventual goal is individualised for two sectors of patients one being the TREAT, these are most likely to benefit and least likely to experience toxicity. The other group would be the REMOVE, these group of the patient's are least likely to respond and most likely to experience toxicity. Pharmacogenomics is the study of how an individual's genetic inheritance affects the body's reponse to drugs and the use of this genetic information to predict the safety, toxicity and efficacy of drugs in individual patient's or groups of patient's. Prognostic versus predictive markers are too used as individual biomarkers or many biomarkers are used as a combination of both predictive and prognostic values. Controlled studies or meta-analysis are required to determine the prognostic

and predictive contributions made by a particular marker. Stage1 NSCLC, relapse ratio is about 40% compared to that of reccurs, but no benefit and reccurs , benefits both together coping to 60%. Predicting outcomes in resected early stages in NSCLC is done by evaluation of micro-metastatic disease and by evaluation of primary tumor, which occurs through molecular analysis by single gene and also can be pathway directed. Predictive markers in individual groups receive different benefits from different treatments.

"NOT EVERYTHING THAT COUNTS CAN BE MEASURED. NOT EVERYTHING THAT CAN BE MEASURED COUNTS."

Role of Pathologist in Lung Cancer Diagnosis in today's world

Dr. Mallikarjuna Vadaguru

CHENNAI

In lung cancer the earlier role of a pathologist was to make the conformation of a malignancy, to distinguish NSCLC (non small cell lung cancer) and SCLC (small cell lung cancer). Distinction between squamous and adenocarcinoma was meaningless because of poor prognosis in both types. The pathologist's present role is to do the histology or cytology to determine the conformation for the presence of malignancy. Special stains like Mucin stain were used. Molecular pathology is a pin point diagnosis provides sub types of lung cancer with immunohistology of the same with help of various targeting biomarkers. The WHO recognises three pre-invasive lesions, squamous dysplasia or carcinoma insitu, atypical adenomatous hyperplasia, diffuse idiopathic neuroendocrine cellular hyperplasia. Non small cell carcinoma conquers 80% of lung cancers, not all lung cancers classified by WHO can be diagnosed from bronchoscopic or core biopsy. NSCLC is of 3 types, adenoicarcinoma, squamous cell carcinoma and large-cell carcinoma. Neuro endocrine lung tumors arise from pulmonary neuroendocrine cells. TTF1 and CD117 also present. Small cell carcinoma infects nearly 20% of lung cancer individuals. Other malignant tumors are salivary gland tumors arising from the tracheobronchial branches. Genomics of lung cancer shows a few common genetic alterations. Modern technology allows determination of genetic alterations from minute quantity of cytology or paraffin embedded samples. Newer chemotherapies and molecular therapies are based on NSCLC subtype. Sensitivity to DNA targetting chemotherapies depends on DNA repair enzymes. Paradigm shift moving from two-part classification to highly complex biological variety of lung cancers.

Approach and Management of Hepatocellular Cancer

Dr. Divyesh Mehta

USA

Hepatocellular carcinoma (HCC) is a global problem with the incidence of > 600,000/year, increasing rapidly. About 80-90% of HCC cases occur in cirrhotic liver. In developed countries, the primary reason for development of HCC include Hepatitis C virus, Hepatitis B virus, Alcohol-related cirrhosis, Nonalcoholic steatohepatitis (NASH), metabolic liver disease. The triad of NASH, obesity and diabetes mellitus are being emphasised as important risk for increasing incidence of HCC. There are now several different scoring systems that have been developed for oncology and can be used in HCC which are Okuda scoring system, CLIP, CUPI, TNM6, JIS, GRETCH, BCLC. Alfa fetoprotein levels in the blood determine the intervention in HCC patients.

Resection is treatment of choice. Transplantation shows the survival at 1 year of 81, 3 year of 67%, 5 year of 57%. The bad signs are >5 CM, bilobar, vascular invasion, nodes and histology. No difference was observed in RFA versus resection. The size of tumors >5 cm and <5 cm have 0% and 50% 5-year survival respectively. The post RFA recurrence is a problem. RFA plus TACE or RFA plus DOX is under evaluation. TACE is palliative with down sizing with no vascular invasion showing 50% of 5 year survival. Radioembolization therapy to liver tumors results in unique imaging findings. Although the classic indication of treatment response is a reduction in tumor size, changes occur in the parenchyma that require proper interpretation of anatomic and functional images. These parenchymal changes include necrosis, lack of enhancement, and specific findings at PET and functional MR imaging. Other benign findings that can occur include pleural effusions, perivascular edema, contralateral hypertrophy, ring enhancement, perihepatic fluid and fibrosis. Complications of radioembolization, including cholecystitis, abscess and bilomas, should be recognized early in the imaging follow-up of these patients. With classic imaging findings and surrogates, response rates range from 20% to 80% in patients treated for hepatocellular carcinoma or metastatic disease to the liver. For Advanced HCC the common treatment options are hormonal therapy, tamoxifen, octreotide, systemic chemotherapy, doxorubicin, 5-FU, Gemcitabine, Capecitabine, Thalidomide, Platinum, interferon and newer agent Sorafenib. Morphology, pathology and biology are to be considered first for the decision analysis. The current need is a new classification, MVI, molecular targets, stem cell data and new drugs.

Management of Complications of Chemotherapy

"Little compelling evidence for use of neuroprotective agents in prevention of platinum induced neuropathy (PIN). patient teaching is key step in management of pain."

DR.P.N.MOHAPATRA

No role of octreotide, oral racecadotril and oral neomycin in prevention of flouropyrimidine induced diarrhea

Dr Ashish Gupta

Zoledronic acid (4 mg every 6 months) prevents AIBLin postmenopausal women and combined with Aromatase Inhibitor may optimize patient outcomes

Dr. Tarini Prasad Sahoo

When patients begin EGFR TKI therapy, they should be advised to Employ a proactive approach in managing skin reactions. Patients should be suggested to use use a thick, alcohol-free emollient cream, sunscreen of SPF 15 or higher, preferably containing zinc oxide or titanium dioxide. .

Dr .Sushree Parida

About 82–96% of those receiving chemotherapy suffer from fatigue during their treatment. We need to question ourselves " Do we really assess fatigue?" Well planned management with Education and counseling, pharmacological and nonpharmacological interventions would benefit patients with chronic fatigue syndrome"

Dr. Chirag Desai

Highlight of the day

- IPersonalization of chemotherapy in breast cancer
- Integrating targeted therapies in MBC
- ISMPO oration
- Molecular markers to aid in treatment of colorectal cancer
- GCRI presentation
- All India Institute of Medical Science presentation

Awards

- Oral award presentations

Glioma and brain mets
- Dr. N. Syed Ismail

Head and Neck cancer
- Dr. Dhiraj Khadakban
- Dr. Bhavesh Bang
- Dr. Mukul Goyal

Lung cancer
- Dr. Rahul Narayan Maddi

DLBCL
- Dr. Arun Philip

Ovarian Cancer
- Dr. Rejiv Rajendranath

Testicular Cancer
- Dr. David Praveen Kumar

Colon Cancer
- Dr. V.L. Balaji

Growth Factors
- Dr. Brijesh Arora

Panel Members of 11th Feb 2011

Lung Cancer

Functional Preservation of Larynx

Complications & its Mgt in HNC

CML

Host Institute presentation

Acharya Harihar Regional cancer Centre and Others

Surgery

Dr. Sanjoy Panda

CUTTACK

Bhuvaneshwar is the second largest tobacco chewers state in India (57%). Now a days, there is a increase in 25-35 aged group patients. The surgery is post –op adjuvant RT, CT is NACT, concurrent, palliation. The oral lesions include Normal mucosa, Pre malignancy and malignacy. Multistep process of carcinogenesis that progresses slowly over a period of time from normal, through dyplasia, to invasive carcinoma. The diagnosis is done through Clinical examination, Scrape cytology, Hp study and others such as oral CDX. The high risk factors are caused by tobacco and alchohol and the sites involved are ventral tongue and floor of mouth. The management involves observation of high risk reduction, ablation surgery, laser, photodynamic therapy and chemoprevention. A survey conducted among 200 engineers revealed that 22% were addicted to gukta and 10% had OSF. OSF is predicted to be very important among, 5 million Indians approximately 0.5% population are surviving with OSF. The orally taken drugs which medically manage OSF are antioxidants, vitamin-A and locally taken are Collagenase, Hyluronidase, Hydrocortisone and the surgical options are opening the mouth by surgery, physiotherapy maintains it. Medicines and abstinence slowly reverts back the mucosa to normal. The current applicable procedures are laser assisted island cuts, mandibulary mucoperiosteal flap, laser assisted coronoidectomy and platysma myocutaneous flap. Laser has substituted knife for most work in trismus surgery offering a better option. The principles are Island of mucosa, scarring strech in opposite direction and mucolisation of the buccal mucosa. Statistics was taken by 25 patients were excellent results were shown in 22 where more than 3.5 cm opening was observed. Release of fibrous bands in OSF by CO_2 laser is a new, effective and less morbid procedure. Physiotherapy and avoidance is equally important for the success of surgical procedure.

Head and Neck Squamous Carcinoma: Oral Therapy

Dr. Ghanashyam Biswa

BHUBANESWAR

Chemotherapy shows response rate ranging from 12% to 37%. The biological agents available currently are cetuximab and nimotuzumab. The future agents are EGFR inhibitors, CDK inhibitors, Src inhibitors, STAT3 inhibitors, Gene therapy: p53, HPV vaccine, COX-2 inhibitors, Green tea extract, FTI. Oral metronomic therapy protocol with Celecoxib + Gefitinib +/Curcumin over 8 months. An early response, symptomatic benifit, disease stabilization, less analgesic usage, less response in alveolar and cutaneous lesion, well tolerated, and good compliance was observed. In an another protocol low dose Lapatinib with fatty food (failed Gefitinib based) showed symptomatic benefit, disease stabilization, less analgesic usage, and acceptable toxicity.

"It's time we pay more, not less, attention to the concept of cure and give our patients the benefit of doubt"

Nanotechnology & Nanopaclitaxel

Application of nanotechnology in oncology aids in diagnosis, treatment and toxicity reduction. In chemotherapy setting, nanotechnology application enhances the efficacy, reduces the toxicity and facilitates ease of administration. Paclitaxel was extensively used before the development of nanotechnology, which resulted in the development of first modulated paclitaxel – nanopaclitaxel without any structural modification ensuring the better efficacy (because of same dose, better distribution, possibility of higher drug dosage, better drug delivery to desired site-targeted delivery), with change in pharmcokinetic, mode of administration and vehicle or carrier which results in ease of adminstration. Albumin bound Paclitaxel, nanopaclitaxel of Fresinus Kabi and other nanopaclitaxel. Thus, these features shows advantage of nanotechnology paclitaxel over conventional paclitaxel. The panel discussed the advantages and disadvantages with the currently availble nanopaclitaxel, based on the evidence and experience. **Moderated by Dr. Dinesh Pendarkar, president of ISMPO with Panel: Dr. Kumar Prabash, Dr. Chanchal Goswamy, Dr. Biswas, Dr. Ravimohan and Dr. Dash.**

Hypersensitivity Reactions: There has been no hypersensitivity reactions reported in the Indian study with nanopaclitaxel with no premdication requirement. But the prescribing informations of one of the product state suggests on the remote possibility of the hypersensitivity reactions. On the neurotoxicity front, there has been not much difference in the incidence of neurotoxicity among the nanopaclitaxel, however, there were studies with Abraxane which clearly shows high incidence of neurotoxicity which was discussed and the question of similarity was unclear. Hematological toxicity needs to be watched for. On the efficacy dose to dose comparison, there was no much difference among the nanopaclitaxel but better than conventional paclitaxel. Weekly regimen can be used as an option with nanpaclitaxel. The nanopaclitaxel shows a clear cut advantage of cost benefit compared to conventional paclitaxel due to toxicity and premedication. In radiosensitising and neoadjuvant setting data is required, however, newer technologies could aid in better outcomes.

Experience Shared

Dr. Ravi Mohan: Nanopaclitaxel found to be effective in rechallanged patients with his experience in ovarian cancer patients prior treatment with paclitaxel. However, rechalenging with paclitaxel sould worsen the neurotoxicity, if patients have existing Paclitaxel. Nanopaclitaxel shows good tolerability in cardiac patients.

Dr. Biswas: In patients with poor performance status, nanoparticle Paclitaxel is well tolerated with improved response.

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