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2011 European Multidisciplinary Cancer Congress

23-27 September, 2011
Stockholm, Sweden

SUMMARY

2011 marked the advent of the European Multidisciplinary Cancer Congress (EMCC), created from a merger of the 16th European CanCer Organisation (ECCO), 36th European Society for Medical Oncology (ESMO), and 30th European Society for Therapeutic Radiology and Oncology (ESTRO) Congresses. The 2011 EMCC capitalized upon the success of the first joint ECCO-ESMO Congress two years ago and the strong collaboration among European cancer organizations, including contributions from European Society of Surgical Oncology (ESSO), European Association for Cancer Research (EACR), European Oncology Nursing Society (EONS) and European Society for Pediatric Oncology (SIOPE). The EMCC emerged as a truly pan-European cancer community effort.

Held in Stockholm, Sweden, 23 - 27 September 2011, EMCC drew 12,768 delegates, 1683 exhibitors and 423 guests. Congress coverage was provided by 380 media representatives who reported on the presentations of 694 invited speakers, in all totaling 15,931 participants. Delegates came from 116 countries around the world, making this a truly global event. The USA was represented by 1144 delegates with 1065 representatives from the UK, followed closely by approximately 1000 attendees from both Germany and France. Over 1000 delegates travelled from as far as China and Japan with Australia also represented, all joining this unique platform to exchange ideas intended to shape oncology care and practice. Twitter provided a new dimension, with 3,192 "tweets" posted during the congress using the hashtag #emcc2011.

INTRODUCTION

The multidisciplinary focus of the Congress expressed in the tagline -"Integrating basic and translational science, surgery, radiotherapy, medical oncology and care"- was reflected in the scientific program developed by Co-Scientific Chairs Jean Charles Soria (ESMO) and Anne-Lise Børresen-Dale (ECCO), Vice Chairs Jean Bourhis and Peter Naredi, and Education Chairs Dirk

Schrijvers (ECCO) and Rolf Stahel (ESMO), together with the help and expertise of the Scientific Committee. The program consisted of 33 individual disciplines and offered 285 sessions, including four Presidential Sessions and 25 Proffered Paper Sessions, reporting state of the art developments in research, treatment and patient care. The Congress is grateful to the 694 cancer specialists who shared their ideas and latest data in over 2000 abstracts and 34 Late Breaking Abstracts, many containing practice-changing information, and all with thought provoking and inspiring analyses.

Fig.1. Chairpersons at the Opening session of 2011 EMCC

Participants in the landmark EMCC were welcomed by Michael Baumann (ECCO President, Congress Chair) and David Kerr (ESMO President, ECCO Board Member) with the promise of a productive conference which would set the stage for successive congress collaborations.

Fig.2. Attendees visiting the ESMO dedicated booth

Besides active participation in creation of the complex educational and scientific Congress program tracks, ESMO enhanced its visibility at the Congress with a dedicated booth, Members Launch, Award ceremony, and educational session with new guidelines presentation.

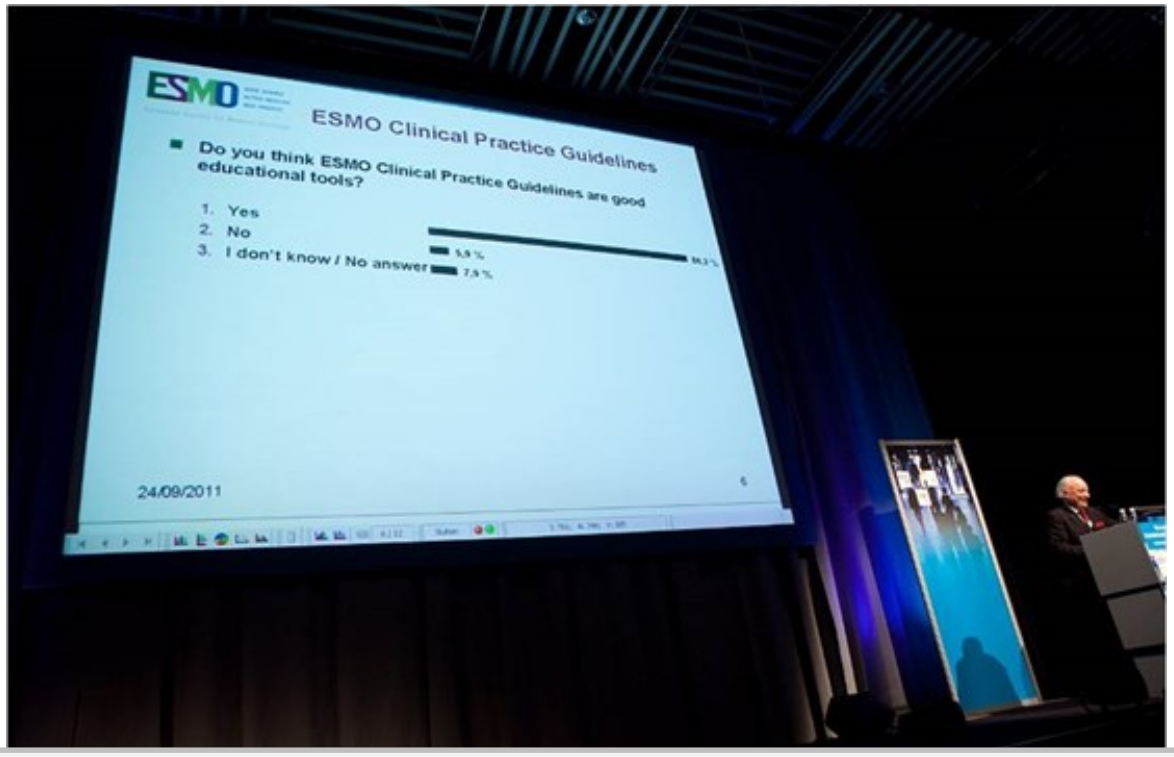

Fig.3. A detail from the ESMO Guidelines Interactive Session

This report will attempt a small overview of the expert scientific presentations made during the congress by medical oncologists, radiotherapists, surgical oncologists, basic scientists, oncology nurses, palliative care professionals and other clinicians involved in cancer diagnosis and treatment.

BASIC SCIENCE

Small-molecule inhibition of Ras oncoprotein

Fang *et al*. reported on a novel approach to target Ras, the major driver of oncogenesis in many tumor types. The group identified and characterized several small-molecules that bind a site next to the SOS interface of the Ras protein, preventing the nucleotide exchange that activates Ras oncoprotein. They screened fragments by Nuclear Magnetic Resonance (NMR) and characterized the candidates by NMR and X-ray crystallography. The subsequent effect upon Ras activation and signaling was described by biochemical and cellular assays. They tested a subset of the Ras-binding molecules and demonstrated that it blocks assembly of the Ras SOS complex by steric hindrance, thus inhibiting nucleotide exchange and preventing Ras activation and signaling. The biological activity of these compounds is being tested further in Ras mutant tumors that are dependent upon nucleotide exchange. (Fang, Abstract LBA 15)

Practice point and future research opportunities

A functionally significant binding pocket plus a compound that competitively inhibits nucleotide exchange and Ras activation have been described, providing new intervention targets for Ras driven oncogenesis

Novel mutations in SF3B1 implicate abnormalities of mRNA splicing, a pathway not previously known as a target in the pathogenesis of myelodysplastic syndromesChemotherapy during pregnancy: Large study on cognitive and cardiac outcome in children with prenatal exposure to chemotherapy

Mutations in SF3B1, a gene coding for a key protein in mRNA processing, have been identified and shown to associate with the presence of ring sideroblasts, a morphological feature indicative of myelodysplastic syndromes (MDS). Papaemmanuil et al. searched for somatically acquired point mutations across all protein-coding exons in the genomes of nine MDS patients by massively parallel sequencing and then identified these recurrent mutations in the SF3B1 gene in 619 patients. They further characterized the prevalence of these mutations by re-sequencing samples of 2087 patients

with MDS, primary cancers and cancer cell lines; SF381 was found to be mutated in 28.1% of MDS patients, 19.3 % of patients with acute myeloid leukaemia and 5.3% of patients with myeloproliferative neoplasms. Similar genetic mutations were also seen in 1% to 5% of other tumor types examined, including breast cancer, multiple myeloma and renal cancer. Mutated SF381 associated with the presence of ring sideroblasts in the bone marrow (P<0.001), which are abnormal red blood cell precursor cells that have a ring of iron-laden mitochondria around the nucleus. Of 123 MDS patients, 34 had the SF3B1 mutation; by multivariate analysis, mutated SF3B1 independently associated with improved overall survival (P=0.028) and a lower risk of leukemic progression (P=0.048). Mutations in SF381 are detectable in blood samples and offer a simple and quick method for MDS diagnosis, which usually is made by invasive bone marrow biopsy that detects ring siderobsts. Mutations in this gene may be predictive for patient outcome and useful in identifying patients with a good prognosis who may benefit from less aggressive treatment. This presentation coincided with the simultaneous publication of a paper about the research in the New England Journal of Medicine. (Papaemmanuil, Abstract LBA 11)

Practice point and future research opportunities

The close association between novel somatic acquired mutations in SF3B1 and ring sideroblasts makes this the first gene to be strongly associated with a specific morphological feature in myelodysplastic syndromes. This molecular lesion has relevant clinical significance, as it is independently associated with a favorable clinical outcome. Besides confirming relevance of this pathway in myelodysplastic syndromes and probably some other epithelial tumors, this discovery may simplify diagnosis and aid in identifying patients who will benefit from optimized treatment.

DNA vaccination against ALK in NSCLC

Voena *et al*. built upon their previous results demonstrating Anaplastic Lymphoma Kinase (ALK) to be an effective oncoantigen for ALK positive lymphoma vaccination to test whether it could also represent a feasible target for vaccination in ALK positive non-small cell lung cancer (NSCLC). They generated transgenic mice ectopically expressing human TFG- or EML4-ALK gene products, that developed multifocal adenocarcinomas similar to human tumors approximately one month after birth. The mice were then vaccinated with 50 ug of either plasmid DNA or mock plasmid DNA. The immune response was determined after 30 days showing a strong ALK specific CTL response was elicited in vaccinated mice that overcame immune tolerance to the ALK protein. Vaccinated mice showed a reduced number of neoplastic foci and a smaller tumor mass compared to controls by NMR measurement. Vaccinated mice showed a significantly increased number of T lymphocytes infiltrating both the tumors and the spared lung. It was also observed that specific CTL activity against ALK and the ability to limit the tumor expansion decreased proportionally to the age of the mice. (Voena, Abstract 1007)

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