NCRI Conference
November 2011

The major problem with the conference was the one common to all good conferences, namely choosing which sessions to attend. Sometimes the choice was clear, but on several occasions I really did want to be in two or three places at once.

The theme of stratified medicine, choosing the right treatment for the right patient, came up time and time again across various sites and treatment types. There were some surprises, particularly in the area of chemoprevention and the lack of positive effects from antioxidants. Another surprise was hearing that about 24% of all cancer patients first present as emergencies and that a similar number have been to their GP with their symptoms between three and five times before getting a diagnosis.

Three sessions in particular struck me.

The First was a session on the Monday morning titled simply "Stratified medicine". The first speaker was Albert Bardelli, from University of Torino Medical School, Italy. He spoke about personalised treatment in colorectal cancer, mentioning that one targeted therapy only benefits about 10% of predicted patients in the absence of further selection. He also made the point that it may well be that not all mutations are "equal". He was followed by Jean Charles Soria, from University XI, Paris. This presentation was about selecting the right therapies for lung cancer patients based on predictive molecular markers. He also highlighted the need for molecular input into multi-disciplinary meetings. The final speaker was David Gonzalez de Castro, from the Royal Marsden in London. He made the observation that molecular markers are only of use if the appropriate drugs are available. He then proceeded to provide a kind of checklist:

1. Find the target
2. Know your enemies & friends (+ve and -ve predictive markers)
3. Put the findings into context
4. If it is stubborn, hit it again! And it may be possible to go back to earlier treatments.

The next was to strike me was the session: Moving Towards a Molecular Rather Than a Risk-based Approach to Selecting Breast Cancer Patients Who Won't Benefit from Standard Treatment. This was hosted by David Cameron from the University of Edinburgh, who pointed out in his introduction that breast cancer is an area where a stratified approach has been validated in the now routine hormonal and HER2 targeting; with newer therapies now being developed for further sub-groups. John Yarnold from the Royal Marsden spoke first and covered the possibility of reducing the burden of radiotherapy. This was in terms both of optimising hypofractionation, partial breast radiation and dose distribution, and in identifying who doesn't need radiotherapy. He touched upon predictive markers for adverse effects.

Next was Luca Giani from Milan, who pointed out that a one-size-fits-all approach to treatment results in both over and under treatment. He made the points that ER, PR and HER2 status alone don't coincide with the molecular subtypes, and that fewer people benefit from chemotherapy that receive it under current guidelines.

The final speaker for this session was Michael Lisanti from Philadelphia. He presented a parasitic model of cancer with mitachondria powering cancer cells and the possibilities for blocking this activity.

One of the reasons that I found this session particularly interesting is that it presented a sound scientific challenge to the view often seen expressed on breast cancer fora by those having treatment for breast cancer that they want to "throw everything at it." BUT, there

is no point in "throwing at it" something that will be of no benefit but which carries a serious health risk.

The last of my Top Three was the Wednesday session on genetic predisposition and the implications for screening, surveillance and management. It considered breast, ovarian, prostate and colorectal cancers. It opened with Douglas Eaton from the University of Cambridge. He spoke about the three main areas of genetic risk for breast cancer: the rare high risk mutations (such as the well known BRCA 1 and 2 gene mutations), the rare moderate risk ones and the common low risk mutations (of which 23 have been found so far with more to come). Different loci carry different levels of risk and can also predict ER status. This could possibly be used to inform decisions on the preventative use of drugs such as tamoxifen. Risk with the common low risk mutations is polygenic and the variants combine multiplicatively. Moreover, genetic risk seems to combine multiplicatively with other risk factors (such as mammographic density). About 200 loci for common cancers have been identified so far.

The second speaker was Harry de Koning from Rotterdam. He discussed issues concerning screening starting with the point that screening programmes are directed at those of average risk, relating this particularly to breast and colorectal cancers. In higher risk populations there can be important differences that might suggest different guidelines for different groups. He illustrated this with guidelines for BRCA 1 and 2 mutation carriers. Screening guidelines are the same, but there is evidence to suggest that while adding mammography to MRI for BRCA1 mutation carriers under 40 gives little benefit, it can contribute to detection of breast cancer in BRCA 2 mutation carriers. There is a need for further work on optimal screening for mutation carriers aged over 60.

The final speaker in this session was Mark Robson from Memorial Sloan-Kettering in New York. He spoke about germline information being used to inform local, systemic and follow-up decisions. In particular it can be useful to know the risk of a second primary - not least because reconstruction options may be more limited after radiotherapy. It was interesting to hear him say that at his Centre the prophylactic mastectomy rate is high and although it wasn't always clear that surgical options had been fully discussed, that was driven by the women themselves (who presumably see no need to have lengthy discussions when they have already decided upon their course of action). He also touched upon germline defects being used as therapeutic drug targets. It has been suggested that BRCA 1 mutation cancers might be hyper sensitive to platinum, although that isn't completely clear. They do seem to respond well to taxanes. This is less so for BRCA 2 mutation cancers, where pathological complete responses are less common.

Other aspects were the possibilities of variants being implicated in radiation-induced tumours and in anthracycline cardio-toxicity.

Those were certainly the highlights for me, but I was also very interested in the plenaries and sessions on screening and prevention, improving cancer survival, survivorship research, and cancer care as disease rather than discipline based. I found the session in which patients shared their experiences of being involved in clinical trials rather disappointing, but that was partly because I was allocated to a group in which the patient who spoke was someone I'd heard speaking earlier in the year at the ABS Conference.

The Poster Abstracts catalogue for the Conference listed a total of 738 posters, displayed in two sessions and grouped thematically. A few had been withdrawn, but clearly there were far too many to see them all by browsing in the time I had available, so I had to be selective.

ICPV's poster ('Independent Cancer Patients' Voice - speaking clearly and being heard') was in Tuesday's session. It gave us an opportunity to meet people and hand out copies

of our Year Book and I thought it was very well received.

There were three posters relating to the ovarian cancer screening study I've been in and I was delighted to see that one of them (presenting a decision making model concerning risk reducing surgery) had won one of the NCRI Prize Awards for PhD students submitting an abstract as first author. Of the other two, one looked at the psychological effects of screening in the context of being recalled after an abnormal result which then turned out not to show a problem. The other investigated the reasons prompting women to withdraw from screening and have risk reducing surgery instead.

I was also interested to see another update on the AZURE trial (looking at adding zolendronic acid to standard adjuvant treatment in primary breast cancer) which suggested a need for further study to look in more detail at the effect of menopausal status.

Others that caught my eye included methods of follow-up; discussing genetic testing with family members; the efficacy of cognitive behavioural interventions to treat menopausal symptoms; increasing recruitment to clinical trials; the provision of information; the prevalence of low vitamin D levels in breast cancer (this one from my own cancer centre) and suggesting that routine testing and, where appropriate, supplementation should be offered.

There was a poster on lifestyle habits and late toxicity after radiotherapy for prostate cancer that I particularly admired for its clarity and beauty of layout.

That is just a brief selection of those I stopped to read. I could easily have spent a whole morning or afternoon on each of the two sessions looking at the posters and chatting with those presenting them, but there just wasn't enough time for that.

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