FEC100) ± H without unexpected toxicity. All pts with hormone receptor-positive tumours received adjuvant hormonal therapy.

Conclusions: These preliminary data confirm the safety of the proposed XT±H combination as neoadjuvant therapy for LABC, with promising pCR rates in the XT+H arm. Weekly XT is particularly attractive for treating HER2-positive tumours because it can be combined with H, the latter being cleared during the perioperative phase, thereby avoiding the risk of overlapping cardiac toxicity with anthracyclines. Additional data on recent pts will be presented at EBCC.

375 Poster

Switching from tamoxifen (TAM) to aromatase inhibitors (AIs) in the adjuvant treatment of breast cancer (BC) patients (pts). Results from the NORA study

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NORA study aimed at investigating modalities of treatment and pattern of relapse in 3511 BC pts, radically treated with surgery during the period 2000–2004, in 77 Oncological Centres.

Switching from TAM to AIs is one of the currently available options for the treatment of BC pts for whom hormone therapy is indicated. Recent trials are suggesting that switching to an AI during the period of the adjuvant therapy improves Disease-Free Survival (DFS) and Time to Distance reccurrence (TTDR). We analysed the switching from TAM to AIs in those patients who received either hormone therapy (HT) alone or chemotherapy (CHT) followed by HT as adjuvant treatment for early BC. 2388 out of 3511 (68%) started TAM, either as the sole therapy (35.3%) or after CHT. AIs have been administered as front-line choice in 392 pts (14.1%). Median age of pts receiving TAM was 58.6 (24–92). Switch was applied in 357 pts (17.2%). Median age of the switched pts was 62 (31–92). TN stage was T1N0 (34.6%), the therapy they have received was CHT followed by HT (59.7%). Median time to switch was 14.8 months (6.9–25.5). Main reasons for switching were gynecological toxicity (47%) and cardiovascular events (11.4%).

Switching from TAM to AIs was a current practice already some years ago, mainly due to TAM adverse events on gynecologic and vascular districts. Estimated DFS in the switched pts are currently under evaluation.

376 Poster

Preoperative dose-dense sequential chemotherapy of epirubicin/cyclophosphamide followed by docetaxel/capecitabine in patients with early breast cancer: preliminary results

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Aims: To evaluate the activity and safety of a dose-dense, sequential chemotherapy of epirubicin/cyclophosphamide (EC) followed by docetaxel/capecitabine (DXe) given preoperatively in patients with early breast cancer not candidate to breast-conserving surgery.

Patients and Methods: This was a Simon's two-step phase II study, recruiting 24 patients in the first step, and an additional 17 patients in the second step (total of 41 patients). Patients with untreated operable breast cancer (T2–3, N0–2, M0) received four cycles of EC (cyclophosphamide, 600 mg/m^2 and epirubicin, 90 mg/m^2) q2 weeks, followed by two cycles of DXe (docetaxel, 36 mg/m^2 days 1, 8, and 15 and capecitabine, 1250 mg/m^2 days 5–18) q 28 days, with pegfilgrastim support. The primary end point was the incidence of pathological complete response (pCR) in the breast. Secondary end points were clinical response, rate of breast conservation, and safety.

Results: Twenty-four out of 26 enrolled patients were evaluable for response to treatment (one patient withdrew from the study for G4 neutropenia after the first EC cycle, and the other for therapy refusal after the 4 EC cycles). A pCR was observed in 6 patients for a total pCR rate of 25%. This met the requirements of the study design for the first step. A clinical response (CR or PR) detected by palpation and by imaging was observed in 23 patients, for an overall response rate of 96%. The rate of breast-conserving surgery was 75%. The treatment was well tolerated: one patient experienced G3 mucositis and another patient required a 25% dose reduction of capecitabine because of hand-foot syndrome.

Conclusion: The dose-dense sequential combination EC/DXe is endowed with good antitumor activity and limited toxicity, allowing a high rate of pCR and breast conservation. Accrual is continuing up to the estimated sample size.

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377 Poster

The expression of Her-2, IGF-1R, IGF-1, ER alpha and ER beta in tamoxifen resistant breast cancers

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Introduction: Tamoxifen resistance poses a significant problem in treating certain oestrogen receptor(ER) positive breast cancers. Recent evidence suggests that over expression of HER-2 receptors and increased signaling in growth factor receptor pathways (EGFR-HER-2, IGF-1R) are responsible for this resistance. The role of ER-beta in Tamoxifen resistance remains controversial. This study examines the gene expression difference in Tamoxifen sensitive and resistant breast cancer.

Methodology: Tamoxifen-resistant breast cancer samples from 11 patients who failed to respond to neoadjuvant Tamoxifen (*Group A*) were obtained. Samples were also obtained from 11 patients who developed recurrence or metastasis at least 1 year after surgery and taking adjuvant Tamoxifen(*Group B*). Samples from 14 patients who had these criterias were employed as Tamoxifen-sensitive controls (*Group C*): considered high risk of developing further metastasis (grade 3 with lymph node metastasis); started on adjuvant Tamoxifen; did not develop evidence of recurrence or metastasis at 6-year follow-up. All samples were ER positive on immunostaining. Relative expressions of ER-alpha, ER-beta, HER-2, IGF-1 and IGF-1R in all these samples were measured using real-time quantitative PCR.

Results: Mean ER-alpha expression was slightly lower in Group A when compared to Group B and C. However, there was little difference in the other gene expressions in all the groups. In Group A, IGF-1R levels significantly increased with HER-2 levels ($P = 0.010$). IGF-1R increased marginally with ER-alpha in Group C ($P = 0.098$) and Group A ($P = 0.000$). In Group B and C, IGF-1 increased significantly with HER-2 and ER-alpha but not in the Group A samples. Only Group C showed a positive correlation between ER beta and IGF-1 ($P = 0.003$) samples. ER-beta increased with IGF-1R in all groups.

Conclusion: Mean ER-alpha was lower in tamoxifen resistant breast cancers. However a larger sample size in future studies is required to detect significant difference in gene expression in these groups. The relationship between (EGFR-)HER-2 and IGF-1R in Tamoxifen-resistance the samples suggests that cross-talk between the two growth factor pathways may contribute to tamoxifen resistance. The stimulatory effect of IGF-1 on ER alpha and ER beta expression may be lost in Tamoxifen-resistant cancers. The role of ER beta in tamoxifen resistance may lie in this relationship.

Thursday, 23 March 2006 16:00–16:45

POSTER SESSION

Locally advanced and recurrent disease

378 Poster

The effect on survival of local recurrence (LR) after breast conserving surgery

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This study is of 687 consecutive cases treated at Nottingham City Hospital in 1990–96 by breast conserving surgery (BCS). Cases were analysed by the Nottingham Prognostic Index (NPI).

Local recurrence (LR) is defined as recurrence within the parenchyma or skin of the treated breast; nodal recurrence is NOT included.