Results: In total, 75 patients with 82 tumours were treated with NACT during the 2 year period. Of these, 31% of the tumours showed pCR. HER2 positivity ($P = 0.011$) and higher Ki67 proliferation index ($P = 0.044$) were significantly associated with pCR using Fisher's exact test and independent *t*-test, respectively. These findings were confirmed on logistic regression, which showed that the strongest predictor of pCR was HER2 positivity, with an odds ratio of 9.3 (95%CI 1.8–47.4), followed by Ki67 index (odds ratio 1.04, 95%CI 1.00–1.08, $P = 0.019$). The factors as a whole function well as a predictive model, as shown by a statically significant Omnibus Tests of Model Coefficients ($P = 0.045$).

Conclusion: Our study has shown that HER2 positivity and higher Ki67 proliferation index are predictive of pCR following NACT in locally advanced breast cancer, in keeping with previously reported findings in the published literature [1]. Although previously published studies have reported a significant association between negative oestrogen receptor status and pCR [1], this was not demonstrated in our study.

Reference

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The Impact of the Addition of Pertuzumab to Neoadjuvant HER2 Targeting on Pathological Complete Response (pCR) Rates: Kent Oncology Centre (KOC) Experience

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Purpose: Neoadjuvant chemotherapy (NACT) with dual HER2 targeting improved pCR rates (ypT0ypN0) compared with herceptin plus chemotherapy in the TRYPHAENA and NeoSphere registration trials. Pertuzumab-containing regimens FEC-THP and TCHP were adopted at KOC following NICE approval (December 2016).

Methods: A retrospective case notes study of 126 (stage I–III) HER2-positive breast cancer patients receiving HER2-directed NACT at KOC was conducted. Single targeting treatment used FEC-TH. Dual targeting regimens were FEC-THP or TCHP. Patients were well matched for age, clinical stage and ER status.

Results: Overall, dual targeting increased pCR rates: 61% (dual) versus 29% (single). pCR rates were higher with dual targeting regardless of ER or nodal status. The highest pCR rate was seen among ER-negative patients receiving dual targeting treatment (76%). Among the 71 patients who received dual target regimens, 23 received FEC-THP and 48 TCHP. Nodal involvement was similar between the two regimens (52% versus 56% node positive, respectively). There were more ER-positive patients in the TCHP group (71% TCHP versus 35% FEC-THP). Overall, pCR rates were comparable (57% FEC-THP versus 63% TCHP). Fifty per cent of ER-positive patients achieved pCR, regardless of regimen. However, nearly all 14 ER-negative patients treated with TCHP achieved pCR (93%) compared with 60% in the FEC-THP group.

Conclusion: A substantial increase in pCR rates was observed with dual targeting, regardless of ER and nodal status, reproducing the registration trial data in real-world clinical practice. pCR rates were greatest in ER-negative patients, regardless of regimen. Ninety-three per cent of the ER-negative subset treated with TCHP achieved pCR.

Identifying and Managing the Barriers to Exercise During and Following Breast Cancer Treatment

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Purpose: Regular exercise has been shown to ameliorate many of the physical consequences of breast cancer treatment, including pain, fatigue and nausea [1]. Furthermore, a large meta-analysis demonstrated that higher physical activity was associated with reduced breast cancer-specific mortality as well as overall mortality [2]. It has been determined that only 37% of patients meet the recommended 150 min of moderate or 75 min of vigorous activity per week [3]. Given the benefits we wanted to promote regular exercise to our patients.

Methods: A qualitative survey was used to identify baseline knowledge of the evidence and to establish the barriers to exercise in a West London breast cancer clinic.

Results: Twenty-four patients were surveyed. Fifty per cent were over 50 years and two-thirds were receiving some form of treatment. All respondents indicated a desire to exercise more and the majority indicated that their exercise frequency had decreased post-diagnosis. The main barriers to exercise identified were fatigue (40%), pain (20%), difficulty accessing gyms/sports facilities (20%) and concerns about exercising while on treatment (10%). Half of those surveyed felt they lacked adequate information about exercising after being diagnosed and 60% were not aware of the health benefits in relation to breast cancer.

Conclusion: The main barriers established were a combination of perceived personal physical limitations, lack of knowledge of the benefit and lack of access to a programme. The information gained was used to promote access to existing resources and raise awareness in our clinics using verbal and written information, including a specially designed info-graphic leaflet created in collaboration with Maggie's Centre. This outlines the benefits of exercise and signposts to local support to address their personal exercise barriers and provide a suitable exercise programme. This leaflet is to be distributed to patients at diagnosis and again at entry into the Open Access Follow Up Clinic.

References

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Pretreatment DPYD Genotyping Reduces the Risk of Capecitabine-associated Severe Toxicities: a Prospective Validation Study

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Purpose: Capecitabine is an oral fluoropyrimidine used for the treatment of metastatic breast cancer (m-BC) [1]. Its catabolism depends on the enzyme dihydropyrimidine dehydrogenase (DPD). Polymorphic variants in the DPYD gene are associated with partial or complete DPD activity and severe toxicities to fluoropyrimidine therapy [2]. Preliminary work previously carried out in our institution suggested that prospective DPYD genotyping is a cost-effective and predictive biomarker of capecitabine toxicity. The aim of this study was to validate these findings.

Methods: We reviewed medical records for consecutive patients with m-BC who underwent DPYD genotyping prior to commencing capecitabine in our institution between December 2014 and December 2017. Patients were tested for four DPYD sequence variants associated with reduced DPD activity. We collected data on patient demographics, presence of DPYD polymorphism and capecitabine-associated toxicities.

Results: Sixty-two patients underwent DPYD testing; average age 58 years (range 28–85). Five (8.4%) were found to carry DPYD genetic polymorphisms associated with reduced DPD activity. Of these, three received alternative chemotherapy, whereas two received dose-reduced capecitabine (50% of dose) without complications; one of these had the dose increased to 75% for cycle 2, resulting in grade 3 plantar-palmar