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Will immunohistochemistry trump genomics in triple negative breast cancer?

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Background: Triple receptor negative breast cancer (TRNBC) refers to an aggressive subtype of breast cancer which has either a very low or no expression of the estrogen receptor (ER), progesterone receptor (PR) and human epidermal growth factor 2 receptor (HER2). We wished to see if there might be a relationship between genomics and immunohistochemistry (IHC) in TRNBC.

Methods: From a total of 134 TRNBC patient records in the Meridian Health tumor registry, 15 were randomly selected for preliminary evaluation. Tissue samples, obtained pathology blocks, underwent molecular testing at NeoGenomics Laboratory and IHC staining from Jersey Shore University Medical Center. The intensity and quantity of tumor staining of paraffin embedded tissue was evaluated for CK5, CK7, CK8, CK14, CK18, CK19, vimentin, CD44, c-kit, P53, P63, topoisomerase II, AR, Ki-67, and GATA-3.

Results: Twenty-six genetic mutations were seen in 15 patients. The mutations found in these TRNBC patients included AXIN1, ARID1A, BAP1, BARD1, BRCA1, BRCA2, CCND1, CREBBP, EGFR, EP300, JAK1, KMT2C, NOTCH3, NRAS, PALB2, PBRM1, PD-L1, PIK3CA, PMS2, PRKCI, RB1, RNF43, RPTOR, SDHB, TERT, and TP53. Of the following frequently seen mutations, i.e. PD-L1, PIK3CA, and TP53, eight patients had one mutation. Five patients had two mutations. Of those five, one patient had PIK3CA and PD-L1, one had PIK3CA and TP53, and three had TP53 and PD-L1. One patient had all three mutations while another had none of these abnormalities. Frequent mutations were seen in multiple patients including KMT2C in three patients, PD-L1 in five patients, PIK3CA in six patients, and TP53 in ten patients. In the PIK3CA patients, Afinitor (everolimus) was the only associated FDA approved therapy. The NRAS, PIK3CA, and TP53 results suggested possible (but non-specific) therapy resistance. Potential clinical trials were suggested by the PIK3CA and TP53 results. Three of the most commonly observed abnormalities had at least one associated IHC marker. Patients with PD-L1 tended to have higher staining scores with CK14 and vimentin. Patients with PIK3CA had lower staining scores with CK5, CK18, CK19, Ki-67 and topoisomerase II than those without PIK3CA. Patients with TP53 had higher staining scores with CK5. There were too few cases to comment on the clinical course and pathology findings.

Conclusions: Our preliminary study shows a possible association between genomics and IHC in TRNBC. Concordance of readily available IHC with genomics could prove to be a useful clinical tool.

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