Plasma levels of immune-oncology checkpoints, chemokines and cytokines were assayed using Bio-Plex Suspension Bead Array platforms.

**Methods**


Methods

analysed using Bio-Plex Manager software 6.0 and results reported as either ng/mL or pg/mL. C-reactive protein (CRP) levels were determined by enzymatic method.

**Aim**

The primary hypothesis was that there was a significant difference in the plasma levels of soluble immune checkpoints, cytokines and chemokines in newly-diagnosed early breast cancer patients and healthy controls are shown in Table 2.

**Results**

Patient characteristics are shown in table 1. Comparison of plasma levels of immune checkpoints, chemokines, and cytokines between breast cancer patients and healthy controls are shown in Table 1.

**Conclusions**

Lower levels of a number of soluble co-stimulatory (n=6/6) and co-inhibitory (n=7/9) immune checkpoints, as well as chemokines (n=2/6) were measured in newly-diagnosed, non-metastatic breast cancer patients compared to healthy controls.

**Lab Method**

Phenotype levels of immune-oncology checkpoints, chemokines, and cytokines were assessed using Bio-Plex Suspension bead arrays (BioRad) or Bio-Rad human magnetic bead panels. The methods were followed according to the manufacturers' specifications. The data was analyzed using Bio-Plex Manager software 6.0 and results reported as either ng/mL or pg/mL. C-reactive protein (CRP) levels were determined by enzymatic method using the Cobas® 6000 c1110 assay (Beckman Instruments Diagnostics). The method was followed according to the manufacturer's instruction. Results are reported as ng/mL.

**Statistical Methods**

The statistical hypothesis was that there was a significant difference in the plasma levels of soluble immune checkpoints, cytokines and chemokines between breast cancer patients and healthy controls. Description statistics were used to describe patient characteristics. The Mann-Whitney U test was used to compare levels of the various test biomarkers between breast cancer patients and healthy controls. Fisher’s exact or Chi-squared tests were used for the analysis of categorical variables. NCSS software version 11 for Windows (USA) was used for statistical analysis.

**Background**

For effective killing of cancer cells an antitumor immune response, a series of events involving different immune cells needs to be initiated and allowed to proceed. The steps in the cancer immunity cycle start with the release of tumor cell antigens, which are recognized by and lead to the killing of cancer cells by cytotoxic T cells. This immune response is instigated by a variety of stimulatory and inhibitory factors.

T cells need two signals for activation: binding of the TCR (T cell receptor) to the MHC (major histocompatibility complex) and activation of co-stimulatory molecules.

Immune checkpoints can stimulate or inhibit these events thereby regulating the functions of immune cells.

Accordingly, checkpoints play important roles in the maintenance of immune homeostasis; immune checkpoints can stimulate or inhibit these events thereby regulating the functions of immune cells.

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