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Immune System Functioning In Renal Cancer Patients

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Commentary

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DESCRIPTION

Few immunotherapy drugs have established a direct correlation between the product's projected mode of action and patient benefit. Because cancer vaccines are anticipated to have a delayed therapeutic effect, identifying the active moiety may allow for the development of an early effectiveness marker. In a multicentre phase III trial, patients with renal cancer who needed first-line treatment for metastatic disease were randomised 1:1 to receive MVA-5T4 or placebo in addition to Sunitinib, IL-2, or IFN-. Following the third and fourth immunizations, antibody responses were measured.

A 5T4 antibody response surrogate (the immune response surrogate; IRS) was produced and then utilised in a survival study to determine therapy benefit. A total of 733 patients were randomised, and 590 patients' immunological responses were evaluated. Within the MVA–5T4-treated group, a strong antibody response to 5T4 was associated with longer life. In the phase III investigation, the IRS was demonstrated to be a significant predictor of treatment benefit as a linear combination of pre-treatment 5T4 antibody levels, haemoglobin, and haematocrit.

Importantly, in an independent dataset of renal, colorectal, and prostate cancer patients treated with MVA–5T4 in phase I–II studies, the IRS was also related with antibody response and survival.

Agents that target the immune system were once the standard of care in metastatic Renal Cell Carcinoma (RCC), but they have been largely displaced by more targeted therapy. Recent discoveries on the regulation of an anti-tumor immune response have resulted in the development of medicines that can stimulate immune responses solely within the tumour, allowing for the prospect of attaining a long-lasting tumour response in the absence of

severe systemic damage. Furthermore, a greater understanding of tumour immunology has increased the possibility of establishing predictive biomarkers of immunotherapy response.

The presence of a cellbound tumor-distinctive immune response was investigated in patients with renal adenocarcinoma (hypernephroma). In a 'Combined Lymphocyte Target Interaction', MLTI-test, the patient's peripheral lymphocytes were mixed *in vitro* with autochthonous malignant and non-malignant cells, as well as allogeneic lymphocytes who's DNA-synthesis had been stopped by Mitomycine-C. If the tumour cells have anything on their surface that makes them foreign to the patient's non-malignant cells, such as tumor-specific antigens, the patient's own lymphocytes should be stimulated to boost DNA-synthesis by autochthonous tumour cells but not by autochthonous non-malignant cells. Peripheral lymphocytes from three of the first six patients studied showed enhanced DNA synthesis after *in vitro* contact with autochthonous kidney cancer cells.

Kidney cancer, particularly clear cell Renal Cell Carcinoma (ccRCC), has long been thought to be immunotherapy sensitive. With recent advances in immunotherapy for solid tumours and the recent approval of the first immune checkpoint inhibitor for ccRCC, we are reviewing the history of immunotherapy in kidney cancer, describing its current state, and looking into the future of a rapidly evolving landscape in kidney cancer immunotherapy. Dendritic cells are the most powerful immune response stimulators, including anticancer responses.

Sunitinib and Sorafenib, multikinase inhibitors, not only decrease angiogenesis and tumour growth, but they also have the potential to interfere with immune system function. Trials combining these multikinase inhibitors with various sorts of immunological modification, particularly cellular treatments, should be sensibly structured to account for all of these complicated consequences, which ultimately warrant more research.

The use of autologous antibody to screen cDNA expression libraries obtained from human malignancies (SEREX) is a potent tool for identifying the structure of tumour antigens recognised by the humoral immune system. SEREX study of 4 renal cancer patients discovered 65 unique antigens (NY-REN-1 to NY-REN-65) reactive with autologous IgG and described them in terms of cDNA sequence, mRNA expression pattern, and reactivity with allogeneic sera. REN-9, -10, -19, and -26 have been linked to human cancer.

REN-9 (LUCA-15) and REN-10 (gene 21) are located on chromosome 3p21.3, near the tumour suppressor gene locus for small cell lung cancer. LKB1/STK11, a gene that is faulty in Peutz-Jeghers syndrome and cancer, is equivalent to REN-19. The bcr gene, which is involved in the (t(9:22)) bcr/abl translocation, encodes REN-26. Three of the antigens in the series have different mRNA expression patterns; REN-3 has a pattern of tissue-specific isoforms, and REN-21 and REN-43 are expressed at a high level in testis compared to 15 other normal organs.

The remaining 62 antigens were widely expressed in normal tissues. In terms of immunogenicity, 20 of the 65 antigens exclusively reacted with autologous sera. Thirty-three antigens responded with sera from healthy donors, demonstrating that they are not just immunogenic in malignancy. The remaining 12 antigens reacted with sera from 5%–25% of cancer patients but not with normal donor sera. Seventy percent of the patients with kidney carcinoma exhibited antibodies against one or more of these 12 antigens.