Solid tumor immunotherapy

With the advent of cancer immunotherapy, there has been a major improvement in patients' quality of life and survival. The growth of cancer immunotherapy has dramatically changed our understanding of the basics of cancer biology and has altered the standards of care (surgery, radiotherapy, and chemotherapy) for patients. Cancer immunotherapy has generated significant excitement with the success of chimeric antigen receptor (CAR) T cell therapy in particular. Clinical results using CAR-T for hematological malignancies have led to the approval of four CD19-targeted and one B cell maturation antigen (BCMA)-targeted cell therapy products by the US Food and Drug Administration (FDA). Also, immune checkpoint inhibitors such as antibodies against Programmed Cell Death-1 (PD-1), Programmed Cell Death Ligand-1 (PD-L1), and Cytotoxic T-Lymphocyte-Associated Antigen 4 (CTLA-4) have shown promising therapeutic outcomes and long-lasting clinical effect in several tumor types and patients who are refractory to other treatments. Despite these promising results, the success of cancer immunotherapy in solid tumors have been limited due to several barriers, which include immunosuppressive tumor microenvironment (TME), inefficient trafficking, and heterogeneity of tumor antigens. This is further compounded by the high intra-tumoral pressure of solid tumors, which presents an additional challenge to successfully delivering treatments to solid tumors [1].

Lu et al. showed that a bispecific nanobioconjugate that enables the decoration of SLAMF7 on the surface of solid tumors induces robust phagocytosis and activates the phagocyte cyclic guanosine monophosphate-adenosine monophosphate synthase-stimulator of interferon genes cGAS-STING pathway, sensitizing the tumors to immune checkpoint blockade. The findings support an immunological conversion strategy that uses nano adjuvants to improve the effectiveness of immunotherapy for solid tumors [2].

Immunotherapy has had considerable success in treating several types of solid cancers, such as melanoma and lung adenocarcinoma [3].

Glioma immunotherapy

Glioma immunotherapy.

Risks

Patients who receive immunotherapy (IT) alone may have an increased rate of Radiation induced necrosis RN/treatment-related imaging changes (TRIC) compared with those who receive chemotherapy (CT) or targeted therapy (TT) alone after stereotactic radiosurgery, whereas receiving any CT may in fact be protective against RN/TRIC. As the use of immunotherapies increases, the rate of RN/TRIC may be expected to increase compared with rates in the chemotherapy era [4].

Metastases

Immunotherapy for metastases.

1)
Guha P, Heatherton KR, O'Connell KP, Alexander IS, Katz SC. Assessing the Future of Solid Tumor Immunotherapy. Biomedicines. 2022 Mar 11;10(3):655. doi: 10.3390/biomedicines10030655. PMID: 35327456; PMCID: PMC8945484.
2)
Lu Y, Huntoon K, Lee D, Wang Y, Ha J, Qie Y, Li X, Schrank BR, Dong S, Gallup TD, Kang M, Zhao H, An Y, Yang Z, Li J, Kim BYS, Jiang W. Immunological conversion of solid tumours using a bispecific nanobioconjugate for cancer immunotherapy. Nat Nanotechnol. 2022 Nov 10. doi: 10.1038/s41565-022-01245-7. Epub ahead of print. PMID: 36357792.
3)
Drake CG, Lipson EJ, Brahmer JR. Breathing new life into immunotherapy: review of melanoma, lung and kidney cancer. Nat Rev Clin Oncol. 2014 Jan;11(1):24-37. doi: 10.1038/nrclinonc.2013.208. Epub 2013 Nov 19. PMID: 24247168; PMCID: PMC4086654.
4)
Colaco RJ, Martin P, Kluger HM, Yu JB, Chiang VL. Does immunotherapy increase the rate of radiation necrosis after radiosurgical treatment of brain metastases? J Neurosurg. 2015 Nov 6:1-7. [Epub ahead of print] PubMed PMID: 26544782.