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Immune checkpoint inhibitors in the treatment of glioblastoma multiforme

POST-CONFERENCE REPORT

ADRIANNA WINNICKA

University of Lodz, Faculty of Biology and Environmental Protection, Pomorska 141/143, 90–236 Lodz, Poland
E-mail: adrianna.winnicka@edu.uni.lodz.pl

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Glioblastoma multiforme (GBM) is among the most common primary tumours of the brain and central nervous system, accounting for about 57% of all diagnosed forms of gliomas. An estimated 5–6 per 100,000 people are diagnosed with GBM each year. Despite extensive research, the median survival of patients diagnosed with GBM is only 12–15 months after diagnosis.

Standard treatment of GBM is based on a multidisciplinary approach – maximal surgical resection, radio-chemotherapy with temozolomide (TMZ) and oral complementary chemotherapy with TMZ. However, due to frequent chemo-resistance and the inability to completely resect the tumour to the exclusion of compromised normal tissue, patients often experience recurrence or progression of the disease. Nevertheless, innovative therapies for GBM are constantly being researched. One of them is immunotherapy, the main goal of which is to mobilize the patient's immune system to recognize and eliminate tumour-transformed cells.

Currently, immunotherapy for GBM includes the use of immune checkpoint inhibitors (ICIs), including mAb anti-PD-1 IgG4, (Niwolumab, Pembrolizumab, Cemiplimab), mAb anti-PD-L1 IgG1 (Atezolizumab, Durvalumab, Avelumab) and mAb anti-CTLA-4 IgG1 (Ipilimumab). ICIs are monoclonal antibodies that, by blocking PD-1 (programmed death receptor 1) or CTLA-4 (cytotoxic T cell antigen 4) proteins, lead to the activation of T cells that fight cancer cells. PD-1 is a protein receptor that inhibits immune activation by binding to PD-L1 and PD-L2 ligands. In contrast, the mechanism by which CTLA-4 inhibits T-cell activation involves competition for ligands with the CD28 molecule.

References

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