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Commentary

Application of Immunotherapy in Cancer Treatment

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DESCRIPTION

The immune system is the core defense against cancer development and progression. Failure of the immune system to recognize and eliminate malignant cells plays an immense role in the pathogenesis of cancer. The paramount achievement in immunotherapy particularly – Immune Check-point Inhibitors (ICI) over the recent decade has brought about major paradigm shift in cancer treatment. ICIs, represented mainly by inhibitory monoclonal antibodies – anti-cytotoxic T-lymphocyte antigen 4 (CTLA-4) (ipilimumab), anti-programmed cell death protein 1 () (PD1pembrolizumab/nivolumab/cemiplimab), Anti-PD-1 Ligand molecules (PD-L-1- atezolizumab/avelumab/durvalumab) reactivate the immune system against tumor cells but can also trigger a myriad of autoimmune side effects, termed immune-related adverse events (irAEs). Immunotherapy related adverse events typically have a delayed onset and prolonged duration compared to adverse events from chemotherapy, and its effective management depends on early recognition and prompt intervention with immune suppression and/or immunomodulatory strategies.

The present study aims to raise awareness about thyroid side effects of immune check-point inhibitors to physicians who are taking care of cancer patients and to specialists - mainly oncologists and endocrinologists who are urged to cooperate for the management of thyroid immunotoxicity.

Until recently, chemotherapy, radiation and surgery were considered the cornerstones of cancer treatment. Advances in immunotherapy have revolutionized tumors treatment. Currently, the most widely used approach is the administration of targeted monoclonal antibodies (mAbs) directed against T cell activation.

Under homeostatic conditions, there is a balance between pro-inflammatory and anti-inflammatory signaling maintained by immune checkpoints. These immune checkpoints are a set of inhibitory and stimulatory pathways that directly affect the function of immune cells. Malignant cells disrupt this balance by promoting an immunosuppressive state that favors immune evasion and tumor growth.

Cancer cells recruit or induce development of regulatory T cells (Tregs), downregulate tumor antigen expression, induce T cell tolerance and/or apoptosis and produce immune suppressive cytokines that stimulate inhibitory immune check-points. This leads to a unique and highly immunosuppressive tumor microenvironment (TME). In an attempt to overcome these immunosuppressive conditions, immune check-point inhibitors act by blocking the effects of selected inhibitory pathways. The best described immune checkpoints are CTLA-4, PD-1, PD-L-1.CTLA-4 is constitutively expressed by regulatory T cells and upregulated after T cells activation, acting as an "OFF" switch. CTLA-4 binds the B7 ligand on antigen presenting cell (APC). Binding CTLA-4, immune check-point inhibitor prevents it from binding with B7, and allows B7 to bind with CD28, in this way inducing the immune system to attack tumor cells. PD-1 is present on T, B, and NK cells, and binds to PD-L-1, expressed by tumor cells, preventing apoptosis of the cell expressing PD-L-1 by the immune system. ICIs, that bind either PD-L-1 or PD-1, prevent this process.The spectrum of thyroid disturbances under ICIs can present as thyrotoxicosis, hypothyroidism, painless thyroiditis, thyroid eye disease and occasionally fever form such as thyroid storm.The incidence of thyroid disorders differs between different ICI classes. Chart-1 represents reported frequencies of hypothyroidism (%) and hyperthyroidism (%). Thyroid dysfunctions are mostly provoked by anti-PD-1 or anti-PD-L-1 mAbs and incidence ranges from 4 to 19.5%.

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