Clinical Thyroidology[®] for the Public

THYROIDITIS

Thyroiditis while receiving immune checkpoint inhibitor therapy for non-thyroid cancers is associated with improved overall survival

BACKGROUND

Cancers are capable of producing molecules to block generation and function of immune cells. On such molecule is programmed cell death ligand 1 (PD-L1) that binds to and inhibits immune cells. Immune checkpoint inhibitors are a new class of cancer chemotherapy agents that target and block molecules such as PD-L1, thus, allowing the body's own immune system to attack and destroy cancer cells. As expected, along with positive effects, these chemotherapy agents may also induce some unfavorable changes in the immune system processes. Since autoimmune thyroid disease is a common cause of thyroid problems, it is not surprising that one of the side effects of immune checkpoint inhibitors is both hyperand hypothyroidism, which may happen up to 29% of cases. Interestingly, some previous publications reported a superior response to the cancer treatment when patients experience immune system related side effects.

This study examines the frequency, course and survival effects of newly diagnosed autoimmune thyroid disease after treatment with immune checkpoint inhibitors.

THE FULL ARTICLE TITLE

Kotwal A et al 2020 PD-L1 inhibitor-induced thyroiditis is associated with better overall survival in cancer patients. Thyroid. Epub Jan 9. PMID: 31813343.

SUMMARY OF THE STUDY

This study was conducted at the Mayo Clinic in Rochester, Minnesota. The medical records of adult cancer patients treated with one of the two PD-L1 inhibitors drugs (either atezolizumab or avelumab) were reviewed. Subjects who developed thyroid disease after starting the treatment were identified. Of 91 patients who were treated with a PD-L1 inhibitor, the majority (86, ~95%) had received atezolizumab. The average age of the patients was 67.9 years, 47% were male, and nearly 2/3s of patients were being treated for primary lung cancer. Thyroid problems occurred in 23 subjects (25%); 61% developed hypothyroidism, 22% developed hyperthyroidism and 17% developed worsening of already existing hypothyroidism. Most patients developed the thyroid problem in about 6 weeks after starting the therapy.

On average, patients were followed for 6 months after the onset of the thyroid disease. During this time, 3 patients had spontaneous recovery.

After looking at mortality rate, the researchers found that a lower number of patients who developed thyroid disease died during the study period (43.5% of patients with a thyroid dysfunction versus 79.4% patients who did not developed thyroid disease).

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

In summary, this study showed that 25% of patients who had received a PD-L1 inhibitor drug also developed autoimmune thyroid disease. Interestingly the survival rate of patients who developed this type of side effect was better compared to the rest.

As more immune checkpoint inhibitors enter the market for treatment of cancer, this and similar studies will be instrumental to improve our knowledge about them.

— Shirin Haddady, MD MPH

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THYROIDITIS, continued

ATA THYROID BROCHURE LINKS

Hypothyroidism (Underactive): <u>https://www.thyroid.org/hypothyroidism/</u> Hyperthyroidism (Overactive): <u>https://www.thyroid.org/hyperthyroidism/</u>

ABBREVIATIONS & DEFINITIONS

Autoimmune thyroid disease: a group of disorders that are caused by antibodies that get confused and attack the thyroid. These antibodies can either turn on the thyroid (Graves' disease, hyperthyroidism) or turn it off (Hashimoto's thyroiditis, hypothyroidism).

Hypothyroidism: a condition where the thyroid gland is underactive and doesn't produce enough thyroid hormone. Treatment requires taking thyroid hormone pills.

Hyperthyroidism: a condition where the thyroid gland is overactive and produces too much thyroid hormone. Hyperthyroidism may be treated with antithyroid meds (Methimazole, Propylthiouracil), radioactive iodine or surgery. Immune checkpoint inhibitors: a new class of cancer chemotherapy agents that target and block molecules produced by cancer cells to block generation and function of immune cells. This allows the body's own immune system to attack and destroy cancer cells.

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