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Commentary

Responses of Patients with Chronic Myeloid Leukemia to Tyrosine Kinase Inhibitors

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DESCRIPTION

Tyrosine Kinase Inhibitors (TKIs) prevent the cellular signaling proteins from being phosphorylated, which is necessary for the growth of tumour cells. The rising use of TKIs in cancer therapy has been associated with a number of endocrine side effects, and thyroid changes, most notably hypothyroidism, represent a well-known occurrence. Thyroid damage ranging from minor follicular cell toxicity to catastrophic thyroiditis, inhibition of thyroid peroxidase, inhibiting iodine uptake, and increased thyroid hormone clearance are just a few of the direct processes responsible for TKI-induced thyroid changes. TKIs' anti-angiogenic effects may also cause indirect thyroid harm. More recently, it has been discovered that TKI-induced thyroid dysfunction may be triggered by thyroid autoimmunity. *De novo* serum thyroid autoantibodies have been found in nearly one-third of oncologic patients monitored before and during sunitinib therapy.

In patients with various solid tumours, thyroid autoimmunity brought on by TKIs has been reported to be related with both hypothyroidism and a superior oncologic response, albeit the exact mechanism(s) involved are yet unknown. Chronic myeloid leukemia with the Philadelphia chromosome is commonly treated with TKIs (Ph-positive CML). These TKIs target the breakpoint cluster region-Abelson murine leukemia viral oncogene homolog (ABL) protein, which is oncogenic in CML patients. Imatinib (first-generation TKI), dasatinib, nilotinib, and bosutinib are the four TKIs that have been approved for use as first-line CML treatments (second-generation TKI).

All second-generation TKIs are successful in cases of failure or resistance, but the criteria for selecting second-line therapy (SLT) are patient-specific and depend on age, comorbidities, and FLT toxicity with TKIs. Only one retrospective mono-centric study has been done on thyroid function in patients with CML who are receiving TKIs treatment, aside from a recent analysis of cases reported by the U.S. Food and Drug Administration.

It demonstrating that thyroid impairment occurs frequently with treatment with first and second generation TKIs. The relevance of thyroid autoimmunity in thyroid dysfunction brought on by TKIs in CML patients, as well as any potential connections between thyroid dysfunction/autoimmunity and therapeutic response, however are not known.

The evaluation of CML therapy response is based on hematological (blood count and spleen volume), cytogenetic (presence of metaphases Ph+ in marrow cells), and molecular (evidence of BCR-ABL transcript) responses. These criteria were created by the European Leukemia Net.

Complete hematological, complete cytogenetic, major molecular and deep molecular responses-the last of which corresponds to progressively lower levels of transcript-are the successive responses obtained after TKI treatment.

The most accurate way to judge the severity of the disease, and in particular to quantify residual disease, is by Molecular Responses (MR). Quantitative RT-PCR of BCR-ABL transcript on leukocytes isolated from a peripheral blood sample is used to assess MR. This transcript's fluctuation over time during TKI treatment as compared to the value found at the time of diagnosis is indicated. To ensure the comparability of data across different laboratories, BCR-ABL transcript levels should be presented using the international scale.

A Siemens Antares color Doppler machine was used to do a thyroid Ultrasound (US)(Siemens, Medical Solutions, and Issaquah, WA). A US examination was performed to assess each subject. Imatinib has frequently been linked to hypothyroidism in patients with solid tumors since the development of TKI therapy; however there is only one mono-centric study available on the thyroid effects seen in CML patients receiving imatinib, nilotinib and dasatinib. This study revealed that thyroid dysfunction is very common (mainly mild subclinical hypothyroidism and transient destructive thyrotoxicosis, generally not requiring specific therapy).

The increase in thyroid hormone requirements caused by a rise in non-de-iodination clearance due to the stimulation of Uri dine diphosphate-glucuronosyltransferases is the most consistent mechanism of imatinib's interference with thyroid function.

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CONCLUSION

This is to blame for "consumption hypothyroidism," which is primarily, though not solely, seen in thyroidectomies individuals taking L-thyroxin. In conclusion it demonstrate that thyroid dysfunction and autoimmune disease are frequently seen in the Chronic Myeloid Leukemia patients receiving Tyrosine Kinase Inhibitors therapy and may help predict a better oncological outcome, corroborate and further our understanding of the thyroid abnormalities caused by TKIs. Since these thyroid abnormalities are typically subclinical, TKIs do not need to be stopped or reduced in dosage.