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TELOMERE LENGTH AND TELOMERASE EXPRESSION ARE SIMILAR IN FAMILIAL AND SPORADIC PAPILLARY THYROID CANCER

Jendrzejewski J, Tomsic J, Lozanski G, Labanowska J, He H, Liyanarachchi S, Nagy R, Ringel MD, Kloos RT, Heerema NA, de la Chapelle A. **Telomere length and telomerase reverse transcriptase gene copy number in patients with papillary thyroid carcinoma.** J Clin Endocrinol Metab. September 7, 2011 [Epub ahead of print].

SUMMARY

BACKGROUND

Papillary thyroid cancer (PTC) is familial in 5% to 10% of cases, but the basis for the inheritance is unknown. One group reported that patients with familial PTC had a shorter-length telomere and that there was increased telomerase activity in patients with familial PTC as compared with those who had sporadic PTC (1). Telomeres are the unique DNA caps on the ends of chromosomes that protect them from degradation. Telomerase is a reverse transcriptase enzyme that is necessary for synthesis of the telomere. The discovery of the telomere and telomerase was the basis for the Nobel Prize in Medicine in 2009. Genetic integrity is lost as telomeres shorten, and this is thought to play a role in aging.

The current study attempted to confirm the findings of the Italian group (1) in patients with familial PTC in the United States.

METHODS

The investigators studied 42 patients with familial PTC from separate families; the proband had to have at least two first- or second-degree relatives with PTC. The results were compared with 65 sporadic cases. The female/male ratio and ages were similar in the two groups. Using white cells, they measured telomere length and telomerase gene copy number by a quantitative real-time polymerase chain reaction assay. In a subset of patients from both groups and in normal controls, telomere length was measured by fluorescence in situ hybridization (FISH) and telomerase mRNA was measured.

RESULTS

The mean telomere length and telomerase gene copy number were nearly identical in both the patients with familial and those with sporadic PTC. When telomere length was measured by FISH, there was also no significant difference between the two groups of patients. In addition, the telomerase mRNA did not differ among the patients with PTC and the controls.

CONCLUSIONS

The results showed no difference between familial and sporadic PTC with regard to telomere length, telomerase copy number, or expression in this cohort, and therefore do not confirm the previous study.

COMMENTARY

It is possible that the differences between the Italian and U.S. studies are due to the selection of patients in each study. Since there is considerable discrepancy between the results of the two studies, it would be desirable to have additional studies of telomere length in familial PTC to clarify this point. From a hypothetical point of view, longer telomeres may prevent cells from degradation. However, it is also possible that increased telomerase activity is a compensatory mechanism to prevent further shortening of the telomere in some cancer cells. Increased telomerase activity has been found in more aggressive thyroid cancers (2).

Based on the current article, it is likely that the genetic basis for familial PTC is still unknown. From a clinical point of view, the only marker is the family history.

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In a recent encounter with a patient who had several first-degree relatives with PTC, I used this history as the basis for my recommendation for thyroidectomy when fine-needle aspiration biopsy of her nodule was inconclusive. In such an instance, a molecular marker could play a decisive role.

— Jerome M. Hershman, MD

References

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