HYPOTHESIS

We hypothesize that high levels of hPL could explain the worse outcomes of DTC in pregnancy.

SUPPORTING ARGUMENTS

a. Human placental lactogen and pregnancy.

Lactogens comprise three closely related peptide hormones: human growth hormone (hGH), human placental lactogen (hPL), and human pro lactin (hPRL). hPRL and hGH originate from the pituitary and hPL is secreted by syncytiotrophoblasts of the placenta. Levels of hPL vary during pregnancy; it can be detected from the sixth week of gestation, increases steadily in the first and second trimesters, and peaks at a constant level in the third.

ABSTRACT

Lactogens comprise three closely related peptide hormones: human growth hormone (hGH), human placental lactogen (hPL), and human prolactin (hPRL). hPRL only binds to PRLR, while both hPL and hGH have receptors (PRLR, GHR) in various organs. hPL binds to PRLR, GHR, and GHR–PRLR with a high affinity regardless of its low (23%) structural homology to PRL. Paradoxically, hPL binds weakly to GHR even though they have significant (85%) amino acid sequence homology. We hypothesized that high levels of hPL could explain the worse outcomes of DTC in pregnancy. A possible mechanism could be that hPL binds GHR–PRLR in thyroid tissue and promotes tumor growth. Alternatively, the high affinity of hPL for PRLRs expressed in thyroid cancer may account for these results. Conversely, hPL binding to GHR, albeit weakly, might also play a role in worsened outcomes. Additional studies must be performed in order for the pathophysiologic mechanisms to be elucidated.

INTRODUCTION

The most frequently seen endocrine tumor is differentiated thyroid cancer (DTC) and it is common in younger women. Numerous factors are known to induce thyroid cell growth in the course of normal pregnancy. Human choriionic gonadotropin, which is analogous to PRL, may have a negative impact on patients with DTC; however, no differences were found between the groups regarding estrogen receptor pattern, sodium/iodide symport (NIS) expression, and B-Raf proto-oncogene, serine/threonine kinase (BRAF) mutations. Consequently, the authors concluded that additional future studies were required because the underlying causes were yet to be revealed.

HYPOTHESIS

We hypothesize that high levels of hPL could explain the worse outcomes of DTC in pregnancy.
The possible role of human placental lactogen in worse outcomes of differentiated thyroid cancer in pregnancy

Kizilgul et al.

HYPOTHESIS

Presence of hPL in breast cancer has a negative influence on prognosis for patients. Contrary to these findings, one study did not find hPL in the serum of patients with breast cancer. The amplification of CSH genes in breast cancers was shown to be related to aneuploidy, lymph node metastases, and overexpression of the Her2/neu oncogene. Moreover, immunohistochemical labelling of hPL in carcinomas further demonstrates the relationship with gene amplification.

hPL is increased in histologic sections as well as serum with placental site trophoblastic tumours. Xiong et al. reported that the expression of placental hormones including human chorionic gonadotropin (hCG), human placental lactogen, and pregnancy-specific 1-glycoprotein in invasive moles and choriocarcinomas further demonstrates the relationship with gene amplification.

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b. Human placental lactogen and cancer

hPL expression has been reported in testicular and ovarian cancers. Immunoactive hPL has been determined in the serum of some patients with breast cancer but not in those with benign breast disease or healthy men and women. Presence of hPL in breast cancer has a negative influence on prognosis for patients. Contrary to these findings, one study did not find hPL in the serum of patients with breast cancer. The amplification of CSH genes in breast cancers was shown to be related to aneuploidy, lymph node metastases, and overexpression of the Her2/neu oncogene. Moreover, immunohistochemical labelling of hPL in carcinomas further demonstrates the relationship with gene amplification.

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CONCLUSION

hPL may have oncogenic action as a consequence of exerting its effect via the same receptors. Additional studies are required to elucidate the pathophysiological mechanisms.

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Hypothetical 3


