Оригінальні дослідження

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E-cadherin and vimentin mRNA expression in papillary thyroid carcinoma samples as possible biomarkers of metastatic process

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Abstract. Epithelial-mesenchymal transition (EMT) has a decisive influence on the process of metastasis (Mts) formation. A key event of EMT is the reorganization of intercellular junctions. E-cadherin plays an important role in tumorigenesis, cancer progression, Mts formation, affecting cancer cells motility and is considered as tumor suppressor. It has been repeatedly proven that E-cadherin is lost during tumor progression and Mts formation. Vimentin is a mesenchymal marker of invasive cancer cells. It is consistently observed to be overexpressed during cancer Mts and is therefore generally acknowledged as a canonical biomarker of type-3 EMT. **The aim** of the study was to compare the mRNA expression of E-cadherin and vimentin in the tissues of patients with nodular goiter, multinodular goiter (MNG), papillary thyroid carcinoma (PTC) with and without Mts to the lymph nodes. **Material and methods.** Postoperative samples of tumor tissue, Mts, nodular goiter, MNG and conditionally normal tissue were used for the studies. The expression of E-cadherin and vimentin mRNA was determined using quantitative real-time polymerase chain reaction (qPCR). **Results.** The obtained data indicate that the expression of the mesenchymal marker vimentin in PTC tissue and Mts significantly exceeds the level of its expression in goiter tissues. Moreover in MNG the vimentin mRNA expression is significantly reduced compared to normal tissue. In Mts the level of mRNA expression of the marker is higher than in the primary tumor. It was shown a moderate suppression of E-cadherin mRNA expression in goiter tissues and a profound suppression of the epithelial marker expression in the PTC and especially in Mts. The decrease in the E-cadherin expression was also observed in goiter tissue. **Conclusions.** Thus, an increase of vimentin and especially decrease in E-cadherin mRNA expression in PTC may be markers of Mts formation. They can also be used for differentiation between benign and malignant thyroid lesions.

Keywords: E-cadherin, vimentin, cell junctions, papillary thyroid carcinoma, metastasis, goiter, multinodular goiter.

The exploration of the regulatory mechanism related to thyroid cancer Mts is of great significance. In the related studies of tumors, it has been shown that the malignant expression, Mts and invasiveness of tumors are all related to EMT [1-3]. EMT is a reversible biological process in which epithelial cells lose their unique features of apicobasal polarity, epithelial markers, intercellular junctions, reorganization of the cytoskeletal architecture, immobility and differentiation and redirect to mesenchymal phenotype with the ability to migrate and invade [2, 4, 5].

EMT is a key process in the dissemination of tumor cells [4]. In most experimental models, epithelial (E-cadherin) and mesenchymal (N-cadherin and vimentin) markers and morphological changes are considered as indicators confirming the process of EMT. In malignant tumors, EMT is initiated by different signaling pathways through the regulation of transcription factors and microRNAs. Zeb, Snail, Twist and Slug are transcription factors that regulate EMT, the process during which epithelial cells become migratory and invasive. All of these proteins inhibit E-cadherin expression, a cell adhesion molecule crucial for epithelial function [4, 6-8].

As a cytoskeletal protein, vimentin filaments support mechanical integrity of the migratory machinery, generation of directional force, focal adhesion modulation and extracellular attachment. However, during EMT it modulates genes for EMT inducers such as Snail, Slug, Twist and ZEB1/2, as well as the key epigenetic factors. In addition, it suppresses cellular differentiation and upregulates their pluripotent potential by inducing genes associated with self-renewability, thus increasing the stemness of cancer stem cells, facilitating the tumour spread and making them more resistant to treatments [5].

The aim of the study was to compare the cadherin and vimentin mRNA expression in conditionally normal tissue and the tissues of patients with goiters, PTC with and without Mts to the lymph nodes.

Material and methods

Postoperative specimens of tumor tissue, Mts, and conditionally normal (non-tumor, histologically unchanged) tissue obtained from the surgical department of the Institute were used for the study. Samples were stored at -80 °C until use. The research protocol was approved by the Ethics Committee of the Institute. Informed consent for further diagnostic and scientific researches of patient biomaterials was signed by all patients.

The mRNA expression of EMT factors was determined using quantitative PCR.

Reactions were carried out using the following instruments: Thermocycler 2720 (Applied Biosystems, USA) and qTower 3 84 G (Analytik Jena, Germany). Data were analysed using instrument software, Microsoft Excel and GraphPad PRISM 10.

All assays were carried out using RNA extracted from anonymised patient samples. RNA was isolated with TRIzol reagent [9]. Briefly, 1 mL of TRIzol reagent was added per 50-100 mg of tissue to the sample and homogenized using a TissueLyser II (Retsch GmbH). Samples were centrifuged for 5 minutes at 12,000×g at 4-10 °C, with following transffering the clear supernatant to a new tube. Lysates were incubated for 5 min to allow complete dissociation of the nucleoprotein complex. Then 0.2 mL of chloroform was added per 1 mL of lysate, and tubes were mixed by shaking. Samples were incubated for 5 min and centrifuged for 15 min at 12,000×g at 4 °C. The aqueous phase was transferred to a new tube and 0.5 mL of isopropanol was added. Samples were incubated for 20 min at -20 °C and centrifuged for 10 min at 12,000×g at 4 °C. Total RNA samples were washed with 75% ethanol, dried and resuspended in 20-50 µL of RNase-free water.

Primers used are listed in the Table.

Table. Primers used

Proteins	Primers
β-actin	Forward: 5′-GAA-ATCGTG-CGTGACATTAA-3′
	Revers: 5′-CCA-GAC-AGC-ACT-GTG-TTG-G-3′
Vimentin	Forward: 5′- GAG-AAC-TTT-GCC-GTT-GAA-GC-3′
	Revers: 5′- GCT-TCC-TGT-AGG-TGG-CAA-TC-3′
E-Cadherin	Forward: 5′-ACA-CTG-CCA-ACT-GGC-TGG-AGA-TTA-3′
	Revers: 5′-TGA-TTA-GGG-CTG-TGT-ACG-TGC-TGT-3′

All oligonucleotides were resuspended in sterile RNase-free water at 100 µM and stored in aliquots at -20 °C.

Conventional RT reactions were set up on ice using pre-cooled reagents. RNA was reverse transcribed in 20 µL volumes in 0.2 µL thin-walled microfuge tubes using component RevertAid RT Kit (ThermoScientific, #K1691). A mixture of 2 µg RNA and 1 µL 100 µM Oligo (dT)18 primer were adjusted to 12 µL with nuclease free water, mixed gently, centrifuged briefly and incubated at 65 °C for 5 min, then chilled on ice. The following components in the indicated order were added, 4 µL 5x Reaction buffer, 1 µL RiboLock RNAse Inhibitor (20U/µl), 2 µL 10mM dNTP Mix, 1 µL RevertAid RT (200U/µL). Following a 5 s spin at 3K RPM in a microfuge, the tubes were transferred to a conventional Thermocycler (Applied Biosystem 2720, USA) with the heated lid set to 112 °C and incubat-

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ed using the following protocol: 42 °C for 1 h, 70 °C for 5 min. 1 μL cDNA aliquots were used for further qPCR analysis.

Quantitative PCR was done using SYBR Green mix (ThermoScientific, #K0251). Reactions were performed in triplicate in a 384-multiwell plate (Axygen, #PCR-384-LC480-W-NF). Gene expressions were normalized to β-actin, and fold differences were calculated using the comparative CT method: 2-(ΔΔCT), where ΔΔCT refers to (normalized tumor/Mts sample) – (normalized control (norma) sample). The cDNA (1 μL) was added to reaction mix, containing 12.5 μL Maxima SYBR Green qPCR Master Mix (2X), primers – 0.3 μM of each and nuclease-free water to 25 μL. Each of reaction mixtures were pipetted into 3 wells of qPCR plate. Plate was sealed and spun for 5 min at 2000 rpm. qPCR reactions were carried out for 40 cycles with 15 s denaturation at 95 °C and 1 min polymerisation at 60 °C. When SYBR Green was used to detect PCR amplicons instead of probes, an additional melt curve was programmed into the run.

Statistical analysis and data presentation were performed using Origin 7.0 software. The results of the study are presented as M±SE. Student's *t*-test was used to compare data groups. Values of $p \leqslant 0.05$ were considered significant.

Results

Patients with PTCs with and without Mts and with goiters were included in the study. Group 1 included patients with goiter, group 2 – with MNG, group 3 – PTC without Mts, group 4 – PTC with Mts, group 5 – Mts tissue.

Housekeeping proteins are essential proteins involved in basic, fundamental cellular functions, like metabolism and cell structure, and are used as internal controls for analysis. We used β-actin as housekeeping protein.

β-actin melting curves show a single peak, indicating that the primers are specific and only one PCR product is being amplified. The melting temperature of amplification product lies between 83 and 84 °C (**Fig. 1**). Melt curve analysis is a crucial quality control step in qPCR to ensure the amplification of a single, specific product, which is essential for accurate gene expression quantification.

The melting temperature of vimentin lies between 78 and 79 °C. Melting curves also show a single peak (**Fig. 2**).

Vimentin was determined in tissue samples of goiter, MNG, PTCs with and without Mts, and Mts

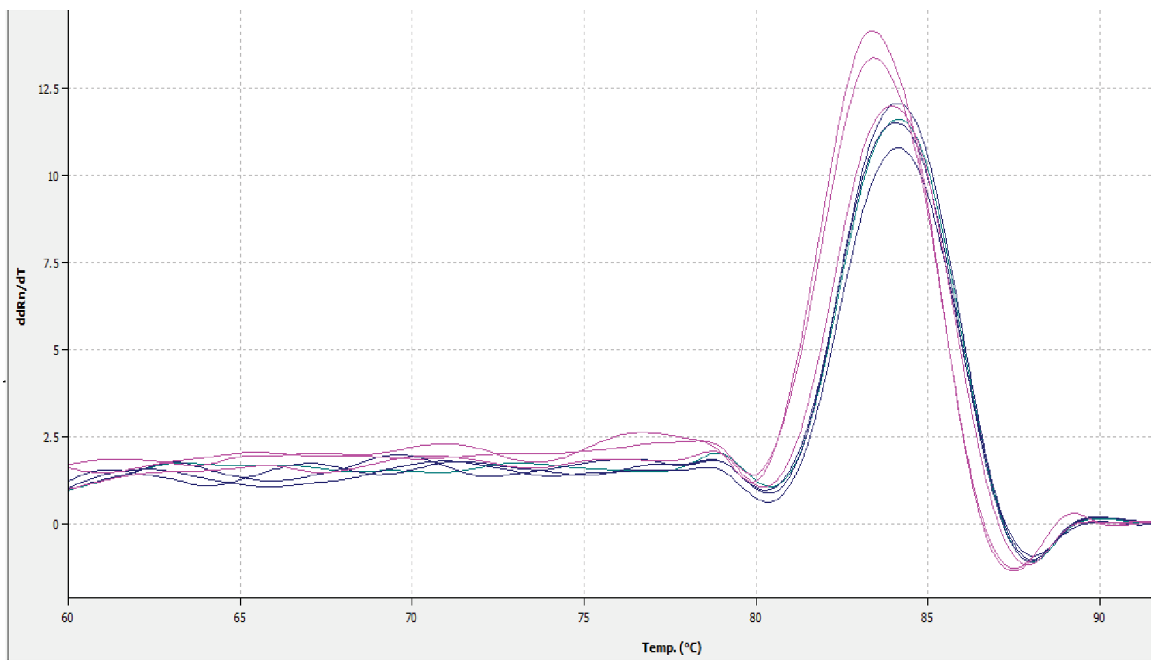

Fig. 1. Melting curves of β-actin.

Note. X-axis shows the melting temperature (60-95 °C). Y axis: ddRn/dT represents the first derivative of the normalized, baseline-corrected fluorescence with respect to temperature. It highlights the temperature at which the maximum rate of fluorescence change occurs, indicating the peak melting point of the PCR product.

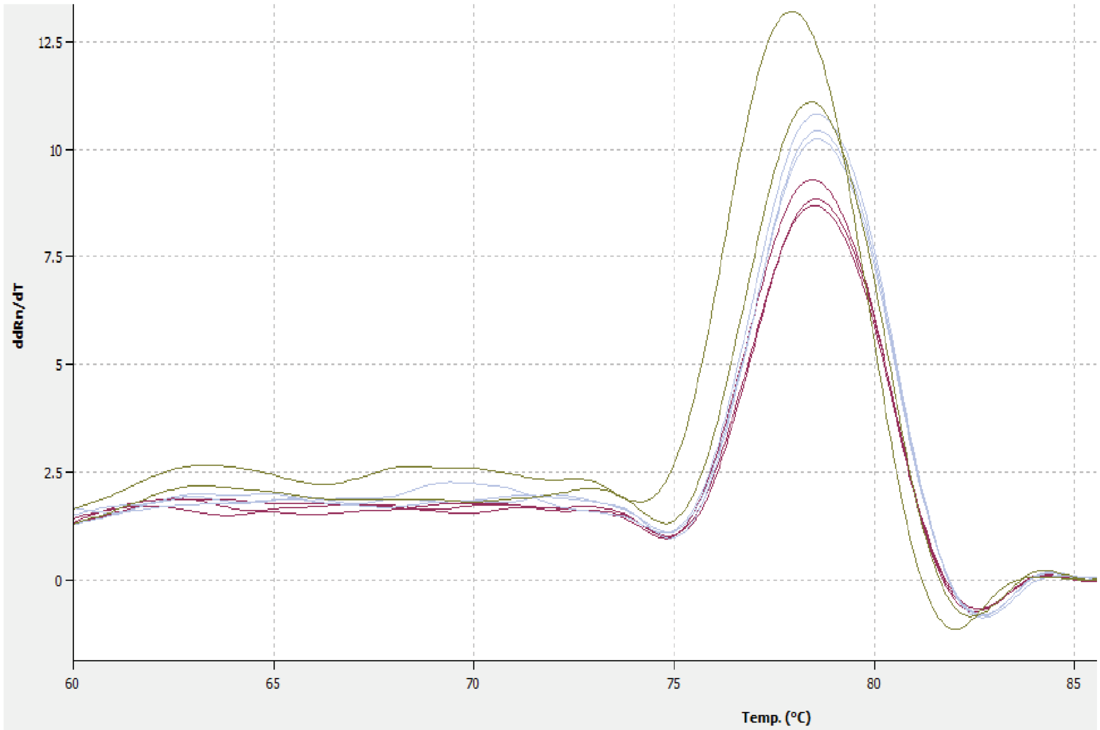

Fig. 2. Vimentin melting curves.

Note. The designations are the same as in fig. 1. Melting temperature is around 78 °C.

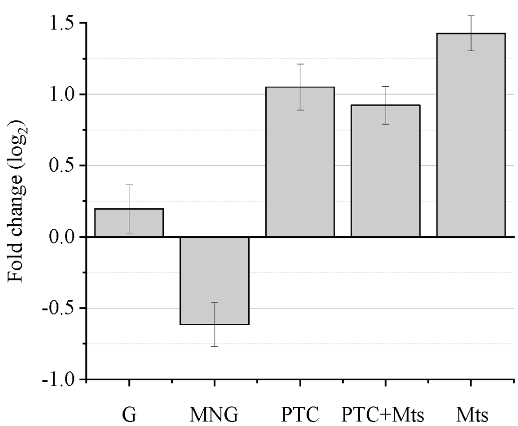

Fig. 3. Determination of vimentin mRNA expression in thyroid samples.

Note. G – goiter.

tissue **(Fig. 3)**.

Figure 3 shows that the expression of the mesenchymal marker vimentin mRNA in PTC tissue and Mts significantly exceeds the level of its expression in goiter tissues ($p \leqslant 0.05$). Moreover, in MNG tissue the vimentin mRNA expression is significantly reduced compared to normal tissue ($p \leqslant 0.05$). Interestingly, in Mts tissue the mRNA expression level of the marker is higher than in the primary tumor ($p \leqslant 0.05$).

Melting curves (n=7) of E-cadherin also show a single peak, indicating that the primers are specific and only one PCR product is being amplified **(Fig. 4)**.

The most important and characteristic event for EMT is a decrease of the E-cadherin expression as an epithelial marker, which indicates the weakening of intercellular connections and the acquisition of an invasive phenotype by the tumor cell. **Fig. 5** show a moderate suppression of E-cadherin expression in goiter tissues and a profound suppression of the expression of the epithelial marker in the PTC. The expression level is lower in PTC with Mts compared to PTC without Mts, and is especially low in the Mts themselves. Attention is drawn also to small but significant decrease in the E-cadherin expression in goiter tissue ($p \leqslant 0.05$). It is possible that there are also processes associated with the weakening of intercellular interaction and a disruption of the tissue structure.

Thus, our data indicate significant differences in the expression of EMT factors between conditionally

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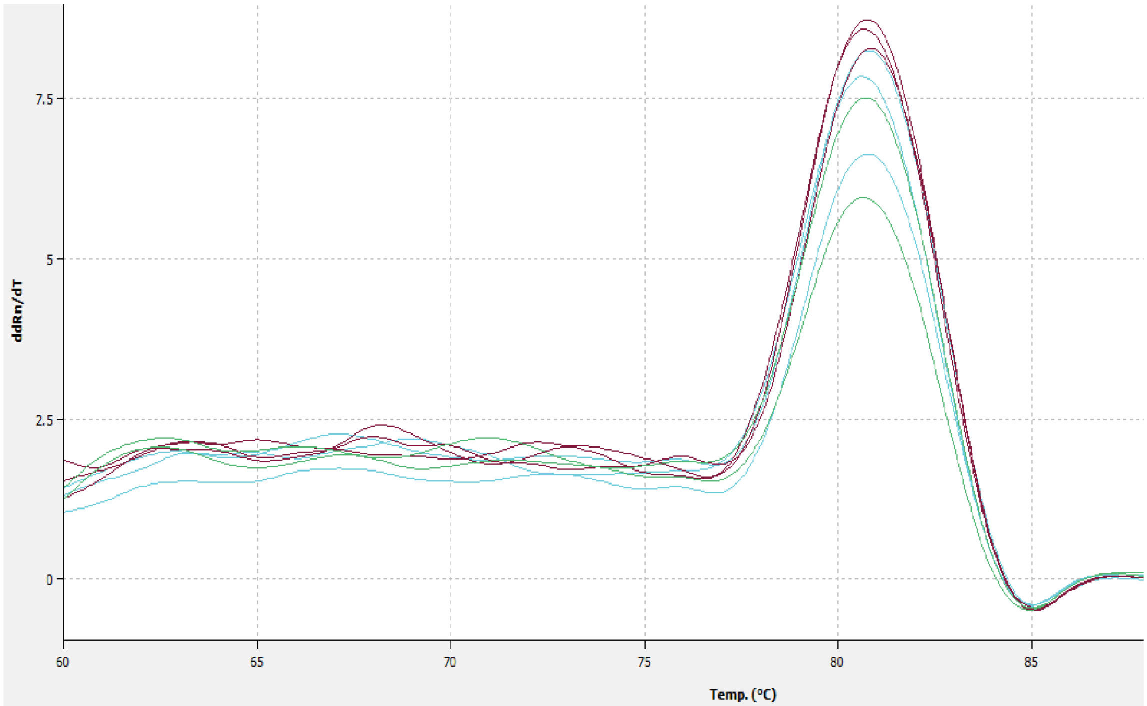

Fig. 4. The melting curves of E-cadherin.

Note. The designations are the same as in fig. 1. Melting temperature is around 81 °C.

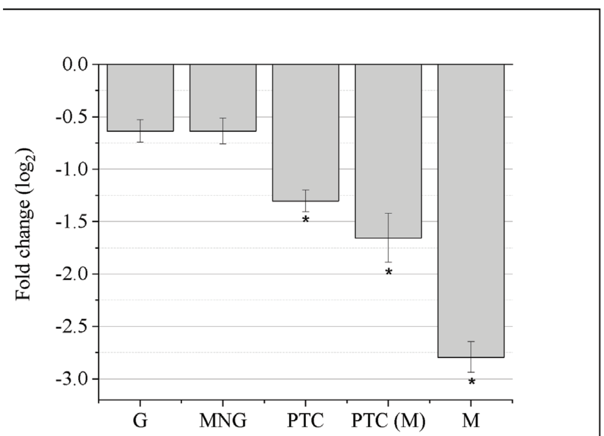

Fig. 5. Determination of E-cadherin mRNA expression in thyroid samples.

*Note. The designations are the same as in Fig. 3. * – significant differences from the previous group, p≤0.05.*

normal thyroid tissue, goiter, tumor tissues and Mts.

Discussion

Cells previously activated by the EMT programme often revert to the epithelial state. This mechanism is called mesenchymal-epithelial transition [10]. In addition to the classical concept of EMT/ mesenchymal-epithelial transition in cancer cells, a recent concept of partial EMT (EM) was introduced, in which cells simultaneously express both epithelial and mesenchymal hybrid markers [4]. This hybrid state makes them metastable, which is a dynamic state enabling cancer cells to induce or revert to EMT. This multishaded EMT concept is known as epithelial–mesenchymal plasticity [11]. The cells in early hybrid EM express both epithelial (E-cadherin) and mesenchymal (vimentin) markers but are less adhesive and rounded in shape. In the late hybrid stage, the mesenchymal markers become more pronounced and the epithelial phenotype is suppressed. Their shape becomes elongated, and adhesion is completely lost. Late hybrid EM stage can lead into a stable mesenchymal state [2].

Vimentin is an important filamentous protein providing structural and functional support to the cell. During initial stages of cancer development, vimentin concentration is very low, however, it increases when cancer starts to invade the surrounding areas [5]. Vimentin is a type III intermediate filament protein alongside other cytoskeletal components, such as microfilaments and microtubules. Its dynamic role in different fundamental cellular processes such as structural support, attachment, migration and signalling is widely accepted [12]. Vimentin is consistently observed to be overexpressed during cancer Mts and is therefore generally acknowledged as a canonical biomarker of type-3 EMT [13]. Several studies have highlighted its central role in the regulation of this complex process [5]. Vimentin filaments protect the cancer cells from mechanical stresses during the migration by providing a viscoelastic framework and support the positioning and integrity of organelles, especially the nucleus, during EMT and cancer progression [14]. In addition, it was reported that vimentin protects the cancer cells from the internal stress of misfolded proteins by directly binding to stress granules and aggresomes, supporting their subsequent destruction [5, 15].

We demonstrate that the expression of the mesenchymal marker vimentin mRNA in PTC tissue and especially Mts significantly exceeds the level of its expression in goiters and conditionally normal tissues.

E-cadherin belongs to calcium-dependent cell adhesion proteins, which are involved in homophilic interactions, forming intercellular contacts. E-cadherin is involved in the mechanisms of intercellular adhesion regulation, cell motility and epithelial cell proliferation. It plays a suppressive role in relation to cell invasiveness. E-cadherin is a protein that promotes cell-cell adhesion in epithelial cells, and its loss is often associated with tumor progression, particularly Mts, through an EMT process [16, 17].

In thyroid carcinoma, the E-cadherin expression is affected by regulating signal pathways, upstream genes and immune microenvironment, thus affecting the occurrence of EMT. These results suggest that it is a new idea to regulate E-cadherin to affect the occurrence of thyroid EMT and improve the malignant expression of tumors. Overall, the malignant phenotype of thyroid cancer is negatively correlated with E-cadherin, and its complex regulatory mechanisms and widely involved cytokines may provide new ideas for the early diagnosis, prognosis and treatment of thyroid cancer [1, 18].

An important task facing endocrine surgery is the identification of reliable markers of Mts in treated PTC. Markers that can be identified at the stage of preoperative investigation are of particular value. Such markers may include factors that take part in EMT, such as Zeb, Snail, Slug, Twist, vimentin and E-cadherin [6, 8, 19-22].

Conclusions

1. There is a moderate suppression of E-cadherin expression in the tissues of nodular and multinodular goiter, and a profound decrease of the expression of this epithelial marker in the thyroid papillary carcinoma tissues, especially in papillary thyroid carcinoma with metastases and in the metastases themselves, which indicates the weakening of intercellular connections and the acquisition of an invasive phenotype by the tumor cells.

2. The expression of the mesenchymal marker vimentin mRNA in the papillary thyroid carcinoma tissues and in metastases significantly exceeds the level of its expression in the normal tissues and the goiter. In multinodular goiter, the vimentin mRNA expression is significantly reduced compared to normal. In metastases, mRNA expression level of the marker is higher than in the primary tumor.

3. The obtained data confirm the initiation of the epithelial-mesenchymal transition process in thyroid papillary carcinoma, especially in papillary thyroid carcinoma with metastases.

4. The extremely small amount of biomaterial (fine-needle biopsy) required to detect vimentin and E-cadherin using real-time polymerase chain reaction makes this approach promising for differentiating benign changes from papillary carcinoma in the clinics.

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Abbreviations

EMT – epithelial-mesenchymal transition

MNG – multinodular goiter

Mts – metastasis

PTC – papillary thyroid carcinoma

qPCR – quantitative real-time polymerase chain reaction

ЕКСПРЕСІЯ мРНК Е-КАДГЕРИНУ ТА ВІМЕНТИНУ В ЗРАЗКАХ ПАПІЛЯРНОЇ КАРЦИНОМИ ЩИТОПОДІБНОЇ ЗАЛОЗИ ЯК МОЖЛИВИХ БІОМАРКЕРІВ МЕТАСТАТИЧНОГО ПРОЦЕСУ

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Резюме. Епітеліально-мезенхімальний перехід (ЕМТ) має вирішальний вплив на процес утворення метастазів (Мтс). Ключовою подією ЕМТ є реорганізація міжклітинних з’єднань. Е-кадгерин відіграє важливу роль у онкогенезі, прогресуванні раку, утворенні Мтс, впливаючи на рухливість ракових клітин і який розглядається як супресор пухлини. Неодноразово було доведено, що Е-кадгерин втрачається під час прогресування пухлини та утворення Мтс. Віментин є мезенхімальним маркером інвазивних ракових клітин. Його надмірна експресія постійно спостерігається під час метастазування раку, тому він є загальновизнаним канонічним біомаркером ЕМТ 3 типу. **Метою** дослідження було порівняння експресії мРНК Е-кадгерину та віментину в тканинах пацієнтів із зобом, багатовузловим зобом (БВЗ), папілярною карциномою щитоподібної залози (ПКЩЗ) без та з метастазами в лімфатичні вузли. **Матеріал і методи**. Для досліджень використовували післяопераційні зразки пухлинної тканини, Мтс, зоба та умовно нормальної тканини. Експресію мРНК Е-кадгерину та віментину визначали за допомогою полімеразної ланцюгової реакції в реальному часі (кПЛР). **Результати**. Отримані дані свідчать про те, що експресія мезенхімального маркера віментину в тканині папілярного раку щитоподібної залози (ПКЩЗ) та метастазах значно перевищує рівень його експресії в тканинах зоба. Більше того, при БВЗ експресія мРНК віментину значно знижена порівняно з нормальною тканиною. У метастазах рівень експресії мРНК маркера вищий, ніж у первинній пухлині. Показано помірне пригнічення експресії мРНК Е-кадгерину в тканинах зоба та значне пригнічення експресії епітеліального маркера в ПКЩЗ та, особливо, в Мтс. Ми також спостерігали зниження експресії Е-кадгерину в тканині зоба. **Висновки**. Таким чином, збільшення віментину та, особливо, зниження експресії мРНК Е-кадгерину в ПКЩЗ можуть бути маркерами утворення Мтс. Їх також можна використовувати для диференціації доброякісних та злоякісних уражень щитоподібної залози.

Ключові слова: Е-кадгерин, віментин, клітинні з’єднання, карциноми щитоподібної залози, метастази, зоб, багатовузловий зоб.

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