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Level of alpha-smooth muscle actin in thyroid tumors, metastasis, blood cells, and plasma

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Abstract. Alpha-smooth muscle actin (α-SMA) is an isoform of the actin protein essential for cell motility, maintenance of cytoskeletal structure, and muscle contraction. Numerous studies indicate that α-SMA is involved in carcinogenesis, epithelial-mesenchymal transition (EMT), metastasis (Mts) formation, and tumor drug resistance. Its expression serves as a marker of cancer-associated fibroblasts (CAFs), which promote tumor growth, invasion, and Mts through interactions with cancer cells. The **aim** of this study was to compare α-SMA levels in tissue samples of follicular adenoma (FA), goiter, papillary thyroid carcinoma (PTC), metastases (Mts), conditionally normal thyroid tissue, as well as in blood plasma and peripheral blood mononuclear cells (PBMC). **Material and methods.** Postoperative samples of tissue, blood plasma, and cells were obtained from the surgical department of the clinic. The amount of α-SMA was determined using enzyme immunoassay kits. **Results.** α-SMA levels in FA and goiter tissues did not differ from those in conditionally normal tissue. A significant difference was observed between normal and PTC tissues with and without metastases. α-SMA levels in PTC tissue without Mts were nearly four times lower than in PTC tissue with Mts. α-SMA expression in metastatic tissue was higher than in normal tissue. Additionally, significant differences in α-SMA levels were detected between normal tissue samples from patients with and without Mts. In blood plasma and PBMC of PTC patients, α-SMA concentration significantly exceeded control levels. **Conclusions.** α-SMA levels in benign thyroid neoplasms did not differ from those in conditionally normal tissue. In the blood plasma and PBMC of PTC patients, α-SMA concentrations were elevated compared to controls; however, no differences were observed between PTC cases with and without Mts. Our findings reveal significant differences in α-SMA concentrations between PTC tumor tissues with and without Mts, which may be useful for predicting metastatic potential.

Keywords: papillary thyroid carcinoma, metastasis, benign neoplasms, alpha-smooth muscle actin.

α-SMA, encoded by the *ACTA2* gene, is an isoform of actin protein. It is crucial for cell movement, maintaining the cell's internal structure (cytoskeleton), and muscle cell contraction. It is normally found in smooth muscle cells of blood vessel walls, the intestines, and other organs. In non-muscle cells, particularly fibroblasts, its expression is a marker for their activation into myofibroblasts. These activated cells are involved in normal wound healing but also play a central role in pathological conditions like fibrosis and cancer progression. α-SMA is an marker of epithelial to mesenchymal transition (EMT). EMT is a process by which tumor cells develop to be more motile and able to me-

tastasize. Progression of tumor cells is always followed by cell composition and extracellular matrix (ECM) component alteration. Increased α-SMA expression and collagen alteration may predict the progressivity of neoplasms. α-SMA is an actin isoform that plays an important role in fibrogenesis. α-SMA can be found in smooth muscle cells, myofibroblasts, and blood vessels [1, 2]. α-SMA correlates with activation of fibroblast to myofibroblast CAFs [3, 4]. Myofibroblasts differ from fibroblasts because of its contractile ability. The phenotype of myofibroblasts in expressing α-SMA and producing extracellular matrix compound is regulated by transforming growth factor-beta [5]. Contractile properties of myofibroblasts are associated with α-SMA expression and are involved in inflammation, wound healing, fibrosis, and carcinogenesis [6]. Carcinoma cells that transform to mesenchymal cells also express α-SMA [7]. α-SMA expression was noted to be higher in serous borderline ovarian tumors that invade the omentum compared to non-invasive. α-SMA, together with vimentin, E-cadherin, and fibronectin are the markers for the EMT process. The EMT is considered one of the steps involved in normal cells to become cancerous [2].

CAFs are key players in cancer development and therapy, and they exhibit multifaceted roles in the tumor microenvironment (TME). From their diverse cellular origins, CAFs undergo phenotypic and functional transformation upon interacting with tumor cells and their presence can adversely influence treatment outcomes and the severity of the cancer. Emerging evidence has highlighted the heterogeneity and plasticity of CAFs, with subtypes identifiable through distinct gene expression profiles and functional properties. CAFs influence cancer development through multiple mechanisms, including regulation of ECM remodeling, direct promotion of tumor growth through metabolic support, promoting EMT to enhance cancer invasiveness and growth, they also aid in angiogenesis, further supporting a metastatic environment as well as stimulating cancer stem cell properties within the tumor. Moreover, CAFs can induce an immunosuppressive TME and contribute to drug resistance and have potential as therapeutic targets [3, 4].

The **aim** of the study was to compare the levels of α-SMA in tissue samples of FA, goiter, PTC, Mts, and conditionally normal tissue, in blood plasma and PBMC.

Material and methods

The research protocol was approved by the Bioethics Commission of SI «V.P. Komisarenko Institute of Endocrinology and Metabolism of the National Academy of Medical Sciences of Ukraine», protocol no. 26-KE dated April 10, 2019. All patients signed informed consent for the use of biomaterials for further diagnostic and scientific research.

Postoperative samples of FA, 2 types of goiters, PTC, Mts, and conditionally normal (non-tumor or morphologically unchanged tissue) tissue, obtained from the surgical department of the Institute's clinic, were used for research. Blood plasma of patients were also analysed. Blood was obtained by standard venipuncture and stored in EDTA tubes. Plasma was separated by centrifugation within 10 minutes after blood sampling. The PBMC were collected using Histopaque 1077 (Sigma, USA) [8]. The concentration of protein in cell lysates was determined according to Bradford [9]. Blood controls were taken from healthy individuals without thyroid and comorbid diseases.

Samples were stored at -80 °C until use. The amount of α-SMA was determined using enzyme immunoassay kits EH1506 (FineTest®, China) (**Fig. 1**). Measurements were performed at an optical wavelength of 450/630 nm on an immunoenzymatic plate analyzer Stat Fax 3200 (Awareness Technology, USA).

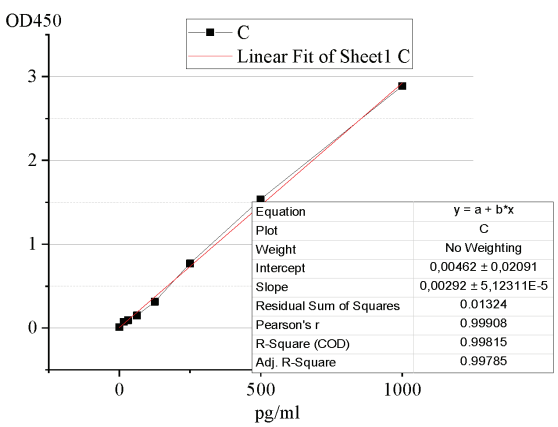

Fig. 1. Calibration curve for calculating the concentration of α-SMA using EH1506 enzyme immunoassay kits.

Patients with PTC, PTC + Mts, FA and goiters participated in the study. Group 1 included 8 sam-

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ples with FA, group 2 – 8 samples with nodular goiter, group 3 – 8 samples with multinodular goiter, groups 4 – 16 samples with PTC without Mts, group 5 – 24 samples with PTC and Mts. The concentration of α-SMA in plasma of 9 patients with PTC without Mts and 10 patients with PTC and Mts was also determined. Blood from 5 individuals without thyroid disease and other chronic diseases, representative for age, was used as a control.

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 and discussion

The level of α-SMA did not differ significantly between conditionally normal tissue and benign neoplasms: FA, nodular and multinodular goiter **(Table)**. The α-SMA concentrations in benign neoplasms are ~ 0.2 ng/mkg of total protein.

Table. α-SMA quantity in the thyroid tissue of patients with FA and goiters

Indicators	ng/mkg	SE	%	SE
CN	0.58262	0.30826	100.00	52.91
FA	0.23936	0.12834	41.08	22.03
CN	0.10957	0.05097	100.00	46.52
NG	0.13156	0.04281	120.07	39.07
CN	0.23844	0.08686	100.00	36.43
MNG	0.17835	0.04248	74.80	17.81

Note. CN – conditionally normal (histologically unchanged) tissue (100% in each group); NG – nodular goiter, MNG – multinodular goiter.

Unlike benign neoplasms, α-SMA amount were significantly higher in PTC tumor tissue compared to normal tissue **(Fig. 2a,b)**. Actin levels in PTC tumor tissue with metastases were almost four times higher than in corresponding normal tissue and PTC tumor tissue without metastases. The difference in α-SMA levels between normal tissue of PTC without metastases and normal tissue of PTC with metastases is noteworthy. This latter finding indicates the tumor's influence on adjacent tissues and is confirmed by other authors [10-12].

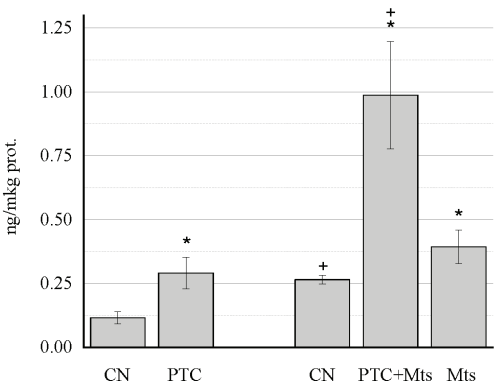

Fig. 2a. α-SMA quantity in the thyroid tissue of patients with PTC.

*Note. CN – conditionally normal tissue. * – significantly different from conditionally normal tissue, p≤0.05; + – significantly different from normal and tumor PTC tissue without Mts, p≤0.05.*

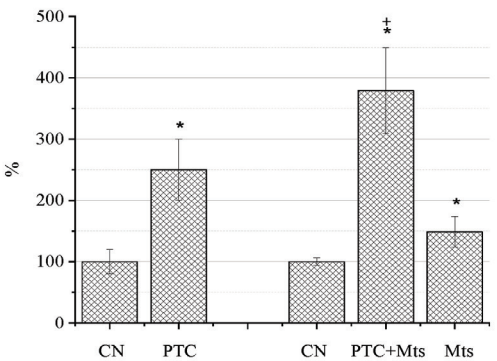

Fig. 2b. Percentage of α-SMA quantity in the thyroid tissue of patients with PTC compared with corresponding conditionally normal tissue.

*Note. CN – conditionally normal tissue. * – significantly different from conditionally normal tissue, p≤0.05; + – significantly different from normal and tumor PTC tissue without Mts, p≤0.05.*

α-SMA concentration in the PBMC of patients with PTC both with, and without Mts was significantly (more than 10 times) higher than in control cells **(Fig. 3)**, which can serve as a diagnostic marker. There was not a difference between two types of tumor tissue.

α-SMA concentration in the blood plasma of patients with PTC both with, and without Mts, also significantly higher than in control plasma **(Fig. 4)**, although the amplitude of changes is significantly lower than in PBMC. There was also not a difference between two types of tumor tissue.

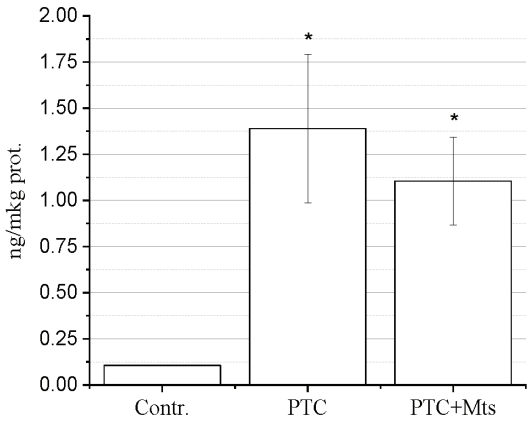

Fig 3. α-SMA concentration in the PBMC of patients with PTC.

*Note. Contr. – cells of healthy volunteers. * – significantly different from control, p≤0.05.*

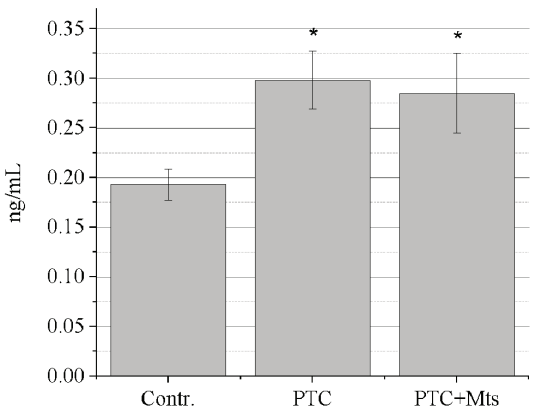

Fig 4. α-SMA concentration in the blood plasma of patients with PTC.

*Note. Control – plasma of healthy people. * – significantly different from control samples, p≤0.05.*

The malignant transformation of tumors is not solely governed by tumor cells, it is also modulated by stromal cells within the TME and by a signaling network. The TME is the surrounding environment in which cancer cells reside and proliferate. It is a highly dynamic and complex network composed of the ECM, diverse signaling molecules, and non-tumor cells, such as immune cells, fibroblasts, and adipocytes. Among these, CAFs – activated fibroblasts, embedded in the TME, which play a critical role in remodeling the ECM during cancer development, have recently attracted attention in tumor research. They not only mediate tumor progression by remodeling the ECM, thereby influencing tumor cell invasion, Mts, angiogenesis, and therapeutic resistance, but also interact with other TME components (such as tumor and immune cells) through multiple complex molecular mechanisms, further exacerbating malignant progression [13, 14].

α-SMA expression in cancer is associated with Mts because it indicates the presence of activated CAFs, which promote tumor invasion and spread. CAFs assist in processes like EMT, which helps cancer cells detach from the primary tumor, and they also contribute to angiogenesis, further supporting a metastatic environment. α-SMA is a marker for CAFs, which are stromal cells that create a more permissive environment for tumor growth and spread. In some cancers, high α-SMA expression is correlated with increased tumor fibrosis, which can be part of the supportive microenvironment for metastatic cells. α-SMA-positive CAFs can promote the formation of new blood vessels, which is essential for tumors to grow and metastasize to new locations. The α-SMA expression is often associated with a higher number of lymph node metastases and a poorer prognosis for patients with certain types of cancer [1, 3, 4].

α-SMA is expressed in myofibroblasts within the TME of thyroid cancer, rather than the cancer cells themselves. Its presence is a marker of CAFs, which are linked to increased tumor growth, invasion, and Mts through their interaction with cancer cells. Elevated levels of α-SMA can indicate more aggressive tumors, particularly when found in the stroma [15-17].

It was shown that expression of α-SMA and matrix metalloproteinases-9 (MMP-9) was higher in PTC tissue compared to normal thyroid tissue. The expression of α-SMA and MMP-9 was slightly, but not significantly, higher in the metastasized tumors and their respective lymph nodes [18]. Our data show the significant difference in α-SMA levels between conditionally normal tissue adjacent to PTC without metastases and PTC with metastases (see Fig. 2a,b).

The results showed that the expression levels of Notch1, TGF-β1, and p-Smad3 in PTC cells and α-SMA in the stroma of PTC were all significantly higher than in nodular goiter and normal thyroid tissues. Further analysis showed that in PTC, higher expression levels of Notch1 and TGF-β1 were closely related with lymph node Mts, whereas the expression α-SMA and p-Smad3, increased significantly with advanced tumor stages. A significant correlation was found between higher TGF-β1

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expression in PTC cells and increased α-SMA levels in the fibroblasts surrounding the cancer cells. TGF-β1 was identified as an important factor from PTC cells that act in a paracrine manner to influence the activation of stromal fibroblasts. These data suggest that the activation of Notch and TGF-β/Smad3 pathways in cancer cells influence tumor growth. Moreover, cancer cell-derived-TGF-β ligands also affect stromal cells in a paracrine fashion and enhance tumor growth [19]. There are also significant differences in the TGF-β1 level between tumor tissues of PTC with and without Mts [20, 21].

Immunohistochemical studies demonstrated that the three fibroblast activation markers (α-SMA, FAPalpha, Tn-C) are consistently expressed in the peritumoral and intratumoral stromal compartment of medullary thyroid carcinoma. Moreover, the extent of desmoplasia as well as the expression of fibroblast markers correlated with the presence of lymph node (LN) metastases. Authors found in a series of 48 thyroid cancers a significant correlation between FAPalpha RNA expression and incidence of LN metastases also in papillary cancers. These findings suggest that the link between specific molecular markers of tumor stromal reaction and locoregional Mts extends from medullary to other thyroid cancer types [22].

In recent years, a large number of markers of tumor aggressiveness, Mts, and radioiodine resistance have been identified in differentiated thyroid carcinomas. These include proliferating cell nuclear antigen [23, 24], the expression of a rare isoform of ribosomal kinase S6K – p60S6K [12], overexpression of MMP [25], transcription factor ZEB1 [26], decrease in level of tight junction protein ZO-1 [27], overexpression of hypoxia-inducible factor-1α [28], TGF-β1 [20, 21] and many others [29]. It is possible that α-SMA quantity in the thyroid tissue of patients with PTC can be another predicting factor of Mts formation.

Conclusions

1. Our data indicate significant differences in the concentration of alpha-smooth muscle actin between tumor tissues of papillary thyroid carcinoma with metastasis, carcinoma tissue without metastasis, conditionally normal tissue and benign neoplasms.

2. The level of actin in the benign neoplasms did not differ from its levels in conditionally normal tissue.

3. In blood plasma and peripheral blood mononuclear cells of patients with papillary thyroid carcinoma the concentration of the alpha-smooth muscle actin exceeded its level in control plasma, but there is no difference between papillary thyroid carcinoma with and without metastasis.

4. Our data indicate significant differences in the concentration of alpha-smooth muscle actin between tumor tissues of papillary thyroid carcinoma with metastasis and tissues of papillary thyroid carcinoma without metastasis. The latter facts may be useful for predicting the formation of metastasis.

Limitations

This study has several limitations. It was conducted at a single centre; therefore, the findings require validation in large-scale, multicenter studies. In addition, the relatively small number of patients in the overall cohort and in individual subgroups may have influenced the measured levels of the studied parameters. There are also certain difficulties with collecting postoperative experimental material.

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Abbreviations

CAFs – cancer-associated fibroblasts
ECM – extracellular matrix
EMT – epithelial-mesenchymal transition
FA – follicular adenoma
Mts – metastasis
PBMC – peripheral blood mononuclear cells
PTC – papillary thyroid carcinoma
α-SMA – alpha-smooth muscle actin
TME – tumor microenvironment

РІВЕНЬ АЛЬФА-ГЛАДКОМ'ЯЗОВОГО АКТИНУ В ПУХЛИНАХ ЩИТОПОДІБНОЇ ЗАЛОЗИ, МЕТАСТАЗАХ, КЛІТИНАХ КРОВІ ТА ПЛАЗМІ

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Резюме. Альфа-гладком'язовий актин (α-SMA) – це ізоформа актинового білка. Він має вирішальне значення для руху клітин, підтримки їхньої внутрішньої структури та скорочення м'язових клітин. Численні дані вказують на те, що α-SMA бере участь у канцерогенезі, епітеліально-мезенхімальному переході (ЕМП), утворенні метастазів (Мтс) та стійкості пухлини до терапії. Його присутність є маркером фібробластів, асоційованих із раком, які пов'язані з ростом, інвазією та метастазуванням пухлини через їхню взаємодію з раковими клітинами. **Метою** дослідження було порівняння рівнів α-SMA в зразках тканин фолікулярної аденоми (ФА), зобу, папілярної карциноми щитоподібної залози (ПКЩЗ), Мтс, умовно нормальної тканини, плазми та периферичних мононуклеарів крові (РВМС). **Матеріал і методи**. Післяопераційні зразки тканини, плазма та клітини крові були отримані з хірургічного відділення клініки. Кількість α-SMA визначали за допомогою наборів для імуноферментного аналізу. **Результати**. Рівні α-SMA в тканині ФА та зобу не відрізнявся від його кількості в умовно нормальній тканині. Спостерігалася значна різниця між нормальною тканиною та тканиною ПКЩЗ із Мтс та без них. Кількість

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α-SMA в тканині ПКЩЗ без Мтс була майже в 4 рази нижчою, ніж у тканині ПКЩЗ із Мтс. Рівень α-SMA в метастазах був вищим, ніж у нормальній тканині. Також спостерігалася значна різниця між нормальною тканиною пацієнтів із Мтс та без них. У плазмі та клітинах крові пацієнтів із ПКЩЗ концентрація α-SMA значно перевищувала контрольний рівень. **Висновки.** Кількість α-SMA в доброякісних новоутвореннях не відрізнялася від рівня актину в умовно нормальній тканині. У плазмі крові та РВМС пацієнтів із ПКЩЗ концентрація α-SMA перевищувала рівень у контрольній плазмі, але різниці між ПКЩЗ із Мтс та без Мтс немає. Наші дані вказують на значні відмінності в концентрації α-SMA між пухлинними тканинами ПКЩЗ із Мтс та тканинами ПКЩЗ без Мтс. Останні факти можуть бути корисними для прогнозування утворення Мтс.

Ключові слова: папілярна карцинома щитоподібної залози, метастазування, доброякісні новоутворення, альфа-гладком'язовий актин.

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