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ORIGINAL INVESTIGATION / ÖZGÜN ARAŞTIRMA

Role of Platelets and Indices in the Clinicopathological Features of Papillary Thyroid Carcinoma

Papiller Tiroid Karsinomunun Klinikopatolojik Özelliklerinde Trombositler ve İndekslerin Rolü

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ABSTRACT

Objective: In this study, the potential relationship between platelet (PLT) count, PLT indices (mean platelet volume [MPV] and plateletcrit [PCT]), and clinicopathological features in patients with papillary thyroid cancer was evaluated.

Method: A total of 196 patients diagnosed with papillary thyroid cancer after total thyroidectomy were included in the study. The preoperative PLT, MPV, and PCT values of the patients were compared with the clinicopathological features of papillary thyroid cancer obtained from pathology reports.

Results: The mean age of the study population was 46±12 years, and the male/female ratio was 29/167. The preoperative PLT count, mean thyroid-stimulating hormone, MPV, and PCT were 288±61 cells/L, 1.9±1.4 mIU/L, 10±0.93, and 0.30%±0.06%, respectively. The preoperative PLT count, MPV, and PCT were significantly higher in patients with tumor size ≥ 1 cm (p=0.005, p=0.001, p<0.001). PLTs in the male group were found to be significantly lower compared with the female group (male: 263±62 cells/L vs female: 292±60 cells/L, p=0.024). In the presence of capsule invasion, PLT, MPV, and PCT values were significantly higher (p=0.046, p=0.021, p=0.08), whereas only PCT values were significantly higher in patients with lymphovascular invasion (p=0.045). In the presence of thyroiditis in nontumor tissue, PLT count and PCT were significantly higher (p=0.043 and p=0.001).

Conclusion: The measurement of PLT count and indices is cost-effective, safe, and always indicated before surgical intervention. Therefore, preoperative PLTs, MPV, and PCT could be used as a predictive marker to foresee adverse clinicopathological features in papillary thyroid carcinoma.

Keywords: Mean platelet volume, platelet, platelet indices, plateletcrit, papillary thyroid cancer

ÖZ

Amaç: Bu çalışmada papiller tiroid kanserli hastalarda trombosit sayısı, trombosit indeksleri (Ortalama trombosit hacmi, trombositkrit) ve klinikopatolojik özellikler arasındaki potansiyel ilişki değerlendirildi.

Yöntem: Total tiroidektomi sonrası papiller tiroid kanseri tanısı alan 196 hasta çalışmaya dahil edildi. Hastaların preoperatif PLT, MPV ve PCT değerleri ile patoloji raporlarındanelde edilen papiller tiroid kanserin klinikopatolojik özellikleri karşılaştırıldı.

Bulgular: Çalışma popülasyonunun ortalama yaşı 46±12 yıl ve erkek/kadın oranı 29/167 idi. Preopratif trombosit sayıları ve ortalama TSH, ortalama trombosit hacmi ve trombositkrit değerleri sırasıyla 288±61 hücre/L, 1,9±1,4 (mIU/L), 10±0,93 ve 0,30±0,06 (%) idi. Tümör boyutu ≥1 cm olan hastalarda ameliyat öncesi trombosit sayısı, ortalama trombosit hacmi ve trombositkrit anlamlı olarak daha yüksekti (p=0,005, p=0,001, p<0,001). Erkek cinsiyette trombositlerin kadın grubuna (kadın: 292±60-erkek: 263±62) (hücre/L) göre anlamlı olarak daha düşük olduğu bulundu (p=0,024). Kapsül invazyonu varlığında trombosit, ortalama trombosit hacmi ve trombositkrit değerleri anlamlı olarak yüksek (p=0,046, p=0,021, p=0,08), lenfovasküler invazyonu olan hastalarda ise yalnızca

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trombositkrit değerleri anlamlı olarak yüksekti (p=0,045). Tümör dışı dokuda tiroidit varlığında trombosit sayısı ve trombositkrit anlamlı olarak yüksekti (p=0,043) (p=0,001).

Sonuç: Trombosit sayısının ve indekslerinin ölçümü uygun maliyetli, güvenlidir ve her zaman cerrahi müdahaleden önce endikedir. Bu nedenle, preoperatif trombositler, ortalama trombosit hacmi ve trombositkrit, papiller tiroid karsinomunda istenmeyen klinikopatolojik özelliği öngörmek için öngörücü bir belirteç olarak kullanılabilir.

Anahtar kelimeler: Ortalama trombosit hacmi, papiller tiroid kanseri, trombosit, trombositkrit, trombosit indeksleri

INTRODUCTION

Cancer and inflammation have a complex relationship based on various physiological processes such as different inflammatory cells, agents, and signal pathways in cancer tissue. Also, tumor microenvironment and inflammatory response play an important role in tumor cell proliferation, survival, angiogenesis, invasion, and metastases.[1] Recent studies have revealed new evidence that inflammation is associated with cancer pathophysiology.[2] Changes in clinical outcomes in cancer patients are associated with the oncological properties of the tumor and the host response to systemic inflammation.[3] In various cancers, hematological components of systemic inflammatory response have been shown to have prognostic value.[4]

Papillary thyroid carcinoma constitutes 1% of all malignancies and 70%–80% of all thyroid cancers and usually has a good prognosis.[5,6] The relationship between inflammation and malignancy in papillary thyroid carcinoma is well established.[7,8] Various studies have shown that patients with thyroiditis have a higher incidence of differentiated thyroid carcinoma,[8] which may indicate a link between thyroid cancer and inflammation. Angiogenesis, metastatic, and proteolytic activities in which PLTs play a role in the background of inflammation and their metabolic roles in cancer pathogenesis are indisputable.[9] In the literature, higher platelet (PLT) levels were associated with a higher risk of recurrence and the risk of metastases in various advanced cancers.[10] In addition to the PLT count, new hematological analyzers have reported new parameters such as PLT dispersion width, mean platelet volume (MPV), and plateletcrit (PCT), known as PLT indices. MPV is a marker associated with functional changes in PLTs that are routinely used and easily detectable from hemograms.[11] MPV shows the average PLT size and reflects PLT production speed and stimulation.[12] PCT demonstrates PLT mass in volume units using MPV and PLT values.[13]

Tumor/host interaction has a significant effect on the results of patients. However, this effect is generally ignored in existing prognostic systems. The evaluation of various immune markers in preoperative papillary thyroid carcinoma cases and the use of postoperative follow-up are still limited and controversial.

The aim of this study was to investigate the potential relationship between PLT count in papillary thyroid carcinoma patients, PLT indices (MPV and PCT), and clinicopathological properties.

METHOD

A total of 196 patients between January 2015 and May 2020 were included in the study. This study has the approval of the local ethics committee (Istanbul Kanuni Sultan Suleyman Training and Research Hospital, Istanbul, Turkey. Date: June 24, 2020, Decision number: 103) and was conducted in accordance with the Declaration of Helsinki. Patients over 18 years of age, who had undergone total thyroidectomy operation, and who were pathologically diagnosed with papillary thyroid cancer were included. Patients who had thyroid carcinoma other than the papillary tumor, who had been admitted with recurrence, known hematological or chronic inflammatory diseases, and who had been using chronic drugs (e.g., steroids) were excluded, to improve the reliability of our results.

The demographic characteristics of the patients, such as age and gender, were recorded. A complete blood count was taken the day before the surgery. Thyroid-stimulating hormone values were measured and recorded one week before the operation. PLT, MPV, and PCT values were determined by the values obtained from the complete blood count.

Histopathological examination of all patients evaluated tumor size, thyroid capsule invasion, lymphovascular invasion, extrathyroidal invasion, bilateral involvement, multifocal tumor presence, and lymph node metastasis positivity. The tumor size was reported as the largest lesion size measured during the histopathological examination. Histological subtypes were evaluated as classic, follicular, and oncocytic variants. Aggressive histology presence, such as tall cell, insular, columnar cell, Hürthle cell, and hobnail variant, has been recorded. The presence of thyroiditis was examined in nontumor tissue.

Median values were calculated for PLT, MPV, and PCT variables. Later, the patients were divided into two subgroups as high and low according to median values. The clinicopathological features were compared.

Statistical Analysis

Continuous variables were expressed as average ± standard deviation, categorical variable frequency, or percentage. The Shapiro-Wilk test was used to determine whether the sample data were distributed normally. The t-test was used when the variance for two groups was equal, and the Mann–Whitney test was used when the variance was not equal. The Chi-squared test was used to compare categorical variables. All analyses were conducted with the Social Sciences Statistical Package for Windows 22.0 (SPSS, Inc., Chicago, IL, USA), and the results with a $p<0.05$ level were significantly accepted.

RESULTS

A total of 214 patients were diagnosed with papillary thyroid carcinoma after total thyroidectomy during the working period. Patients with known hematological disorders, past history of malignancy, active infection and chronic drug use (e.g., steroids), other chronic inflammatory diseases such as diabetes mellitus, rheumatoid arthritis, malignancies, and pregnancy were excluded. Of the 214 patients, 196 met the inclusion criteria. The mean age of the study population was 46±12 years, and the male/female ratio was 29/167. Demographic characteristics and hematological data are shown in Table 1.

PCT in the male group was found to be significantly lower (p=0.024). The mean PLT count was 275±58 cells/L, MPV was 10±0.96 fL, and PCT was 0.28%±0.05% in 94 patients with tumor size less than 1 cm. The mean PLT count was 299±61 cells/L, MPV was 10±0.85 fL, and PCT was 0.32%±0.06% in 102 patients with tumor size larger than 1 cm. The count of preoperative PLT, MPV, and PCT was significantly higher in patients with tumor size ≥ 1 cm (p=0.005, p=0.001, p<0.001). The mean PLT count was 302±58 cells/L, MPV was 10±0.70 fL, and PCT was 0.33%±0.06% in 28 patients with capsule invasion. The mean PLT count was 285±61 cells/L, MPV was 10±0.95 fL, and PCT was 0.30%±0.06% in 168 patients without capsule invasion. In the presence of capsule invasion, PLT, MPV, and PCT values were significantly higher (p=0.046, p=0.021, p=0.008). The mean PLT count was 298±52 cells/L, MPV was 10±0.97 fL, and PCT was 0.32%±0.06% in 39 patients with lymphovascular invasion. The mean PLT count was 285±63 cells/L, MPV was 10±0.74 fL, and PCT was 0.30%±0.06% in 157 patients without lymphovascular invasion. PCT values were significantly higher in patients with lymphovascular invasion (p=0.045). The mean PLT count was 297±52 cells/L, MPV was 10±0.96 fL, and PCT was 0.29%±0.07% in 79 patients with thyroiditis in nontumor tissue. The mean PLT count was 282±66 cells/L, MPV was 10±0.88 fL, and PCT was 0.31%±0.05% in 117 patients without thyroiditis in nontumor tissues. In the presence of thyroiditis in nontumor tissue, PLT count and PCT were significantly higher (p=0.043 and p=0.001). There was no significant difference between PLT, MPV, and PCT values in the presence of extrathyroidal invasion (p=0.677, p=0.234, p=0.939). Fifteen patients (83%) were classified as T3a due to intrathyroidal dissemination and 3 patients (16%) as T3b because of strep muscle invasion. Eight (72%) of 11 (5.6%) patients with lymph node metastasis were reported as N1a and 3 (27%) as N1b. PLT, MPV, and PCT values were not significantly different in the presence of lymph node metastasis (p=0.556, p=0.715, p=0.544). The relationship of PLT indices with the clinicopathological properties of papillary thyroid carcinoma is presented in Table 2.

Table 1. Demographic characteristics and hematological data

	Mean±SD	
Age (years)	46±12	
<45 (n=90)	35±5.9	
≥45 (n=106)	55±7.9	
Gender		
Female, n (%)	167 (85)	
Male, n (%)	29 (15)	
TSH (mIU/L)	1.9±1.4	Range 0.01–8
PLTs (cells/L)	288±61	Range 128–536
MPV (fL)	10±0.93	Range 6.6–14
PCT (%)	0.30±0.06	Range 0.14–0.55

TSH: Thyroid-stimulating hormone; PLT: Platelet; MPV: Mean platelet volume; PCT: Plateletcrit

Comparison of low and high PLT, MPV, and PCT subgroups (Table 3) once again showed that age, sex, and histological subtype did not correlate with PLT, MPV, and PCT; however, higher PLT, MPV, and PCT values were associated with larger tumor size (p=0.031, p=0.002, p<0.001). High MPV and PCT values were associated with capsule invasion (p=0.007, p=0.032), high PLT and PCT values were associated with lymphovascular invasion (p=0.029, p=0.044). Multifocality, bilaterality, lymph node metastasis, extrathyroidal invasion, presence of thyroiditis, and aggressive histology did not differ between the groups.

Table 2. Relationship of PLT indices with the clinicopathological features of PCT

	n	PLTs (cells/L)	p	MPV (fL)	p	PCT (%)	p
Gender							
Female	167	292±60		10±0.93		0.30±0.06	
Male	29	263±62	0.021	10±0.95	0.948	0.27±0.06	**0.024**
Age (years)							
<45	90	296±62		10±0.91		0.31±0.06	
≥45	106	281±59	0.138	10±0.95	0.742	0.29±0.06	0.172
Tumor size							
<1 cm	94	275±58		10±0.96		0.28±0.05	
≥1 cm	102	299±61	**0.005**	10±0.85	**0.001**	0.32±0.06	**<0.001**
Capsule invasion							
Yes	28	302±58		10±0.70		0.33±0.06	
No	168	285±61	**0.046**	10±0.95	**0.021**	0.30±0.06	**0.008**
Lymphovascular invasion							
Yes	39	298±52		10±0.97		0.32±0.06	
No	157	285±63	0.099	10±0.74	0.380	0.30±0.06	**0.045**
Multifocality							
Yes	63	289±60		10±0.99		0.30±0.06	
No	133	288±62	0.742	10±0.78	0.634	0.30±0.06	0.606
Bilaterality							
Yes	48	299±60		10±0.98		0.30±0.06	
No	148	284±1.7	0.123	10±0.77	0.887	0.31±0.06	0.142
Lymph node metastasis							
Yes	11	288±61	0.556	10±0.65	0.715	0.30±0.05	0.544
N1a	8	298±39		10±0.62		0.30±0.03	
N1b	3	281±102		11±0.32		0.31±0.10	
No	185	293±56		10±0.94		0.30±0.06	
Extrathyroidal invasion							
Yes	18	299±78	0.677	10±0.94	0.234	0.30±0.06	0.939
T3a	15	303±84		10±0.82		0.31±0.08	
T3b	3	277±32		10±0.96		0.28±0.05	
No	178	287±59		10±0.81		0.30±0.08	
Thyroiditis							
Yes	79	297±52		10±0.96		0.29±0.07	
No	117	282±66	**0.043**	10±0.88	0.863	0.31±0.05	**0.032**
Aggressive histology							
Yes	191	303±68.22		10±0.93		0.30±0.06	
No	5	287±61	0.604	10±0.73	0.866	0.32±0,07	0.573
Histologic subtype							
Classic	113	291±65		10±0.96		0.30±0.07	
Follicular	83	284±56	0.588	10±0.88	0.929	0.30±0.05	0.701

PLT: Platelet; PCT: Plateletcrit; MPV: Mean platelet volume

Table 3. Comparison of clinicopathological features in the low and high PLT and indices

	Low PLT (<286.5×109 cells/L)cells/L)	High PLT (≥286.5×109	p	Low MPV (<10.8 fL)	High MPV (≥10.8 fL)	p	Low PCT (<0.29%)	High PCT (≥0.29%)	p
Gender									
Female	80 (48%)	87 (52%)	**0.015**	77 (46%)	90 (54%)	0.414	64 (38%)	103 (62%)	0.312
Male	21 (72%)	8 (28%)		11 (38%)	18 (62%)		14 (48%)	15 (52%)	
Age (years)									
<45	41 (46%)	49 (54%)	0.123	40 (44%)	50 (56%)	0.906	33 (37%)	57 (63%)	0.410
≥45	60 (57%)	46 (43%)		48 (45%)	58 (55%)		45 (43%)	61 (57%)	
Tumor size									
<1 cm	56 (60%)	38 (40%)	**0.031**	53 (56%)	41 (44%)	**0.002**	53 (56%)	41 (44%)	**<0.001**
≥1 cm	45 (44%)	57 (56%)		35 (34%)	67 (66%)		25 (25%)	77 (75%)	
Capsule invasion									
Yes	10 (36%)	18 (64%)	0.070	6 (21%)	22 (79%)	**0.007**	6 (21%)	22 (79%)	**0.032**
No	91 (54%)	77 (46%)		82 (49%)	86 (51%)		72 (43%)	96 (57%)	
Lymphovascular invasion									
Yes	14 (36%)	25 (64%)	**0.029**	14 (36%)	25 (64%)	0.207	10 (26%)	29 (74%)	**0.044**
No	87 (55%)	70 (45%)		74 (47%)	83 (53%)		68 (43%)	89 (57%)	
Multifocality									
Yes	30 (48%)	33 (52%)	0.451	28 (44%)	35 (56%)	0.930	23 (37%)	40 (63%)	0.517
No	71 (53%)	62 (47%)		60 (45%)	73 (55%)		55 (41%)	78 (59%)	
Bilaterality									
Yes	20 (42%)	28 (58%)	0.116	22 (46%)	26 (54%)	0.881	15 (31%)	33 (69%)	0.164
No	81 (55%)	67 (45%)		66 (45%)	82 (55%)		63 (43%)	85 (57%)	
Lymph node metastasis									
Yes	4 (36%)	7 (64%)	0.300	4 (36%)	7 (64%)	0.558	2 (18%)	9 (82%)	0.132
N1a	3 (38%)	5 (62%)		4 (50%)	4 (50%)		1 (13%)	7 (87)	
N1b	1 (33%)	2 (67%)		0 (0%)	3 (100%)		1 (33%)	2 (67%)	
No	97 (52%)	88 (48%)		84 (45%)	101 (55%)		76 (41%)	109 (59%)	
Extrathyroidal invasion									
Yes	7 (39%)	11 (61%)	0.260	10 (56%)	8 (44%)	0.340	5 (28%)	13 (72%)	0.274
T3a	5 (33%)	10 (67%)		8 (53%)	7 (47%)		4 (27%)	11 (73%)	
T3b	2 (67%)	1 (33%)		2 (67%)	1 (33%)		1 (33%)	2 (67%)	
No	94 (53%)	84 (47%)		78 (44%)	100 (56%)		73 (41%)	105 (59%)	
Thyroiditis									
Yes	36 (46%)	43 (54%)	0.170	34 (43%)	45 (57%)	0.667	28 (35%)	51 (65%)	0.306
No	65 (56%)	52 (44%)		54 (46%)	63 (54%)		50 (43%)	67 (57%)	
Aggressive histology									
Yes	2 (40%)	3 (60%)	0.600	1 (20%)	4 (80%)	0.237	2 (40%)	3 (60%)	0.992
No	99 (52%)	92 (48%)		87 (46%)	104 (54%)		76 (40%)	115 (60%)	
Histologic subtype									
Classic	55 (49%)	58 (51%)	0.350	50 (44%)	63 (56%)	0.831	42 (37%)	71 (63%)	0.381
Follicular	46 (55%)	37 (45%)		38 (46%)	45 (54%)		36 (43%)	47 (57%)	

PLT: Platelet; MPV: Mean platelet volume; PCT: Plateletcrit

DISCUSSION

PLT count and PLT indices can be used as markers in cancer patients in addition to cardiovascular, cerebrovascular, thromboembolic, and inflammatory diseases.[10] PLTs have a role in promoting systemic inflammation, proliferation, survival, migration, and angiogenesis, as well as increasing chemokines that contribute to cancer development and progression with the release of regulatory T cells, suppressing antitumor immunity.[14,15] Cancer cells have been shown to secrete myeloid growth factors such as granulocyte colony-stimulating factor, IL-1, IL-6, and tumor necrosis factor-alpha, resulting in leukocytosis, PLTs, and neutrophilia.[16–18] Our study has the pioneer comparison of clinicopathological properties with PLT indices in papillary thyroid carcinoma and concludes that the PLT count was significantly higher in tumors larger than 1 cm and in patients with capsule invasion. These features are prognostic and hope that PLT counts may be a parameter that can be used to determine prognosis in papillary thyroid carcinoma. Additionally, the PLT count is also significantly high in the presence of thyroiditis in nontumor tissue, which emphasizes the importance of PLTs in inflammation.

Increased proinflammatory cytokines cause proliferation, and megakaryocytes are converted into PLTs.[19] Examining the role of PLTs in infection and inflammation in their study, Klinger MH et al.[20] reported that megakaryocytic thrombocytosis may be caused by the release of proinflammatory agents such as IL-1, IL-2, and IL-6. However, PLTs ensure the release and production of vascular endothelial growth factor, which plays a role in tumor angiogenesis and immunization.[21] The count of PLT in different organ cancer surged; PLT levels were found to be significantly lower in cases of nonsmall cell lung cancer and epithelial ovarian cancer, whereas no changes were observed in breast and colon cancer.[22,23] We have demonstrated the importance of PLT count and PLT indices both in thyroid cancer and thyroiditis tissue.

If PLTs have an important place in tumor angiogenesis, MPV can also be used as a marker for angiogenesis, reflecting PLT activity. Larger PLTs are more metabolically and enzymatically active than smaller PLTs.[24] MPV shows PLT function. MPV has been investigated in many types of cancer. In patients with osteosarcoma, Gou et al.[25] examined the prognostic value of MPV and concluded that MPV may be an independent prognostic factor. Similarly, Zhu et al.[26] examined the diagnostic value of PLT indices in colorectal cancer and found that MPV was associated with vascular invasion. Baldane et al.[27] reported significantly higher MPV levels compared with benign goiters and healthy people and significantly reduced tumor removal. In our study, high MPV values were also associated with tumor size and capsule invasion in papillary thyroid carcinoma. MPV values are thought to have an active role in evaluating papillary thyroid carcinoma prognosis along with PLT count.

There are not many studies about PCT in thyroid cancer in the literature. PCT is an index of PLT mass, calculated using PLT and MPV.[13] PCT can be used to determine the need for transfusion.[28] Machairas et al.,[29] in their studies comparing multinodular goiter and papillary thyroid cancer, found that PCT did not differ significantly between multinodular goiter and papillary cancer cases, but it was higher in T3 tumors than T1 and T2 tumors in papillary cancer cases. Yaylaci et al.[30] found no significant difference for PCT in their study comparing benign goiters and thyroid cancers. Oncel et al.[22] compared patients with benign ovarian masses with a healthy control group. PCT measurements were found to be higher but not significant in epithelial ovarian cancer patients. There is evidence in the literature that PCT can be used in both diagnostic and prognosis in colorectal cancer.[26] Wang et al.[31] concluded that PCT in resectable lung cancer may be a potential prognostic factor. PCT values were found to be significantly higher in case of capsule invasion and tumor size larger than 1 cm in our study. In addition, significantly high PCT values were found in the presence of lymphovascular invasion. In addition to PLT count, PCT was significantly high in the presence of thyroiditis in nontumor tissue.

Our study has limitations because of retrospective design and demonstrating only single-center results. Also, the numbers of patients were limited. However, our results have reflected highly selected patient results, as conditions that can alter PLT count or indices result have been excluded in the data. All the hematological parameters have been analyzed in only one laboratory to minimize the analytic differences.

In conclusion, The *BRAF* mutation, combined testing of *RET/PTC, NTRK, RAS*, and *PAX8-PPARγ* expressed microRNAs, also appears to be a promising diagnostic approach for distinguishing benign from malignant thyroid neoplasm.[32] In fact, they are highly costly and could not be obtained widely all over the world but the measurement of PLT count and indices is effective, safe, and easily obtained. Therefore, preoperative PLT, MPV, and PCT may be valuable to predict adverse clinicopathological outcomes in papillary thyroid carcinoma.

Disclosures

Ethics Committee Approval: The study was approved by the Istanbul Kanuni Sultan Suleyman Training and Research Hospital Ethics Committee (No: 103, Date: 24/06/2020).

Informed Consent: Written informed consent was obtained from all patients.

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