

lesions by performing RNA-sequencing and bioinformatics analysis on a panel of 12 fresh tissue samples comprising HPV(+) cervical intraepithelial neoplasia 3 (CIN3) and carcinoma at FIGO IA1-IIB stages, plus normal epithelium. PB samples were obtained from women with CIN3 and stage IA cancer immediately prior to treatment; PB from healthy women was used as control. Subsets of PB lymphocytes were phenotyped using multicolor flow cytometry.

Results Among the differentially expressed genes identified, there were a considerable number of genes that, according to Gene Ontology, are responsible for inflammatory and innate immune responses (including interferon type I and II pathways) and belong to the system of self/non-self DNA/RNA recognition, with multiple anti-inflammatory factors found to be down-regulated, while a spectrum of interferon-stimulated genes, anti-viral/anti-microbial factors, pro-inflammatory cytokines, as well as markers of immune suppression were found up-regulated in invasive cancer. Accordingly, 'Influenza A' and 'Pyrimidine metabolism' KEGG pathways appeared to be significantly enriched. SPEED enrichment analysis revealed the TLR-, TNF alpha-, and IL1-dependent signalings among the top pathways lying behind the alterations of gene expression patterns observed at the initial stage of invasion. PPI network analysis confirmed close interrelation of differently expressed genes encoding molecular components of inflammatory response and virus recognition system. Among the circulating lymphocyte functional markers that may mirror the described immune alterations, the expression of CD161 in iNKT/NK-like T/NK cells (defined by CD3/CD56, Va24Ja18/Vb11-TCR, and CD4/CD8), the level of CD27 and delta2/delta1 ratio in Tgammadelta subpopulation (defined by CD3/TCRgd), and co-expression pattern of PD1/PDL1/LAG3/TIM3 in 4 subsets of CD4/CD8 T cells defined by the level of CD25/CD127, as well as in NK/NKT cells, were measured, and specific correlated differences between the control and cancer groups and between different lymphocyte populations were detected.

Conclusion The findings suggest deep involvement of the inflammation-associated and IC-mediated mechanisms and coordinate contribution of various T cell subsets with innate-like properties in initiation and promotion of invasive growth of cervical carcinoma both at local and systemic levels.

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THE EFFECT OF OTHER HIGH-RISK HPV TYPES ON CERVICAL INTRAEPITHELIAL NEOPLASIA AND CANCER

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Introduction/Background Cervical cancer is the most common gynecologic cancer in worldwide with an incidence of 13,1/100.00 and has a high mortality rate of 6,9/100.000. High-risk human papilloma virüs (HPV) is the main cause of cervical squamous intraepithelial lesions and invasive cervical cancer. HPV 16 and HPV 18 are the most leading types in cervical cancer and cervical neoplasms. Some studies found a significant effect of other high-risk HPV types on cervical carcinogenesis some found non-significant. The effect on cervical carcinogenesis of co-infections with other high risk HPV types

remains unclear. The purpose of this study is to evaluate the influence risk of cervical carcinogenesis of the other high risk HPV types.

Methodology From January 2016 to May 2020, patients who screened with cotest (pap smear and HPV DNA) and had a high risk HPV DNA positivity underwent a colposcopic analyses and biopsy enrolled the study. Patients evaluated from A Gynaecologic Oncologist or a trained fellow of Gynaecologic Oncology at department of Gynecologic Oncology of Ankara University Faculty of medicine. Patients who have a high risk HPV positivity, age between 25–65 and non vaccinated for HPV included in the study. The exclusion criteries were pregnant patients, age <25 and >65, treated before for cervical intraepithelial neoplasia, missing medical records, radiation therapy and total hysterectomy history.

Results Table 1 summarizes the demographic data of the patients. CIN2+ results are seen mostly at HPV 16 group (n=60, 36,4%) then respectively nonHPV 16/18 (n=40, 24,2%), HPV 18 (n= 36,21,8%), non 16/18+16/18 (n=29, 17,6%) group. CIN 2+ results was found in 44,2% (n= 73), 35,8% (n=59), 12,7% (n=21), 7,3% (n=12) of patients with NILM, HSIL,ASCUS and LSIL respectively. Postmenopausal status taken as a reference; a significant difference was observed in premenopausal patients (OR= 2,688, 95% CI = 1,494–4,836). Gravity and number of colposcopic biopsy

Abstract 495 Table 1

Variables		
Age	Mean±SD	41,93±9,63
Gravidity	Mean±SD	1,93±1,33
	Median (Min.-Max.)	2,00 (0,00-8,00)
Menopausal Status, n(%)	Premenopause	388 (74,6)
	Postmenopause	132 (25,4)
Smoking, n(%)	No	349 (67,1)
	Yes	171 (32,9)
Contraception Method, n(%)	Intrauterine device	54 (10,4)
	Oral contraceptives	29 (5,6)
	Tubal ligation	19 (3,7)
	Condom	73 (14,0)
	No method usage	334 (64,2)
	Other	11 (2,1)
Number of colposcopic biopsy	Mean±SD	1,65±0,76
	Median (Min.-Max.)	2,00 (1,00-5,00)
Condyloma, n(%)	Negative	459 (88,3)
	Positive	61 (11,7)
HPV Genotype, n(%)	16	151 (29,0)
	18	71 (13,7)
	Non16/18	228 (43,8)
	Non16/18+16/18	70 (13,5)
Cytology results, n(%)	HSIL	71 (13,7)
	LSIL	45 (8,6)
	ASCUS	95 (18,3)
	NILM	309 (59,4)
	ECC results, n(%)	
	CIN1	3 (1,8)
	CIN2	9 (5,6)
	CIN3	11 (6,9)
	Carcinoma	14 (8,8)
	Normal	123 (76,9)

Abstract 495 Table 2

≥ CIN2 Colposcopic Biopsy Results (According to HPV Genotypes and Cytology Results)					
HPV Genotypes	Cytology Results				
	NILM n - (%)	ASCUS n - (%)	LSIL n - (%)	HSIL n - (%)	TOTAL n - (%)
HPV 16	24 (40)	6 (10)	2 (3,3)	28 (46,7)	60 (100) - (36,3)
HPV 18	20 (55,6)	5 (13,9)	1 (2,8)	10 (27,8)	36 (100) - (21,8)
non HPV 16/18	17 (42,5)	6 (15)	7 (17,5)	10 (25)	40 (100) - (24,2)
non 16/18+16/18	12 (41,4)	4 (13,8)	2 (6,9)	11 (37,9)	29 (100) - (17,7)
TOTAL n - (%)	73 (44,2)	21 (12,7)	12 (7,3)	59 (35,8)	165 (100)

had a statistically significant effect on CIN2+ results (OR= 1,155, 95% CI=1,006–1,326, OR= 1,964, 95% CI= 1,531–2,519). nonHPV16/18 had taken as a reference the other HPV groups had a statistically significant effect on CIN2+ results, HPV 16 (OR= 3,099, 95% CI= 1,933–4,968), HPV 18 (OR= 4,834, 95% CI=2,715–8,608), nonHPV16/18 +HPV16/18 (OR=3,324, 95% CI= 1,851–5,969). The most effective variable on CIN2+ results is endocervical curettage (OR= 28,571, 95% CI=17,355–47,037). The effect of cytology results ASCUS had taken as reference value, NILM and LSIL had no significant effect (p=0,759 and p= 0,553 respectively). HSIL had a statistically significant effect on results (OR= 17,325, 95% CI=7,883–38,077). Table 2 shows HPV genotypes and association of <CIN2 and CIN2+ results. **Conclusion** Almost fifty percent of HPV 18 and nonHPV16/18+HPV16/18 types are associated with CIN2+ lesions. Non16/18 HPV types are associated with a 17% percent of CIN2+ lesions. According to cytology results 44,2% of patients have negative cytology and non16/18 HPV types have 42,5% percent of negative cytology. So non16/18 HPV types are not mostly associated with high grade lesions but detected high lesions are mostly associated with negative cytology.

Disclosures

Table 1 :Demographic data of the patients

Table 2 : HPV genotypes and association of <CIN2 and CIN2+ results.

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UROLOGICAL COMPLICATIONS OF CERVICAL CANCER TREATMENT : A RETROSPECTIVE ANALYSIS OF 420 PATIENTS

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Introduction/Background Cervical cancer is oftentimes plagued by several urological complications during or post treatment. Early disease is mainly managed with radical hysterectomy, while more advanced disease is usually treated by chemoradiation. Although urological complications of cervical cancer

treatment have declined during the past decades, owing to improvements in various therapeutic modalities, the incidence of those complications has not yet precisely defined.

Methodology Cervical cancer patients between 2009 and 2020 were retrospectively reviewed from the cancer database of our tertiary institution.

Results 420 women were diagnosed with cervical cancer of any stage in our cancer hospital. 122 (29%) of those women had early stage disease and thus were managed with radical hysterectomy (RH); the remaining 294 (71%) underwent chemoradiation, chemotherapy, or palliative therapy. 5 out of 122 RH patients (4%) experienced urological adverse events, and namely intraoperative ureteric injury, intraoperative and urinary bladder injury and postoperative ureteral necrosis. One patient (0.8%) was managed with primary end to end ureteral anastomosis, another (0.8%) with intraoperative bladder repair, one patient (0.8%) had Boari flap formation, and two (1.6%) underwent ureteral reimplantation (reoperation on the 10th and 14th postoperative day respectively). In 24 RH patients (19.6%) prophylactic cystoscopic ureteral stenting had taken place before the operation. As for the non RH group (294 patients) 10 (3.4%) had prophylactic cystoscopic stenting, while 3 patients (1%) underwent nephrostomy placement.

Conclusion Cervical cancer management –either surgical or conservative –is often accompanied by various urological complications. Prophylactic ureteral stenting, meticulous surgical technique, prompt diagnosis and management of urological adverse events are of paramount importance when dealing with cervical cancer.

Disclosures The authors declare no conflicts of interest.

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CONCURRENT CHEMORADIOTHERAPY FOLLOWED BY SURGERY FOR CERVICAL CANCER: A MULTICENTER RETROSPECTIVE STUDY OF 126 CASES

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Introduction/Background Cervical cancer is the third leading cause of cancer death in women worldwide. Radio-