patients were divided into 2 groups: group 1 - HPV-positive (n = 14) and group 2 - HPV-negative (n = 48). CVF proteomic analysis was carried out by the bottom-up method (HPLC-MS/MS) on an Agilent 1100/7 Tl LTQ-FT Ultra mass spectrometer (Thermo Electron, Germany). The search for protein identification and semiquantitative analysis was carried out against the UniProt Knowledgebase (UniProtKB, version 16.05.2014) using the MaxQuant software, version 1.1.1.2.

Results None of the vaccinated women showed cervical pathology by cytology. In 6 cases, a biopsy of the cervix was performed in the presence of HPV carcinogenic risk together with mild changes in the cervix epithelium during colposcopy. The histological diagnosis in all cases (n = 6) corresponded to CIN1.

In total, more than 419 different proteins were detected in CVF samples. The level of 34 CVF proteins were significantly different in HPV positive group compared to HPV negative patients (p<0.05). Among them, 9 proteins were involved in the innate immunity processes (APOB, FABP5, GRN, HP, MUC5AC, OLFM4, PKP1, QSOX1, S100A8).

Conclusion A high incidence of HPV was revealed in Russian women previously vaccinated against HPV (23%). CIN 1 was detected in 6 HPV-positive vaccinated women (43%) with normal cytology (NILM). Proteomic analysis of CVF can be proposed as a non-invasive method for detecting biomarkers for early diagnosis of CIN. This work was supported by RSF grant No. 18-75-10097.

Disclosures Nothing to disclose.

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THE ROLE OF KI-67, P16 AND BCL-2 IN THE MANAGEMENT OF CERVICAL NEOPLASIA

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Introduction/Background Cervical cancer, a common gynecological tumor, has a high mortality and it seriously threatens the health of the women. The biomarkers of cell proliferation and apoptosis indicate the early carcinogenesis and are useful for future patient monitoring. HPV persistent infection causes overexpression of P16, but this could be also in normal tissue. P16 is important and useful for cervical cancer screening, but combined with other biomarker - Ki-67, which is a marker of cell proliferation. In normal tissues, the simultaneous expression of P16 and Ki-67 is less likely to occur. Bcl-2 is an intracellular membrane protein which prevents apoptotic cell death and it can be used as a biomarker, too.

Methodology We selected a number of 40 paraffin embedded specimens of cervical tissue from patients diagnosed with cervical pathology, who were admitted in our department from 1-st of January 2018 till 31-th of December 2019. The specimens groups were formed by L-SIL (10), H-SIL (10), scuamous cervical carcinoma – SCC (10) and nontumoral cervical tissue (10) as control group. For all the specimens was performed the histopatological exam and the immunohistochemistry for Ki-67, P16 protein and Bcl-2 protein. Expression of Ki 67, P16 protein and Bcl-2 was detected and the diagnostic values were analyzed.

Results Positive rates of Ki 67 and P16 expression in H-SIL and SCC groups were significantly higher than those in L-SIL

and control group. In our study the expression's intensity of P16 and Ki-67 was positively correlated with the degree of cervical lesions. The immunostaining for Bcl-2 is highly expressed in cervical cancer tissue, compared with nontumoral cervical tissue. The difference is not well expressed compared to H-SIL and L-SIL.

Conclusion Cervical cancer is the malignant tumor with a known etiology, so that prophylactic measures could be taken. The combination of P16 and Ki-67 can identify patients with high risk of SCC and reduce the rate of misdiagnosis. This is of high value for the differential diagnosis between SCC and H-SIL. Bcl-2 is an important regulator of apoptosis. The relationships of tumor genesis with anti-apoptotic genes and proapoptotic genes have been confirmed. Combined with other biomarkers, Bcl-2 could be usefull in assesing the patients' prognosis.

Disclosures I have nothing to disclose.

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PREOPERATIVE MEASUREMENT OF TUMOR VOLUME IN EARLY CERVICAL CANCER. IS IT RELIABLE?

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Introduction/Background Maximum diameter-based tumour measurement is the standard method to asses tumour size and staging pre and postoperatively. Traditionally, clinically estimation of tumour size was the preferred preoperative measuring tool.

Nowadays, thanks to the availability of advanced imaging techniques, preoperative staging could be done more precisely.

Several studies have analysed the correlation between the tumour size measured with MRI and ultrasound and final pathology findings.

In this study we analyse not only the correlation of diameter-based tumour size, but also the correlation of tumour volume estimation.

Methodology A secondary analysis of the SUCCOR study was performed (European patients with FIGO 2009 stage IB1 cervical cancer that underwent radical hysterectomy from January 1st, 2013 to December 31st, 2014). Patients with previous conization were excluded. Patients with at least 3 different tumour measurements both in MRI or ultrasound and in the final pathology report were included. The 3 diameters measured to calculate the volume were defined as: craniocaudal diameter (dcc); anteroposterior diameter (dap) and the largest lateral diameter (dl).

Tumour volume estimation was calculated using the ellipsoid formula (V = dcc x dap x dl x π /6). Intraclass Correlation Coefficient (ICC) was applied to study the correlation of diameter-based tumour size and tumour volume estimation between MRI and pathology report and Ultrasound and pathology report.

Results 693 patients were included in the final analysis of SUCCOR study. 137 of them had both preoperative MRI with 3 different measures (Anteroposterior, Craniocaudal and largest lateral diameter) and pathology report. 81 patients had the 3 diameters measured preoperatively by ultrasound.

When performing a preoperative MRI, the ICC between MRI and final pathology for maximum diameter size was 0.71 (0.61–0.78) and for tumour volume 0.53 (0.38–0.64).

Preoperative ultrasound compared to final pathology report showed a ICC of 0-87 (0.8-0.91) for maximum diameter size and 0.64 (0.4-0.78) for tumour volume measurement.

Conclusion Maximum diameter size showed a good correlation (ICC=0.75-0.9) with the pathology report when measured preoperatively by ultrasound and a moderate correlation (ICC=0.5-0.75) when measured by MRI. For tumour volume measurement both ultrasound and MRI showed a moderate correlation with the final pathology report.

Disclosures All authors contributed to writing the manuscript and read and approved the final manuscript.

The authors declare no conflict of interest.

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SHOULD WE REALLY ABANDON MINIMALLY INVASIVE SURGERY IN EARLY-STAGE CERVICAL CANCER? ONCOLOGICAL RESULTS OF LAPAROSCOPICALLY ASSISTED RADICAL VAGINAL HYSTERECTOMY

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Introduction/Background Recent evidence indicates that some minimally invasive surgery (MIS) approaches, such as laparoscopic- and robotic-assisted radical hysterectomy, offer lower survival rates to patients with early-stage cervical cancer compared with open radical hysterectomy. We evaluated the oncological results of a different MIS approach, that of laparoscopically assisted radical vaginal hysterectomy (LARVH) in the treatment of patients with early-stage cervical cancer.

Methodology From January 2001 to December 2018, patients with early-stage cervical cancer (IA1 with lymphovascular invasion, IA2, IB1, and IIA < 2 cm; FIGO 2009) were treated by LARVH. Colpotomy and initial closure of the vagina were performed following the Schauta procedure, avoiding manipulation of the tumor. Laparoscopic sentinel lymph node (SLN) biopsy was performed in all cases. Women treated between 2001 and 2011 also underwent systematic bilateral pelvic lymphadenectomy after SLN biopsy. Adjuvant radiotherapy or chemo-radiotherapy was administered according to standard guidelines.

Results One hundred fifteen patients were included. Intraoperative complications occurred in nine patients (7.8%). Adjuvant radiotherapy or chemoradiotherapy was administered to 35 (30.4%) and three (2.6%) patients, respectively. After a median follow-up of 87.8 months (range 1–216), seven women (6%) presented recurrence (three pelvic and two paraaortic recurrences, and two had distant metastases). Four women died (mortality rate 3.4%). The three and 4.5-year disease-free survival rates were 96.7% and 93.5%, respectively, and the overall survival was 97.8% and 94.8%, respectively.

Conclusion LARVH offers excellent disease-free and overall survival in women with early stage cervical cancer and can be considered as an adequate MIS alternative to open radical hysterectomy.

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IMPACT OF AGE ON CANCER SPECIFIC SURVIVAL IN PATIENTS WITH LOCALLY ADVANCED CERVICAL CANCER

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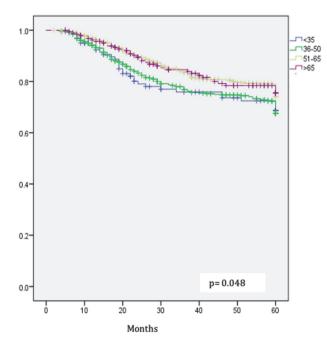
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Introduction/Background Cervical Cancer (CC) is uncommon in very young (<35 years) and in geriatric women (> 65 years), age as a prognostic factor is still controversial. The extremes of life had certain risk factors for being diagnosed with locally advanced cervical cancer (LACC); one of them is that in young women, there is a belief that the disease does not occur; therefore, lack of knowledge of the signs, symptoms and, as an essential factor, a lack of adherence to screening is common. In women older 65 years, the screening has been suspended, explaining how this group of women tend to be diagnosed in advanced stages.

This work aims to compare sociodemographic, clinical, and pathological characteristics, response to treatment, disease-free survival, overall survival, and cancer-specific survival in patients with LACC treated with concurrent chemoradiotherapy, clustered by age.

Methodology It is a retrospective study in patients with LACC treated at the National Cancer Institute of Mexico City from 2005 to 2014. A descriptive, comparative, and survival and cancer specific analysis was conducted.

Results From a total of 2,091 patients with LACC, we found 125 patients (9.7%) younger than 35 years (group 1), 533 (41.35), age between 36–50 years (group 2), 444 (34.4%) between 51–65 years (group 3) and 189 (14.6%) of patients 66 years or older. The general characteristics are found in table 1. More than 50% of women from group 4



Abstract 445 Figure 1 Cancer specific survival