DEFINING THE LONGITUDINAL RISK OF CIN3+ IN PATIENTS WITH LESS THAN CIN2 COLPOSCOPY FOLLOWING INDEX HIGH GRADE CYTOLOGY

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Objectives To determine the baseline and cumulative risk of CIN3 and invasive cervical cancer in patients with <CIN2 colposcopy following a high-grade screening cytology

Methods By linking administrative databases including cytology, pathology, cancer registries and physician billing history, a population-based cohort study was performed on women with <CIN2 initial colposcopy between January 2012– December 2013 including only women with antecedent high grade cytology (ASC-H, HSIL, invasive squamous cell carcinoma(SCC), adenocarcinoma, AGC or AIS. Three and five-year risks of CIN3 and invasive cervical cancer were generated using Kaplan Meier survival analysis.

Results Among 4168 women with ASC-H, HSIL, SCC or adenocarcinoma on screening cytology, the 3/5-year CIN3 incidence rates were 17.7%/20.0%(no biopsy), 13.0/15.1%(negative biopsy) and 18.9%/20.0%(LSIL biopsy) while for AGC/AIS(n=944) cytology, the respective 3- and 5-year rates of CIN3 were 7.42%/8.39%(no biopsy), 7.41%/9.26%(negative biopsy) and 7.695/7.69%(LSIL biopsy). The 3 and 5-year invasive cancer rates were: 1.25%/1.68% (no biopsy), 0.78%/1.04% (negative biopsy) and 0%/0%(LSIL biopsy) for ASC-H, HSIL, SCC or adenocarcinoma and 1.12%/1.54% (no biopsy) , 0.46%/0.46%(negative biopsy) and 0%/0.0% (LSIL biopsy) after AGC/AIS screening cytology. By screening cytology, participants referred for HSIL had the highest 3- and 5-year rates of CIN3(18.9% and 21%), compared to AGC (7.22%/8.28%) and ASC-H(18.9%/18%). The 3- and 5-year invasive cancer were 1.38%/1.75% for HSIL, 0.85%/1.17% for AGC and 0.91%/1.36% for ASC-H.

Conclusions In patients referred for high grade cytology where colposcopy shows <CIN2, the subsequent risk of invasive cancer at 5 years is sufficiently elevated to warrant closer surveillance in colposcopy. Risk is slightly less significant for AGC or AIS cytology.

ROLE OF EXTENSIVE PROCESSING OF RISK-REDUCING SALPINGO-OOPHORECTOMY SPECIMENS IN BRCA1/2 GERMLINE PATHOGENIC VARIANT CARRIERS

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Objectives We assessed the importance of extensive processing of risk-reducing salpingo-oophorectomy (RRSO) specimens with regard to 1) detecting serous tubal intra-epithelial carcinoma (STIC) or high-grade serous carcinoma (HGSC) at RRSO and 2) development of HGSC in the follow-up after normal RRSO in BRCA1/2 germline pathogenic variant (gPV) carriers.

Methods From Hereditary Breast and Ovarian cancer in the Netherlands (HEBON) study, BRCA1/2 gPV carriers who underwent RRSO between 1995 and 2018 were included. Pathology reports of RRSOs were retrieved from the Dutch pathology registry and extent of processing was assessed. To confirm diagnoses of STIC/HGSC at RRSO or HGSC after normal RRSO, tissue slides of RRSO specimens with (pre) malignancy and from women who developed HGSC after RRSO were reviewed. Fisher’s exact and Mann-Whitney U test were used to compare the extent of processing between the groups.

Results In total 2557 women, of which 1624 BRCA1, 930 BRCA2, and 3 with both BRCA1/2 gPV with 10.5 years of median follow-up were included. 8 isolated STICs and 30 HGSCs at RRSO were found, with 16 HGSCs after normal RRSO. Women with STIC/HGSC at RRSO more often had totally embedded fallopian tubes and ovaries (81.6 and 84.2 versus 61.1 and 65.9% respectively; p=0.01 and p=0.02). Women who did not have their RRSO specimen totally embedded had a 6 times higher risk to develop HGSC during follow-up.

Conclusions Extensive processing of RRSO specimens of BRCA1/2 gPV carriers increased detection of STIC/HGSC at RRSO and subsequently resulted in a risk-reduction for developing HGSC after normal RRSO.

IS IT TIME TO TEST ALL ENDOMETRIAL CARCINOMAS FOR P53 MUTATION?

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Objectives Historically, p53 mutation has been considered a diagnostic marker for serous endometrial carcinoma. Of the recently adopted molecular subtypes, p53 abnormal (copy number high) is the most aggressive. The aim of this study was to explore the histologic and clinical characteristics of p53 abnormal endometrial carcinoma regardless of their histologic subtype or grade.

Methods A total of 146 p53 mutated endometrial carcinoma cases were included (44 cases from the Karolinska Institute, 37 cases from Bern University Hospital and 65 cases from the TCGA database). Based on availability, 1–2 representative digital slides from each case were reviewed. Morphologic, molecular, clinical and follow up data were recorded if available. Survival analysis was performed only on p53 abnormal molecular subtypes.

Results A significant number of p53 abnormal cases (24.2%) classified as low grade (FIGO 1 and 2) endometrioid carcinomas. There was no significant difference in survival among different histologic subgroups (p=0.60). There was no significant difference in survival among low grade (FIGO1 or 2) vs high grade (FIGO3) tumors (p=0.98). Low