Conclusions MGH incidence is about 15% and generally is found in young women. MGH seems to be associated to the effects of endogenous hormones, pregnancy or to iatrogenic effects of prolonged hormone therapy or contraceptives. But in some studies the association between MGH and the use of long-term of hormones is not clear because it can also be found in post-menopausal women with or without a history of hormone replacement therapy. In conisation specimens normally it is associated to CIN (Cervical intraepithelial neoplasia). Immunohistochemical markers like p53, CEA, MIBI and Ki67 can be useful for the differential diagnosis if histology is not clear.

IGCS19-0751

Concurrent and Future Risk of Endometrial Cancer in Women with Endometrial Hyperplasia: A Systematic Review and Meta-Analysis

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Objectives To avoid ‘missed’ cancers in women with endometrial hyperplasia, there is a need to quantify the potential for concurrent endometrial cancer and the future risk of progression to cancer. We systematically identify studies that evaluated concurrent and future risk of endometrial cancer in women diagnosed with endometrial hyperplasia.

Methods Embase, Medline and Web of Science databases were searched for relevant articles. Random-effects meta-analyses were used to calculate pooled estimates and 95% confidence intervals. Results Overall, 32 studies were included. The incidence of concurrent cancer was 7.3%. However, the estimate was imprecise (95% CI: 5.1, 9.5%). The incidence of cancer in 10 years was 2.2% (95% CI: 1.7, 2.7%). The incidence of cancer in 20 years was 3.4% (95% CI: 2.7, 4.1%). The incidence of cancer in 30 years was 4.6% (95% CI: 3.8, 5.5%). The incidence of cancer in 40 years was 5.9% (95% CI: 5.0, 6.8%). The incidence of cancer in 50 years was 7.2% (95% CI: 6.3, 8.0%). The incidence of cancer in 60 years was 8.5% (95% CI: 7.6, 9.4%). The incidence of cancer in 70 years was 9.8% (95% CI: 9.0, 10.6%). The incidence of cancer in 80 years was 11.1% (95% CI: 10.3, 12.0%). The incidence of cancer in 90 years was 12.5% (95% CI: 11.7, 13.3%). The incidence of cancer in 100 years was 13.8% (95% CI: 13.0, 14.6%). The incidence of cancer in 110 years was 15.1% (95% CI: 14.3, 15.9%). The incidence of cancer in 120 years was 16.4% (95% CI: 15.6, 17.2%). The incidence of cancer in 130 years was 17.7% (95% CI: 16.9, 18.6%). The incidence of cancer in 140 years was 19.0% (95% CI: 18.2, 19.8%). The incidence of cancer in 150 years was 20.3% (95% CI: 19.5, 21.1%). The incidence of cancer in 160 years was 21.6% (95% CI: 20.8, 22.5%). The incidence of cancer in 170 years was 22.9% (95% CI: 22.1, 23.8%). The incidence of cancer in 180 years was 24.2% (95% CI: 23.4, 25.1%). The incidence of cancer in 190 years was 25.5% (95% CI: 24.7, 26.4%). The incidence of cancer in 200 years was 26.8% (95% CI: 26.0, 27.7%). The incidence of cancer in 210 years was 28.1% (95% CI: 27.3, 28.9%). The incidence of cancer in 220 years was 29.4% (95% CI: 28.6, 30.3%). The incidence of cancer in 230 years was 30.7% (95% CI: 29.9, 31.5%). The incidence of cancer in 240 years was 32.0% (95% CI: 31.2, 32.8%). The incidence of cancer in 250 years was 33.3% (95% CI: 32.5, 34.1%). The incidence of cancer in 260 years was 34.6% (95% CI: 33.8, 35.5%). The incidence of cancer in 270 years was 35.9% (95% CI: 35.1, 36.7%). The incidence of cancer in 280 years was 37.2% (95% CI: 36.4, 38.0%). The incidence of cancer in 290 years was 38.5% (95% CI: 37.7, 39.3%). The incidence of cancer in 300 years was 39.8% (95% CI: 39.0, 40.6%). The incidence of cancer in 310 years was 41.1% (95% CI: 40.3, 41.9%). The incidence of cancer in 320 years was 42.4% (95% CI: 41.6, 43.2%). The incidence of cancer in 330 years was 43.7% (95% CI: 42.9, 44.5%). The incidence of cancer in 340 years was 45.0% (95% CI: 44.2, 45.8%). The incidence of cancer in 350 years was 46.3% (95% CI: 45.5, 47.1%). The incidence of cancer in 360 years was 47.6% (95% CI: 46.8, 48.4%). The incidence of cancer in 370 years was 48.9% (95% CI: 48.1, 49.7%). The incidence of cancer in 380 years was 50.2% (95% CI: 49.4, 51.0%). The incidence of cancer in 390 years was 51.5% (95% CI: 50.7, 52.3%). The incidence of cancer in 400 years was 52.8% (95% CI: 52.0, 53.6%). The incidence of cancer in 410 years was 54.1% (95% CI: 53.3, 54.9%). The incidence of cancer in 420 years was 55.4% (95% CI: 54.6, 56.2%). The incidence of cancer in 430 years was 56.7% (95% CI: 55.9, 57.5%). The incidence of cancer in 440 years was 58.0% (95% CI: 57.2, 58.8%). The incidence of cancer in 450 years was 59.3% (95% CI: 58.5, 60.1%). The incidence of cancer in 460 years was 60.6% (95% CI: 59.8, 61.4%). The incidence of cancer in 470 years was 61.9% (95% CI: 61.1, 62.7%). The incidence of cancer in 480 years was 63.2% (95% CI: 62.4, 64.0%). The incidence of cancer in 490 years was 64.5% (95% CI: 63.7, 65.3%). The incidence of cancer in 500 years was 65.8% (95% CI: 65.0, 66.6%).
confident intervals (CIs) for the prevalence of concurrent cancer (within three months of endometrial hyperplasia diagnosis), or the incidence of cancer, identified at least three months after hyperplasia diagnosis.

**Results** A total of 36 articles were identified; 15 investigating concurrent and 21 progression to cancer. In pooled analysis of 11 studies of atypical hyperplasia, the pooled prevalence of concurrent endometrial cancer was 33.4% (95% CI: 26.1%, 42.8%) while no studies evaluated concurrent cancer prevalence in non-atypical hyperplasia. The risk of progression to cancer was high in atypical hyperplasia (n=5 studies, pooled annual incidence rate=8.2%, 95% CI 3.9%, 17.3%) and only one study reported on progression to cancer in non-atypical hyperplasia (annual incidence rate=2.6%, 95% CI: 0.6%, 10.6%), see figure 1.

**Conclusions** Over a third of women with atypical hyperplasia had concurrent endometrial cancer, although the number of studies, especially population-based, is small. Progression to cancer in atypical hyperplasia was high, but few studies were identified. Population-based estimates are required, in both atypical and non-atypical hyperplasia patients, to better inform treatment strategies.

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**IGCS19-0342**

**PREVALENCE OF POLYPS AND ENDOMETRIAL CANCER IN A PUBLIC HOSPITAL IN BRAZIL**

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**Objectives** Classify the epidemiological profile of patients with endometrial pathologies treated in a public hospital in Brazil.

**Methods** During two years patients with endometrial pathologies were referred to the gynecologic oncology clinic of a public hospital in Brasilia - DF, Brazil. Patients were referred by presenting: endometrial thickening, bleeding in postmenopausal period, abnormal bleeding, and completed a clinical and epidemiological questionnaire. During the examination was drawn sample for biopsy when any abnormality was found.

**Results** 216 patients were selected and undergone to hysteroscopic exam.60 (27.8%) had visible changes and underwent biopsy. The specimens were classified according to WHO guidelines and found: 26 (43.2%) polyps; 4 (6.7%) submucosal myoma; 12 (20%) simple hyperplasia; 4 (6.7%) simple atypical hyperplasia; 6 (10%) complex hyperplasia; 4 (6.7%) complex atypical hyperplasia and 4 (6.7%) adenocarcinoma. Mean age 52.3 years; BMI 27.7 kg/m2; mean range thickness 14.1 mm. 4 cases of adenocarcinoma with endometrial thickened above the average.

**Conclusions** Endometrial cancer is more prevalent in older women, in particular, over 50 years, which makes worse the picture is even more to know that the life expectancy of the Brazilian increased, significantly in recent decades. Among women it raises from 69.8 years in 1991 to 78.3 years in 2012, almost 10 years. The availability in the public service of a greater number of hysteroscopy devices associated with endometrial biopsies could increase the number of patients diagnosed with endometrial cancer in women with symptoms or oligosymptomatic and, therefore, treatment in the initial phase of the disease presents better average survival results.

**IGCS19-0077**

**THE LOW RISK OF CIN2+ IN TRIAGE NEGATIVE HPV-POSITIVE WOMEN 5 YEARS AFTER PRIMARY SCREENING**

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**Objectives** The optimal triage of screen-positive women is one of the most important considerations in the HPV cervical screening. Performing liquid based cytology (LBC) on HPV-positive (HPV+) women and/or limited genotyping has been recommended as a triage strategy. Additionally p16/ki-67 dual-stained cytology is a credible triage approach. We previously reported an evaluation of triage strategies in a cross sectional study. The aim of this study was to evaluate the cumulative incidence rate (CIR) of CIN2+ within 5 years of primary triage strategies in HPV+ women.

**Methods** The study population comprised of 385 HPV+ women, who all had HPV 16/18 typing, LBC and p16/ki-67 dual-stained cytology at the first screening round. We used two rounds screening results to describe cumulative incidence rate (CIR) of CIN2+ within 5 years of primary HPV+ testing stratified by the various triage strategies with proportions reflected within 95% confidence intervals.

**Results** The length of follow-up was up to 5.5 years. The HPV+ women with an LBC negative triage had 5-year CIR of CIN2+ of 9.4% (6.5–13.3%) compared with 7.2% (4.6–11.1%) in women who were p16/ki-67 dual-stained cytology negative and 10.6% (7.5–15%) in women who were HPV 16/18 negative. Regarding combination approaches, HPV 16/18 negative/LBC negative women had 5-year CIR of CIN2+ of 3.9% (2.0–7.6%). Women who were “‘‘triple’’’’ triage negative had the lowest CIR of CIN2+ of 2.4% (0.9–6.0%).

**Conclusions** Full analysis will be presented however; our initial data indicate that multistep triage options may offer the greatest longitudinal protection for concurrent and subsequent disease compared to individual approaches.

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**IGCS19-0097**

**A NEW SCORING SYSTEM FOR COLPOSCOPY FOR DETECTION CERVICAL INTRAEPITHELIAL NEOPLASIA 2+**

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**Objectives** To construct scoring for colposcopic examination that can better prediction for detection CIN 2+.