

Review

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Review

Endometrial Cancer After Endometrial Ablation: A Narrative Review

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Simple Summary

After endometrial ablation (EA) persistent uterine bleeding indicate that no method of endometrial ablation (EA) eliminates the entire endometrium and hysteroscopy shows distorted and scarred uterine cavity in the majority of women. These observations raise concerns regarding presentation, assessment and stage of potential post-ablation endometrial cancer (PAEC) developing in residual endometrium. To address these concerns, we conducted a systematic search for reports of endometrial cancer (EC) associated with or after EA using multiple data bases from its inception through 2025. Based on 12 studies, at follow up of 1.9-25 years, 43 ECs were identified in 39,795 women with a history of EA; summary incidence of 0.11% (range 0.0 - 1.59%). Based on 43 evaluable cases, the mode and time to presentation, investigation, diagnosis, and stage of PAEC were not altered by EA. We conclude that EA reduces the risk EC significantly, likely due to quantitative reduction in endometrium volume and EA eliminating occult pre- or malignant endometrial cells which are vulnerable to ablation techniques.

Abstract

Post-endometrial ablation persistent uterine bleeding indicates that no method of endometrial ablation (EA) eliminates the entire endometrium and hysteroscopy shows distorted and scarred uterine cavity in the majority of women. These observations raise concerns regarding presentation, assessment and stage of potential post-ablation endometrial cancer (PAEC) developing in residual endometrium. To address these concerns, we conducted a systematic search for reports of endometrial cancer (EC) associated with or after EA using multiple data bases imputing keywords of EC after EA and possible combinations of first- and second-generation EA techniques associated with EC from its inception in the 1980s through 2025. After excluding irrelevant publications, we identified 86 ECs associated with EA described in 20 case reports (N=20), four case series (N=18), eleven cohort studies (N=21), one registry (N=27) and five reviews. Based on 12 relevant studies, at follow up of 1.9-25 years, 43 ECs were identified in 39,795 women with a history of EA; summary incidence of 0.11% (range 0.0 - 1.59%). Based on the remaining 43 evaluable cases of PAEC, the mode and time to presentation, investigation, diagnosis, and stage of PAEC were not altered by EA. We conclude that EA has a protective effect reducing the risk EC significantly, likely due to quantitative reduction in endometrium that can potentially become malignant and the EA process eliminating occult pre- or malignant endometrial tissues which are vulnerable to ablation techniques. The mode and time to presentation, the diagnostic work-up, including endometrial biopsy and hysteroscopy, and stage of PAEC are not altered by EA.

Keywords: endometrial ablation; endometrial cancer; endometrial cancer after endometrial ablation; endometrial cancer associated with endometrial ablation; post-ablation endometrial cancer; post-ablation endometrial biopsy; post-endometrial ablation hysteroscopy

1. Introduction

Endometrial ablation (EA) refers to destruction or elimination of the endometrium using thermal (heat or cold) energy or electro-mechanical removal. It was introduced in the 1980s as a safer, less skill-dependent alternative to hysterectomy for women with abnormal uterine bleeding (AUB) of benign pathology who are unable or unwilling to tolerate or have failed traditional medical/conservative therapies. Soon after the utilization of multiple EA methods, post-endometrial ablation bleeding patterns indicate that no method of endometrial ablation (EA) eliminates the entire endometrium in the majority of women. Consequently, concerns were raised regarding timely diagnosis of potential post-ablation endometrial cancer (PAEC) due to incomplete elimination of the endometrium leaving endometrial nest(s) which could result in sequestered island(s) of endometrial carcinoma (EC) [1].

Furthermore, post-EA hysteroscopy indicated that EA causes significant distortion and scarring of the uterine cavity which could impede and/or mask finding sequestered island(s) of PAEC that could escape detection and possibly delay diagnosis by preventing uterine bleeding which is a common clinical hallmark of EC that prompts investigation [2]. Therefore, PAEC in a distorted uterine cavity may not declare itself with bleeding and be inaccessible to standard evaluation by biopsy and/or hysteroscopy resulting in obscuring and/or delaying diagnosis, upstaging the disease and worsening prognosis.

Conversely, it is argued that EA may reduce the risk of EC since it reduces quantitatively endometrial volume that can potentially become malignant or by eliminating occult pre- or malignant elements, which are vulnerable to EA [3–5]. PAEC concerns are underscored by the fact that EC is the commonest gynecologic malignancy and it is strongly associated with obesity which has reached epidemic proportions [6]. Notably, the estimated relative risk for EC in women with obesity follows a positive correlation reaching odds ratio of approximately 20 in women with BMI ≥ 40 [7]. Also, Mendelian randomization analysis including all reported risk factors of EC, indicates that for every five extra BMI units, EC increases by 88%; a much larger increased risk than that reported in conventional observational studies and meta-analyses [8].

Therefore, the changing landscape of increased prevalence of obesity in women and its strong association with increased risk of EC [7,8], together with the widespread utilization of EA to treat AUB, raises looming challenges as healthcare providers are facing large cohorts of obese and nonobese women who had EA entering their sixth and seventh decades of life—peak years for EC. In this review, we examined all publications describing EC associated with EA to determine incidence, mode and time to presentation, diagnostic work-up, including endometrial biopsy and hysteroscopy and stage of PAEC at hysterectomy.

2. Methods

We conducted a systematic search for English language reports of EC associated with EA using MEDLINE, Google Scholar, PubMed, EMBASE and Cochrane Library data bases from the inception of EA in the 1980s through 2025. We used keywords of EC after EA, EC associated with EA, post-EA EC, post-EA endometrial biopsy, post-EA hysteroscopy. Relevant publications included 20 case reports (N=20) [9–27], four case series (N=18) [28–31], twelve cohort studies (N=21) [4,32–42], one National registry (N=27) [43] and five reviews [2,25,44–46] providing information on 86 EC associated with EA.

3. Results

3.1. Incidence of PAEC

The estimated summary incidence of PAEC, constructed from 11 cohort studies [4,32–34,36–42], and one national registry [43] is 0.11% (range 0.00 – 1.59%) at follow up of 1.9 to 25 years (Table 1). We excluded one additional cohort study reporting incidence 0.11% (4 EC/3,769 patients) because three patients had TCRE for postmenopausal bleeding (PMB) while taking hormone replacement therapy (HRT) and had preoperative risk factors for EC including obesity, hypertension and endometrial polyps during TCRE. The 4th patient had selective resection of polyps and curettage only without having EA [35].

Table 1. Incidence of endometrial cancer (EC) after endometrial ablation (EA). TCRE-transcervical endometrium resection, REA-rollerball endometrial ablation, N/A-nonapplicable, NSS-no statistical significance, HR-hazard ratio, RR-relative risk, IR-Incident risk.

Author [Reference]	Study	No. of EA	Follow-up (years)	Number of EC	Incidence (%)	Statistics
Panoskaltsis [32]	Prospective	193	Median 6 years (5-8)	0	0	N/A
Neuwirth [33]	Retrospective	466	5,063 women-years	2	0.43	NSS
Cooper [34]	RCT	263	6 years (5-7)	1	0.38	N/A
Krogh [36]	Retrospective	367	4,037 women-years	3	0.82	NSS
Grochmal [37]	Prospective	287	20 years	0	0	N/A
Cooper [38]	Retrospective	11,299	Median 6.2 years (2.7-10.8)	2	0.02	N/A
Dood [39]	Retrospective	4,776	Median 4.1 years (1.9- 7.2)	3	0.06	HR=0.45 (0.15-1.40) p=.17
Morelli [40]	Retrospective	63	---	1	1.59	N/A
Singh [4]	Retrospective	1,521	Median 10 years 19,733 women-years	0	0.00	RR=0.01 (0.0-0.28) p=.001
Soini [41]	Retrospective	5,484	39,892 women-years	3	0.05	IR=0.56 (0.12-1.64)
Kalampokas [42]	Retrospective	901	21.5 years (18-25)	2	0.22	P=.001
Flöter Rådestad [43]	Registry	14,175	Median 7.1 years (3.1-13.3)	27	0.19	IR=0.53 (0.03-0.53) for REA IR=1.27 (0.86-1.88) for TCRE
Total	12 Studies	39,795	Median 8.5 years (1.9-25)	43	0.11 (0 -1.59)	

For additional context, we provide a summary of each study.

Panoskaltsis et al reported on their first 193 women, average age 41 (range 24–56) after TCRE and rollerball endometrial ablation (REA) in UK. At a median follow up of 6 years (range 5-8), there was one case of atypical endometrial hyperplasia (AEH) without evidence of EC [32].

Neuwirth et al examined incidence of EC in 509 women with normal endometrial histology who had hysteroscopic EA in two US centers. Among 466 cases, there were two (0.43%) PAEC in a total of 5,063 woman-years; expected incidence was 1.66 cases in an age-matched group with known length of follow-up from the U.S. SEER data; similar to the expected EC incidence in the general population [33].

Cooper et al reported long-term outcomes following microwave endometrial ablation (MEA) or TCRE in Scotland. Of 263 treated women, 236 returned questionnaires. After a minimum of five and maximum of seven years, 20 women in the MEA (16%) and 33 in the TCRE arm (25%) had hysterectomy including one case (1/263; 0.38%) of EC post-MEA [34].

Krogh et al reported on a survey of 367 Danish women following EA during 1990-1996; 52 had EA twice and one three times using rollerball for the fundal area and TCRE for the remaining cavity. Of these, 141 (47%) had hormonal treatment (HT), 35% of which had HT before EA. At 11 years, 82% had returned questionnaires and three women (0.82%) reported EC in 4,037 women-years. According to the Danish National Health Board, the age-standardized incidence of EC rate was 17/100,000 women at age 45 in 1993, which corresponds to the mean age of the women in the middle of the EA period, providing a calculated expected number of 6.8 EC. They concluded that EA does not increase incidence of EC and HT does not influence the course of events [36].

Cooper et al reported risk of further gynaecological surgery and gynaecological cancer following hysterectomy or EA performed between 1989 and 2006 in a population-based retrospective cohort study in Scotland. A total of 37,120 women had hysterectomy, 11,299 underwent EA without a subsequent hysterectomy and 2,779 underwent EA followed by subsequent hysterectomy. The median (interquartile range) duration of follow-up was 11.6 years (7.9 - 14.8) and 6.2 years (2.7 - 10.8) in the hysterectomy and EA (without hysterectomy) cohorts, respectively. In the EA group, two women (0.02%) developed EC [38].

Dood et al investigated whether EA is associated with increased risk or delayed diagnosis of PAEC compared with medical management in women with AUB in a multi-centered retrospective cohort study in UK, between June 1994 and September 2010. All women underwent TCRE, using second-generation EA devices or medical management. Of 234,721 women, 4,776 underwent EA and 229,945 received medical treatment, including combined estrogen-progestin (30,731), progestin-alone (40,457), levonorgestrel intrauterine system (LNG-IUS, 3,588) or expectant/other nonhormone medications. During a median period of 4.07 years (interquartile range, 1.88 - 7.17), EC developed in 3 (0.06%) women in the EA group and 601 (0.26%) in the medical management group (ablation HR, 0.45; CI 0.15 - 1.40; $p = 0.17$). This corresponded with a study-specific annual EC incidence rate of 59.6/100,000 women, 19.3/100,000 in women who underwent EA, and 60.3/100,000 in women who received medical management. There was no difference in EC incidence when comparing first-and second-generation EA methods with medical management [33]. Comparing women who underwent EA with women who received the LNG-IUS, there was a notable change in the hazard ratio (ablation HR, 6.04; CI, 0.61–60.2; $p = .13$). However, this observation was based on EC rates of only 3 of 4 776 and 1 of 3,558 women in the EA and LNG-IUS groups, respectively. In secondary analysis, the LNG-IUS was found to have lower rate of EC than all other treatments (LNG-IUS HR, 0.12; CI, 0.02–0.83; $p = .03$) [39].

Morelli et al reported one EC in 63 women having annual post-EA follow-up and transvaginal ultrasound between January 2000 and August 2014. All women received TCRE or REA or thermal balloon endometrial ablation (TBEA). Nine (14.3%) patients had PMB. Among those with no bleeding, one exhibited endometrial fluid collection and hysteroscopy plus endometrial biopsy identified EC (IA, G2). No patients with uterine bleeding had EC; bleeding attributed to atrophy [40].

Singh et al reported on a retrospective observational study in UK, including 1,521 women (mean age 48 ± 6.3 years) having EA from January 1994 through December 2011. During 18 years, 1,022

women (67.19 %) had first-, and 499 (32.81 %) second-generation EA; TCRE in 843 (55.42 %), bipolar radiofrequency (RF) in 245 (16.11 %), MEA in 243 (15.98 %). At a median follow-up of 10 years (19,733 women-years), none developed EC compared to 261 women who developed EC during the study period in the Yorkshire Cancer Registry database corresponding to a lifetime risk of EC in the general population of approximately 2-3%. (RR 0.0135; CI 0.0007–0.2801; P = 0.0054) [4].

Soini et al examined risk of EC, breast cancer and hysterectomy rate after EA in a retrospective cohort of 5,484 women with EA at mean age (SD, range) 42 years (4.4, 30-49 in Finland between 1997 and 2014). The primary outcome was cancer incidences in the EA cohort compared with those in the background population of the same age. During the study period, the age standardized incidence rate of EC in Finland was 14.2/100,000 women-years adjusted for age according to the World Standard Population. During follow up of 39,892 women-years, the standardized incidence ratio for EC was 0.56 (CI 0.12–1.64); three (0.05%) observed compared with five expected cases. Of the three PAEC, two were diagnosed at an early stage and one at unknown stage [41].

Kalampokas et al assessed incidence of EC after EA (TCRE/REA) in a prospective observational cohort study from Aberdeen Royal Infirmary between February 1990 through December 1997. To assess risk of EC, each woman was matched by age to the annual observed incidence of EC in northeast Scotland for each year from the date of EA until 2015. During the 7-year study period, 901 eligible women (mean age 42.3 ± 5.7 years; range 26-50 years) underwent EA. Of these, 204 (22.6%) subsequently had hysterectomy for reasons other than EC, and 695 (77.1%) did not. The overall incidence of EC was 0.2% (2/901); the risk of developing EC after EA was calculated as 11.1/100,000 women-years. The mean expected incidence for all women and the subgroup with no hysterectomy was estimated to be 26.5 and 35.6 occurrences per 100,000 women-years, respectively ($P < 0.001$). They concluded that the risk of EC could be significantly reduced but not eliminated by EA [42].

Flöter Rådestad et al analyzed data from the Swedish National Patient Registry and National Quality Registry for Gynecological Surgery (SweGCG) of women who had TCRE or REA performed between 1997 and 2017. Women were followed until hysterectomy, diagnosis of EC, or death based on National Cancer Registry and the National Death Registry. Primary aim was to determine long-term incidence of EC after TCRE and REA in a nationwide population. Secondary aim was to assess TCRE versus REA separately. Expected incidence for EC was calculated using Swedish data retrieved from the NORDCAN project taking into account differences of age and follow-up time. In total, 17,296 women (mean age 45.1 years) underwent TCRE ($n = 8,626$) or REA ($n = 8,670$). Excluded were 3,121 (18.0%) who had hysterectomy for benign causes. During a median follow-up of 7.1 years (interquartile range 3.1 - 13.3 years), the cumulative numbers of EC were 25 (0.3%) after TCRE and 2 (0.02%) after REA, respectively. The expected number of PAEC was estimated to be 15 cases. The observed incidence was significantly lower than expected (population-based estimate) after REA but not after TCRE, giving a standardized incidence ratio of 0.13 (CI 0.03 - 0.53) after REA and 1.27 (CI 0.86 - 1.88) after TCRE. Median times to EC were 3.0 and 8.3 years after TCRE and REA, respectively. They concluded that there was a significant reduction of EC after REA, suggesting a protective effect, whereas TCRE showed an incidence within the expected rate [43].

3.2. Circumstances, Characteristics and Outcomes of Patients with PAEC

Of the 86 EC, 43 cases provided no patient details and were included as incident cases in the 12 cohort studies to determine the incidence of PAEC (Table 1). The remaining 43 cases provided sufficient patient information to determine the circumstances, characteristics and outcomes of patients with PAEC and are divided into 5 groups shown in Tables 2–6.

Table 2. Data of 12 cases of endometrial cancer (EC) present during endometrial ablation (EA). NAEH-nonatypical endometrial hyperplasia, HBSO-hysterectomy and bilateral salpingo-oophorectomy; TCRE-transcervical endometrial resection; REA-rollerball endometrial ablation; HRT-hormone replacement therapy; SH-simple hyperplasia, RF-radiofrequency.

Author [reference]	Age at EA (yr)	BMI	Pre-EA biopsy	Type of & time to EA from biopsy	Time to HBSO	Stage of EC
Dwyer [9]	38	>30	Secretory	3 months: TCRE-EC	<3 months	No residual EC, I
Klein [17]	52 PMB. Hart disease	--	Proliferative Given HRT	3 months: REA. D&C-EC	<1 month	Ovarian metastasis II, G3
Steed [19]	41	26	Proliferative	2 months: REA/TCRE EC, G1	3 months	No residual EC I
Smith [28]	3 cases. <50 yr	--	Benign	Bipolar RF/D&C- EC	Unknown	No residual EC x 3 I x 3
Argall [30]				3 months: D&C-EC, G1		
	40	--	Benign	1 month: D&C-EC, G1	3 months	No residual EC. IA, G1
	48	--	Benign		2 months	No residual EC. IA, G1
	50	--	NAEH	11 months: D&C-EC, G1	2 months	No residual EC. IA, G1
	51	--	Benign	17 months: D&C-EC, G1	1 month	No residual EC. IA, G1
	47	--	Benign	1 month: D&C-EC, G1	34 months	IA, G1
	53	--	Unknown	Unknown: D&C-EC, G2	60 months	IA, G2

Table 3. Data of 3 cases of endometrial cancer (EC) likely present during endometrial ablation (EA). BMI-body mass index, REA-rollerball endometrial ablation, TBEA-thermal balloon endometrial ablation, AEH-atypical endometrial hyperplasia.

Author [reference]	Age at EA (yr)	BMI	Pre-EA biopsy	Type of & time to EA	Time to & presentation from EA to diagnosis & treatment	Treatment Stage of EC
Ramy [12]	38	>40	Complex hyperplasia	4 months: REA, no biopsy.	6 months: Persistent bleeding. Hysteroscopy, D&C-EC	HBSO, nodes IA, G1

Baggish [15]	50 PMB	>30	Adenomatous hyperplasia	6 months: REA, no biopsy.	6 months: Persistent bleeding. Pap smear-EC cells	HBSO-EC I, G1
Lee [20]	36	36	Complex hyperplasia	1 month: TBEA, biopsy- AEH	2 months: Persistent bleeding. Vaginal hysterectomy-EC	BSO IA, G1

Table 4. Data of 3 cases with atypical endometrial hyperplasia (AEH) being present during endometrial ablation (EA). BMI-body mass index, TCRE-transcervical resection of the endometrium, REA-rollerball endometrial ablation, CH-complex endometrial hyperplasia, AEH-atypical endometrial hyperplasia, HBSO-hystetectomy and bilateral salpingo-oophorectomy.

Author [referen]	Age/BM I	Pre-EA Biopsy	Type of EA & pathology	Time to presentation	Evaluation	Treatment	Stage
du Toit [11]	39/42	Insufficient	TCRE-AEH. No treatment	1 year. Bleeding. Pap smear-EC cells	Hysteroscopy, excessive bleeding	Converted to HBSO-EC	IA, G1
Horowitz [14]	63/>40	Adenomatou s hyperplasia	REA/D&C- AEH. Refused hysterectomy	14 months. Persistent bleeding & umbilical mass	D&C, biopsy umbilical mass. EC both, poorly differentiated	HBSO, nodes	IV. Died 4 months
Igbal [16]	50/--	CH. Changed to AEH following EC diagnosis	Danazol 800 mg X 4 weeks. TCRE-benign endometrium	3 years. Heavy bleeding	Hysteroscopy, biopsy-EC.	HBSO	IB, G2

Table 5. Data of 9 women with PAEC having been treated with endometrial ablation (EA) for postmenopausal bleeding (PMB). NAEH-nonatypical endometrial hyperplasia, REA-rollerball endometrial ablation, HRT-hormone replacement therapy.

Author [reference]	Ag/BMI. Comorbidity	Pre-EA biopsy	Type EA	Time to and presentation	Evaluation	Treatment	Stage
Margolis [13]	55/>30. Diabetes, hypertension. Progesterone	NAEH	REA Benign. Premarin treatment	3 years Amenorrhea. Urinary stress incontinence	None	HBSO-EC Bladder Suspension	IC, G1 >50% invasion
Sagiv [21]	57/? No risks	Proliferative	TCRE Secretory	3 years Bleeding/pain	Hysteroscopy , biopsy unsuccessful.	HBSO-EC nodes	IC, G1 >50% invasion
Gaia [35]	63/21 Hypertension HRT 8 yr	Benign	TCRE Benign HRT 10 yr	10 years Bleeding/HRT	Hysteroscopy , D&C-EC	HBSO nodes	IB, G2-3
	60/33 Hypertension	Benign	D&C only Benign	8 years Bleeding	Hysteroscopy , D&C- EC	HBSO nodes	IC, G1
	53/39. Hypertension HRT 3y	Benign	TCRE Benign, ADM	5 years Bleeding/HRT	Hysteroscopy , D&C-EC	HBSO, nodes	IA, G1

	56/34. Hypertension	NAEH	TCRE NAEH	6 years Bleeding/HRT	Hysteroscopy , D&C-EC	HBSO nodes	IB, G1
Tsafrir [29]	66/? Hypertension	Benign	TCRE	6 years, Bleeding	Biopsy-EC	HBSO	IA
	62/? Hypertension	Benign	TCRE	6 years bleeding	Biopsy-EC	HBSO + adjuvant	III. Died 9-month
Wortman [31]	56/? Breast cancer	Inactive	TCRE Benign	5 years Intense pain, no bleeding	No attempt	HBSO	III. Mets to ovary

Table 6. Patient demographics, mode of endometrial ablation (EA), presentation, diagnosis, treatment and stage of PAEC of 16 premenopausal women with appropriate indications for EA. BMI-body mass index, HBSO-hysterectomy bilateral salpingo-oophorectomy, NAEH-nonatypical endometrial hyperplasia.

Author [Reference]	Age/BMI	Comorbidity	Pre-EA biopsy	Type of EA Biopsy	Time to presentation	Evaluation	Treatment	Stage
Copperman [10]	51/>30	Diabetes hypertension Colon cancer	NAEH Treated with progestin	Coagulation	5 years. PMB	Biopsy-EC	HBSO, nodes	II/III, G2
Brooks- Carter [18]	50/70	Diabetes hypertension 14wk uterus	Proliferative	Coagulation Myomectomy	14 months. Bleeding	Hysteroscopy biopsy-EC	Refused HBSO, Radiation	--
Areia [22]	48/<30	No risks	NAEH Progestin- 10 GnRH-a-4 mo	Coagulation Benign	30 months. Pain, no bleeding	No attempt	HBSO-EC	IB, G1
Le Marrec [24]	48/--	Breast cancer	Proliferative	TBEA Proliferative	6 years. PMB	D&C-EC	HBSO nodes	IA, G2
AlHilli [25]	47/36	Hypertension	Secretory	Bipolar RF	5 years. PMB	Hysteroscopy Biopsy-EC	VHBSO	IA, G1
Wortman [26]	40/24	No risks	Secretory	Bipolar RF	2 years. Bleeding, pain	Hematometra TCRE-EC	HBSO No residual EC	IA, G1
	41/--	--	Proliferative	Bipolar RF Proliferative	7 years. Bleeding, pain	Biopsy failed	TAH-EC Endometriosis	IA, G1
	33/--	--	--	Coagulation	17 years. PMB, pain	TCRE-EC	HBSO, nodes No residual EC	!, G3
Wortman [31]	45/24	--	--	TBEA Proliferative	10 years. PMB	Biopsy-EC	HBSO	I G1
	42/--	Diabetes sleep apnea	Proliferative 1y before EA	TBEA	8 years. Bleeding, pain	TCRE-EC	HBSO	IA, G1
	49/--	--	Secretory	Bipolar RF	7 years. PMB, pain	Hysteroscopy, Biopsy-EC	HBSO nodes	IA, G1
Kalampokas [42]	48/37	--	Benign	EA/TCRE	18 years. PMB	Biopsy-EC	HBSO	IA
	39/28	--	benign	EA/TCRE	18 years. PMB	Biopsy-EC	HBSO	IA
Lopez [23] abstract	50/>30	Hypertension	Proliferative GnRH-a	TCRE Proliferative	2 years, PMB	Hysteroscopy biopsy-EC	HBSO	IA, G1

MacMahon [27] abstract	48/34	--	--	TBEA	8 years, PMB	TCRE- EC	HBSO	IA
Tsafirir [29] abstract	37/<25	No risks	Benign	TCRE	6 years, Bleeding	Biopsy-EC	HBSO	IB

Presence of EC during EA: Table 2 includes 12 women who already had EC present during EA [9,17,28,30]. Of these, 7 had benign, two proliferative and one each secretory, simple hyperplasia (nonatypical endometrial hyperplasia, NAEH) and unknown pre-EA endometrial pathology. The time from pre-EA biopsy to EA was within 3 months in 6, unknown in 4, and 11 and 17 months in 2 cases. In two cases, hysterectomy and bilateral salpingo-oophorectomy (HBSO) was performed at 34- and 60-months after EC diagnosis and the corresponding stage was IA, G1 and IA, G2, respectively. No residual EC was found in 9 (75%) cases.

Likely presence of EC during EA: In three additional cases, EC was likely present during EA (Table 3) [12,15,20]. In one woman having EA for PMB, pre-EA biopsy was adenomatous endometrial hyperplasia (NAEH) [15] while the other two had complex endometrial hyperplasia (NAEH) [12,20]; the latter upgraded to atypical endometrial hyperplasia (AEH) during EA one month later [20]. All 3 cases had persistent bleeding and stage 1A, G1 EC was diagnosed with endometrial biopsy and hysterectomy within 6 months.

Presence of atypical endometrial hyperplasia (AEH) during EA: There were 3 cases of AEH identified during EA with pre-EA biopsy showing insufficient, adenomatous and complex hyperplasia (Table 4) [11,14,16]. In two women, EA was performed for PMB. The BMI was >40 in two and unknown in one. The patient with complex hyperplasia was given Danazol 800 mg daily for 4 weeks and the TCRE specimen was reported as benign. However, the original pre-EA biopsy was re-examined and upgraded to AEH following hysterectomy for heavy PMB 3-years post-EA showing EC (IB, G2) [16]. In one patient no treatment was offered after the TCRE showed AEH. At 1-year post-EA, she had spotting and Pap smear identified EC cells and EC (IA, G1) was confirmed following hysterectomy [11]. A 63-year-old with BMI >40 refused hysterectomy after AEH, presented with umbilical metastasis of EC (stage IV) 14 months later, and died 4 months post hysterectomy [14].

PAEC in women having EA for PMB: Nine PAEC were diagnosed in women having TCRE (N=7), one REA and one having D&C only for PMB (Table 5) [13,21,29,31]. Eight women had risk factors for EC with two taking combined HT and one progesterone only. Pre-EA biopsy was benign in 7 and nonatypical endometrial hyperplasia (NAEH) in 2 women. During EA, pathology was benign in four, unknown in three and NAEH in two women. At a median of 6 years (range 3-10), seven (77.8%) women presented with bleeding, one with pain without bleeding and one with urinary stress incontinence (USI). Hysteroscopy and D&C/biopsy diagnosed EC in six (75%) and was unsuccessful and not attempted in the other two women. Two PAEC were found after hysterectomy; one for USI and one for pain without bleeding. Seven PAEC were stage IA-IC and two stage III.

3.3. PAEC in Women with Appropriate Indications for EA

Excluding 27 cases (Tables 2–5) and the 43 additional incident cases because of insufficient patient information, there remain only 16 cases with pertinent information on patient characteristics, circumstances, and appropriate indications for EA (Table 6) [10,18,22–27,29,31,42].

Patient characteristics and demographics: The median age and (range) at the time of EA were 47.5 years (33-51). The BMI was not reported in 5 and it was <30 in 5, ≥30 - <39 in 5, ≥40 in 1 woman. Comorbidities were not provided in 6 women while at least one of hypertension, diabetes, colon and breast cancer were reported in 7. No risks were reported in 3 women. Pre-EA endometrial biopsy was reported as benign in 3, proliferative in 5 (1 taken 1year prior to EA), secretory in 3, NAEH in 2 (1

adenomatous, 1 simple) and not provided in 3 women. The method of EA was reported as coagulation or TCRE in 8, bipolar radiofrequency in 4 and TBEA in 4 women. Endometrial pathology during EA was not reported in 11 cases. It was proliferative in 4 and benign in 1 woman.

Presentation of PAEC: Presentation of PAEC was vaginal bleeding in 15 (93.8%); 5 patients had pain associated with bleeding and 10 were postmenopausal. The woman with no post-EA bleeding received hysterectomy for pain 30 months post-EA and EC (IB, G1) was found. The median time to presentation was 6.5 years, range 1 to 18.

Diagnosis of PAEC: Diagnosis of PAEC was achieved in 14 of 15 (93.3%) attempted procedures using endometrial biopsy in 5, D&C in 1, hysteroscopy and biopsy in 4 and TCRE in 4 women. PAEC was diagnosed after hysterectomy in 2 women; one after failed biopsy and the other having hysterectomy for intense pain without bleeding. PAEC was reported as stage I in 13 (81%), II/III in 2 (1.3%), unknown in 1 woman.

4. Discussion

Incidence of PAEC: In our review, 3 studies found no statistical difference in risk reduction of EC after EA [33,39,41] likely due to inadequate power. Statistically significant reduction was reported by 3 adequately powered studies [4,42,43] with an overall summary incidence 0.11% (Table1). Comparing women having EA with women having LNG-IUS to treat AUB, Dood et al reported that there was a notable but not statistical difference favoring LNG-IUS treatment versus EA based on EC rates of 3/4,776 (0.06%) and 1/3,558 (0.03%) women in the EA and LNG-IUS groups, respectively [39]. However, in secondary analysis the LNG-IUS had lower rate of EC than all other treatments (LNG-IUS HR, 0.12; CI, 0.02–0.83; p = .03) [39]. Dood’s study provides additional support that EA reduces significantly the risk of EC since it is at least equivalent to the risk reduction provided by the LNG-IUS which has been shown to be the most effective prevention measure for EC [47,48]. Large-scale observational studies describe up to a 78% reduction in EC risk among LNG-IUS users, particularly if used long-term [47,48]. Furthermore, in women with a BMI ≥40 kg/m², modelling suggests that the LNG-IUS could be a cost-effective approach for primary prevention of EC in high-risk women [49,50]. Applying the same analogy, the marked reduction in incidence of EC after EA indicates that EA might be considered an appropriate preemptive minimally invasive approach to reduce the risk of EC in high-risk women with or without AUB. However, it must be pointed out that since the follow up in women in our review is variable and limited [median 8.5 years (1.9-25)] and the age of PAEC diagnosis was 54 (10 years earlier than the median age of EC in the general population), the incidence of PAEC likely will increase in time as women reach their end of life.

In the last 30 years, the incidence of EC worldwide has increased by 132% and is set to continue to rise in response to an ageing population and increasing global rates of obesity and diabetes-both being risk factors for EC [51]. The increased prevalence of obesity in women and its association with increased risk of EC [7,8] together with the widespread utilization of EA to treat AUB in these women raises the possibility of a higher incidence of EC in obese women undergoing EA than in the general population. Regardless, we found that in the short-term EA has a protective effect since the summary incidence of EC from all studies was approximately 1 per 1,000 women, compared to a life-time risk of 2 to 3% in the general population. The reduction in risk is likely due to dual effect of EA reducing endometrial volume and also destroying/eliminating occult pre- or malignant EC cells. The latter mechanism is supported by the fact that no residual cancer was found in hysterectomy specimens in several cases in the present review (Table 2, 9 of 12; Table 6, 2 of 16 cases) and other prior publications [3–5]. Based on similar experience, we have reported that hysteroscopic EA can be an effective definitive treatment in selected women with AUB and AEH and even EC in women who refuse or are at high-risk for hysterectomy and are compliant with regular long-term follow-up [3].

In our review, the method of EA was not always provided to determine if one method is more effective in reducing EC. Flöter Rådestad et al reported that there was a significant reduction of EC after REA, suggesting a protective effect, whereas TCRE showed an incidence within the expected rate [43]. Dood et al did not find any difference in EC incidence when comparing first-and second-generation EA methods with medical management [39]. This discrepancy in outcomes after different methods of EA likely reflects variations in patient characteristics and selection, surgical technique and/or method used and surgeon's experience and expertise.

Our review also underscores the importance of endometrial sampling during EA in cases of non resectoscopic EA methods since at least 12 cases of EC were identified by additional sampling during EA and treated timely; all with benign pre-EA endometrial biopsy (**Table 2**). Interestingly, although hysterectomy was delayed 34 and 60 months in two women, the stage of EC was IA, G1-2 in both cases. To minimize the risk of missing significant intrauterine pathology in women undergoing EA, we advocate and perform total TCRE in all women with PMB, in the absence of benign (within 6 months) endometrial biopsy, in the presence of risk factors (including BMI >30) for endometrial neoplasia, and in the presence of any intrauterine lesions. For patients in whom REA or nonhysteroscopic EA is performed, we advocate and perform endometrial curettage and hysteroscopy immediately prior to EA for additional sampling, evaluate the endometrial cavity and to thin the endometrium. As a result, we have identified several cases of incidental miscellaneous uterine malignancies including undiagnosed EC in 5,750 women undergoing primary resectoscopic EA with benign pre-ablation endometrial biopsy performed by the senior author (G.A.V) from 1990 through December 2017 [52]. The value of additional sampling during EA is also underscored in **Table 3** (EC likely present during EA) indicating that even in the presence of pre-EA biopsy showing endometrial hyperplasia, no intra-EA biopsy was done in two women. In the third case the Pre-EA biopsy was upgraded from complex to AEH after EC diagnosis, which is a pre- or coexisting lesion with EC.

Presentation of PAEC: The presentation of PAEC in 15 women (**Table 3-5**) and in the additional 16 cases with appropriate indications for EA (**Table 6**) was vaginal bleeding in 13 (86.7%) and 15(93.8%), respectively. This is similar to the findings of a systematic review and meta-analysis reporting that in women with PMB, the pooled prevalence of EC was 91% (95% CI, 87-93), irrespective of tumor stage [53].

Diagnosis of PAEC: Diagnosis of EC was achieved by hysteroscopy and D&C/biopsy in 8 of 9 (88.9%) attempted procedures in the 15 cases with inappropriate indications and in 14/15 (93.3%) attempted procedures in the 16 cases with appropriate indications (**Table 6**). This is similar to that reported in the general population where biopsy and hysteroscopy have shown high effectiveness and similar rates in diagnosing EC [54,55].

Age at diagnosis of PAEC: The median age of diagnosis of EC in the general population is 64 years. In the 16 women with appropriate indications for EA, the median age and (range) of diagnosis of PAEC was 54 (42-66) years. This is calculated from the median age of 47.5 years at EA and the median time to presentation of 6.5 years. Flöter Rådestad et al reported median times to EC of 3.0 and 8.3 years after TCRE and EA, respectively [43] while Dood et al concluded that there was no delay in diagnosis of EC when comparing EA versus medical management of AUB [39].

Stage of PAEC at diagnosis: In our review, the stage of PAEC in women with appropriate indications was reported as stage I in 13 (81%), II/III in 2 (1.3%), unknown in 1 woman (**Table 6**). Interestingly, although the time to presentation in 9 women with PMB treated with EA, was 3 to 10 years, the stage of EC was I in 7 (77.8%) and III in 2 (2.2%) women (**Table 5**); similar to premenopausal women (**Table 6**). These are similar to those found in Tables 2-4 (Stage I, 73.3%) and those reported in the general population reported as stage I (65-75%); II (5-10%); II (10-15%), and IV (5-10%) [56].

Our review provides additional cases of PAEC, one more registry and more robust evidence supporting finding of a recent review by Oderkerk et al reporting on 11 studies involving 38 EC among 29,102 patients with a history of EA. The authors found that the incidence of endometrial cancer ranged from 0.0% to 1.6%. Among these cases, vaginal bleeding was the presenting symptom in 71% of patients, endometrial sampling was successful in 89% of described cases and 90% of the pathology showed early-stage endometrioid adenocarcinoma (FIGO Stage I). The authors concluded that previous EA is not associated with the development of EC, diagnostic work-up is not impeded by previous EA and EC after endometrial ablation are not detected at an advanced stage [46].

5. Conclusions

Based on the available evidence, we conclude that EA decreases significantly the risk of EC in the short-term, likely due to quantitative reduction in endometrial volume and eliminating occult pre- or malignant endometrium which is vulnerable to EA. However, the incidence of PAEC might increase in time as women with EA reach their end of life. The mode and time to presentation, diagnostic work-up, including endometrial biopsy and hysteroscopy and stage of PAEC are not altered by EA. Endometrial sampling during EA identifies additional cases with pre- or malignant endometrium and we advocate that all patients undergoing any method of EA have intra-operative endometrial sampling regardless of pre-EA pathology.

6. Future Directions

We propose that EA be evaluated further for long-term effects on PAEC and be considered as a pre-emptive therapy in women at high risk for EC with and without AUB.

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References

1. DeCherney, A.H.; Diamond, M.P.; Lavy, G.; Polan, M.L. Endometrial ablation for intractable uterine bleeding: hysteroscopic resection. *Obstet Gynecol.* 1987;70:668–670.
2. Valle, R.F.; Baggish, M.S. Endometrial carcinoma after endometrial ablation: high-risk factors predicting its occurrence. *Am J Obstet Gynecol.* 1998;179:569–572.
3. Edris, F.; Vilos, G.A.; Al-Mubarak, A.; Ettler, H.C.; Hollett-Caines, J.; Abu-Rafea, B. Resectoscopic surgery may be an alternative to hysterectomy in high-risk women with atypical endometrial hyperplasia. *J Minim Invasive Gynecol.* 2007 Jan-Feb;14(1):68-73.
4. Singh, M.; Hosni, M.M.; Jones, S.E. Is endometrial ablation protective against endometrial cancer? A retrospective observational study. *Arch Gynecol Obstet* 2016;293:1033–1037.
5. Dion, L.; Agostini, A.; Golfier, F.; et al. In which cases should endometrial destruction be performed during an operative hysteroscopy? Clinical practice guidelines from the French College of Gynaecologists and Obstetricians (CNGOF). *J Gynecol Obstet Hum Reprod* 2021;50:102188.
6. GBD 2021 Adult BMI Collaborators. Global, regional, and national prevalence of adult overweight and obesity, 1990–2021, with forecasts to 2050: a forecasting study for the Global Burden of Disease Study 2021. *Lancet.* 2025 Mar 8;405(10481):813-838.

7. Wise, M.R.; Jordan, V.; Lagas, A.; Showell, M.; Wong, N.; Lensen, S.; Farquhar, C.M. Obesity and endometrial hyperplasia and cancer in premenopausal women: A systematic review. *Am J Obstet Gynecol.* 2016 Jun;214(6):689.e1-689.e17.
8. Hazelwood, E.; Sanderson, E.; Tan, V.Y.; et al. Identifying molecular mediators of the relationship between body mass index and endometrial cancer risk: a Mendelian randomization analysis. *BMC Med.* 2022 Apr 19;20(1):125.
9. Dwyer, N.A.; Stirrat, G.M. Early endometrial carcinoma: 16. An incidental finding after endometrial ablation for dysfunctional uterine bleeding. *Br J Obstet Gynaecol* 1991;98:733-734.
10. Copperman, A.B.; DeCberney, A.H.; Olive, D.L. A case of endometrial cancer after endometrial ablation for dysfunctional uterine bleeding. *Obstet Gynecol* 1993;183:640-642.
11. du Toit, G.C.; van der Merwe, J.P.; Kruger, T.F. Endometrial carcinoma after transcervical resection of the endometrium. *S Afr Med J Suid-Afrikaanse tydskrif vir geneeskunde.* 1994;84(6):359–60.
12. Ramey, J.W.; Koonings, P.P.; Given, F.T.; et al. The process of carcinogenesis for endometrial adenocarcinoma could be short: Development of a malignancy after endometrial ablation. *Am J Obstet Gynecol* 1994;170:1370-1371.
13. Margolis, M.T.; Thoen, L.D.; Boike, G.M.; et al. Asymptomatic endometrial carcinoma after endometrial ablation. *Int J Gynaecol Obstet* 1995;51:255–258.
14. Horowitz, I.R.; Copas, P.R.; Aaronoff, M.; et al. Endometrial adenocarcinoma following endometrial ablation for postmenopausal bleeding: A case report. *Gynecol Oncol* 1995;56:460-463.
15. Baggish, M.S.; Ringgenberg, E.; Sze, E.H.M. Adenocarcinoma of the corpus uteri after endometrial ablation. *J Gynecol Surg* 1995;11:91-94.
16. Iqbal, P.K.; Paterson, M.E. Endometrial carcinoma after endometrial resection for menorrhagia. *Br J Obstet Gynaecol.* 1997;104(9):1097–1098.
17. Klein, Z.; Markovitch, O.; Altaras, M.; Beyth, Y.; Fishman, A. Advanced endometrial adenocarcinoma following endometrial ablation: a case report and review of the literature. *Int J Gynecol Cancer.* 1997;7(2):163–165.
18. Brooks-Carter, G.N.; Killackey, M.A.; Neuwirth, R.S. Adenocarcinoma of the endometrium after endometrial ablation. *Obstet Gynecol* 2000;96:836–837.
19. Steed, H.L.; Scott, J.Z. Adenocarcinoma diagnosed at endometrial ablation. *Obstet Gynecol.* 2001;97(5 Pt 2):837-9.
20. Lee, J.K-S.; Gupta, J.; McCarthy, K.; Gornall, R.J. Endometrial adenocarcinoma following thermachoice balloon endometrial ablation. *J Obstet Gynaecol J Inst Obstet Gynaecol.* 2005;25(8):825–826.
21. Sagiv, R.; Ben-Shem, E.; Condrea, A.; Glezerman, M.; Golan, A. Endometrial carcinoma after endometrial resection for dysfunctional uterine bleeding. *Obstet Gynecol.* 2005;106(5 Pt 2):1174–1176.
22. Areia, A.; Branco, M.; Frutuoso, C.; et al. Endometrial adenocarcinoma after endometrial ablation. A case report. *Eur J Gynaecol Oncol* 2006;27:432–433.
23. Jimenez Lopez, J.S.; Bravo, V. Endometria after endometrial ablation: a case report. *Journal of minimally invasive gynecology*, 2007;14:S120. Abstract
24. Le Marrec, A.; Lavoue, V.; Morcel, K.; et al. Occurrence of endometrial cancer six years after treatment with thermal balloon ablation (Thermachoice): first case report. *Eur J Obstet Gynecol Reprod Biol* 2010;150:219–220.
25. AlHilli, M.M.; Hopkins, M.R.; Famuyide, A.O. Endometrial cancer after endometrial ablation: systematic review of the medical literature. *J Minim Invasive Gynecol.* 2011;18:394–400.
26. Wortman, M.; Dawkins, J.C. Post-ablation endometrial carcinoma (PAEC) following radiofrequency endometrial ablation: a case report and its implications for management of endometrial ablation failures. *Surg Technol Int.* 2016;24:161–166.
27. MacMahon, C.; Hatti, A.; Bakour, S.; Ewies, A.A.A. Challenges of endometrial assessment after ablation in women with postmenopausal bleeding - A case series. *J Obstet Gynaecol.* 2018;38(3):432-434.
28. Smith, S.E.; Baecher-Lin, L. Endometrial Cancer Diagnosed at the Time of Novasure Endometrial Ablation: A Case Series Report. *Journal of minimally invasive gynecology*, 2011;18(6):S99-S100. Abstract

29. Tsafirir, Z.; Schiff, L.; Sangha, R.; et al. Endometrial cancer following endometrial ablation. *J min Invas Gynecol* 2014;21:S148
30. Argall, E.; Jovanovic, A.; Figueroa, R.; Mayer, A.; Nelson, B.; Roland, P. Effects of endometrial ablation on treatment planning in women with endometrial cancer. *J Minim Invasive Gynecol.* 2016;23:281–285.
31. Wortman, M.; Vilos, G.A.; Vilos, A.G.; Abu-Rafea, B.; Dwyer, W.; Spitz, R. Postablation endometrial carcinoma. *JSLs.* 2017 Apr-Jun; 21(2): e2017.00011.
32. Panoskaltis, T.A.; Bassett, P.A.; Frappell, J.M. Transcervical endometrial resection in 197 women. hysterectomy rate and associated risk factors after a 5-8-year follow-up period. *Gynaecological Endoscopy* 2002;11:99–106.
33. Neuwirth, R.S.; Loffer, F.D.; Trenhaile, T.; et al. The incidence of endometrial cancer after endometrial ablation in a low-risk population. *J Am Assoc Gynecol Laparosc.* 2004;11:492–494.
34. Cooper, K.G.; Bain, C.; Lawrie, L.; et al. A randomised comparison of microwave endometrial ablation with transcervical resection of the endometrium; follow up at a minimum of five years. *BJOG* 2005;112:470–5.
35. Gaia, G.; Botchorishvili, R.; Canis, M.; et al. Endometrial cancer following endometrial resection. *Gynecol Surg.* 2007;4:179–185.
36. Krogh, R.A.; Lauszus, F.F.; Guttorm, E.; Rasmussen, K. Surgery and cancer after endometrial resection. Long-term follow-up on menstrual bleeding and hormone treatment by questionnaire and registry. *Arch Gynecol Obstet.* 2009 Dec;280(6):911-916.
37. Grochmal, S.A.; Sherry, S.J.; Erian, J.; Chandakas, S. Long-Term Results from Endometrial Ablation/Resection with Nd:YAG Laser and Electrosurgery: A Twenty Year Follow-Up. *Journal of minimally invasive gynecology*, 2010-11, Vol.17 (6), p.S105-S105
38. Cooper, K.; Lee, A.; Chien, P.; Raja, E.; Timmaraju, V.; Bhattacharya, S. Outcomes following hysterectomy or endometrial ablation for heavy menstrual bleeding: retrospective analysis of hospital episode statistics in Scotland. *BJOG* 2011;118:1171–1179.
39. Dood, R.L.; Gracia, C.R.; Sammel, M.D.; Haynes, K.; Senapati, S.; Strom, B.L. Endometrial cancer after endometrial ablation vs. medical management of abnormal uterine bleeding. *J Minim Invasive Gynecol.* 2014 Sep-Oct;21(5):744-52.
40. Morelli, M.; Rocca, M.L.; Mociaro, R.; et al. Sonographic findings in postmenopausal women with a prior endometrial ablation: interpretation and management of women with endometrial thickening and bleeding. *J Minim Invasive Gynecol.* 2015 Mar-Apr;22(3):489-494.
41. Soini, T.; Rantanen, M.; Paavonen, J.; et al. Long-term Follow-up After Endometrial Ablation in Finland: Cancer Risks and Later Hysterectomies. *Obstet Gynecol* 2017;130:554–60.
42. Kalampokas, E.; McRobbie, S.; Payne, F.; Parkin, D.E. Endometrial cancer after endometrial ablation or resection for menorrhagia. *Int J Gynaecol Obstet.* 2018 Jul;142(1):84-90.
43. Flöter Rådestad, A.; Dahm-Kähler, P.; Holmberg, E.; et al. Long-term incidence of endometrial cancer after endometrial resection and ablation: A population based Swedish gynecologic cancer group (SweGCG) study. *Acta Obstet Gynecol Scand.* 2022 Aug;101(8):923-930.
44. Gimplelson, R.J. Not so benign endometrial hyperplasia: endometrial cancer after endometrial ablation. *J Am Assoc Gynecol Laparosc.* 1997;4:507–511.
45. Gkrozou, F.; Antoun, L.; Tsonis, O.; Daniilidis, A.; Paschopoulos, M. Endometrial Cancer Following Endometrial Ablation: An Overview of Published Literature. *Indian Journal of Gynecologic Oncology.* 2022 Jun;20(2):26-32.
46. Oderkerk, T.J.; van de Kar, M.R.D.; Cornel, K.M.C.; et al. Endometrial cancer after endometrial ablation: a systematic review *International Journal of Gynecologic Cancer* 2022;32:1555-1560.
47. Soini, T.; Hurskainen, R.; Grénman, S.; Mäenpää, J.; Paavonen, J.; Pukkala, E. Cancer risk in women using the levonorgestrel releasing intrauterine system in Finland. *Obstet Gynecol* 2014;124:292–299.
48. Jareid, M.; Thalabard, J.C.; Aarflot, M.; Bøvelstad, H.M.; Lund, E.; Braaten, T. Levonorgestrel-releasing intrauterine system use is associated with a decreased risk of ovarian and endometrial cancer, without increased risk of breast cancer. Results from the NOWAC Study. *Gynecol Oncol.* 2018 Apr;149(1):127-132.

49. Derbyshire, A.E.; Allen, J.L.; Gittins, M.; Lakhiani, B.; Bolton, J.; Shaw, J.; et al. PROgesterone Therapy for Endometrial Cancer Prevention in Obese Women (PROTEC) Trial: A Feasibility Study. *Cancer Prev Res (Phila)*. 2021 Feb;14(2):263-274.
50. Bernard, L.; Kwon, J.S.; Simpson, A.N.; Ferguson, S.E.; Sinasac, S.; Pina, A.; Reade, C.J. The levonorgestrel intrauterine system for prevention of endometrial cancer in women with obesity: A cost-effectiveness study. *Gynecol Oncol*. 2021 May;161(2):367-373.
51. Baker-Rand, H.; Kitson, S.J. Recent Advances in Endometrial Cancer Prevention, Early Diagnosis and Treatment. *Cancers (Basel)*. 2024 Mar 1;16(5):1028.
52. Vilos, G.A.; Edris, F.; Abu-Rafea, B.; Hollett-Caines, J.; Ettler, H.C.; Al-Mubarak, A. Miscellaneous uterine malignant neoplasms detected during hysteroscopic surgery. *J Minim Invasive Gynecol*. 2009;16:318–325.
53. Clarke, M.A.; Long, B.J.; Del Mar Morillo, A.; Arbyn, M.; Bakkum-Gamez, J.N.; Wentzensen, N. Association of Endometrial Cancer Risk With Postmenopausal Bleeding in Women: A Systematic Review and Meta-analysis. *JAMA Intern Med*. 2018 Sep 1;178(9):1210-1222.
54. Sakna, N.A.; Elgendi, M.; Salama, M.H.; Zeinhom, A.; Labib, S.; Nabhan, A.F. Diagnostic accuracy of endometrial sampling tests for detecting endometrial cancer: a systematic review and meta-analysis. *BMJ Open*. 2023 Jun 23;13(6):e072124.
55. Clark, C.; Cazzolla, A.; Colonna, G.; Loizzi, V.; Cormio, G.; Lopez, S. Hysteroscopy vs. Vabra in Endometrial Cancer Diagnosis: A Systematic Review of the Literature. *Cancers (Basel)*. 2025 Mar 28;17(7):1145.
56. National Cancer Institute. Cancer Stats Facts: Uterine Cancer (Corpus Uteri) [Internet]. Surveillance, Epidemiology, and End Results Program. Available from: <https://seer.cancer.gov/statfacts/html/corp.html>

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