Case Report

Endometrial Cancer After Endometrial Ablation: Systematic Review of Medical Literature

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ABSTRACT

Data are limited regarding the occurrence of endometrial cancer after endometrial ablation (EA). A systematic review of the English-language medical literature was performed of cases of endometrial cancer after EA. This review included the present case report involving a 47-year-old woman with a diagnosis of stage IA, grade 1 endometrial adenocarcinoma 5 years after radiofrequency EA. The systematic literature review identified 22 endometrial cancer cases occurring after EA. Most (76.5%) were stage I at diagnosis. Time to endometrial cancer diagnosis ranged from 2 weeks to 10 years following EA. All but 3 cases involved patients with known endometrial cancer risk factors. To our knowledge, the present case is the first reported occurrence of endometrial cancer after radiofrequency EA. Endometrial cancer has been detected after EA at variable intervals. Occurrence of endometrial cancer after EA is low, yet it continues to be difficult to quantify through retrospective analyses.

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Endometrial carcinoma is the most common gynecologic malignant lesion in the United States, with an incidence in the general population of 2% to 3% and a prevalence worldwide of 1 per 1000 [1,2]. Postmenopausal bleeding is the primary initial symptom, and greater than 90% of patients exhibit some form of abnormal uterine bleeding [1]. Furthermore, approximately 50% of endometrial cancers occur in women with associated risk factors including unopposed estrogen stimulation, obesity, diabetes mellitus, and hypertension [2]. Women with chronic anovulation are at increased risk of endometrial cancer after endometrial ablation (EA) [3]. Premenopausal patients with menorrhagia or abnormal uterine bleeding must be thoroughly evaluated, and a diagnosis of endometrial cancer must be ruled out before they undergo EA [3].

Heavy menstrual blood loss is an important health concern that affects 10% to 35% of women of reproductive age [4,5], and accounts for approximately 12% of referrals to outpatient gynecology clinics [5]. A shift toward minimally invasive treatment of menorrhagia has occurred in the last decade; currently, EA represents approximately 60% of procedures performed to treat menorrhagia [6]. How likely that endometrial cancer will occur after EA has remained an unanswered question since adoption of this treatment in the 1980s. Because EA is a relatively new technology, data on the incidence of endometrial cancer after EA are insufficient to draw important conclusions. Most cases reported in the medical literature were detected after first-generation EA techniques such as endometrial resection that relied on operative hysteroscopic skills [7]. Since the late 1990s, global EA with second-generation devices has largely replaced first-generation hysteroscopic EA procedures [8]. Second-generation EA technologies include thermal balloon ablation (TheraChoice; Ethicon, Inc., Somerville, NJ), microwave ablation (Microsulis; Microsulis Medical Ltd., Denmead Hampshire, Hampshire, England), cryoablation (Her Option; CooperSurgical, Inc., Trumbull, CT), ablation with free-circulating fluid (hydrothermablation), and radiofrequency ablation (NovaSure; Hologic, Inc., Bedford, MA) [8,9]. Their benefit resides in ease of use, equivalent treatment outcomes compared with first-generation techniques, and fewer adverse effects [9].
A reasonable assumption is that through total destruction of the endometrium, the incidence of endometrial cancer after EA may be reduced. Conversely, there is concern that the diagnosis of endometrial cancer may be delayed because of the consequent intrauterine scarring and distortion of the uterine cavity after EA [10]. This scarring and distortion could result in cancer being diagnosed at an advanced stage. Evidence suggests that the incidence of endometrial cancer after EA is unchanged in patients who undergo EA compared with patients who do not [7]. In a large retrospective cohort study, Neuwirth et al [7] demonstrated that the expected incidence of endometrial cancer in the general population was similar to the actual incidence of endometrial cancer after EA. However, their study used first-generation EA techniques. In addition, the patient population investigated was described as at low risk without taking into account predisposing risk factors for endometrial cancer such as obesity, chronic anovulation, and diabetes.

With the transition to second-generation EA over the last decade, the occurrence of endometrial cancer after EA must be reevaluated. The objective of this study was to review and summarize the clinical manifestation of all endometrial cancers after EA described in the literature to date. Also reported is the first known case of endometrial cancer after radiofrequency EA at our institution and, to our knowledge, the first reported case in the literature of endometrial cancer in relation to radiofrequency EA.

**Case Report**

A 47-year-old woman, gravida 2, para 2, with previous vaginal deliveries and a history remarkable for hypertension and obesity (body mass index [BMI], 36) was evaluated at the Abnormal Uterine Bleeding Clinic in December 2009. The patient had undergone radiofrequency EA to treat menorrhagia in 2004, and had experienced amenorrhea for 5 years. An endometrial biopsy specimen obtained before

![Fig. 1](image1)

Hysterectomy specimen showing zone of cancer in the lower uterine segment and obliteration of the fundus by uterine synechiae due to radiofrequency endometrial ablation.

![Fig. 2](image2)

Flow chart of systematic review analysis. EA = endometrial ablation; EC = endometrial cancer.
EA demonstrated secretory endometrium. The uterine cavity was 4 cm long at ablation, and a global treatment effect was evident at postablation hysteroscopy. The patient had a 6-month history of painless vaginal bleeding that varied from spotting consisting of a brownish yellow discharge to passage of dime-sized blood clots.

An initial evaluation of the patient’s abnormal uterine bleeding was undertaken by office hysteroscopy. A 3-mm hysteroscope was used, but it could be advanced only to 4 to 5 cm, demonstrating a contracted uterine cavity consistent with the history of EA. When the cavity was gently probed with the hysteroscope, a small cavity was created, and a copious amount of tissue was visible. An endometrial biopsy catheter inserted to 4.5 cm obtained a profuse amount of tissue. Histopathologic evaluation demonstrated a FIGO (International Federation of Gynecology and Obstetrics) grade 1 endometrioid endometrial cancer arising in a background of atypical complex endometrial hyperplasia. Transvaginal ultrasonography revealed a 3-mm endometrial stripe and a heterogeneous echotexture of the myometrium.

The patient subsequently underwent vaginal hysterectomy and bilateral salpingo-oophorectomy. The final pathology report was of a FIGO stage IA, grade 1 endometrioid endometrial carcinoma arising in a background of complex endometrial hyperplasia. Transvaginal ultrasonography revealed a mass in the lower uterine segment that measured $3.6 \times 1.7 \times 1.1$ cm. The mass was superficial and confined to the endometrium without evidence of myometrial invasion. The uterine cavity was obliterated completely, consistent with postablation uterine synechiae; this area was free of cancer and inaccessible histologically. The cancer zone was located exclusively in the lower portion of the uterine cavity, where regenerating endometrium was present (Fig. 1). Postoperative recovery was unremarkable.

### Materials and Methods

A systematic search of MEDLINE, EMBASE, the Cochrane Library, WoS, and SCOPUS from database inception to February 2010 was performed. All publications referring to endometrial ablation and endometrial cancer were searched. Two reviewers (M.M.A. and M.R.H.) independently reviewed titles and identified abstracts. Exclusion criteria included a diagnosis of endometrial cancer made before or at EA and non–English-language publications. Agreement in study selection was evaluated using $\kappa$ statistics. The primary objective of the analysis was to identify all endometrial cancer after EA cases described in the literature.

### Results

The primary search identified 234 abstracts. Of these, 205 reports were excluded on review of titles and abstracts (Fig. 2). Of the other 29 reports, 3 were in a language other than English and 1 was not retrievable. On final review, 8 of 29 reports did not fulfill the study inclusion criteria. Mean (SD) $\kappa$ agreement was 0.94 (0.04).
## Table 2

Clinical manifestation of endometrial carcinoma after endometrial ablation in case reports

<table>
<thead>
<tr>
<th>Source, year</th>
<th>Age at cancer diagnosis, yr</th>
<th>BMI at cancer diagnosis</th>
<th>Risk factors for endometrial cancer</th>
<th>Preablation disease</th>
<th>Postablation symptoms</th>
<th>Evaluation before hysterectomy</th>
<th>Pathologic findings after hysterectomy</th>
<th>Time from ablation to cancer diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>AlHilli et al [12], 2007</td>
<td>47</td>
<td>36</td>
<td>Obesity, hypertension</td>
<td>Secretory endometrium</td>
<td>Irregular vaginal bleeding</td>
<td>Hysteroscopy, biopsy, US</td>
<td>Stage IA, grade 1 endometrial adenocarcinoma</td>
<td>5 yr</td>
</tr>
<tr>
<td>Gaia et al [12], 2007</td>
<td>73</td>
<td>21</td>
<td>Hypertension, postmenopausal status, HRT</td>
<td>None</td>
<td>Irregular vaginal bleeding</td>
<td>Hysteroscopy, D&amp;C</td>
<td>Stage IB, grade 2–3 mixed clear-cell/endometrioid carcinoma</td>
<td>10 yr</td>
</tr>
<tr>
<td>68</td>
<td>33</td>
<td>Hypertension, obesity, postmenopausal status</td>
<td>None</td>
<td>Irregular vaginal bleeding</td>
<td>MRI, hysteroscopy, D&amp;C</td>
<td>Stage IC, grade 1 endometrioid adenocarcinoma</td>
<td>8 yr</td>
<td></td>
</tr>
<tr>
<td>58</td>
<td>39</td>
<td>Hypertension, obesity, postmenopausal status</td>
<td>None</td>
<td>Irregular vaginal bleeding</td>
<td>Hysteroscopy</td>
<td>Stage IA, grade 1 adenocarcinoma</td>
<td>5 yr</td>
<td></td>
</tr>
<tr>
<td>62</td>
<td>34</td>
<td>Obesity, HRT</td>
<td>None</td>
<td>Irregular vaginal bleeding</td>
<td>US, endometrial biopsy</td>
<td>Stage IB, grade 1 endometrioid adenocarcinoma</td>
<td>6 yr</td>
<td></td>
</tr>
<tr>
<td>Areia et al [13], 2006</td>
<td>52</td>
<td>NA</td>
<td>None</td>
<td>Simple hyperplasia → atrophic endometrium</td>
<td>Pelvic pain</td>
<td>None</td>
<td>Stage IA, grade 1 endometrioid adenocarcinoma; complex atypical hyperplasia</td>
<td>33 mo</td>
</tr>
<tr>
<td>Sagiv et al [14], 2005</td>
<td>60</td>
<td>NA</td>
<td>None</td>
<td>Proliferative endometrium</td>
<td>Pain, irregular bleeding</td>
<td>US; endometrial biopsy not successful because of cervical stenosis</td>
<td>Stage IB, grade 1 endometrioid adenocarcinoma</td>
<td>3 yr</td>
</tr>
<tr>
<td>Lee et al [15], 2005</td>
<td>36</td>
<td>36</td>
<td>Obesity, polycystic ovary syndrome</td>
<td>Complex atypical endometrial hyperplasia</td>
<td>None</td>
<td>None</td>
<td>Stage IA, grade 1 endometrioid endometrial carcinoma</td>
<td>2 mo</td>
</tr>
<tr>
<td>Brooks-Carter et al [16], 2000</td>
<td>55</td>
<td>70</td>
<td>Obesity, DM, hypertension</td>
<td>Proliferative endometrium/ submucous myoma</td>
<td>Irregular vaginal bleeding</td>
<td>Pipelle endometrial biopsy</td>
<td>Atypical hyperplasia and well-differentiated villoglandular adenocarcinoma → no final pathologic diagnosis (patient declined hysterectomy and opted for radiation therapy)</td>
<td>5 yr</td>
</tr>
<tr>
<td>Klein et al [17], 1997</td>
<td>52</td>
<td>NA</td>
<td>Postmenopausal status</td>
<td>Proliferative endometrium</td>
<td>None</td>
<td>Endometrial biopsy, US</td>
<td>Stage III, grade 2 endometrial carcinoma</td>
<td>2 wk</td>
</tr>
<tr>
<td>Iqbal and Paterson [18], 1997</td>
<td>53</td>
<td>NA</td>
<td>None</td>
<td>Benign endometrium</td>
<td>Irregular vaginal bleeding</td>
<td>Hysteroscopy, endometrial biopsy</td>
<td>Stage IB, grade 2 endometrioid adenocarcinoma</td>
<td>3 yr</td>
</tr>
<tr>
<td>Margolis et al [19], 1995</td>
<td>58</td>
<td>NA</td>
<td>Hypertension, DM, obesity</td>
<td>Adenomatous complex hyperplasia</td>
<td>Asymptomatic, stress urinary incontinence</td>
<td>None</td>
<td>Stage IB, grade 2 endometrioid adenocarcinoma</td>
<td>3 yr</td>
</tr>
<tr>
<td>64</td>
<td>NA</td>
<td>Hypertension, DM, obesity, postmenopausal status</td>
<td>Endometrial polyp with adenomatous hyperplasia</td>
<td>Irregular vaginal bleeding</td>
<td>Hysteroscopy and D&amp;C</td>
<td>Stage IV endometrial adenocarcinoma with skin metastases</td>
<td>1 yr</td>
<td></td>
</tr>
</tbody>
</table>
Overall, 17 studies in the literature reported endometrial cancer occurring after EA [2,7,11–25]. A total of 21 cases were described, including 3 retrospective chart reviews (1 with 4 descriptive case reports), 3 descriptive literature reviews, and 11 case reports. Reference was made to 22 endometrial cancer cases after EA (Table 1). Of the 22 cases, 5 were included in 2 retrospective reviews [7,11] that did not contain any clinical information pertaining to the cases (Table 1). Hence, only 17 of 22 cases were included in the final analysis (Table 2). In 2 literature reviews [2,23], the described cases overlapped with the cases identified in our systematic review. One case in each of these papers, which was described by Dwyer and Stirrat [26], was excluded from the initial analysis because the patient received a diagnosis of endometrial cancer at EA.

The EA methods described consisted of transcervical resection of the endometrium (10 of 22; 45.5%), rollerball ablation (6 of 22; 27.3%), and coagulation resectoscopy (2 of 22; 9.1%). In 2 cases (9.1%), the ablation type was not specified. There was a single report of endometrial cancer after thermal balloon EA (ThermaChoice) [15], which is the first case of endometrial cancer after second-generation EA reported in the literature. The present case is the first report in the literature to describe the occurrence of endometrial cancer after radiofrequency EA.

Of the 17 cases in the final analysis, mean (SD; 95% CI) patient age at cancer diagnosis was 54.4 (9.9; 49.7–59.2) years. Of the 8 cases in which BMI was described, mean BMI was 38.9, and 11 patients (64.7%) were obese by description. Six patients (35.3%) were postmenopausal at EA, and underwent the procedure to treat postmenopausal bleeding. In addition, 6 patients (35.3%) had a preablation diagnosis of complex atypical or adenomatous hyperplasia. Overall, 10 of 17 patients (58.8%) underwent EA performed in the clinical setting of postmenopausal bleeding or complex atypical endometrial hyperplasia. In total, 14 patients (82.4%) had at least 1 known risk factor for endometrial cancer including hypertension, diabetes, and obesity.

In 13 patients (76.5%), the primary symptom after EA was irregular vaginal bleeding, and 1 patient had pelvic pain exclusively at 33 months after EA [13]. One case report described a patient with no symptoms at presentation and in whom hysterectomy was performed as part of a procedure to treat incontinence [19]. Of note, 2 patients had endometrial carcinoma at EA, although the diagnosis was not known until 2 weeks and 2 months, respectively, after the procedure [15,17]. Endometrial cancer was diagnosed as stage I in 13 patients (76.5%). Brooks-Carter et al [16] described a case in which the stage of endometrial cancer was unknown. The patient underwent palliative treatment for a post-EA diagnosis of well-differentiated adenocarcinoma, and hysterectomy was not performed. Single endometrial cancer cases of stage II, III, and IV were reported individually [17,20,22]. The patient with stage IV endometrial cancer had initially refused hysterectomy to treat postmenopausal bleeding that had been ongoing for 3 years. A periumbilical nodule that
have been proposed as 2 likely mechanisms [2,10]. The areas of the endometrium; the presence of islands of regeneration occurred in the lower uterine segment several years after EA.

Diagnosis of Endometrial Cancer After EA

Ensuring complete destruction of the endometrium after EA is complicated. In general, the adequacy of ablation is assessed at inspection at hysteroscopy. However, data demonstrate that regenerating endometrium persists behind an intrauterine scar that may eventually become obstructed [7,10]. Herein lies the main concern with EA in which endometrial cancer may be masked or its diagnosis delayed.

The systematic search of the literature demonstrated that patients with a diagnosis of endometrial cancer after EA typically had abnormal uterine bleeding, with the exception of 1 patient who had no symptoms and another patient who had pelvic pain after EA. Furthermore, the interval between EA and endometrial cancer diagnosis varied between 6 months and 10 years, and the duration of this did not influence the ability to ascertain the diagnosis of endometrial cancer or the stage of the disease at diagnosis.

Because the risk of endometrial cancer generally increases by approximately 2- to 3-fold between ages 50 to 70 years, it is possible that women who have undergone EA in the last 2 decades are now entering the age group at risk for endometrial cancer [1,2]. These patients should be evaluated with caution, and a diagnosis of endometrial cancer should be taken into consideration.

Patients with bleeding after EA often create a diagnostic dilemma because the basic tools for evaluation of abnormal uterine bleeding often fail to yield substantial results. In addition, the anatomical distortion caused by EA renders hysteroscopy and sonohysterography difficult, and the features normally expected at pelvic ultrasonography are altered [30–32]. This view has shaped the general consensus on the evaluation of abnormal uterine bleeding after EA. However, systematic review and the present case report suggest that endometrial sampling and investigation of abnormal uterine bleeding may be difficult to perform, yet are feasible and often of high yield. Only in 2 cases (11.8%) was a preoperative diagnosis with hysteroscopy or endometrial biopsy not successful at presentation because of cervical stenosis and intrauterine adhesions, respectively.

Of note, 76.5% of endometrial cancer cases after EA reported in the literature were diagnosed at stage I. This finding is consistent with the general presentation of endometrial angiogenesis such as vascular endothelial growth factor are crucial in the overall process [26,28].

A percentage of patients continue to have persistent or recurrent menorrhagia after EA. Not uncommonly, residual endometrium may be identified at hysteroscopy, pathologic assessment, or magnetic resonance imaging after EA in these patients [27,29,30]. It is unknown whether unopposed estrogen stimulation in patients at risk of endometrial cancer may promote cancer in these regenerating areas of the endometrium. However, excess estrogen stimulation is a well-known risk factor for endometrial cancer and has a critical role in its development [1].

Discussion

Case Report

Concerns have been proposed about the possibility that symptoms of endometrial cancer may be masked through endometrial destruction with EA [10]. The present patient had a 6-month history of bleeding after a prolonged period of amenorrhea, which prompted evaluation. In addition, she had specific risk factors for endometrial cancer including obesity and hypertension. This presentation is consistent with endometrial cancer after EA in the case reports reviewed, which highlights that patient history, clinical findings, and risk factors may alert physicians to the possibility of endometrial cancer after EA.

Furthermore, the present patient had stage IA endometrial cancer at presentation, as was observed in most cases reported in the literature. Hence, despite the interval between EA and endometrial cancer, the diagnosis of endometrial cancer does not seem to be masked by the underlying process of endometrial destruction.

The present patient had a background of endometrial hyperplasia with atypia at final pathologic analysis, in which endometrial cancer was identified. This finding is not uncommon in endometrial cancer [15,23–25]. Most important, the area of the uterus that seemed to have endured the effects of EA was obliterated completely, and the lower segment, which was accessible at hysteroscopy, contained the area of endometrial cancer. It is unknown whether the area in which endometrial cancer was identified represents an untreated area at ablation 5 years previously or whether endometrial regeneration occurred in the lower uterine segment several years after EA.

Endometrial Repair and Regeneration

It is difficult to determine how cancer develops in ablated areas of the endometrium; the presence of islands of regenerating endometrium below the basalis layer and adenomyosis have been proposed as 2 likely mechanisms [2,10]. The process of human tissue repair involves inflammation, tissue formation, and tissue remodeling [26,27]. This process normally is cyclic during menstruation. Molecular and hormonal impulses including matrix metalloproteinases and estrogen and progesterone are responsible for endometrial breakdown and regeneration. In addition, factors involved in endometrial angiogenesis such as vascular endothelial growth factor are crucial in the overall process [26,28].

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Of note, 76.5% of endometrial cancer cases after EA reported in the literature were diagnosed at stage I. This finding is consistent with the general presentation of endometrial
cancer: stage I disease is diagnosed in 73% of patients, and stage II in 10% [1]. Furthermore, survival rates for stage I and II disease are as high as 91% at 5 years [1]. The present analysis suggests that endometrial cancer does not seem to be diagnosed at a later stage after EA and that the patterns of occurrence of endometrial cancer after EA are generally consistent with those of endometrial cancer without EA.

**Patient Selection**

Differentiating between normal and abnormal bleeding after EA also is difficult [24]. Recognizing the difference is important because the initial symptom of endometrial cancer most frequently is vaginal bleeding. However, endometrial cancer is unique in that patient risk factors are largely predictive of disease occurrence.

In 1 series, patients older than 70 years who had diabetes and were nulliparous had an 87% likelihood of developing atypical endometrial hyperplasia or endometrial cancer [1]. However, only 3% who did not demonstrate all 3 risk factors were observed to have clinically significant disease [1].

Among the 17 patients identified through a systematic review who had endometrial cancer after EA, 6 (35.3%) had complex atypical hyperplasia or adenomatous hyperplasia at preoperative histologic testing. As many as 40% of patients with a diagnosis of complex atypical endometrial hyperplasia have coexistent endometrial cancer at diagnosis [33]. In patients with such abnormal endometrial disease, the risk of future endometrial cancer is as high as 30% [34]. Therefore, it is not surprising that all patients with endometrial cancer diagnosed within 12 months of EA had preexistent abnormal endometrial disease, which suggests disease progression or undiagnosed endometrial cancer at EA. Furthermore, compared with women with heavy menstrual blood loss, patients with postmenopausal bleeding are at 8-fold increase of risk of endometrial cancer [35]. Despite this detail, 6 of 17 endometrial cancer cases reported were in women who underwent EA because of postmenopausal bleeding; 2 of those patients had unrecognized endometrial cancer at EA [15,17]. Only 3 of the 17 women did not have any risk factors for endometrial cancer. Therefore, the importance of rigorous patient selection and the need to perform EA only in patients with heavy menstrual blood loss in the absence of documented endometrial histologic disease cannot be overemphasized.

Many investigators advocate close postoperative surveillance of patients after EA [24,36]. At our institution, patients are followed up at 3 months postoperatively, and as necessary thereafter. A diagnostic procedure is attempted or baseline imaging is performed in all patients with recurrent vaginal bleeding or symptoms of endometrial cancer after EA. Patients at high risk of endometrial cancer or with a history of endometrial hyperplasia are referred for hysterectomy.

**Treatment of Endometrial Cancer**

Our case report cites an interesting observation. In our patient, endometrial cancer was confined to the lower uterine segment, where the effects of the ablation were not prominent. The upper uterine segment seemed to be completely obliterated by uterine synechiae that resulted from the ablation. This finding suggests that EA may limit the potential for endometrial carcinoma in patients at risk. Indeed, EA has been proposed as a treatment of endometrial cancer. This treatment approach has been described in patients who are poor candidates for surgery and in whom progesterone is contraindicated such as those with complicated cardiac disease [37–39]. Sharp et al [37] described the use of microwave EA in treatment of grade 2 endometrioid endometrial cancer in an 89-year-old woman. The patient was free of disease at 36 months after the procedure. Second-generation EA is now being considered as a palliative measure in the treatment of bleeding due to advanced endometrial cancer [40].

To further explore the effect of EA on the incidence of endometrial cancer, Horowitz et al [41] conducted an elegant study in animals. New Zealand white rabbits received unopposed estrogen for 18 months after rollerball EA. EA was performed at laparotomy, and both uterine horns were ablated. In the study group, 3 of 17 rabbits (17.6%) had endometrial hyperplasia or adenocarcinoma compared with 4 of 16 rabbits in the control group (25%). The study did not show a statistically significant decrease in the incidence of endometrial cancer in the rabbits that received ablation and were exposed to estrogen compared with control rabbits; however, it was underpowered to detect any meaningful difference. Ultimately, long-term longitudinal studies in human beings are required to determine the effect of EA on the incidence of endometrial cancer.

**Conclusion**

The incidence of endometrial cancer after EA continues to be undefined, although it does not seem to be increased [7]. Herein are reported all known endometrial cancer cases after EA: 22 individual endometrial cancer cases after all types of EA including first- and second-generation EA procedures. To our knowledge, the present case report is the first known case in the literature of endometrial cancer after radiofrequency EA. Although the underlying mechanisms of first- and second-generation EA are similar, there is no indication that the pathophysiologic characteristics of endometrial cancer in either case are different.

Whether EA prevents the occurrence of endometrial cancer or delays its diagnosis has yet to be determined, and requires large longitudinal studies. It is unknown what the appropriate postablation surveillance should constitute. However, it seems that most patients with endometrial cancer after EA demonstrate symptoms such as bleeding and pelvic pain. In most cases, a preoperative diagnosis is feasible, contrary to the concerns that the diagnosis of endometrial cancer is delayed and may be difficult to achieve.

Endometrial cancer was observed to occur in most patients with risk factors for endometrial cancer such as complex atypical endometrial hyperplasia, obesity, hypertension,
and diabetes. This finding highlights the importance of careful patient selection and the need for adequate patient evaluation and counseling before and after the procedure. Prompt thorough evaluation of postablation symptoms is of prime importance. The EA procedure should be restricted to premenopausal patients who do not have a risk factor for endometrial cancer and who have documented normal endometrial histopathologic features at preablation evaluation.

References


