Anastrozole versus Tamoxifen Treatment in Postmenopausal Women with Endocrine-Responsive Breast Cancer and Tamoxifen-Induced Endometrial Pathology

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Abstract

Purpose: To investigate the effect of switching from adjuvant tamoxifen to anastrozole (Arimidex) treatment in postmenopausal women with endocrine-responsive breast cancer and histologically proven tamoxifen-induced benign endometrial pathology.

Experimental Design: Two hundred twenty-six postmenopausal women who had received adjuvant tamoxifen 20 mg/d (≥12 months, ≤48 months) and developed abnormal vaginal bleeding and/or an asymptomatic endometrial thickness >10 mm [measured by transvaginal ultrasound (TVUS)] were subjected to hysteroscopy and dilation and curettage (D&C). Thereafter, 171 patients were randomized in a phase III study to continue tamoxifen treatment (n = 88) or switch to anastrozole 1 mg/d (n = 83). Patients were monitored for ≤42 months using TVUS at 6-monthly intervals.

Results: At study entry, there were no significant differences in vaginal bleeding, endometrial thickness, and histologic findings between the two treatment groups. Throughout the treatment period, there was no significant difference in recurrent vaginal bleeding between groups [anastrozole, 4 of 83 (4.8%); tamoxifen, 9 of 88 (10.2%); P = 0.18]. Six months after randomization, the mean endometrial thickness for patients who switched to anastrozole was significantly reduced compared with those who continued tamoxifen treatment (P < 0.0001). Significantly fewer anastrozole patients required a repeat hysteroscopy and D&C compared with those on tamoxifen [4 of 83 (4.8%) and 29 of 88 (33.0%), respectively; P < 0.0001]. Repeat hysteroscopy and D&C revealed endometrial atrophy in all 4 cases in the anastrozole group and 14 polyps, 8 hyperplasias, and 7 atrophies in the tamoxifen group.

Conclusions: Switching from tamoxifen to anastrozole treatment significantly reduced the need for a second hysteroscopy and D&C due to recurrent vaginal bleeding or thickening of the endometrium in postmenopausal breast cancer patients with tamoxifen-induced endometrial abnormalities.

Breast cancer is a major cause of morbidity and mortality, with more than 1 million new cases and 410,000 deaths reported worldwide each year (1). For many years, the selective estrogen receptor modulator tamoxifen has been the standard treatment for postmenopausal women with hormone-sensitive breast cancer. Although tamoxifen is an estrogen receptor antagonist in breast tissue, it also acts as an estrogen agonist in other tissues such as the endometrium. This agonist effect in the endometrium can stimulate proliferation, which increases the risk of polyps, hyperplasia, and endometrial cancer by 2- to 4-fold compared with patients not receiving tamoxifen (2–10). It has been reported that 10% of tamoxifen-treated patients will develop tamoxifen-induced endometrial pathology within 5 years, leading to operative intervention (11).

Although the American Society of Clinical Oncology Technology Assessment (12) has recently recommended that the optimal hormonal therapy for postmenopausal women with hormone-sensitive early breast cancer should now include an aromatase inhibitor, the 9th St. Gallen International Consensus Conference did not reach a consensus on the optimal use of aromatase inhibitors on the treatment of primary breast cancer (13). The American Society of Clinical Oncology recommendations stated that the incidences of life-threatening adverse events, such as endometrial cancer, with an aromatase inhibitor are significantly reduced compared with tamoxifen. This has been shown in the Arimidex, Tamoxifen, Alone or in Combination (ATAC) trial, where initiating treatment with anastrozole (Arimidex) in postmenopausal...
women with early breast cancer has been shown to significantly reduce the incidence of endometrial cancer, vaginal bleeding, and discharge compared with initiating treatment with tamoxifen (14). Moreover, subprotocols of the Arimidex, Tamoxifen, Alone or in Combination trial have also indicated a reduced incidence of endometrial abnormalities and medical interventions in the anastrozole group compared with the tamoxifen group (15–18), suggesting that anastrozole may have a protective effect against endometrial cancer (19).

There are currently no published prospective data that detail the effect of anastrozole in patients with tamoxifen-induced endometrial pathology. Therefore, the aim of this study was to evaluate the effect of switching from adjuvant tamoxifen to anastrozole on recurrent vaginal bleedings (primary end point) and endometrial thickening (secondary end point) in postmenopausal breast cancer patients with histologically verified tamoxifen-induced endometrial changes.

**Patients and Methods**

**Eligibility criteria.** Between January 1998 and January 2003, 226 eligible postmenopausal women with endocrine-responsive, invasive breast cancer, who were receiving adjuvant tamoxifen treatment and had suspected endometrial changes (abnormal vaginal bleeding and/or thickened endometrium), were referred to our hospital for invasive diagnostic measures (Fig. 1).

The postmenopausal state was defined as no regular menstruation within 1 year of primary treatment of breast cancer or after a bilateral salpingo-oophorectomy. After immunohistochemical assessment, patients were defined as “endocrine responsive” if the estimated estrogen and progesterone receptor content of the primary tumor was ≥20% and ≥10%, respectively; “uncertain endocrine responsive” if receptor content was 1% to 19% and 1% to 9%, respectively; and “endocrine nonresponsive” if the estrogen and progesterone receptor content at the primary tumor was undetectable.

A hysteroscopy, in addition to dilation and curettage (D&C), was done in all patients and polyps were resected by operative hysteroscopy. Postmenopausal women with histopathologically confirmed endometrial polyps, hyperplasia, or glandulocystic atrophy, an estimated life expectancy of ≥5 years (irrespective of breast cancer diagnosis), and no severe or relevant comorbidity (performance status Eastern Cooperative Oncology Group ≤2, Charlson ≤3) were included in the trial. Patients had to be accessible for treatment and follow-up. There were no age limitations in this trial. Clandulocystic atrophy was defined as an apparent increase in endometrial thickness detected by transvaginal ultrasound (TVUS), which may be a result of tamoxifen-induced edema of cystically dilated endometrial glands with periglandular stromal and myometrial condensation whereas the overlying epithelium remains atrophic (20).

Patients were excluded from the study if they had histopathologically proven endometrial carcinoma or atypical hyperplasia, if they were receiving concurrent treatment with other hormonal agents, or if no written consent had been obtained (Fig. 1).

**Trial design.** In this open-label phase III trial, 173 patients were prospectively randomized to either continued treatment with tamoxifen 20 mg/d or switch to anastrozole 1 mg/d. Patients were randomized by “complete randomization” (synonymous simple randomization), which is a simple coin-tossing procedure, and “random number generation” was used to prepare the randomization sequence (21).

The study was conducted in accordance with the Declaration of Helsinki and informed consent was obtained from each patient.

The primary end point of the study was the incidence of renewed vaginal bleeding. The secondary end point was change in endometrial thickness (defined as the width of the combined thickness of the anterior and posterior sides of the endometrium) as detected by TVUS.

For the 171 patients included in the analysis, a normal endometrial thickness (<5 mm) had been confirmed by TVUS in 67% of patients before the initiation of adjuvant tamoxifen. The endometrial thickness in the remaining 33% asymptomatic patients was not known at the time of beginning with adjuvant tamoxifen treatment.

**Follow-up.** Assessments for patients receiving randomized treatment were made at 6-monthly intervals by the same gynecologist for up to a maximum of 42 months. Endometrial thickness and morphology were measured using TVUS in the longitudinal plane with a 5.0-MHz transvaginal probe. Data on other adverse events were not collected and evaluated as part of the study.

Patients with renewed abnormal vaginal bleeding following treatment were subjected to a repeat hysteroscopy and D&C immediately. Patients with an endometrial thickness >10 mm and nonsuspicious morphology required repeated TVUS after 3 months. In the event of any further increase in endometrial thickness, or if the endometrium showed any suspicious morphology (nonhomogeneous endometrium with different density, cysts, no clear boundary between endometrium and myometrium), hysteroscopy and D&C were done. Further treatment in such cases was decided on an individual basis; patients stopped...
endocrine treatment, persisted with the assigned study treatment, or switched treatment. It was recommended that randomized treatment should continue up to a maximum of 5 years or until tumor recurrence, loss of follow-up, or troubling adverse events.

Statistical methods. Using a two-tailed $\chi^2$ test with a significance level of $\alpha = 0.05$ and a power of 0.80, a total of 170 patients were required to show a significant decrease of recurrent vaginal bleeding from $33\%$ to $15\%$. Determination of sample size was achieved by using the software nQuery (22).

Statistical analysis was done on a per protocol basis. A two-tailed $t$ test was used to calculate the differences between the means of independent samples that had continuous variables and, depending on the number of cases, the $\chi^2$ test or Fisher’s exact test was used to compare categorical variables. All statistical tests were two sided.

The body mass index (BMI) of each patient was calculated by dividing weight (kg) by height$^2$ (m). Mean TVUS measurements (SD) were calculated in each patient group for all the examination dates.

Results

Patients. Of the 226 postmenopausal breast cancer patients who had previously received tamoxifen 20 mg/d for $\geq 12$ and $\leq 48$ months, 173 (76.5%) were included in the study (Fig. 1). Following randomization, one patient was withdrawn due to breast cancer recurrence and another one was withdrawn due to missing ultrasound data; neither received study medication. A total of 83 patients were randomized to switch to anastrozole and 88 patients were randomized to continue tamoxifen treatment. The two treatment groups were well balanced in terms of baseline patient and tumor characteristics (Table 1).

At the time of study entry, there was no significant difference in the incidence of vaginal bleeding and an endometrial thickness $>10$ mm between the anastrozole and tamoxifen groups ($P = 0.64$; Table 2). Of the 48 patients (57.8%) within the anastrozole group and 55 patients (62.5%) within the tamoxifen group who presented with an endometrial thickness $>10$ mm, 27 and 26 patients, respectively, presented with vaginal bleeding. A total of 53 patients were followed for a maximum period of 42 months. The mean duration of tamoxifen treatment before the first endometrial pathology was observed was similar between the two treatment arms (Table 1). The duration of endocrine treatment after randomization, and overall, was longer in the anastrozole group [32.2 (9.7) and 58.6 (6.6) months, respectively; $P = 0.18$] compared with the tamoxifen group [30.3 (8.9) and 57.8 (6.7) months, respectively; $P = 0.26$]. However, this difference did not reach statistical significance.

### Table 1. Patient demographics, treatment duration, and baseline tumor characteristics

<table>
<thead>
<tr>
<th></th>
<th>Anastrozole 1 mg/d ($n = 83$)</th>
<th>Tamoxifen 20 mg/d ($n = 88$)</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (range), y</td>
<td>57.9 (46-78)</td>
<td>58.9 (46-83)</td>
<td>0.34*</td>
</tr>
<tr>
<td>Mean BMI (range), kg/m$^2$</td>
<td>28.5 (16-52)</td>
<td>27.7 (19-52)</td>
<td>0.35*</td>
</tr>
<tr>
<td>Mean duration of tamoxifen before endometrial pathology, mo (SD)</td>
<td>25.4 (11.2)</td>
<td>27.2 (9.8)</td>
<td>0.18*</td>
</tr>
<tr>
<td>Mean duration of endocrine treatment after randomization, mo (SD)</td>
<td>32.2 (9.7)</td>
<td>30.3 (8.9)</td>
<td>0.18*</td>
</tr>
<tr>
<td>Tumor size (%), mm</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>$\leq 20$</td>
<td>35 (42.2)</td>
<td>45 (51.1)</td>
<td>0.24*</td>
</tr>
<tr>
<td>$&gt;20$</td>
<td>48 (57.8)</td>
<td>43 (48.9)</td>
<td></td>
</tr>
<tr>
<td>Nodal status (%)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>0</td>
<td>42 (50.6)</td>
<td>53 (60.2)</td>
<td>0.21*</td>
</tr>
<tr>
<td>1</td>
<td>41 (49.4)</td>
<td>35 (39.8)</td>
<td></td>
</tr>
<tr>
<td>American Joint Committee on Cancer stage (%) $^*$</td>
<td></td>
<td></td>
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<tr>
<td>Stage I</td>
<td>19 (22.9)</td>
<td>31 (35.2)</td>
<td>0.20*</td>
</tr>
<tr>
<td>Stage IIA/B</td>
<td>41 (49.4)</td>
<td>38 (43.2)</td>
<td></td>
</tr>
<tr>
<td>Stage IIIA/B</td>
<td>23 (27.7)</td>
<td>19 (21.6)</td>
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<tr>
<td>Endocrine responsiveness $^*$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Responsive</td>
<td>63 (75.9)</td>
<td>66 (75.0)</td>
<td>0.45*</td>
</tr>
<tr>
<td>Uncertain</td>
<td>15 (18.1)</td>
<td>18 (20.5)</td>
<td></td>
</tr>
<tr>
<td>Nonresponsive</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Unknown</td>
<td>5 (6.0)</td>
<td>4 (4.5)</td>
<td></td>
</tr>
<tr>
<td>Surgical therapy (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breast-conserving therapy</td>
<td>22 (26.5)</td>
<td>34 (38.6)</td>
<td>0.09*</td>
</tr>
<tr>
<td>Mastectomy</td>
<td>61 (73.5)</td>
<td>54 (61.4)</td>
<td></td>
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<tr>
<td>Adjuvant systemic therapy (%)</td>
<td></td>
<td></td>
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<tr>
<td>Tamoxifen</td>
<td>48 (57.8)</td>
<td>60 (68.2)</td>
<td>0.16*</td>
</tr>
<tr>
<td>Chemotherapy followed by tamoxifen</td>
<td>35 (42.2)</td>
<td>28 (31.8)</td>
<td></td>
</tr>
</tbody>
</table>

*Two-sided $t$ test.
$^*$Pearson’s $\chi^2$ test.
$^*$See ref. 35.

$^*$Patients were defined as endocrine responsive if the immunohistochemically estimated estrogen and progesterone receptor content at the primary tumor was $>20\%$ and $>10\%$, respectively; uncertain endocrine responsive if $1\%$ to $19\%$ and $1\%$ to $9\%$, respectively; and endocrine nonresponsive if the estrogen and progesterone receptor content was undetected.
Primary end point (renewed vaginal bleeding). Throughout the treatment period, there was no significant difference in the number of patients experiencing renewed vaginal bleeding between the anastrozole and tamoxifen groups [4 (4.8%) and 9 (10.2%) patients, respectively; \( P = 0.18 \)]. Of the 62 patients with histologically confirmed atrophy, 23 (37.1%) reported vaginal bleeding before randomization and 11 (17.7%) reported vaginal bleeding after.

Secondary end point (endometrial thickness). The mean endometrial thickness at time of admittance study entry was comparable in the anastrozole and tamoxifen groups \([12.4 (±5.8) \text{ and } 12.9 (±5.6) \text{ mm, respectively; } P = 0.59] \). Six months after randomization, the endometrial thickness was significantly lower in those patients who switched to anastrozole \([3.3 (±1.2) \text{ mm]} \) compared with those continuing tamoxifen treatment \([6.7 (±2.4) \text{ mm]; } P < 0.0001 \). A significant difference between the two groups could be found throughout the entire treatment period (Fig. 2). In the anastrozole group, no patients presented with an endometrial thickness >10 mm. In contrast, 30 patients \((34.1\%\) within the tamoxifen group presented with an endometrial thickness >10 mm \((11-24 \text{ mm}; P < 0.0001) \). Eight of these patients reported vaginal bleeding. Twenty-six of these 30 patients \((86.7\%\) required a second D&C. Significantly fewer patients in the anastrozole group underwent a repeat hysterectomy and D&C due to recurrent vaginal bleeding or thickening of the endometrium compared with those who continued tamoxifen treatment \([4 (4.8\%) \text{ versus } 29 (33.0\%) \text{ patients; } P < 0.0001] \).

Histopathologic findings. Of the four patients in the anastrozole group who required repeat D&C due to vaginal bleeding, endometrial atrophy was found in all four cases. Of the 29 patients in the tamoxifen group undergoing second hysterectomy and D&C, polyps were found in 14 cases \((48.3\%\), hyperplasia in 8 cases \((27.6\%\), and atrophy in 7 cases \((24.1\%\). Two of the eight patients with hyperplasia had atypical hyperplasia and underwent hysterectomy. The results of the first and second gynecologic investigations were consistent in 21 of the 33 patients \((63.6\%) \text{ assessed (} P = 0.003) \).

Additional findings. There was no significant difference in the frequency of breast cancer recurrences during endocrine treatment between the two treatment groups \((7.2\%\text{ in the anastrozole group and } 9.1\%\text{ in the tamoxifen group; } P = 0.66) \). Examination of a possible influence of BMI, age, and duration of endocrine treatment on the occurrence of first and second endometrial pathology revealed no significant correlation.

Discussion

This open-label randomized trial was designed to evaluate the effects of switching to anastrozole following adjuvant tamoxifen treatment \((≥12 \text{ months, } ≤48 \text{ months})\) compared with remaining on tamoxifen in postmenopausal patients with breast cancer and histologically confirmed tamoxifen-induced endometrial pathology \((\text{polyps, hyperplasia, and glandulocystic atrophy})\). The rationale behind switching to anastrozole was to avoid invasive procedures necessitated by recurrent abnormal vaginal bleeding and/or thickened endometrium in tamoxifen-treated patients.

Although there was no significant difference in recurrent vaginal bleeding between the two treatment arms throughout the treatment period, significantly fewer patients receiving anastrozole required repeat hysterectomy and D&C compared with those who continued tamoxifen treatment. Additionally, the analyses show a significant reduction in mean endometrial thickness in patients who switched to anastrozole compared with those who continued on tamoxifen treatment. These data are in line with our earlier study investigating the effects of tamoxifen on the endometrium in postmenopausal women.

<table>
<thead>
<tr>
<th>Table 2. Endometrial pathology at randomization</th>
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<tbody>
<tr>
<td><strong>Anastrozole 1 mg/d (n = 83)</strong></td>
</tr>
<tr>
<td><strong>Endometrial pathology at randomization (%)</strong></td>
</tr>
<tr>
<td>Vaginal bleeding</td>
</tr>
<tr>
<td>Endometrial thickness &gt;10 mm, no bleeding</td>
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<tr>
<td>Vaginal bleeding + endometrial thickness &gt;10 mm</td>
</tr>
<tr>
<td><strong>Histologic findings of first hysteroscopy and D&amp;C (%)</strong></td>
</tr>
<tr>
<td>Endometrial polyps</td>
</tr>
<tr>
<td>Hyperplasia</td>
</tr>
<tr>
<td>Glandulocystic atrophy</td>
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</table>

*Pearson’s \(\chi^2\) test.
with breast cancer (11). Our previous data showed a significant increase in endometrial thickness during tamoxifen treatment compared with placebo.

To date, no other published randomized studies have evaluated endocrine treatment in patients with tamoxifen-induced endometrial pathology. However, consistent with our findings, a retrospective analysis of the Arimidex, Tamoxifen, Alone or in Combination trial (n = 9,366) shows that initiating treatment with anastrozole rather than tamoxifen can reduce the risk of gynecologic adverse events, including vaginal bleeding, vaginal discharge, and endometrial cancer (14). Further analyses of the Arimidex, Tamoxifen, Alone or in Combination trial have also indicated a protective effect of anastrozole against endometrial cancer compared with expected rates in an untreated, age-matched population. In contrast to this protective effect of anastrozole, tamoxifen treatment resulted in an increase in relative risk for endometrial cancer to 2.68 (95% confidence interval, 1.34-4.80; ref. 19).

A study by Berliere et al. (23) evaluated the effects of aromatase inhibitors (anastrozole or letrozole) on the endometrium of 31 breast cancer patients who had developed local recurrence or pernicious metastatic evolution of their breast tissue during tamoxifen treatment. The authors concluded that aromatase inhibitor treatment did not reduce endometrial thickness. Patients were grouped into those who have never been treated for endometrial pathology before or during tamoxifen therapy (n = 27) and those who had been treated for endometrial polyps before initiation of tamoxifen therapy (n = 4). The mean endometrial thickness evaluated by TVUS before and 2 years after initiation of aromatase inhibitor treatment was found to be 9.2 and 8.5 mm, respectively, in the untreated group, and 7.5 and 7.2 mm, respectively, in the treated group. During follow-up, one case of polyps was observed in each group. In the present study, the mean endometrial thickness decreased to within reference ranges (3-4 mm) in patients receiving anastrozole but not in those receiving tamoxifen (7-9 mm). The greater decrease in endometrial thickness observed in this study following treatment with anastrozole could possibly be explained by a higher endometrial thickness before randomization (12.4 mm with anastrozole and 12.9 mm with tamoxifen). It is also important to consider that hysteroscopy and D&C may have contributed to the reduction in endometrial thickness by removing the pathologic cause (e.g., polyps and hyperplasia). Preliminary clinical trial results have shown that switching to an aromatase inhibitor significantly reduces tamoxifen-induced endometrial thickening (measured by TVUS) after 6 months (P =0.021; ref. 24). In addition, 12 months of anastrozole treatment in 11 obese postmenopausal women with endometrial hyperplasia resulted in an atrophic endometrium in all cases (25).

It has been hypothesized that the increased incidence of endometrial pathology with adjuvant treatment may be associated with patient age and BMI. In postmenopausal women, endometrial thickness before and during adjuvant treatment has been shown to be negatively correlated with age and positively correlated with BMI, possibly as a result of increased aromatase activity in fatty tissues (11, 26, 27). However, the analyses in the present study showed that BMI, age, and duration of endocrine treatment showed no significant correlation with the occurrence of primary and secondary endometrial pathologies.

As the present trial was not double-blinded, a diagnostic bias cannot be ruled out. However, as the frequency of renewed vaginal bleeding (as an objective variable) observed in the tamoxifen group was at least twice that observed in the anastrozole group, a bias seems to be unlikely. The reliability of TVUS was also unclear when the study was initiated. It is now known that this is not the best method for assessing tamoxifen-induced endometrial changes due to the high false-positive rate (11, 28). However, it has recently been reported that TVUS is appropriate for screening the endometrium for intrauterine pathology before endocrine treatment in women with breast cancer (with an endometrial thickness of 3 mm as a cutoff for needing further investigations with hysteroscopy and endometrial biopsy; refs. 29, 30).

Avoiding invasive procedures is one of the potential benefits of switching from tamoxifen to anastrozole treatment for postmenopausal breast cancer patients. Although efficacy was not an end point of the current study, the Italian Tamoxifen Arimidex trial, which has recruited 446 postmenopausal women who have undergone surgery for node-positive endocrine-responsive tumors, has shown that patients who switch to anastrozole show a reduction in the risk of relapse and death compared with those who continue tamoxifen treatment (median follow-up, 36 months; ref. 31). These data are further supported by combined analyses of the Austrian Breast and Colorectal cancer Study Group Trial 8 and Arimidex-Nolvadex 95 Trial, in which 3,224 postmenopausal women are being evaluated to assess whether switching to anastrozole after 2 years of tamoxifen is more effective than continuing tamoxifen treatment (32). In this combined analysis, the hazard ratio for recurrence-free survival with anastrozole compared with tamoxifen treatment was 0.59 (95% confidence interval, 0.42-0.82; P < 0.0018) regardless of the nodal status. Thus, the inclusion of a third-generation aromatase inhibitor in the endocrine treatment sequence for postmenopausal breast cancer patients significantly improves outcomes compared with tamoxifen treatment alone.

The American Society of Clinical Oncology Technology Assessment states that postmenopausal women with endocrine-responsive breast cancer may consider switching to an aromatase inhibitor after 2 to 3 years of treatment with tamoxifen to reduce the risk of tumor recurrence (12). In addition, the report states that treatment with an aromatase inhibitor significantly reduces the incidence of life-threatening adverse events, including endometrial cancer, compared with tamoxifen treatment (12). Indeed, recent clinical trial data have shown that patients who initiate treatment with anastrozole have a significantly lower risk of developing endometrial cancer and other gynecologic disorders (vaginal bleeding and discharge) than those who initiate treatment with tamoxifen (14, 16, 18, 33, 34). Although there was no significant difference between groups in renewed vaginal bleeding throughout the treatment period in this study, our results show that switching from tamoxifen to anastrozole significantly reduces the need for a second hysteroscopy and D&C due to renewed vaginal bleeding or endometrial thickening in postmenopausal breast cancer patients with tamoxifen-induced endometrial abnormalities.

Acknowledgments

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References


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