Evolving Issues in the Clinical and Managed Care Settings on the Management of Menopause Following the Women's Health Initiative

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ABSTRACT

BACKGROUND: Publication of the Women's Health Initiative (WHI) trial results in 2002 significantly reduced physician and patient confidence in and acceptance of hormone replacement therapy (HRT) as an appropriate treatment option for menopause-associated vasomotor symptoms (VMS). This was true despite the fact that the WHI trial was a primary prevention study conducted in postmenopausal women and was not designed to evaluate the efficacy of HRT in the treatment of VMS.

OBJECTIVE: To review data from the WHI, including recent analyses, demonstrating the risks and benefits of HRT in postmenopausal women, to describe changes in menopause treatment guidelines and HRT use since publication of early WHI results nearly 6 years ago, and to identify opportunities for improving the quality of care in perimenopausal women.

SUMMARY: Early results from the WHI demonstrated that the risks of long-term HRT in postmenopausal women outweighed the benefits, leading study investigators to conclude that HRT should not be initiated or continued for the primary prevention of coronary heart disease (CHD) in postmenopausal women. Treatment guidelines published by several professional and managed care organizations continue to advocate the use of HRT for treatment of moderate-to-severe VMS. Nevertheless, physician and patient confidence in HRT has declined, as evidenced by a decrease in new HRT prescriptions and an increase in the discontinuation rate of HRT immediately following publication of the preliminary WHI results. Recent analyses demonstrate that the risk for CHD in postmenopausal women is largely dependent upon the age of the woman and the number of years since menopause, with a lower risk for CHD in women aged 50 to 59 years and in women who experienced menopause within the previous 10 years. The highest risk for CHD was evident in women aged 70 to 79 years and in women who experienced menopause 20 or more years ago. Although these data do not support the use of HRT as a primary prevention strategy in postmenopausal women, they do suggest the need to further evaluate the benefits and risks of HRT in perimenopausal women based on patient-specific characteristics, including age and time since menopause.

CONCLUSION: Menopausal women present a unique opportunity for health care providers to improve the quality of care among women, not only as it relates to the treatment of VMS, but also as it relates to osteoporosis and cardiovascular disease, 2 common comorbidities in perimenopausal and postmenopausal women.

J Manag Care Pharm. 2008;14(3)(suppl S):S7-S13

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Introduction

The use of hormone replacement therapy (HRT), the gold standard in the management of moderate-to-severe menopause-associated vasomotor symptoms (VMS) (i.e., hot flashes, night sweats),[1-3] has received increased media attention and has been debated among the health care community since the early results of the Women's Health Initiative (WHI) were published in 2002.[4] The WHI found an increased risk of coronary heart disease (CHD), breast cancer, stroke, and venous thromboembolism in postmenopausal women taking HRT (WHI, 2002).[4] Further, results from the Heart and Estrogen/Progestin Replacement Study Follow-Up (HERS II)[5,6] and the Million Women Study[7] were released, adding more evidence that HRT in postmenopausal women may do more harm than good, as evidenced by increased risks of breast cancer[7] and venous thromboembolism,[6] and the absence of a cardioprotective effect.[5]

Since these data were published in 2002-2004, researchers have continued to analyze the data from the WHI to determine if the observed deleterious effects of HRT are limited to a specific subset of women. The purpose of this article is to (1) review data from the WHI, including recent analyses, demonstrating the risks and benefits of HRT in postmenopausal women; (2) to describe changes in menopause management guidelines and HRT use since the publication of the WHI results; (3) to summarize management guidelines for conditions related to menopause; and (4) to identify opportunities for improving the quality of care in perimenopausal women.

Women's Health Initiative

Study Design

The WHI was a multiphase, multicenter, randomized, double-blind, placebo-controlled, primary prevention trial started in 1993 that was designed to evaluate the efficacy and safety of (1) a low-fat diet; (2) HRT (2 parallel studies of estrogen [0.625 mg conjugated equine estrogen]) plus progestin [2.5 mg medroxyprogesterone acetate] in women with a uterus or estrogen alone in women who had had a hysterectomy); and (3) calcium and vitamin D supplementation.[8] Each of these interventions was aimed at reducing specific morbidities (i.e., diet: breast and colorectal cancers and CHD; HRT: CHD, other cardiovascular disease [CVD], and hip and other fractures; calcium and vitamin D: hip and other fractures and colorectal cancer). Importantly, this trial was not designed to evaluate the benefits or risks of HRT when used for management of menopause-associated VMS. Postmenopausal women aged between 50 and 79 years who did not have a history of breast cancer were eligible for inclusion in the study. The first phase of the study was a controlled clinical trial in which subjects were randomized to the diet study or to the HRT study. After 1 year of study participation, subjects were eligible for inclusion in the calcium plus vitamin D

TABLE 1 *Relative and Absolute Risks of Major Clinical Outcomes in the Estrogen Plus Progestin Substudy of the Women's Health Initiative*[a]

Outcome	Relative Risk (95% Confidence Interval)[b] Estrogen + Progestin Vs. Placebo	Absolute Risk (No. per 10,000 Person Years)	
		Estrogen + Progestin (N = 8506)	Placebo (N = 8102)
Coronary heart disease events	1.24 (1.00-1.54)	39	33
Nonfatal myocardial infarction[c]	1.28 (1.00-1.63)	31	25
Coronary heart disease deaths	1.10 (0.70-1.75)	8	8
All strokes	1.31 (1.02-1.68)	31	24
Ischemic stroke	1.44 (1.09-1.90)	26	18
Deep vein thrombosis	1.95 (1.43-2.67)	26	13
Pulmonary embolism	2.13 (1.45-3.11)	18	8
Invasive breast cancer	1.24 (1.01-1.54)	41	33
Invasive colorectal cancer	0.56 (0.38-0.81)	9	16
Endometrial cancer	0.81 (0.48-1.36)	6	7
Total fracture	0.76 (0.69-0.83)	152	199
Hip fracture	0.67 (0.47-0.96)	11	16
Total mortality[d]	0.98 (0.82-1.18)	52	53
Global index[d,e]	1.15 (1.03-1.28)	170	151

[a] *Final, centrally adjudicated data after a mean follow-up of 5.6 years, unless otherwise noted.*

[b] *Nominal (unadjusted) confidence intervals.*

[c] *Includes silent myocardial infarction.*

[d] *Data not centrally adjudicated; mean follow-up = 5.2 years.*

[e] *Global index = the earliest occurrence of CHD events, invasive breast cancer, stroke, pulmonary embolism, endometrial cancer, colorectal cancer, hip fracture, or death due to other causes.*

Data from References 4, 10-15, and 45.

study. Women deemed ineligible for the controlled clinical trial, and those unwilling to enroll, were eligible to participate in the observational arm of the study. The planned average follow-up period was 9 years. The remainder of the discussion of the WHI will focus on the HRT study.

Objectives

The primary objective of the HRT study was prevention of CHD events, defined as acute nonfatal myocardial infarction (MI), (definite or probable) requiring hospitalization, silent MI, or CHD death.[4,9] Secondary objectives were measures of other CVD, hip or other fractures, and endometrial (in women with a uterus), colorectal, or other cancers. Invasive breast cancer was identified as a primary adverse outcome. A global index, defined as the earliest occurrence of CHD events, invasive breast cancer, stroke, pulmonary embolism (PE), endometrial cancer (in women with a uterus), colorectal cancer, hip fracture, or death due to other causes, was used to measure the relative risk to benefit ratio of HRT.

Results

Estrogen Plus Progestin in Women With a Uterus

A total of 16,608 women were included in the estrogen plus progestin (N = 8,506) versus placebo (N = 8,102) study.[4] Baseline characteristics were similar between treatment groups. The mean age was 63.3 years at baseline, 84% of participants were Caucasian, and mean body mass index was 28.5 kg/m^2. The prevalence of CVD was low, affecting 7.7% of the population, and participants were not considered to be at increased risk for breast cancer.

This arm of the study was stopped early, after an average follow-up of 5.2 years, because an interim (10th semiannual) analysis revealed that the risks of estrogen plus progestin in women with a uterus outweighed the benefits.[4] Specifically, the stopping boundary for invasive breast cancer had been crossed, and the global index suggested overall harm (hazard ratio [HR], 1.15; 95% confidence interval [CI], 1.03-1.28). Final, centrally adjudicated results for select major clinical end points were available and are included in Table 1. Note the significantly increased risks of stroke (primarily nonfatal ischemic stroke), deep venous thrombosis (DVT), PE, and invasive breast cancer, and the significant reductions in risk of invasive colorectal cancer and total and hip fractures.[10-13] The overall risk of CHD was increased in the HRT group relative to the placebo group; however, it did not reach statistical significance (HR, 1.24; 95% CI, 1.00-1.54).[14] Analysis of risk of CHD events by presence (HR, 1.44; 95% CI, 0.77-2.70) or absence (HR, 1.23; 95% CI, 0.97-1.55) of CHD at baseline, defined as a history of MI or revascularization procedure, revealed no significant difference between treatment groups with regard to risk of CHD events ($P = 0.66$).[14] There were no significant between-group differences with respect

TABLE 2 *Relative and Absolute Risks of Major Clinical Outcomes in the Estrogen Alone Substudy of the Women's Health Initiative*[a]

Outcome	Relative Risk (95% Confidence Interval)[b] Estrogen Vs. Placebo	Absolute Risk (No. per 10,000 Person Years)	
		Estrogen (N = 5310)	Placebo (N = 5429)
Coronary heart disease events	0.95 (0.79-1.16)	53	56
Nonfatal myocardial infarction[c]	0.91 (0.73-1.14)	40	43
Coronary heart disease deaths	1.01 (0.71-1.43)	16	16
All strokes	1.37 (1.09-1.73)	45	33
Ischemic stroke	1.55 (1.19-2.01)	38	25
Deep vein thrombosis	1.47 (1.06-2.06)	23	15
Pulmonary embolism	1.37 (0.90-2.07)	14	10
Invasive breast cancer	0.80 (0.62-1.04)	28	34
Colorectal cancer[d]	1.08 (0.75-1.55)	17	16
Total fracture	0.71 (0.64-0.80)	144	197
Hip fracture	0.65 (0.45-0.94)	12	19
Total mortality[d]	1.04 (0.88-1.22)	81	78
Global index[d,e]	1.01 (0.91-1.12)	192	190

[a] *Final, centrally adjudicated data after a mean follow-up of 7.1 years, unless otherwise noted.*
[b] *Nominal (unadjusted) confidence intervals.*
[c] *Includes silent myocardial infarction.*
[d] *Data not centrally adjudicated; mean follow-up = 6.8 years.*
[e] *Global index = the earliest occurrence of CHD events, invasive breast cancer, stroke, pulmonary embolism, colorectal cancer, hip fracture, or death due to other causes.*
Data from References 9, 16-20.

to nonfatal MI, CHD deaths, endometrial cancer, or total mortality (Table 1).[4,14,15]

The imbalance in the risk-to-benefit ratio was identified using time-trend analyses. The cumulative hazards revealed that the between-group differences in CHD began shortly after randomization and remained consistent throughout the follow-up period.[4] Differences in stroke, on the other hand, did not become apparent until 1 to 2 years after randomization, and continued to diverge throughout the follow-up period. Differences in the risk for PE began shortly after randomization and continued to diverge throughout the follow-up period. The risk for breast cancer was similar in both treatment groups during the first 4 years of the study, after which time the risk of breast cancer increased at a much faster rate in the HRT group than in the placebo group. Differences in favor of HRT in reductions in the risks for hip fractures and colorectal cancer became apparent shortly after randomization and after 3 years, respectively. Collectively, these results led the authors of the WHI to conclude that long-term HRT should not be initiated or continued for the primary prevention of CHD in postmenopausal women with an intact uterus.[4]

Estrogen Alone in Women Without a Uterus

A total of 10,739 women were included in the estrogen alone (N = 5,310) versus placebo (N = 5,429) study.[9] Baseline characteristics were similar between treatment groups. The mean age was 63.6 years at baseline, 75% of participants were Caucasian, and mean body mass index was 30.1 kg/m^2. Patients were considered to be at average risk for CHD and breast cancer.

As in the HRT arm in women with a uterus, the HRT arm in women without a uterus was also stopped early, after an average follow-up of 6.8 years.[9] However, unlike the HRT arm in women with a uterus, which was discontinued at the recommendation of the independent data and safety monitoring board because of health risks,[4] the HRT arm in women without a uterus was discontinued by the National Institutes of Health (NIH), the sponsors of the study.[9] It was believed that no additional benefits or risks of estrogen therapy would be demonstrated if the study continued for the final planned year, and it was not considered acceptable to subject the study participants to the increased risk of stroke that had been identified during earlier interim analyses.[9]

Estrogen therapy was associated with significant increases in the risks of stroke (primarily nonfatal ischemic stroke) and DVT (Table 2).[16,17] Nonsignificant increases in the risks of PE and colorectal cancer were also observed,[9,16] as were significant reductions in total fractures and hip fractures[18] and nonsignificant reductions in the risks for CHD and breast cancer.[19,20] The reduced risk of breast cancer in women treated with estrogen was an unexpected finding and contrasts the findings in women with a uterus who were treated with estrogen plus progestin. The HR for the global index, a measure of the relative risk-to-benefit ratio, was 1.01 (95% CI, 0.91-1.12), indicating neutrality.[9] Interestingly, the HR for CHD was slightly higher in the estrogen arm than in the placebo arm

early in the study but gradually declined with time.[19] By the end of the follow-up period, the HR for CHD was lower in the estrogen group than in the placebo group; however, this difference did not reach statistical significance at any time during the study. Analysis of risk for CHD events by presence (HR, 1.12; 95% CI, 0.69-1.80) or absence (HR, 0.93; 95% CI, 0.75-1.15) of CHD at baseline revealed no significant difference in the risk of future CHD events ($P=0.33$).[19]

Between-group differences in the cumulative HRs for stroke and hip fracture became apparent early in the study, whereas differences in the cumulative HR for breast cancer became apparent at 2 years; all continued to diverge throughout the follow-up period.[9] No apparent between-group differences in cumulative HRs were observed for CHD, PE, colorectal cancer, death, or the global index. The statistical analysis did not show any benefit from HRT in terms of CHD; therefore, as in the estrogen plus progestin arm in women with an intact uterus, the authors concluded that long-term estrogen therapy should not be initiated or continued for the primary prevention of CHD in postmenopausal women without a uterus.[9]

Clinical Implications

The results of the WHI led health care providers and patients to change the way they prescribed and used HRT, respectively. These changes occurred despite continued recommendation from professional societies, such as the American Association of Clinical Endocrinologists (AACE)[1] and the North American Menopause Society (NAMS),[2,3] managed care organizations,[21] and FDA-approved product labeling of available therapies to use HRT at the lowest effective dose for the shortest duration possible for the management of moderate-to-severe menopause-associated VMS. These organizations recognized that the patient population included in the WHI was not representative of the typical menopause patient, as women in the WHI were older and postmenopausal. Thus, they did not change their recommendations for HRT for the management of VMS in menopausal women based on the results of the WHI study; however, they did caution against the use of HRT as a primary or secondary CHD prevention strategy.[1,2,21]

Major changes were evident in the preferences and prescribing patterns of health care providers following release of the results from the WHI. Results from several small surveys revealed a more conservative approach to HRT among family practitioners and internists than among gynecologists following the WHI.[22,23] In fact, gynecologists, especially those who completed their residency prior to 1994, expressed a high degree of skepticism about the results of the WHI, held a stronger belief in the benefits of HRT, and were less concerned about the risks of HRT.[22,24,25]

A nation wide survey that combined results from the National Ambulatory Medical Care Survey and the National Hospital Ambulatory Medical Care Survey evaluated changes over time in the number and rate of physicians' office visits that included a prescription for HRT between 2001 and 2003.[26] Office visits for HRT among women aged 40 years and older declined significantly between 2001 and 2003, from a high of 41.4% in 2001, to 32.7% in 2002, to 26.0% in 2003 ($P=0.002$).[26] These changes translate into a reduction in the number of office visits during which a prescription for HRT was written from 26.5 million visits in 2001 to 16.9 million visits in 2003 ($P<0.002$). Similar findings were observed in an evaluation of the National Disease and Therapeutic Index database, which tracks the number of physicians' office visits during which a prescription is written, and the National Prescription Audit database, which tracks the number of prescriptions filled at retail pharmacies. Between 1995 and 2001, there was a gradual increase in the number of prescriptions of HRT dispensed in the United States from 58.3 million in 1995 to 91.0 million in 2001.[27] Between 2001 and 2003, however, the annual number of prescriptions for HRT fell to 56.9 million (2003 figures annualized based on January 2003-July 2003 data), a more dramatic decline than the increase observed during the preceding 6 years. Prescription data from 5 health maintenance organizations (HMOs) showed that the decline in the total number of prescriptions for HRT between September 1999-June 2002 and December 2002, was a consequence of both a significant increase in prescription discontinuations and a significant decrease in the number of new prescriptions written with changes becoming evident immediately following publication of the preliminary results of the WHI.[28]

The widespread release of the results of the WHI included patients as a target audience. Study results were disseminated via NIH press releases (www.nhlbi.nih.gov/whi/press_releases.htm), patient education materials developed for managed care members,[21] mass media, and health care providers.[29] Results from a telephone survey of 670 women in 1 HMO revealed that most patients (93%) had heard about the WHI findings, but less than one-quarter (23%) actually knew what the study results were.[29] Despite the apparent lack of understanding of the benefits and risks of HRT, 56% of women surveyed attempted to discontinue their HRT within 6 to 8 months after study publication. Patients whose HRT was prescribed by their gynecologist were more likely to attempt to discontinue therapy (59.6%) than those whose HRT was prescribed by their primary care provider (49.3%).[29] This finding conflicts with the more conservative view of HRT among family practitioners and internists than among gynecologists described above.[22,23]

Study Limitations

The WHI investigators acknowledge the evaluation of a single dose of a single formulation of estrogen (0.625 mg conjugated equine estrogen), with or without a single dose of progestin (2.5 mg medroxyprogesterone acetate), via a single route of administration (oral) as a limitation of the WHI.[4,9] Thus, the study design does not allow the results to be extrapolated to other doses, formulations, or routes of administration of estrogen (with or without progestin). Other limitations identified by the authors include higher than expected drop-in and drop-out rates, early discontinuation of the 2 study arms, which preclude accurate assessment of long-term

effects, and the inability to distinguish the effects of estrogen from those of progestin in the combination therapy study in women with a uterus.

A major limitation of the study design was the patient population. The WHI enrolled older, postmenopausal women, with an average age of 63 years at baseline.[4,9] This patient population is thought to be at increased risk for subclinical CHD relative to younger, perimenopausal women, a hypothesis supported by the presence of increasingly higher prevalence of CHD risk factors and pre-existing CVD with increasing age and years since menopause in WHI participants.[30] Subgroup analyses of risk of CHD events by age revealed a nonsignificant trend toward a reduction in risk among women aged 50 to 59 years treated with estrogen alone (HR, 0.63; 95% CI, 0.36-1.08), with less of a benefit in women aged 60 to 69 years (HR, 0.94; 95% CI, 0.71-1.24); the highest risk was in women aged 70 to 79 years (HR, 1.11; 95% CI, 0.82-1.52) (*P* value for interaction = 0.07).[19] Although this trend was not mirrored in women treated with estrogen plus progestin (HR, 1.27, 1.05, and 1.44, respectively), a similar trend was observed when women were grouped by years since menopause.[14] In women treated with estrogen plus progestin who experienced menopause within the previous 10 years, the HR for CHD events was 0.89; in women who had experienced menopause 10 to 19 years ago, or 20 or more years ago, the risk was higher (HR, 1.22 and 1.71, respectively) (*P* value for interaction = 0.33).[14] A secondary analysis of the WHI combined both arms of the HRT study and evaluated the risk of CHD in relation to age and years since menopause.[30] Similar, nonsignificant trends were evident in the combined analysis. When analyzed by age at baseline, HRs were 0.93, 0.98, and 1.26, respectively, in women aged 50 to 59 years, 60 to 69 years, and 70 to 79 years (*P* value for trend = 0.16). When analyzed by years since menopause (< 10, 10-19, ≥ 20), HRs were 0.76, 1.10, and 1.28, respectively (*P* value for trend = 0.02). Although only studying a surrogate marker and not a clinical outcome, results from the WHI Coronary-Artery Calcium analysis suggest a benefit of estrogen therapy in preventing heart disease in women aged 50 to 59 years at study enrollment.[31] Coronary calcification correlates well with the extent of underlying atherosclerosis and the risk of future cardiovascular events.[32] Treatment lasted a mean of 7.4 years and imaging occurred at a mean of 1.3 years after the end of the trial.[31] Women randomly assigned to receive estrogen had significantly less coronary-artery calcification than women randomly assigned to placebo.[31] Collectively, these data support the need to weigh the benefits and risks of HRT in menopausal women based on patient-specific characteristics, including age and time since menopause. They do not support a one-size-fits-all approach to HRT.

Management of Conditions Related to Menopause

The role of HRT in the management of menopause-associated VMS has been reviewed. Briefly, the AACE and NAMS recommend the use of estrogen (in women without a uterus) or estrogen plus progestin (in women with a uterus) at the lowest effective dose for the shortest duration possible for the management of VMS.[1,2] They do not advocate the use of HRT for the primary or secondary prevention of CHD. CVD and osteoporosis are common conditions in menopausal and postmenopausal women, and the menopausal transition creates a unique opportunity to initiate preventive or treatment strategies in these women.

Cardiovascular Disease

CVD is the leading cause of death among women in the United States.[33] By age group, it is the second leading cause of death among women aged 45 to 64 years, second only to cancer, and is the leading cause of death among women aged 65 years and older. Preventive strategies include lifestyle modifications (e.g., diet, weight loss, exercise, smoking cessation), and appropriate management of underlying risk factors, such as obesity, especially abdominal obesity, hypertension, dyslipidemia, insulin resistance, and diabetes through nonpharmacologic and pharmacologic intervention, as necessary.[1,34] The AACE suggests that HRT, specifically estrogen therapy, may offer some clinical benefit in women without CHD who are in the early stages of menopause (i.e., within 5 years of symptom onset), but should not be used in older postmenopausal women (i.e, those whose symptom onset occurred ≥ 5 years ago) or in women with pre-existing CHD.[1]

Osteoporosis

Osteoporotic bone loss is a common occurrence that affects an estimated 55% of Americans aged over 50 years.[35] Osteoporosis affects women at a disproportionately higher rate than men, with women accounting for 80% of cases.[35] Osteoporosis increases the propensity for falls and fractures, the latter of which are estimated to occur 3 times as frequently in women as in men.[36]

Both the AACE and the NAMS advocate a diet rich in calcium and vitamin D, regular weight-bearing exercise, smoking cessation, limited alcohol consumption, and in certain patients, a bisphosphonate (e.g., alendronate, risedronate, ibandronate) for the prevention of osteoporosis.[37,38] Results from the WHI showed that calcium and vitamin D supplementation in healthy postmenopausal women resulted in a small but significant improvement in hip bone density; however, supplementation did not significantly reduce the incidence of hip fractures.[39] Furthermore, supplementation increased the risk of kidney stones.[39] Pharmacologic treatment options include HRT, bisphosphonates, selective estrogen receptor modulators (e.g., raloxifene), salmon calcitonin, and teriparatide (recombinant human parathyroid hormone). Readers are encouraged to consult the AACE and NAMS guidelines for a detailed explanation of benefits, risk, and dosing considerations for these agents.

Opportunities for Improving Quality of Care in Perimenopausal Women

The uncertainty about the benefits and risks of HRT that followed the initial release of the WHI results may be confounded by the

more recent finding that the risks of HRT may correlate with age of onset of menopausal symptoms or years since menopause. Although patient education initiatives followed publication of the initial WHI results, it is clear that continued education on VMS and its management is needed. In fact, in the telephone survey of 670 women described earlier, only 57% considered the quality of information they received about the WHI to be good and only 23% actually knew what the study results were.[29] However, the need for improved education about HRT was apparent well before the WHI study results were published. In fact, the Commission on Women's Health, a 5-year initiative aimed at increasing public awareness of women's health issues and the quality of health care, found that only 34% of women aged 50 years and older were receiving HRT in 1998, an increase from 23% in 1993.[40] This undertreatment was accompanied by a lack of appropriate counseling on HRT. In fact, of the 2,850 women surveyed, only 38% reported receiving counseling from their health care provider on HRT within the previous year. These results support the findings of a smaller Gallup survey of 833 women aged 45 to 60 years conducted in 1993, in which only 36% of respondents reported receiving the majority of their information about menopause from their physician and 69% reported being somewhat or very satisfied with the information they received.[41] Of the women surveyed, 84% reported that their physician had discussed HRT with them, but only 42% reported using HRT to relieve menopausal symptoms. Results from the Management of Menopause survey in 2000 provided further evidence of the need to provide additional counseling on the treatment of menopause, as evidenced by a 73% exposure score, a 52% breadth score, and a 33% quality score.[42]

Not only does menopause present an opportunity for primary care practitioners and gynecologists to educate women on the symptoms and management of typical menopause-associated symptoms, such as VMS, it also presents an opportunity to educate women on preventive care strategies for CVD, osteoporosis, and diet and weight management. The fact that breast cancer risk only developed after 4 years of treatment in the estrogen plus progestin arm of the WHI and not at all in the estrogen alone arm should put some patients and health care providers at ease about using HRT for short periods to prevent VMS. The need for improved education on these topics was demonstrated in the Commission on Women's Health Initiative described in the previous text. In 1998, a mere 36% of women surveyed reported being very familiar with osteoporosis compared with 30% of women surveyed in 1993.[40] The number of women who reported receiving counseling on exercise, diet/weight, calcium intake, and smoking cessation from their physician within the previous year averaged 49%, 46%, 41%, and 29%, respectively, in 1998. In 2004, there was only a 19% compliance rate among Medicare plans with the HEDIS measure of osteoporosis management in women aged 67 years and older.[43] This metric required a bone mineral density test or a prescription for an agent for osteoporosis in women who had had a fracture.[43] Thus, quality of care improvements, including improved counseling and early implementation of preventive and treatment strategies, may lead to improvements in several HEDIS measures, including osteoporosis management in women who had a fracture, fall risk management, osteoporosis testing in older women, control of high blood pressure, cholesterol management for patients with cardiovascular conditions, medical assistance with smoking cessation, and physical activity in older adults.[44]

Conclusions

Publication of the preliminary results of the WHI led to significant changes in the management of menopausal symptoms over the past several years. Recent analyses challenge the preliminary results, citing differences in risk of CHD based on age and years since menopause. Additional studies are warranted to determine the effect of HRT on CHD risk in those women beginning menopause in whom HRT is considered the standard of care for treatment of moderate-to-severe VMS. Women of menopausal age are at increased risk for CVD and osteoporosis, and data suggest that the quality of care for all 3 of these conditions is lacking. Thus, menopause presents a unique opportunity for health care providers to channel women seeking treatment for their menopausal symptoms into a preventive or treatment program for CVD and/or osteoporosis, as necessary. Such a proactive approach may lead to improvements in several HEDIS measures for CVD and osteoporosis.

DISCLOSURES

Andrea Lukes discloses that there was no financial relationship or financial interest relating to the topic of this activity. Lukes has received research/grant support from CDC/APTR, Xanodyne, AMS, Ethicon, and Cytyc, and is a consultant to Xanodyne, AMS, Cytyc, Daiichi, and Conceptus. Lukes was responsible for the entire study concept and design of this article. She performed all the data collection, data interpretation, writing, and revision of this article.

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