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Effects of estrogen with and without progestin on urinary incontinence

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Abstract

Context: Menopausal hormone therapy has long been credited with many benefits beyond the indications of relieving hot flashes, night sweats, and vaginal dryness, and it is often prescribed to treat urinary incontinence (UI).

Objective: To assess the effects of menopausal hormone therapy on the incidence and severity of symptoms of stress, urge, and mixed UI in healthy postmenopausal women.

Design, setting, and participants: Women's Health Initiative multicenter double-blind, placebo-controlled, randomized clinical trials of menopausal hormone therapy in 27,347 postmenopausal women aged 50 to 79 years enrolled between 1993 and 1998, for whom UI symptoms were known in 23,296 participants at baseline and 1 year.

Interventions: Women were randomized based on hysterectomy status to active treatment or placebo in either the estrogen plus progestin (E + P) or estrogen alone trials. The E + P hormones were 0.625 mg/d of conjugated equine estrogen plus 2.5 mg/d of medroxyprogesterone acetate (CEE + MPA); estrogen alone consisted of 0.625 mg/d of conjugated equine estrogen (CEE). There were 8506 participants who received CEE + MPA (8102 who received placebo) and 5310 who received CEE alone (5429 who received placebo).

Main outcome measures: Incident UI at 1 year among women without UI at baseline and severity of UI at 1 year among women who had UI at baseline.

Results: Menopausal hormone therapy increased the incidence of all types of UI at 1 year among women who were continent at baseline. The risk was highest for stress UI (CEE + MPA: relative risk [RR], 1.87 [95% confidence interval {CI}, 1.61-2.18]; CEE alone: RR, 2.15 [95% CI, 1.77-2.62]), followed by mixed UI (CEE + MPA: RR, 1.49 [95% CI, 1.10-2.01]; CEE alone: RR, 1.79 [95% CI, 1.26-2.53]). The combination of CEE + MPA had no significant effect on developing urge UI (RR, 1.15; 95% CI, 0.99-1.34), but CEE alone increased the risk (RR, 1.32; 95% CI, 1.10-1.58). Among women experiencing UI at baseline, frequency worsened in both trials (CEE + MPA: RR, 1.38 [95% CI, 1.28-1.49]; CEE alone: RR, 1.47 [95% CI, 1.35-1.61]). Amount of UI worsened at 1 year in both trials (CEE + MPA: RR, 1.20 [95% CI, 1.06-1.36]; CEE alone: RR, 1.59 [95% CI, 1.39-1.82]). Women receiving menopausal hormone therapy were more likely to report that UI limited their daily activities (CEE + MPA: RR, 1.18 [95% CI, 1.06-1.32]; CEE alone: RR, 1.29 [95% CI, 1.15-1.45]) and bothered or disturbed them (CEE + MPA: RR, 1.22 [95% CI, 1.13-1.32]; CEE alone: RR, 1.50 [95% CI, 1.37-1.65]) at 1 year.

Conclusions: Conjugated equine estrogen alone and CEE + MPA increased the risk of UI among continent women and worsened the characteristics of UI among symptomatic women after 1 year.

Conjugated equine estrogen with or without progestin should not be prescribed for the prevention or relief of UI.

Comment in

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