The specialist authority for menopause & post reproductive health

British Menopause Society

Primary prevention of coronary heart disease in women

The British Menopause Society (BMS) is the specialist authority for menopause and post reproductive health in the UK. The BMS educates, informs and guides healthcare professionals, working in both primary and secondary care, on menopause and all aspects of post reproductive health.

BMS consensus statements are developed by the BMS medical advisory council to address key disorders and controversial topics relating to menopause and post reproductive health. They reflect new studies and recent medical and scientific information from articles published in professional journals, together with informal consensus.

The consensus statements are evidence-based, comprehensively referenced and peer reviewed and they are updated when guidance changes and/or new data becomes available.

Summary

Coronary heart disease (CHD) is a leading cause of death in women. Observational studies have consistently shown estrogen to help prevent CHD in postmenopausal women. The large randomized controlled Women's Health Initiative (WHI) trial initially did not confirm these observational findings. However, further analyses of the WHI study as well as meta-analyses of randomised clinical trials of hormone replacement therapy (HRT) and of the observational Nurses' Health Study have now found that the timing of onset of HRT use is important and that estrogen may have an important protective role in CHD, particularly in women initiating treatment below age 60 years. This BMS consensus statement will examine the evidence regarding HRT and non-estrogen therapies (lipid lowering agents, aspirin, antihypertensives, antidiabetic medications, SERMs) as well as diet, lifestyle and smoking cessation in the primary prevention of CHD in women.

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Introduction

Coronary heart disease (CHD) is the leading cause of death in women.[1] In the UK, CHD accounts for 8% of all female deaths. Although CHD is rarely the cause of death before the sixth decade it is the most common cause after the age of 60 years. With an expanding and ageing female population CHD is an important health issue. Pharmacological and non-pharmacological prevention strategies will be examined.

Estrogen-based hormone replacement therapy

Until the end of the 20th century estrogen was thought to protect against CHD.[2] Many cohort studies showed that HRT was associated with a 40-50% reduction in the incidence of CHD. The effects were the same for both estrogen alone and combined HRT. However, the randomised controlled Women's Health Initiative (WHI) trials initially did not confirm the findings of the observational studies.[3-8]

WHI used conjugated equine estrogens 0.625 mg daily in hysterectomised women with the addition of medroxyprogesterone acetate (MPA) 2.5 mg in non-hysterectomised women aged 50-79 years. However, it has become apparent that there are differences between these HRT regimens, and that the timing of initiating HRT use may be crucial as may the dose at initiation and type of hormones used.

Combined versus estrogen alone HRT

WHI found an early, albeit transient, increase in coronary events in the combined but not the estrogen alone arm. Overall, there was no significant effect of HRT.[4] The WHI used MPA as the progestogen and there is little information about other progestogens which may have different effects on lipids, glucose, insulin metabolism and coagulation. Analysis of the WHI estrogen-alone study showed a non-significant reduction in CHD which became a statistically significant reduction in women initiating treatment below age 60 years during the long term follow up of the trial.[9] Furthermore, in the observational Nurses' Health Study, women beginning HRT near menopause had a significantly reduced risk of CHD (RR = 0.66, 95% CI 0.54-0.80 for estrogen alone; RR = 0.72, 95% CI 0.56-0.92 for estrogen with progestogen).[10]

Oral versus transdermal therapy

Whether transdermal is better than oral delivery on CHD risk is uncertain.[11,12]

Estrogen dose

A possible explanation for early cardiovascular harm in the WHI study is an increase in coagulation activation or abnormal cardiovascular remodelling. Both of these effects are dose dependent. Thus, although estrogen has the potential to cause vascular benefit (as shown in observational and animal studies), high doses have the potential to cause vascular harm.[2] The results combining the two arms of WHI are consistent with this hypothesis: reduced risk was found in women aged 50–69 years and increased risk in older women.[8] Thus the dose of estrogen used in WHI may have been inappropriately high for older women and does not reflect UK practice where dose at initiation is usually decreased with increasing age.

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Timing of treatment

Women in the WHI who started combined HRT within 10 years of the menopause had a lower risk of CHD than women who started later.[8] Combining the two HRT arms found that women who initiated hormone therapy closer to menopause tended to have reduced CHD risk compared with the increase in CHD risk among women more distant from menopause.

Stratification of the WHI estrogen-alone study by age showed a significant reduction in a composite of coronary events and procedures in the 50-59 years age group[5,6] and an eventual significant reduction in events compared with placebo during long term follow up.[9] Subsequent meta-analyses of randomised controlled trials have confirmed a significant reduction in CHD events in those women initiating HRT below age 60 years[13] or within 10 years of menopause onset.[14] No reductions, but also no increases, were shown in those women initiating HRT above age 60 years or beyond 10 years postmenopause. Furthermore, significant reductions in CHD death have also been observed in women initiating HRT below 60 years, with non-significant reductions in those initiating above 60 years of age.[15] A longer duration randomised clinical trial of HRT or no treatment in women within the very early postmenopause showed a significant reduction with HRT of a composite endpoint of myocardial infarction, death and admission to hospital with heart failure.[16] No increase in stroke, venous thromboembolism or any form of cancer was seen during the 10 years of HRT use.

A post-hoc WHI sub-study in hysterectomized women aged below 60 years compared calcified plaque in the coronary arteries, a marker for atheromatous-plaque burden which is predictive of future risk of cardiovascular events.[17] Mean coronary-artery calcium score after trial completion was lower among women receiving estrogen than among those receiving placebo. However, there were no baseline measurements and no measurements carried out in other age groups, so the value of these data is limited.

A placebo controlled randomised trial of HRT initiated within 6 years of onset of menopause compared with initiation beyond 10 years postmenopause followed changes in carotid artery intima-media thickness over 6 years. There was a significant reduction seen with HRT in the early starters but progression of atheroma in the late starters was the same as with placebo.[18]

Premature Ovarian Insufficiency (POI) and early menopause

Women with untreated POI or early menopause are at increased risk of CHD.[12] Regulatory bodies recommend the use of HRT in POI at least up to the average age of the natural menopause.[19]

Non-estrogen based pharmacological therapies

Lipid lowering agents

Statins

Statins (3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors), were proposed as the mainstay for primary prevention of coronary heart disease in women.[20,21] A meta-analysis of six trials, including a total of 11,435 women without cardiovascular disease, assessed the effects of lipid-lowering medications. Lipid lowering did not reduce total mortality (RR, 0.95; 95% confidence interval [CI], 0.62-1.46), CHD mortality (RR, 1.07; 95% CI, 0.47-2.40), nonfatal

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myocardial infarction (RR, 0.61; 95% CI, 0.22-1.68), revascularization (RR, 0.87; 95% CI, 0.33-2.31), or CHD events (RR, 0.87; 95% CI, 0.69-1.09).[22] Furthermore, statins have been associated with increases in type 2 diabetes mellitus.[23] However, a more recent meta-analysis has concluded that there is in fact evidence for a benefit of statins for the primary prevention of CHD in women.[24]

Fibrates

Fibrates have been studied in coronary heart disease since the 1970s. The results have been confusing – starting with initial benefits in small studies but contradicted by either minimal benefits in the Coronary Drug Project or adverse non-cardiovascular effects in the World Health Organization Clofibrate Study.[25] More recently a Cochrane review found only a minor effect in primary prevention (absolute risk reduction <1%).[26] Whilst lipid-lowering is clearly essential in the management of women with established CHD, its role in primary prevention is less well established. Nevertheless, it would seem prudent to use these agents in women with hypercholesterolaemia identified as being at increased CHD risk.

Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors

PCSK9 is an enzyme which is involved in lipoprotein homeostasis. Inhibition of this enzyme leads to profound falls in LDL cholesterol. Monoclonal antibodies have been developed which effectively lower LDL cholesterol by up to 70% and are accompanied by reductions in atherosclerotic cardiovascular events.[27] However, the treatment has to be administered by subcutaneous injection every 2 or 4 weeks and is currently very expensive.

Aspirin

Prophylactic use of aspirin for primary prevention of coronary heart disease has been recommended. However, the Women's Health Study (WHS) of 39,876 healthy women randomized to aspirin 100 mg every other day or placebo for 10 years showed a null effect of aspirin on the primary trial endpoint of non-fatal myocardial infarction, nonfatal stroke, or cardiovascular death (RR 0.91, 95% CI: 0.80-1.03).[28] Overall mortality and cardiovascular death from any cause were also unaffected by aspirin. Within the null finding was no effect on fatal or non-fatal myocardial infarction (RR 1.02, 95% CI: 0.84-1.25). A more recent appraisal found that the risks of significant bleeding in the stomach and brain outweigh any possible benefit for primary CHD prevention.[29] There is therefore insufficient evidence to recommend low-dose aspirin use for primary CHD prevention in women, although it is recommended in women with established CHD and possibly in women considered to be at increased risk for stroke. A possible reduction in colorectal carcinoma with aspirin awaits further evaluation.

Antihypertensives

Meta-analysis of prospective data on over one million adults (40-69 years) has shown that an increase of 20 mm Hg systolic or 10 mm Hg diastolic in usual blood pressure doubles the death from CHD.[30] Hypertension is the most prevalent chronic disorder, and systolic blood pressure increases after menopause.[31] At the age of 60 years over 50% of women are hypertensive. Lifestyle modification and pharmacotherapy are the main therapeutic options to decrease morbidity and mortality.

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Treating hypertension reduces the risk of CHD. Some HRT combinations, such as estradiol and drospirenone, reduce blood pressure, and their use could be considered in hypertensive women requiring HRT.[32]

Anti-diabetic treatments

Diabetes mellitus is a major risk factor for CHD.[33] The prevalence of diabetes increases sharply with increasing age and is around 6% overall in the UK population, with about 90% of these cases being type 2 diabetics. Lifestyle measures are an extremely important part of diabetes management. Of the therapeutic options, metformin is by far the most commonly prescribed drug for type 2 diabetes whilst the use of sulfonylureas has diminished considerably.[34] Newer agents have become available, including gliptins, GLP-1 analogues and sodium-glucose co-transporter 2 (SGLT2) inhibitors. Both the GLP-1 analogues and the SGLT2 inhibitors have been shown to have favourable effects of cardiovascular outcomes.[35] HRT has also been shown to reduce the incidence of new diabetes in postmenopausal women[36,37] Thus in diabetics, current prevention of CHD is based on management of established cardiovascular risk factors with lifestyle adjustments, anti-diabetic drugs, lipid lowering agents and antihypertensive therapy.

HRT cannot at present be recommended solely for the prevention of diabetes, but this effect should be considered in the management of postmenopausal women considered at increased risk for diabetes development.[38]

Preventing metabolic syndrome

The metabolic syndrome is a combination of metabolic disturbances, including abdominal obesity, dyslipidaemia, glucose intolerance and hypertension.[39] The coexistence of three or more of these factors constitutes the syndrome and increases the probability of developing diabetes, as well as increasing the risk of coronary and cardiovascular mortality. Menopause itself results in a metabolic syndrome and thus will contribute to a worsening of any pre-existing features of the syndrome.[40] Thus metabolic syndrome needs to be treated, and HRT should be considered a part of this management.

Selective estrogen receptor modulators: raloxifene

Raloxifene is a Selective Estrogen Receptor Modulator (SERM). These compounds possess estrogenic actions in certain tissues and anti-estrogenic actions in others. It was thought that it could be cardio-protective from its effects on lipids, but the Raloxifene Use for the Heart (RUTH) study found that it did not reduce the risk of coronary heart disease overall, and it increased the risk of fatal stroke and venous thromboembolism.[41] However, age stratification showed that there was a reduction in CHD in those initiating raloxifene below age 60 years.[42] Another SERM, lasofoxifene, was also shown to reduce CHD and stroke events in postmenopausal osteoporotic women, but there was an increased risk of venous thromboembolism.[43]

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Diet and lifestyle

Body mass index and obesity

One of the outcomes noted in the Nurses' Health Study was that there is a gradient of coronary risk for body mass index (BMI), with the heaviest category of women having a three-fold risk for CHD compared with lean women.[44] Much evidence has focused on the distribution of fat with a more android morphology (apple) representing a higher cardiac risk than a more gynoid (pear) shape. Thus a waist circumference of 76.2 cm (30 in) or more was associated with more than a 2-fold higher risk of CHD.

Diet

Low rates of coronary heart disease in the Mediterranean region were noted many years ago.[45] This has been attributed to the Mediterranean diet characterised by a high intake of vegetables, legumes, fruits and cereals (in the past largely unrefined); a moderate to high intake of fish; a low intake of saturated lipids but a high intake of unsaturated lipids, particularly olive oil; a low to moderate intake of dairy products, mostly cheese and yogurt; a low intake of meat; and a modest intake of ethanol, mostly as wine. Adherence to the diet reduces risk of death from coronary heart disease.[46] The INTERHEART case control study showed that a diet containing fruit and vegetables and regular alcohol consumption was associated with a reduced risk of myocardial infarction.[47]

Smoking

Smoking increases the risk of myocardial infarction. Compared to non-smokers, female current smokers have a relative risk of myocardial infarction of 2.24 (range 1.85-2.71) which is greater than that seen in men.[48] The INTERHEART study estimated that 29% of heart attack cases in Western Europe are due to smoking and smokers and former smokers are at almost twice the risk of a heart attack compared to never smokers.[47] Indeed it is now known that smoking just one cigarette per day substantially increases the risk of cardiovascular disease.[49]

Exercise and physical activity

Lower fitness levels are associated with 4.7-fold increased risk for CHD.[50] European guidelines recommend >150 minutes of endurance exercise training per week at moderate to vigorous intensity.[51] Walking appears to be as effective as vigorous exercise in women.[52]

Summary practice points

- Estrogen may have a protective role in CHD prevention especially if initiated in women below age 60 years or within 10 years of onset of menopause.
- Healthcare professionals should consider the potential benefits of estrogen for the reduction of all-cause mortality and prevention of CHD in their decision making when initiating HRT in healthy postmenopausal women below age 60 years or within 10 years of menopause onset. Use of HRT solely for primary prevention of CHD is off label.
- Women with POI or early menopause should take estrogen to reduce the risk of CHD.
- Lipid-lowering agents are probably beneficial in primary prevention.

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- Aspirin cannot be recommended for primary prevention of CHD but may protect against stroke.
- Treating hypertension reduces the risk of CHD.
- In diabetics, prevention of CHD is based on management of established cardiovascular risk factors through both lifestyle measures and pharmacotherapy.
- In metabolic syndrome, prevention of CHD is based on management of established cardiovascular risk factors through both lifestyle measures and pharmacotherapy.
- Stopping smoking, reducing obesity, improving diet and undertaking regular exercise are key lifestyle measures to reduce coronary heart disease.

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Author: John Stevenson in collaboration with the medical advisory council of the British Menopause Society.

REVIEWED: MARCH 2026

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