Emerging vascular risk factors in women: Any differences from men?

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The incidence and severity of both traditional and emerging cardiovascular disease (CVD) risk factors as well as the response to treatment may differ between genders. In this narrative review, several emerging CVD risk factors (i.e. inflammatory and haemostatic markers, endothelial dysfunction, homocysteine, lipid disorders, microalbuminuria/proteinuria, coronary artery calcium score, arterial stiffness, periodontitis, inflammatory bowel syndrome, obstructive sleep apnea, impaired glucose metabolism, metabolic syndrome and non-alcoholic fatty liver disease) are discussed in the context of gender differences.

Overall, women are more likely to have higher C-reactive protein and adiponectin levels as well as a higher prevalence of metabolic syndrome and non-alcoholic steatohepatitis compared with men. In contrast, men have greater tumor necrosis factor-alpha, homocysteine and uric acid levels as well as higher coronary artery calcium score than women. Furthermore, arterial stiffness, obstructive sleep apnea syndrome, non-alcoholic fatty liver disease, progression of renal dysfunction and impaired fasting glucose are more prevalent in men. Men are also reported to have smaller low-density lipoprotein particles than women, whereas men and postmenopausal women have higher levels of serum cholesterol and triglyceride sub-classes compared with premenopausal women.

Hormone replacement therapy (HRT) may also affect vascular risk in women. Briefly, postmenopausal women have a higher CVD risk
compared with premenopausal women. Estrogens have been reported to exert protective vascular effects in animal and observational but randomized clinical trials did not report such effects in older women, even suggesting the possibility of an increased CVD risk in this setting, especially with combined estrogen plus progestin therapy. Therefore, early initiation of HRT in perimenopausal women is important in order to gain maximum benefit with less risk. Overall, the decision to initiate HRT should be individualized based on the risk/benefit balance as HRT type, duration, administration route and dose may differentially affect CVD markers. HRT should not be administered for CVD prevention.

Further larger prospective studies are needed to establish these gender-specific relationships. Current data should be taken into consideration when assessing and treating CVD risk in women.


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