EARLY MENOPAUSE: CAUSES AND CONSEQUENCES FOR WOMEN'S HEALTH

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Abstract. *This article examines the etiology of premature menopause and analyzes its multifaceted impact on women's health. Through a comprehensive literature review of international research, the paper explores the genetic, autoimmune, iatrogenic, and lifestyle factors that contribute to early menopause onset. The article further investigates the short and long-term health consequences, including increased risks of cardiovascular disease, osteoporosis, cognitive decline, and psychological effects. The findings underscore the importance of early diagnosis, appropriate medical interventions, and holistic management approaches to mitigate health risks associated with premature menopause.*

Keywords*: premature menopause, premature ovarian insufficiency, women's health, cardiovascular risk, osteoporosis, hormone replacement therapy*

Introduction. Menopause represents a natural biological transition in a woman's reproductive life, typically occurring between the ages of 45 and 55. However, approximately 1-4% of women experience premature menopause, defined as the cessation of ovarian function before the age of 40 [1]. This condition, also known as premature ovarian insufficiency (POI), is characterized by amenorrhea, elevated gonadotropin levels, and estrogen deficiency [2]. The early onset of menopause presents significant concerns for women's health due to the prolonged exposure to hormonal deficiencies.

The etiology of premature menopause is complex and multifactorial, involving genetic, autoimmune, iatrogenic, and environmental factors. Understanding these causative factors is crucial for early identification of at-risk individuals and implementation of preventive strategies. Moreover, premature menopause has far-reaching consequences for women's health, affecting not only reproductive capacity but also cardiovascular, skeletal, cognitive, and psychological well-being [3].

Main Section. The pathogenesis of premature menopause involves accelerated follicular atresia or a reduced initial follicular pool, leading to premature depletion of ovarian follicles. Several etiological factors have been identified, though approximately 50-60% of cases remain idiopathic [4].

Genetic factors play a significant role in the development of premature menopause. Familial clustering has been observed, with mothers and sisters of affected women having a 6-fold higher risk of developing premature menopause [5]. Chromosomal

abnormalities, particularly those involving the X chromosome, have been implicated in approximately 10-13% of cases. Turner syndrome (45,X) and fragile X premutation (FMR1) are among the most common genetic causes [1]. Other genetic mutations affecting folliculogenesis, steroidogenesis, and DNA repair mechanisms have also been identified as contributors to premature ovarian failure [6].

Autoimmune disorders represent another significant etiological category, accounting for approximately 20-30% of premature menopause cases [2]. Autoimmune ovarian insufficiency can occur in isolation or as part of polyglandular autoimmune syndromes. Thyroid autoimmunity, particularly Hashimoto's thyroiditis, is frequently associated with premature menopause. Other autoimmune conditions include Addison's disease, rheumatoid arthritis, systemic lupus erythematosus, and myasthenia gravis [3]. The presence of anti-ovarian antibodies, though not diagnostically reliable, has been detected in many women with immune-mediated premature menopause.

Iatrogenic causes constitute a growing proportion of premature menopause cases due to advances in cancer treatments and their improved survival rates. Chemotherapeutic agents, particularly alkylating agents, can induce ovarian toxicity by damaging primordial follicles [7]. The risk varies according to the agent used, cumulative dose, and the woman's age at treatment. Similarly, radiotherapy involving the pelvis can cause direct ovarian damage, with effects dependent on the radiation dose, field, and the woman's age. Surgical interventions, including bilateral oophorectomy and certain gynecological procedures affecting ovarian blood supply, can also lead to premature ovarian failure [5].

Environmental and lifestyle factors have been increasingly recognized as contributors to premature menopause. Cigarette smoking has been consistently associated with earlier menopause, with smokers experiencing menopause 1-2 years earlier than non-smokers [8]. This effect appears dose-dependent and may be mediated through polycyclic aromatic hydrocarbons that accelerate follicular atresia. Other potential environmental factors include exposure to endocrine-disrupting chemicals, pesticides, and industrial compounds, though the evidence remains inconclusive [4]. Nutritional status, body mass index, and physical activity patterns may also influence the timing of menopause, with extreme thinness and intensive exercise potentially contributing to earlier onset [6].

Cardiovascular consequences represent one of the most significant concerns. Estrogen has protective effects on the cardiovascular system through multiple mechanisms, including favorable lipid modifications, vasodilation, and anti-inflammatory properties [9]. Women with premature menopause experience a 50% higher risk of coronary heart disease and a significantly increased risk of stroke compared to women with normal-onset menopause [3]. This risk is inversely proportional to the age at menopause and is particularly pronounced in women experiencing menopause before age 40. Lipid abnormalities, including elevated total cholesterol and LDL levels, reduced HDL levels, and increased triglycerides, develop rapidly following premature menopause [7].

Additionally, increased arterial stiffness, endothelial dysfunction, and higher blood pressure have been documented in women with early menopause.

Skeletal health is profoundly affected by premature estrogen deficiency. Estrogen plays a crucial role in maintaining bone mineral density by inhibiting osteoclast-mediated bone resorption [5]. Women with premature menopause experience accelerated bone loss, leading to a 1.5-3 times higher risk of osteoporosis and fragility fractures [1]. The duration of estrogen deficiency correlates directly with fracture risk, making women with premature menopause particularly vulnerable. Studies have demonstrated significantly lower bone mineral density at multiple skeletal sites in women with premature menopause compared to age-matched controls [8].

The management of premature menopause requires a comprehensive approach. Hormone replacement therapy (HRT) remains the cornerstone of treatment for most women with premature menopause who have no contraindications. Unlike women with natural menopause, for whom HRT decisions involve careful risk-benefit analysis, women with premature menopause are generally advised to use HRT until at least the average age of natural menopause (approximately 51 years) to mitigate health risks. Studies have demonstrated that HRT effectively reduces cardiovascular risk, maintains bone mineral density, alleviates vasomotor and urogenital symptoms, and may offer neuroprotective benefits in women with premature menopause.

Conclusion. Premature menopause represents a significant women's health concern with complex etiology and far-reaching health implications. Genetic factors, autoimmune disorders, iatrogenic causes, and environmental influences contribute to its development, though many cases remain idiopathic. The consequences extend beyond reproductive capacity, affecting cardiovascular, skeletal, neurological, and psychological health due to prolonged estrogen deficiency. The evidence reviewed in this article underscores the importance of early diagnosis, appropriate medical interventions, and holistic management approaches for women experiencing premature menopause. Hormone replacement therapy, when not contraindicated, plays a central role in mitigating health risks and should generally be continued until the average age of natural menopause.

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