



PATHOGENETIC ASPECTS AND PREVENTIVE APPROACHES TO REPRODUCTIVE DISORDERS IN WOMEN OF REPRODUCTIVE AGE LIVING IN CONDITIONS OF SEVERE IODINE DEFICIENCY AND TUBERCULOSIS

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ABSTRACT

Reproductive health in women of reproductive age depends on the integrity of endocrine regulation, immune balance, and metabolic stability. Any long-term disturbance affecting these systems may result in functional or persistent reproductive disorders. Severe iodine deficiency and tuberculosis are among the conditions that exert profound systemic effects and may significantly impair reproductive function, especially when they coexist.

Reproductive health in women of reproductive age depends on the integrity of endocrine regulation, immune balance, and metabolic stability. Any long-term disturbance affecting these systems may result in functional or persistent reproductive disorders. Severe iodine deficiency and tuberculosis are among the conditions that exert profound systemic effects and may significantly impair reproductive function, especially when they coexist.

Iodine is an essential micronutrient required for the synthesis of thyroid hormones, which play a central role in regulating metabolism, energy balance, immune responses, and reproductive processes. Thyroid hormones are closely involved in the regulation of the hypothalamic-pituitary-ovarian axis, ensuring coordinated hormonal signaling necessary for normal follicular development, ovulation, and endometrial preparation. In conditions of iodine deficiency, reduced thyroid hormone production leads to disruption of these regulatory mechanisms, creating a background for endocrine imbalance.

Severe iodine deficiency is often associated with subclinical or overt thyroid dysfunction, which may remain undiagnosed for long periods. In women of reproductive age, thyroid hormone insufficiency may alter gonadotropin secretion and interfere with ovarian steroidogenesis. As a result, menstrual irregularities, anovulatory cycles, and luteal phase insufficiency may develop, negatively affecting fertility and reproductive potential.

Tuberculosis represents a chronic infectious disease characterized by prolonged inflammation, intoxication, and immune dysregulation. Its long-lasting course places a significant burden on adaptive mechanisms and hormonal homeostasis. Chronic infection activates stress-related endocrine pathways, including increased cortisol production, which

may suppress reproductive hormone secretion and exacerbate existing endocrine disturbances. In women with iodine deficiency, these effects may be particularly pronounced due to reduced compensatory capacity of the endocrine system.

From a pathogenetic standpoint, the coexistence of iodine deficiency and tuberculosis creates a complex interaction between endocrine, immune, and metabolic factors. Thyroid dysfunction reduces tissue sensitivity to reproductive hormones, while chronic inflammation associated with tuberculosis disrupts immune–endocrine communication. These processes reinforce one another, forming a self-sustaining pathological cycle that promotes reproductive dysfunction.

Impairment of folliculogenesis and ovulation is one of the key pathogenetic outcomes of this interaction. Inadequate thyroid hormone levels slow metabolic processes within ovarian tissue, while inflammatory mediators further compromise ovarian responsiveness. In addition, alterations in endometrial development may occur due to hormonal imbalance, resulting in reduced receptivity and unfavorable conditions for implantation.

Preventive strategies aimed at reducing the impact of these factors on reproductive health should be multifaceted. Ensuring adequate iodine intake through population-based and individual preventive measures remains a cornerstone of endocrine health preservation. Monitoring thyroid function in women living in iodine-deficient regions is essential for early detection and correction of hormonal disturbances.

At the same time, effective tuberculosis control plays a critical role in preventing long-term systemic consequences. Early diagnosis, appropriate treatment, and follow-up of tuberculosis reduce chronic inflammatory burden and improve overall physiological resilience. In women of reproductive age, such measures may help preserve endocrine stability and reproductive function.

Preventive approaches should also emphasize integrated clinical assessment, including evaluation of thyroid status, menstrual function, and reproductive history in women affected by tuberculosis. Health education and awareness programs may further enhance early recognition of reproductive risk factors and promote timely medical intervention.

Conceptual Diagram. Pathogenetic Interaction Between Iodine Deficiency, Tuberculosis, and Reproductive Disorders



The diagram illustrates the synergistic interaction between iodine deficiency and tuberculosis, highlighting their combined impact on endocrine regulation and reproductive function through interconnected pathogenetic pathways.

Clinical and Preventive Implications of Endocrine and Reproductive Dysregulation

An important aspect of reproductive disorders associated with severe iodine deficiency and tuberculosis is their gradual and often subclinical development. In many cases, endocrine disturbances evolve silently, without pronounced clinical manifestations, especially at early stages. This feature complicates timely diagnosis and increases the likelihood of persistent reproductive dysfunction by the time medical attention is sought. In women of reproductive age, such latent progression represents a serious challenge, as even mild hormonal imbalance may significantly affect fertility and reproductive outcomes.

The endocrine system plays a central coordinating role in maintaining reproductive health. Thyroid hormones, in particular, act as modulators of ovarian responsiveness and endometrial maturation. When iodine deficiency leads to impaired thyroid hormone synthesis, adaptive mechanisms of the reproductive system become limited. Under these conditions, even minor additional stressors, such as chronic infection, may shift the balance toward functional decompensation. Tuberculosis, characterized by long-term inflammation and immune activation, represents one of the most significant stress factors capable of aggravating endocrine vulnerability.

Clinical observations described in the literature indicate that women living in iodine-deficient regions often present with nonspecific complaints, including fatigue, emotional instability, and menstrual irregularities. These symptoms are frequently attributed to chronic

infection or general somatic weakness, while underlying endocrine disturbances remain unrecognized. As a result, reproductive disorders may be underestimated, and preventive measures are delayed. This highlights the need for increased clinical awareness of the endocrine–reproductive consequences of iodine deficiency in patients with tuberculosis.

Another important consideration is the impact of endocrine imbalance on reproductive aging. Chronic disruption of hormonal regulation may accelerate functional exhaustion of the reproductive system, reducing the reproductive window and increasing the risk of infertility. In women affected by tuberculosis, prolonged inflammatory activity and metabolic stress further contribute to this process. The combination of iodine deficiency and chronic infection may therefore have long-term implications extending beyond immediate reproductive outcomes.

From a preventive perspective, the identification of high-risk groups is of particular importance. Women of reproductive age living in iodine-deficient regions and diagnosed with tuberculosis should be considered a vulnerable population requiring targeted monitoring. Preventive strategies should not be limited to infection control alone but should incorporate systematic evaluation of endocrine status, especially thyroid function. Early recognition of thyroid dysfunction allows timely correction and may prevent irreversible reproductive damage.

Nutritional interventions aimed at ensuring adequate iodine intake represent a fundamental preventive measure. Population-based iodine supplementation programs have demonstrated effectiveness in reducing the prevalence of iodine deficiency disorders; however, individual assessment remains necessary for women with chronic diseases. In the context of tuberculosis, nutritional support should be integrated into comprehensive care, as malnutrition and micronutrient imbalance may compromise both immune defense and endocrine regulation.

Equally important is the role of interdisciplinary collaboration in managing reproductive health risks. Cooperation between endocrinologists, phthisiologists, gynecologists, and primary care providers is essential to ensure holistic assessment and management. Such an approach facilitates early detection of endocrine disturbances, appropriate reproductive counseling, and timely referral for specialized care when needed.

Health education also plays a critical role in prevention. Increasing awareness among women about the importance of micronutrient balance, thyroid health, and reproductive well-being may encourage earlier healthcare-seeking behavior. Educational initiatives targeting communities in iodine-deficient regions can contribute to improved understanding of reproductive risks associated with chronic infections and endocrine imbalance.

In addition, regular follow-up and long-term observation should be emphasized in women recovering from tuberculosis. Even after successful completion of anti-tuberculosis therapy, residual endocrine and reproductive disturbances may persist. Continued monitoring of thyroid function and reproductive health status allows assessment of recovery dynamics and identification of late complications.

Taken together, these considerations underline the complexity of reproductive disorders developing in the context of severe iodine deficiency and tuberculosis. The multifactorial nature of these conditions necessitates a comprehensive preventive framework

that addresses endocrine balance, nutritional adequacy, infection control, and reproductive health monitoring. Integrating these elements into routine clinical practice may significantly reduce the burden of reproductive disorders and improve long-term outcomes for women of reproductive age.

In conclusion, severe iodine deficiency combined with tuberculosis forms an unfavorable pathogenetic environment that predisposes women of reproductive age to endocrine and reproductive disorders. Understanding these mechanisms provides a scientific basis for preventive strategies focused on micronutrient sufficiency, infection control, and integrated reproductive health monitoring. Such an approach is essential for improving reproductive outcomes and reducing long-term health consequences in this vulnerable population.

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