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CLINICAL AND FUNCTIONAL CHARACTERISTICS OF TESTICULAR DEVELOPMENT AND SPERMATOGENESIS IN OVERWEIGHT ADOLESCENT BOYS LIVING IN IODINE-DEFICIENT REGIONS

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Abstract
Adolescent obesity combined with environmental iodine deficiency creates multifactorial risks for male reproductive maturation. This cross-sectional study examined 184 boys aged 14 to 17 years from iodine-deficient areas, comparing testicular volume, spermatogenic markers, and hypothalamic-pituitary-gonadal axis function between normal-weight and overweight cohorts. Overweight adolescents demonstrated significantly reduced testicular volumes, lower serum testosterone concentrations, impaired sperm parameters, and disrupted thyroid homeostasis compared to controls, suggesting synergistic endocrine disruption warranting targeted clinical intervention.

Keywords: Adolescence, spermatogenesis, testicular-volume, iodine-deficiency, overweight, testosterone, hypothyroidism, hypogonadism, puberty, follicle-stimulating-hormone

Introduction

Male reproductive capacity established during adolescence remains vulnerable to metabolic and environmental perturbations. Iodine deficiency affects approximately 1.88 billion individuals globally, with endemic regions demonstrating thyroid dysfunction rates exceeding 40 percent in pediatric populations. Concurrently, adolescent obesity prevalence has tripled over three decades, reaching 18.5 percent in developing nations. Thyroid hormones regulate Sertoli cell proliferation, germ cell differentiation, and steroidogenesis through genomic and non-genomic pathways. Adipose tissue produces aromatase enzymes converting testosterone to estradiol, potentially disrupting androgen-estrogen homeostasis critical for spermatogenesis. The intersection of iodine insufficiency and excess adiposity during pubertal maturation may synergistically impair testicular development, yet quantitative clinical characterization remains limited. This investigation examined testicular morphology, spermatogenic function, and hormonal profiles in overweight adolescents from iodine-deficient environments.

Literature Review

Previous investigations established that subclinical hypothyroidism in adolescence correlates with delayed sexual maturation and reduced testicular volumes ranging from 8 to 12 milliliters compared to euthyroid peers. Studies from iodine-deficient regions documented thyroid-stimulating hormone elevations above 4.5 microinternational units per milliliter in 32 to 47 percent of adolescent males,

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though reproductive sequelae received minimal attention. Research examining obesity effects on male fertility demonstrated inverse relationships between body mass index and total testosterone, with reductions of 3.0 to 4.8 nanomoles per liter per 10 kilogram per square meter increment. Semen analysis investigations revealed sperm concentration decreases of 24 to 38 percent in obese versus normal-weight adolescents, yet mechanistic understanding remains incomplete. Critical gaps exist regarding combined effects of iodine deficiency and metabolic dysfunction on pubertal testicular development, particularly quantitative assessment of spermatogenic parameters and hormonal interrelationships in this vulnerable population.

Methodology

This cross-sectional comparative study was conducted from September 2023 through August 2024 in three districts of Uzbekistan classified as moderate iodine-deficient regions with median urinary iodine concentrations between 52 and 78 micrograms per liter. The investigation enrolled 184 male adolescents aged 14 to 17 years recruited through secondary schools and pediatric endocrinology clinics following institutional ethics committee approval and written informed consent from legal guardians.

Participants were stratified into two groups based on body mass index percentiles according to World Health Organization growth references. The overweight group comprised 96 adolescents with body mass index at or above the 85th percentile for age, ranging from 25.3 to 32.7 kilograms per square meter. The control group included 88 age-matched adolescents with body mass index between the 15th and 75th percentiles, ranging from 18.2 to 23.1 kilograms per square meter. Exclusion criteria eliminated subjects with diagnosed endocrine disorders including diabetes mellitus and congenital adrenal hyperplasia, chromosomal abnormalities such as Klinefelter syndrome, cryptorchidism or other congenital urogenital malformations, acute or chronic systemic illnesses, current pharmacological therapy affecting reproductive or thyroid function, and previous thyroid supplementation within six months. Anthropometric measurements included height recorded to the nearest 0.1 centimeter using stadiometers and weight measured to 0.1 kilogram precision on calibrated scales. Body mass index was calculated as weight in kilograms divided by height in meters squared. Waist circumference was measured at the midpoint between the lowest rib and iliac crest. Testicular volume assessment utilized Prader orchidometer with measurements recorded for both testes separately, then averaged for analysis. Tanner staging for genital and pubic hair development was performed by trained pediatric endocrinologists.

Venous blood samples were collected between 08:00 and 09:00 hours following overnight fasting. Serum was separated within 30 minutes and analyzed within four hours or stored at minus 20 degrees Celsius for maximum seven days. Thyroid function assessment measured thyroid-stimulating hormone and free thyroxine using electrochemiluminescence immunoassays with reference ranges of 0.4 to 4.0 microinternational units per milliliter and 10.3 to 24.5 picomoles per liter respectively. Reproductive hormones quantified included total testosterone by liquid chromatography-tandem mass spectrometry with reference range 8.4 to 28.7 nanomoles per liter for Tanner stages four through five, luteinizing hormone and follicle-stimulating hormone measured via immunoassay with expected ranges 1.5 to 9.3 and 1.4 to 18.1 international units per liter respectively. Semen analysis was performed in 142 participants who provided samples after 48 to 72 hours sexual abstinence following

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World Health Organization 2021 guidelines. Parameters evaluated included semen volume measured in milliliters, sperm concentration as million per milliliter, total sperm count, progressive motility percentage, and normal morphology percentage assessed using strict Kruger criteria. Samples were analyzed within 60 minutes of collection by experienced laboratory technicians blinded to participant group assignment.

Results

The overweight group demonstrated significantly elevated mean body mass index of 28.4 plus or minus 2.6 kilograms per square meter compared to 20.7 plus or minus 1.8 kilograms per square meter in controls, representing a 37.2 percent difference. Waist circumference measured 91.3 plus or minus 8.4 centimeters versus 72.6 plus or minus 6.2 centimeters, indicating 25.7 percent greater central adiposity in overweight subjects. Tanner staging revealed delayed progression, with 68.8 percent of overweight adolescents at stage three or four compared to 84.1 percent of controls at stages four or five. Testicular volume averaged 14.2 plus or minus 3.1 milliliters in overweight participants versus 18.6 plus or minus 2.8 milliliters in normal-weight controls, representing a statistically significant 23.7 percent reduction. Right testicular volume measured 14.5 plus or minus 3.3 milliliters compared to 18.9 plus or minus 3.0 milliliters, while left testicular volume was 13.9 plus or minus 3.2 milliliters versus 18.3 plus or minus 2.9 milliliters respectively. The proportion of subjects with bilateral testicular volumes below 15 milliliters reached 64.6 percent in the overweight group compared to 18.2 percent in controls. Thyroid function analysis revealed mean thyroid-stimulating hormone concentrations of 5.8 plus or minus 2.3 microinternational units per milliliter in overweight adolescents versus 2.9 plus or minus 1.1 microinternational units per milliliter in controls, representing a 100 percent elevation. Free thyroxine levels measured 12.4 plus or minus 2.7 picomoles per liter compared to 16.8 plus or minus 3.2 picomoles per liter, indicating 26.2 percent lower concentrations. Subclinical hypothyroidism, defined as thyroid-stimulating hormone above 4.0 microinternational units per milliliter with normal free thyroxine, occurred in 62.5 percent of overweight participants versus 15.9 percent of controls.

Reproductive hormone assessment demonstrated total testosterone concentrations of 11.3 plus or minus 3.8 nanomoles per liter in the overweight cohort versus 19.7 plus or minus 4.2 nanomoles per liter in controls, representing a 42.6 percent reduction. Luteinizing hormone measured 4.8 plus or minus 2.1 international units per liter compared to 5.6 plus or minus 2.4 international units per liter, while follicle-stimulating hormone concentrations were 8.7 plus or minus 3.9 international units per liter versus 6.2 plus or minus 2.8 international units per liter respectively, indicating relative gonadotropin dysregulation. Semen analysis from 73 overweight and 69 control participants revealed mean sperm concentrations of 28.4 plus or minus 14.6 million per milliliter versus 52.3 plus or minus 18.7 million per milliliter, representing a 45.7 percent decrease. Total sperm count averaged 76.8 plus or minus 42.3 million compared to 148.6 plus or minus 56.2 million in controls. Progressive motility measured 38.2 plus or minus 11.4 percent versus 52.7 plus or minus 13.8 percent, indicating 27.5 percent reduced motility. Normal morphology assessment showed 3.8 plus or minus 1.6 percent versus 6.4 plus or minus 2.1 percent using strict criteria. Oligozoospermia, defined as concentration below 15 million per milliliter, occurred in 28.8 percent of overweight subjects compared to 7.2 percent of controls.

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Discussion

These findings demonstrate substantial impairment of testicular development and spermatogenic function in overweight adolescents from iodine-deficient regions, suggesting synergistic pathophysiological mechanisms. The observed 23.7 percent reduction in testicular volume exceeds isolated obesity effects reported in previous literature, indicating additional environmental contribution. Iodine deficiency disrupts thyroid hormone production essential for Sertoli cell maturation during the prepubertal proliferative phase, potentially limiting seminiferous tubule capacity. Elevated thyroid-stimulating hormone concentrations in 62.5 percent of overweight participants reflect combined nutritional insufficiency and metabolic stress, as obesity-associated inflammation may further suppress deiodinase enzyme activity reducing peripheral thyroid hormone conversion. The 42.6 percent testosterone reduction represents clinically significant hypogonadism with potential long-term reproductive consequences. Excess adipose tissue increases aromatase expression, converting testosterone to estradiol and establishing hyperestrogenic states that suppress hypothalamic gonadotropin-releasing hormone pulsatility through negative feedback. Simultaneously, adipocyte-derived leptin at supraphysiological concentrations may desensitize hypothalamic receptors, further blunting luteinizing hormone secretion. The relatively preserved luteinizing hormone levels despite marked testosterone deficiency suggest primary testicular dysfunction rather than isolated central hypogonadism, consistent with impaired Leydig cell steroidogenic capacity.

Semen parameter deterioration, particularly the 45.7 percent sperm concentration decrease, indicates functional spermatogenic disruption beyond morphological testicular hypoplasia. Thyroid hormones regulate tight junction proteins between Sertoli cells maintaining the blood-testis barrier essential for meiotic progression. Hypothyroidism disrupts this microenvironment, increasing germ cell apoptosis and reducing mature spermatid production. Additionally, testosterone insufficiency limits androgen-dependent spermatogenic phases including spermiogenesis and spermiation. The 28.8 percent oligozoospermia prevalence in mid-to-late adolescence portends concerning fertility implications, as these represent formative years establishing lifelong reproductive potential.

Overweight adolescent boys in iodine-deficient regions exhibit markedly reduced testicular volumes, impaired spermatogenesis, and hypogonadotropic hypogonadism reflecting synergistic endocrine disruption. The 42.6 percent testosterone reduction and 45.7 percent sperm concentration decrease represent clinically significant reproductive dysfunction. These findings necessitate integrated screening programs addressing both nutritional iodine supplementation and obesity management to preserve male reproductive health during critical pubertal development.

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