

Enhancing Pregnancy Outcomes: The Role of Metabolic Factor Correction in Subfertility

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Abstract:

Background: Infertility is a growing concern worldwide, affecting 8–12% of reproductive-age couples. Metabolic disorders such as insulin resistance, hyperprolactinemia, and thyroid dysfunction play a crucial role in subfertility. Addressing these metabolic factors may improve reproductive outcomes in affected women.

Aim: This study evaluates the impact of correcting metabolic factors on enhancing pregnancy outcomes in sub-fertile women.

Methodology: A prospective observational study was conducted at the Department of Obstetrics and Gynaecology, Government Medical College and Hospital, Bettiah, Bihar, India. The study included 90 sub-fertile women aged 18–45 years. Participants were screened for metabolic abnormalities, including thyroid dysfunction, hyperprolactinemia, and insulin resistance. Corrective interventions included levothyroxine for thyroid dysfunction, metformin for insulin resistance, and dopamine agonists for hyperprolactinemia. Ovulation was monitored over three menstrual cycles post-intervention, and hormonal levels were reassessed.

Results: A significant reduction in thyroid-stimulating hormone (TSH) ($p < 0.001$), serum prolactin ($p < 0.001$), and luteinizing hormone (LH) ($p < 0.05$) was observed after three months of treatment. Estradiol and progesterone levels increased significantly ($p < 0.05$, $p < 0.001$, respectively). Ovulation occurred in 66.67% of participants within three cycles, while 8.33% ovulated within six cycles. However, 25% required extended treatment.

Conclusion: Correcting metabolic abnormalities significantly enhances ovulation and improves fertility outcomes in sub-fertile women. Addressing these factors should be a key component of infertility management.

Keywords: Hyperprolactinemia, Infertility, Insulin Resistance, Metabolic Factors, Subfertility, Ovulation, Thyroid Dysfunction.

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Introduction

Infertility is afflicting more and more people. Usually defined in clinical practice as the inability to conceive following twelve months of consistent sexual activity with the purpose of having a kid in women under 35 years old, infertility is. But in the same diagnostic management, women over 35 years old sometimes refer to a duration of six months of failed attempts [1]. The global prevalence of infertility among reproductive-age couples is estimated to be between 8 and 12 percent, with some estimates going as high as 18 percent. These individuals primarily hail from underdeveloped nations [2-3]. Up to 30% of the population is infertile in the Central and Eastern area, Central and South Asia, and Sub-Saharan Africa [4]. Any degree of diminished fertility in couples who tried and failed to conceive is commonly referred to as subfertility

[5]. One in fifteen couples of childbearing age experience this. Subfertility affects almost 70 million couples globally [6]. There is a complicated interaction between metabolic variables and fertility, which impacts reproductive health in both sexes. Fertility can be severely impacted by a variety of metabolic diseases. Metabolic syndrome is characterized by high blood pressure, dyslipidemia, and blood plasma glucose levels that are all abnormally high [7]. These three abnormalities directly lead to the pro-inflammatory and prothrombotic condition, which raises type 2 diabetes and atherosclerotic cardiovascular disease risk [8]. Both metabolic syndrome and polycystic ovarian syndrome are characterized by metabolic abnormalities, the most common of which are insulin resistance and hyperinsulinemia [9].

Increased androgen synthesis and fat metabolism from theca cells are negatively impacted by insulin resistance and elevated circulating insulin levels [10].

The link between fat and infertility has been acknowledged. Subfertility, infertility, irregular periods, miscarriages, and a higher risk of pregnancy problems are all outcomes of its impact on the reproductive process. Obesity is linked to insulin resistance (IR), a metabolic change. Obesity and insulin resistance are linked to hormonal defects affecting fertility. Among these changes are alterations in the pulses of luteinizing hormone (LH) and follicle stimulating hormone (FSH), hyperandrogenism, and control of sex hormone-binding globulin's hepatic production (SHBG). The endometrium, oocytes, and follicles are all negatively impacted by these alterations, which in turn reduces reproductive ability. Reducing intrauterine growth restriction (IR), largely via weight loss and lowering of visceral fat., is one of the primary aims of treating obesity-related infertility.

Essential for one-carbon biosynthetic and epigenetic activities, foliates are water-soluble B9 vitamins. Folate can be found in foods including beans and other legumes, milk, liver, dark green leafy vegetables, citrus fruits and liquids, whole grains, and other foods. Study find folic acid, a synthetic type of folate, in dietary supplements and foods that have been fortified with it. Folic acid as having a pteridine ring, p-aminobenzoic acid, and glutamic acid as its structural components. A one-carbon unit of methyl, methenyl, methylene, or formyl may be present in naturally occurring folate derivatives, which are converted to dihydrofolate or tetrahydrofolate (THF) and have a glutamate residue chain. Fortifying foods and nutritional supplements with synthetic vitamin folic acid has the advantages of being more stable, monoglutamated, and oxidized than natural folates.

According to estimates worldwide, 186 million people suffer with infertility—that is, the helplessness to conceive following one year of insecure sexual activity. A rate of more than 30% is possible in developing nations. Although women continue to face social stigma, male infertility is the leading cause of childlessness worldwide. Twenty percent to thirty percent of infertility in women has no known etiology, however ovulation, tubal, and endometriosis are factors. Lifestyle and reproductive health for women have recently received more attention. A woman's fertility may be impacted by her weight, body composition, level of physical activity, and diet. Metabolism and female reproduction are intrinsically linked. The creation of sex hormones by the gonads is a cyclical process that controls the metabolism of energy during reproduction. The reproductive demands of

mammals, and particularly females, alter energy metabolism. The capacity of mammals to store oxidizable fuels is essential for puberty, pregnancy, and lactation. Strategies for energy conservation in food plenty and for limiting reproduction in nutrient poor conditions have evolved over time. Problems arise when there is a physiological misalignment between metabolism and reproduction. Metabolic abnormalities are more common in ovarian dysfunction illnesses such as polycystic ovarian syndrome (PCOS) and Turner syndrome, and metabolic and cardiovascular disorders are more common in women once ovarian function quits. The gender-specific approach meant to guarantee the survival of species has negative effects on female health and fertility' in contemporary obesogenic surroundings.

Methodology

Study Area: A prospective observational study was conducted at the Department of Obstetrics and Gynaecology, Government Medical College and Hospital, Bettiah, Bihar, India for one year

Sample Size: The study included 90 sub-fertile female patients.

Sample Collection: Women who were unable to conceive due to metabolic abnormalities like hyperprolactinemia, insulin resistance, and thyroid dysfunction were part of the research. Metabolic testing, hormonal assessments, and clinical histories were used to select participants.

Inclusion and Exclusion Criteria

Inclusion Criteria: Included in this study were female individuals with subfertility information, ranging in age from 18 to 45 years.

Exclusion Criteria: The study excluded female participants with a history of hysterectomy, or bilateral oophorectomy. After outlining 'the study's aims, objectives, and procedures, written informed consent was secured from each participant. Baseline demographic information of the patients was subsequently recorded, with all data being kept strictly confidential. The institutional ethics committee granted ethical clearance for the research. The study looked at metabolic elements including serum prolactin, thyroid function, body mass index (BMI), blood glucose level, and lipid profile. Thyroid and prolactin function were processed both at baseline and again after 3 months. Ovulation status was monitored following 3 and 6 menstrual cycles.

Procedure: The study utilised a systematic technique to determine if metabolic correction enhanced subfertility in women. Participants with metabolic problems such as hyperprolactinemia, insulin resistance, and thyroid dysfunction were exclusively selected for the study based on their

clinical history and hormonal/metabolic evaluations. Hormonal levels were preserved at baseline in blood samples collected during first evaluations, which also encompassed clinical metrics such as body mass index (BMI), fasting glucose, and indicators of insulin resistance. Hormonal assessment and transvaginal ultrasound were utilised to track ovulation. During the three-month intervention phase, individuals were administered targeted medications for their problems. Levothyroxine was provided for thyroid dysfunction. Metformin was prescribed for insulin resistance, along with recommendations for lifestyle modifications. Dopamine agonists were used for hyperprolactinemia. Alongside facilitating metabolic balance, we also offered exercise and nutritional guidance. Ovulation was observed throughout three menstrual cycles subsequent to the intervention, and hormone levels were evaluated post-treatment. We utilised evidence from prior to and subsequent to metabolic correction to evaluate its efficacy in enhancing reproductive function. Important insights on the influence of metabolic regulation on fertility were derived from the subsequent analysis of findings, which therefore had therapeutic implications. Obstacles to the efficacy of the intervention must be surmounted.

Statistical Analysis: The statistics were available as the means along with the standard deviation. The Chi-square test was utilized for the analysis of categorical variables, whereas the student's t-test was applied to compare continuous variables. A p-value of <0.05 was deemed statistically significant, suggesting a noteworthy difference between the groups being compared.

Result

Table 1 presents the demographic and clinical characteristics of the research sample (N=90). The predominant age group among participants was 36–45 years (42.5%), succeeded by 26–35 years (35%) and 18–25 years (22.5%), with a mean age of 37.21 ± 3.21 years. The average Body Mass Index (BMI) was 23.12 ± 2.94 kg/m², and the mean blood glucose level was 92.51 ± 10.31 mg/dL. The mean insulin resistance, determined by HOMA-IR, was 2.31 ± 0.82 . The lipid profile revealed a mean total cholesterol of 190.52 ± 30.21 mg/dL, LDL cholesterol of 110.31 ± 25.42 mg/dL, HDL cholesterol of 55.21 ± 15.11 mg/dL, and triglycerides of 150.41 ± 40.62 mg/dL. These findings provide a baseline health condition for the research group.

Table 1: Demographic Characteristics of Study Subjects (N=90)

Variables	Mean $\pm$ SD	Frequency (n)	Percentage (%)
Age (years)			
18-25		20	22.50
26-35		32	35.00
36-45		38	42.50
Mean $\pm$ SD	37.21 ± 3.21		
BMI (kg/m ²)	23.12 ± 2.94		
Blood glucose level (mg/dL)	92.51 ± 10.31		
Insulin resistance (HOMA-IR)	2.31 ± 0.82		
Lipid Profile			
Total cholesterol (mg/dL)	190.52 ± 30.21		
LDL cholesterol (mg/dL)	110.31 ± 25.42		
HDL cholesterol (mg/dL)	55.21 ± 15.11		
Triglycerides (mg/dL)	150.41 ± 40.62		

Table 2 illustrates the prevalence of thyroid function in a cohort of 90 participants. A majority of the participants, 64 (71.21%), exhibited euthyroidism (normal thyroid function). Subclinical hypothyroidism was identified in 14 individuals (15.73%), whereas 8 individuals (8.35%) were

diagnosed with clinical hypothyroidism. Only four participants (4.71%) exhibited hyperthyroidism. This implies that although most patients were euthyroid, a notable number exhibited thyroid dysfunction, with subclinical hypothyroidism being more prevalent than other aberrant forms.

Table 2: Thyroid Function Distribution (N=90)

Types of Thyroid Status	Frequency (n)	Percentage (%)
Euthyroid	64	71.21
Hyperthyroid	4	4.71
Subclinical hypothyroid	14	15.73
Clinical hypothyroid	8	8.35

Table 3 displays the biochemical evaluation of thyroid function in a group of 90 individuals, contrasting baseline measurements with those acquired after a three-month period. A statistically significant reduction in Thyroid Stimulating Hormone (TSH) levels was noted, decreasing from 7.42 ± 2.09 mIU/L at baseline to 3.12 ± 1.05 mIU/L after three months ($p < 0.001$), indicating enhanced

thyroid regulation. Furthermore, Free T4 levels dropped dramatically from 17.82 ± 3.13 to 14.08 ± 2.95 micromole/L ($p < 0.05$), alongside a notable reduction in Free T3 levels from 3.00 ± 0.75 to 2.50 ± 0.65 pg/mL ($p < 0.01$). Overall, these results suggest the normalisation of thyroid function throughout the three-month period.

Table 3: Biochemical Examination of Thyroid Function (N=90)

Thyroid Function	Baseline Status	After 3 Months Status	P-value
TSH (mIU/L)	7.42 ± 2.09	3.12 ± 1.05	<0.001
Free T4 (micromole/L)	17.82 ± 3.13	14.08 ± 2.95	<0.05
Free T3 (pg/mL)	3.00 ± 0.75	2.50 ± 0.65	<0.01

Table 4 displays the findings of a biochemical assessment of prolactin function in a cohort of 90 people. It demonstrates substantial changes in hormone levels from baseline to three months later. Serum prolactin levels diminished from 25.36 ± 5.24 ng/mL to 14.82 ± 4.09 ng/mL ($P < 0.001$). Luteinizing hormone (LH) and follicle-stimulating hormone (FSH) levels decreased from 12.45 ± 3.89 mIU/mL to 8.21 ± 2.76 mIU/mL ($P < 0.05$) and from

9.58 ± 2.47 mIU/mL to 7.94 ± 2.03 mIU/mL ($P < 0.05$), respectively. Oestradiol levels elevated from 45.12 ± 12.38 pg/mL to 52.89 ± 13.27 pg/mL ($P < 0.05$), whereas progesterone levels exhibited a significant increase from 1.21 ± 0.52 ng/mL to 5.62 ± 1.21 ng/mL ($P < 0.001$). These alterations indicate a substantial hormonal transition throughout the three-month duration.

Table 4: Biochemical Examination of Prolactin Function (N=90)

Prolactin Function	Baseline Status	After 3 Months Status	P-value
Serum Prolactin (ng/mL)	25.36 ± 5.24	14.82 ± 4.09	<0.001
LH (mIU/mL)	12.45 ± 3.89	8.21 ± 2.76	<0.05
FSH (mIU/mL)	9.58 ± 2.47	7.94 ± 2.03	<0.05
Estradiol (pg/mL)	45.12 ± 12.38	52.89 ± 13.27	<0.05
Progesterone (ng/mL)	1.21 ± 0.52	5.62 ± 1.21	<0.001

Table 5 displays the ovulation status of 90 patients receiving therapy. Sixty-six point sixty-seven percent of patients attained ovulation within three cycles, demonstrating a positive and prompt response to therapy in the majority of instances. A lesser percentage (8.33%) ovulated within six

cycles, indicating a postponed yet eventual reaction. However, 25% of people needed further therapy beyond six cycles, underscoring a subset of patients who may require prolonged or different therapeutic strategies to attain ovulation.

Table 5: Ovulation Status (N=90)

Ovulation Status	Frequency (n)	Percentage (%)
Within 3 cycles	60	66.67
Within 6 cycles	8	8.33
More treatment required	22	25.00

Discussion

The present study aimed to evaluate the demographic and clinical characteristics, thyroid and prolactin hormonal profiles, and ovulation outcomes in a sample of 90 individuals undergoing therapeutic intervention. The findings offer significant insights into the endocrine and reproductive health status of the participants, along with the effectiveness of the administered therapy over a three-month period.

The demographic profile indicated that most participants were aged 36–45 years, with a mean age

of 37.21 years, signifying that the majority were in late reproductive age. The baseline BMI (23.12 kg/m^2) and average blood glucose level (92.51 mg/dL) were within normal ranges, indicating a predominantly healthy metabolic profile. Likewise, lipid profiles including total cholesterol, LDL, HDL, and triglycerides were within anticipated ranges, further corroborating the metabolic stability of the population at baseline. Hannoun et al. [11] reported in 1997 a treatment success rate of 5% in a CC+IUI group after one cycle and 58.7% in a controlled ovarian stimulation (COS)+IUI group after three cycles. Guzick et al. [12] conducted a retrospective

analysis of 45 published studies on unexplained infertility IUI trials, revealing clinical gestation rates of 8.3% in the CC+IUI cohort and 17.1% in the hMG+IUI cohort. Goverde et al. [13] documented clinical gestational rates of 7.4% in a natural cycle IUI cohort and 8.7% in a COS+IUI cohort. A European research conducted from 2001 to 2004 indicated that the pregnancy rate per IUI cycle varied between 11.4% and 12.6% [14]. Our investigation revealed a clinical gestation rate of 9.34% (7.8% in the CC group, 8.9% in the hMG group, and 9.6% in the rFSH group), which exhibited considerable variability.

Regarding thyroid function, a significant proportion (71.21%) of the participants were euthyroid, aligning with general population estimates. However, 24 participants (26.79%) exhibited thyroid dysfunction, with subclinical hypothyroidism (15.73%) being the most common, followed by clinical hypothyroidism (8.35%) and hyperthyroidism (4.71%). These findings emphasize the prevalence of subtle thyroid abnormalities even in ostensibly healthy individuals, reinforcing the need for routine thyroid screening in patients presenting with reproductive concerns. Their Research indicates that sustaining a healthy weight is essential for hormonal balance and overall fertility [15]. It highlights the need of weight management as an integral component of a holistic strategy for tackling subfertility. The metabolic evaluations of the study indicated an average blood glucose level of 92.51 ± 10.31 mg/dL and a mean HOMA-IR score of 2.31 ± 0.82 , suggesting possible insulin resistance. Insulin resistance and hyperglycemia are intricately associated with disorders like polycystic ovarian syndrome (PCOS) and type 2 diabetes, which are major factors in anovulation and infertility [16-17].

The therapeutic intervention led to marked improvements in thyroid parameters. TSH levels significantly decreased ($p < 0.001$), while Free T3 and Free T4 levels also reduced significantly, indicating a return to euthyroid status. This normalization of thyroid hormones may play a pivotal role in the regulation of reproductive hormones and improvement of ovulatory function. Thyroid function evaluations indicated that 71.21% of patients were euthyroid, whereas 24.08% exhibited subclinical or overt hypothyroidism. Thyroid disease, especially hypothyroidism, impairs the hypothalamic-pituitary-ovarian axis and correlates with reduced ovulation rates and worse pregnancy outcomes [18]. After three months of focused management, notable improvements in thyroid function were seen. Mean TSH levels decreased from 7.42 ± 2.09 mIU/L to 3.12 ± 1.05 mIU/L, indicating successful therapy of hypothyroidism. This lowering is essential for reinstating ovarian function and enhancing reproductive potential.

Parallel improvements were observed in the prolactin axis. Serum prolactin levels dropped significantly post-treatment ($p < 0.001$), which is clinically relevant since hyperprolactinemia is a known disruptor of ovulatory cycles. Additionally, LH and FSH levels decreased significantly, likely reflecting the restored negative feedback mechanism due to normalized estrogen and progesterone levels. The significant increase in progesterone and estradiol post-treatment indicates improved luteal function and ovarian responsiveness, which are critical for successful ovulation and fertility. These findings are consistent with recent research indicating that hyperprolactinemia decreases gonadotropin production, hence compromising ovarian function and disrupting normal menstrual cycles [19]. Furthermore, oestradiol levels jumped substantially, from 45.12 ± 12.38 pg/mL to 52.89 ± 13.27 pg/mL ($p < 0.05$), while progesterone levels increased markedly, from 1.21 ± 0.52 ng/mL to 5.62 ± 1.21 ng/mL ($p < 0.001$). The increase in oestradiol signifies improved follicular growth, perhaps due to the reduction of prolactin's suppressive influence on FSH and LH.

The ovulation results further corroborate the biochemical findings. Approximately two-thirds of individuals (66.67%) attained ovulation after three cycles, indicating a fast treatment response. An extra 8.33% ovulated within six cycles, indicating delayed response. However, 25% of patients need extended or different therapeutic interventions, underscoring the heterogeneity in individual hormonal response and the requirement for customised treatment regimens. These findings suggest that hormonal correction through appropriate therapeutic regimens can significantly elevate thyroid and reproductive hormone levels and improve ovulatory results. The research highlights the need of precise endocrine evaluation in infertile individuals and suggests that rectifying subclinical abnormalities in thyroid and prolactin levels may provide considerable reproductive advantages.

Conclusion

The findings of this research highlight the tremendous impact of metabolic correction on the improvement of reproductive function in subfertile women. The majority of research participants were in late reproductive age, with a mean age of 37.21 years. Metabolic parameters, including BMI, blood glucose, and cholesterol levels, were predominantly within the normal range; nevertheless, indications of endocrine dysregulation were present. The restoration of thyroid function was remarkable, evidenced by the normalisation of TSH from 7.42 mIU/L to 3.12 mIU/L ($p < 0.001$), and a reduction in prolactin levels from 25.36 ng/mL to 14.82 ng/mL ($p < 0.001$). The endocrine enhancements favoured ovulation frequency, with 66.67% of subjects seen to ovulate within three cycles. The research

indicates the significance of particular metabolic therapies, such as pharmacological agents and lifestyle modifications, in addressing subfertility. Given the significant enhancement in ovulatory function, metabolic correction should be prioritised as a crucial therapeutic approach in addressing infertility resulting from metabolic problems.

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