Use validated indices (Partial Mayo Score, endoscopic findings) at every visit to formally categorize disease activity. Treatment intensity and monitoring frequency should be dictated by the patient's highest documented severity, as this predicts complication risk.

2. Treat to an Objective Target.

Aim for clinical and endoscopic remission (mucosal healing). Utilizing fecal calprotectin and scheduled sigmoidoscopy to guide therapy reduces long-term risks of hospitalization, colectomy, and colorectal neoplasia.

3. Establish a Structured Surveillance Protocol.

For patients with a history of extensive, moderate to severe colitis, implement a disciplined schedule for dysplasia surveillance

4. Educate Patients on Complication Red Flags

Proactively teach patients to recognize and urgently report symptoms indicating severe flare (high fever, profuse bleeding, severe pain) or complication (worsening pain, distension suggesting toxic megacolon). This enables rapid intervention.

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УДК 616.379-008.64:618.3-037

A. Errati

Scientific supervisor: PhD, Associate Professor S. A. Shut

Educational Establishment
"Gomel State Medical University"
Gomel, Republic of Belarus

GESTATIONAL DIABETES AND ITS RISK FACTORS

Introduction

Gestational diabetes mellitus (GDM) is characterized by high blood sugar levels that first appear or are identified while a woman is pregnant (GDM) is often divided into two classes: (GDM) of class A1, which depends on diet, and (GDM) of class A2, which requires pharmacological treatment of hyperglycaemia [1]. The benefits of diagnosing GDM have been known for a long time; numerous studies have shown that the prevention and treatment of GDM reduces the risk of adverse pregnancy outcomes and that women with GDM are at high risk of developing type 2 diabetes mellitus in the postpartum period [2, 3]. Due to the strikingly high perinatal mortality rate among pregnant women with diabetes, treatment strategies for GDM have continued to change over the past few decades. Treatment strategies for GDM have changed over time, starting with the World Health Organization (WHO) changing glucose thresholds and the adoption of comprehensive criteria by the International Association of Diabetes and Pregnancy Groups (IADPSG) in 2013 [3, 4]. Risk factors for developing gestational diabetes are key points that can reduce risks during pregnancy [5].

Goal

1. To identify clinical and laboratory risk factors for GDM.
2. To characterize systemic metabolic alterations through comprehensive profiling.
3. To distinguish GDM-specific patterns from normal pregnancy changes.

Material and methods of research

We conducted a retrospective analysis of 67 pregnant women (24–28 weeks gestation) divided into non-diabetic (n=39) and GDM (n=28) groups based on IADPSG criteria [4]. Laboratory parameters including complete blood count, urinalysis, and biochemical panels were analysed with comparison of mean values between groups.

Statistical analysis was performed using standard methods. For continuous variables presented in table 1 (e.g., glucose, CRP, ALT), the Student's t-test for independent samples was used to compare the mean values between the Non-Diabetes and GDM groups. For categorical variables presented in table 2 (e.g., prevalence of obesity, hypertension), the Chi-square (X^2) test was used to compare the frequencies between the two groups. A p-value of less than 0.05 was considered statistically significant [7].

The results of the research and their discussion (Table 1).

Table 1 – Key Laboratory Parameters in GDM vs Non-Diabetic Pregnancy

Parameter	Non-Diabetes (n=39)	GDM (n=28)	Change	P-value	Significance
Glucose (mmol/L)	4.48	5.36	0.88	<0.001	Highly Significant
CRP (mg/L)	5.8	9.2	3.4	<0.001	Highly Significant
ALT (U/L)	41.5	56.3	14.8	<0.001	Highly Significant
AST (U/L)	36.2	48.7	12.5	<0.001	Highly Significant
Proteinuria (g/L)	0.012	0.018	0.006	0.012	Significant
Haemoglobin (g/L)	121.6	120.5	−1.1	0.58	Not Significant
Ferritin (μg/L)	16.8	15.2	−1.6	0.21	Not Significant

The GDM group demonstrated expected hyperglycaemia with 19.6% higher fasting glucose ($p < 0.001$), confirming diagnostic classification. More notably, substantial and highly significant elevations in inflammatory markers (CRP increased 58.6%, $p < 0.001$) and liver enzymes (ALT elevated 35.7%, $p < 0.001$) indicate systemic metabolic disturbance beyond glucose intolerance. The proteinuria increase (50%, $p=0.012$) suggests early renal stress. While the ferritin reduction and slight haemoglobin decrease were observed, these differences were not statistically significant ($p > 0.05$), suggesting that although there is a trend, the iron metabolism alterations in this GDM cohort require further investigation with a larger sample size (Table 2).

Table 2 – Prevalence of Clinical Conditions

Condition	Non-Diabetes (n=39)	GDM (n=28)	Difference	P-value	Significance
Obesity	7.50%	53.60%	46.10%	<0.0001	Highly Significant
Gestational hypertension	15%	25%	10%	0.30	Not Significant
Hemorrhoids	0%	17.90%	17.90%	0.009	Highly Significant
Latent iron deficiency	0%	17.90%	17.90%	0.009	Highly Significant

Obesity prevalence was dramatically and significantly higher in the GDM group (7.1-fold increase, $p < 0.0001$), reinforcing its central role in GDM pathophysiology. The exclusive presence

of haemorrhoids (p=0.009) and latent iron deficiency (p=0.009) in GDM patients is statistically significant and suggests these conditions may represent additional manifestations of metabolic disturbance rather than coincidental findings. Although gestational hypertension was more common in the GDM group (25% vs. 15%), this difference did not reach statistical significance (p=0.30), possibly due to the sample size.

Our comprehensive analysis reveals that gestational diabetes mellitus represents a systemic metabolic disorder extending beyond isolated glucose intolerance. The highly significant elevations in inflammatory markers (CRP) support the paradigm of GDM as a low-grade inflammatory state, potentially contributing to insulin resistance and vascular complications. The substantial and significant hepatic enzyme increases (ALT, AST) suggest hepatic involvement possibly related to fatty liver changes, indicating that liver function monitoring should be incorporated into GDM management. Renal parameters showed early stress indicators with significantly increased proteinuria, highlighting potential renal implications. While a haematological paradox of lower ferritin was noted, it was not statistically significant in this sample, suggesting the relationship between GDM and iron metabolism is complex and may involve inflammatory sequestration rather than simple deficiency. The dramatic and statistically robust obesity association underscores adiposity as a primary risk factor, supporting pre-pregnancy weight management as a preventive strategy. These findings collectively advocate for expanded GDM management approaches that address multisystem metabolic alterations rather than focusing exclusively on glycaemic control.

Conclusions

GDM is associated with comprehensive metabolic alterations affecting inflammatory pathways, hepatic function, and renal integrity, as confirmed by statistically significant differences in CRP, ALT, AST, and proteinuria.

The strong and highly significant association with obesity ($p<0.0001$) highlights the importance of pre-conception weight management as a key preventive strategy.

The significant association with haemorrhoids and latent iron deficiency (p=0.009) suggests these conditions may be linked to the metabolic disturbances of GDM and warrant clinical attention.

Our results support expanding GDM management beyond glucose monitoring to include assessment of liver function, inflammatory status, and renal parameters, with consideration of individualized nutritional approaches.

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