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Case Analysis and Management Strategies of Glucocorticoid-Induced Metabolic Syndrome during Pregnancy

Li Tang, Weihua Yang, Qiuping Wang*

Department of Obstetrics, Guangming District People's Hospital, Shenzhen, China
Email: *58839530@qq.com

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Abstract

Gestational Metabolic Syndrome (GMS) is a unique cluster of metabolic abnormalities characterized by pre-pregnancy overweight/obesity, dysregulated glucose/lipid metabolism, and elevated blood pressure, significantly increasing risks of adverse pregnancy outcomes and long-term maternal-fetal cardiovascular diseases. This article reports a case of a 36-year-old woman with gestational diabetes mellitus (GDM) who developed acute GMS following dexamethasone administration for threatened preterm labor. The patient developed acute metabolic decompensation 24 hours post-dexamethasone administration, with fasting blood glucose peaking at 13.3 mmol/L and triglycerides exceeding 18.2 mmol/L. Multidisciplinary intervention resulted in favorable maternal and neonatal outcomes. This case highlights the importance of individualized monitoring and early intervention to mitigate metabolic decompensation risks in GDM patients receiving glucocorticoid therapy.

Keywords

Gestational Metabolic Syndrome, Glucocorticoids, Metabolic Disorders, Case Report, Management Strategies

1. Introduction

Gestational metabolic syndrome (GMS) refers to the pathological conditions of insulin resistance, glucose and lipid metabolism disorders and abnormal blood pressure regulation caused by hormone changes in pregnant women. Its core performance is multiple metabolic imbalances such as elevated blood glucose, abnormal blood pressure, elevated triglyceride (TG) and decreased high-density lipoprotein [1]. Preeclampsia and gestational diabetes mellitus (GDM) were first es-

tablished as independent risk factors for cardiovascular disease in women by the American Heart Association (AHA) 2011 guidelines for the prevention and treatment of cardiovascular disease in women [2]. Studies have shown that patients with such pregnancy complications not only present the typical characteristics of metabolic syndrome (including central obesity, insulin resistance, etc.), but also have a significantly higher risk of cardiovascular disease and type 2 diabetes in the long term after delivery than the general population [3]-[6]. Glucocorticoid (GC) is widely used in premature fetal lung maturation, but its diabetogenic and lipolytic effects pose a significant risk for patients with GDM. GC exacerbates insulin resistance through PEPCK/G6 pase upregulation and GLUT4 inhibition, and promotes lipolysis through HSL activation, which together trigger GMS in susceptible individuals [7]. This is a case report of a case of GMS caused by the use of glucocorticoids in gestational diabetes mellitus in our hospital, which was treated in time and had a good outcome. This case provides a reference value for clinical GMS prevention, treatment and prognosis.

2. Case Presentation

A 36-year-old primigravida (G1P0) was admitted on June 15, 2021, at 30 + 6 weeks of gestation due to "vaginal bleeding for 3 hours." No abnormalities were detected during prenatal check-ups in the early stages of pregnancy. Pregnancy 26+ weeks: Diagnosed with GDM via oral glucose tolerance test (OGTT): fasting blood glucose (FBG) 6.23 mmol/L, 1-hour postprandial glucose 10.57 mmol/L, 2-hour postprandial glucose 11.6 mmol/L. Managed with diet and exercise, achieving FBG 5.1 - 5.7 mmol/L and normal postprandial glucose levels. Pregnancy 30 + 6 weeks: Presented with painless vaginal bleeding (50 mL). Ultrasound revealed a marginal placenta previa (17 mm from cervical os). Diagnosed with threatened preterm labor and admitted for monitoring.

Treatment and Complications:

Dexamethasone 6 mg intramuscularly (q12h × 4 doses) was administered for fetal lung maturation. 24 hours post-treatment: Metabolic deterioration, including nausea, diaphoresis, blood pressure 128/86 mmHg, heart rate 96 bpm, and oxygen saturation 99%. Laboratory findings: FBG 13.3 mmol/L, 2-hour postprandial glucose 19.6 mmol/L, "milky" serum appearance, Triglycerides (TG) > 8.47 mmol/L, total cholesterol Total Cholesterol (TC) > 12.93 mmol/L, HDL-C 0.68 mmol/L (**Table 1**). According to Niu Jianmin's criteria [1], this patient met three diagnostic indicators: Pre-pregnancy BMI: 28.4 kg/m^2 (≥25 kg/m^2); otal cholesterol (TC) > 12.93 mmol/L (normal < 5.2 mmol/l), HDL-C 0.68 mmol/L (normal 1.25 - 1.55 mmol/l)); Hypertriglyceridemia: TG > 8.47 mmol/L (normal < 1.7 mmol/L).

Management:

- Immediate dexamethasone discontinuation.
- Fluid resuscitation and insulin therapy: Initial bolus with normal saline (15 - 20 mL/kg/h), followed by continuous insulin infusion (0.1 U/kg/h). Transitioned to subcutaneous insulin (14 U pre-meal, 14 U bedtime detemir) upon glucose

stabilization.

- Lipid-lowering therapy: Xuezhikang (0.6 g bid).
- Outcomes: Metabolic parameters improved within 24 hours (Table 2). Discharged after 3 days with outpatient monitoring. Delivered vaginally at 36 + 1 weeks (neonatal weight 2900 g, Apgar 9 - 10). Postpartum metabolic normalization: TG decreased from 8.47mmol/L to 5.17 mmol/L; TC from >12.93 mmol/L to 5.77 mmol/L (Table 3).

Explicate:

Xuezhikang (generic: red yeast rice extract; active ingredient: lovastatin 2.5 -3.0 mg/capsule), administered at 0.6 g twice daily. Its HMG-CoA reductase inhibitory activity reduces hepatic cholesterol synthesis, while monacolin K modulates lipid metabolism Dose selection was based on pregnancy-specific safety protocols. Xuezhikang has the effect of regulating abnormal blood lipids, which can reduce blood cholesterol, triglycerides, low-density lipoprotein cholesterol, and increase high-density lipoprotein cholesterol; Inhibit the formation of atherosclerotic plaque; Protect vascular endothelial cells; Inhibit lipid deposition in the liver.

Table 1. Alterations in TG and TC levels before and after administration of dexamethasone.

Parameter	Pre-Dexamethasone	Post-Dexamethasone (Peak)
Triglycerides	2.1 mmol/L	>18.2 mmol/L
Total Cholesterol	4.8 mmol/L	>12.93 mmol/L

Table 2. Blood glucose fluctuations.

Date	Fasting (mmol/l)	2h-PAB (mmol/l)	2h-PAL (mmol/l)	BD (mmol/l)	2h-PAD (mmol/l)	Bedtime (mmol/l)
16/6	5.1	8.6	8.5	6.7	9	8.6
17/6	9.2	13.1↑	9.3	9	12.6↑	10.1
18/6	8.2	8.3↑	8.8	8.1	11.1↑	9.1
19/6	8.5	8.9	8.2	6.1	9.7	8.9
20/6	5.6	6.2	7.6	6.2	8.6	7.6

2h-PAB: 2 hours after breakfast; 2h-PAL: 2 hours after lunch, BD; before dinner; 2h-PAD: 2 hours after dinner.

Table 3. Blood lipid change table.

Date	TG (<1.7 mmol/l)	TC (<5.3 mmol/l)	HDL-C (1.25 - 1.55 mmol/l)
15/6	2.1	4.8	1.2
16/6	8.47	>12.93	0.68
17/6	18.2	>12.93	0.81
22/6	7.23	10.82	0.64
24/7 (48h-postpartum)	5.17	5.77	0.75

TG (triglycerides); TC (total cholesterol); HDL-C (high-density lipoprotein cholesterol).

3. Discussion

The clinical research of metabolic syndrome (MetS) can be traced back to the 1920s. With the deepening of multi-dimensional research, domestic scholars have formulated clinical diagnostic criteria for metabolic syndrome (MetS) applicable to Chinese population [8], which stipulates that if any three of the following four indicators are met, the diagnosis can be made: 1) Prepregnancy body mass index (BMI) ≥ 25 kg/m^2; 2) Systolic/diastolic blood pressure ≥ 140/90 mmHg; 3) Diagnosis history of gestational diabetes (GDM); 4) Triglyceride (TG) levels ≥ 3.23 mmol/L. Based on this standard, the prevalence of MetS in GDM patients and preeclampsia (PE) patients was 13.6% and 26.2%, respectively [8]. GDM, as a metabolic disease unique to pregnancy, is characterized by increased insulin resistance and insufficient compensatory function of pancreatic beta cells in the middle and late stages of pregnancy. The pathogenesis involves the interaction of multiple factors such as fluctuations in pregnancy related hormone levels, genetic susceptibility, and dietary structure. It is worth noting that MetS and GDM have significant similarities in pathological and physiological aspects, including insulin resistance, abnormal lipid metabolism, endothelial dysfunction, and chronic inflammatory status. Bautista Castano *et al.* [9] confirmed through a retrospective cohort study involving 6887 samples that pregnant women with pre pregnancy overweight (BMI ≥ 25 kg/m^2) have a significantly higher risk of developing GDM compared to normal weight pregnant women (RR = 2.13). The prospective cohort study by Chatzi team [10] further revealed that the risk of developing late stage GDM in pregnant women diagnosed with MetS during early pregnancy increased by 3.17 times, with the most significant correlation between fasting blood glucose ≥ 5.6 mmol/L and GDM during early pregnancy (OR = 4.92). Pregnancy induced hypertension (HDCP), as a unique complication of the perinatal period, is characterized by multidimensional metabolic abnormalities including abnormal blood pressure, insulin resistance, endothelial damage, coagulation fibrinolysis system imbalance, and lipid factor disorder. Long term follow-up data shows that the incidence of postpartum metabolic syndrome and cardiovascular disease is significantly increased in HDCP patients. Srinivas *et al.* [11] found that the incidence of MetS in late pregnancy was significantly higher in patients with preeclampsia (PE) compared to healthy pregnant women, suggesting a potential pathological association between the two. The retrospective study by Ray's team [12] further confirms that pre pregnancy MetS increases the risk of placental dysfunction by 2.8 times, which often leads to serious complications such as HDCP and placental abruption. It should be emphasized that GDM and PE not only pose maternal and fetal risks during the perinatal period, but also significantly increase the long-term risk of postpartum cardiovascular and metabolic diseases. Therefore, it is recommended to implement early metabolic intervention for mothers with a history of GDM and PE, including comprehensive measures such as lifestyle adjustments, nutritional optimization, and dynamic monitoring of metabolic indicators, to reduce the risk of long-term metabolic syndrome related diseases.

In this case, the pregnant woman was diagnosed with GDM during mid pregnancy, and dietary guidance and exercise to control blood sugar had a good effect. MetS appeared after treatment with dexamethasone for threatened preterm birth. The metabolic syndrome related abnormalities in pregnant women with diabetes after glucocorticoid use may be related to the interaction of the following multiple factors. It is necessary to comprehensively analyze the physiological effects of glucocorticoid, the pathological basis of GDM, and the metabolic characteristics of pregnancy:

1) The interference of glucocorticoids on glucose metabolism. The exacerbation of insulin resistance in GCs promotes glucose production by activating key hepatic gluconeogenesis enzymes such as PEPCK and G6Pase, while inhibiting glucose uptake by adipose and muscle tissue (downregulating GLUT4 expression), leading to elevated blood glucose levels. GDM patients themselves have insulin resistance and insufficient beta cell compensation function, and GCs further exacerbate this pathological state, forming a vicious cycle; Impairment of β-cell function: Long term exposure to high-dose GCs may directly inhibit insulin secretion by pancreatic β-cells and mediate β-cell apoptosis through oxidative stress and inflammatory factors (such as IL-6, TNF-α), weakening blood glucose regulation.

2) The synergistic effect of lipid metabolism disorder. GCs activate hormone sensitive lipase (HSL), promote fat breakdown, release free fatty acids (FFA) into the bloodstream, and lead to high levels of free fatty acidemia. Accumulation of FFA in the liver and muscles can exacerbate insulin resistance and promote the synthesis of very low-density lipoprotein (VLDL), leading to hypertriglyceridemia. GCs activate the 11 β-HSD1 enzyme in visceral adipose tissue, increase local cortisol activity, and promote visceral fat deposition, while adipokines such as leptin and resistin secreted by visceral fat further worsen metabolic disorders.

3) The amplification effect of inflammation and oxidative stress. GDM patients themselves have elevated levels of inflammatory factors (such as TNF-α, IL-6) derived from adipose tissue and placenta. Although GCs have short-term anti-inflammatory effects, long-term use may promote infiltration of inflammatory cells in adipose tissue (such as macrophage polarization), exacerbate systemic inflammatory responses, and interfere with insulin signaling pathways (such as inhibition of IRS-1 phosphorylation). Oxidative stress is enhanced, and GCs can reduce the activity of antioxidant enzymes such as SOD and GPx, increase the generation of reactive oxygen species (ROS), lead to mitochondrial dysfunction and DNA damage, exacerbate insulin resistance and endothelial damage.

4) Water sodium retention and blood pressure regulation imbalance. Renin angiotensin system (RAS) activation: GCs enhance angiotensinogen expression and sodium reabsorption, leading to increased blood volume and peripheral vascular resistance, further elevating blood pressure. Pregnant women with GDM combined with chronic inflammation and endothelial dysfunction may be more sensitive to the pressor effect of GCs. Endothelial dysfunction: GCs inhibit the synthesis of nitric oxide (NO), promote the release of endothelin-1 (ET-1), exacerbate

endothelial dependent vasodilation dysfunction, and increase the risk of gestational hypertension and long-term cardiovascular disease.

5) The specificity of metabolism during pregnancy. The synergistic effect of placental hormones, such as progesterone and placental prolactin (hPL) secreted by the placenta during pregnancy, has an antagonistic effect on insulin. The synergy between GCs and these hormones may amplify insulin resistance effects. Fetal maternal metabolic competition: GCs are transported to the fetus through the placenta, which may affect fetal metabolic programming. At the same time, the mother needs to maintain a high metabolic state to meet the nutritional needs of the fetus. The application of GCs may disrupt this balance and accelerate the development of metabolic abnormalities.

4. Conclusions

The mechanism of metabolic syndrome (GMS) induced by glucocorticoids (GCs) in pregnant women with diabetes mellitus (GDM) involves multiple pathophysiological interactions. In this case, the patient experienced acute hyperglycemia (fasting blood glucose 13.3 mmol/L) and severe hypertriglyceridemia (TG 18.2 mmol/L) 24 hours after receiving dexamethasone at 30 + 6 weeks of pregnancy, consistent with the typical pattern of GC induced metabolic deterioration proposed by Bautista Castano *et al.* [9]. GCs further exacerbate the inherent insulin resistance in GDM patients by activating key gluconeogenesis enzymes (such as PEPCK) and inhibiting peripheral tissue GLUT4 expression [10]. In addition, the accumulation of visceral fat and release of FFA mediated by GCs can amplify oxidative stress response through inflammatory factors such as TNF-α and IL-6, leading to impaired beta cell function [11]. Clinical management needs to balance the benefits of GCs in promoting fetal lung maturation with metabolic risks. In this case, timely discontinuation of medication and insulin combined lipid-lowering treatment successfully reversed metabolic disorders, confirming the necessity of multidisciplinary intervention. Here, we call for the management of high-risk pregnant women with MetS to achieve the following points:

1) Highly value pregnant women diagnosed with GDM and HCDP during pregnancy, strengthen supervision, intervene early, and reduce the incidence of MetS and long-term cardiovascular and metabolic disorders; 2) Strictly control the dosage and timing of GCs when diagnosing GDM and HCDP in pregnant women, and adjust the insulin dosage in a timely manner for GDM patients; 3) Assess metabolic status before medication; During the medication period, blood glucose monitoring should be conducted at least 4 times a day; 4) Timely treatment of MetS, discontinuation of medication, rapid fluid replacement, glucose control, blood pressure reduction, electrolyte correction, multidisciplinary consultation, comprehensive management of patients; 5) High risk individuals using glucocorticoids in MetS should be alert to acute metabolic decompensation, and it is recommended to establish a full cycle management pathway of "pre medication evaluation monitoring post medication follow-up". 6) For patients who need to receive medium and high

doses of GCs or have risk factors for diabetes, it is recommended to conduct blood glucose screening and more strict blood glucose control. The treatment methods include changing lifestyle, using oral medication, and insulin therapy.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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