

Evaluation of Thyroid Profile in Gestational Diabetes Mellitus

Lakshman Lal¹, Atima Bharti¹, Rajiv Kumar Mahli², Arshi Perween²

¹Professor and Head, Department of Biochemistry, MGM, Jamshedpur, Jharkhand, India, ²Assistant Professor, Department of Obstetrics and Gynecology, RIMS, Ranchi, Jharkhand, India, ³Tutor, Department of Biochemistry, MGM, Jamshedpur, Jharkhand, India, ⁴Senior Resident, Department of Obstetrics and Gynecology, RIMS, Ranchi, Jharkhand, India

Abstract

Introduction: Gestational diabetes mellitus (GDM) is when women without diabetes develop high blood sugar level during pregnancy.

Materials and Methods: This study comprised 100 patients with GDM selected from Obstetrics and Gynaecology Department of Rajendra Institute of Medical Sciences, Ranchi. All these patients were clinically euthyroid at the time of assessment and there was no history of thyroid disorder.

Results: Highest serum thyroid stimulating hormone value is 26.0 (μ IU/ml) and lowest values <0.2 (μ IU/ml) among GDM patients. Highest values of free T4 is 15.0 (μ g/dl) and lowest value is 0.30 (μ g/L). Value of glycosylated hemoglobin in gestational diabetes is in the range of 4.4-12.8% which is statistically significant. 100 GDM patients were studied. 19% patients had raised thyroid hormone, 23% had low level, and 58% patients were euthyroid.

Conclusion: The incidence of hypothyroidism is more in GDM compared to hyperthyroidism.

Key words: Gestational diabetes mellitus, Hypothyroidism, Thyroid stimulating hormone

INTRODUCTION

Gestational diabetes mellitus (GDM) is when women without diabetes develop high blood sugar level during pregnancy.¹ It increases the risk of preeclampsia, depression, and requiring a cesarean section. Babies born to mother with poorly treated GDM are at increased risk of being too large, having hypoglycemia, and jaundice. If untreated can result in still birth. Children are higher risk of being overweight and developing Type 2 diabetes in later life.¹ GDM affects 3-9% of pregnancies depending on the population studied.² It affects 1% of those under the age of 20 and 13% of those over the age of 44. Diabetes and thyroid disease are two common endocrine disorders observed in the adult population with insulin and thyroid

being intimately involved in cellular metabolism and thus excess or deficit of either of these hormone could result in functional derangement of the other.

In euthyroid individuals with diabetes mellitus the serum T3 levels, basal thyroid stimulating hormone (TSH) level, and TSH response to thyrotropin releasing hormone (TRH) may all be influenced by glycemic status.³ Poorly controlled diabetes may also result in impaired TSH response to TRH or loss of normal nocturnal TSH peak. TSH response may be normalized with improvement in glycemic status but even with good diabetic control the normal nocturnal TSH peak may not be restored in C-peptide negative patients, i.e., those with totally absent pancreatic beta-cell function.⁴

MATERIALS AND METHODS

Our study comprised 100 patients with GDM selected from Obstetrics and Gynecology Department of Rajendra Institute of Medical Sciences, Ranchi. These patients were both insulin dependent and insulin independent diabetic being treated either by insulin injection or oral sulfonylurea drugs. Patients with clinical and biochemical evidence of

Access this article online



www.ijss-sn.com

Month of Submission : 10-2016
Month of Peer Review : 11-2016
Month of Acceptance : 11-2016
Month of Publishing : 12-2016

Corresponding Author: Dr. Lakshman Lal, 21 Ashashree Garden, Near RK Mission Ashram, Morabadi, Ranchi, Jharkhand, India.
E-mail: lakshmanlal37@gmail.com

hepatic or renal dysfunction as well as those with other significant diabetic complications were excluded from the study. All those patients were clinically euthyroid at the time of assessment and had no history of suggestive disease.

Blood Glucose Estimation

Fasting blood was collected with a caution to avoid any hemolysis. Glucose estimation was done by glucose oxidase/peroxidase method.

Calculation:

$$\text{Total glucose in mg/dl} = \frac{\text{Absorbance of the test sample}}{\text{Absorbance of the standard}} \times 100$$

Thyroid function test will be done by enzyme-linked immunosorbent assay method.

Quantitative determination of glycosylated hemoglobin in blood by ion exchange resin method.

RESULTS

1. Blood sugar level in GDM. Blood sugar level (mg/dl)

GDM	Fasting		2 h postprandial	
	Range	Mean±SD	Range	Mean±SD
Diabetes	100-200	124.57±27.77	130-330	162.5±34.24

SD: Standard deviation

Highest fasting blood sugar level was 210 mg/dl and highest postprandial was 330 mg/dl.

2. Serum TSH in GDM was in the range of 0.10-26.0 (μIU/ml) mean 5-34 (Figure 2)
3. Serum T4 in GDM was in the range of 0.50-14.2 (μg/dl) mean was 7.41 (μg/dl)
4. Serum T3 in GDM was in the range of 30-280 (μg/dl) mean 121.20
5. Serum FT4 in GDM was in the range of 0.30-15.0 (μg/dl) mean 1.77 (μg/dl) (Figure 1)
6. Glycosylated hemoglobin in GDM was in the range of 4.4-12.8% mean 7.22%
7. 100 GDM patients were studied. 19 patients had raised thyroid hormone, 23 patients had low level, and 58 patients were euthyroid.

DISCUSSION

GDM occurs in 2-9% of all pregnancies^{5,6} and associated with substantial rates of maternal and paternal complications. The risk of perinatal mortality is not increased but the risk of macrosomia is high. Other perinatal risks include

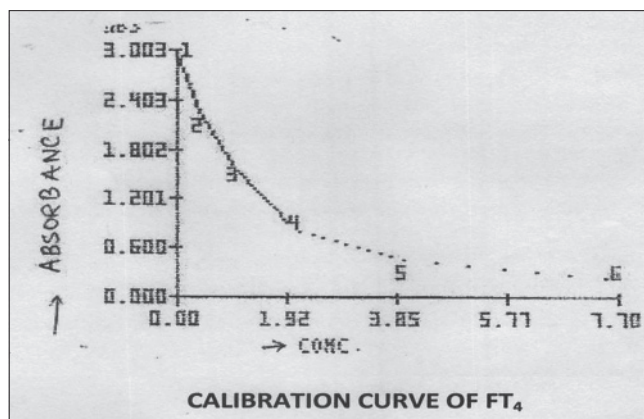


Figure 1: Calibration curve of FT₄

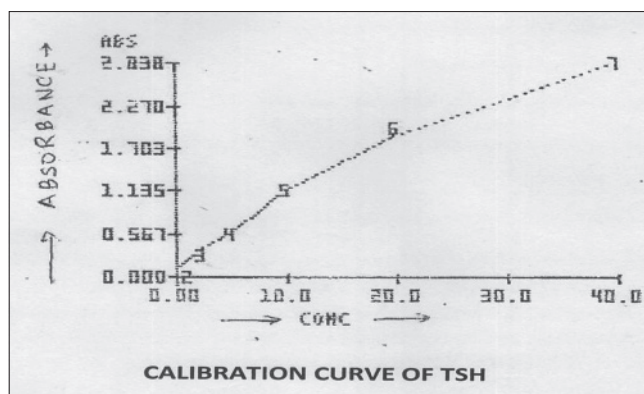


Figure 2: Calibration curve of thyroid stimulating hormone

shoulder dystocia, birth injuries such as bone fractures, nerve palsies, and hypoglycemia. Gestational diabetes is strong risk factor for diabetes in later life.⁸

Overtly abnormal amounts of thyroid hormone either excessive because of Grave's thyrotoxicosis or deficient because of hypothyroidism can have profound effect on glucose metabolism and insulin secretion. The resulting increase insulin resistance, glucose intolerance, and dyslipidemia are usually reversible when normal thyroid hormone levels are restored.⁹⁻¹¹ It is well known that marked insulin resistance progressively increases with gestational age during pregnancy.¹² Because of similar effects of thyroid hormone and pregnancy on glucose metabolism, it seems reasonable to posit that women either abnormally high or low level of thyroid hormone would be more likely to develop gestational diabetes. In our study, patients with GDM were evaluated for the functional status of thyroid gland. Among 100 GDM patients investigated 42% had abnormal thyroid hormone level. These finding show a high incidence of abnormal thyroid hormone level (low or raised) in diabetic population. This observation is in agreement with report of Smithson.^{13,14} Mean of serum T4 in GDM was 7.41 and SD ± 2.78. This finding is in accordance with Saunders *et al.*¹⁵

Glycosylated hemoglobin gives average blood glucose level of preceding 90 days. In uncontrolled or poorly control diabetes, there is an increase glycosylation of number of proteins including hemoglobin and α -crystalline of lenses. During diabetes excess glucose present in blood reacts with hemoglobin.¹⁶ In our study, the mean of glycosylated hemoglobin in GDM was 7.22%. Thyroid dysfunctions and autoantibodies in early pregnancy are associated with increased risk of GDM and adverse birth outcome.¹⁷ Thyroid antibodies and subclinical hypothyroidism occur more frequently in pregnant women with insulin-dependent diabetes mellitus than in healthy pregnant women.¹⁸

CONCLUSION

In our study, high incidence of abnormal thyroid hormone level was noted among GDM patients. Recent publications have confirmed that gestational diabetes is a disease with adverse perinatal outcome, and is susceptible to effective management. Hence, during pregnancy thyroid evaluation and blood sugar estimation should be included in antenatal routine investigations. Women with GDM should have an annual fasting glucose performed for life and referred for prepregnancy counseling if contemplating a further pregnancy.

REFERENCES

- Gestational Diabetes NIDDK September; 2014. Available at <http://www.https://en.m.wikipedia.org>. [Last retrieved on 2016 Jul 31].
- Donovan PJ, McIntyre HD. Drugs for gestational diabetes. *Aust Prescr* 2010;33:141-4.
- Schlienger JL, Anceau A, Chabrier G, North ML, Stephan F. Effect of diabetic control on the level of circulating thyroid hormones. *Diabetologia* 1982;22:486-8.
- Coiro V, Volpi R, Marchesi C, Capretti L, Speroni G, Caffarri G, *et al.* Influence of residual C-peptide secretion on nocturnal serum TSH peak in well-controlled diabetic patients. *Clin Endocrinol (Oxf)* 1997;47:305-10.
- Hoffman L, Nolan C, Wilson JD, Oats JJ, Simmons D. Gestational diabetes mellitus—management guidelines. The Australasian Diabetes in Pregnancy Society. *Med J Aust* 1998;169:93-7.
- American College of Obstetrician and Gynaecologist. Clinical Management Guidelines for Obstetric Gynaecologist. ACOG Practice Bulletin No. 30. Washington, DC: American College of Obstetrician and Gynaecologist; 2001.
- Blank A, Grave GD, Metzger BE. Effects of gestational diabetes on perinatal morbidity reassessed. Report of the International Workshop on Adverse Perinatal Outcomes of Gestational Diabetes Mellitus, December 3-4, 1992. *Diabetes Care* 1995;18:127-9.
- O'Sullivan J. The Boston gestational diabetes studies. In: Sutherland HW, Stowers JN, Pearson DW., Editors. *Carbohydrate Metabolism in Pregnancy and the Newborn*. London: Springer-Verlag; 1989. p. 287-94.
- Handisurya A, Pacini G, Tura A, Gessl A, Kautzky-Willer A. Effects of T4 replacement therapy on glucose metabolism in subjects with subclinical (SH) and overt hypothyroidism (OH). *Clin Endocrinol (Oxf)* 2008;69:963-9.
- Tene C, Zárate A, Basurto L, Islas S, Revilla C, Ochoa R, *et al.* Correction of insulin resistance in methimazole-treated patients with Graves disease. *Rev Invest Clin* 2001;53:531-5.
- Al-Shoumer KA, Vasanthy BA, Al-Zaid MM. Effects of treatment of hyperthyroidism on glucose homeostasis, insulin secretion, and markers of bone turnover. *Endocr Pract* 2006;12:121-30.
- Lind T, Billewicz WZ, Brown G. A serial study of changes occurring in the oral glucose tolerance test during pregnancy. *J Obstet Gynaecol Br Commonw* 1973;80:1033-9.
- Smithson MJ. Screening for thyroid dysfunction in a community population of diabetic patients. *Diabet Med* 1998;15:148-50.
- Suzuki Y, Nanno M, Gemma R, Tanaka I, Taminato T, Yoshimi T. The mechanism of thyroid hormone abnormalities in patients with diabetes mellitus. *Nihon Naibunpi Gakkai Zasshi* 1994;70:465-70.
- Saunders J, Hall SE, Sönksen PH. Thyroid hormones in insulin requiring diabetes before and after treatment. *Diabetologia* 1978;15:29-32.
- Sampson MJ, Gopaul N, Davies IR, Hughes DA, Carrier MJ. Plasma F2 isoprostanes: Direct evidence of increased free radical damage during acute hyperglycemia in Type 2 diabetes. *Diabetes Care* 2002;25:537-41.
- Karakosta P, Algakis D, Georgiou V, Roumeliotaki T, Fthenore E, Vassilaki M, *et al.* Thyroid dysfunction and autoantibodies in early pregnancy are associated with increased risk of gestational diabetes and adverse birth outcome. *J Clin Endocrinol Metab* 2012. 97:4464-72.
- Olivieri A, Valensise H, Magnoni F, Medda E, De Angelis S, D'Archivio M, *et al.* High frequency of antithyroid autoantibodies in pregnant women at increased risk of gestational diabetes mellitus. *Eur J Endocrinol* 2000;143:741-7.

How to cite this article: Lal L, Bharti A, Mahli RK, Perween A. Evaluation of Thyroid Profile in Gestational Diabetes Mellitus. *Int J Sci Stud* 2016;4(9):135-137.

Source of Support: Nil, **Conflict of Interest:** None declared.