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Original Research Article

Study of post hypoglycaemic hyperglycemia on patients posted for surgery (on the day of surgery)

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Abstract: Diabetes Mellitus is one of the common non-communicable diseases and is found to be one of the top five global causes of premature death. It is a metabolic disorder associated with increased risk of perioperative infection and postoperative cardiovascular morbidity and mortality. Strict glycemic control is considered as a perioperative goal in surgical patients. But there is no overall consensus on the optimal perioperative management of the diabetic patient. This case control study is done to understand the incidence of post hypoglycaemic hyperglycemia in diabetic patients posted for surgery (on the day of surgery). 50 participants were selected, 30 were diabetic patients and 20 were non diabetic control group who were all planned for surgery. Their blood sugar levels were estimated in pre dinner, midnight and fasting state. These values give a clear picture on the increased incidence of post hypoglycaemic hyperglycemia in diabetics than the control non- diabetics posted for surgery. Thus this study helps in calculating the insulin dose in pre operative period. This helps in blood sugar control in diabetics posted for surgery and thereby decrease the perioperative morbidity and mortality.
Keywords: Surgery, post hypoglycaemic hyperglycemia, fasting blood sugar, perioperative outcome.

INTRODUCTION:

Diabetes Mellitus is one of the common non-communicable diseases and is found to be one of the top five global causes of premature death. It is a metabolic disorder associated with increased risk of perioperative infection and postoperative cardiovascular morbidity and mortality [1, 2]. Strict glycemic control is considered as a perioperative goal in surgical patients [3–4]. But there is no overall consensus on the optimal perioperative management of the diabetic patient [5–6]. Surgery is a stress inducing factor for diabetics which lead to increase in cortisol and catecholamine levels to [7, 8]. Increased cortisol and catecholamines reduce insulin sensitivity, while heightened sympathetic activity reduces insulin secretion while simultaneously increasing growth hormone and glucagon secretion [9, 10]. Surgery changes normal metabolic patterns and trigger gluconeogenesis, glycogenolysis, proteolysis, lipolysis, and ketogenesis ultimately resulting in hyperglycemia and ketosis [11].

Diabetes Mellitus is one of the common diseases complicating surgery. Patients with diabetes have a higher incidence of morbidity and mortality following surgery. With increasing number of diabetic patients undergoing surgery and increased risk of diabetic complications, strict perioperative assessment and management becomes important. Mortality and post operative infections are higher in diabetics than non diabetics. Strict control of serum glucose is important to minimize infection [12]. Perioperatve hyperglycemia increases risk of postoperative mortality, cardiovascular, respiratory, neurologic and infectious morbidity [13, 14].

Good control of blood sugar is important on the day of surgery on anaesthetic point and also to decrease the perioperative morbidity, mortality and post surgical complications.

This study on the blood sugar levels at pre dinner and fasting state gives a clear picture on pre operative insulin dose calculation and blood sugar control on the day of surgery.

AIM AND OBJECTIVE:

1. The objective of the study is to find the incidence of post hypoglycaemic hyperglycemia in patients posted for surgery.
2. The purpose of the study is to correct the incidence of fasting hyperglycemia in patients undergoing surgery and thus to improve the outcome of surgery.

MATERIALS AND METHODS:

This case- control study was conducted in Sree Balaji Medical College and hospital for a period of 2 months. 50 participants were selected 30 were diabetic patients and 20 were non diabetic control group who were all planned for surgery. This study was approved by the institutional ethical committee. After getting informed consent from the participants the fasting blood samples were collected by standard aseptic techniques at 2AM and 6AM. Serum was separated. Fasting blood sugar was estimated by GOD-POD method using Mindray kit in autoanalyser. Data collected was analysed using SPSS package.

RESULTS:

Table-1: Denote Fasting blood sugar on the day of surgery is higher in diabetics than non diabetics.

	GROUP 1	GROUP 2	GROUP 3	NON DIABETICS
PRE DINNER BLOOD SUGAR mg/dl	175.6	155.7	164.5	107.43
BLOOD SUGAR AT 2AM mg/dl	50.8	160.1	195.1	98

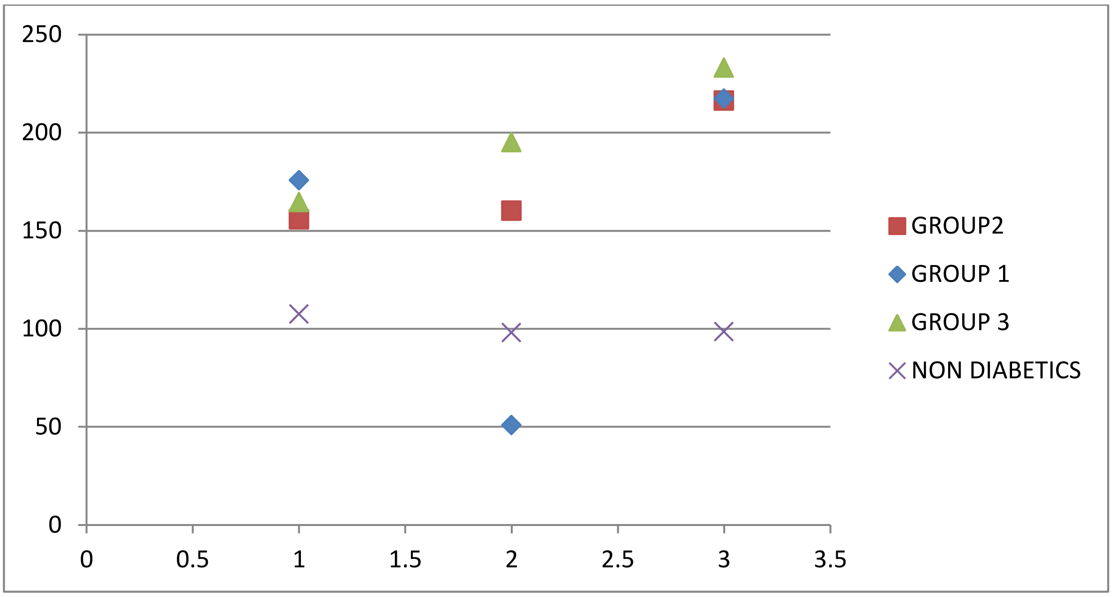

Fig-1: shows fall /rise in blood sugar at 2AM which is been followed by increased fasting blood sugar value at 6AM.

By this study, it is been observed that the participants had various level of fluctuation in blood sugar at 2AM and 6AM. The blood sugar values at 2AM and 6AM was divided in to three groups (group 1, group 2, group 3) according to the range in blood sugar values. The mean blood sugar values at 2AM were found to be 50.80, 160.10, and 195.10 depending on the predinner insulin dose. The fasting blood sugar values at 6AM were found to be high 217.20 ± 15mg/dl. By analysing these values, P- Value is found to be less than 0.001, which is extremely statistically significant and confidence interval is 95%.

The control study group (non- diabetic patients posted for surgery) showed mean blood value of 107.43mg/dl at pre- dinner time and 98.50mg/dl at fasting state. P- Value is 0.0030, very statistically significant, and confidence interval 95%.

DISCUSSION:

The stress response to surgery is mainly due to increased secretion of pituitary hormones and activation of the sympathetic nervous system [12]. Patients experiencing surgery develop a hypermetabolic stress which results in hyperglycemia and insulin resistance. This response leads to increased level of endogenous hepatic glucose production eventhough insulin stimulated peripheral glucose uptake is reduced. Cortisol secretion from the adrenal cortex increases due to surgery, as a result of stimulation by ACTH. Cortisol level increases from baseline values of around 400 nmol litre^{-1},and may reach >1500 nmol litre^{-1}

This increased cortisol affects carbohydrate, fat and protein metabolism. It also promotes protein breakdown and gluconeogenesis in the liver. All these results in hyperglycemia and protein loss.

Risk of infection is increased with severe hyperglycemia, because of monocyte abnormalty and polymorphonuclear neutrophil function, decreased intracellular bactericidal activity, and glycosylation of immunoglobulins [15, 16]. Hyperglycemia affects blood coagulation process which leads to increase in prothrombin fragments and D-dimers which finally results in platelet aggregation and thrombosis [17].

Hyperglycemia leads to inflammation and activation of proinflammatory cytokines. Ischemic preconditioning which is an intrinsic myocardial protective mechanism is affected by poor blood sugar control [18]. Thus, hyperglycemia interacts at the cellular and biochemical level in many ways, which may be responsible for the adverse effects associated with hyperglycemia.

In the preoperative period the glycemic control is aimed to achieve fasting plasma glucose of 100-200mg% and a post prandial plasma glucose 140-200mg%.

Insulin is the drug of choice in peri operative blood sugar control .Insulin provides protection from endothelial dysfunction, which may prevent organ failure and death [19]. The anti-inflammatory effects of insulin, decreases levels of proinflammatory cytokines, adhesion molecules, and acute phase proteins. Insulin also has anti-thrombotic, anti-atherogenic properties [20–22]. Insulin treatment causes arterial vasodilation and capillary recruitment, via activation of the nitric oxide pathway [23] and improves myocardial perfusion [24].

The midnight fall in blood sugar level evokes a counter regulatory hormone response and causes increase in blood sugar level. This rebound hyperglycemia, results in increase in predinner dose of insulin which worsens the situation. In this type of post hypoglycemichyperglycemia the predinner insulin dose has to be reduced. Similar type, Somogyi phenomenon is also to be kept in mind.

Few diabetics present with predinner and 2AM blood sugar value to be similar but the 6AM fasting blood sugar to be high. This could be due to early morning surge of growth hormone and cortisol. This type of blood sugar presentation is Dawn phenomenon and this can be corrected by administration of intermediate acting insulin at bedtime. Very few patients present with increased blood sugar level at 2AM and 6AM. This is found to be due to inadequate dose of predinner insulin dose or due to waning away of the insulin activity. This is corrected by giving small dose of regular insulin before dinner along with the intermediate acting insulin.

CONCLUSION:

The present study shows a clear picture about the incidence of post hypoglycaemic hyperglycemia mainly due to increased counter regulatory hormones. Maintaining good blood sugar control during perioperative period results in better outcome following surgery. Diabetic medicines need to be changed before and after surgery to maintain blood sugar control. Thus management of diabetic patients in perioperative period is very important and need strict monitoring and better understanding among physician, surgeons and anaesthetists. This study gives information about the preoperative insulin dose. Thus metabolic assessment and management of diabetes must begin early and maintenance of euglycemia during perioperative period will reduce the morbidity and mortality.

REFERENCES:

1. Malone DL, Genuit T, Tracy JK, Gannon C, Napolitano LM; Surgical site infections: reanalysis of risk factors. J Surg Res 2002; 103(1):89.
2. Lee TH, Marcantonio ER, Mangione CM, Thomas EJ, Polanczyk CA, Cook E *et al.;* Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. Circulation 1999; 100(10):1043.
3. Van G, den Berghe MD, Wouters P, Weekers F, Verwaest C, Bruyninckx F *et al.;* “Intensive insulin therapy in critically ill patients,” The New England Journal of Medicine, 2001; 345(19): 1359–1367.
4. Coursin DB, Murray MJ, “How sweet is euglycemia in critically ill patients?” Mayo Clinic Proceedings, 2003; 78(12) 1460–1462. ·
5. Hirsch B, McGill JB; “Role of insulin in management of surgical patients with diabetes mellitus,” Diabetes Care, 1990; 13(9): 980–991.
6. Gill GV, Alberti KGMM; “The care of the diabetic patient during surgery,” in International Textbook of Diabetes Mellitus, John Wiley & Sons, New York, NY, USA, 2003.
7. Zaloga GP; “Catecholamines in anesthetic and surgical stress,” International Anesthesiology Clinics, 1988; 26(3): 187–198.
8. Madsen SN, Engquist A, Badawi I, Kehlet H; “Cyclic AMP, glucose and cortisol in plasma during surgery,” Hormone and Metabolic Research, 1976; 8(6): 483–485.
9. Werb MR, Zinman B, Teasdale SJ, Goldman BS, Scully HE, Marliss EB; “Hormonal and metabolic responses during coronary artery bypass surgery: role of infused glucose,” The Journal of Clinical Endocrinology & Metabolism, 1989; 69(5): 1010–1018.
10. Wright PD, Johnston IDA; “The effect of surgical operation on growth hormone levels in plasma,” Surgery, 1975; 77(4): 479–486.
11. Changes in normal metabolic patterns due to surgery trigger gluconeogenesis, glycogenolysis, proteolysis, lipolysis, and ketogenesis ultimately resulting in hyperglycemia and ketosis
12. Desborough JP, Hall GM; Endocrine response to surgery. In: Kaufman L. Anaesthesia Review, Edinburgh*:* Churchill Livingstone*,* 1993*; 10:* 131*–48.*
13. Outtara A, Lecomte P, Le Manach Y, Landi M, Jacqueminet S, Platonov I, *et al.;* Poor intraoperative blood glucose control is associated with a worsened hospital outcome after cardiac

surgery in diabetic patients. Anesthesiology. 2005; 103(4):687–94.

14. Doenst T, Wijeysundera D, Karkouti K, Zechner C, Maganti M, Rao V *et al.;* Hyperglycemia during cardiopulmonary bypass is an independent risk factor for mortality in patients undergoing cardiac surgery. J Thoracic & Cardiovascular Surgery. 2005; 130(4):1144–50.
15. Rassias AJ, Marrin CA, Arruda J, Whalen PK, Beach M, Yeager MP; Insulin infusion improves neutrophil function in diabetic cardiac surgery patients. Anesthesia Analgesia. 1999; 88(5):1011–6.
16. Rassias AJ, Givan AL, Marrin CA, Whalen K, Pahl J, Yeager MP; Insulin increases neutrophil count and phagocytic capacity after cardiac surgery. Anesthesia Analgesia. 2002; 94(5):1113–9.
17. Lefebvre PJ, Scheen AJ; The postprandial state and risk of cardiovascular disease. Diabetic Med. 1998; 15(Suppl 4):S63–8.
18. Kersten JR, Toller WG, Gross ER, Pagel PS, Warltier DC; Diabetes abolishes ischemic preconditioning: role of glucose, insulin, and osmolality. Am J Physiol Heart Circ Physiol. 2000; 278(4):H1218–24.
19. Langouche L, Vanhorebeek I, Vlasselaers D, Vander Perre S, Wouters PJ, Skogstrand K.*et al.;* Intensive insulin therapy protects the endothelium of critically ill patients. J Clinical Investigation. 2005; 115(8):2277–86.
20. Dandona P, Mohanty P, Chaudhuri A, Garg R, Aljada A; Insulin infusion in acute illness. J Clinical Investigation. 2005; 115(8):2069–72.
21. Collet JP, Montalescot G, Vicaut E, Ankri A, Walylo F, Lesty C *et al.;* Acute release of plasminogen activator inhibitor-1 in ST-segment elevation myocardial infarction predicts mortality. Circulation. 2003; 108(4):391–4.
22. Marfella R, Siniscalchi M, Esposito K, Sellitto A, De Fanis U, Romano C *et al.;* Effects of stress hyperglycemia on acute myocardial infarction: role of inflammatory immune process in functional cardiac outcome. Diabetes Care. 2003; 26(11):3129–35.
23. Coggins M, Lindner J, Rattigan S, Jahn L, Fasy E, Kaul S *et al.;* Physiologic hyperinsulinemia enhances human skeletal muscle perfusion by capillary recruitment. Diabetes. 2001; 50(12):2682–90.
24. Laine H, Nuutila P, Luotolahti M, Meyer C, Elomaa T, Koskinen P *et al.;* Insulin-induced increment of coronary flow reserve is not abolished by dexamethasone in healthy young men. J Clinical Endocrinol Metabol. 2000; 85(5):1868–73.
25. Gao F, Gao E, Yue TL, Ohlstein EH, Lopez BL, Christopher TA *et al.;* Nitric oxide mediates the antiapoptotic effect of insulin in myocardial ischemia-reperfusion: the roles of PI3-kinase, Akt, and endothelial nitric oxide synthase phosphorylation. Circulation. 2002; 105(12):1497–502.