Journal of Pharmaceutical Research International

33(59B): 240-248, 2021; Article no.JPRI.80267
ISSN: 2456-9119
(Past name: British Journal of Pharmaceutical Research, Past ISSN: 2231-2919, NLM ID: 101631759)

Preoperative Management of Diabetic Patients, an Overview

Hashem Bark Awadh Abood [a*#], Ali Ahmed Al-Qadhi [b≡], Ossama Saed Alhindi [c≡], Mohammed Jaafar A. Al Sheef [dφ], Abdullah Saleh Salman Alwadani [e], Nura Jamal T. Alsaddah [f], Sulaiman Mohammed Mimish [g], Abdulelah Ahmed Alabdulwahab [hφ], Ammar Abdullah Basardah [i], Mohamed Eltahir Musa [i], Abdullah Suwaymil Alrashdi [j], Khalid Mohsen Alzahrani [k], Bassam Ibrahim AlJohani [l] and Batool Mohammed S. Alshehri [b]

[a] Dr Samir Abbas Hospital , Jeddah King Fahad hospital, Albaha, Saudi Arabia.
[b] Imam Abdulrahman Bin Faisal University, Dammam, Saudi Arabia.
[c] Ibn Sina National College, Jeddah, Saudi Arabia.
[d] Taibah University, Saudi Arabia.
[e] GP, Algrah PHC, Saudi Arabia.
[f] MCH Makkah, Saudi Arabia.
[g] Batterjee Medical College, Saudi Arabia.
[h] King Faisal University, Saudi Arabia.
[i] General practitioner, Care National Hospital, Riyadh, Saudi Arabia.
[j] KKH, Ha'il, Saudi Arabia.
[k] King Fahad Hospital, Jeddah, Saudi Arabia.
[l] University of Hail, Hail, Saudi Arabia.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/JPRI/2021/v33i59B34375

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/80267

Review Article

Received 14 October 2021
Accepted 16 December 2021
Published 17 December 2021

[#] Consultant General Surgery;
[≡] Medical Intern;
[φ] Intern;
**Corresponding author*

ABSTRACT

Glycemic control is critical in the perioperative setting, especially in diabetic patients. The consequences of surgical tension and anesthesia on blood sugar levels are distinct, and should be considered in order to maintain optimal glycemic control. Each stage of surgery presents its own set of challenges in terms of keeping glucose levels within the target range. Furthermore, there are some surgical conditions that necessitate specific glucose management protocols. Authors hope to highlight the most crucial factors to consider when developing a perioperative diabetic regimen, while still allowing for specific adjustments based on sound clinical judgement. Overall, by carefully managing glycemic control in perioperative patients, we may be able to reduce morbidity and mortality while improving surgical outcomes.

Keywords: Glycemic control; diabetes; diabetic patients.

1. INTRODUCTION

Diabetes is a long-term condition characterized by the body's inability to appropriately digest blood glucose. It's either insulin-dependent or insulin-independent. Diabetes mellitus type 1 (T1DM) is characterized by a deficiency in the beta cells' insulin secretion [1]. Pancreatitis is an autoimmune disease that affects the pancreas, causing the destruction of the beta cells that secret insulin [1]. While Peripheral insulin resistance and an insulin-secretory malfunction of varying severity characterize Type 2 diabetes [2].

Diabetes has well-defined prevalence and diagnosis criteria. In the United States, around 29.1 million people (nearly 9.3% of the total population) have diabetes. Around 27% of these 29.1 million cases, or 8.1 million, are undiagnosed [3]. Furthermore, according to a study conducted by the World Health Organization (WHO), diabetes affects an estimated 347 million individuals globally [4]. Between 2010 and 2030, developing nations are expected to see a 69% increase in adults with diabetes, whereas developed countries will see a 20% increase [5].

Diabetes mellitus (DM) causes a slew of systemic disorders that necessitate surgical intervention. It's noted that diabetic patients are subjected to surgical procedures. Procedures are performed at a higher rate in diabetics than in non-diabetics patients [6]. Hyperglycemia has a detrimental impact on the human body's whole organ system. The danger of a diabetes-related perioperative complication. Diabetic patients have a higher mortality rate than non-diabetics. The number of people receiving surgery has also climbed. Long-term blood glucose control lowers the rate of diabetes considering the seriousness of the complications [7-11].

Glucose metabolism is primarily controlled by the liver and pancreas. In peripheral tissues, glucose metabolism happens to a large extent. The liver absorbs glucose and stores it as glycogen, as well as performing gluconeogenesis and glycogenolysis. The catabolic hormones adrenaline, glucocorticoids, and growth hormone raise blood glucose levels, while the pancreatic beta cell secretes insulin in reaction to the elevated blood glucose level, which reduces blood glucose levels. Because peripheral tissues use glucose for energy, blood glucose levels are lower [12].

The autoimmune death of pancreatic islet b cells causes T1DM, which causes the body to lack its ability to release insulin. Because it most usually presents in childhood or adolescence, it is also known as juvenile-onset diabetes. Insulin is produced in the pancreatic beta cells and has a variety of tasks, the most important of which is to regulate glucose metabolism. Insulin is involved in glycogen production, lipogenesis, and growth, in addition to glucose consumption. Hyperglycemia occurs when glucose cannot be delivered into cells and adipose tissue due to a lack of insulin. Hyperglycemia is described as a fasting glucose level of more than 140 mg/dL, which can cause ketoacidosis and other serious metabolic problems. Patients with type 1 diabetes rely on exogenous insulin since they lack the ability to manufacture insulin on their own [13,14].

Adults above the age of 35 are more likely to develop T2DM. It's a complicated metabolic condition defined as a reduction in pancreatic insulin secretion, as well as varying contributions from diminished insulin action or insulin resistance in target tissues, primarily muscle and the liver. Obesity and an unhealthy life are two main risk factors for type 2 diabetes. If people with type 2 diabetes lose weight and follow a physician-directed weight loss program that involves rigorous diet control and exercise, they may not need to take oral anti-diabetic medicine or insulin [15].

Treatment of diabetes depends on its type as type 1 diabetes is managed using insulin. Rapid-acting (lispro), short-acting (regular, Lente), intermediate-acting (NPH), and long-acting (ultralente) insulin formulations are available. One to two injections of intermediate or long-acting insulin, with or without regular or lispro insulin, are used in standard insulin therapy. Multiple (three or more) daily injections of insulin, employing combinations of insulin, such as normal or lispro insulin three times daily, adjusted before meals, and NPH before sleep, or via continuous subcutaneous insulin infusion are referred to as intensive insulin therapy. Insulin dosage calculation is still based on guesswork. Patients with type 1 diabetes require an insulin dosage of 0.5 to 1.0 U/kg/d on average [16,17].

Sulfonylureas, meglitinides, biguanides, a-glucosidase inhibitors, and thiazolidinediones are some of the drugs used to treat T2DM. Sulfonylureas and meglitinides are known as oral hypoglycemic agents because they can produce hypoglycemia. The other drugs, known as oral anti-hyperglycemic agents, are used to treat a variety of diseases. Sulfonylureas and meglitinides cause the pancreatic beta cells to release more insulin, lowering blood glucose levels. Sulfonylurea medicines such as gliburide and glimepiride are routinely prescribed, whereas meglitinides include repaglinide and nateglinide. In the presence of insulin, biguanides (metformin) suppress hepatic glucose synthesis. These medicines also boost anaerobic glycolysis, improve glucose uptake and utilization in muscle, and reduce glucose absorption in the intestine [18-21].

1.1 Evaluation of Preoperative Glycemic Control in Patients with Diabetes

To avoid hyperglycemia and hypoglycemia, glycemic management is indicated prior to admission to surgery based on patient-specific objectives. During the hospital stay, glycemic variability should also be evaluated. Fasting, stress, infection, glucocorticoids, and other factors can cause glycemic fluctuation. During the anesthesia consultation, two criteria are used to assess glycemic control: I glycated haemoglobin (HbA1c); and (ii) blood sugar (glucose) levels. Hypoglycemia and hyperglycemia should be treated since they can have negative preoperative implications and cause the intervention to be delayed [22,23].

1.2 HbA1c

HbA1c, which measures glycemic control over the past three months, can be used to assess the quality of glycemic control prior to consultation and treatment adaption to specific goals . According to certain research, a high HbA1c level in a diabetic patient is linked to a higher risk of morbidity and death, as well as an increased risk of early postoperative infection and myocardial infarction. The risk increases by 40% for every one% increase in HbA1c. If HbA1c is more than 7.8%, the risk of sternal infection is 5-fold higher (OR = 5.3). In undiagnosed diabetes patients, HbA1c > 7% appears to have a negative predictive value. HbA1c levels and mean blood sugar levels have a link (. If HbA1c is very high (> 9%), surgery should be postponed (unless in an emergency) since it indicates a lack of glycemic control, exposing the patient to severe metabolic problems [24-26].

1.3 Recent Blood and Capillary Glycemic Values

Many studies have found a link between admission glycaemia (> 2 g/L or 11 mmol/L) and postoperative morbidity and mortality rates, as well as a 10-fold increased risk of problems when glycaemia is present before surgery. Blood sugar levels can be used to stratify the risk of sternal infection after cardiac surgery, for example: a blood sugar level of less than 1.80 g/L (10 mmol/L) prior to the intervention reduces the risk of infection, death and length of stay. Capillary blood sugar levels should be monitored during the preoperative consultation and in the days leading up to the intervention. Even if an appropriate HbA1c value is observed, a recent disequilibrium (hyper- or hypoglycemia) could affect perioperative care. The HbA1c/mean blood sugar relationship does not apply in this circumstance [27-32].

1.4 Identification of Episodes of Hypoglycemia

Hypoglycemia is a therapeutic concern in all diabetic patients, but it is especially prevalent in hospitalized diabetic patients due to more significant glycemic fluctuations even when the precise effect of anesthetic or surgery has yet to be determined, these states of insecurity can contribute to glucose regulation abnormalities, such as hypoglycemia. Treatment with insulin secretors (hypoglycemic sulphonamides/glinides) or insulin therapy frequently results in hypoglycemia. It primarily affects T1D patients; however it can also affect T2D diabetics at any moment. Hypoglycemia is caused by a conflict between a lack of glucose supply and poorly suited insulin or insulin-secretor treatment. These conditions are more common in the perioperative period, due to prolonged fasting or inconsistent meal consumption. It is critical to use caution when administering medicines that have a direct hypoglycemic effect.

Any inexplicable malaise in a diabetic patient should be treated as a hypoglycemic episode until proven otherwise, even if the blood sugar level at the time of measurement appears to be normal. Whatever the blood sugar level, serious hypoglycemia is defined by the requirement for help from another person. Some hypoglycemic episodes are more difficult to detect, especially when hypoglycemia occurs frequently, diabetes is a long-term condition, this scenario affects almost 40% of T1D patients, 10% of T2D patients on insulin, and a small%age of T2D patients taking oral anti-diabetic medications. [33,34]

1.5 Perioperative Management

Since the host's defenses against infection are weakened in hyperglycemic patients, they are more susceptible to infection. Hyperglycemia also slows wound healing by reducing wound tensile strength and interfering with collagen production. If blood glucose is not regulated, healing at the anastomotic site is substantially hampered. The risk of cerebrovascular infarction, and renal ischemia increases as the incidence of coronary artery disease rises in diabetics. The risk of hyperglycemic hyperosmolar non-ketotic states (HONKS) is higher in type 2 diabetes patients than diabetic ketoacidosis and hyperosmolar non-ketotic coma (HONC), which both have a higher mortality rate. Insulin resistance can also be caused by hyperglycemia due to glucose toxicity [35-41].

1.6 Preoperative Evaluation

Preoperative management of diabetic patients aims mainly to [42,43] :

(a)Lowering overall morbidity and mortality in patients
(b)Avoiding extreme hyperglycemia or hypoglycemia.
(c) Maintaining electrolyte and fluid balance in the body
(d) Ketoacidosis prevention.

The patient's diabetes condition and surgical risk factors should be assessed first. The anesthetic method to be utilized, the specifics of the surgery to be done, and laboratory values are all significant considerations. Potential concerns should be recognized, treated, and stabilized before surgery in the case of elective surgery. The major goal of preoperative evaluation is to assess metabolic control and any diabetes-related comorbidity, such as cardiovascular disease, autonomic neuropathy, and nephropathy. Diabetes individuals are more likely to experience silent myocardial ischemia. As a result, all diabetes patients should have their ECGs checked, although echocardiography and stress tests are frequently recommended. Diabetic autonomic neuropathy can cause perioperative hypotension, therefore look for resting tachycardia, peripheral neuropathy, and orthostatic hypotension. To rule out diabetic nephropathy, serum creatinine and creatinine clearance rate (CCR) should be assessed.[42]

1.7 Control of Blood Glucose

Some recent studies have revealed that oral hypoglycemic medications can be used to effectively manage perioperative diabetes; nevertheless, it should be noted that their side effect is the requirement of additional clinical monitoring. Because of inconsistent absorption and unpredictable blood glucose concentrations, subcutaneous insulin administration, it may be a less favored method. Intravenous infusion of rapid-onset soluble (short-acting) insulin, such as that found in Glucose-insulinpotassium (GIK) systems, is a safe and effective way to treat perioperative hyperglycemia.[43-44] To counteract the catabolic process that occurs as a result of surgery, we should give the patient enough insulin. Glucose is required to address both the increased metabolic needs resulting from surgical stress and the basic metabolic requirements.

Patients with type 2 diabetes on diet alone, don’t need insulin in their management, while those who use oral hypoglycemic agent (OHA) and need major surgery, they should stop oral hypoglycemic agent 48 hours before surgery and move to short acting insulin. In this case, the Alberti (GIK) regimen is relatively safe because it provides both insulin and glucose in the same solution. Metformin should not be discontinued because the risk of lactic acidosis is exceedingly minimal. Metformin should not be used in the morning [45-49].

Long-acting insulin should be stopped at least 24 hours before surgery for patients with type 1 diabetes. The use of intermediate-acting insulin can be continued until the night before surgery. Insulin should be administered to every type 1 diabetes patient having surgery. The glucose-insulin infusion is an effective way to avoid the negative metabolic effects of hunger and surgical stress. GIK infusion is a simple and effective approach to manage blood glucose levels, and it should be started prior to surgery. Throughout the perioperative period, blood glucose levels should be monitored often and correctly. The level of glucose should be controlled between 6.6 and 9.9 mmol/L [50].

Low blood sugar is a potentially fatal condition that is treated with oral carbohydrates, IV glucose, or glucagon administered via various routes. Glucagon is appealing to the diabetic population due to its ease of use and safe administration, which does not necessitate the use of a patent IV. Patients with low consciousness cannot safely consume the oral carbohydrates required to raise their blood sugar without risk of aspiration, and obtaining IV access in the diabetic population can be difficult, preventing prompt administration of IV glucose. In the event of IV medication extravasation, IV glucose also poses the risk of severe tissue necrosis. Glucagon is a reliable method of raising the patient's glucose levels and relieving severe hypoglycemia long enough for more definitive correction of the patient's glucose levels by mouth, especially when IV access is unavailable or has failed [51].

1.8 Anesthetic Considerations: [52-58]

The most stable analgesia and anesthetic should be used to decrease stress.

To minimise the hunger time, diabetic patients should be the first cases in the morning.

Diabetics may have both regional and general anesthetic, but regional one reduces stress.

Only appropriate analgesics should be utilized.

Catecholamine secretion is modulated by spinal or epidural anaesthesia, which prevents hyperglycemia and ketoacidosis. This effect could last for a long time after surgery.

Beta blocker medicines raise blood glucose levels in those who don't have diabetes, but they also wreak havoc on sugar control in diabetics and mask warning signs of hypoglycemia.

Patients with gastroparesis should be given a prokinetic medication prior to receiving general anaesthesia to reduce the risk of gastric acid aspiration.

Because consciousness is hampered by severe hypoglycemia, recovery fromgeneral anesthesia can be delayed.

It's possible you'll have trouble breathing (stiff joint syndrome). As a result, a complex intubation protocol should be used, and the case should be handled by a trained anesthetist.

1.9 Glucose, Electrolytes and Fluid Management

For basal energy needs and to avoid hypoglycemia, and ketosis during surgery, most experts recommend that diabetic patients be given roughly 5 g of glucose each hour. If the situation is extremely stressful, more glucose may be required. The concentration of dextrose in the IV fluid is determined by the expected time of treatment and the patient's limits. For example, for relatively brief or simple procedures, 5% dextrose in water (contains 50 g glucose per L) is suitable. Using 10% dextrose (contains 100 g glucose per L) at 50 mL per hour for prolonged procedures will prevent excessive fluid delivery. If fluid restriction is critical, a 20 or 50% dextrose solution can be administered through a central line. If extra fluids are required (for example, to replace blood loss they should not contain dextrose [59-60].

Pre and post-surgery, serum potassium levels should be checked. During surgery, a number of events can affect serum potassium levels. Insulin promotes potassium uptake into cells, but dehydration and hyperglycemia can cause potassium to leak out of cells and into the bloodstream. Due to the exchange of intracellular potassium for external hydrogen ions, acidosis can cause hyperkalemia. 10 to 15 mEq potassium should be provided per 500 mL of

10% glucose-containing liquids in diabetics with normal kidney function and normal blood potassium levels. This should be modified based on potassium levels in the blood [61,62].

Any other intraoperative fluids, such as 0.9% sodium chloride solution, should be glucose-free. Ringer's lactate solution should be avoided because lactate is a gluconeogenic precursor that is rapidly digested, resulting in a rise in blood glucose levels. Lactate, on the other hand, plays a little influence in acid-base disturbances [63].

1.10 Emergency Surgery

Most diabetic patients who require emergency surgery do not have good metabolic control at the time and may develop diabetic ketoacidosis (in type 1 diabetics) or hyperglycemic hyperosmolar syndrome (in type 2 diabetics). Patients must always be fully stabilized before anesthesia and surgery, with the exception of some life-threatening surgical circumstances (torrent and uncontrolled hemorrhage or profoundly impaired airway). If possible, surgery should be postponed for 4-6 hours to maximize metabolic condition. [51-64]

2. CONCLUSION

Diabetes mellitus is a historical chronic disease that affects the life of its patients, so proper dealing with diabetic patients is important. Operative intervention for diabetic patients requires a lot of preoperative assessment, and many parameters to be evaluated to achieve appropriate preoperative evaluation, treatment of complications, and good glycemic and metabolic control that results in an improved outcomes and reduce perioperative morbidity and death.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. [Guideline] Diagnosis and classification of diabetes mellitus. Diabetes Care. 2010 Jan;33 Suppl 1:S62-9.
2. Keller DM. New EASD/ADA Position Paper Shifts Diabetes Treatment Goals. Medscape Medical News. Available:http://www.medscape.com/viewarticle/771989. Accessed: October 15, 2012.
3. Centers for Disease Control and Prevention, National Diabetes Statistics Report: Estimates of Diabetes and Its Burden in the United States, US Department of Health and Human Services, Atlanta,Ga, USA, 2014.
4. Kharroubi AT, Darwish HM. Diabetes mellitus: The epidemic of the century. World J Diabetes. 2015;6(6):850-867. DOI:10.4239/wjd.v6.i6.850
5. Shaw JE, Sicree RA, Zimmet PZ. Global estimates of the prevalence of diabetes for 2010 and 2030," Diabetes Research and Clinical Practice. 2010; 87(1):4–14.
6. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2009;32 Suppl 1(Suppl 1):S62-S67. DOI:10.2337/dc09-S062
7. Introduction: Standards of Medical Care in Diabetes-2021. (2021). Diabetes care, 44(Suppl 1), S1–S2. Avaialble:https://doi.org/10.2337/dc21-Sint
8. Godoy DA, Napoli MD, Biestro A, Lenhardt R. Perioperative Glucose Control in Neurosurgical Patients. Anesthesiology Research and Practice [Internet]. 2012 [Cited 2014 Apr 26];2012. Available:http://www.hindawi.com/journals/arp/2012/ 690362.
9. Turina M, Fry DE, Polk HC Jr. Acute Hyperglycemia and the Innate Immune System: Clinical, Cellular, and Molecular Aspects. Critical Care Medicine. 2005;33(7):1624-33.
10. American Diabetes Association. Medical Management of Type 2 Diabetes. 8th ed. Meneghini L, Ed. Alexandria, VA, American Diabetes Association; 2020
11. Siegel KR, Ali MK, Zhou X, Ng BP, Jawanda S, Proia K, Zhang X, Gregg EW, Albright AL, Zhang P. Cost-effectiveness of interventions to manage diabetes: has

the evidence changed since 2008? Diabetes Care. 2020;43:1557–1592

12. American Diabetes Association. 13. Children and Adolescents: Standards of Medical Care in Diabetes-2020. Diabetes Care. 2020;43(Suppl 1):S163–S182.

    Avaialble:https://doi.org/10.2337/dc20-S013.
13. American Diabetes Association. 16. Diabetes Advocacy: Standards of Medical Care in Diabetes-2019. Diabetes Care. 2019l42(Suppl 1): S182–S183.

    Avaialble:https://doi.org/10.2337/dc19-S016
14. Rajaei E, Jalali MT, Shahrabi S, Asnafi AA, Pezeshki SMS. HLAs in Autoimmune Diseases: Dependable Diagnostic Biomarkers? Curr Rheumatol Rev. 2019;15(4):269-276.
15. Inzucchi S. Oral antihyperglycemic therapy for type 2 diabetes: scientific review. JAMA 2002;287(3):360–72.
16. Barnett P, Braunstein G. Diabetes mellitus. In: AndreoliT, Carpenter C, Griggs R, et al, editors. Cecil essentials of medicine. 5th edition. Philadelphia7 WB Saunders Company. 2001:590 3.
17. Tuomilehto J. The emerging global epidemic of type 1 diabetes. Curr Diab Rep. 2013 Dec;13(6):795-804.
18. Inzucchi S. Oral antihyperglycemic therapy for type 2 diabetes: scientific review. JAMA 2002;287(3):360–72.
19. Luna B, Feinglos MN. Oral agents in the management of type 2 diabetes mellitus. Am Fam Physician 2001; 63(9):1747–56.
20. Stumvoll M, Nurjhan N, Perriello G, et al. Metabolic effects of metformin in non-insulin-dependent diabetes mellitus. N Engl J Med. 1995;333:550–4.
21. Johansen K. Efficacy of metformin in the treatment of NIDDM: meta-analysis. Diabetes Care. 1999;22:33–7.
22. Haute Autorite´ de Sante´. Recommandation popur la pratique clinique. Stra-te´ gie me´dicamenteuse du contro^le glyce´mique du diabe` te du type 2013;2.

    Avaialble:[http://www.hassante.fr/portail/upload/docs/application/pdf/2013-02/10irp04_reco_diabete_type_2.pdf].
23. Meynaar IA, Eslami S, Abu-Hanna A, van der Voort P, de Lange DW, de Keizer N. Blood glucose amplitude variability as predictor for mortality in surgical and medical intensive care unit patients: a multicenter cohort study. J Crit Care. 2012;27:119–24.
24. Sato H, Carvalho G, Sato T, Lattermann R, Matsukawa T, Schricker T. The association of preoperative glycemic control, intraoperative insulin sensitivity, and outcomes after cardiac surgery. J Clin Endocrinol Metab. 2010;95: 4338– 44.
25. Halkos ME, Lattouf OM, Puskas JD, Kilgo P, Cooper WA, Morris CD, et al. Elevated preoperative hemoglobin A1c level is associated with reduced longterm survival after coronary artery bypass surgery. Ann Thorac Surg. 2008;86: 1431–7.
26. O'Sullivan CJ, Hynes N, Mahendran B, Andrews EJ, Avalos G, Tawfik S, et al. Haemoglobin A1c (HbA1C) in non-diabetic and diabetic vascular patients. Is HbA1C an independent risk factor and predictor of adverse outcome? Eur J Vasc Endovasc Surg. 2006;32:188–97.
27. Lazar HL, Chipkin SR, Fitzgerald CA, Bao Y, Cabral H, Apstein CS. Tight glycemic control in diabetic coronary artery bypass graft patients improves perioperative outcomes and decreases recurrent ischemic events. Circulation. 2004;109: 1497–502.
28. Bhamidipati CM, LaPar DJ, Stukenborg GJ, Morrison CC, Kern JA, Kron IL, et al. Superiority of moderate control of hyperglycemia to tight control in patients undergoing coronary artery bypass grafting. J Thorac Cardiovasc Surg. 2011;141:543–51.
29. Desai SP, Henry LL, Holmes SD, Hunt SL, Martin CT, Hebsur S. Strict versus liberal target range for perioperative glucose in patients undergoing coronary artery bypass grafting: a prospective randomized controlled trial. J Thorac cardiovasc Surg. 2012;143:318–25.
30. Fish LH, Weaver TW, Moore AL, Steel LG. Value of postoperative blood glucose in predicting complications and length of stay after coronary artery bypass grafting. Am J Cardiol. 2003;92:74–6.
31. Furnary AP, Zerr KJ, Grunkemeier GL, Starr A. Continuous intravenous insulin

infusion reduces the incidence of deep sternal wound infection in diabetic patients after cardiac surgical procedures. Ann Thorac Surg. 1999;67:352–60.
[Discussion 360-362].

32. Nathan DM, Kuenen J, Borg R, Zheng H, Schoenfeld D, Heine RJ, et al. Translating the A1C assay into estimated average glucose values. Diabetes Care. 2008;31:1473–8.
33. Brutsaert E, Carey M, Zonszein J. The clinical impact of inpatient hypoglycemia. J Diabetes Complications. 2014;28:565–72.
34. Schopman JE, GeddesJ, Frier BM. Prevalence of impaired awareness of hypoglycaemia and frequency of hypoglycaemia in insulin-treated type 2 diabetes. Diabetes Res Clin Pract. 2010;87:64–8.
35. Golden SH, Peart-Vigilance C, Kao WH, Brancati FL. Perioperative Glycemic Control and the Risk of Infectious Complications in a Cohort of Adults with Diabetes. Diabetes Care. 1999;22: 1408-14.
36. Zerr KJ, Furnary AP, Grunkemeir GL, Bookin S, Kanhere V, Starr A. Glucose Control Lowers the Risk of Wound Infections in Diabetics after Open Heart Operations. Ann Thorac Surg.1997;63:356-61.
37. Furnary AP, Zerr KJ, Grunkemeir GL, Starr A. Continuous Intravenous Insulin Infusion Reduces the Incidence of Deep Sternal Wound Infection in Diabetic Patients after Cardiac Surgical Procedures. Ann Thorac Surg. 1999;67:352-62.
38. Gottrup F, Andreassen TT. Healing of Incisional Wounds in Stomach and Duodenum: The Influence of Experimental Diabetes. J Surg Res. 1981;31:61-68.
39. McMurry JF Jr. Wound Healing with Diabetes Mellitus. Better Glucose Control for Better Wound Healing in Diabetes. Surg Clin North Am. 1984;64:769-78.
40. Verhofstad HJ, Hendriks T. Complete Prevention of Impaired Anastomotic Healing in Diabetic Rats Requires Preoperative Blood Glucose Control. Br J Surg. 1996;83:1717-21.
41. Rodriguez BL, Lau N, Burchfiel CM, Abbott RD, Sharp DS, Yano K, et al. Glucose Intolerance and 23-Year Risk of Coronary Heart Disease and Total Mortality: The Honolulu Heart Program. Diabetes Care. 1999;22(8):1262-65.
42. Rehman HU, Mohammed K. Perioperativemanagement of diabetic patients. Current Surgery. 2003;60(6): 607–611.
43. Meneghini LF. Perioperative management of diabetes: translating evidence into practice," Cleveland Clinic Journal of Medicine. 2009;76(4):S53–S59.
44. Tetsuro T, Hiroshi K, Yoshihiko T, Miyako K, Hiroshi N, Ritsuko YH, et al. Asymptomatic Coronary Heart Disease in Patients with Type 2 Diabetes with Vascular Complications: A Cross-Sectional Study. BMJ Open [Internet]. 2011[cited 2014 Apr 26];1(2).
Available:http://bmjopen.bmj.com/content/1/2/e000139.full.
45. Furnary AP, Wu Y, Bookin SO. Effect of Hyperglycemia and Continuous Intravenous Insulin Infusions on Outcomes of Cardiac Surgical Procedures: The Portland Diabetic Project. Endocrine Practice. 2004;10(Suppl 2):S21-33.
46. Krinsley JS. Association between Hyperglycemia and Increased Hospital Mortality in a Heterogeneous Population of Critically Ill Patients. Mayo Clin Proc. 2003;78:1471-78.
47. Thompson J, Husband DJ, Thai AC, Alberti KG. Metabolic Changes in the Non-Insulin-Dependent Diabetic Undergoing Minor Surgery: Effect of Glucose-Insulin-Potassium Infusion. Br J Surg. 1986;73:301-304.
48. Thomas DJ, Platt HS, Alberti KG. Insulin-Dependent Diabetes during the Peri-Operative Period. An Assessment of Continuous Glucose-Insulin- Potassium Infusion, and Traditional Treatment. Anesthesia. 1984;39:629-37.
49. Joana S, Susana C, Vitor M, Luis C, Camila T, Pedro F, et al. Metformin-Induced Lactic Acidosis: A Case Series. J Med Case Reports. 2007;1:126.
50. Albarti KGMM. Diabetes and Surgery. Anaesthesiology. 1991;74:209-11.
51. Kedia N. Treatment of severe diabetic hypoglycemia with glucagon: an underutilized therapeutic approach. Diabetes Metab Syndr Obes. 2011;4: 337-46.
52. McAnulty GR, Robertshaw HJ, Hall GM. Anaesthetic Management of Patients with

Diabetes Mellitus. Br J Anaesthesia. 2000;85:80-90.
53. Escalante DA, Kim DK, Garber AJ. Atherosclerotic Cardiovascular Disease. In: DeFronzo RA, editor. Current Therapy of Diabetes Mellitus. St. Louis: Mosby. 1998:176-82.
54. Wicklmayr M, Rett K, Dietz G, Mehnart H. Comparison of Metabolic Clearance Rates of MCT/LCT and LCT Emulsions in Diabetics. J Parenteral Enteral Nutr. 1988;12:68-71.
55. McAnulty GR, Robertshaw HJ, Hall GM. Anaesthetic Management of Patients with Diabetes Mellitus. Br J Anaesth. 2000; 85:80-90.
56. Salzarulo HH, Taylor LA. Diabetic “Stiff Joint Syndrome” as a Cause of Difficult Intubation. Anesthesiology. 1986;64: 366-68.
57. Sinclair RCF, Faleiro RJ. Delayed Recovery of Consciousness after Anaesthesia. Contin Educ Anaesth Crit Care Pain. 2006;6(3):114-18.
58. Cueni-Villoz N, Devigili A, Delodder F, Cianferoni S, Feihl F, Rossetti AO, et al. Increased Blood Glucose Variability during Therapeutic Hypothermia and Outcome after Cardiac Arrest. Crit Care Med. 2011;39(10):2225-31.
59. Schade DS. Surgery and diabetes. Med Clin North Am 1988;72:1531-43.
60. Hirsch IB, Paauw DS, Brunzell J. Inpatient management of adults with diabetes. Diabetes Care. 1995;18:870-8.
61. Gill GV, Albarti K. The Care of the Diabetic Patient during Surgery. In: Alberti K, Zimmett P, DeFronzo RA, Keen H, editors. International Textbook of Diabetes Mellitus. 2nd ed. Chickester: John Wiley & Sons; 1997:1243-53.
62. Albarti KGMM, Thomas DJB. The Management of Diabetes During Surgery. Br J Anaesth. 1979;51: 693-710
63. Watkins PJ, Smith JS, Fitzgerald MG, Malins JM. Lactic Acidosis in Diabetes. Brit med J. 1969;1:744-47.
64. Wall RT. Endocrine Disease. In: Hines RL, Marschaff KE, editors. Anaesthesia and Co-Existing Disease. 7th ed. India: Elsevier. 2009:365-406.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle5.com/review-history/80267