Therapeutic Hypothermia induced Hypokalemia and Hyperglycemia in Cardiac Surgery

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Abstract

Hyperglycemia is common in critically ill patients and has been associated with poor outcomes. The effect of hypothermia, whether induced or spontaneous, on insulin resistance and hyperglycemia is not well understood and sometimes overlooked. In cardiac surgery hypothermia impairs blood glucose homeostasis and insulin sensitivity (¹). However, the impact of therapeutic hypothermia on blood glucose levels and insulin requirements is unknown (¹).

Keywords: Therapeutic Hypothermia, Hyperglycemia.

Introduction

Hyperglycemia has been associated with poor neurological outcomes for patients treated with therapeutic hypothermia, but it is unclear whether hyperglycemia directly contributes to deleterious outcomes or is simply a marker for overall poor health (²). Hypothermia indirectly increases the glucose levels in the blood stream, resulting in hyperglycemia. As hypothermia is initiated, sympathetic activity is increased resulting in elevated levels of catecholamines and free fatty acids, results in decreased insulin secretion and increased tissue resistance to insulin (³). The increase in catecholamine release is compounded by impaired peripheral glucose uptake at the tissue level because of hypothermia (³). The decrease of is also caused by cooling of the islets of Langerhans, responsible for insulin secretion (⁴). A third effect of hypothermia is hypokalemia (⁵). Hypokalemia is associated with insulin resistance and hyperglycemia (⁶). Therefore, three factors play a role in increasing glucose levels in hypothermic conditions: increased catecholamine levels, hypothermic effect on the pancreas, and hypothermic induction of hypokalemia (³).

In cardiac surgery hypothermia is an important method to protect organs during low flow. But hypothermia induced hypoglycaemia is also a challenge to surgical outcome. In surgical patients, perioperative hyperglycemia increases risk of
postoperative mortality, and cardiovascular, respiratory, neurologic, and infectious morbidity \(^{(1)}\).

**Method**

In this study 50 consecutive adult patients were taken in a randomised nonblinded manner elective open heart surgery like ASD, VSD, MVR, DVR, CABG ON PUMP under general anaesthesia, moderate hypothermia (28-32 °C) and alpha stat pH management.

Preoperative exclusion criteria were
1) Less than 18 years,
2) Diabetic patients,
3) Organ dysfunction.

During surgery we divided total CPB Bypass period in to three phases- Cooling Phase, Plateau Phase and Rewarming Phase. We monitored nasopharyngeal temperature. In cooling phase we monitored the blood glucose level and serum potassium every after 2°C decrease of body temperature. During Plateau phase the temperature was remain same and we check glucose every after 20 minutes. In rewarming phase we check blood glucose level and serum potassium every after 2°C decrease of body temperature.

We found a higher blood glucose level and hypokalemia with decreasing temperature. We added insulin as our institutional protocol. During bypass we added insulin as bolus dose according to our institutional protocol (Table-1).

**Table-1. Protocol for bolus insulin dose.**

| Blood glucose level (mg/dl) | Insulin Bolus Units (IU) |
|-----------------------------|--------------------------|
| 170-180                     | 5                        |
| 170-190                     | 8                        |
| 190-200                     | 10                       |
| >200                        | 15                       |

The Society of Thoracic Surgeons recommends insulin for cardiac surgical patients when glucose concentrations are persistently >180 mg/dL \(^{(7)}\)

**Result**

In our study we found that blood glucose level was increasing with decreasing temperature. Before anaesthesia an average blood glucose level of 50 patients was 90-100mg/dl. After anaesthesia it was 100-110 mg/ dl. Just 10 minutes after the starting of CPB bypass we found a raised blood glucose level (average-180-190 mg/dl) and hypokalemia. We added insulin as our protocol and hypokalemia is also corrected. But after starting the cooling phase the blood glucose level was increased with decreasing body temperature. Then insulin was added but blood glucose level was remaining same. Sometimes we found a raised blood glucose level. We found that hyperglycemia and hypokalemia both increased simultaneously in cooling phase. But in rewarming phase hyperglycemia and also the requirement of potassium both were gradually decreased with increasing temperature. We had no need to correct potassium except 8 cases during rewarming phase.

![Plot 1: Changes of blood glucose level](Image)

Plot 1 is showing the changes of blood glucose level with respect to the change of temperature in both cooling phase and rewarming phase.

Temperature is plotted horizontally in °C and blood glucose is plotted vertically in mg/dl unit.

![Plot 2: Requirement of potassium](Image)

Plot 2 is showing the requirement of potassium to correct hypokalemia during cooling phase.
Temperature is plotted horizontally and requirement of potassium in meq is plotted vertically.
In both plot we found a large difference between 37°C-35°C. This is because at that time CPB was initiated and a dilution problem was occurred.

Discussion
Hyperglycemia accompanies initiation of CPB regardless of the anaesthetic technique (8,9,10). Concentrations of glucose, insulin, and glucagon are higher during normothermic than hypothermic CPB (11,12).
Markedly high serum catecholamine concentrations, which occur during CPB, inhibit the pancreatic β-cell the insulin secretory response to hyperglycemia CPB (13), during moderate hypothermia, which results blood glucose concentrations raise steadily at initiation of CPB. In our study we found it from Plot-1. Despite marked hyperglycemia, insulin concentrations decline from their control values during hypothermic bypass (14,15,16).
Counter-regulatory hormones also decline from pre-bypass concentrations during hypothermic bypass (17).
Another effect of hypothermia is hypokalemia (18). Hypokalemia is associated with insulin resistance and hyperglycemia (19). With rewarming, insulin concentrations rise spontaneously to appropriate high levels; nonetheless, blood glucose remains elevated (20).

Conclusion
In cardiac surgery therapeutic hypothermia is most important and also the Hypothermia induced hyperglycemia and hypokalemia is most common. Hyperglycemia and hypokalemia both increase with decreasing temperature and both occurs simultaneously. These two factors are most important for outcome of cardiac surgery.

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