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Mini Review

# Monitoring of Blood Glucose Level in Surgical Intensive Care Unit Patients with Sepsis

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## Abstract

Sepsis is a life-threatening condition that arises when the body's response to infection injures its own tissues and cause organs dysfunction, clinically characterized by an acute changes of 2 points or greater in the SOFA score, clinical criteria for septic shock include sepsis with fluid-unresponsive hypotension, serum lactate level greater than 2 mmol/L, and the need of vasopressor to maintain mean arterial pressure of 65 mm Hg or greater [1]. There are many pathophysiological changes during sepsis and septic shock, and one of the most striking is metabolic derangement. Among the metabolic changes, hyperglycemia is the most important [2,3].

**Keywords:** Blood Glucose; Sepsis; Hyperglycemia

Hyperglycemia is a common feature of the critically ill in general and of patients with sepsis in particular. Even a moderate degree of hyperglycemia appears harmful for the outcome of critically ill patients, since maintenance of normoglycemia (blood glucose levels < or =110 mg/dL) with intensive insulin therapy has shown to improve survival and reduce morbidity in prolonged critically ill patients in surgical intensive care units [2]. But some casess of sepsis may be complicated by the occurance of hypoglycemia, which is caused by dysbalance between glucose production and glucose consumption [4,5].

## Incidence and outcome of sepsis

Sepsis with acute organ dysfunction is common and frequently fatal and represents a significant health care burden. The incidence and associated mortality and morbidity rates of severe sepsis are commonly underestimated. Sepsis affects more than 750,000 patients each year in the United States; it is the 10th leading cause of death and one of the leading causes for admission to the intensive care unit [6]. The estimated mortality from sepsis is 20–30%, meaning that approximately 500,000 patients survive their septic episode annually in the United States alone. Mounting evidence has since demonstrated that survivors of sepsis have a higher long-term risk of death and a lower health-related quality of life when compared with the general population [7].

## Etiology

Sepsis can be a response to any class of microorganism. Microbial invasion of the blood stream is not essential for the development of sepsis. Local or systemic spread of microbial signal molecules or toxins can also elicit a response. The occurrence of gram-negative sepsis has diminished over the years to 25-30% in 2000. Gram-positives account for the majority of cases up to 30-50%. In 11-19% cases, the etiology is polymicrobial in nature. Fungi, Viruses and parasites account for 14%, but their frequency could be underestimated [8]. Lastly cultures may be negative in 30% of cases, mainly in patients with community-acquired sepsis who are treated with antibiotics before admission [9]. The source of infection is usually endogenous. Infections of the chest, abdomen, genitourinary system and primary blood stream cause more than 80% of sepsis. Rates of pneumonia, bacteremia and multiple site infection have steadily increased over time, whereas abdominal infections have remained unchanged and genitourinary infections have decreased. Rarely, the source may even be exogenous [10].

## Glucose metabolism in sepsis

Enhanced peripheral glucose uptake and utilization, increased glucose production, depressed glycogen synthesis, glucose intolerance, and insulin resistance are the key manifestations of altered glucose metabolism [11]. The goal of these metabolic changes is to provide adequate organ supply of glucose as an energy substrate [12]. Increased glucose uptake is mediated by effects of the counterregulatory hormones and by cytokine effects [13]. Gluconeogenesis is a main factor in the hyperglycemic response, increased gluconeogenesis is supplied by stimulated Cori cycle activity and enhanced glucose–alanine cycle turnover. Additional effectors of

increased gluconeogenesis are counter-regulatory hormones and direct cytokine stimulation [14]. Under normal conditions, glycemic control is achieved by the inhibitory effects of insulin and by a feedback control of gluconeogenesis. During sepsis, glycemic control is disturbed due to insulin resistance and the loss of feedback control [15]. Insulin resistance may be associated with impaired insulin receptor binding or impairment in the activation of early or intermediate components of the insulin signaling pathway and/or with defects in glucose transporter [16].

### Hypoglycemia with sepsis

The pathophysiological explanation for this dysregulation may be related to classical endocrine mechanisms, like hyperinsulinemia, defective hypoglycemic counter regulation (e. g. adrenal failure) and/or to consequences of severe infection per se [17]. There was no defect in hypoglycemic counterregulatory hormones, judged by high concentrations of glucagon, catecholamines, growth hormone and cortisol [18]. Hyperinsulinemia, which has been suggested to be the cause of septic hypoglycemia [19], was ruled out by normal postabsorptive insulin concentrations prior to and at the time of hypoglycemia. Portal insulin concentrations may have been elevated, which is supported by increased C-peptide concentrations. Although insulin inhibits hepatic glucose production in healthy subjects, it should be noted that this effect of insulin is greatly reduced in severe infection [20]. Therefore, it remains to be defined whether elevated portal insulin concentrations are an important factor in the pathogenesis of septic hypoglycemia. There were no clues to the presence of other hypoglycemic factors like fulminant liver failure, hypoglycemic drugs, alcoholism or long-term starvation. Hypotension per se is not a risk factor for hypoglycemia as humans in traumatic hypovolemic or cardiogenic shock develop hyperglycemia [21]. *In vivo* or *in vitro* consumption of glucose by bacteria has been mentioned as a potential cause for hypoglycemia but proved to be equally unlikely [22]. The time course, however, strongly suggests the hypoglycemia to be the consequence of severe sepsis in this patient. Glucose homeostasis is profoundly changed during sepsis. In the early phase hyperglycemia is found, which is explained by augmented hepatic glucose production through accelerated glycogenolysis and gluconeogenesis [23]. With progression of sepsis profound hypoglycemia occurs preterminally in animals , due to enhancement of glucose uptake by the tissues despite declining blood glucose concentrations and a failure of the liver to maintain glucose output at an elevated level [24]. The question arises which factors cause these alterations in glucose homeostasis during septic shock. The stimulation of glycogenolysis and gluconeogenesis can largely be explained by the interaction of counterregulatory hormones like glucagon, catecholamines and cortisol [15]. In general no deficiencies in counter-regulatory hormones are found in sepsis. Additionally, the effects of insulin on hepatic glucose production and peripheral glucose uptake are markedly blunted in sepsis [25]. It is important to recognize that pathologically low blood glucose also occurs secondary to impaired endogenous glucose production even in the absence of exogenous insulin. Accordingly, episodes of hypoglycemia are also a marker of illness severity; for example, a retrospective observational study of 102 critically ill patients with an episode of severe hypoglycemia reported that 27.5% had not received insulin within 12 hours of the hypoglycemic episode [26].

### Glycemic control

Hyperglycemia is independently associated with increased ICU mortality [27]. Strict control of the blood glucose concentration is considered important because it may reduce mortality and morbidity; however, hypoglycemia is significantly higher inpatients with tight glucose control using intensive insulin therapy. [28]. Glycemic control to a moderately tight range is not inferior to euglycemia and clearly safer in critically ill patients [29].

## Conclusion

We concluded that frequent blood glucose measurement is one element of the routine intensive monitoring that all critically ill patients receive following admission to intensive care units. Transient increase in blood glucose concentration (hyperglycemia) is very common in this patient. Although decrease in blood glucose concentration (hypoglycemia) is rare but has very bad prognosis with significant increase in mortality rate.

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