# Stress Hyperglycemia in Patients with Sepsis

Pitiporn Rattanataweeboon MD*,
Warakarn Vilaichone MD*, Sathit Vannasaeng MD*

** Divisions of Endocrinology and Critical Care Medicine, Department of Medicine, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand*

***Background:*** *Patients with acute stress from stroke or myocardial infarction may develop hyperglycemia, even in the absence of diabetes mellitus. Stress hyperglycemia increases morbidity and mortality in these patients. There has been no study to evaluate stress hyperglycemia in patients with sepsis.*
***Material and Method:*** *A prospective cohort study in patients with sepsis admitted in the Department of Medicine of Siriraj Hospital during 2006-2007 was done. Data were collected on admission blood glucose, HbA1c and other factors which may predict the outcomes of sepsis.*
***Results:*** *Data were collected from 70 patients with sepsis. The prevalence of stress hyperglycemia was 42.3% in this study. We found no differences in clinical findings, laboratories, interventions and outcomes between groups of stress and non-stress hyperglycemia. Multivariate analysis showed that only APACHE II score and use of a mechanical ventilator were associated with mortality.*
***Conclusion:*** *The prevalence of stress hyperglycemia in patients with sepsis was high. We cannot conclude that stress hyperglycemia did not affect the mortality and morbidity outcome mainly because of the small number of subjects which may be not enough to detect statistical significance.*

***Keywords****: Hyperglycemia, Sepsis, Stress, Physiological*

***J Med Assoc Thai 2009; 92 (Suppl 2): S88-94***
***Full text. e-Journal:*** *http://www.mat.or.th/journal*

A high proportion of patients suffering from acute stress such as stroke[1] or myocardial infarction[2] may develop hyperglycemia, even in the absence of a preexisting diagnosis of diabetes. Both human and animal studies suggest that this is not a benign occurrence and that stress-induced hyperglycemia is associated with a high risk of mortality after both stroke[3] and myocardial infarction[4]. Previous studies showed that the prevalence rate of stress hyperglycemia in myocardial infarction varied from 36-71 percent and 46-84 percent in non-diabetic and diabetic patients, respectively. The mortality rates were 3 percent in non-diabetic patients without stress hyperglycemia, 15 percent in non-diabetic patients with stress hyperglycemia and was as high as 43 percent in diabetic patients with stress hyperglycemia[5]. The prevalence of stress hyperglycemia in stroke patients also varied from 8-63 percent and 39-83 percent in non-diabetic and diabetic patients, respectively[6] . Non-diabetic patients with stress hyperglycemia had a 3.28-fold higher risk of mortality than those without stress hyperglycemia[6].

In sepsis, change in body metabolism may increase the risk for hyperglycemia. The levels of counter regulatory hormones including catecholamines, glucagon, cortisol and growth hormone increase in sepsis and these hormones enhance hepatic glycogenolysis and gluconeogenesis. Furthermore, inflammatory cytokines including interleukin -1, interleukin-6 and tumor necrosis factor-A also decrease insulin mediated glucose uptake in peripheral tissue. These insulin resistances found in hepatic and peripheral tissue may be attributable to hyperglycemia in sepsis. The degrees of hyperglycemia and insulin resistance are directly proportional to the severity of the stress[7]. Hyperglycemia motivates secretion of inflammatory cytokine and induces phagocytic dysfunction because high blood sugar level decreases adherence, chemotaxis, phagocytosis, and bacterial killing of neutrophils[8]. Furthermore, laboratory study showed that high blood glucose level

*Correspondence to: Vannasaeng S, Division of Endocrinology, Department of Medicine, Faculty of Medicine Siriraj Hospital, Bangkok 10700, Thailand.*

of more than 200 mg per deciliter caused phagocytic dysfunction[9]. Meta-analysis of Asia Pacific Cohort Collaboration study found that overall mortality rates from sepsis in diabetic patients were increased to 1.98-fold (95% CI =1.49-2.63)[10] . It is possible that hyperglycemia may increase the mortality outcome in patients with sepsis.

Van Den Berghe et al showed that the use of intensive insulin therapy in surgical intensive care unit (ICU) to control blood glucose at a level between 80 and 110 mg per deciliter reduced mortality by 34 percent, bloodstream infections by 46 percent, acute renal failure requiring dialysis or hemofiltration by 41 percent, red-cell transfusions by 50 percent, and critical-illness polyneuropathy by 44 percent[11]. Patients receiving intensive therapy were less likely to require prolonged mechanical ventilation and intensive care[11]. A later study in a medical ICU showed that intensive insulin therapy did not significantly reduce in-hospital mortality. However, morbidity was significantly reduced as shown by the prevention of newly acquired kidney injury, accelerated weaning from mechanical ventilation, and accelerated discharge from the ICU and the hospital[12]. This study also showed that intensive control of blood sugar level resulted in reduced mortality in subgroup of patients with sepsis.

Sepsis is the stressful condition in which prevalence and outcome of stress hyperglycemia with no intervention as related to mortality has never been studied. We prospectively evaluated the prevalence of stress hyperglycemia and mortality in patients with sepsis admitted in the Department of Medicine of Siriraj Hospital.

## Material and Method

### *Patients*

This study included patients who were admitted to the medical wards at Siriraj Hospital with diagnosis of sepsis according to the criteria shown below. None of them had any history of diabetes and had HbA1c level of 6.5 percent or less at admission. The patients with hemolytic anemia, thalassemia, hemoglobinopathy, chronic renal failure and those who previously received corticosteroids were excluded.

### *Definitions*

Sepsis is defined as systemic inflammatory response syndrome (SIRS) that has a proven or suspected microbial etiology. SIRS is defined by the presence of at least 2 of following conditions.

1) Oral temperature of > 38°C or < 36°C.

2) Respiratory rate of > 20 breaths/min or PaCo 2 of < 32 torr.

3) Heart rate of > 90 beats/min.

4) Leukocyte count of > 12,000/mL or < 4,000/mL or > 10% bands.

Septic shock is defined as sepsis with hypotension (systolic blood pressure < 90 mmHg or 40 mmHg less than patient's normal blood pressure) for at least 1 hour despite adequate fluid resuscitation or need for vasopressors to maintain systolic blood pressure $\geq$ 90 mmHg or mean aterial pressure $\geq$ 70 mmHg[13]. APACHE II (Acute Physiology and Chronic Health Evaluation II)[14] is a severity of disease classification system. An integer score from 0 to 71 is computed based on several measurements. The point score is calculated from 12 routine physiological measurements (blood pressure, body temperature, heart rate, respiratory rate, oxygenation, arterial pH, serum sodium, serum potassium, serum creatinine, haematocrit, white blood cell count and Glasgow coma score), information on previous health status and some information obtained at admission (such as age). Higher scores imply a more severe disease and a higher risk of death[15,16]. SOFA (Sequential Organ Failure Assessment) score[17] is a scoring system to determine the extent of a person's organ function or rate of failure. The score is based on six different scores, one each for the respiratory, cardiovascular, hepatic, coagulation, renal and neurological systems. The higher score correlates well with greater mortality[18] . Stress hyperglycemia is defined as the level of fasting plasma glucose (FPG) being more than 100 mg per deciliter or random plasma glucose (RPG) being more than 140 mg per deciliter in individauals without diabetes, and having HbA1c being equal to or less than 6.5 percent at admission[19,20]. Fasting plasma glucose is defined as plasma glucose obtained in the morning after 8 hours of fasting and within 48 hours after admission. Random blood glucose is defined as plasma glucose obtained anytime within 48 hours after admission and receiving intravenous dextrose infusion of less than 5 mg/kg/min[21]. Both fasting and random blood sugar levels were obtained before corticosteroid administration. Acute renal failure is defined as a level of serum creatinine level which is twice the level obtained on admission or a peak level of serum creatinine level of more than 2.5 mg per deciliter[12].

### *Data collection*

Plasma glucose measurement was done within

48 hours after admission. We also collected clinical data within 48 hours after admission on age, sex, preadmission co-morbid diseases, sites of infection, antibiotic use, bacterial culture and results of a sensitivity test, central line insertion, use of mechanical ventilators, insulin therapy, total parenteral nutrition, corticosteroid therapy, vasopressor administration, the presense of septic shock, APACHE II (Acute Physiology And Chronic Health Evaluation) score, SOFA (Sequential Organ Failure Assessment) score. Blood samples were measured for HbA1c, albumin, and cortisol levels prior to corticosteroid administration.

***Study outcomes***

The primary goal of the study was the prevalence of stress hyperglycemia in patients with sepsis. Secondary outcomes were the effects of stress hyperglycemia on hospital stay, days in ICU, mortality in the first 30 days after admission as well as more than 30 days after admission, and overall mortality.

***Statistical analysis***

Continuous variables are presented as either means (± SD) or median. Categorical variables are presented as numbers and percentages. The baseline

**Table 1.** Baseline clinical characteristics of the patients

| Variables (Mean ± SD) | No stress hyperglycemia (n = 40) | Stress hyperglycemia (n = 30) | p-value |
|---|---|---|---|
| Age (yrs) | 63.9 ± 16.3 | 64.4 ± 17.5 | 0.80 |
| Sex, male/female (n) | 16/24 | 15/15 | 0.40 |
| Preadmission cormorbidities, n (%) | | | |
| Chronic neurological disease | 7 (17.5) | 9 (30) | 0.22 |
| Cardiac disease | 4 (10) | 7 (23) | 0.13 |
| Chronic liver disease | 6 (15) | 7 (23) | 0.37 |
| Malignancy | 14 (35) | 13 (43) | 0.47 |
| Chronic pulmonary disease | 3 (7.5) | 3 (10) | 0.71 |
| Chronic renal disease | 2 (5) | 1 (3.3) | 0.73 |
| Septic shock, n (%) | 20 (50) | 14 (46.6) | 0.78 |
| Intervention, n (%) | | | |
| Central line | 10 (25) | 9 (30) | 0.64 |
| Mechanical ventilator | 15 (37.5) | 17 (56.6) | 0.11 |
| Vasopressor use - dopamine | 15 (37.5) | 10 (33.3) | 0.72 |
| - norepineprine | 9 (22.5) | 6 (20) | 0.80 |
| - epineprine | 1 (2.5) | 0 | 0.38 |
| Corticosteroid supplement | 11 (27.5) | 10 (33.3) | 0.59 |
| Total parenteral nutrition | 0 | 1 (3.3) | 0.24 |
| Insulin use | 7 (17.5) | 7 (23) | 0.55 |
| APACHE II score (median) | 20.7 ± 8.9 (19) | 23.5 ± 9 (25) | 0.20 |
| SOFA score (median) | 6.8 ± 4.4 (6) | 8.2 ± 4.4 (8) | 0.20 |
| FPG (mg/dl ), (range) | 84.0 ± 13.8 (67-96) | 125.6 ± 14.9 (110-150) | 0.001 |
| Random BG (mg/dl) (range) | 105.4 ± 22 (77-137) | 156.0 ± 14.8 (141-190) | <0.0001 |
| HbA1c (%) (range) | 5.8 ± 0.4 (4.9-6.5) | 6.0 ± 0.4 (5.1-6.4) | 0.12 |
| Albumin (g/dl) (range) | 2.6 ± 0.7 (1.6-4.4) | 2.8 ± 0.7 (1.5-4.3) | 0.20 |
| Cortisol (μg/dl) (range) | 28.6 ± 16.1 (3.3-3.4) | 36.3 ± 21 (3.6-63.4) | 0.88 |
| Complications, n (%) | | | |
| Neurological complication | 4 (10) | 4 (13) | 0.66 |
| Cardiac complication | 8 (20) | 4 (13) | 0.46 |
| GI complication | 7 (17.5) | 9 (30) | 0.22 |
| Acute renal failure | 15 (37.5) | 14 (46.6) | 0.44 |
| Outcomes | | | |
| Hospital stay, days (median) | 16.7 ± 17.2 (11) | 14.9 ± 16.53 (8) | 0.60 |
| First 30 days mortality, n (%) | 19 (47.5) | 18 (60) | 0.30 |
| After 30 days mortality, n (%) | 0 | 1 (3.3) | 0.30 |
| Over all mortality, n (%) | 19 (47.5) | 19 (63.3) | 0.20 |

**Table 2.** Univariate analysis of factors associated with mortality

| Variables | Odds ratio (95% confidence interval) | p-value |
|---|---|---|
| Stress hyperglycemia | 1.9 (0.73-5.0) | 0.24 |
| Pulmonary infection | 3.5 (1.28-9.6) | 0.02 |
| Gram negative drug resistance infection | 3.5 (1.1-11.2) | 0.05 |
| Central line use | 4.5 (1.3-15.7) | 0.02 |
| Ventilator use | 19.6 (5.5-69.9) | <0.001 |
| Norepinephrine administration | 4.5 (1.1-17.6) | 0.04 |
| Acute renal failure | 2.8 (1.04-7.7) | 0.06 |
| APACHE II score $\geq$ 20 | 9.6 (3.2-29) | <0.001 |
| SOFA score $\geq$ 10 | 13.0 (2.7-62.6) | 0.001 |
| Albumin level $\leq$ 3.5 g/dl | 4.1 (0.8-22.2) | 0.07 |
| Cortisol level $\geq$ 32.5 μg/dl | 10.4 (3.0-35.6) | 0.001 |

characteristics between groups were compared by use of t-test for continuous variables and Chi-square test for non-continuous variables. Logistic regression analysis with forward stepwise method was used to evaluate factors independently associated with the outcomes as measured by odds ratio with 95% confidence interval. P-value of less than 0.05 is considered as of statistical significance.

## Results

A total of 70 patients with clinical sepsis were enrolled. Their median FPG, RPG and HbA1c levels were 111 (range, 67-150) mg per deciliter, 122 (range, 51-190) mg per deciliter and 5.9 (range, 4.9-6.5)%, respectively. Thirty of them had stress hyperglycemia and 40 had no stress hyperglycemia .The prevalence of stress hyperglycemia in sepsis patients was 42.3%. Among 70 patients, 38 of them (19 or 63.3% with stress hyperglycemia and 19 or 47.5% without stress hyperglycemia) died resulting in the overall mortality rate of 54.3 %. As shown in Table 1, with exception of hyperglycemia, there were no differences in mean ages, sex, clinical characteristics of the patients including laboratory findings, interventions and outcomes between groups of stress hyperglycemia and no stress hyperglycemia. After univariate analysis (as shown in Tables 2 and 3), lung infection, gram-negative sepsis with multi-drug resistances, insertion of central line, mechanical ventilator use, norepineprine administration, high APACHE II score, and high SOFA score were associated with mortality. Although low serum albumin level as defined as being less than 3.5 g per deciliter was not significantly associated with mortality (p = 0.07, Table 2) , but it was significantly lower (p = 0.04) in those who died as compared to those who survived (Table 3). Patients who died had mean cortisol level higher than that of the survivors (37.9 vs. 24.7 μg per deciliter, p = 0.002) and a cortisol level of more than 32.5 μg per deciliter was associated with higher mortality (p = 0.001). However, after multivariate analysis as shown in Table 4, only the APACHE II score and ventilator use remained significantly associated with outcomes (p = 0.034 and p = 0.01, respectively).

**Table 3.** Univariate analysis of continuous variables associated with mortality by t-test

| Variables (mean $\pm$ SD) | Outcomes | | p-value |
|---|---|---|---|
| | Survived (n = 32) | Dead (n = 38) | |
| Albumin level | 2.8 $\pm$ 0.7 | 2.5 $\pm$ 0.6 | 0.04 |
| APACHE II score | 16.5 $\pm$ 6.1 | 26.4 $\pm$ 8.5 | <0.001 |
| SOFA score | 5.2 $\pm$ 3.4 | 9.2 $\pm$ 4.5 | <0.001 |
| Cortisol level | 24.7 $\pm$ 15.4 | 37.9 $\pm$ 19.2 | 0.002 |

## Discussion

We found that, in sepsis patients who were not diagnosed as having diabetes before, the prevalence of stress hyperglycemia was rather high being nearly half of the patients studied. There has been no study on stress hyperglycemia in patients with sepsis, thus no comparison could be made on this prevalence data. In the present study, we could not find any significant association between stress hyperglycemia and mortality outcome. There may be several reasons to explain this negative finding. Firstly, stress hyperglycemia is defined by the rise of blood glucose level when the patients have severe stress such as acute

**Table 4.** Multivariate analysis of factors associated with mortality

| Variables | Odds ratio (95% confidence interval) | p-value |
|---|---|---|
| Stress hyperglycemia | 1.7 (0.3-10.2) | 0.59 |
| Pulmonary infection | 2.0 (0.4-9.5) | 0.39 |
| Gram negative drug resistance infection | 1.6 (0.3-7.5) | 0.57 |
| Central line use | 0.2 (0.02-2.8) | 0.25 |
| Ventilator use | 10.7 (1.8-64.0) | 0.01 |
| Norepinephrine administration | 4.7 (0.5-42.2) | 0.17 |
| Acute renal failure | 0.4 (0.1-3) | 0.40 |
| APACHE II score | 1.2 (1.01-1.4) | 0.03 |
| SOFA score | 0.9 (0.7-1.2) | 0.57 |
| Albumin level | 0.6 (0.2-1.6) | 0.26 |
| Cortisol level | 1.0 (0.96-1.06) | 0.60 |

myocardial infarction and stroke in either diabetic or non-diabetic patients. In previous studies[4-6], definitions of stress glucose concentrations vary from 120 mg per deciliter to 198 mg per deciliter (on admission) or 110 mg per deciliter to 144 mg per deciliter (8 hours of fasting in the morning after admission). In our study, stress hyperglycemia was defined as fasting plasma glucose of more than 100 mg per deciliter or random plasma glucose level of more than 140 mg per deciliter. Previous studies in myocardial infarction and stroke found that stress hyperglycemia was associated with increased morbidity and mortality [4-6]. Meta-analysis of 32 cohort studies in non-diabetic patients with stroke found that stress hyperglycemia when defined as admission glucose being greater than 108 mg per deciliter was associated with 3-fold increase in hospital mortality[6]. In our study, the fasting stress glucose level was not associated with mortality possibly because we used a lower cut-off point of 100 mg per deciliter for diagnosis of stress hyperglycemia which might have included less severe hyperglycemic patients. Secondly, stress hyperglycemia in sepsis patients may be less harmful than that in patients with stroke and myocardial infarction. Thirdly and most likely, it is possible that the number of patients used in our study, although being adequate for measuring prevalence rate of stress hyperglycemia, is yet too small to yield enough power to detect the difference in the major outcome, which is the mortality rate in patients with stress hyperglycemia as compared to those without it. Finally, the level of blood glucose in the higher range of stress hyperglycemia may not predict mortality but control of hyperglycemia to a lower level between 80-110 mg per deciliter by insulin may be a better predictor of morbidity and mortality in critical patients[11,12].

From univariate analysis, we found that pulmonary infection, resistant gram negative infection, central line use, ventilator use, norepinephrine administration, higher APACHE II score, higher SOFA score, albumin and cortisol levels were associated with mortality. Serum cortisol level was correlated with APACHE II score because of increased corticosteroid secretion in response to stress in sepsis. However, after multivariate analysis, only APACHE II score and mechanical ventilator use were significantly associated with mortality. These imply that the severity of sepsis is the most important factor associated with mortality and severe patients need more frequent use of a mechanical ventilator.

In conclusion, the prevalence of stress hyperglycemia in sepsis patients was 42.3%. We cannot conclude that stress hyperglycemia is not associated with unfavorable outcomes mainly because of our small sample size. With the high prevalence of stress hyperglycemia, it may yet be shown to contribute to the unfavorable outcomes in patients with sepsis if further study with a larger sample size could show its association with mortality or major morbidity. The high APACHE II score and use of mechanical ventilators predicted high mortality.

**Acknowledgement**

This study was supported by research grant from the Faculty of Medicine Siriraj Hospital.

**References**

1. Melamed E. Reactive hyperglycaemia in patients with acute stroke. J Neurol Sci 1976; 29: 267-75.
2. Sewdarsen M, Jialal I, Vythilingum S, Govender G, Rajput MC. Stress hyperglycaemia is a predictor

of abnormal glucose tolerance in Indian patients with acute myocardial infarction. Diabetes Res 1987; 6: 47-9.
3. Mankovsky BN, Metzger BE, Molitch ME, Biller J. Cerebrovascular disorders in patients with diabetes mellitus. J Diabetes Complications 1996; 10: 228-42.
4. Capes SE, Hunt D, Malmberg K, Gerstein HC. Stress hyperglycaemia and increased risk of death after myocardial infarction in patients with and without diabetes: a systematic overview. Lancet 2000; 355: 773-8.
5. Bellodi G, Manicardi V, Malavasi V, Veneri L, Bernini G, Bossini P, et al. Hyperglycemia and prognosis of acute myocardial infarction in patients without diabetes mellitus. Am J Cardiol 1989; 64: 885-8.
6. Capes SE, Hunt D, Malmberg K, Pathak P, Gerstein HC. Stress hyperglycemia and prognosis of stroke in nondiabetic and diabetic patients: a systematic overview. Stroke 2001; 32: 2426-32.
7. Buczkowska EO. Alterations of blood glucose homeostasis during septic or injury stress—hyperglycemia. Wiad Lek 2002; 55: 731-44.
8. Gyurko R, Siqueira CC, Caldon N, Gao L, Kantarci A, Van Dyke TE. Chronic hyperglycemia predisposes to exaggerated inflammatory response and leukocyte dysfunction in Akita mice. J Immunol 2006; 177: 7250-6.
9. Trager K, DeBacker D, Radermacher P. Metabolic alterations in sepsis and vasoactive drug-related metabolic effects. Curr Opin Crit Care 2003; 9: 271-8.
10. Woodward M, Zhang X, Barzi F, Pan W, Ueshima H, Rodgers A, et al. The effects of diabetes on the risks of major cardiovascular diseases and death in the Asia-Pacific region. Diabetes Care 2003; 26: 360-6.
11. Van den Berghe G, Wouters P, Weekers F, Verwaest C, Bruyninckx F, Schetz M, et al. Intensive insulin therapy in the critically ill patients. N Engl J Med 2001; 345: 1359-67.
12. Van den Berghe G, Wilmer A, Hermans G, Meersseman W, Wouters PJ, Milants I, et al. Intensive insulin therapy in the medical ICU. N Engl J Med 2006; 354: 449-61.
13. Munford RS. Severe sepsis and septic shock. In: Kasper DL, Braunwald E, Fauci AS, Hauser SL, Longo DL, Jameson JC, editors. Harrison's principle of internal medicine. 16th ed. New York: McGraw-Hill; 2005: 1607.
14. Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. Crit Care Med 1985; 13: 818-29.
15. Giangiuliani G, Mancini A, Gui D. Validation of a severity of illness score (APACHE II) in a surgical intensive care unit. Intensive Care Med 1989; 15: 519-22.
16. Sudarsanam TD, Jeyaseelan L, Thomas K, John G. Predictors of mortality in mechanically ventilated patients. Postgrad Med J 2005; 81: 780-3.
17. Ferreira FL, Bota DP, Bross A, Melot C, Vincent JL. Serial evaluation of the SOFA score to predict outcome in critically ill patients. JAMA 2001; 286: 1754-8.
18. Vincent JL, de Mendonca A, Cantraine F, Moreno R, Takala J, Suter PM, et al. Use of the SOFA score to assess the incidence of organ dysfunction/failure in intensive care units: results of a multicenter, prospective study. Working group on "sepsis-related problems" of the European Society of Intensive Care Medicine. Crit Care Med 1998; 26: 1793-800.
19. Suleiman M, Hammerman H, Boulos M, Kapeliovich MR, Suleiman A, Agmon Y, et al. Fasting glucose is an important independent risk factor for 30-day mortality in patients with acute myocardial infarction: a prospective study. Circulation 2005; 111: 754-60.
20. Hadjadj S, Coisne D, Mauco G, Ragot S, Duengler F, Sosner P, et al. Prognostic value of admission plasma glucose and HbA in acute myocardial infarction. Diabet Med 2004; 21: 305-10.
21. Rosmarin DK, Wardlaw GM, Mirtallo J. Hyperglycemia associated with high, continuous infusion rates of total parenteral nutrition dextrose. Nutr Clin Pract 1996; 11: 151-6.

## ระดับกลูโคสในเลือดสูงจากภาวะเครียดในผู้ป่วยภาวะพิษเหตุติดเชื้อ

*ปิติพร รัตนทวีบุญ, วรการ วิไลชนม์, สาธิต วรรณแสง*

***ภูมิหลัง**: ผู้ป่วยซึ่งมีภาวะเครียดอย่างเฉียบพลันจากโรคหลอดเลือดสมองหรือโรคหลอดเลือดหัวใจอาจมีระดับกลูโคสในเลือดสูงผิดปกติ โดยที่ไม่ได้เป็นโรคเบาหวาน ระดับกลูโคสที่สูงในภาวะเครียดนี้เพิ่มอัตราตาย และอัตราป่วยในผู้ป่วยเหล่านี้ อย่างไรก็ดียังไม่มีการศึกษาความชุกและผลของระดับกลูโคสในเลือดสูงจากภาวะเครียดในผู้ป่วยภาวะภาวะพิษเหตุติดเชื้อมาก่อน*

***วัสดุและวิธีการ**: ศึกษาไปข้างหน้าในผู้ป่วยภาวะพิษเหตุติดเชื้อซึ่งรับไว้รักษาในภาควิชาอายุรศาสตร์ โรงพยาบาลศิริราช ระหว่างปี พ.ศ. 2549-2550 โดยรวบรวมข้อมูลระดับกลูโคสในเลือดเมื่อแรกรับไว้รักษา ระดับฮีโมโกลบิน เอวันซี และปัจจัยอื่น ซึ่งอาจมีผลต่อภาวะพิษเหตุติดเชื้อ*

***ผลการศึกษาและสรุป**: ได้ทำการศึกษาในผู้ป่วยภาวะพิษเหตุติดเชื้อจำนวน 70 ราย พบว่าอัตราความชุกของภาวะกลูโคสในเลือดสูงเท่ากับร้อยละ 42.3 การวิเคราะห์ข้อมูลชนิดหลายตัวแปรพบว่า คะแนน อปาเช และการใช้เครื่องช่วยหายใจ มีความสัมพันธ์อย่างอิสระคือ เพิ่มอัตราตายในผู้ป่วยเหล่านี้ ผลการศึกษาไม่พบว่าภาวะกลูโคสในเลือดสูง จากเครียดเพิ่มอัตราตายหรืออัตราป่วยในผู้ป่วยภาวะพิษเหตุติดเชื้อ เนื่องจากจำนวนผู้ป่วยอาจน้อยเกินไปจนไม่สามารถตรวจพบความแตกต่างทางสถิติได้*