

# Incidences and Outcomes of Hyperglycemic Crises: A 5-Year Study in a Tertiary Care Center in Thailand

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**Objective:** To assess the incidences and outcomes of hyperglycemic crises.

**Material and Method:** A retrospective study of diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) in adults with type 1 or type 2 diabetes mellitus (DM) admitted to Thammasat Hospital between 2006 and 2010 was performed. Incidences, precipitating causes, clinical and laboratory characteristics, and treatment outcomes of hyperglycemic crises were obtained via medical record review. Multivariate logistic regression analysis was used to determine predictors for mortality.

**Results:** Eighty-three patients were eligible and included. The mean age was  $54.9 \pm 17.7$  years old. Most subjects had type 2 DM (86.7%). The 5-year incidence of hyperglycemic crises was 7.46%. Diabetic ketoacidosis occurred more frequently than HHS (4.67% vs. 1.71%). During the hyperglycemic episodes, the mean plasma glucose level on admission was  $741.3 \pm 320.8$  mg/dL. Infections were the most common precipitating factor [61/83 (73.5%)], followed by non-compliance with treatments [35/83 (42.2%)]. Treatment complications included recurrent hyperglycemia (69.9%), hypokalemia (48.2%), hyponatremia (21.7%), and hypoglycemia (15.7%). The overall mortality rate of hyperglycemic crises was 8.4% (5.8% in DKA, 15.8% in HHS and 8.3% in the overlap of both conditions). The most common causes of death were infections [5/7 (71.4%)]. By multivariate analysis, serum sodium level on admission was independently associated with mortality (adjusted odds ratio 1.08, 95% CI 1.01-1.16,  $p = 0.03$ ).

**Conclusion:** Hyperglycemic crises were common in the authors' setting. Diabetic ketoacidosis occurred more frequently but had a lower mortality rate than HHS. Complications from hyperglycemic crisis treatment could be prevented by close monitoring, while high serum sodium level on admission was a predictor for mortality. Strategies to prevent infections and improve treatment compliance are needed to reduce the incidence of hyperglycemic crises among patients with DM.

**Keywords:** Hyperglycemic crises, Mortality rate, Complications, Adults, Thai

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Hyperglycemic crises, diabetic ketoacidosis (DKA), and hyperosmolar hyperglycemic state (HHS) are the most serious acute complications of diabetes mellitus (DM), which result in significant morbidity and mortality. The pathogenesis of these hyperglycemic crises is the combination of insulin deficiency and increased counter-regulatory hormones. In consequence, hyperglycemia develops as a result of increased gluconeogenesis, accelerated glycogenolysis, and impaired glucose utilization by peripheral tissues, while lipolysis causes ketoacidosis.

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In Western countries, the annual incidence of DKA was about 4.6 to 8 episodes/1,000 diabetic patients<sup>(1)</sup>. Recent epidemiologic studies indicated that hospitalizations for DKA were increasing<sup>(2-4)</sup>. Although DKA is common in patients with type 1 DM, it can occur in patients with type 2 DM. The mortality rate of DKA in adult patients was less than 1%<sup>(3)</sup>. However, more than 5% of mortality rate was associated with advanced age and concomitant life-threatening illnesses<sup>(5,6)</sup>. The mortality rate of HHS was reported to be as much as 20% higher than DKA<sup>(7)</sup>.

In Thailand, a medical school hospital reported the average annual incidence of DKA during the period from 1970 to 1988 was 16%<sup>(8)</sup>. More recently, another hospital reported the incidence of DKA was 11.4% between 2000 and 2007 (unpublished

data from Rajavithi Hospital in 2008). Although the treatments for DKA and HHS have been improving for the last 5 years, data on incidences and outcomes of DKA and HHS in Thailand are lacking. Therefore, the present study aimed to describe the incidences and outcomes of hyperglycemic crises in adult patients with DM from 2006 to 2010.

## Material and Method

### Study design and population

A medical record of all episodes of hyperglycemic crises in both type 1 and type 2 DM adult patients (age 18 years old or more) occurring during the period from 2006 to 2010 in Thammasat Hospital was performed. Thammasat Hospital is a 570-bed tertiary-care hospital in central Thailand and is affiliated with Thammasat University Medical School. Eligible patients were identified using World Health Organization (WHO) ICD-10 codes, E10.0, E10.1, E11.0 and E11.1. In patients who presented with more than one episode of hyperglycemic crises, only the first episode was analyzed. The present study protocol was approved by the Research and Ethic Committee of Thammasat University.

### Study definitions and data collection

During the medical record review, diagnoses of DKA and HHS were confirmed according to the American Diabetes Association (ADA) criteria<sup>(3)</sup>. The criteria for diagnosing DKA include serum glucose being more than 250 mg/dL, positive serum or urine ketones and wide anion gap with metabolic acidosis (serum bicarbonate less than 18 mmol/L and anion gap more than 10 or arterial pH less than 7.30). Patients with HHS were diagnosed by the criteria including serum glucose being more than 600 mg/dL and effective serum osmolality being more than 320 mOsm/kg with or without alteration of consciousness.

Data on demographics, duration of DM, current treatment, precipitating factors for hyperglycemic crises, laboratory data, treatment complications, and outcomes of hyperglycemic crises were collected. Serum and urine ketones were measured using the sodium nitroprusside method on Urisys 1800 urinalysis analyzer (Roche Diagnostics, Germany). Hypoglycemia was defined as serum glucose or capillary blood glucose of less than 70 mg/dL in the first 24 hours after admission due to insulin overtreatment or inadequate dextrose solution supplement. The recurrence of hyperglycemia was defined as hyperglycemia (serum glucose or capillary blood glucose being more than

250 mg/dL in DKA or 300 mg/dL in HHS) in the first 24 hours after resolution of DKA or HHS. Hyponatremia was defined as serum sodium level of more than 145 mmol/L in the first 24 hours after admission due to inappropriate fluid management and hypokalemia was defined as serum potassium level of less than 3.5 mmol/L in the first 24 hours after admission due to inadequate or no potassium replacement.

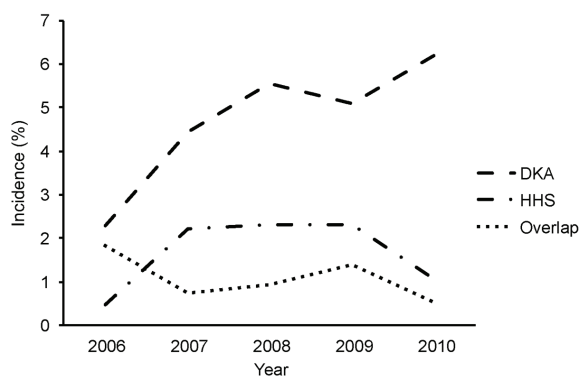
### Statistical analysis

Analyses were performed using SPSS version 13.0 (SPSS, Chicago, IL, USA). The incidences of hyperglycemic crises were described in annual percentage. Categorical data was compared using Chi-square test or Fisher exact test, as appropriate. Continuous data was compared using student t-test and one-way analysis of variance (ANOVA), as appropriate. All p-value were two-tailed and p less than 0.05 was considered statistically significant. Multivariate analysis was used to determine factors associated with mortality. Adjusted odds ratio and 95% confidence intervals were calculated.

## Results

### Incidence of hyperglycemic crises and patients' characteristics

Eighty-three eligible patients with hyperglycemic crises were identified. The annual incidences of each hyperglycemic crisis are shown in Fig. 1. Diabetic ketoacidosis occurred two to three times more frequently than HHS throughout the 5-year period. The incidence of DKA had increased over



DKA = diabetic ketoacidosis; HHS = hyperosmolar hyperglycemic state; Overlap = overlap of DKA and HHS

Fig. 1 Five-year annual incidences of different types of hyperglycemic crises in Thammasat Hospital

time while the incidence of HHS and the overlap of both conditions had not changed significantly.

Demographic and clinical characteristics of the 83 patients are summarized in Table 1. The most common diagnosis of hyperglycemic crises was DKA [n = 52 (62.6%)] followed by HHS [n = 19 (22.9%)] and the overlap of both conditions [n = 12 (14.5%)]. The mean age was  $54.9 \pm 17.7$  years. Patients with HHS were significantly older than the other groups. These 83 patients had DM for the mean duration of  $5.5 \pm 6.3$  years; 72 patients (86.7%) had type 2 DM. The most common precipitating factor for hyperglycemic crises was infection [n = 61 (73.5%)], followed by noncompliance with treatments [n = 35 (42.2%)]. The remaining precipitating factors were cardiovascular diseases (myocardial infarction, stroke), thyrotoxicosis, no previous DM treatment, and unknown precipitators. Hyperglycemic crisis was the first presentation and contributed to the diagnosis of DM in 18 patients (21.7%).

Laboratory data on admission for patients with DKA, HHS and the overlap of both conditions are shown in Table 2. The mean serum glucose level was  $741.3 \pm 320.8$  mg/dL. Patients with the overlap of both conditions had significantly higher mean serum glucose than the other groups ( $p < 0.001$ ). The mean serum sodium level was  $134.7 \pm 13.5$  mmol/L (corrected mean serum sodium level  $144.9 \pm 14.2$  mmol/L). Patients with HHS had significant higher mean serum sodium level than the other groups ( $p < 0.001$ ).

#### Complications and outcomes of hyperglycemic crises

The mortality rates of hyperglycemic crises are summarized in Table 3. Seven patients died; five from infections and two from myocardial infarction. The overall mortality rate was 8.4%. Patients with HHS had the highest mortality rate. The complications of hyperglycemic crisis treatment are summarized in Table 4. The recurrence of hyperglycemia (69.9%)

**Table 1.** Demographic and clinical characteristic of patients with hyperglycemic crises

	DKA (n = 52)	HHS (n = 19)	Overlap (n = 12)	All (n = 83)	p-value
Age (years $\pm$ SD)	$48.9 \pm 17.6$	$67.1 \pm 12.5$	$61.7 \pm 13.2$	$54.9 \pm 17.7$	<0.001
Sex					
Male	24 (46.2)	10 (52.6)	5 (41.7)	39 (47.0)	
Female	28 (53.8)	9 (47.4)	7 (58.3)	44 (53.0)	0.82
BW (kg $\pm$ SD)	$61.0 \pm 15.0$	$60.6 \pm 11.0$	$55.3 \pm 8.9$	$60.1 \pm 13.4$	0.40
Type of DM					
Type 1	9 (17.3)	0 (0)	2 (16.7)	11 (13.3)	
Type 2	43 (82.7)	19 (100)	10 (83.3)	72 (86.7)	0.15
Duration (years)	$5.8 \pm 6.6$	$5.0 \pm 5.1$	$5.0 \pm 6.9$	$5.5 \pm 6.3$	0.87
Treatment type					
Diet	1/37 (2.7)	1/12 (8.3)	2/7 (28.6)	4/56 (7.1)	0.06
Oral	16/37 (43.2)	7/12 (58.3)	1/7 (14.3)	24/56 (42.9)	
Insulin	13/37 (35.1)	4/12 (33.3)	4/7 (57.1)	21/56 (37.5)	
Combination	7/37 (18.9)	0 (0)	0 (0)	7/56 (12.5)	
Precipitating factor*					
Infection	35	16	10	61	0.79
Non-compliance with treatments	25	6	4	35	
CVD	2	0	0	2	
Others	7	3	2	12	

Data were presented in number (%) unless otherwise indicated

DKA = diabetic ketoacidosis; HHS = hyperosmolar hyperglycemic state; Overlap = overlap of DKA and HHS; BW = body weight; CVD = cardiovascular diseases composed of myocardial infarction and stroke

\* Each patient could have more than one precipitating factors

**Table 2.** Laboratory data on admission of patients with hyperglycemic crises

	DKA (n = 52)	HHS (n = 19)	Overlap (n = 12)	All (n = 83)	p-value
Serum glucose (mg/dL)	627.2 ± 304.6	813.8 ± 170.7	1,121.0 ± 247.8	741.3 ± 320.8	<0.001
HbA1c (%)	12.1 ± 3.0	11.6 ± 3.5	12.1 ± 2.7	12.0 ± 3.0	0.83
Serum ketone					
Negative	0 (0)	14 (73.7)	0 (0)	14 (16.9)	<0.001
Positive	52 (100)	5 (26.3)	12 (100)	69 (83.1)	
Urine ketone					
Negative	6 (11.5)	12 (63.2)	1 (8.3)	19 (22.9)	<0.001
Positive	46 (88.5)	7 (36.8)	11 (91.7)	64 (77.1)	
Electrolytes (mmol/L)					
Na	127.8 ± 8.2	149.6 ± 12.0	141.0 ± 12.4	134.7 ± 13.5	<0.001
K	5.0 ± 1.0	4.6 ± 0.9	4.7 ± 1.0	4.8 ± 1.0	0.25
Cl	91.7 ± 8.7	110.5 ± 13.3	102.3 ± 13.2	97.5 ± 13.2	<0.001
HCO <sub>3</sub>	9.8 ± 5.2	20.1 ± 5.8	10.2 ± 4.6	12.2 ± 6.7	<0.001
Effective osmolality* (mOsm/kg)	290.4 ± 12.5	334.5 ± 23.2	334.3 ± 27.0	310.5 ± 31.7	<0.001
pH ABG	7.18 ± 0.2	7.35 ± 0.1	7.17 ± 0.1	7.22 ± 0.2	<0.001
BUN (mg/dL)	34.0 ± 36.0	84.2 ± 60.7	56.0 ± 22.6	48.7 ± 46.0	<0.001
Cr (mg/dL)	2.5 ± 4.5	3.5 ± 2.9	2.8 ± 0.8	2.8 ± 3.9	0.63
Anion gap**	26.3 ± 5.2	19.0 ± 6.5	28.5 ± 6.2	24.9 ± 6.5	<0.001

Data were presented in number (%). Continuous variables were presented in mean ± SD

DKA = diabetic ketoacidosis; HHS = hyperosmolar hyperglycemic state; Overlap = overlap of DKA and HHS; ABG = arterial blood gas; BUN = blood urea nitrogen; Cr = creatinine

\* Effective serum osmolality = 2 (measured Na (mmol/L)) + glucose (mg/dL)/18

\*\* Anion gap = Na - (Cl + HCO<sub>3</sub>) (mmol/L)

**Table 3.** Mortality rates among patients with hyperglycemic crises

	DKA (n = 52)	HHS (n = 19)	Overlap (n = 12)	All (n = 83)	p-value
Death	3 (5.8)	3 (15.8)	1 (8.3)	7 (8.4)	0.41
Causes of death					
Infection	2 (66.7)	2 (66.7)	1 (100)	5 (71.4)	0.79
MI	1 (33.3)	1 (33.3)	0 (0)	2 (28.6)	

Data were presented in number (%)

DKA = diabetic ketoacidosis; HHS = hyperosmolar hyperglycemic state; Overlap = overlap of DKA and HHS; MI = myocardial infarction

and hypokalemia (48.2%) were common treatment complications. There were significant differences in rates of hypernatremia (11.5% in DKA, 36.8% in HHS and 41.7% in overlap groups) and hypokalemia (36.5% in DKA, 63.2% in HHS and 75% in overlap groups) during the treatment between the three groups ( $p = 0.01$  and  $p = 0.02$ , respectively).

Demographic, clinical and laboratory characteristics, on admission and complications were

compared between patients who died and survived (Table 5). The most common cause of death was infection [5/7 (71.4%)]. By univariate analysis, patients who died had significantly higher serum sodium level, serum chloride level, effective osmolality, BUN, and creatinine levels than those who survived. By multivariate logistic regression analysis, serum sodium level on admission was independently associated with mortality (adjusted odds ratio = 1.08, 95% confidence

**Table 4.** Complications of treatment for hyperglycemic crises

	DKA (n = 52)	HHS (n = 19)	Overlap (n = 12)	All (n = 83)	p-value
Hypoglycemia	8 (15.4)	3 (15.8)	2 (16.7)	13 (15.7)	0.99
Causes of hypoglycemia					
Insulin overtreatment	6/8 (75.0)	3/3 (100)	2/2 (100)	11/13 (84.6)	0.48
Inadequate dextrose solution	2/8 (25.0)	0/3 (0)	0/2 (0)	2/13 (15.4)	
Recurrence of hyperglycemia	40 (76.9)	10 (52.6)	8 (66.7)	58 (69.9)	0.14
Causes of recurrent hyperglycemia					
Insulin discontinuation	9/40 (22.5)	2/10 (20.0)	1/8 (12.5)	12/58 (20.7)	0.92
Inadequate insulin therapy	30/40 (75.0)	8/10 (80.0)	7/8 (87.5)	45/58 (77.6)	
No overlap of insulin during transition period	1/40 (2.5)	0/10 (0)	0/8 (0)	1/58 (1.7)	
Hyponatremia	6 (11.5)	7 (36.8)	5 (41.7)	18 (21.7)	0.01
Hypokalemia	19 (36.5)	12 (63.2)	9 (75.0)	40 (48.2)	0.02
Causes of hypokalemia, No. (%)					
No K replacement	7/19 (36.8)	6 (50.0)	4 (44.4)	17 (42.5)	0.76
Inadequate K replacement	12/19 (63.2)	6 (50.0)	5 (55.6)	23 (57.5)	

Data were presented in number (%)

DKA = diabetic ketoacidosis; HHS = hyperosmolar hyperglycemic state; Overlap = overlap of DKA and HHS

**Table 5.** Demographic data, clinical characteristics, laboratory data on admissions and complications of the death and survival groups

	Death (n = 7)	Survival (n = 76)	p-value
Age (years)	64.9 ± 17.6	54.0 ± 17.6	0.16
Sex			
Male	2 (18.6)	37 (48.7)	0.44
Female	5 (71.4)	39 (51.3)	
Blood sugar (mg/dL)	708.0 ± 226.4	774.4 ± 329.1	0.71
HbA <sub>1c</sub> (%)	10.9 ± 1.4	12.1 ± 3.1	0.08
Electrolytes (mmol/L)			
Na	150.0 ± 12.3	133.3 ± 12.8	0.01
K	4.6 ± 1.2	4.9 ± 0.9	0.59
Cl	110.7 ± 12.9	96.3 ± 12.6	0.03
HCO <sub>3</sub>	13.8 ± 4.2	12.1 ± 6.9	0.36
Corrected Na* (mmol/L)	159.7 ± 14.7	143.6 ± 13.3	0.03
Effective osmolality (mOsm/kg)	339.3 ± 33.6	307.9 ± 30.4	0.05
BUN (mg/dL)	96.9 ± 75.5	44.3 ± 40.3	0.003
Cr (mg/dL)	7.0 ± 11.3	2.4 ± 2.1	0.002
Hypoglycemia	3 (42.9)	10 (13.2)	0.07

Data were presented in number (%). Continuous variables were presented in mean ± SD

DKA = diabetic ketoacidosis; HHS = hyperosmolar hyperglycemic state; Overlap = overlap of DKA and HHS; ABG = arterial blood gas; BUN = blood urea nitrogen; Cr = creatinine

\* Corrected Na = [(serum glucose (mg/dL) - 100)/100] x 1.6

**Table 6.** Multivariate analysis for factors associated with mortality among patients with hyperglycemic crises

Factor	Adjusted odds ratio (95% confidence interval)	p-value
Serum sodium level	1.08 (1.01-1.16)	0.03
Creatinine level	1.16 (0.95-1.41)	0.14
Hypoglycemia	3.53 (0.40-30.85)	0.26
Age	1.02 (0.94-1.10)	0.67

interval 1.01-1.16,  $p = 0.03$ ) after adjusting for age, creatinine and hypoglycemia during the treatment (Table 6).

### Discussion

From the present study, the annual incidence of DKA increased from 2.29% in 2006 to 6.22% in 2010. This trend was similar to that reported in the studies from USA and Korea<sup>(2,3)</sup>. However, the annual incidences of HHS and the overlap of both conditions had not increased over time. Most of the patients with DKA had type 2 DM (86.7%). This was due to the fact that the prevalence of type 2 DM was higher than that of type 1 DM<sup>(9-11)</sup>. The present study revealed 12 patients (14.5%) diagnosed with overlap of both DKA and HHS. Two patients with type 1 DM were diagnosed with this condition. The previous studies also reported that HHS could occur in type 1 DM<sup>(12,13)</sup>. However, the diagnosis of diabetes type in the present study was not definitive due to lack of autoantibody data. The overall mean duration of DM before developing hyperglycemic crises was  $5.5 \pm 6.3$  years, which was similar to those reported in previous studies<sup>(2,10,14)</sup>. Like the other studies, the common precipitating factors of DKA and HHS were infections and non-compliance with treatments<sup>(1,2,9-11,15,16)</sup>. These results emphasized the importance of infection prevention and drug compliance during sick-day to prevent hyperglycemic crises.

In the present study, the recurrence of hyperglycemia and hypokalemia were common complications. The causes of hypokalemia were no or inadequate potassium replacement. Therefore, it is important to monitor and replace potassium according to the ADA guideline<sup>(3)</sup>. The recurrence of hyperglycemia was caused by inappropriate insulin discontinuation, therapy, and overlap during the transition period. Hence, physicians should adjust insulin depending on serum glucose level and balance between the caloric

intake from glucose and insulin administration. During the transition period, the patients should receive the overlap of 1 to 2 hours between discontinuation of intravenous insulin and the administration of subcutaneous insulin to prevent the recurrence of hyperglycemia. Nevertheless, no recurrence of hyperglycemic crises occurred in the present study.

The mortality rate of HHS was 15.8% and higher than that of DKA. This result was consistent with those observed 18-35% higher mortality rate in HHS as compared to DKA in other studies<sup>(9,14,17-20)</sup>. According to ADA, the mortality rate of DKA in adult patients is less than 1%. The higher mortality rate in the present study emphasized the importance of aggressive treatment and monitoring process. This high mortality was consistent with the rate of 7 to 20% in the previous studies from resource-limited settings<sup>(9,10,14,17,18)</sup>. The authors found that the most common cause of death following hyperglycemic crises was infections that were similar to those reported in the previous studies<sup>(2,9,10,14,21)</sup>. Several factors associated with mortality in DKA and HHS patients have been identified previously. These included advanced age<sup>(14,17,18,21)</sup>, alteration of mental status<sup>(17)</sup>, serum urea level<sup>(19)</sup> and the degree of hyperosmolarity<sup>(21)</sup>. In the present study, the authors found that only serum sodium level on admission was independently associated with mortality. Because serum sodium levels could indicate the patients' volume status, the high level of serum sodium on admission might indicate the severity of dehydration, which could result in renal insufficiency, treatment difficulties, and mortality. In addition, hypernatremia might be implied to the patients who could not access free water by themselves, thus reflecting their underlying disabilities and comorbidities that increased risk for mortality<sup>(22)</sup>.

There were notable limitations in the present study. First, given the nature of retrospective study, missing data and misclassification biases could occur. Second, small sample size limited the detection of other factors that might be associated with mortality, such as age and other comorbidities. Third, information about clinical presentations of hyperglycemic crises was not retrievable through medical record review process, thus, these variables were not taken into account for analysis of predictors for mortality. Although the authors found that the serum sodium level on admission was significantly associated with mortality, there might be other confounding factors such as vital signs or Acute Physiology and Chronic Health Evaluation II (APACHE II) score that were

not adjusted for the multivariate analysis. Lastly, generalization of the study findings might limit to tertiary-care hospital settings in Thailand.

In conclusion, the annual incidence of DKA had been increasing in Thammasat Hospital, while the incidence of HHS had been stable but HHS had contributed to the highest mortality rate. Prevention of infections and improving patients' compliance with DM treatment would reduce the incidences of hyperglycemic crises. Close monitoring was required for patients with high sodium level on admission and during treatment of hyperglycemic crises. Strategies including sick-day management education and appropriate treatment of complications should be incorporated into management of hyperglycemic crises.

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#### Potential conflicts of interest

None.

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## อุบัติการณ์การเกิดและผลลัพธ์ของภาวะฉุกเฉินเนื่องจากระดับน้ำตาลในเลือดสูง: การศึกษาระยะเวลา 5 ปี ในศูนย์ดูแลตติยภูมิในประเทศไทย

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**วัตถุประสงค์:** เพื่อที่จะศึกษาอุบัติการณ์และผลลัพธ์ของภาวะฉุกเฉินจากน้ำตาลในเลือดสูงของผู้ป่วยเบาหวาน

**วัตถุประสงค์และวิธีการ:** ใช้การศึกษาข้อมูลย้อนหลังของผู้ป่วยที่มีภาวะฉุกเฉินจากน้ำตาลในเลือดสูง ในผู้ป่วยเบาหวานชนิดที่ 1 และ 2 ที่เข้ารับการรักษาในโรงพยาบาลธรรมศาสตร์เฉลิมพระเกียรติ ปี พ.ศ. 2549-2553 โดยนำข้อมูลบันทึกทางการแพทย์มาศึกษาเพื่อหาอุบัติการณ์ลักษณะทางคลินิก ผลทางห้องปฏิบัติการ และผลลัพธ์ของการรักษา

**ผลการศึกษา:** ผู้ป่วยที่เข้าการศึกษามีจำนวน 83 คน อายุเฉลี่ย  $54.9 \pm 17.7$  ปี ส่วนใหญ่เป็นเบาหวานชนิดที่ 2 (ร้อยละ 86.7) อุบัติการณ์ของการเกิดภาวะฉุกเฉินจากน้ำตาลในเลือดสูงของผู้ป่วยเบาหวานที่เข้ารับการรักษาในโรงพยาบาลธรรมศาสตร์เฉลิมพระเกียรติ 5 ปี คิดเป็นร้อยละ 7.64 การเกิด *diabetic ketoacidosis* (DKA) พบได้บ่อยกว่า *hyperosmolar hyperglycemic state* (HHS) (ร้อยละ 4.67 เทียบกับ 1.71) ช่วงที่เกิดภาวะฉุกเฉินจากน้ำตาลในเลือดสูง ผู้ป่วยมีระดับน้ำตาลเฉลี่ย  $741.3 \pm 320.8$  มิลลิกรัม/เดซิลิตร พบว่าการติดเชื้อเป็นปัจจัยกระตุ้นที่พบบ่อยสุด [61/83 (ร้อยละ 73.5)] รองลงมาเป็นจากผู้ป่วยไม่รักษาเบาหวานต่อเนื่อง ภาวะแทรกซ้อนหลังการรักษา ได้แก่ การเกิดระดับน้ำตาลกลับมาสูง (ร้อยละ 69.9) ภาวะโพแทสเซียมในเลือดต่ำ (ร้อยละ 48.2) ภาวะโซเดียมในเลือดสูง (ร้อยละ 21.7) และระดับน้ำตาลในเลือดต่ำ (ร้อยละ 15.7) เป็นต้น อัตราการเสียชีวิตโดยรวมของภาวะฉุกเฉินจากน้ำตาลในเลือดสูงคิดเป็นร้อยละ 8.4 (อัตราการเสียชีวิตจาก DKA คิดเป็นร้อยละ 5.8 จาก HHS คิดเป็นร้อยละ 15.8 และจากทั้ง 2 ภาวะร่วมกันคิดเป็นร้อยละ 8.3) โดยสาเหตุการตายที่พบบ่อยสุดคือการติดเชื้อ [5/7 (ร้อยละ 71.4)] และพบว่าระดับโซเดียมที่สูงขึ้นมีความสัมพันธ์กับอัตราการตาย (*adjusted odds ratio* 1.08 (95% CI 1.01-1.16),  $p = 0.03$ )

**สรุป:** ภาวะฉุกเฉินจากน้ำตาลในเลือดสูงเป็นภาวะที่พบได้บ่อย DKA เกิดได้บ่อยกว่า แต่อัตราการเสียชีวิตน้อยกว่า HHS ภาวะแทรกซ้อนที่พบได้บ่อยหลังการรักษาควรป้องกันโดยติดตามอย่างใกล้ชิด และพบว่าระดับโซเดียมที่สูงเมื่อแรกรับเข้าโรงพยาบาลเป็นตัวบ่งชี้ถึงอัตราการตายการป้องกันการติดเชื้อ และการแนะนำให้รักษาอย่างสม่ำเสมอจะช่วยลดการเกิดภาวะฉุกเฉินจากน้ำตาลในเลือดสูงในผู้ป่วยเบาหวานได้