Hyperglycaemic crisis in the Eastern Cape province of South Africa: High mortality and association of hyperosmolar ketoacidosis with a new diagnosis of diabetes

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Objectives. To describe the frequencies, presenting characteristics (demographic, clinical and biochemical) and outcomes (duration of admission and mortality rates) for various types of hyperglycaemic crisis.

Methods. Retrospective review of medical records of patients with hyperglycaemic crisis admitted to Nelson Mandela Academic Hospital, Mthatha, E Cape, from 1 January 2008 to 31 December 2009. Outcome measures were duration of admission and mortality.

Results. Data were available for 269 admissions (response rate 81.0%), 169 females and 100 males. Admissions for hyperglycaemia (HG, *N*=119), and non-hyperosmolar diabetic ketoacidosis (NHDKA, *N*=97) were more frequent than those for hyperosmolar hyperglycaemic state (HHS, *N*=29) and hyperosmolar diabetic ketoacidosis (HDKA, *N*=24). Duration of admission was similar in all groups. Mortality was high in all groups, but was higher in patients with HDKA (37.5%, risk ratio (RR) 3.88, 95% confidence interval (CI) 1.41 - 10.67, p=0.009), HHS (31.0%, RR 2.91, 95% CI 1.09 - 7.75, p=0.033) and HG (19.5%, RR 1.56, 95% CI 0.75 - 3.21, p=0.236) than in those with NHDKA (13.4%). HDKA (62.5%) was associated with new-onset diabetes more often than NHDKA (27.8%), HHS (44.8%) or HG (17.6%) (p<0.0001). An altered level of consciousness was more frequent in HDKA than NHDKA admissions (RR 5.71, 95% CI 1.90 - 17.17, p=0.002).

Conclusions. Duration of hospital stay was similar across groups. Mortality rates were high in all groups. New-onset diabetes, altered level of consciousness and mortality were more characteristically associated with HDKA than any of the other types of hyperglycaemic crisis. Optimal glycaemic control in known diabetic patients will reduce rates of hyperglycaemic crisis admissions.

S Afr Med J 2010; 100: 822-826.

Diabetic hyperglycaemic crisis is a major reason for high-care unit admissions,[1] with mortality rates of up to 30%.[2-9] It is characteristically classified as ketoacidotic or hyperosmolar.[10] Presentations with the combination of ketoacidosis and hyperosmolality are recognised,[6,7,11-16] and have variably been described as ketoacidosis with hyperosmolality or hyperosmolar state with ketoacidosis, depending on whether ketoacidosis or hyperosmolality was considered dominant.[15]

Therapy and outcome of treatment for hyperglycaemic crisis is related to the presence of hyperosmolality and ketoacidosis.[1] Hyperosmolality is associated with the use of hypotonic fluids, anticoagulation and the more frequent occurrence of vascular thrombosis,[17] while ketoacidosis may necessitate the use of sodium bicarbonate.[18]

Hyperglycaemic hyperosmolar non-ketoacidosis is associated with a higher morbidity and mortality than diabetic ketoacidosis,[6,7,19] which is mainly attributed to hyperosmolality, older age[6,7] and the more frequent association of hyperosmolar non-ketoacidosis with type 2 diabetes.[19] Few studies[6,7,11,13] have described the clinical and laboratory features and outcomes of treatment for hyperosmolar diabetic ketoacidosis. A single report from Africa[11] included only 8 cases.

This study investigated the frequencies, presenting characteristics and outcomes of admissions for various types of hyperglycaemic crisis at Nelson Mandela Academic Hospital, Walter Sisulu University (WSU), Mthatha. The hospital is the referral centre for primary and secondary health care facilities in the O R Tambo district of the Eastern Cape province of South Africa, which has a population of about 1.7 million people.[20] Like the rest of the province the O R Tambo district has poorly developed health infrastructure and a disproportionately high burden of communicable diseases, particularly HIV/AIDS, tuberculosis and neurocysticercosis.

Methods

A retrospective review of hospital records of patients with hyperglycaemic crisis admitted to the medical department of Nelson Mandela Academic Hospital from 1 January 2008 to 31 December 2009 was performed. Hyperglycaemic crisis referred to admissions in which insulin infusion and intravenous fluid rehydration were initiated. Data from the records were socio-demographic information, diabetes type, precipitating cause for hyperglycaemic crisis, serum biochemical measurements at presentation, duration of admission and in-hospital deaths. The study was approved by the Ethics Committee of WSU.

Admissions were categorised as follows: (*i*) non-hyperosmolar diabetic ketoacidosis (NHDKA); (*ii*) hyperosmolar diabetic ketoacidosis (HDKA); (*iii*) hyperosmolar hyperglycaemic state (HHS); and (*iv*) hyperglycaemia (HG). Definitions used were: NHDKA – blood glucose >13.9 mmol/l, ketosis, serum bicarbonate < 18 mmol/l and calculated effective serum osmolality ≤320 mosmol/kg; HDKA – blood glucose >13.9 mmol/l, ketosis, serum bicarbonate

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<18 mmol/l, and calculated effective serum osmolality >320 mosm/kg; HHS – blood glucose >33.3 mmol/l, serum bicarbonate ≥18 mmol/l, and calculated effective serum osmolality >320 mosm/kg; and HG – blood glucose >13.9 mmol/l, serum bicarbonate ≥18 mmol/l, and calculated effective serum osmolality ≤320 mosm/kg. Ketosis referred to at least 1+ ketonuria on dipstick urinalysis or trace ketonaemia on qualitative laboratory serum analysis. Acidosis and severe acidosis were diagnosed on the basis of serum bicarbonate levels of <18 mmol/l and <10 mmol/l, respectively. Ketoacidosis referred to ketosis with serum bicarbonate level <18 mmol/l. Effective osmolality was calculated as: [2(serum sodium in mmol/l) + plasma glucose in mmol/l]. Type of diabetes (type 1 and type 2) was based on the diagnosis before the index hyperglycaemic crisis. New diabetes referred to patients without a previous diagnosis of diabetes. Impaired level of consciousness was defined as a Glasgow Coma Scale <15, hypernatraemia as uncorrected serum sodium >150 mmol/l, hypokalaemia as serum potassium <3.5 mmol/l, hyperkalaemia as serum potassium >5.5 mmol/l, hypochloraemia as serum chloride <94 mmol/l, and hyperchloraemia as serum chloride >104 mol/l. Precipitating factors for hyperglycaemic crisis refer to factors including co-morbidities that are associated with deterioration in glycaemic levels.

Treatment consisted of intravenous fluids and insulin administered according to standard protocols.[10] Blood glucose levels were monitored hourly and dipstick urinalysis for ketones done 4-hourly, while serum electrolytes, urea and creatinine were measured once daily. Potassium replacement as 20 mmol of potassium chloride was added to each litre of rehydration fluid, after hyperkalaemia had been excluded. Use of anticoagulation in patients with hyperosmolality and sodium bicarbonate therapy in severe acidosis was rare.

Statistical analysis

Data were presented as the median (interquartile range) for continuous variables and proportions (%) with (number of admissions) for discrete variables. The non-parametric Kruskal-Wallis one-way ANOVA was used to compare continuous variables between the four types of hyperglycaemic crisis, with post-hoc comparisons of mean ranks of all pairs of groups. Proportions between two groups were compared using Pearson's chi-square test. Proportions across all groups were compared using Pearson's chi-square test with a Bonferroni adjustment. All tests were two-sided and a p-value <0.05 was considered significant. Logistic regression models were fitted to compare risk of mortality, altered level of consciousness and onset of new diabetes between the four types of hyperglycaemic crisis. Risk was expressed as risk ratio (RR), using the NHDKA as the baseline risk group. Data were captured on a customised MS Excel spread sheet and analysed using SPSS version 15 (SPSS, Inc, Chicago, IL, USA) and Statistica version 9 (StatSoft, Tulsa, Oklahoma, USA).

Results

There were 764 diabetes-related admissions to the medical wards during the study; 332 were for DKA, HHS and HG. Complete data on key variables such as age, gender, type of diabetes, blood glucose, serum sodium, serum bicarbonate, ketosis and duration of admission (Table I) were available for 269 admissions, 169 females and 100 males (response rate 81%). They comprised 119 HG (44.2%), 97 NHDKA (36.1%), 29 HHS (10.8%) and 24 HDKA (8.9%). Ten patients admitted 2 - 7 times accounted for a total of 33 admissions (Table II). There were readmissions for NHDKA, HDKA and HG but not for HHS. The HHS group included 6 admissions with blood glucose levels below 33.3 mmol/l. Seven admissions with blood glucose levels below 13.9 mmol/l were observed, 4 for NHDKA, 1 for HDKA and 2 for HG; this was attributed to initial treatment at the referring health facilities, which typically took the form of normal saline infusion and 10 units intravenous bolus of soluble insulin. The 63 admissions for which records were not available had the following diagnoses: DKA (N=11), HHS (N=5) and HG (N=47).

The mean ages in the two DKA groups were similar, and significantly lower than in the HHS and HG groups (Table I). No person with HHS was younger than 40 years or had type 1 diabetes, in contrast to other groups. Females dominated all groups except the HHS group, which had a majority of males.

Precipitating factors were identified in 36.1 - 44.8% of admissions across the four groups. The predominant factors were infections (N=62), cerebrovascular disease (N=17) and poor drug compliance (N=11). Of the 17 cases of cerebrovascular disease, 12 were in the HG group, 3 in the HHS group, 2 in the HDKA group and none in the NHDKA group. One of the 10 patients who died had type 1 diabetes and died during her 7th admission. The median durations of hospitalisation were comparable in all groups.

The four groups had equally high glycated haemoglobin (HbA1c) levels at admission (Table III): 1.6% of NHDKA, 0% of HDKA, 0% of HHS and 1.3% of HG patients had an HbA1c <7% at admission. Hypernatraemia and hyperchloraemia were more prevalent in the HDKA group than in the other groups. Hypochloraemia was the dominant serum chloride abnormality in NHDKA. Acidosis was documented in all groups. The proportions of admissions with severe acidosis were comparably high in both ketoacidotic groups; 97 (53.6%) in NHDKA versus 24 (54.2%) in HDKA.

Mortality was high in all groups (Table I), and was significantly higher in patients with HDKA (RR 3.88, 95% confidence interval (CI) 1.41 - 10.67, p=0.009), HHS (RR 2.91, 95% CI 1.09 - 7.75, p=0.033) and HG (RR 1.56, 95% CI 0.75 - 3.21, p=0.236) than in those with NHDKA (Table IV).

There were similar proportions of patients with type 1, type 2 and new diabetes in the NHDKA group, while the vast majority of HDKA admissions were patients with new diabetes (Table I). Taking the NHDKA group as a baseline risk group, patients with HDKA and HHS had a significantly higher risk of new diabetes (Table IV).

An altered level of consciousness was commonest in the HDKA group, followed by HHS, NHDKA and lastly HG. The rates of altered level of consciousness were similarly high in the HDKA and HHS groups (p=0.714, but higher in both hyperosmolar groups than in the NHDKA and HG groups (p=0.001 for HDKA v. NHDKA, p=0.007 for HHS v. NHDKA, and p=0.000 for HDKA v. HG and HHS v. HG, respectively). The NHDKA and HG groups had similar rates of altered level of consciousness (p=0.146). Similar to the above analyses, patients with HDKA and HHS had a significantly higher risk of altered level of consciousness.

Discussion

This study found unacceptably high mortality rates across all groups, higher representation of newly diagnosed diabetes in the HDKA group than in the other groups, and markedly elevated admission HbA_{1c} levels in all groups.

The overall mortality rate of 20.2% (deaths per all admissions) in the study, with a range from 13.4% for NHDKA to 37.5% for HDKA, is higher than the rates of 2.7 - 7.7% for NHDKA and 0 - 9.6% for HDKA from centres[6,7,11] that admit patients with hyperglycaemic crisis into high-care units. Patients with hyperglycaemic emergencies in Jamaica,[13] also managed in the medical wards, had lower mortality rates than ours (6.7%, 25% and 20.3% for NHDKA, HDKA and HHS, respectively), indicating that additional factors contribute to our high mortality rates. Although the higher mortality rate for HDKA than

Table I. Demographic characteristics, presenting clinical characteristics and outcomes of various types of hyperglycaemic crisis

	NHDKA	HDKA	HHS	HG	*p*-value
Age (yrs)	33(24 - 48)[‡¶]	24(19 - 44)[‡¶]	65(54 - 74)[*†]	58(39 - 68)[*†]	<0.0001
Age >40 yrs[§]	46.4%	41.7%	100%	84.9%	<0.0001
Ages of patients with new diabetes (yrs)[§]					<0.0001
<25	11.1%	46.7%	0%	4.8%	
25 - 39	55.6%	26.7%	0%	14.3%	
≥40	33.3%	26.7%	100%	81%	
	(27)	(15)	(29)	(21)	
Females[§]	61.9%	87.5%	41.4%	63.9%	0.007
	(97)	(24)	(29)	(119)	
Type of diabetes[§]					
Type 1	34%	8.3%	0%	10.1%	<0.0001
Type 2	38.1%	29.2%	55.2%	72.3%	
New diabetes	27.8%	62.5%	44.8%	17.6%	
	(97)	(24)	(29)	(119)	
Precipitating factor present	36.1%	37.5%	44.8%	41.2%	0.80
	(97)	(24)	(29)	(119)	
Unconsciousness[§]	37.3%	77.3%	72.2%	27.6%	<0.0001
	(75)	(22)	(18)	(76)	
Duration of admission (d)	7 (5 - 10)	9 (5 - 12)	6 (4 - 11)	7 (4 - 11)	0.57
	(97)	(24)	(29)	(119)	
Mortality rate[§]	13.4%	37.5%	31%	19.5%	0.02
	(97)	(24)	(29)	(118)	

Data are median (interquartile range) or percentages (number of admissions). Numbers in brackets after the percentages are the number of admissions with data available for variable of interest.

Non-parametric post-hoc tests: [*]$p<0.05$ v. NHDKA; [†]$p<0.05$ v. HDKA; [‡]$p<0.05$ v. HHS; [¶]$p<0.05$ v. HG; [§]$p<0.05$ for χ^2 test. χ^2 test for categorical variables and Kruskal-Wallis test for continuous variables.

NHDKA = non-hyperosmolar diabetic ketoacidosis; HDKA = hyperosmolar diabetic ketoacidosis; HHS = hyperosmolar hyperglycaemic state; HG = hyperglycaemia.

NHDKA agrees with other reports,[6,7,11,13] the former had a similar mortality rate to HHS despite the patients being more than 20 years younger. This underscores the need to distinguish between these forms of ketoacidosis, as the combination of hyperosmolality and ketoacidosis had a worse prognosis than ketoacidosis alone despite both ketoacidotic groups being relatively young. A tendency to longer duration of admission in HDKA than NHDKA has been reported.[11] We are unable to provide an explanation for our patient groups having comparable periods of hospital stay.

Admissions for HDKA, in contrast to NHDKA, were likely to be of patients with a new diagnosis of diabetes. Although hyperosmolality in HHS is explained by an age-related increase in the renal threshold for glucose and reduced sensitivity of the thirst centre, it is not clear what the underlying mechanism is in our patients with HDKA, who were relatively young (41.7% were aged >40 years, compared with 100% for HHS) and had new-onset diabetes. We do not know whether they had an elevated renal threshold for glucose or an increased thirst threshold. It is also possible that as HDKA admissions were mainly of newly diagnosed diabetes subjects, who were not aware of their diabetes status, they may have presented relatively late or not taken adequate fluids to militate against ongoing renal losses (which an informed diabetic patient would have known it was necessary to do). This may explain why hypernatraemia and an altered level of consciousness were more prevalent in HDKA than NHDKA admissions. However, we have no data on the duration of symptoms or severity of polydipsia before presentation. Admissions for HHS and HG with a serum bicarbonate level below 18 mmol/l were as categorised, as ketosis was absent at presentation The factors probably contributing to low serum bicarbonate in these ketone-negative patients include uraemic and lactic acidosis, although lactate was not routinely measured in our patients.

While longitudinal studies,[3,21,22] particularly in populations of black African ancestry, have shown that most patients with ketoacidosis as the first manifestation of diabetes have features of type 2 diabetes, we were unable to adequately characterise the newly diagnosed diabetic patients with NHDKA or HDKA as having type 1 or type 2 diabetes. Few patients had information on a family history of diabetes, a personal or family history of auto-immune diseases,

Table II. Profiles of patients with repeated admissions for hyperglycaemic crisis

Patient No.	Gender	Age (yrs)	Diabetes type	No. of admissions	Types of hyperglycaemic crisis	Outcome
1	F	23	1	7	5 times for NHDKA and 2 times for HG	Died at last admission
2	F	51	2	3	2 times for HG and once for NHDKA	Survived all admissions
3	F	51	2	2	2 times for HDKA	Survived all admissions
4	F	16	1	4	4 times for NHDKA	Survived all admissions
5	F	69	2	3	3 times for HG	Survived all admissions
6	M	85	2	5	3 times for HG and once each for NHDKA and HDKA	Survived all admissions
7	F	13	1	2	Once each for NHDKA and HG	Survived all admissions
8	F	31	1	2	Once each for NHDKA and HG	Survived all admissions
9	M	32	1	3	3 times for NHDKA	Survived all admissions
10	F	65	2	2	2 times for HG	Survived all admissions

NHDKA = non-hyperosmolar diabetic ketoacidosis; HDKA = hyperosmolar diabetic ketoacidosis; HG = hyperglycaemia.

weight, height, waist circumference, presence of acanthosis nigricans or C-peptide levels. Laboratory markers of islet auto-immunity were not performed as this test was not available.

The very high HbA_{1c} level at presentation in all groups is notable. Although admissions for HDKA were predominantly in persons newly diagnosed with diabetes, all 15 admissions for HDKA, including 8 in newly diagnosed diabetic patients for whom data on HbA_{1c} level were available, had HbA1c levels above 10%. The finding that an HbA_{1c} <7% was only noted in 2 of 174 admissions (including 56 patients with newly diagnosed diabetes) with HbA_{1c} levels indicates that hyperglycaemic crisis in our setting is preceded by chronic hyperglycaemia regardless of whether the patient is known to have diabetes or newly diagnosed. Indeed an elevated HbA_{1c} level has been reported to be associated with unprovoked ketoacidosis, and the suggested mechanism is glucotoxicity to the beta cell.[23] Universal screening for diabetes is not recommended, but improving glycaemic control in our patients already diagnosed with diabetes will reduce the occurrence of hyperglycaemic crisis.

Public health measures to reduce deaths from hyperglycaemic crisis are urgently needed. This includes educating the general population on diabetes prevention through a healthy lifestyle and highlighting its common presenting symptoms. Impediments to achieving good glycaemic control in diabetic patients, particularly at referring health care facilities, must be addressed as chronic hyperglycaemia preceded most admissions for hyperglycaemic crisis. A follow-up to a study from Soweto[23] reporting high mortality in hyperglycaemic crisis described significantly reduced deaths following improved education and care of patients, particularly those who were most prone to hyperglycaemic decompensation.[24]

We plan to review outcomes for hyperglycaemic crisis admissions in order to assess the impact of our recently established high-care unit and accurately ascertain the type of diabetes in our new-onset diabetes patients with hyperglycaemic crisis

We acknowledge the assistance of the medical wards clerk, Miss Q Duba, in the retrieval of patient case records.

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Table III. Presenting serum biochemical measurements in various types of hyperglycaemic crisis

	NHDKA	HDKA	HHS	HG	*p*-value
Blood glucose (mmol/l)[§]	31.1 (24.1 - 38)[‡]	37.8 (22.4 - 57.1)[‡]	58.6 (41.2 - 76.4)[*†¶]	27.1 (18 - 33.8)[‡]	
	(97)	(24)	(29)	(119)	<0.0001
Blood glucose >33.3 mmol/l[§]	34%	50%	79.3%	25.2%	<0.0001
	(97)	(24)	(29)	(119)	
HbA_{1c} (%)	12.7 (10.5 - 15.6)	12.5 (11.6 - 15.8)	13 (11.6 - 15.2)	12.2 (10.4 - 14.6)	0.50
	(62)	(15)	(17)	(80)	
Hypernatraemia[§]	0%	66.7%	20.7%	0%	<0.0001
	(97)	(24)	(29)	(119)	
Hypokalaemia	10.6%	17.4%	20.7%	11.9%	0.47
	(94)	(23)	(29)	(118)	
Hyperkalaemia[§]	22.3%	13%	24.1%	9.3%	0.04
	(94)	(23)	(29)	(118)	
Hypochloraemia[§]	45.4%	8.3%	10.7%	41.5%	<0.0001
	(97)	(24)	(28)	(118)	
Hyperchloraemia[§]	13.4%	83.3%	50%	26.7%	<0.0001
	(97)	(24)	(28)	(75)	
Acidosis[§]	100%	100%	31%	9.2%	<0.0001
	(97)	(24)	(29)	(119)	

Data are median (interquartile range) or percentages (number of admissions). Numbers in brackets after the percentages are the number of admissions with data available for variable of interest.

Non-parametric post-hoc tests: [*]$p<0.05$ v. NHDKA; [†]$p<0.05$ v. HDKA; [‡]$p<0.05$ v. HHS; [¶]$p<0.05$ v. HG; [§]$p<0.05$ for χ^2 test. χ^2 test for categorical variables and Kruskal-Wallis test for continuous variables.

NHDKA = non-hyperosmolar diabetic ketoacidosis; HDKA = hyperosmolar diabetic ketoacidosis; HHS = hyperosmolar hyperglycaemic state; HG = hyperglycaemia.

Table IV. Logistic regression analysis of risk of death, new diabetes and altered level of consciousness at presentation in various groups of hyperglycaemic crisis

Group	Death		New diabetes		Altered level of consciousness	
	RR (95% CI)	*p*-value*	RR (95% CI)	*p*-value*	RR (95% CI)	*p*-value*
HDKA	3.88 (1.41 - 10.67)	0.009	4.32 (1.69 - 11.04)	0.002	5.71 (1.90 - 17.17)	0.002
HHS	2.91 (1.09 - 7.75)	0.033	2.11 (0.90 - 4.96)	0.088	4.36 (1.40 - 13.55)	0.011
HG	1.56 (0.75 - 3.21)	0.236	0.56 (0.29 - 1.06)	0.075	0.60 (0.30 - 1.20)	0.148
NHDKA	1		1		1	

* Wald test, NHDKA = baseline risk group.

NHDKA = non-hyperosmolar diabetic ketoacidosis; HDKA = hyperosmolar diabetic ketoacidosis; HHS = hyperosmolar hyperglycaemic state; HG = hyperglycaemia.

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Accepted 3 August 2010.