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Original Research Paper

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A STUDY OF CLINICAL AND BIOCHEMICAL PROFILE OF PATIENTS OF DIABETIC KETOACIDOSIS

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ABSTRACT

OBJECTIVES: To study the clinical and biochemical profile of patients of diabetic ketoacidosis admitted. To compare various parameters of clinical and biochemical profile before and after treatment.

METHODS: 30 adult patients with diabetic ketoacidosis admitted in medicine department of Geetanjali medical college and hospital, meeting the inclusion criteria, over a period of 1 years (Jan 2018 to Dec 2018). Patients fulfilling diagnostic criteria of DKA and inclusion criteria were enrolled. Diagnostic criteria are plasma glucose >250 mg/dl, blood pH <7.30, serum bicarbonate <15 mEq/l, urine ketones positive.

RESULTS: 24 patients had type 2 diabetes (80%) and 6 (20%) patients had type 1 diabetes. Average age at the time of presentation was 44.20 ± 13.17 years. The commonest precipitating factor was infection (83.33%) followed by irregular or no treatment (40%) and other factors (16.67%). The most common clinical features at the time of presentation were breathlessness, vomiting, fever, abdominal pain, dehydration and acidotic breathing. The mean values for RBS, pH, HCO3 were 449.45 ± 71.45, 7.19 ± 0.10, and 10.42 ± 3.48 respectively at the time of presentation. There was no significant difference in clinical and biochemical profile of patients with type 1 and type 2 diabetes. Mortality rate was 3.33%.

CONCLUSION: Most common clinical features at the time of presentation are breathlessness, vomiting, fever, abdominal pain, dehydration and acidotic breathing. There is no significant difference in the clinical and biochemical profile of patients with type 1 and type 2 diabetes. Mortality rate in diabetic ketoacidosis is 3.33% and the most notable predictors of poor prognosis are; severity of altered sensorium, severity of comorbid condition, severe dehydration, severe acidosis.

KEYWORDS : Diabetic Ketoacidosis; Mortality; Predictors; Precipitating Factors.

INTRODUCTION

An absolute or relative lack of Insulin results in genesis of Diabetes Mellitus. Diabetes results in abnormal levels of glucose in the bloodstream. [1] Diabetes mellitus (DM) refers to a group of common metabolic disorders that share the phenotype of hyperglycaemia. Depending on the etiology of the DM, factors contributing to hyperglycaemia include reduced insulin secretion, decreased glucose utilization, and increased glucose production. The metabolic dysregulation associated with DM causes secondary pathophysiologic changes in multiple organ systems that impose a tremendous burden on the individual with diabetes and on the health care system.[2]

Diabetic ketoacidosis is reported to be responsible for more than 100 000 hospital admissions per year in the US, and accounts for 4-9% of all hospital discharge summaries among patients with diabetes[3]. Around 10% of patients presenting with ketoacidosis in India (20% in the western countries) are freshly diagnosed to have diabetes[4]. The patient may present with wide range of manifestations like ketosis, ketoacidosis, pre-coma and coma but often these manifestations are submerged in the clinical presentation of precipitating illness.[5]

Majority of the patients presenting with diabetic ketoacidosis are known diabetics on treatment and the commonest precipitating factors are infections (sepsis) and omission of insulin.[6] The commonest presenting complaints include nausea, vomiting, polyuria, polydipsia and main clinical findings include dehydration, acidotic respiration and confusion or coma.7

This study will help to understand natural history of disease, prevalence among males and females and its precipitating factors and hence increase awareness to avoid occurrence of the dreadful complication of the disease i.e. diabetic ketoacidosis.

OBJECTIVES

1. To know the distribution and drug history in known cases of diabetes mellitus and newly diagnosed diabetics.
2. To know the dose and duration of insulin required to clear urine ketone bodies and correlation with biochemical tests.

METHODS

All the patients of diabetes suffering from DKA and admitted in medicine department and who follows following selection criteria were enrolled in the study.

INCLUSION CRITERIA:

1. Age >18 years & <80 years
2. Diagnosed or freshly detected case of either type 1 or 2 diabetes.
3. Patient diagnosed to be having diabetic ketoacidosis.
4. Patients primarily admitted for other disease but having accidental detection of diabetic ketoacidosis.
5. Patient with pre-existing co-morbid conditions.

EXCLUSION CRITERIA:

1. Age <18 years & >80 years
2. Patients diagnosed to have hyperosmolar nonketotic acidosis.

RESULTS

Out of 30 cases of diabetic ketoacidosis, 24 (80%) were having type 2 diabetes mellitus and 6 (20%) were having type 1 diabetes mellitus. Out of 30 patients, 16 were male (53.33%) and 14 were female (46.67%). Maximum number of cases 14 (46.7%) observed were in the age group 41- 50 years. Minimum number of cases 1 (3.3%) were in the age group 11-

20, 31-40, 71-80 years. Majority of patients are in the age group 41-60, that is 21 patients (70%).

In this study, maximum number of patients i.e., 11 (36.7%) were diabetic for 0-1 years. Patients with diabetes more than 10 years were 4 (13.3%). Mean duration is 5.71 ± 6.6. Out of 30 patients, 21 (69%) had a family history of diabetes. Amongst 6 cases of type 1 DM, 2 (6.66%) gave family history of diabetes. Amongst 24 case of type 2 DM, 19 (63.27%) gave family history of diabetes. Maximum number of patients were on OHAs alone 10 (33.3%), 4 (13.3%) were on insulin therapy alone, 9 (30%) patients were on both insulin and OHAs and 7 (23.3%) were not on any medications as they were newly diagnosed diabetics after their hospital admission.

Table 3: Precipitating Factors in diabetic ketoacidosis

Precipitating Factors	Type of Diabetes		Total (n=30)
	Type-1	Type-2	
Treatment related:	5(41.67%)	7(58.33%)	12(40%)
• Irregular	2(40%)	3(60%)	5(16.67%)
• No treatment	3(42.86%)	4(57.14%)	7(23.33%)
Infection:	2(8%)	23(92%)	25(83.33%)
• UTI	1(11.11%)	8(88.89%)	9(30%)
• Septicemia	1(14.29%)	6(85.71%)	7(23.33%)
• Septic Shock	0(0%)	1(100%)	1(3.33%)
• Respiratory tract infection	0(0%)	4(100%)	4(13.33%)
• Acute gastro enteritis	0(0%)	2(100%)	2(6.67%)
• Hepatitis	0(0%)	1(100%)	1(3.33%)
• Necrotizing Fasciitis	0(0%)	1(100%)	1(3.33%)
Others:	0(0%)	5(100%)	5(16.67%)
• Myocardial Infarction	0(0%)	2(100%)	2(6.67%)
• Cardiogenic shock	0(0%)	1(100%)	1(3.33%)
• Cerebrovascular accident (CVA)	0(0%)	1(100%)	1(3.33%)
• Surgery	0(0%)	1(100%)	1(3.33%)

Table 4: Clinical profile in diabetic ketoacidosis before and after treatment

	Pre (N=30)	Post (N=29)
Breathlessness	20(66.7%)	3(10.35%)
Vomiting	20(66.7%)	1(3.45%)
Abdominal pain	9 (30.0%)	1(3.45%)
Fever	15(50.0%)	0(0%)
Dehydration	23(76.7%)	0(0%)
Mental status: Conscious Drowsy Stupor Coma	 18(60%) 10(33.3%) 1(3.33%) 1(3.33%)	29(100%) 0(0%) 0(0%) 0(0%)
Acidotic breathing	14(46.7%)	0(0%)

Out of 30 patients, 20 (66.7%) were having vomiting, 15 (50%) with fever and 9 (30%) with abdominal pain. 23 (76.7%) were dehydrated and 20(66.7%) were breathless at the time of admission out of which 14(46.7%) were having acidotic breathing. 18(60%) patients were conscious, 10(33.3%) patients were drowsy and 1(3.33%) patient was stuporous, 1(3.33%) was in coma state at the time of admission. Conscious level of all patient who survived i.e., 29 patients improved with treatment. After treatment, breathlessness present in 3(10.35%), abdominal pain in 1(3.33%), and vomiting in 1 (3.33%) patient.

On admission, out of 30 cases, 17(56.7%) patients were having 4+, 11(36.7%) were having 3+ and 2(6.7%) were having 2+ urine ketone bodies. Urine ketone bodies were completely washed out i.e. nil in all survived patients and were positive in expired patient at the time of death.

In my study group having maximum number of patients i.e. 10 (33.3%) required insulin dose between 26-50units for clearance of urine ketone bodies.

Only 1 patient required <25 units for clearance of urine ketone bodies and 4 (13.3%) patients required >100 units to clear urine ketone bodies.

Majority of patients i.e. 12 (40%) in this study group required 13-24 hours for the clearance of urine ketone bodies from the start of insulin therapy. 4 (13.3%) patients required <12 hr and 1 (3.3%) 73-84 hours. In this study we observed that the patient who died, was comatose at the time of presentation, had septicaemic shock, type 2 diabetic, severely dehydrated, duration of insulin therapy given was 12 hours and dose of insulin given was 65 IU.

Before urine ketone bodies was cleared out of the system patient could not be recovered from shock state and hence died. RBS was very high 500 at time of presentation. She was in severe acidosis pH 7.15 and HCO3 was 4.7.

In this study, there was a difference in the biochemical parameters between the patients who survived and expired; but before patient could receive insulin treatment sufficient to make difference in requirement and duration of insulin, she could not be recovered from septic shock.

DISCUSSION

In present study, diabetic ketoacidosis was more common in Type 2 diabetes mellitus (80%) than Type 1 diabetes mellitus (20%). In a study by Rao et al, 22(81%) had type2 diabetes and 5(19%) had type 1 DM.[8] The duration of diabetes in our patients varied from 15 days to 30 years with mean of 5.71 ± 6.6. Maximum number of cases i.e. 11 (36.7%) were in the age group 0-1 year. In a study by Newton et al the duration of diabetes was 18.3 ± 14.4 years.[9] In our study, the commonest precipitating factor was infection seen in 83.33% of patients. Amongst infections urinary tract infection was the commonest cause seen in 30% followed by septicemia in 23.33% patients. In this study, Rao et al found infection (43%) as commonest precipitating factor. Among infection, urinary tract infections were most common, followed by septicemia and pneumonia.[8]

Newton et al reported infection as the leading precipitating factor in 41%.[10] In our study the most common symptoms were vomiting (66.7%) and breathlessness (66.7%). Abdominal pain and fever were present in 9% and 15% of patients respectively. The most commonly found signs were dehydration (76.7%), and acidotic breathing (46.7%). Altered sensorium was found in 39.99% of patients.Rao et al observed the clinical features in his study were as follows: vomiting (40%), breathlessness (36%), fever (28%) and abdominal pain (24%).[8] In our study RBS values ranged from 274-500mg/dl with mean 449.45 ±71.45. pH ranged from 6.88 – 7.32 with mean 7.19 ± 0.1 Bicarbonate ranged from 4 – 17 with mean 10.42 ± 3.48. There was no significant difference found in the biochemical profile of type 1 and type 2 DM patients. The patient who expired had severe acidosis and all parameters fell in to the severe diabetic ketoacidosis criteria. Rao et al showed the similar findings where there were no significant differences in type 1 and type 2 diabetes developing diabetic ketoacidosis.[8]

In this study, we found that majority of patients required insulin dose between 26-50 units i.e. 10 (33.3%) patients, followed by 51-75 units i.e. 8 (26.7%) patients and 76-100 units i.e. 6 (20%) patients, for clearing urine ketone bodies. Insulin was given for 13-24 hours as infusion in majority of patients i.e., 12 (40%) patients to clear urine ketone bodies. Before we could administer full dose of insulin required to recover from diabetic ketoacidosis and to wash out ketone bodies, patient

expired due to septicaemic shock. 65 unit of insulin was given over period of 12 hours.

A study by Stamatis P reported that higher the dose of insulin required and more the duration of insulin therapy, as intensive infusion therapy, to clear urine ketone bodies; worse is the prognosis.[9]

In this study we found that poor prognostic factors were severity of acidosis, severity of altered sensorium, severity of dehydration, high RBS at the time of presentation and comorbid conditions. Patient who expired was in septicemic shock. Another study by Pinnies JA reported that septic shock was the most frequent cause of death (31%) and poor prognostic indicators were older age, hypotension, low Na, pH and HCO3, and high urea.[11]

CONCLUSION

There is no significant difference in the clinical and bioche mical profile of patients with type 1 and type 2 DM devel oping ketoacidosis. Some of the clinical and biochemical parameters may indicate bad prognosis; most notably, severity of altered sensorium, severity of comorbid condition, severe dehydration, severe acidosis, doses and duration of insulin required for clearing urine ketones.

REFERENCES

1. Samreen Riaz: Diabetes mellitus. Scientific Research and Essay Vol. 4 (5) pp. 367-373, May, 2009.
2. Powers AC. Diabetes Mellitus. In: Longo DL, Kasper DL, Jamson JL, Fauci AS, Hauser SL, Loscalzo J, editors. Harrison's Principles of Internal Medicine. 18th Edition, United States: McGraw-Hill; 2012.
3. Umpierrez GE, Kitabchi AE. Treat Endocrinol. Diabetic ketoacidosis: risk factors and management serategies. 2003;2(2):95-108.
5. Akhtar J, Jabbar A, Islam N, Khan MA. Prevalence and clinical profile in diabetic ketoacidosis. J Pak Med Assoc 1993 Jul; 43(7):137-9.
4. V. Seshiah. Acute complications of diabetes mellitus. In: Shah SN, editor. API Text Book of Medicine. 7th Edition, India: API; 2003.
6. Matoo VK, Nalini K, Dash RJ, et al. Diabetic ketoacidosis – A clinical study. JAPI 1991 May; 39(5):379-81.
7. Rajsoorya C, Wong SF, Chew LS. Clinical and biochemical profile in diabetic ketoacidosis. Singapore Med J 1993 Oct; 34(5):381-4.
8. D Rao V, B Pradhan, Y Mallikarjuna, R Reddy. Clinical profile of diabetic ketoacidosis in adults. Health Renaissance 2012; Vol 10 (No.2); 80-86
9. Stamatis P, Efstathiou, Aphrodite G, Tsioulos, Loannis et al. A mortality prediction model in diabetic ketoacidosis. Clin Endocrinol 2002; 57:595- 601.
10. Newton CA, Raskin P. Diabetic Ketoacidosis in Type 1 and Type 2 Diabetes Mellitus: Clinical and Biochemical Differences. Arch Intern Med. 2004;164(17):1925-1931. doi:10.1001/archinte.164.17.1925.
11. Pinnies JA, Cairo G, Gaztambide S, Vazquez JA. Course and prognosis of 132 patients with diabetic ketotic and non-ketotic hyperosmolar state. Diabetes Metab 1994 Jan-Feb; 20(1):43-8