ORIGINAL RESEARCH PAPER

Volume-8 | Issue-4 | April-2019 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A STUDY OF BIOCHEMICAL PROFILE AND OUTCOME IN PATIENTS OF DIABETIC KETOACIDOSIS ADMITTED IN SHETH LG HOSPITAL

Medicine

Dr Sangita Rathod Head of Unit, Associate Professor, Department of Medicine, AMC Met Medical College, LG Hospital, Maninagar, Ahmedabad

Dr Bhavesh Gurjar 2nd Year Resident, Department of Medicine, AMC Met Medical College, LG Hospital, Maninagar, Ahmedabad

Dr Poojan Parmar* 1st Year Resident, Department of Medicine, AMC Met Medical College, LG Hospital, Maninagar, Ahmedabad *Corresponding Author

ABSTRACT

BACKGROUND : DKA is a well known and major acute metabolic complication seen in diabetic pt (type 1 as well as type 2), if not recognized in initial stage it may turn into life threatening condition, characterized by hyperglycemia,acidosis and ketonuria , DKA can be inaugural or complicate known diabetes.

Objective:THIS STUDY WAS PERFORMED TO EVALUATE BIOCHEMICAL PROFILE OF DKA PATIENT ADMITTED IN SHETH LG HOSPITAL

DESIGN: The study was a cross sectional study conducted over a period of 6 months in AMC MET medical college,Ahmedabad.

MATERIALS AND METHODS: Clinical profile of 50 diabetic pt admitted in emergency with the diagnosis of DKA were analysed.

RESULTS: Most common age group: 45-60 year

Gender: male

Mode of treatment: oral hypoglycemic agents>insulin

Mean duration of diabetes: 6-10year

Mean HB: 8-10 gm/dl

Mean RBS: 400-550

Mean HBA1C:6-10%

Mean ph: 7.00-7.24

Mean Na+: 135-145

Mean K+:3.5-5.5

Mean Creatinine: >1.0

Mean Blood urea: >18

Most common symptoms: nausea and vomiting > abdominal pain.

Out of 50 patients 49 were discharged with proper guidance of treatment of DM and followup in medical opd and 1 pt was succumbed to death .

KEYWORDS

INTRODUCTION:

Diabetic ketoacidosis is serious acute metabolic complication in diabetic pt, characterized by absolute or relative insulin deficiency with an overall mortality up to 5%.(1) Main pathophsiology being insulin deficiency , increased counter regulatory hormones like glucagon and steroids and peripheral insulin resistance leading to hyperglycemia. DKA is seen in type 2 as well in type 1 diabetic with increased prevalence when it is imparted by severe insulin resistance in conditions like hypertension, obesity, acute myocardial infarction, septicemia, tuberculosis etc.. Type 2 diabetics have longer hospital stay and mortality when compared to type 1 due regards to the associated co-morbidities seen in DM-2. (2) The incidence of DKA depends on age, sex, duration of diabetes and presence of other co morbidities. DKA is also found to be the initial presentation of diabetes or precipitated by many factors like noncompliance to treatment , acute stress conditions and most common being infection.(3)

Together with hyperglycemic coma, diabetic ketoacidosis (DKA) is the most severe acute metabolic complication of diabetes mellitus DM(1).DKA is defined by the triad hyperglycemia, acidosis, and ketonuria. DKA can be inaugural or complicate known diabetes.(2). Although DKA is evidence of poor metabolic control and usually indicates an absolute or relative imbalance between the patient's requirements and the treatment, DKA-related mortality is low among patients who receive standardized treatment, which includes administration of insulin, correction of hydroelectrolytic disorders, and management of the triggering factors (which are often cessation of insulin therapy, an infection, or a myocardial infarction).In general, DKA is always described to be closely linked to type 1 DM. The occurrence of DKA has been thought to indicate the underlying significant and irreversible ß-cell damage that classifies these diabetic patients as type 1 DM . However, many DKA patients do have clinical course and metabolic features of type 2 DM also. Recent epidemiologic studies estimate that hospitalizations for DKA have increased during the past 2 decades.(5) Part of this increased frequency of admissions may be related to the increased prevalence of type 2 diabetes. With the changes in the frequency of DKA and the increased incidence of DKA in patients with type 2 diabetes mellitus, the question may be posed of whether there has been any change in the clinical or laboratory characteristics of the patients with DKA who present to the emergency department. In India, there have been few investigations focusing on the type 2 diabetic patients who suffered from DKA episodes. We therefore undertook to examine the clinical and lab characteristics of diabetic patients who presented with DKA to our hospital.

This present study is performed to assess the biochemical profile and outcome of DKA in diabetic patients.

MATERIALS AND METHODS:

A cross sectional study was done including 50 diabetic pt with DKA admitted in Department of medicine, AMC MET MEDICAL COLLEGE, LG HOSPITAL, MANINAGAR, AHMEDABAD.

INCLUSION CRITERIA:

All patients of type1/2 diabetes with DKA fulfilling the diagnostic criteria as following taken for study:

-plasma glucose of >250 mg/dl

-a serum bicarbonate level of <15

-arterial blood ph <7.35

-all pt are investigated with urine analysis, blood urea, serum creatinine levels, plasma glucose, HBA1C, ABGA, s.electrolytes.

& Age (>18yr).;

EXCLUSION CRITERIA:

Patients with hyperosmolar state and hyperglycemia without ketoacidosis.

Pregnant females.
Age <18 yr.

STATISTICAL ANALYSIS:
Data were entered in MS excel 2010; Data was presented in form of frequency and percentage.

RESULTS:
AGE AND GENDER VARIATION SEEN IN DKA PT:

AGE	18-30 YEAR	2
	31-45	18
	46-60	27
	>60	3
GENDER	MALE	28
	FEMALE	22

Hereby our study shows that most common age group is 46-60 yr(27) followed by 31-45 yr (18).

Various clinical symptoms seen in DKA pt :

CLINICAL SYMPTOMS	PERCENTAGE (%)
NAUSEA, VOMITTING	98.07
ABDOMINAL PAIN	61.53
POLYURIA,POLYDYPSIA	36.28
FEVER	34.61
WEAKNESS	30.76
ALTERED SENSORIUM	30.76
SHORTNESS OF BREATH	17.30

Hereby study shows that 98% of DKA pt has complains of nausea and vomiting > abdominal pain (61.53%),followed by polyuria and polydipsia.

Among Precipitating Factors For Dka, Non Compliance To Therapy (66%)is The Most Common One Followed By Infections (pneumonia, Uti,tb-30%) , Followed By Other Factors Such As (mi,cva-4%).
AMONG CO-MORBIDITIES IN DKA PATIENT HYPERTENSION WAS MOST COMMON (34%)>OBESITY (22%)>DIABETIC RETINOPATHY & NEPHROPATHY (16%)>CKD (9.61%)>CVD (6%).

BIOCHEMICAL INVESTIGATIONS:

METABOLIC ACIDOSIS	MILD(7.25-7.30	1
(n=50)	MODERATE(7.00-7.24	47
	SEVERE(<7.0)	2
RBS	>450mg/dl	37
(n=50)	<450 mg/dl	13
HBA1C	>9	18
(n=50)	<9	32

DISCUSSIONS:
hereby study shows that most of the patients at time of admission have RBS >450mg/dl, with HBA1c>9, with ABGA showing severe metabolic acidosis with Ph between 7.00-7.24.

VARIATIONS IN RFT AND S.ELECTROLYTES SEEN IN DKA PATIENTS:

S .NA +		S.K+		S. creatinine		Blood Urea	
<135	13%	<3.5	21%	0.8-1.0	48%	>18	80%
135-145	77%	3.5-5.5	38%	>1.0	52%	<18	20%
>145	10%	>5.5	60%				

The study shows that most of the patients of DKA had S.Na+ of 135-145 range, S. K+ of >5.5 range, S.Creat of >1.0 and urea >18 which indicates most of the patients had normal S.Na+ with hyperkalemia and altered renal function.

Correlation Of Comorbidities With Compliance To Treatment:

	Compliant Patient To Therapy	Non-compliant Patient To Therapy	Total
Co-morbidities present	9	32	41
Co- Morbidities Absent	6	3	9
Total	15	35	50

P value of above table =0.0245
HERBY WE GOT P VALUE OF 0.0245 THAT IS STATISTICALLY SIGNIFICANT (<0.058) WHICH SUGGEST THAT MORE CO-MORBIDITIES ARE MORE IN PATIENTS WHO ARE NON COMPLAINT TO TREATMENT.

There were 8 patients of type 1 and 42 patients of type 2 diabetes mellitus.

DISCUSSIONS:
In developing country like India poor socio-economic status in patients with T2DM incline to have poor compliance and poor glycemic control so any precipitating factor tends to land them in a state of DKA. Mean age of patients in present study was 50 years. Among clinical symptoms nausea and vomiting were most common, followed by abdominal pain in DKA patients .Similarly in present study altered sensorium, complaining of polyuria and polydypsia and shortness of breath was reported in 30%, 37% and 26% DKA patients respectively. Present study has revealed that DKA patients were having more than one precipitating factor like 66% who had poor compliance also had infection like pneumonia and TB. Hence, it can be said that presence of non-compliance to treatment is an important precipitating factor which indicates that prevalence of DKA can be reduced by proper education of patients about their illness and harm of non-compliance. Pneumonia was the most common (24%) infection in our study precipitating DKA. Other factors such as hyperglycemia, leukocyte dysfunction, macro vascular disease and acidosis predispose the diabetic with ketoacidosis to common and rare infections. This is agreement with the other's which showed that infection of any site is an important precipitating factor in causing DKA.(3). The possible reason for such a high mortality rates in Indian patients may be due to delayed presentation and poor socioeconomic conditions which influence the selection of better antibiotics for the treatment. An underlying deficiency of insulin, with elevation in counter-regulatory hormones, is found in DKA. There are three proposed mechanisms of DKA in type 2 diabetics: 1) Insulinopenia, 2) Elevation of counter regulatory stress hormones, and 3) Increase in free fatty acids(1). Some authors/studies believe that only the first one of these, insulinopenia, is significant in type 2 diabetics.The stressors precipitating DKA have been postulated to cause a relative rather than a definitive deficiency of insulin. Possible causes for this relative insulin deficiency include impairment of insulin secretion due to chronic exposure of insulin secreting or islet cells to high levels of glucose of free fatty acids.6Additional causes are hypokalemia, which can impair insulin secretion, and prolonged fasting, which increases the rate of ketosis, and may decrease insulin secretion. Although elevation of counter-regulatory stress hormones and free fatty acids are postulated mechanisms We did not measure the C-peptide levels and therefore we can only hypothesize about the possible role of insulinopenia in the development of DKA in our patients. However, it is hard to ignore the possible role of counter regulatory hormones in these patients as 66% of these patients were having a stressful event prior to development of DKA. Increased secretion of glucagon (as well as other counteregulatory hormones such as cortisol, catecholamines, and growth hormone) in response to stress from 1) overwhelming infection 2) infarction of tissue 3) other severe illness further suppress insulin secretion to perpetuate a downward spiral. In our study of adult DKA patients, the mean age was 45.3 years, with insignificant male predominance for both types of diabetes. However, type 2 diabetic patients were significantly older than that of type 1. There is a common perception that significant\physical stress (other than the effects of persistent hyperglycemia per se) is a prerequisite for patients with type 2 diabetes to develop DKA. An adequate insulin secretory response is restored in these patients by aggressive management of diabetes. Infection is the major precipitating factor, occurring in 43% of our patients, with urinary tract infections and pneumonia being the most common infections. Similar reports have been described by other investigators also.(2,3)We found that in many instances, an acute illness, such as cerebro-vascular accident or myocardial infarction, has precipitated the DKA by the release of counter regulatory hormones. Therefore, DKA should be considered in patients with diabetes who have a concurrent infection, stroke, myocardial infarction, or other serious illness. These intercurrent illnesses should be sought and treated aggressively. Omission of insulin therapy for a variety of reasons has also been shown to be the leading precipitating cause of DKA.2 In our study also nearly thirty five per cent patients discontinued treatment prior to development of DKA. Many of our “previously diagnosed” type 2 patients had no obvious precipitating cause other than prolonged omission of oral hypoglycemic therapy.

The rate of insulin discontinuation and a history of poor compliance accounts for more than half of DKA admissions in rural and minority populations(6).Several cultural and socioeconomic barriers, such as low literacy rate, limited financial resources, and limited access to health care, in medically indigent patients may explain the lack of compliance and why DKA continues to occur in such high rates in rural patients. Although symptoms of nausea, emesis, and abdominal pain are related to the presence of ketonemia. It was observed that symptoms of osmotic diuresis did not bring people to medical attention, but nausea, vomiting, and abdominal pain do.

CONCLUSION:

There is need of taking active measures to rule out DKA in DM and comatose patients in order to prevent further complications and mortality. Mortality mainly depends on the general condition of the patient, as well as the coexistent medical illness and time of onset of therapy as well as compliance to therapy. Hence education regarding life style modification, diet and treatment adherence must be done in each and every patient of DM.

REFERENCES:

1- HARRISON'S PRINCIPLES OF INTERNAL MEDICINE 20TH EDITION & Gosmanov AR, Gosmanova EO, Dillard-Cannon E. Management of adult diabetic ketoacidosis. Diabetes Metabolic Syndrome. Obes Targ Ther 2014; 7:255–64.
2. Xu Y, Bai J, Wang G, Zhong S, Su X, Huang Z et al. Clinical profile of diabetic ketoacidosis in tertiary hospitals in China: a multicentre, clinic-based study. Diabet Med: J Br Diabet Assoc 2016;33:261–8.
3. Wang Y, Desai M, Ryan PB, DeFalco FJ, Schuemie MJ, Stang PE et al. Incidence of diabetic ketoacidosis among patientswith type 2 diabetes mellitus treated with SGLT2inhibitors and other antihyperglycemic agents. diabetes research and clinical practice 2017; 128: 83–90.
4. Kitabchi AE, Umpierez GE, Miles JM, Fisher JN. Diabetes care 2009; 32:1335-43.
5. Barski L, Nevzorov R, Harman-Boehm I, Jotkowitz A, Rabaev E, Zektser M, et al. Comparison of diabetic ketoacidosis in patients with type-1 and type-2 diabetes mellitus. Am J Med Sci. 2013;345:326-30.
6. Wilson JF. Diabetic Ketoacidosis- The Clinics. Ann Int Med. 2010;152:ITC 1-1.
7. Seth P, Kaur2 H, Kaur M. Clinical Profile of Diabetic Ketoacidosis: A Prospective Study in a Tertiary Care Hospital. Journal of Clinical and Diagnostic Research 2015; 9: OC01-4. 1
8. Kitabchi AE, Umpierrez GE, Murphy MB, KreisbergRA.Hyperglycemic crises in adult patients with diabetes: a consensus statement from the American DiabetesAssociation. Diabetes Care 2006;29:2739–48.
9. Adhikari PM, Mohammed N, Pereira P. Changing profile of diabetic ketosis. J Indian Med Assoc. 1997;95: 540-42.
10. National Center for Health Statistics. National hospital discharge and ambulatory surgery data. [article online]. Available from http://www.cdc.gov/nchs/about/major/hdasd/nhds.htm. Accessed 9 March 2018.