Original

CLINICAL AND BIOCHEMICAL ASPECTS OF DIABETIC KETOACIDOSIS AND IT'S OUTCOME A 5-YEAR EXPERIENCE IN A SAUDI TEACHING HOSPITAL

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ABSTRACT

الهدف هو دراسة المظاهر الإكلينيكية و الكيميائية للحماض الكيتوني السكري الحاد و طريقة علاجه و نتائج العلاج في مستشفى جامعة الملك عبد العزيز بجدة و مقارنتها بالدراسات التي أجريت قبل عشر سنوات. . تم دراسة ملفات المرضى الذين تم تنويمهم في المستشفى في الفترة ما بين يناير 1994 م إلى 1998 م بسبب الحماض الكيتوني السكري الحاد. تم تسجيل مدة إصابة المرضى بالسكري و طريقة العلاج، الأعراض قبل التنويم، النتائج الكيميائية و نسبة الوفاة. . تم تنويم 102 حالة. 86% كانت إصابتهم بالسكر معروفة. من الأسباب التي أدت إلى الحماض الكيتوني السكري الحاد هو توقف العلاج و الالتهابات التعفنية خصوصا التهاب المجاري البولية. من الأعراض التي لها علاقة بالتنويم إضطرابات الجهاز الهضمي خصوصا عند مرضى السكري النوع الأول. نسبة الوفاة 1%. وقد لوحظ تحسن في معدل التنويم و الوفاة مقارنة بالعشر سنوات الماضية. .

Objective: To study the clinical and biochemical profile of diabetic ketoacidosis and it's treatment outcome in King Abdul Aziz University Hospital, and to compare it with those reported 10-years previously.

Key Word: Diabetic Ketoacidosis, Presentation, Precepitating Factors, Outcome, Saudi Arabia

Introduction

Diabetic ketoacidosis is an acute metabolic complication of diabetes mellitus that can be life threatening. It occurs mainly in known diabetics[1,2] and is caused by absolute or relative lack of insulin.The mortality rate of DKA has shown a dramatic fall from almost 100% (in the era before introduction of insulin in 1920) to 1-9% in the present time[3,4,]. Although many reports have shown infection to be the main precipitating factor[3,5,6,7] other recent reports have shown poor compliance to be the major cause especially in urban black population[8,9]. Previous studies investigating the cause of DKA reported that in 14-32%of cases no identifiable cause could be found [9,10,11] and there was a significant relation between this and young age group[12]. The prevalence of diabetes mellitus is increasing world wide and in Saudi Arabia it increased from 4.95% in 1985[13] to 7.4% in 1995[14]. A previous study reported from KAUH in 1984[15] showed admission rate due to DKA was 123 cases over 2 years, it also showed poor compliance to be a major precipitating factor for DKA (65.9%) with a mortality rate of 4.8%. In this study we report our observations on 102 episodes of DKA admitted to KAUH over a 5- year period and we discuss the clinical , biochemical profile, treatment outcome of these episodes and compare it with the previous study.

Materials And Methods

This study included patients with diabetic ketoacidosis (DKA)who were admitted to the medical ward of King Abdulaziz University Hospital (KAUH) during the period from January 1994 to December 1998. The total number of patients with DKA was 102. Diagnosis of DKA was defined as follow(1):(i) plasma glucose > 250 mg% (13.8 mmol/l); (ii) serum bicarbonate < 15mmol/l;(iii) arterial pH < 7.3; (iv) ketonuria(as measured by nitroprusside method). All patients who were less than 14 years were excluded from this study. Detailed information for every participated patient was obtained regarding : duration of diabetes; type of outpatient treatment for diabetes;reason of stopping treatment if any; previous episodes of DKA; symptoms before hospitalization. Cases were diagnosed as first presentation if they have no prior history of diabetes mellitus. Data compilation was made with respect to clinical findings on admission, blood pressure, level of consciousness,

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state of hydration, biochemical parameters (including blood sugar, serum bicarbonate, arterial pH and blood gases on admission), mode of therapy, and outcome.

Analysis of data was conducted using the Statistical Package for Social Sciences(SPSS 7.5). A two tailed student's t test and Chi-square test were used as appropriate. Results were considered significant if P value was less than 0.05.

Results

Of the total admissions due to DKA 85(86.3%) were patients known to have D.M, while 17 (13.7%) were patients with no prior history of D.M. Of the known diabetics 66 patients (75%) were type I and 22 (25%) were type II who were significantly older with longer duration of diabetes mellitus (p 0.001). Also from those patients known to have diabetes 72 (81.8%) were on insulin and 16 (18.2%) on oral hypoglycemic agents. The overall sex ratio M:F was 1.1:1 with no sex difference between known and new diabetics or type I diabetics while it was 2.1:1 in type II (not statistically significant). Both new and known diabetics were thin with no significant difference in their BMI. (Table 1).

Precipitating Factors: Poor compliance and inadequate treatment was the cause of DKA in 50 (56.8%) admissions, 41 (46.6%) type I and 9 (10.2%) type II (p0.08). The reason for stopping treatment was either financial, to try local remedies or they didn't want to have insulin injections. (Table 1) Infection precipitated DKA in 46(44.1%) of the total cases, UTI being the commonest occured in 24 patients (23.5%). No significant difference was found between new and known diabetics or type I and II. (Table 2) In 17 patients(16.7%) no precipitating cause could be identified with significant relation only to those less than 20 years of age (p 0.001). One patient had myocardial infarction and another one had cerebrovascular accident as a precipitating cause of DKA.

Presentation: The majority of patients with DKA 91 patients (89.2%) present with gastrointestinal manifestations including anorexia, abdominal pain, nausea, vomiting and diarrhea.There was no significant difference between new and known diabetics but these symptoms were more common in type I diabetics 63 patients(78.8%) versus17 patients (21.3%) (p 0.01). (Table 1). Central nervous system manifestations as headache, drawsiness were found in 35 (34.3%) patients, one patient had loss of consciousness. There was no significant difference between new and known diabetics or type I and II.

Biochemical parameters: The biochemical parameters on admission are shown in the (Table I). The highest plasma glucose in our series was 59.4 mmol/l. All parameters were comparable between new and known diabetics, difference was found in pH where type I present with more severe acidosis.

Treatment and Outcome: Treatment consisted of correction of fluid deficit and electrolyte abnormalities. Insulin was given as I.V bolus followed by continuos adjustable infusion. Sodium bicarbonate was given only if pH< 7.1. In addition appropriate antibiotics were used in patients with infection. There was one death in our series due to extensive myocardial infarction with an over all mortality 1%.

Discussion

Diabetic ketoacidosis occurs most often in patients with known diabetes especially type I. The main presenting symptoms being gastrointestinal, hence, instructing the patients about the risk of these symptoms as an early sign of DKA is essential. Poor compliance and infections were the main precipitating factors. Decreased admission rate, improved patient's compliance and decreased mortality compared to that 10 years ago reflects better patient's understanding of diabetes and it's complication.

Diabetic ketoacidosis is an acute metabolic decompensation that occurs mainly in patients known to have diabetes mellitus[1,2,10,11,23]. In our series 86.3% were known diabetics, and 13.7% had DKA as their first clinical presentation, a finding comparable with several other reports[15,8,16]. It is generally known that DKA occurs more frequently in type I diabetics [12,17,18,,19] this fact is also evident from our study which reveals that 75% of known diabetics were type I versus 25% type II. We found no over all sex differences but DKA occurs more in thin males

with type II diabetes, an observation that was also noticed in the Apachi Indians[20]. In our study poor compliance and inadequate treatment was the main precipitating factor for DKA which occurred in 56.8% of patients most of them were type I. This figure is different from other studies[3,5,6,7,21,22,23,24] which has shown infection to be the most frequent precipitating factor. These studies, however, didn't consider financial resources and diabetic education, but, finding similar to our results were found in urban -African American population[9,8]. Poor compliance was responsible for 65.9% of cases reported from KAU hospital 10 years previously. This might reflect improved patient education and better understanding of diabetes and it's complications. Infection was the second major cause , found in 44.1% of total cases with no difference between new and known diabetics. UTI was the most common infection, similar to what was reported before[15,16] Of total admissions 16.7% had no identifiable precipitating factor for their DKA, a finding reported in several studies[9,10,11,23]. However a significant relation was found between unknown precipitating factor and patients below the age of 20 years (p0.001) a finding also previously reported[12]. Gastrointestinal symptoms were a major type of presentation[16,24] especially in type I and this was shown in our patients where 89.2% of our patients presented with gastrointestinal manifestations. Severe acidosis on presentation was noticed more in type I which was due to severe metabolic decompensation due to decreased insulin reserve. We observed that mortality was 1% and this represent a large fall in mortality in comparison to that of KAUH 10 years previously when it was 4.8% and this decrease in mortality due to DKA has been noticed in other studies[25]. Another observation was the admission rate which showed a dramatic fall from 123 episodes over 2 years to 102 episodes over 5 years and this probably represented better patient education and good standard of hospital care. Although hospital admission due to DKA was decreasing, inspite of an increasing prevalence of diabetes mellitus, DKA was a common complication in type I diabetics. Admission and fatality due to DKA would further decrease by more adequate management, improving patient education program and treatment of underlying illness particularly infection.

References

1. Taft-P, Stocking JR, Harrison JW, Cameron-DP. Diabetic ketacidosis :it's causes and prevention. Med-J 1968 Aug 2:825-829
2. Beigelman PM: Diabetic ketacidosis(diabetic coma) 482 episodes in 257patients:experience of three years. Diabetes 1971 20:490-500
3. Ketabechi AE, Fisher JN, Murphy MB, RumbarkMJ, Diabetic Ketoacidosis and hyperglycemic hyperosmolar nonketotic state: In Kahn CR, Weir GC,eds. Joslin's diabetes mellitus, 13th edn. Philadelphia; Lea&Febiger, 1994: 738-770.
4. Hockaday TDR, Alberti-KGGM Diabetic coma Clin.Endocrinol. Metab1972;1:751-788.
5. Wachetl-TJ, Tetu-Mouradjian-LM;Goldman-DL. Hyperosmolarity and acidosis in diabetes mellitus three year experience in Rhode island. J-Gen-Intern-Med. 1991; 6(6):495-502
6. Matoo-VK, Nalini-K, Dash-RJ Clinical profile and treatment outcome of diabetic ketoacidosis J-Assoc-Physician-India. 199;39(5):379-81
7. Bhoraskar-AS. Diabetic ketoacidosis-clinical presentation. J-Diab-Assoc India1995;35(3):73-78
8. Umpierrez-GE, Kelly-JP, Navarrete-JE, Casals-MM, Kitabchi-AE. Hyperglycemic crises in urban black.Arch-Intern-Med. 1997; 24,157(6):669-75
9. Musy-VC;Lee-JK, Crawford-R;Klatka-MA, McAdams-D;Philips-LS. Diabetes in urban-African-American.cessation of insulin therapy is the major precipitating cause if diabetic ketoacidosis.Diabetes-Care.1995;18(4):483-89
10. Snorgaard-O, Eskildsen-PC, Vadstrup-S, Nertup-J Diabetic ketoacidosis in Denmark:epidemiology, incidence rate, precipitating factors and mortality rates J.Intern.Med. 1989 ;226: 223-228
11. Soler-NG, Bennett-MA, Fitzgerald-MG, Malins-JM Intensive care in the management of diabetic ketoacidosis. Lancet 1973; 1:951-954
12. Thompson-CJ, Cummings-P, Chalmers-J, Newton-RW Abnormal insulin treatment behavior:a major cause of diabetic ketoacidosis in young adult. Diabete-Med. 1995;12(5): 429-32
13. Fatani-H, Mira-SA, Elzuber. APrevalence of diabetes mellitus in rural Saudi Arabia Diabetes care 1987; 10(2):180-183

14. Alhasmi-MAF, Alswailem-A, Wasry-AS, Sulimani-R;Alswailem-APrevalence of diabetes mellitus in Saudi Arabia Saudi. Med.J. 1995;16(4):294-299

15. Mira SA, Fatani HH, Elzubeir AG, Elsabbagh S. Diabetic Ketoacidosis A report of 123 Saudi diabetics. Saudi Med. J. 1987; 8(4) : 364-368.

16. Rajasoorya-C, Wong-SF, Chew-LS Diabetic ketoacidosis a study of 33 episodes Singapore-Med-J.1993;34(5):381-4

17. Berger W. Diabetic emergencies. Schweiz-Rundsch-Med-Prax. 1997;18;86(8):308-13

18. Escobedo-de-la-pena-J, Rico-verdin-B Incidence and fatality of the acute and chronic complications of diabetes mellitus in Mexico. Salud-Publica-Mex. 1996; 38(4):236-42

19. Berne C. Diabetic coma - an unnecessary death.Nord-Med. 1992;107(8-9):213-4,216

20. Wilson-C;Krakoff-J;Gohdes D. Ketoacidosis in Apache Indians with NIDD Arch-Intern-Med. 1997; 13;157(18):2098-100

21. Wangnoo-SK, Zargar-AH, Mattoo-V, Reshi-AR, Dar-AF.Continuos glucose monitoring in diabetic ketoacidosis using the glucose monitor GM-1320 Diabetes Bulletin 1989;9:142-145

22. Hockaday-TDR, Alberti-KGGM. Diabetic coma. Br JHosp-Med.1972;7:183-198

23. Johnson-DD, Palumbo-PJ, Chu-C Diabetic ketoacidosis in a community-based population. Mayo- Clin-Proc 1980;55:83-88

24. Zarger-AH, Sofi-FA, Masoodi-SR, Laway-BA, Wani-AI. Clinical biochemical and therapeutic aspects of diabetic ketoacidosis and it's outcome Saudi Med-J. 1998;19(4):446-52

25. Wetterhal-SF, Olson-DR, Destefano-F, et al. Trends in diabetes and diabetic complication. Diabetes-Care.1992;15(8):960-7

Table 1., Comparison between type I & type II diabetics according to some variables:

Variables	Type I n=66	Type II n=22	P value
Age(yrs) Mean + SD	23.92 + 10.66	57.41 + 16.49	< 0.001
Sex (M:F)	1:1.1	2.1:1	0.11
BMI Mean + SD	22.05+4.32	23.12+ 4.89	0.37
Duration of D.M(yrs) Mean + SD	5.51 + 4.38	9.64+ 5.96	< 0.001
Poor compliance No. (%)	41 (46.6%)	9 (10.2%)	0.08
Presentation with GIT manifestation No. (%)	63 (78.8)	17 (21.3)	0.01
Blood suger(mmol/l) Mean + SD	30.52+ 7.51	32.30+ 10.51	0.39
pH Mean +SD	7.11+ 0.10	7.18+ 0.12	0.02
Bicarbonate(mmol/l) Mean + SD	8.36+ 3.62	9.31+3.39	0.27

Table 2., Distribution of diabetic patients according to type of diabetes and infection:

Variables	Type I n=25	Type II n=14	Total n=39
Chest infection No. (%)	4 (16%)	6 (42.9%)	10 (25.6%)
U.T.I No. (%)	14 (56%)	6 (42.9%)	20 (51.3%)
Dental abcess No. (%)	1 (4%)		1 (2.6%)
Tonsillitis No. (%)	3 (12%)		3 (7.7%)
Foot infection No. (%)		2 (14.3%)	2 (5.1%)
Others No. (%)	3 (12%)		3 (7.8)