P2-P092

Hypertriglyceridemia in Type 1 Diabetes children during Diabetic Ketoacidosis; Relation to DKA severity and Glycemic control

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Background and Objective

Diabetic ketoacidosis (DKA) is by far a serious and potentially life-threatening complication of T1D[1]. During an episode of DKA, an increase in TG (in 30-50% of cases) and total cholesterol levels (TC) was reported that probably results from temporary impairment of lipoprotein lipase (LPL) activity[2]. The incidence of acute pancreatitis and cerebral edema is increased in patients with DKA and hypertriglyceridemia[3]. Severe hypertriglyceridemia can increase risk of acute pancreatitis, especially with TG levels higher than 1,000–1,772 mg/dl[4]. The aim of our study was to evaluate the prevalence of hypertriglyceridemia at onset of DKA in T1D patients and assess its relation to DKA severity and glycemic control after 3 months.

Methodology

This cross-sectional study was conducted on random cohort of 84 children with T1D presenting with DKA episode at DEMPU, Cairo University over 6 months period. The study was approved by the Research Ethics Committee of Cairo University. Children with T2D, with diabetes secondary to post-surgical pancreatectomy, cystic fibrosis, or steroid therapy, or those using thyroxin therapy or any lipid lowering medications as well as those who have family history of dyslipidemia were excluded from the study. All patients included in the study (after taking informed consents from their legal guardians) were evaluated clinically for conscious level assessment (using Glasgow coma scale) as well as biochemical analysis for BG, ABG, serum electrolytes (Na, K) kidney functions (urea, creatinine). Serum triglycerides were measured initially during DKA presentation then at 48 h of DKA management (insulin therapy). HbA1c (%) levels were measured 3 months later.

Results

Among the 84 cases,43 (51.19%) were males and 41 (48.81%) were females with mean age 7.1±2.65 years (ranging between 2.5 and 13 years). Sixty-eight (80.95%) were newly diagnosed and 16 (19.05%) were known to be diabetic. Mean BG level at presentation was 490.86±278.74 mg/dl and median triglyceride level was 237.75 mg/dl [Table1].

There was a significant difference in prevalence of hypertriglyceridemia at onset of DKA and after 48 hrs of management (p<0.001). In our cohort, 74 patients (88.1%) had hypertriglyceridemia at onset of DKA ranging from 75.8-10500 mg/dl with significant improvement in TG after 48 hrs of DKA management (p<0.001). Hypertriglyceridemia resolved completely in 41 of them after 48 hrs, while 33 patients still had hypertriglyceridemia.

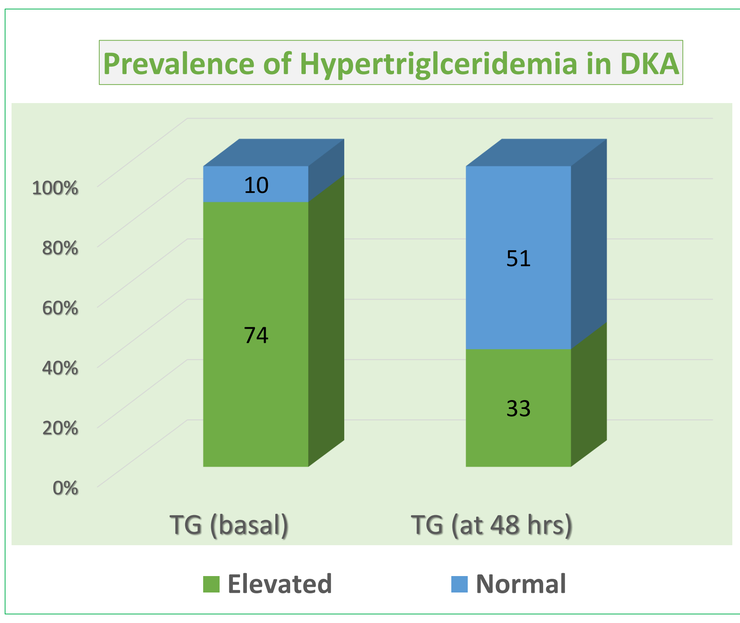

Figure (1): Prevalence of hypertriglyceridemia within the study group

When correlating basal serum TG with other study parameters in our cohort, a significant positive correlation was found with BG level (r= 0.703, p=0.005), while a significant negative correlation was found with serum bicarbonate and GCS (i.e. conscious level) with a p value of 0.012 & 0.022 respectively. On the other hand, correlating TG after 48 hrs with different study parameters showed a significant positive correlation with BG level (r= 0.704, p=0.005) and a significant negative correlation with pH, serum bicarbonate and GCS (p=0.01, 0.004 & 0.013 respectively) [Table 2].

When insulin requirements and HbA1c were assessed 3 months later in the study group, no significant correlation was found between triglycerides (either basal or after 48 hrs of DKA) and glycemic control or insulin requirements (TDD).

Comparing between different study parameters in relation to the onset of DKA (newly diagnosed with first attack of DKA & those known to be diabetic presenting with DKA), a significant difference was reported between both groups in mean difference of TG at 0 and at 48 h (p=0.049) [Table 3]. When the duration of hospital stay and ICU stay was studied in relation to different study parameters, both correlated positively with initial TG.

Table (1): Metabolic and biochemical study parameters

	Range	Mean±SD	Median (IQR)
Wt SDS	-3.08 - 2.33	-0.39±1.06	-0.24 (1.27)
RBG (mg/dl)	249-1170	490.86±278.74	419.5 (178)
pH	6.9-7.34	7.16±0.12	7.1 (0.24)
HCo3 (mEq/l)	1.9-14	8.51±3.01	7.55 (4.25)
GCS	12-15	14.52±0.86	15 (1.25)
TG1 (mg/dl)	75.8-10500	393.53±1126.3	237.75 (128.85)
TG2 (mg/dl)	65.8-1850	160.96±197.17	115.5 (133.93)
HbA1c after 3 mo (%)	6.8-11	8.47±1.03	8.5 (1.075)
TDD of insulin after 3 mo (IU/kg/d)	0.66-1.8	1.18±0.26	1.185 (0.315)

Wt SDS: weight, **RBG:** random blood glucose, **GSC:** Glasgow coma scale ,**TG1:** initial triglycerides, **TG2:** triglycerides after 48 hrs, **TDD**: total daily dose

Table (2): Correlation between TG1&TG2 and other study parameter

	TG1		TG2	
	r	p-value	r	p-value
Age (years)	-0.166	0.130	-0.142	0.201
RBG (mg/dl)	0.703	**0.005**	0.704	**0.005**
pH	-0.187	0.089	-0.283	**0.010**
HCo3 (mEq/l)	-0.274	**0.012**	-0.314	**0.004**
GCS	-0.250	**0.022**	-0.270	**0.013**
HbA1c after 3mo (%)	0.056	0.611	0.055	0.624
TDD of insulin after 3mo (IU/kg/d)	-0.005	0.965	0.049	0.657
Duration of total hospital stay (h)	0.528	**< 0.001**		
Duration of ICU stay (h)	0.370	**< 0.001**		

RBG: random blood glucose, **GSC:** Glasgow coma scale, **TG1:** initial triglycerides, **TG2:** triglycerides after 48 hrs, **TDD**: total daily dose

Table (3): Comparison between new onset and known T1D regarding study parameters.

		Onset of T1D		*p*-value
		New	Known	
Sex	*Male*	33 (48.5%)	10 (62.5%)	0.314
	Female	35 (51.5%)	6 (37.5%)	
Age (yrs)		6.86±2.57	8.13±2.83	0.086
RBS (mg/dl)		508.75±298.3	383.5±65.76	0.577
pH		7.16±0.109	7.15±0.14	0.681
HCo3 (mEq/L)		8.24±3.083	9.66±2.45	0.090
GCS		14.5±0.89	14.63±0.719	0.603
TG1 (mg/dl)*		224.9 (141)	297 (249)	0.565
TG2 (mg/dl)*		119 (98)	104 (117)	0.869
HbA1C (%)		8.53±1.047	8.24±0.96	0.324
Insulin TDD after 3 mo (IU/kg/d)		1.177±0.262	1.2±0.24	0.703
TG mean difference (mg/dl)		255.21±1041.4	146.44±124.67	**0.049**

RBG: random blood glucose, **GSC:** Glasgow coma scale, **TG1:** initial triglycerides, **TG2:** triglycerides after 48 hrs, **TDD**: total daily dose

* Regarding TG, median values were used in comparison instead of mean due to outliers.

Conclusion

Hypertriglyceridemia was detected in most patients of T1D during episodes of DKA that significantly declined with insulin therapy. Serum TG correlated with the DKA severity and BG levels. However, it did not affect glycemic control or insulin dose later on.

Bibliography

[1] **Kearney T, & Dang C.** Diabetic and endocrine emergencies. **Postgrad Med J.** 2007;83(976):79-86.
[2] **Nocoń-Bohusz J, Wikiera B, Basiak A et al.** LPL gene mutation as the cause of severe hypertriglyceridemia in the course of ketoacidosis in a patient with newly diagnosed type 1 diabetes mellitus. **Pediatr Endocrinol Diabetes Metab.** 2016 Feb ;21(2):89-92.
[3] **Saengkaew T, Sahakitrungruang T, Wacharasindhu S, & Supornsilchai V.** DKA with severe hypertriglyceridemia and cerebral edema in an adolescent boy: A case study and review of the literature. **Case Rep Endocrinol.** 2016; 2016: 7515721
[4] **Athyros VG, Giouleme OI, Nikolaidis NL et al.** Long-term follow-up of patients with acute hypertriglyceridemia-induced pancreatitis. **J Clin Gastroenterol.** 2002;34(4): 472-475.