

Case report

Diabetic Ketoacidosis with Severe Dyslipidemia in a Pediatric Patient with New-Onset Type 1 Diabetes Mellitus: A Case Report

Sarah Alfaqaih^{1*} , Soad Albira^{1,2} , Ebtesam Ahmed^{1,2} ¹Department of Pediatrics, Faculty of Medicine, University of Misurata, Misurata, Libya²Pediatric Intensive Care Unit, Misurata Medical Center, Misurata, LibyaEmail: sarah.alfaqaih@med.misuratau.edu.ly

Keywords

Diabetic Ketoacidosis,
Dyslipidemia,
Hypertriglyceridemia,
Pediatric.

ABSTRACT

Diabetic ketoacidosis (DKA) is a life-threatening complication of type 1 diabetes mellitus (T1DM). Although mild hypertriglyceridemia (HTG) is common in DKA, severe HTG (triglycerides >2000 mg/dL) is rare in pediatric patients and markedly increases the risk of acute pancreatitis (AP). This report describes a unique case of new-onset T1DM presenting with DKA and extreme dyslipidemia, highlighting management challenges. An 11-year-old girl presented with fatigue, dehydration, and Kussmaul breathing. Laboratory tests confirmed DKA (pH 7.01, blood glucose 490 mg/dL) and showed a severely lipemic blood sample. Initial management included fluid resuscitation and an intravenous insulin infusion. Although DKA resolved within 48 hours, triglyceride (TG) levels surged from 1271 to 3967 mg/dL on the fourth day after reducing the intravenous insulin infusion rate from 0.1 to 0.05 IU/kg/h, accompanied by mild abdominal pain. AP was ruled out by ultrasound, and enzyme levels were normal. Management required readjusting the insulin infusion rate and initiating oral lipid-lowering agents (fenofibrate, rosuvastatin, and omega-3). TG levels normalized within 20 days, though low-density lipoprotein (LDL) remained elevated, consistent with the beta shift phenomenon. A significant family history of hyperlipidemia suggested a genetic predisposition. Severe HTG is a critical complication of pediatric DKA that requires prompt recognition and treatment. Intravenous insulin remains the primary treatment, but clinicians must monitor for rebound lipid levels after acidosis resolves. Oral lipid-lowering therapies are effective adjuncts, especially when plasmapheresis is unavailable. This case emphasizes the need for individualized management and long-term lipid monitoring in pediatric T1DM.

Introduction

Type 1 diabetes mellitus (T1DM) is a common endocrine disorder among children, characterized by acute insulin deficiency [1, 2]. Diabetic ketoacidosis (DKA) is a potentially life-threatening complication of newly diagnosed or poorly controlled diabetes mellitus and is the initial presentation of T1DM in 30-55% of cases [2-4]. Reported complications from delayed or improper diagnosis and management of DKA include cerebral edema, acute pancreatitis (AP), hypertriglyceridemia (HTG), pulmonary edema, peripheral venous thrombosis, and rhabdomyolysis [5]. Mild HTG is frequently reported during the course of treatment for diabetes and diabetic ketoacidosis. However, severe HTG has been reported in up to 8% of adults with DKA, while a limited number of cases have been reported in younger age groups [6]. HTG in younger age groups is defined as an increased fasting plasma TG concentration above the 95th percentile for age and gender [7]. Severe HTG is defined as a TG level above 1000 mg/dL in adults and between 1000-1999 mg/dL in pediatric age groups, and very severe HTG is >2000 mg/dL [8]. HTG can be primary, resulting from underlying genetic mutations, or secondary to other disorders such as diabetes mellitus (DM), obesity, and medications (e.g., estrogen) [9, 10]. The most commonly cited mechanism for HTG associated with DKA is increased lipid production and decreased clearance. Increased secretion of free fatty acids (FFAs) and amino acids from adipocytes and muscles, along with elevated counter-regulatory hormones, drives increased hepatic gluconeogenesis and glycogenolysis. Increased hepatic uptake of FFAs leads to greater production of very low-density lipoprotein (VLDL), which causes HTG [11]. In addition, insulin deficiency in DKA decreases lipoprotein lipase (LPL) activity, which is required for clearance of VLDL and chylomicrons from plasma. After insulin administration, LPL is upregulated, leading to excessive conversion to LDL [12]. It has been proposed that its development might be worsened by a concurrent genetic tendency toward hyperlipidemia [13, 14] because LDL has a longer half-life; it may persist even when triglycerides and VLDL have normalized. This is explained by the beta shift phenomenon but remains poorly established [15, 16]. This phenomenon is rarely reported in the pediatric age group [17, 18].

The incidence of severe HTG with DKA in the pediatric population is unknown, with limited reported cases in the literature [6, 17-43]. Across all documented cases, prompt diagnosis and management are required to prevent the risk of acute pancreatitis, a well-known and severe consequence of HTG. We report a presentation in an 11-year-old girl with DKA,

severe HTG, hypercholesterolemia, and a beta-shift phenomenon. To our knowledge, this is the first case reported in the region in a pediatric patient.

Case presentation

An 11-year-old girl presented with a one-day history of fatigue and lightheadedness. She was previously normal; she was evaluated for celiac disease because of poor weight gain around 6 weeks before presentation and was normal. She presented to the emergency room with Kussmaul breathing and pallor. She was drowsy but oriented, moderately to severely dehydrated, and systemic examination was unremarkable apart from mild abdominal distension; anthropometrics: weight 27 Kg, height 137 cm [3rd centile], BMI 15 [3rd-15th centile]. She had cold extremities and weak, rapid pulses. Her vital signs were: heart rate (121 beats/min), respiratory rate (49 breaths/min), temperature (36.4 °C), and blood pressure (110/79 mmHg). Her initial blood glucose was 490mg/dL by glucometer, and the urine dipstick showed glucose (+4) and ketones (+3). The patient received a normal saline fluid bolus (20mL/kg) and was transferred to the PICU for further management (Table 1).

In the PICU, her initial arterial blood gas (ABG) showed severe metabolic acidosis: pH 7.01, HCO₃ 3.5 mmol/L, pCO₂ 13.7 mmHg, and anion gap 25.4 mmol/L. Routine blood investigations clearly showed a lipemic sample. (Figure 1-a) The laboratory specialist could not process the lipemic sample, and most investigations were unreliable for assessing the patient's overall condition.

The patient started on hydration therapy, and insulin infusion was initiated at 0.1 IU/kg/hour. The general condition improved after 12 hours of insulin therapy, despite the ABG results. After 24 hours, blood analysis was performed; the sample remained milky but had improved compared with the initial sample. (Figure 1-b, c) TG 1271 (mg/dL), TC 1422 (mg/dL); the follow-up lipid profile results are shown in Figure 2.

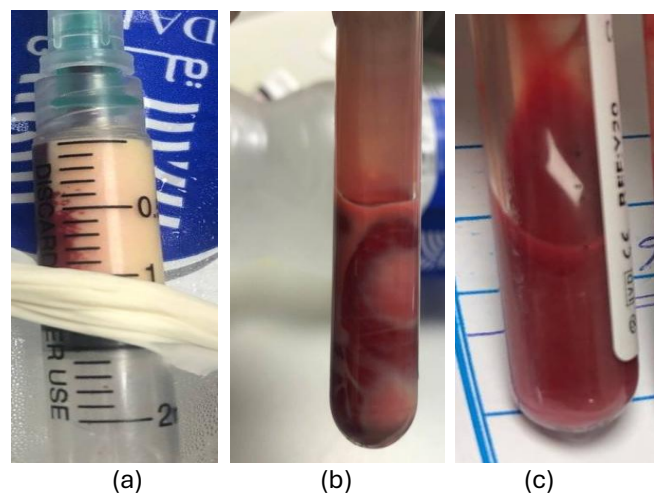


Figure 1. (a) Day-1 blood sample, (b) Day-2 blood sample, (c) Day-7 blood sample

On the third day of PICU admission, she complained of epigastric abdominal pain, mildly tender to palpation. Acute pancreatitis (AP) was suspected, and she was evaluated with abdominal ultrasound, which showed no evidence of AP and normal liver architecture. Serum amylase and lipase were initially normal: 6.8 U/L and 27.3 U/L, respectively. DKA resolved, which was defined by resolution of metabolic acidosis and ketonuria; minimal oral feeding was allowed, and the insulin infusion was decreased to 0.05 IU/kg/hour.

On the fourth day, laboratory evaluations showed a surge in the lipid profile: TG 3967 (mg/dL [normal range <200mg/dL]) and TC 1018 (mg/dL [normal range <200mg/dL]). The insulin infusion was readjusted to 0.1 IU/kg/hour, and fluids were managed with 5-10% glucose saline as required. The following day, TG declined to 2487 (mg/dL) and TC to 1235 (mg/dL), with clinical improvement in abdominal pain. Antilipid medication was started on the fifth day with daily fenofibrate 67mg, rosuvastatin 10 mg, and omega-3 supplementation. The next day, SC insulin was started, and the patient was prepared for discharge.

The endocrinology team closely followed her. Review of the family history revealed a significant history of familial hypertriglyceridemia, with an affected father and type 2 diabetes mellitus. After 20 days of follow-up, the lipid profile results were TG (87.6 mg/dL), HDL (51.3 mg/dL [normal range ≥ 35mg/dL]), and VLDL (16.2 mg/dL), and the TC/HDL ratio was 26.4 (normal < 4.5), TC and LDL remained elevated at 558 mg/dL and 478.3 mg/dL, respectively. We stopped fenofibrate and continued on rosuvastatin 10 mg daily and omega-3 1000 mg. TC and LDL showed a declining trend at the next follow-up investigations and returned to normal levels at 60 days of follow-up; TG (79.3 mg/dL), TC (113 mg/dL), LDL (65.6 mg/dL [normal range 10-130 mg/dL]), HDL (40.8 mg/dL), TC/HDL ratio (2.76), and she continued omega-3 daily dose only. She reported no side effects of concern during her pharmacological therapy. At the last follow-up before this report (100 days),

TG (100 mg/dl), TC (240 mg/dl), HDL (97.6 mg/dl), LDL (150 mg/dl), TC/HDL ratio (2.45), and HbA1c 6.8% (Figure 2). The patient continued the same management plan, and the importance of proper glycemic control and diet was emphasized. The family was counseled about genetic testing to exclude a genetic component as the cause of dyslipidemia, especially with a positive family history; because of financial considerations, it was postponed.

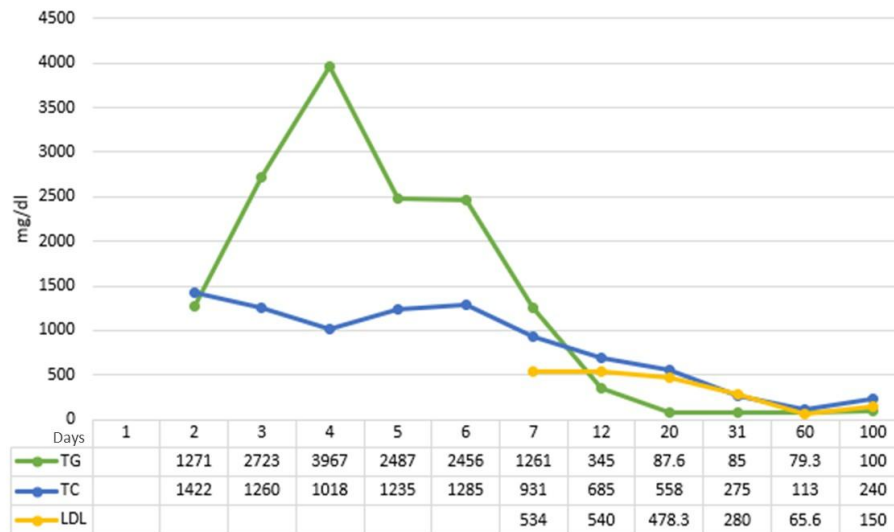


Figure 2. The line graph shows lipid profile results.

Table 1. Inpatient laboratory values

Lab value	Day 1	Day 2	Day3	Day 7	Reference value
PH	7.01	7.25	7.35	-	7.35-7.45
pCo2	13.7	16.2	35.8	-	35-45 mmHg
Hco3	3.6	7.02	19	-	22-26 mEq/L
glucose level	490	285	179	158	70-120 mg/dl
Hemoglobin	-	23.5	19.5	12	11.0-14.0 g/dL
Total leucocyte count	-	10	4.1	6.29	4000-11000/ μ L
Absolute neutrophil count	-	6.47	2.3	2.59	1500-8500/ μ L
Absolute lymphocyte count	-	1.97	1.4	2.95	1500-7000/ μ L
Platelet	-	210	230	360	150000-350000/ μ L
creatinine	-	1.75	0.74	0.34	0.3-1.1 mg/dl
Na	138	145	140	135	135-147 mmol/L
K	3.4	3	3.8	3.9	3.5-5.3 mmol/L
Cl	-	114	-	108.6	95-110 mmol/L
serum urea	31	10	16.9	18	<50 mg/dl
S. amylase	6.8	-	37.6	12	25-115 U/L
S. Lipase	27.3	-	14.8	13	8-78 U/L
ALT	288	404	163	82.3	<=31 U/L
AST	1670	434	725	98.3	<=31 U/L
Total Bilirubin	-	1.2	0.12	0.7	0.22-1.20 mg/dL
Direct Bilirubin	-	0.6	0.06	0.06	0.1-0.4 mg/dl
Total protein	8.8	-	5.1	6.3	6.6-8.7 g/dl
Serum albumin	5.8	-	5	4.6	3.6-5.1 g/dl
C-Reactive Protein	622	442	20	2	< 5
Procalcitonin (PCT)	0.05	-	-	-	<0.05 ng/mL
HbA1c	17	-	-	13	< 5.7%
Anti-GAD	-	-	26.9	-	<10 IU/ml
anti-IA2	-	-	659	-	<10 IU/ml
C-peptide	-	-	0.4	-	1.0-4.8 ng/ml

TSH	-	-	-	1.445	0.4 - 4.0 mIU/L
FT4	-	-	-	11.95	10 to 23 pmol/L
Urine glucose	+4	+3	+	+	Negative
Urine ketone	+3	+1	Neg	Neg	Negative

Discussion

Hypertriglyceridemia is an uncommon complication of DKA in pediatric populations. Our case has one of the highest reported TG levels in pediatric cases without AP. Detecting HTG in DKA is critical, given the risk of AP. One of the largest retrospective cross-sectional studies found that 59% of documented cases of AP have an HTG > 2000 mg/dl in the adult population [44]. There is no documented estimate of the incidence of severe HTG with AP in pediatric DKA; it has been described in multiple reported cases in the literature. The occurrence of severe HTG with DKA has been reported in newly diagnosed T1D [17-28, 30, 31, 33, 34, 36-42], known cases of T1D [6, 29, 32, 35, 43], or MODY [35], as thoroughly reviewed in reported cases. Our patient presented with life-threatening DKA, severe HTG, and hypercholesterolemia as a presentation of newly diagnosed T1D. She was treated with fluid hydration and an insulin infusion. Notably, after resolution of metabolic acidosis and a decrease in the insulin infusion requirement, a subsequent peak in the lipid profile was observed. As a result, the insulin infusion was readjusted until TG and TC levels plateaued. Similar cases have been reported in the literature and require higher insulin infusion rates [17, 18, 25, 31, 40, 42].

Regarding insulin infusion duration, limited data exist on pediatric HTG treatment, whereas in adults with diabetes and HTG, an average of 55.8 h of I.V. insulin infusion was needed to reduce TG to less than 500 mg/dl [45]. In reported pediatric cases under 18 years of age, infusion time ranges from 14 h to 7 days. In our case, as with most reported instances, the lipemic sample was the sole indicator of severe dyslipidemia. Routine screening of triglyceride (TG) levels at the time of T1D presentation is not a standard practice, so the true prevalence of severe hypertriglyceridemia (HTG) in newly diagnosed diabetes mellitus (DM) remains unknown. The American Diabetes Association guidelines recommend obtaining a lipid profile shortly after diagnosis and once glycemic control is achieved [46]. Across the cases we reviewed in the literature and our case, we highlight the importance of prompt detection and treatment to prevent complications associated with severe HTG. This evidence was supported by Richardson et al. [10], who conducted the largest retrospective study on HTG, reviewing all causes of severe HTG in children aged 17 years or older (1999–2016). They identified 124 cases of severe HTG, with approximately 19% linked to T1D and 15% associated with DKA [10].

Although our case did not develop AP, severe HTG required prompt treatment to prevent complications related to it. There is no universal guideline for management of severe HTG associated with DKA. Still, all approaches agree that IV insulin therapy and fluid resuscitation are the main options of treatment, as has been reported in all cases reviewed. Additional interventions were required in the associated cases; for instance, plasmapheresis or exchange was needed in a limited number of cases with a severe TG level of 16334 mg/dl, associated with AP, corneal cloudiness, and unresponsiveness to the initial approach [22, 40].

As our case had mild abdominal pain and AP was ruled out, plasmapheresis was not an option for treatment. We started oral lipid-lowering agents: fenofibrate, rosuvastatin, and Omega-3, which is similar to other reported cases [18, 29-32, 41] but only one case combined a statin with fenofibrate. In our case, there is a significant family history of familial hypertriglyceridemia (patient's father and grandmother), which encouraged our decision to start a statin as part of the approach. Macke et al. reported, among their case series, three cases with a positive family history of dyslipidemia: case (A), a 16-year-old male treated with fenofibrate and omega-3; case (B), a 7-year-old boy; and case (E), a 5-year-old girl, who did not receive any anti-lipid medication [18]. Our case report had no side effects from fenofibrate or statin therapy. The statin therapy is well-tolerated and approved in the pediatric population [47] while fenofibrate is not routinely approved for children and is generally considered an off-label treatment reserved for severe, refractory pediatric hypertriglyceridemia > 400mg/dl [48]. Recently, a controlled trial showed fenofibrate as a safe and effective agent for TG-lowering in the pediatric age group [49].

We think the timing of HTG normalization cannot be predicted and varies by case and treatment approach, ranging from less than one day to several months. In our case, TG, VLDL, and HDL normalized at 20 days of treatment, but her total cholesterol and low-density lipoprotein (LDL) were elevated slightly above normal levels after one and a half months of stopping rosuvastatin. This course was described as a beta-shift phenomenon, but the exact mechanism of occurrence is not fully understood [16]. This has been reported in pediatric cases in the published literature [17, 18]. In our case and in previously reported cases, we emphasize that proper glycemic and nutritional control are the main goals for further follow-up, although we cannot exclude genetic causes in our case.

We cannot overlook underlying genetic causes as contributors to HTG and treatment response. Genetic testing was not available for most reported cases, including ours; only 4 of 33 cases reviewed had a documented genetic study. Kilimci et al. [43] identified a heterozygous variant, p.Asn318Ser (NM_000237.3: c.953A>G), in the 6th exon of the LPL gene in their reported patient, with conflicting pathogenicity (ClinGen: CA251887). Nocoń-Bohusz et al. [26] detected a heterozygous LPL gene variant (c.1018+34 A>G) in the presented case, whereas Park and Skala-Zaborowski reported no LPL gene

pathogenic variants [35, 36] The data above suggest that most cases of diabetic lipemia result from a quantitative deficiency of lipoprotein lipase secondary to insulin deficiency. In contrast, genetic mutations in LPL or its primary cofactors play a smaller role in hypertriglyceridemia [14]. In our case, the normalization of triglyceride levels goes against heritable dyslipidemia, although genetic testing is recommended because of a strong family history of dyslipidemia.

Conclusion

Severe hypertriglyceridemia (HTG) is a rare but serious complication of pediatric diabetic ketoacidosis (DKA) that can cause acute pancreatitis. Our case showed that while initial insulin therapy and fluids are essential for DKA resolution, careful monitoring and insulin adjustment are needed because lipid levels can fluctuate, especially after acidosis. With no standard guidelines for pediatric HTG in DKA, management should be tailored, using intravenous insulin and fluids as main treatments. Oral lipid-lowering drugs such as fenofibrate, rosuvastatin, and Omega-3 Fatty Acids proved effective in our patient, especially given a family history of dyslipidemia, offering an alternative when plasmapheresis is unaffordable.

References

1. Glaser N, Fritsch M, Priyambada L, Rewers A, Cherubini V, Estrada S, et al. ISPAD clinical practice consensus guidelines 2022: Diabetic ketoacidosis and hyperglycemic hyperosmolar state. *Pediatr Diabetes*. 2022;23(7):835-56. doi: 10.1111/pedi.13406. PubMed PMID: 36250645.
2. Jefferies C, Cutfield SW, Derraik JG, Bhagvandas J, Albert BB, Hofman PL, et al. 15-year incidence of diabetic ketoacidosis at onset of type 1 diabetes in children from a regional setting (Auckland, New Zealand). *Scientific reports*. 2015;5(1):10358.
3. Hamman RF, Bell RA, Dabelea D, D'Agostino RB, Jr., Dolan L, Imperatore G, et al. The SEARCH for Diabetes in Youth Study: Rationale, Findings, and Future Directions. *Diabetes Care*. 2014;37(12):3336-44. doi: 10.2337/dc14-0574.
4. Naccarato CQ, Azevedo N, Liberatore RDR, Jr. Analysis of complications related to diabetic ketoacidosis in pediatric patients at a University Hospital: a cross-sectional study. *J Pediatr (Rio J)*. 2025;101(3):445-50. Epub 20250312. doi: 10.1016/j.jped.2025.01.009. PubMed PMID: 40037551; PubMed Central PMCID: PMC12039515.
5. Valaiyapathi B, Ashraf A. Hospital management of severe hypertriglyceridemia in children. *Current Pediatric Reviews*. 2017. doi: 10.2174/1573400514666180117092707.
6. Saengkaew T, Sahakitrungruang T, ... DKA with severe hypertriglyceridemia and cerebral edema in an adolescent boy: a case study and review of the literature. *Case Reports in ...* 2016. doi: 10.1155/2016/7515721.
7. Cruz-Bautista I, Huerta-Chagoya A, Moreno-Macías H, Rodríguez-Guillén R, Ordóñez-Sánchez ML, Segura-Kato Y, et al. Familial hypertriglyceridemia: an entity with distinguishable features from other causes of hypertriglyceridemia. *Lipids Health Dis*. 2021;20(1):14. Epub 20210215. doi: 10.1186/s12944-021-01436-6. PubMed PMID: 33588820; PubMed Central PMCID: PMC7885394.
8. Gagnon CA, Ashraf AP. Beyond the Guidelines: Perspectives on Management of Pediatric Patients with Hypertriglyceridemia. *Curr Atheroscler Rep*. 2024;26(11):617-28. Epub 20240930. doi: 10.1007/s11883-024-01237-z. PubMed PMID: 39347913; PubMed Central PMCID: PMC11519174.
9. Shah AS, Wilson DP. Primary hypertriglyceridemia in children and adolescents. *Journal of clinical lipidology*. 2015;9(5):S20-S8.
10. Richardson T, Aslibekyan S, Ashraf A. Clinical characteristics and sequelae of severe hypertriglyceridemia in pediatrics. *Endocrine Practice*. 2018.
11. Qu Shen QS, Zhang Ting ZT, Dong H. Effect of hepatic insulin expression on lipid metabolism in diabetic mice. 2016.
12. Vergès B. Lipid disorders in type 1 diabetes. *Diabetes & metabolism*. 2009;35(5):353-60.
13. Karagianni C, Stabouli S, Roumeliotou K, Traeger-Synodinos J, Kavazarakis E, Gourgiotis D, et al. Severe hypertriglyceridaemia in diabetic ketoacidosis: clinical and genetic study. *Diabetic medicine*. 2004;21(4):380-2.
14. Radhakutty A, Shen J, Hooper A, Miller S, Burnett J, Mah P, et al. Quantification and genotyping of lipoprotein lipase in patients with diabetic lipaemia. *Diabetic medicine*. 2014;31(12):1702-7.
15. Gardner DG, Shoback D. *Greenspan's basic & clinical endocrinology*. 2018.
16. Barnett ML, Casipit C, Lam J, ... SAT-268 Beta-Shift Phenomenon Following Resolution of Diabetic Ketoacidosis: A Case Report. *Journal of the ...* 2025.
17. Vitale R, Laffel L. New-onset type 1 diabetes in a child with diabetic ketoacidosis and severe hypertriglyceridemia without pancreatitis. *JCEM case reports*. 2023.
18. Macke C, Al-Gadi I, Bansal N, Lyons S, ... Hypertriglyceridemia in New-Onset Type 1 Pediatric Diabetes. *Pediatric ...* 2025. doi: 10.1155/pedi/8425032.
19. CYWINSKI JS, WALKER FA, WHITE H, TRAISMAN HS. Juvenile diabetes mellitus associated with acute pancreatitis. *Acta Paediatrica*. 1965;54(6):597-603.
20. Blackett P, Holcombe J, Alaupovic P, ... Plasma lipids and apolipoproteins in a 13-year-old boy with diabetic ketoacidosis and extreme hyperlipidemia: researchgate.net; 1986.
21. Slyper AH, Wyatt DT, Brown CW. Clinical and/or biochemical pancreatitis in diabetic ketoacidosis. *Journal of Pediatric Endocrinology & Metabolism*. 1994;7(3):261.
22. Lutfi R, Huang J, Wong H. Plasmapheresis to treat hypertriglyceridemia in a child with diabetic ketoacidosis and pancreatitis. *Pediatrics*. 2012.
23. Williamson S, Alexander V, Greene SA. Severe hyperlipidaemia complicating diabetic ketoacidosis. *Archives of disease in childhood*. 2012;97(8):735-.

24. Aboulhosn K, Arnason T. Acute pancreatitis and severe hypertriglyceridaemia masking unsuspected underlying diabetic ketoacidosis. Case Reports. 2013.
25. Wolfram P, MacDonald M. Severe hypertriglyceridemia causing acute pancreatitis in a child with new onset type I diabetes mellitus presenting in ketoacidosis. Journal of pediatric intensive 2013. doi: 10.3233/pic-13053.
26. Nocoń-Bohusz J, Wikiera B, Basiak A, ... LPL gene mutation as the cause of severe hypertriglyceridemia in the course of ketoacidosis in a patient with newly diagnosed type 1 diabetes mellitus. Pediatric Endocrinology 2016.
27. Fick T, Jack J, Pyle-Eilola A, ... Severe hypertriglyceridemia at new onset type 1 diabetes mellitus. Journal of Pediatric 2017. doi: 10.1515/jpem-2017-0008.
28. Sharma P, Kumar M, ... Severe hypertriglyceridemia causing pancreatitis in a child with new-onset type-I diabetes mellitus presenting with diabetic ketoacidosis: pmc.ncbi.nlm.nih.gov; 2017.
29. Zaher F, Boubagura I, Rafi S, ... Diabetic ketoacidosis revealing a severe hypertriglyceridemia and acute pancreatitis in type 1 diabetes mellitus. Case Reports in 2019. doi: 10.1155/2019/8974619.
30. Yagnik P, Desai P, Modem V. Hypertriglyceridemia with acute pancreatitis in pediatric diabetic ketoacidosis: a case report: cureus.com; 2019.
31. Baidwan B, Walsh E, Skelton J, ... Severe hypertriglyceridemia with new-onset type 1 diabetes in diabetic ketoacidosis: pmc.ncbi.nlm.nih.gov; 2020.
32. Sari TE, Rini EA, Khairunnisa K. Very Severe Hypertriglyceridemia in Children with Diabetic Ketoacidosis. Jurnal Kedokteran Syiah Kuala. 2021;21(2).
33. Chreith A, Hijazia K, Doya L, ... Severe dyslipidemia associated with diabetic ketoacidosis in newly diagnosed female of type 1 diabetes mellitus: academic.oup.com; 2021.
34. Basta C, Ramones K, Agarwal S, Marino G, ... Severe hypertriglyceridemia: a rare complication of diabetic ketoacidosis in a 3-year-old with SARS-CoV-2 infection: Elsevier; 2021.
35. Park H, Kim M, Kim J, Lee S, ... Hypertriglyceridemia with acute pancreatitis in a 14-year-old girl with diabetic ketoacidosis: synapse.koreamed.org; 2022.
36. Skala-Zamorowska E, Kaminska H, ... Severe hypertriglyceridemia in the course of ketoacidosis in a patient with newly diagnosed type 1 diabetes mellitus. Disaster and 2022.
37. Katebi L. A Case of Hypertriglyceridemia in a Child 12 Years Old with Diabetic Ketoacidosis: sid.ir; 2023.
38. Daxay GJ, Yadav N, Anigani K, Saini SK, Kumari S, Saini AK. Severe diabetic ketoacidosis complicated with severe dyslipidemia, cerebral edema and acute kidney injury in a pediatric girl with type 1 diabetes mellitus: a case study. 2023.
39. Arora A, Khan O, Gupte S, Patil M, Mane S, ... A case of diabetic ketoacidosis with severe hypertriglyceridemia and slowly resolving metabolic acidosis: cureus.com; 2024.
40. Huang S, Song F, Gao K, Song Y, Chen X. Plasma exchange treatment of a diabetic ketoacidosis child with hyperlipidemia to avoid pancreatitis: a case report: frontiersin.org; 2024.
41. Kulkarni N, Sarangi B, ... Severe hypertriglyceridemia in an adolescent female with diabetic ketoacidosis: A case report: journals.lww.com; 2024.
42. Sooin S, Kaur G, Kaul V, Batra D. Severe Hypertriglyceridemia in a Child with Refractory, Fatal Diabetic Ketoacidosis: A Case Report with Review of Literature: journals.lww.com; 2024.
43. Kilimci D, Bal A, Ozkinay F, Ersoy B. Lipoprotein Lipase (LPL) Gene Mutation in a Girl With Diabetic Ketoacidosis, Acute Pancreatitis, and Hypertriglyceridemia: cureus.com; 2025.
44. Amblee A, Mohananeey D, Morkos M, Basu S, Abegunde AT, Ganesh M, et al. ACUTE PANCREATITIS IN PATIENTS WITH SEVERE HYPERTRIGLYCERIDEMIA IN A MULTI-ETHNIC MINORITY POPULATION. Endocr Pract. 2018;24(5):429-36. Epub 20180302. doi: 10.4158/ep-2017-0178. PubMed PMID: 29498913.
45. von Oettingen JE, Rhodes ET, Wolfsdorf JI. Resolution of ketoacidosis in children with new onset diabetes: Evaluation of various definitions. Diabetes Res Clin Pract. 2018;135:76-84. Epub 20171028. doi: 10.1016/j.diabres.2017.09.011. PubMed PMID: 29111277; PubMed Central PMCID: PMC6013285.
46. Tiwari D, Aw TC. The 2024 American Diabetes Association guidelines on Standards of Medical Care in Diabetes: key takeaways for laboratory. Exploration of Endocrine and Metabolic Diseases. 2024;1(4):158-66. doi: 10.37349/eemd.2024.00013.
47. Fiorentino R, Chiarelli F. Statins in Children, an Update. Int J Mol Sci. 2023;24(2). Epub 20230110. doi: 10.3390/ijms24021366. PubMed PMID: 36674877; PubMed Central PMCID: PMC9862804.
48. Kalra S, Gandhi A, Kalra B, Agrawal N. Management of dyslipidemia in children. Diabetology & Metabolic Syndrome. 2009;1(1):26. doi: 10.1186/1758-5996-1-26.
49. Dalili S, Koohmanaee S, Hassanzadeh Rad A, Nemati P, Mashayekhi-Sardoo H, Bayat R, Shahrokhi M. Fenofibrate vs. Omega-3 in Pediatric Hypertriglyceridemia: A Randomized Controlled Trial. J Pediatr Health Care. 2026. Epub 20260204. doi: 10.1016/j.pedhc.2025.12.021. PubMed PMID: 41636674.