



Contents lists available at ScienceDirect

AAACE Clinical Case Reports

journal homepage: www.aaaceclinicalcasereports.com

Case Report

Plasmapheresis as an Early Treatment for Severe Hypertriglyceridemia, Acute Pancreatitis, and Diabetic Ketoacidosis

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ARTICLE INFO

Article history:

Received 7 January 2021

Accepted 24 March 2021

Available online 6 April 2021

Key words:

acute pancreatitis

diabetic ketoacidosis

plasmapheresis

severe hypertriglyceridemia

type 1 diabetes

ABSTRACT

Objective: Severe hypertriglyceridemia (SHTG; plasma triglycerides >1000 mg/dL) is a rare but serious complication in children who develop diabetic ketoacidosis (DKA) from uncontrolled or new-onset type 1 diabetes.

Methods: We present the case of a severely malnourished 16-year-old with a 10-month history of presumed type 2 diabetes managed with lifestyle modifications and metformin, who presented with SHTG, acute pancreatitis, and DKA. On examination, there was no evidence of lipemia retinalis, cutaneous xanthomas, or xanthelasma. He was initially treated with an insulin infusion and intravenous fluids. Despite this treatment, his pancreatitis symptoms worsened and lipase level increased, necessitating 2 courses of plasmapheresis that immediately resolved his symptoms and dramatically improved his clinical status. He was discharged on hospital day 5. During his hospital admission, islet cell antigen 512, insulin, glutamic acid decarboxylase 65, and zinc transporter 8 autoantibodies were positive in the presence of insulinopenia, consistent with type 1 diabetes.

Results: Hypertriglyceridemia and hypercholesterolemia did not recur during follow-up, suggesting that the underlying mechanism for SHTG was insulin deficiency.

Conclusion: This report of SHTG, DKA, and pancreatitis in an adolescent highlights the safe, early initiation of plasmapheresis as an effective treatment. To our knowledge, plasmapheresis has rarely been used so early in the course of treatment for an adolescent with SHTG, DKA, and acute pancreatitis.

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Introduction

Diabetic ketoacidosis (DKA) is a serious metabolic complication of uncontrolled type 1 diabetes and, rarely, type 2 diabetes (T2D). DKA is defined as hyperglycemia, ketonemia or moderate or large ketonuria, and metabolic acidosis occurring from insulin deficiency.¹ Severe hypertriglyceridemia (SHTG; triglycerides >1000

mg/dL) and pancreatitis secondary to SHTG are very rare but serious complications in pediatric patients with DKA. While the standard treatment of SHTG involves intravenous (IV) fluids and insulin infusion, plasmapheresis is rarely reported with this clinical triad in the pediatric literature.^{2,3} We present our experience with laboratory discrepancies that complicated the diagnosis and with the utilization of plasmapheresis early in the disease course, which led to rapid stabilization of the patient and early discharge.

Abbreviations: DKA, diabetic ketoacidosis; IV, intravenous; SHTG, severe hypertriglyceridemia; T2D, type 2 diabetes.

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Case Report

A 16-year-old Hispanic male presented to his physician with decreased appetite and foul-smelling urine. His body mass index was 17.2 kg/m² (7 %ile), point-of-care glucose level was 283 mg/dL,

<https://doi.org/10.1016/j.aaace.2021.03.009>

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and urine dipstick was positive for glucose and ketones. Despite ketonuria, he was diagnosed with T2D and initially managed with lifestyle modifications. Three months after his initial presentation, his HbA1C reached 8.6% (70 mmol/mol) (reference range, <5.7%) and he was started on metformin. A fasting lipid panel was normal (cholesterol 146 mg/dL [reference range <170 mg/dL]; high density lipoprotein (HDL), 58 mg/dL [reference range >40 mg/dL]; low-density lipoprotein, 66 mg/dL [reference range <110 mg/dL]; very-low-density lipoprotein, 22 mg/dL; and triglycerides, 111 mg/dL [reference range <90 mg/dL]). The family reported excellent compliance with metformin and dietary modifications.

Ten months after his diagnosis, he presented to the emergency room with severe malnutrition, nausea, vomiting, and abdominal pain for 4 days and worsening hyperglycemia over 3 weeks (glucose levels, 200–300 mg/dL). His weight on admission was 47 kg (2 %ile), and his body mass index was 15 kg/m² (<1 %ile). Pertinent family history included a father with hypercholesterolemia and T2D diagnosed at age 37 and managed with metformin as well as a paternal uncle and paternal grandmother with T2D. There was no family history of myocardial infarction, early stroke, or pancreatitis.

On examination, the patient had moderate abdominal tenderness in both upper quadrants. He was alert and oriented, with no focal neurologic deficits. He had no eruptive cutaneous xanthomas, lipemia retinalis, tuberous xanthomas, or palmar crease xanthomas. Laboratory results showed mild acidosis based on an arterial blood gas of pH 7.32 (reference range, 7.35–7.45). However, a metabolic panel revealed a bicarbonate level of 8 mmol/L (reference range, 20–30 mmol/L) (Table 1). Urinalysis was positive for glucose and ketones. His blood appeared milky, prompting additional testing that revealed severely elevated triglycerides (28040 mg/dL) with elevated levels of cholesterol, low-density lipoprotein, and alanine transaminase. A repeat lipase showed an increase from 40 to 744 U/L over the course of a few hours, suggesting acute pancreatitis. Alcohol and drug testing were not performed due to low suspicion in the patient. Medications known to cause pancreatitis or hypertriglyceridemia were also excluded. He received 1 L of IV fluids and morphine for pain.

Upon arrival at the pediatric intensive care unit, his pH decreased to 7.23, despite fluid resuscitation. His β -hydroxybutyrate level was 8.9 mmol/L (reference range, 0.2–0.8 mmol/L); c-peptide, 0.2 ng/mL (reference range, 0.8–4.0 ng/mL); and insulin, <1 μ U/mL (reference range, <20 μ U/mL). His liver enzymes continued to be elevated, with aspartate aminotransferase at 80 U/L (reference range, <40 U/L) and alanine transaminase at 420 U/L (reference range, <60 U/L). HbA1C was elevated to 12.9% (117 mmol/mol). An abdominal ultrasound showed an abnormal, enlarged, inhomogeneous pancreas and no evidence of hepatosplenomegaly or gallstones.

With an elevated anion gap, acidosis, and ketones raising concern for DKA, he received IV hydration and was started on a regular insulin infusion at 0.1 U/kg/h that was increased to 0.15 U/kg/h the following morning when the level of triglycerides did not improve (Fig. 1). Twenty hours after initiating DKA therapy, his triglyceride levels remained >2000 mg/dL and his lipase levels remained elevated at 768 U/L. His abdominal pain acutely worsened and was no longer controlled by narcotic medications. As a result, 21 hours after admission, he received his first course of plasmapheresis, dramatically decreasing his triglycerides to 451 mg/dL and improving his pain. He received a second course of plasmapheresis at 40 hours, based on current practice guidelines,⁴ which decreased his triglycerides to 69 mg/dL. He started a low-fat diet 42 hours after admission, which he tolerated well. While clinically stable for discharge, he remained an inpatient for 2

Table 1

Laboratory Results with Reference Ranges Upon Arrival at the Emergency Room

Laboratory test (reference range)	Value
pH (7.35–7.45)	7.32
pCO ₂ (35–40 mm Hg)	31
HCO ₃ (20–30 mmol/L)	8
Anion gap (5–15 mmol/L)	22
Glucose (70–110 mg/dL)	211
ALT (<60 U/L)	395
Total Cholesterol (<170 mg/dL)	1014
LDL (<110 mg/dL)	732
Triglycerides (<90 mg/dL)	28040
Lipase (<400 U/L)	40

Abbreviations: ALT = alanine transaminase; HCO₃ = bicarbonate; LDL = low-density lipoprotein.

additional days to receive diabetes education and initiate insulin treatment and was discharged on hospital day 5. Diabetes auto-antibodies were positive for islet cell antigen 512, insulin, glutamic acid decarboxylase 65, and zinc transporter 8, confirming a diagnosis of type 1 diabetes. Hypertriglyceridemia did not recur during 19 months of follow-up.

Discussion

The exact mechanisms involved in the triad of DKA, SHTG, and acute pancreatitis are not entirely understood. As observed in our patient, patients with DKA have abnormal glucose and lipid metabolism that can result in hypertriglyceridemia.⁵ Insulin deficiency prevents the synthesis of lipoprotein lipase, an enzyme that hydrolyzes triglycerides in lipoproteins into free fatty acids and glycerol.^{6–8} Therefore, during DKA, increased fat mobilization by the activation of lipolysis and inhibition of lipoprotein lipase leads to an increase in triglycerides.^{7,9} Insulin also promotes the storage of free fatty acids in adipocytes, preventing high concentrations of potentially toxic free fatty acids in the plasma. Acute pancreatitis secondary to SHTG may be explained by elevated concentrations of cell-damaging free fatty acids produced from the hydrolysis of triglycerides by pancreatic lipase.¹⁰

There are various primary causes of SHTG, including polygenic and monogenic disorders of hypertriglyceridemia. In our patient, there was strong support for insulin deficiency as the cause, given his hyperglycemia, a severely elevated β -hydroxybutyrate level, and a low bicarbonate level. However, his initial bicarbonate level did not correlate with the pH, leading to uncertainty about the presence of metabolic acidosis. Additionally, triglyceride levels reaching 40176 mg/dL due to uncontrolled type 1 diabetes are not commonly seen. Given the likelihood of DKA, treatment with insulin infusion and hydration was initiated. After rehydration, the arterial pH levels met the diagnostic criteria for DKA, and the insulin infusion was continued. When the triglyceride levels did not significantly improve, the insulin infusion was further increased. However, the triglyceride levels remained severely elevated, and the patient experienced worsening acute pancreatitis symptoms, prompting plasmapheresis.

There are no clear guidelines in the literature on managing the triad of DKA, SHTG, and acute pancreatitis in the pediatric population.¹¹ In patients with SHTG, immediate lowering of triglycerides is possible with an insulin infusion.⁶ Additionally, previous pediatric case reports on this clinical triad have noted reductions of triglyceride levels with IV fluids and insulin therapy alone (Table 2).^{12–20} However, a study by Richardson et al¹¹ noted that in pediatric patients with SHTG, diabetes, and/or DKA who received IV insulin, the average time for the level of triglycerides to decline to

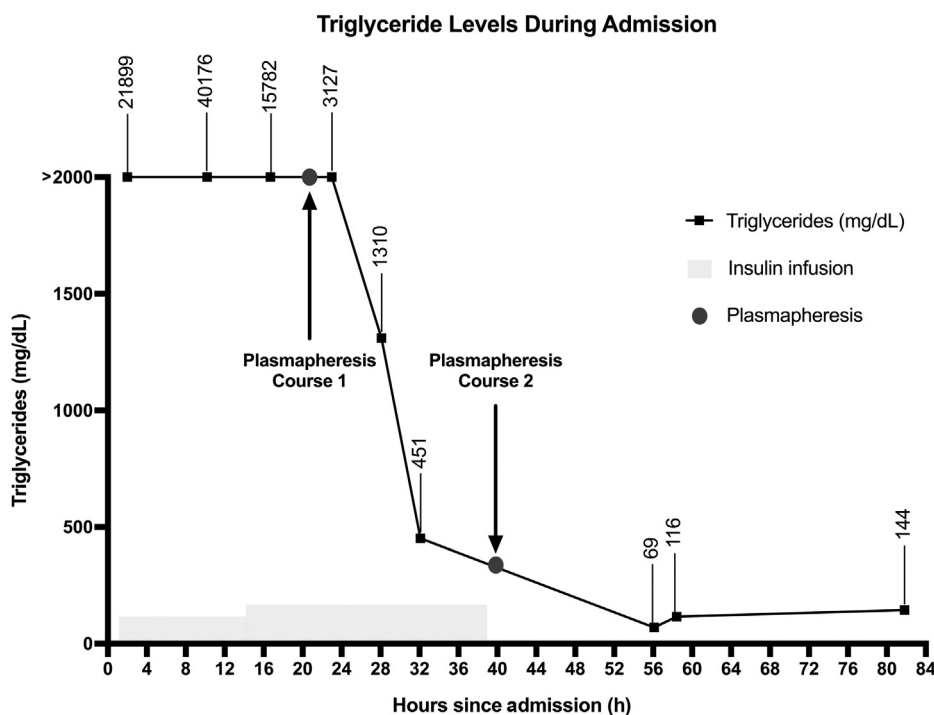


Fig. 1. Triglyceride levels (mg/dL) during the patient's hospital stay. The treatments that the patient received are also noted.

<1000 mg/dL was 3 days and to decline to <500 mg/dL was 23.8 days. The first reported case of plasmapheresis in a pediatric patient with DKA, SHTG, and acute pancreatitis was prompted by renal insufficiency, while the second was prompted by altered mental status.^{2,3} In our patient, the acute increase in severe abdominal pain that was unresponsive to narcotics and the lack of response to hydration and insulin led to the initiation of plasmapheresis, thereby avoiding risks of prolonged SHTG (ie, stroke, ischemia, and infection). The risks of plasmapheresis, including stroke, ischemia, and anaphylaxis, were also considered by the team.

To our knowledge, only 13 cases of pediatric DKA, hypertriglyceridemia, and pancreatitis have been reported to date (Table 2). As previously mentioned, treatment for pediatric patients with the triad of DKA, SHTG, and acute pancreatitis has focused on IV fluids and insulin therapy. However, 2 patients with this triad were successfully treated with plasmapheresis, resulting in triglyceride reduction and a significantly improved clinical status.^{2,3} The longest reported hospital stay for the triad was 20 days,²⁰ while the shortest stay was 5 days.¹² In our case, the early initiation of plasmapheresis stabilized the patient for discharge on day 3, preventing significant morbidity. The remaining 2 days in the hospital were devoted to diabetes education and initiating insulin therapy.

As alluded to previously, this case presented some challenges in the diagnosis of DKA. Severely elevated triglyceride levels may artificially elevate or lower other laboratory test results, necessitating cautious interpretation.^{21–23} The patient's initial arterial blood gas results did not strictly meet the diagnostic criteria for DKA. However, a metabolic panel revealed a low bicarbonate level, an elevated anion gap, and significantly elevated β -hydroxybutyrate and ketone levels, which were consistent with DKA. When interpreting laboratory results in the setting of SHTG, it is important to recognize that hypertriglyceridemia can falsely lower bicarbonate levels^{21–23} due to its space-occupying effect, in which the volume displaced by the high concentration of lipids decreases the aqueous phase of the sample. This interference can occur with

both enzymatic and indirect ion-selective electrode measurement methods.²² The discordance between pH and bicarbonate values presented an initial challenge in determining the severity of DKA and the safest course of treatment. In this case, the safest option was to treat the patient for presumed DKA.

SHTG can also result in pseudonormoglycemia when measuring blood glucose levels using a glucometer. As explained by the space-occupying effect, with triglycerides accounting for approximately one third of our patient's blood volume, this artificially decreased the blood sugar concentration detected by the patient's home glucometer. This could result in patients not checking urine for ketones when blood sugars are elevated and delays in seeking medical care, potentially delaying the diagnosis of DKA.²³ In the outpatient setting, practitioners should cautiously interpret glucose readings if a patient has suspected SHTG because the true glucose concentration is likely to be underestimated. Apart from the aforementioned tests impacted by hypertriglyceridemia, including glucose and bicarbonate, other spectrophotometric method-based tests may also be affected. In the context of this case report, tests such as aspartate aminotransferase and alanine transaminase may be severely affected, and results should be interpreted with caution. The space-occupying effect of SHTG will inevitably impact electrolyte readings using indirect ion-selective electrode methods, most significantly affecting sodium results, resulting in so-called pseudohyponatremia. Retesting using a direct method to measure electrolyte activity, such as a blood gas instrument, will provide accurate results and guide clinical IV fluid decisions.

Conclusion

When considering plasmapheresis in the clinical triad, hydration and insulin should be the first therapeutic steps because the resolution of DKA can generally improve SHTG.⁹ Based on our experience, once DKA resolves, it may be beneficial to initiate plasmapheresis to treat SHTG and acute pancreatitis, especially if

Table 2
Reported Pediatric Cases of Hypertriglyceridemia, Diabetic Ketoacidosis, and Pancreatitis, Including Patient Presentation, Treatment, and Length of Hospital Stay

Title (year)	PubMed ID	Age (years)	Sex	Clinical symptoms prior to treatment	Treatment	Plasmapheresis	Length of hospital stay
Acute pancreatitis induced by diabetic ketoacidosis with major hypertriglyceridemia: report of 4 cases (2020) ¹²	32313705	12	F	Nausea, vomiting, abdominal pain, polyuria, polydipsia, dehydration	IV fluid, insulin infusion	No	5 days
Acute pancreatitis induced by diabetic ketoacidosis with major hypertriglyceridemia: report of 4 cases (2020) ¹²	32313705	12	F	Abdominal pain, fever, decreased bowel sounds, polyuria, polydipsia, weight loss	IV fluid, insulin infusion, mechanical ventilation, vasopressors, broad-spectrum antibiotics	No	Patient died 12 days after ICU admission
Severe hypertriglyceridemia: a rare and harmful complication in diabetic ketoacidosis, treated successfully with plasmapheresis (2019) ³	N/A	14	F	Headaches, somnolence	IV fluid, insulin infusion	Yes	8 days
Diabetic ketoacidosis revealing a severe hypertriglyceridemia and acute pancreatitis in type 1 diabetes mellitus(2019) ¹³	30723557	14	M	Severe abdominal pain, vomiting, steatorrhea	IV fluid, insulin infusion, fenofibrate, broad-spectrum antibiotics, hydrocortisone	No	Not available, but patient was stabilized for 7 days and then transferred to another department
Hypertriglyceridemia with acute pancreatitis in pediatric diabetic ketoacidosis: a case report (2019) ¹⁴	30891384	16	F	Polyuria, polydipsia, weight loss, decreased energy, Kussmaul respirations, altered mental status responding only to painful stimuli, signs of poor perfusion, oral thrush, moderate to severe dehydration, xanthomas	IV fluid, IV insulin bolus, insulin infusion, sodium bicarbonate bolus	No	Not available
Severe hypertriglyceridemia causing pancreatitis in a child with new-onset type I diabetes mellitus presenting with diabetic ketoacidosis (2017) ⁷	28400692	4	F	Vomiting, abdominal pain, progressive breathing difficulty, lethargy, polyuria, polydipsia, 3 kg weight loss, dehydration	IV fluid, insulin injections	No	8 days
Acute pancreatitis and severe hypertriglyceridemia masking unsuspected underlying diabetic ketoacidosis (2013) ¹⁵	24005972	18	F	Anorexia, nausea, vomiting, left upper quadrant pain, abdominal cramps	IV fluid, insulin infusion, antiemetics, narcotic pain control, broad-spectrum antibiotics	No	8 days
Severe hypertriglyceridemia causing acute pancreatitis in a child with new-onset type I diabetes mellitus presenting in ketoacidosis (2013) ¹⁶	24455446	10	F	Kussmaul respirations, abdominal pain, nausea, vomiting, weakness, polydipsia, polyuria, nocturia, weight loss	IV fluid, insulin infusion (0.1-0.2 units/kg/h)	No	7 days
Plasmapheresis to treat hypertriglyceridemia in a child with diabetic ketoacidosis and pancreatitis (2012) ²	22201145	10	F	Abdominal pain, poor perfusion, shock	IV fluid, insulin infusion, fenofibrate	Yes	Not available
Treatment of severe hypertriglyceridemia with continuous insulin infusion (2011) ¹⁷	24804116	10	F	Moderate to severe dehydration, Kussmaul respirations, abdominal pain, anorexia, polydipsia, vomiting, hyperventilation	IV fluid, insulin infusion(0.5-1 IU/kg/h), nasogastric decompression, analgesia with meperidine and metamisole, bicarbonate, vasopressors	No	2 weeks
Clinical and/or biochemical pancreatitis in diabetic ketoacidosis (1994) ¹⁸	7820221	14	F	Disorientation, bilious vomiting, diarrhea, fluctuating level of consciousness, hypoactive bowel sounds, guaiac-positive vomit, decreased urine output, transient renal failure	Not available	No	Not available, but patient kept with nothing by mouth until day 11 of hospitalization
Diabetic lipemia in childhood diabetic ketoacidosis: a clue to coexisting acute pancreatitis (1980) ¹⁹	6778677	11	M	Kussmaul respirations, lipemia retinalis, periumbilical tenderness, lethargy, polydipsia, polyuria, polyphagia	IV fluid, insulin infusion, nasogastric decompression	No	18 days
Juvenile diabetes mellitus associated with acute pancreatitis (1965) ²⁰	5832154	12	F	Anorexia, weakness, moderately dehydrated, semiconscious, Kussmaul respirations, abdominal tenderness	IV fluid, regular insulin, antispasmodic medications, penicillin, streptomycin, blood transfusion	No	20 days

Abbreviations: F = female; ICU = intensive care unit; IV = intravenous; M = male, N/A = not applicable.

the clinical status deteriorates. In our case, therapeutic plasma exchange was initiated early and appropriately in the course of acute pancreatitis, which led to faster resolution of symptoms, advancement of diet, and early discharge.²⁴ The best candidates for plasmapheresis may be those with severe acute pancreatitis, triglyceride levels ≥ 1000 mg/dL after initial resuscitation with IV fluids, and those with signs of organ failure.²⁵ The ideal timing for initiation of apheresis is within 24 to 96 hours after the onset of symptoms and after glycemic control measures have been initiated. With limited data on its use, pediatric randomized controlled trials are needed to determine if plasmapheresis is an effective and safe tool in the management of SHTG, DKA, and pancreatitis.

Ethics Approval and Consent to Participate

Ethics approval and consent were not required by our institutional review board to publish this case report.

Consent for Publication

The case report was discussed with the patient's mother, and all questions were answered. Verbal informed consent to publish was given to the corresponding author by the patient's mother.

Availability of Data and Material

All data presented in the study are available and can be verified through the patient's electronic medical record.

Disclosure

The authors have no multiplicity of interest to disclose.

Acknowledgments

This manuscript was supported by the National Institutes of Health Grant K12 DK094714. The content is solely the responsibility of the authors and does not necessarily represent the official view of the NIH.

Author Contributions

L.M.N. and V.B. conceived and presented the idea. A.M.K., P.S., V.B., and L.M.N. performed an extensive literature search. L.M.N. collected the patient data. A.M.K. and L.M.N. drafted the manuscript and designed the figure. R.Z.S. reviewed and edited the manuscript. All authors approved the manuscript for publication.

References

1. Wolfsdorf JJ, Glaser N, Agus M, et al. ISPAD Clinical Practice Consensus Guidelines 2018: diabetic ketoacidosis and the hyperglycemic hyperosmolar state. *Pediatr Diabetes*. 2018;19(suppl 27):155–177.
2. Lutfi R, Huang J, Wong HR. Plasmapheresis to treat hypertriglyceridemia in a child with diabetic ketoacidosis and pancreatitis. *Pediatrics*. 2012;129(1):e195–e198.
3. Lee JM, Razavi A, Le AV, Williams R. Severe hypertriglyceridemia: a rare and harmful complication in diabetic ketoacidosis, treated successfully with plasmapheresis. *Pediatrics*. 2019;144(2 MeetingAbstract):390.
4. Schwartz J, Padmanabhan A, Aqai N, et al. Guidelines on the use of therapeutic apheresis in clinical practice—evidence-based approach from the Writing Committee of the American Society for Apheresis: the Seventh Special Issue. *J Clin Apher*. 2016;31(3):149–162.
5. Simons-Linares CR, Jang S, Sanaka M, et al. The triad of diabetes ketoacidosis, hypertriglyceridemia and acute pancreatitis. How does it affect mortality and morbidity?: A 10-year analysis of the National Inpatient Sample. *Medicine (Baltimore)*. 2019;98(7), e14378.
6. Poonuru S, Pathak SR, Vats HS, Pathak RD. Rapid reduction of severely elevated serum triglycerides with insulin infusion, gemfibrozil and niacin. *Clin Med Res*. 2011;9(1):38–41.
7. Sharma PK, Kumar M, Yadav DK. Severe hypertriglyceridemia causing pancreatitis in a child with new-onset type-I diabetes mellitus presenting with diabetic ketoacidosis. *Indian J Crit Care Med*. 2017;21(3):176–178.
8. Hahn SJ, Park J-H, Lee JH, Lee JK, Kim K-A. Severe hypertriglyceridemia in diabetic ketoacidosis accompanied by acute pancreatitis: case report. *J Korean Med Sci*. 2010;25(9):1375–1378.
9. Donelli D, Morini L, Trenti C, Santi R, Arioli D, Negri EA. Plasma exchange for the treatment of transient extreme hypertriglyceridemia associated with diabetic ketoacidosis and acute pancreatitis. *Eur J Case Rep Intern Med*. 2018;5(5), 000853.
10. Yang F, Wang Y, Sternfeld L, et al. The role of free fatty acids, pancreatic lipase and Ca⁺ signalling in injury of isolated acinar cells and pancreatitis model in lipoprotein lipase-deficient mice. *Acta Physiol (Oxf)*. 2009;195(1):13–28.
11. Richardson T, Aslibekyan S, Ashraf AP. Clinical characteristics and sequelae of severe hypertriglyceridemia in pediatrics. *Endocr Pract*. 2018;24(9):789–795.
12. Bouchaala K, Bahloul M, Bradii S, Kallel H, Chtara K, Bouaziz M. Acute pancreatitis induced by diabetic ketoacidosis with major hypertriglyceridemia: report of four cases. *Case Rep Crit Care*. 2020;2020:7653730.
13. Zaher FZ, Boubagura I, Rafi S, Elmghari G, Elansari N. Diabetic ketoacidosis revealing a severe hypertriglyceridemia and acute pancreatitis in type 1 diabetes mellitus. *Case Rep Endocrinol*. 2019;2019:8974619.
14. Yagnik PJ, Desai PH, Modem VM. Hypertriglyceridemia with acute pancreatitis in pediatric diabetic ketoacidosis: a case report. *Cureus*. 2019;11(1), e3844.
15. Aboulhosn K, Arnason T. Acute pancreatitis and severe hypertriglyceridaemia masking unsuspected underlying diabetic ketoacidosis. *BMJ Case Rep*. 2013;2013, bcr2013200431.
16. Wolfgram PM, Macdonald MJ. Severe hypertriglyceridemia causing acute pancreatitis in a child with new onset type I diabetes mellitus presenting in ketoacidosis. *J Pediatr Intensive Care*. 2013;2(2):77–80.
17. Rodríguez Santana Y, Nimo Román A, García Sáez I, López Alvarez JM, Consuegra Llapur E, González Jorge R. Treatment of severe hypertriglyceridemia with continuous insulin infusion. *Case Rep Crit Care*. 2011;2011:293917.
18. Slyper AH, Wyatt DT, Brown CW. Clinical and/or biochemical pancreatitis in diabetic ketoacidosis. *J Pediatr Endocrinol*. 1994;7(3):261–264.
19. Winter RJ, Herr TJ, Stone NJ, Traisman HS. Diabetic lipemia in childhood diabetic ketoacidosis: a clue to coexisting acute pancreatitis. *Diabetes Care*. 1980;3(6):706–707.
20. Cywinski JS, Walker FA, White H, Traisman HS. Juvenile diabetes mellitus associated with acute pancreatitis. *Acta Paediatr Scand*. 1965;54(6):597–602.
21. Cartier JL, Look DC, Dunn JP. Pseudohypobicarbonatemia in a patient presenting with suspected diabetic ketoacidosis. *AAACE Clin Case Rep*. 2018;4(2):e108–e111.
22. Rifkin SI, Shaub B. Factitious hypobicarbonatemia associated with profound hyperlipidemia. *Ren Fail*. 2014;36(7):1155–1157.
23. Rumbak MJ, Hughes TA, Kitabchi AE. Pseudonormoglycemia in diabetic ketoacidosis with elevated triglycerides. *Am J Emerg Med*. 1991;9(1):61–63.
24. Zhang KY, Cox KL, Sellers ZM. Plasmapheresis for hypertriglyceridemia-induced acute pancreatitis in a child: a case report and brief review of the literature. *Pancreas*. 2017;46(7):e58–e59.
25. Click B, Ketchum AM, Turner R, Whitcomb DC, Papachristou GI, Yadav D. The role of apheresis in hypertriglyceridemia-induced acute pancreatitis: a systematic review. *Pancreatol*. 2015;15(4):313–320.