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# Acute pancreatitis concomitant with diabetic ketoacidosis: a cohort from South China

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## Abstract

**Objective:** To evaluate the clinical characteristics of acute pancreatitis (AP) concomitant with diabetic ketoacidosis (DKA) in a cohort from South China and identify factors associated with early detection of DKA in AP patients.

**Methods:** Inpatient medical records of AP concomitant with DKA were retrospectively reviewed.

**Results:** Forty-eight patients with AP concomitant with DKA were enrolled in this study. The results indicated that comorbidity history of diabetes mellitus and mental status of not alert on admission were factors associated with DKA in AP patients. Compared with patients without DKA, patients with DKA showed significantly higher rates of hypertriglyceridemia and lower rates of gallstones than those without DKA. AP patients with concurrent DKA had higher levels of serum triglycerides, longer lengths of hospital stays, and higher complication rates of systemic inflammatory response syndrome and acute kidney injury.

**Conclusion:** AP patients might have higher risks of concomitant DKA if presenting as not alert upon admission or if they have past medical histories of diabetes mellitus. Serum triglyceride levels were significantly higher in AP patients with DKA. DKA raised the severity of AP, but did not increase in-hospital mortality.

## Keywords

Acute pancreatitis, diabetic ketoacidosis, risk factors, diabetes mellitus, serum triglycerides, hypertriglyceridemia

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## Introduction

Acute pancreatitis (AP) and diabetic ketoacidosis (DKA) are critical conditions that require early recognition and treatment in the emergency department. It has been infrequently reported in prior literature that DKA can coexist with AP.[1–5] AP is present in approximately 11% of DKA patients,[6] while the DKA prevalence in AP patients is approximately 1.2% to 26.4%.[7] A significantly higher mortality rate was found in patients with concomitant DKA and AP compared with AP-only patients.[7] Timely diagnosis and management in these cases is expected to contribute to better outcomes.

Abdominal pain is a classical presenting symptom in AP patients, which may mask the coexisting DKA, as >40% of DKA patients present with abdominal pain on admission.[8] If DKA was overlooked, resolving acidosis and dehydration could be further delayed, resulting in severe adverse outcomes. Although the occurrence of AP concomitant with DKA is uncommon in emergency visits, it could be easily overlooked due to the similarity of their clinical presentations. This presents great challenges for emergency physicians. Therefore, this study focused on analyzing the risk factors for the co-existence of DKA in AP patients to facilitate early identification. Morbidity and mortality in these patients were also investigated and compared with AP-only patients.

## Materials and methods

### Cohort subjects

We retrospectively reviewed the inpatient medical records of patients who were hospitalized in the First Affiliated Hospital of Sun Yat-Sen University in South China from January 2003 to December 2018. In total, there were 2476 consecutive patients diagnosed with AP over this time period who fulfilled at least two of the following three diagnostic features:[9] (i) abdominal pain characteristic of AP; (ii) serum lipase or amylase levels that were at least three times the upper limit of the normal range; and (iii) characteristic findings of acute pancreatitis on cross-sectional imaging (computed tomography or magnetic resonance imaging) or transabdominal ultrasonography. DKA diagnoses were made according to standard criteria.[10] Patients who were <18 years old, pregnant, or without complete data profiles (as outlined in Data Collection) were excluded. Finally, among the AP patients, 48 with a simultaneous diagnosis of DKA were enrolled as eligible patients in this study.

To adjust for potential cohort imbalances, propensity score matching (PSM) was performed on the AP with DKA and AP without DKA cohorts at a ratio of 1:1.25 using age and gender. Another 60 age- and gender-matched AP patients without DKA were randomly selected as the control group. Clinical files of these 108 patients were reviewed, and data were extracted.

The Atlanta criterion was used to classify the severity and types of AP.[9] The severity of AP was divided into three grades: mild acute pancreatitis (MAP), moderately severe acute pancreatitis (MSAP), and severe acute pancreatitis (SAP). SAP was defined as persistent organ failure for more than 48 hours. Additionally, AP was subdivided into two types: interstitial edematous and necrotizing AP. Interstitial edematous pancreatitis was defined as diffuse (or occasionally localized) enlargement of the pancreas due to inflammatory edema, while necrotizing pancreatitis was defined as necrosis of the pancreatic parenchyma, the peripancreatic tissue, or both.

### Data collection

We documented demographic information (age and sex), body mass index (BMI),

history of comorbidities (diabetes mellitus, hypertension, hyperlipemia, chronic obstructive pulmonary disease, and coronary atherosclerotic heart disease), and etiologies of AP (alcohol consumption, gallstones, hypertriglyceridemia [HTG], autoimmune, and medications). The diagnosis of hypertriglyceridemic AP was considered when AP was found with HTG (>5.67 mmol/L) in the absence of other causative factors of AP.[11] All laboratory test results were collected. The definition of hyperlipidemia (HLP) was based on the Chinese Joint Committee for Developing Chinese Guidelines on Prevention and Treatment of Dyslipidemia in Adults (JCDCG) guidelines.[12] According to the JCDCG definition, hyperlipidemia included hypercholesteremia (total cholesterol ≥5.2 mmol/L), HTG (serum triglycerides ≥1.7 mmol/L), high LDL cholesterol (≥3.4 mmol/L), and mixed hyperlipidemia. Clinical symptoms including the presence or absence of abdominal pain, body temperature, and mental status on admission were also taken from medical records. We collected information regarding the clinical course of AP including hospitalization course and complications.

### *Statistical analysis*

Statistical analyses were performed using IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, NY, USA). SPSS propensity score matching was applied, and logistic regression analysis was performed using nearest neighbor matching. The ratio for matching was 1:1.25 using a caliper of width equal to 0.2 of the standard deviation of the logit of the propensity score. Continuous variables are described as mean ± standard deviation (SD) or median (interquartile range) for variables with skewed distribution and were compared with the Student's t-test or the Mann–Whitney U test where appropriate. Categorical variables are expressed as percentages (%) and were compared with the Pearson $\chi^2$ test, continuity-adjusted chi-square test, or Fisher's exact test. Variables with $P < 0.05$ in prior comparisons between the two groups were adjusted by multivariate logistic regression analyses to evaluate associated factors that contributed to the coexistence of AP with DKA. Odds ratios (ORs) and corresponding 95% confidence intervals (CIs) of the variables were calculated. Results were considered significant if they achieve a *P*-value of <0.05.

## Results

Fifty-two of the 2476 AP patients were diagnosed with AP concomitant with DKA over the past 15 years, with an overall prevalence of 2.10%. Among them, one patient was 14 years old, one was pregnant, and two others had incomplete data profiles; these cases were excluded. Therefore, 48 AP patients with concomitant DKA were enrolled in this study.

### *Comparison of demographic and clinical features of AP patients with and without DKA upon admission*

A comparison of the demographic and clinical characteristics upon admission between patients with and without DKA is shown in Table 1. In the DKA group, 19 patients (39.58%) were female; the mean age of AP diagnosis with DKA was 42.52 years, ranging from 20 to 87 years; the mean BMI was 22.58 kg/m$^2$, and seven (14.58%) patients were heavy smokers with histories of 7 to 32 years. No significant difference was found regarding age, gender, BMI, tobacco use, or alcohol consumption compared with the non-DKA group.

Compared with the patients without DKA, patients with DKA showed significantly higher rates of diabetes mellitus history (87.50% vs. 15.00%, $p < 0.01$) and

**Table 1.** Comparison of demographic and clinical features upon admission of AP patients with and without DKA.

| | DKA* (n = 48) | non-DKA* (n = 60) | *P* value |
|---|---|---|---|
| Female | 19 (39.58) | 23 (38.33) | 0.90 |
| Age (years) | 42.52 ± 15.29 | 47.81 ± 15.57 | 0.08 |
| BMI (kg/m$^2$) | 22.58 ± 5.84 | 23.61 ± 2.61 | 0.32 |
| Tobacco abuse | 15 (31.25) | 18 (30.00) | 0.87 |
| Alcohol consumption | 12 (25.0) | 18 (30.0) | 0.79 |
| History of comorbidities | | | |
| Diabetes Mellitus | 42 (87.50) | 9 (15.0) | <0.01 |
| Hyperlipidemia | 35 (72.92) | 21 (35.0) | <0.01 |
| Hypertension | 15 (31.25) | 14 (23.33) | 0.36 |
| COPD | 3 (6.25) | 4 (6.67) | 1.0 |
| CAD | 10 (20.83) | 7 (11.67) | 0.19 |
| Manifestations upon admission | | | |
| Abdominal pain | 38 (79.17) | 55 (91.67) | 0.06 |
| Fever | 11 (22.92) | 7 (11.67) | 0.12 |
| Not alert | 17 (35.42) | 4 (6.67) | <0.01 |

*Data are presented as the mean ± SD or as the number of patients (%). BMI: body mass index.
AP, acute pancreatitis; DKA, diabetic ketoacidosis; BMI, body mass index; COPD, chronic obstructive pulmonary disease; CAD, coronary artery disease.

hyperlipidemia history (72.92% vs. 35.00%, $p < 0.01$). Other comorbidities were not significantly different between these patients. Patients' mental status were categorized into alert, stupor, and drowsy, the numbers in each category were 31 (64.58%), 13 (27.08%), and 4 (8.33%), respectively. It was noted that more patients were not alert (35.42% vs. 6.67%, $p < 0.01$) in the DKA group compared with the non-DKA group. The clinical manifestations on admission were nonspecific between groups when compared by abdominal pain (79.17% vs. 91.67%,) and fever (22.92% vs. 11.67%).

## *Multivariate analysis of risk factors for the coexistence of AP with DKA*

Table 2 shows the factors associated with the coexistence of AP and DKA upon admission. When biochemical parameters are absent upon admission, it is a challenge for emergency physicians to be aware of this complex clinical case and prescribe related tests. In our study, we aimed to recognize the patients at high risk from past medical histories and/or physical examinations. We found that the coexistence of AP with DKA was more likely to occur in patients with comorbidity histories of diabetes mellitus (adjusted OR: 55.128, 95% CI: 10.469–290.306, $p < 0.01$) and in those who presented as not alert (adjusted OR: 32.547, 95% CI: 4.701–225.322, $p < 0.01$). However, a history of hyperlipidemia was not significantly associated.

## *Etiology characteristics of AP patients with and without DKA*

The etiologies of AP are shown in Table 3. Compared with patients without DKA, patients with DKA showed significantly higher rates of HTG (66.67% vs. 28.33%, $p < 0.01$) and lower rates of gallstones (10.42% vs. 45.00%, $p < 0.01$). No significant differences were found among other common causes of AP between these patients.

**Table 2.** Adjusted odds ratios of variables associated with AP concomitant with DKA by multivariate analysis.

| | $\beta$ | S.E. | Wald | P value | Adjusted OR (95%CI) |
|---|---|---|---|---|---|
| Diabetes Mellitus | 4.010 | 0.848 | 22.378 | <0.01 | 55.128 (10.469–290.306) |
| Hyperlipidemia | 0.909 | 0.781 | 1.354 | 0.245 | 2.481 (0.537–11.460) |
| Not alert | 3.483 | 0.987 | 12.446 | <0.01 | 32.547 (4.701–225.322) |

AP, acute pancreatitis; DKA, diabetic ketoacidosis.

**Table 3.** Comparison of the etiologies of AP in patients with and without DKA.

| | DKA* (n = 48) | non-DKA* (n = 60) | P value |
|---|---|---|---|
| Gallstones | 5 (10.42) | 27 (45.0) | <0.01 |
| Alcohol consumption | 7 (14.58) | 9 (15.0) | 0.95 |
| Hypertriglyceridemia | 32 (66.67) | 17 (28.33) | <0.01 |
| Idiopathic | 3 (6.25) | 7 (14.58) | 0.53 |
| Medications | 1 (2.08) | 0 (0) | 0.91 |

*Data are presented as the number of patients (%).
AP, acute pancreatitis; DKA, diabetic ketoacidosis.

## *Comparison of clinical course and laboratory findings of AP patients with and without DKA*

A comparison of clinical events and laboratory results of patients with and without DKA is shown in Table 4. With regard to the classification of AP in the DKA group, 17 (35.42%) patients were SAP, 15 (31.25%) were MSAP, and 16 (33.33%) were MAP. More patients were SAP compared with the non-DKA group (35.42% vs. 13.33%, $p < 0.01$). However, no significant difference was found regarding necrotizing AP (22.92% vs. 10.00%).

In the DKA group, the causes of death for the two patients were multiple organ dysfunction syndrome and severe sepsis, respectively. The inpatient mortality rate was comparable with the non-DKA group (4.17% vs. 5.0%). There was also no significant difference in the intensive care unit (ICU) admission rate, duration of ICU, or the rates of hospital readmission at 30 days after discharge. However, AP patients with concurrent DKA were more likely have longer hospital stays ($13.44 \pm 6.25$ days vs. $7.02 \pm 3.33$ days, $p < 0.01$).

Regarding complications, patients with concomitant AP and DKA were more likely to have systemic inflammatory response syndrome (SIRS) (60.42% vs. 15.00%, $p < 0.01$) and acute kidney injury (AKI) (29.17% vs. 8.33%, $p = 0.02$) on admission. However, there was no statistically significant difference with regard to concurrent infection or respiratory distress requiring intubation.

By comparing laboratory findings, more patients in the DKA group had HTG (66.67% vs. 18.33%, $p < 0.01$). Serum triglyceride levels in patients with DKA were significantly higher than in patients without DKA. Moreover, patients with concurrent DKA had lower serum levels of ionized calcium ($Ca^{2+}$, $p < 0.01$), carbon dioxide combining power ($CO_2CP$, $p < 0.01$), and higher levels of random glucose ($p < 0.01$), anion gap (AG, $p < 0.01$), blood urea nitrogen (BUN, $p = 0.04$), lactic dehydrogenase (LDH,

**Table 4.** Comparison of clinical course and laboratory findings in AP patients with and without DKA.

| | DKA* (n = 48) | non-DKA* (n = 60) | *P* value |
|---|---|---|---|
| Classification of AP | | | |
| SAP | 17 (35.42) | 8 (13.33) | <0.01 |
| Necrotizing AP | 11 (22.92) | 6 (10.00) | 0.07 |
| Hospitalization course | | | |
| LOS (days) | 13.44 ± 6.25 | 7.02 ± 3.33 | <0.01 |
| ICU admission | 7 (14.58) | 6 (10.00) | 0.47 |
| ICU duration (days) | 3.50 (2.00–5.75) | 5.00 (4.00–6.50) | 0.29 |
| In-hospital mortality | 2 (4.17) | 3 (5.00) | 0.76 |
| Hospitalization 30 days after discharge, n (%) | 2 (4.17) | 1 (1.67) | 0.69 |
| Complications on admission | | | |
| SIRS | 29 (60.42) | 9 (15.00) | <0.01 |
| Concurrent infection | 15 (31.25) | 24 (40.0) | 0.35 |
| AKI | 14 (29.17) | 5 (8.33) | 0.02 |
| Respiratory distress requiring intubation | 4 (8.33) | 3 (5.00) | 0.56 |
| Laboratory findings | | | |
| HTG | 32 (66.67) | 11 (18.33) | <0.01 |
| Serum triglycerides (mmol/L) | 7.10 (2.65–12.82) | 2.10 (1.58–6.72) | <0.01 |
| Serum amylase (U/L) | 384.50 (227.0–623.0) | 428.0 (188.20–1162.75) | 0.06 |
| Serum lipase (U/L) | 973.0 (680.0–1684.50) | 1659.0 (517.13–3214.75) | 0.08 |
| $Ca^{2+}$ (mmol/L) | 2.08 (1.93–2.18) | 2.28 (2.14–2.46) | <0.01 |
| Random glucose (mmol/L) | 18.65 (13.70–22.80) | 6.95 (5.60–8.10) | <0.01 |
| $CO_2CP$ (mmol/L) | 11.0 (7.0–15.0) | 24.0 (22.0–25.0) | <0.01 |
| AG (mmol/L) | 25.89 ± 8.23 | 15.34 ± 3.94 | <0.01 |
| WBC ($\times 10^9$/L) | 12.79 (9.67–17.52) | 11.83 (10.15–14.96) | 0.23 |
| BUN (mmol/L) | 7.90 (6.00–13.29) | 5.00 (3.25–7.20) | 0.04 |
| Creatine(μmol/L) | 108.50 (72.0–147.0) | 72.0 (55.0–88.75) | 0.05 |
| LDH (U/L) | 492.50 (342.0–519.0) | 307.0 (225.0–410.0) | <0.01 |
| ALT (U/L) | 85.0 (33.0–200.0) | 52.50 (27.0–73.0) | <0.01 |
| TBIL (μmol/L) | 33.77 ± 21.31 | 18.60 ± 8.12 | 0.02 |
| PCT (ng/ml) | 3.30 (0.61–4.22) | 2.30 (1.07–3.93) | 0.41 |
| NT-proBNP (pg/mL) | 577.40 (174.85–1468.00) | 407.40 (267.80–577.40) | 0.23 |
| hsTnT (ng/ml) | 0.01 (0.00–0.07) | 0.00 (0.00–0.06) | 0.43 |

*Data are presented as the mean ± SD, the number of patients (%), or median(interquartile range). AP, acute pancreatitis; DKA, diabetic ketoacidosis; SAP, severe acute pancreatitis; LOS, length of stay; ICU, intensive care unit; SIRS, systemic inflammatory response syndrome; AKI, acute kidney injury; HTG, hypertriglyceridemia; $Ca^{2+}$, ionized calcium; $CO_2CP$, carbon dioxide combining power; AG, anion gap; WBC, white blood (cell) count; BUN, blood urea nitrogen; LDH, lactic dehydrogenase; ALT, alanine aminotransferase; TBIL, total bilirubin; PCT, procalcitonin; NT-proBNP, N-terminal B-type natriuretic peptide; hsTnT, high-sensitivity troponin T.

$p < 0.01$), alanine aminotransferase (ALT, $p < 0.01$), and total bilirubin (TBIL, $p = 0.02$).

## Discussion

In this single-center retrospective cohort study of 2476 AP patients, we found that DKA presented in 52 cases (2.10%), which was similar to a previous study.[7] The coexistence of AP and DKA has been recognized since 1969 and reported infrequently during the past 40 years.[1,3,4,13–18] Abdominal pain, a classical feature of AP, can be found in a number of patients with DKA, so the

diagnosis of DKA is often delayed when AP is the initial diagnosis on presentation.[1,3] Usually, discovering DKA in AP patients is based solely on associated laboratory findings (i.e., plasma glucose, serum AG and bicarbonate, presence of urine ketones). Delayed recognition of DKA may be detrimental to AP patients because such patients require more aggressive volume replacement and strict control of hyperglycemia.[6]

It is challenging for emergency physicians to realize these concurrent cases and prescribe related tests to identify them early. By comparing the demographic and clinical presentations on admission between the DKA and non-DKA groups, we found that more patients in the DKA group presented as not alert and had medical histories of diabetes mellitus or hyperlipidemia. The differences in diabetes mellitus and mental status of not alert were also significant in the multivariate analysis, indicating that they were strong risk factors for the existence of DKA in AP patients. This prompted us to wonder if emergency physicians should screen for DKA when AP was the initial diagnosis on presentation and the patients presented as not alert or had past medical histories of diabetes mellitus.

DKA is a life-threatening condition that requires management in the ICU in most cases. In our study, we found that the presence of DKA increased the severity of AP as there were more SAP cases compared with the non-DKA group ($p = 0.01$). Additionally, DKA raised the Ranson and APACHE II scores of AP patients,[19] which demonstrated that the DKA group had higher levels of random glucose, AG, BUN, LDH, ALT, and TBIL, but lower levels of $Ca^{2+}$ and $CO_2CP$ on admission. Patients with concurrent DKA had more severe organ dysfunction and acid-base disturbance, which were in accordance with the data from Quintanilla-Flores et al.[20] Additionally, the existence of AP aggravated the severity of DKA due to marked acidosis, hyperglycemia, and increased depletion of intravascular volume.[6] Simons-Linares et al.[7] recently performed the largest cohort study to date on the coexistence of AP and DKA, in which 2.8 million patients were hospitalized with AP and 33,356 had concomitant DKA. They found that patients with concomitant AP and DKA had higher complication rates of AKI, SIRS, shock, acute respiratory distress syndrome, sepsis, and ileus, which resulted in longer hospital stays and higher inpatient mortality rates compared with AP-only patients. However, in our study, although we found that patients in the DKA group had higher complication rates of SIRS and AKI on admission, the inpatient mortality rates were similar to patients in the non-DKA group, as were the ICU admission rates and ICU durations. Notably, patients in the DKA group still had longer hospital stays, which indicated a more critical condition.

Compared with patients without DKA, AP patients with DKA showed significantly higher rates of an etiology of HTG. The role of HTG in the coexistence of AP and DKA has already been discussed in prior studies.[6,13,19] It is believed that the link between DKA and AP is usually mediated through HTG, but whether DKA is the cause of AP (DKA→HTG→AP) or a complication of AP (HTG→AP→DKA) is still unclear.[7] Furthermore, AP may induce DKA without HTG, as AP can destroy pancreatic beta cells, resulting in a transient insulin deficiency that triggers DKA.[3] In this study, we noticed that serum triglyceride levels were significantly higher in patients with DKA than in those without DKA. This indicated that HTG might play an important role in the pathogenesis of these cases.

Our study had limitations. First, a major limitation was the small sample size. Some of the factors that might be expected to predict the existence of DKA in AP could not be measured in the multivariate analysis.

Moreover, as discussed above, only two in-hospital deaths were documented in the DKA group, and no significant difference in in-hospital mortality rates was found between the two groups. This might also result from the limited number of patients enrolled in this cohort. Second, as a retrospective study, we were not able to control all the variables studied or the treatment adjustments; therefore, some variables were not compared in the study. A future prospective study with a larger sample size is necessary to confirm our findings and further explore the risk factors and prognosis for the coexistence of AP with DKA.

In summary, we discovered that AP patients might have higher risks of concomitant DKA if they present as not alert upon admission or had past medical histories of diabetes mellitus. Emergency physicians should screen for the existence of DKA if AP is suspected in such patients. Serum triglyceride levels were significantly higher in AP patients with DKA. DKA raised the severity of AP, but did not increase in-hospital mortality rates.

## Declaration of conflicting interest

The authors declare that there is no conflict of interest.

## Ethical approval

No ethical approval was needed for this retrospective study. Data were obtained with permission from the medical director offices of the respective centers.

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## Informed consent

The Ethical Committee of the participating centers remitted that the data could be collected without contacting the patients.

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## References

1. Aboulhosn K and Arnason T. Acute pancreatitis and severe hypertriglyceridaemia masking unsuspected underlying diabetic ketoacidosis. *BMJ Case Rep* 2013; 2013: pii: bcr2013200431.
2. Fujishiro M, Horita A, Nakagawara H, et al. Severe hypertriglyceridemia possibly masked acute pancreatitis and led to a difficult diagnosis in an obese patient with ketoacidosis-onset type 2 diabetes. *Intern Med* 2017; 56: 2611–2616.
3. Kim JH and Oh MJ. Acute pancreatitis complicated with diabetic ketoacidosis in a young adult without hypertriglyceridemia: a case report. *Korean J Gastroenterol* 2016; 68: 274–278.
4. Singla AA, Ting F and Singla A. Acute pancreatitis secondary to diabetic ketoacidosis induced hypertriglyceridemia in a young adult with undiagnosed type 2 diabetes. *JOP* 2015; 16: 201–204.
5. Zaher FZ, Boubagura I, Rafi S, et al. Diabetic ketoacidosis revealing a severe hypertriglyceridemia and acute pancreatitis in type 1 diabetes mellitus. *Case Rep Endocrinol* 2019; 2019: 8974619.
6. Nair S, Yadav D and Pitchumoni CS. Association of diabetic ketoacidosis and acute pancreatitis: observations in 100 consecutive episodes of DKA. *Am J Gastroenterol* 2000; 95: 2795–2800.
7. 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: e14378.

8. Pant N, Kadaria D, Murillo LC, et al. Abdominal pathology in patients with diabetes ketoacidosis. *Am J Med Sci* 2012; 344: 341–344.
9. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis–2012: revision of the Atlanta classification and definitions by international consensus. *Gut* 2013; 62: 102–111.
10. Kitabchi AE, Umpierrez GE, Murphy MB, et al. Hyperglycemic crises in adult patients with diabetes: a consensus statement from the American Diabetes Association. *Diabetes Care* 2006; 29: 2739–2748.
11. Carr RA, Rejowski BJ, Cote GA, et al. Systematic review of hypertriglyceridemia-induced acute pancreatitis: a more virulent etiology? *Pancreatology* 2016; 16: 469–476.
12. Joint Committee for Developing Chinese guidelines on Prevention and Treatment of Dyslipidemia in Adults. Chinese guidelines on prevention and treatment of dyslipidemia in adults. *Zhonghua Xin Xue Guan Bing Za Zhi* 2007; 35: 390–419.
13. Timilsina S, Timilsina S, Mandal A, et al. Triad of diabetic ketoacidosis, hypertriglyceridemia, and acute pancreatitis: severity of acute pancreatitis may correlate with the level of hypertriglyceridemia. *Cureus* 2019; 11: e4930.
14. Shinotsuka M and Tokuda Y. Milk pancreatitis with hypertriglyceridemia and diabetic ketoacidosis. *J Gen Fam Med* 2017; 18: 422–424.
15. Seth A, Rajpal S, Saigal T, et al. Diabetic ketoacidosis-induced hypertriglyceridemic acute pancreatitis treated with plasmapheresis-recipe for biochemical disaster management. *Clin Med Insights Gastroenterol* 2014; 7: 51–53.
16. Nair S and Pitchumoni CS. Diabetic ketoacidosis, hyperlipidemia, and acute pancreatitis: the enigmatic triangle. *Am J Gastroenterol* 1997; 92: 1560–1561.
17. Kumar P, Sakwariya A, Sultania AR, et al. Hypertriglyceridemia-induced acute pancreatitis with diabetic ketoacidosis: a rare presentation of type 1 diabetes mellitus. *J Lab Physicians* 2017; 9: 329–331.
18. Nielsen OS and Simonsen E. A case of transient diabetes mellitus in connection with acute pancreatitis. *Acta Med Scand* 1969; 185: 459–461.
19. Wang YC, Attar BM, Hinami K, et al. Concurrent diabetic ketoacidosis in hypertriglyceridemia-induced pancreatitis how does it affect the clinical course and severity scores? *Pancreas* 2017; 46: 1336–1340.
20. Quintanilla-Flores DL, Rendón-Ramírez EJ, Colunga-Pedraza PR, et al. Clinical course of diabetic ketoacidosis in hypertriglyceridemic pancreatitis. *Pancreas* 2015; 44: 615–618.