Pre-operative management of a patient with tubo-ovarian abscess and diabetic ketoacidosis

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ABSTRACT

Background: Diabetic ketoacidosis (DKA) is a serious consequence of uncontrolled diabetes, especially in patients complicated by certain infections. Patients with DKA often require surgery; however, at the same time are more vulnerable to unintended postoperative complications. Therefore, proper pre-operative management in these patients is prominent. This study aimed to summarize pre-operative management of a patient with DKA and tubo-ovarian abscess.

Case Presentation: A 45-year-old female was referred to Dr. Soetomo Hospital Surabaya with a chief complaint of fever, right abdominal pain, and white vaginal discharge. The patient had a history of uncontrolled DM since 2014. Ultrasonography examination revealed the presence of a hyperechoic mass in the uterine fundus (size: 6.39 x 7.36 cm). Initial laboratory examination indicated leukocytosis, hyperglycemia, hyponatremia, proteinuria, and ketoacidosis condition. The patient was diagnosed with DKA, type 2 diabetes mellitus (T2DM), tubo-ovarian abscess. Before undergoing surgery to remove the abscess, the patient received fluid therapy (NaCl 0.9%); insulin therapy (rapid-acting insulin 2x4 IU intravenous (IV), bicarbonate 100 mEq in 400 mL normal saline/24 hours IV, and rapid-acting insulin pump 1 IU/hour); diet therapy, and antibiotic therapy (ampicillin 1 gr every 6 hours IV, gentamycin 240 mg every 24 hours IV, and metronidazole 500 mg every 8 hours IV). Regular examination was performed, which included the evaluation of random blood glucose (RBG) every 3 hours, serum electrolytes every 24 hours, repeat blood gas analysis every 24 hours, blood culture, and vaginal swabs.

Conclusion: Euglycemia is the main goal of perioperative treatment in order to enhance postoperative results. Careful blood glucose management should be performed before surgery to decrease patients’ morbidity and mortality. It must also be highlighted that regular patient monitoring through clinical and laboratory markers is essential to treat DKA successfully.

Keywords: Diabetic ketoacidosis, tubo-ovarian abscess, hyperglycemia, metabolic acidosis, ketones.


INTRODUCTION

Diabetes mellitus (DM) is a significant problem and with 629 million people estimated to have DM by 2045.1 Uncontrolled hyperglycemia, metabolic acidosis, and an increased concentration of body ketone are considered the hallmarks of diabetic ketoacidosis (DKA). It is a serious consequence of diabetes, most frequently affecting people with type 1 diabetes mellitus (T1DM). In people with type-2 diabetes mellitus (T2DM), although rare, DKA may also occur. In fact, a study reported that DKA was found predominantly in T2DM patients as compared to T1DM in Indonesia.2 Hyperglycemia, dehydration, and acidity lead to relative or absolute insulin insufficiency, causing a DKA condition. An infection, a newly diagnosed diabetes, or noncompliance with therapy are the most frequent causes of this condition.3,4

Patients with DM have been reported to undergo surgery more frequently than those without DM, yet at the same time are vulnerable to complications that might occur during or postoperatively, as diabetes affects the microvascular and macrovascular systems. Furthermore, diabetic patients undergoing surgery are also at higher risk of having DKA, and those with already symptomatic of DKA are at a higher risk of developing adverse post-operative outcomes if their glycemic levels are not stabilized.5,6 Therefore, proper surgical treatments, especially pre-operatively, are prominent. To lower perioperative mortality and morbidity, diabetic patients and those at risk for prediabetes must have a thorough preoperative examination. An optimization of preoperative comorbidities is part of this preoperative examination. In order to prevent unintended hypoglycemia and hyperglycemic episodes during the perioperative phase, anti-diabetic medication regimes must also be optimized.7,8

Preoperative management of patients with DKA is often challenging in a clinical setting. Many factors, such as unpredictable fasting duration before surgery, improper administration of intravenous medication which is potentially hazardous, inappropriate antibiotic therapy to treat infection, and the stress response to surgery may result in fatal postoperative outcomes.9-13 Thus, proper pre-operative management for patients with DKA accompanied by infection is of critical importance. This study aimed to summarize pre-operative management of a patient with DKA and tubo-ovarian abscess.
CASE PRESENTATION

A 45-year-old female patient was referred from the Obstetrics and Gynecology Department with myoma uteri subserous, a tubo-ovarian abscess (TOA) dextra with differential diagnosis as endometrioma, intra-uterine device (IUD) in situ, unregulated T2DM and class I obesity (BMI 33.2 kg/m²). The main complaints were fever and weakness for the past one week. The patient also complained of right lower abdominal pain and white vaginal discharge during the same period of time. One day prior to admission to the hospital, the complaints worsened. However, cough, shortness of breath, painful urination, diarrhea, or sores on the body were denied. The patient had a history of DM since 2014, for which she received metformin 3x500 mg. However, the patient was found not compliant to medication and routine glycemic control.

Physical examination revealed a fair general condition, with a Glasgow Coma Scale (GCS) of E4V5M6. Vital signs showed blood pressure of 130/80 mmHg, heart rate of 90 x/minute, respiratory rate of 28x/minute, temperature of 38°C, and oxygen saturation of 99.0% (without oxygen supplementation). Body mass index (BMI) was 33.2 kg/m² (weight: 85 kg and height: 160 cm). The head and neck examinations exhibited no signs of anemia, icterus, cyanosis, or dyspnea. Heart examination suggested a normal heart sound (S1 and S2). No murmur or gallop was found. The lung examination indicated symmetrical chest movement without retraction and vesicular sound without rhonchi and wheezing. Abdominal examination indicated pain in right-lower abdomen and suprapubic regions. Vaginal/vulva toucher was fluxus (-) and flor albus (+). The extremities examination showed a warm, dry, and red acral without edema. The results of the electrocardiogram (ECG), chest x-ray, and abdomen x-ray were within normal limits, whereas ultrasonography examination revealed uterus anteflexed with 7.56 x 4.4 cm in size; hyperechoic mass 6.39 x 7.36 cm in fundus uterus; cystic masse in adnexa parametrium sinistra, internal echo positive, with size of 9.1 x 9.3 cm; and IUD in situ (Figure 1).

Figure 1. Ultrasonography examination revealed IUD in situ and left ovarian cysts.

Figure 2. Detailed of the findings during the surgery: (A) Uterine myoma and left ovarian cyst; (B) Multiple uterine myoma and IUD in situ; and (C) Tubo-ovarian abscess (TOA).
Table 1. The results of the patient laboratory test

<table>
<thead>
<tr>
<th>Lab Parameters (unit)</th>
<th>Results</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Initial test</td>
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<tr>
<td>Blood Analysis</td>
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<tr>
<td>Hemoglobin (g/dL)</td>
<td>14.1</td>
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<tr>
<td>Hematocrits (%)</td>
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<tr>
<td>Leukocytes (/µL)</td>
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<tr>
<td>Platelets (/µL)</td>
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<td>Neutrophils (%)</td>
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<td>Lymphocytes (%)</td>
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<tr>
<td>Neutrophil-to-lymphocyte Ratio (%)</td>
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</tr>
<tr>
<td>Partial thromboplastin time (seconds)</td>
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<tr>
<td>Activated Partial Thromboplastin Time (seconds)</td>
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<tr>
<td>C-Reactive Protein (mg/L)</td>
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<tr>
<td>Procalcitonin (ng/mL)</td>
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<tr>
<td>Blood Urea Nitrogen (mg/dL)</td>
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<tr>
<td>Serum Creatinine (mg/dL)</td>
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<td>Serum Glutamic Oxaloacetic Transaminase (U/L)</td>
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<td>Serum Glutamic Pyruvic Transaminase (U/L)</td>
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<tr>
<td>HbA1C (%)</td>
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<tr>
<td>Random Blood Glucose (mg/dL)</td>
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<tr>
<td>Albumin (g/dL)</td>
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<tr>
<td>Sodium (mmol/L)</td>
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<td>Potassium (mmol/L)</td>
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<td>+3</td>
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<tr>
<td>Leukocytes</td>
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<td>Nitrite</td>
<td>Negative</td>
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<tr>
<td>Urine glucose</td>
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</tr>
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<td>SO2 (%)</td>
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Initial laboratory examination (April 1, 2022) showed elevated neutrophil-lymphocyte ratio (NLR) (9.4%), C-reactive protein (CRP) (28.4 mg/L), and procalcitonin (2.42 ng/mL) levels, likely suggesting the presence of inflammation or infection. Increased levels of RBG (322.0 mg/dL), HbA1C (14.0%), and urine glucose (+4) were also recorded, justifying unregulated diabetes mellitus. Sodium level (131.0 mmol/L) in the blood indicated hyponatremia and urine protein test (+1) suggested proteinuria. Urine and blood gas analysis (BGA) also revealed the occurrence of ketoacidosis (urine ketones: +3; arterial pH: 7.2; pCO2: 15 mmHg; HCO3: 6.1%; base excess: -21.0 mmol/L) (Table 1).

Based on all the anamnesis, history taking, physical examinations, and laboratory findings, the patient was diagnosed with diabetic ketoacidosis, unregulated T2DM, class I obesity (BMI 33.2 kg/m²), myoma uteri subserous, TOA dextra with differential diagnose endometrioma and IUD in situ. The patient was then treated with NaCl 0.9% 2 liters/2 hours, continued with 80 drops per minute/4 hours, 30 drops per minute/18 hours, and 20 drops per minute/24 hours to replace dehydration losses; rapid-acting insulin 2x4 IU intravenous (IV), sodium bicarbonate 100 mEq in 400 mL normal saline/24 hours IV; rapid-acting insulin pump 1 IU/hour; ampicillin 1 gr every 6 hours IV; gentamycin 240 mg every 24 hours IV; metronidazole 500 mg every 8 hours IV. The target RBG level was 160–200 mg/dL in pre-, durante, and post-surgery.
Regular examination was performed if the patient's RBG levels were 160-200 mg/dL, which included the evaluation of RBG every 3 hours, serum electrolytes every 24 hours, repeat BGA every 24 hours, blood culture, and vaginal swab.

On the second day of treatment, the patient still complained of abdominal pain. Vital signs were within normal limits (Table 1) and the patient was in fair general condition (GCS of E4V5M6). Laboratory examinations indicated RBG 160 mg/dL and hyponatremia (sodium level: 131 mmol/L). DKA condition improved, whereas myoma uteri subserous, TOA dextra with differential diagnosis of endometrioma was still observed. The patient received an IV infusion of ringer-acetate 1500 mL/24 hours, rapid-acting insulin pump 1 IU/hour, IV sodium bicarbonate 100 mEq in 400 mL normal saline/24 hours, IV ampicillin 1 gr/6 hours, IV gentamicin 240 mg/24 hours, and IV metronidazole 500 mg/8 hours therapies. RBG level was evaluated every 3 hours, serum electrolytes every 24 hours, and repeat BGA every 24 hours. The patient then underwent surgery to remove the abscess on the tubo-ovary and revealed enlarged uterus with multiple myoma uteri subserous with size 5 x 6 cm; ovarian cyst on adnexa parametrium sinistra with 8x7 cm in size. The left salpingectomy was conducted and adherence grade III-IV was found at between adnexa parametrium dextra and sigmoid; leaking ovarian dextra cyst producing with 200cc pus. Adhesiolysis, salpingectomy dextra, and total abdominal hysterectomy were conducted. Detailed of the findings during the surgery are presented in Figure 2.

On the third day of treatment, the patient feels slight pain in the surgical wound. Vital signs indicated a slightly increased heart rate (107 x/minute) and SpO2 of 98% with a nasal cannula of 3 liters per minute (lpm). The patient was in a fair general condition with GCS of E4V5M6. Laboratory examination indicated leukocytosis, thrombocytosis, and hyperglycemia. The patient was diagnosed with T2DM post-DKA, TOA dextra, ovarian cyst sinistra, multiple myoma uteri, adhesion grade III-IV post-exploratory laparotomy. The treatments included ringer-acetated 1500 mL within 24 hours IV; rapid-acting insulin pump 1 IU/hour stop; subcutaneous levensir 0-0-10 IU; subcutaneous novorapid 6-6-6 IU; ampicillin 1 gr every 6 hours IV; gentamicin 240 mg every 24 hours IV; metronidazole 500 mg every 8 hours IV. Evaluation of RBG, postprandial blood glucose, and serum electrolytes was performed every 24 hours.

On the seventh day of treatment, the patient reported no complaints. Vital signs were within normal limits and the patient was in a fair general condition (GCS E4V5M6). Laboratory examinations revealed elevated platelets (586.000/μL) and CRP (12,6 mg/dL) levels. RBG level was 139 mg/dL. The patient was diagnosed with T2DM post-DKA and tube-ovarian abscess post-exploratory laparotomy. The patient received treatment with subcutaneous levermir 0-0-14 IU, subcutaneous novorapid 10-10-10 IU, and oral clindamycin 450 mg every 6 hours. The patient was planned to be discharged.

DISCUSSION

TOA, a complicated infected mass of the adnexa, develops as a result of pelvic inflammatory disease (PID). A TOA typically presents with an adnexal mass, fever, an increased white blood cell count, lower abdominal-pelvic pain, and/or vaginal discharge; however, the symptoms may also present in a wide range of ways. Any clinical concern for this diagnosis requires urgent evaluation and treatment as the abscess might rupture and cause a life-threatening sepsis.15-16 The patient in the current study mainly complained of fever and weakness, accompanied by the presence of white vaginal discharge. The patient admitted using intrauterine devices and had a history of uncontrolled T2DM since 2014.

Occasionally, the patient with diabetes may present with DKA. Uncontrolled hyperglycemia, metabolic acidosis, and an increment in body ketone concentration are the hallmarks of DKA. Hyperglycemia, dehydration, and acidity result in relative or absolute insulin insufficiency that worsen DKA condition. An infection, newly diagnosed diabetes, or noncompliance to therapy are the most frequent causes of the condition.17-20 In addition, patients with DKA presented with an elevated body temperature, tachycardia, tachypnea, or leukocytosis for which a predisposing infection state was documented.21 The patient in this study presented with fever, abdominal pain, hyperglycemia, leukocytosis, proteinuria, hyponatremia, and ketoacidosis (Table 1). The patient was diagnosed with DKA, T2DM, and TOA with differential diagnosis as an endometrioma.

Acidosis has been divided into two categories, namely metabolic acidosis, which is controlled by bicarbonate, and respiratory acidosis, which involves changes in carbon dioxide (HCO3).22-24 A decrease in serum HCO3 of less than 24 mEq/L and an increase in the hydrogen ion concentration in the systemic circulation are the hallmarks of metabolic acidosis. DKA can be categorized as mild, moderate, or severe depending on how severe the metabolic acidosis is and whether there are any signs of changed mental status.25 Based on the arterial pH status (7.2), the level of serum bicarbonate (6.1 mEq/L), the presence of urine ketone (+3), and mental status (GSC E4V5M6) (Table 1), the patient in the present study might be categorized into mild-moderate DKA.

Euglycemia is the main goal of perioperative treatment, which also aims to enhance postoperative results.26 Extended preoperative fasting should be avoided and careful blood glucose monitoring should be started and continued during surgery. Point-of-care diagnostics, venous phlebotomy, or analysis of blood gas samples can do this. Despite not meeting the requirements for glucose testing, capillary and arterial whole blood glucose can nevertheless be utilized to direct insulin administration in the operating room.27

Before undergoing surgery, patients with DKA need to have their DKA treated. Currently, the best management for DKA is using a fixed rate intravenous insulin infusion (FRIIII) as it promotes faster resolution of the DKA, and this therapy should be maintained until the ketosis has resolved. However, it is also salutary to note that DKA may mimic the surgical abdomen, and before performing emergency surgery in patients with diabetes, DKA must be excluded as the cause.
of the acute abdomen. Postoperatively, the patient should be managed in a critical care environment, and the FRIII therapy should be continued unless the ketosis state has been successfully treated. Once the ketosis is resolved, the FRIII can either be converted to the variable rate IV insulin infusion (VRIII) or the patient can resume their usual medication once they eat and drink normally.

Careful blood glucose management has been linked to a lower death rate in surgical patients and decreased perioperative problems. Frequent glucose monitoring should be used in insulin-using individuals to make sure that glucose readings are within normal ranges. In patients with hyperglycemic crises, the goals of therapies are to increase circulatory volume and tissue perfusion; gradually lower serum glucose and osmolality; correct electrolyte imbalance; and identify co-morbid precipitating reasons and treat them as soon as possible. It must be highlighted that regular patient monitoring in relation to the aforementioned objectives by clinical and laboratory markers is essential for the successful treatment of DKA.

Approximately 6 L of total body water are lost in the volume-depleted phases of DKA. As a result, the goal of the initial fluid therapy is to increase intravascular volume and ensure proper urine flow. Isotonic saline should be administered as the first fluid at a rate of 15-20 mL per kg of body weight per hour, or 1-1.5 L over the first hour. The choice of fluid for additional replenishment relies on the degree of hydration, the level of serum electrolytes, and the production of the urine. A 0.45% NaCl infused at 4-14 mL/kg/hour is recommended for patients who are hyper- or eunatremic, and 0.9% NaCl given at a comparable rate is suggested for patients with hyponatremia. Over the course of 12 to 24 hours, it is intended to make up half of the predicted water and salt deficiency. In the current case, the patient's therapy included rehydration using NaCl 0.9% 2 L/2 hours, continued by 80 drops per minute/4 hours, 30 drops per minute/18 hours, and 20 drops per minute/24 hours.

Insulin administered in physiological dosages is the cornerstone of DKA therapy. Only after the serum potassium level is greater than 3.3 mmol/L can insulin be administered. Utilizing an intravenous bolus of regular insulin (0.1 U/kg body weight) in DKA, followed by 0.1 U/kg/hr continuous infusion of regular insulin is recommended. The insulin rate should be reduced to 0.05 U/kg/hr when plasma glucose reaches 200–250 mg/dL. This should be followed, as specified, by a shift in hydration fluid to D5 12 NS. In order to keep blood glucose levels in DKA between 150 and 200 mg/dL until it is resolved, the insulin infusion rate needs to be modified.

The effectiveness and affordability of subcutaneous rapid-acting insulin analogs (lispro or aspart) in the treatment of individuals with uncomplicated mild to moderate DKA have been demonstrated in a number of clinical investigations. The patients can either received an initial dosage of 0.3 U/kg followed by 0.2 U/kg every 2 hours until their blood glucose level was below 250 mg/dL, or received an initial dose of 0.2 U/kg followed by 0.1 U/kg every hour. The insulin dosage was then cut in half to 0.05 or 0.1 U/kg, as appropriate, and given every 1 or 2 hours until the DKA was resolved. Regular insulin is best administered via continuous IV infusion due to its quick half-life and simple titration compared to subcutaneous regular insulin, which has a slower onset of action and a longer half-life. It is crucial to note that there are no studies to support the IV use of fast-acting insulin analogs in patients with severe DKA, therefore this recommendation should be made clear. Again, since these medications are administered subcutaneously, they could not work in patients who have significant fluid depletion. The patient in this study was given fast intravenous regulation with rapid-acting insulin 2x4 IU and rapid-acting insulin pump 1 IU/hour. After DKA was resolved, insulin pump was replaced by subcutaneous long-acting insulin (Levemir 0-0-10 IU) and rapid-acting insulin (novorapid 6-6-6 IU).

The fundamental source of the underlying acid-base imbalance should be addressed in the management of metabolic acidosis. For sepsis and diabetic ketoacidosis, for instance, sufficient fluid resuscitation and correction of electrolyte imbalances are required. Antidotes for poisoning, dialysis, antibiotics, and the delivery of bicarbonate in specific circumstances are further treatments to consider. Diabetes-related ketoacidosis frequently results from bacterial infections. In this situation, postponing antibiotic therapy is linked to higher rates of morbidity and mortality. However, unnecessary antimicrobial therapy delivery may potentially have a deleterious effect on the prognosis.

CONCLUSION

We reported a case of a 45-year-old female patient with a chief complaint of fever, right lower abdominal pain, and vaginal white discharge. The patient has a history of uncontrolled T2DM since 2014. Based on all the medical history, physical examination, and laboratory findings, the patient was diagnosed with DKA, T2DM, and TOA with differential diagnosis endometrioma. The therapies given consisted of fluid, insulin, diet, and antibiotic therapies. Euglycemia is the main goal of perioperative treatment, which also aims to enhance postoperative results. Careful blood glucose management should be performed prior to surgery to decrease morbidity and mortality of patients; steer clear hyperglycemia; upkeep fluid and electrolyte balance in the body; avoid ketoacidosis; and establish specific glycemic goal levels, with stable patients having less than 140 mg/dL and critical patients having less than 180 mg/dL. It must be highlighted that regular patient monitoring in relation to the aforementioned objectives by clinical and laboratory markers is essential for the successful treatment of DKA.

PATIENT CONSENT

The patient had agreed and signed informed consent regarding publishing this clinical case in an academic journal without exposing the patient's identity.

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CONFLICTS OF INTEREST
The authors declare no conflict of interest regarding the manuscript.

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AUTHOR CONTRIBUTION
Both authors contributed equally to the study.

REFERENCES