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DIABETIC KETOACIDOSIS IN PREGNANCY CAUSED BY MULTIPLE RISK FACTORS: A CASE REPORT

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ABSTRACT
Pregnant patients with diabetes are susceptible to diabetic ketoacidosis (DKA) but this condition is still underreported. Unknown risk factors during pregnancy will increase morbidity and mortality of DKA. The interaction between multiple risk factors will certainly increase the burden of health care. This report aims to discuss the management of DKA in pregnancy with multiple risk factors, from diagnosis to prevention of complications. This study is descriptive study with case report design. Data were obtained from anamnesis, physical, and supporting examinations at Wangaya Hospital. Data were analyzed qualitatively and presented narratively according to case report format. This paper reports DKA in 39-year-old female with 24 weeks gestational age with shortness of breath. This was her fourth pregnancy. The patient had no history of diabetes and had never undergone pregnancy control. The patient had increased blood pressure and Kussmaul's breathing. Supporting examinations showed leukocytosis, hyperglycemia, bacteriuria, ketonuria, proteinuria, metabolic acidosis, low potassium, while chest x-ray showed pneumonia. The patient was diagnosed DKA with severe preeclampsia, pneumonia, urinary tract infection, and hypokalemia. Unfortunately, the patient experienced IUFD. After aggressive fluid replacement, intravenous insulin, electrolyte correction, treat the causes, and maternal monitoring, the patient showed significant improvement. Various adaptations during pregnancy can lead to DKA. Missed glucose homeostasis screening, oxidative stress, aging of β cells, inflammatory environment with various cytokines, to vascular dysfunction can be pathways for DKA in pregnancy. DKA in pregnancy is a challenge that requires proper risk factor screening. Optimal management and close monitoring are needed to achieve good prognosis.

Keywords: diabetic ketoacidosis; pregnancy; risk factors

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INTRODUCTION

The prevalence of type 2 diabetes mellitus (DM) is now increasing in developing countries with a decreasing age of onset. Women of reproductive age can also experience diabetes. Various forms of diabetes can overlap with pregnancy (Coetzee et al., 2023). Globally, the prevalence of pregnant patient with DM varies widely. A recent meta-analysis reported that global pregnant patients with DM had reached 14.7% based on the International Association of Diabetes and Pregnancy Study Groups (IADPSG) criteria (Saeedi et al., 2021). Pregnant patients with DM are susceptible to diabetic ketoacidosis (DKA). Although DKA in pregnancy is an obstetric emergency, epidemiological data on this condition are still underreported. Studies state that reports of DKA in pregnancy are only around 0.5%-10% of all cases. Maternal death due to DKA is rare, but fetal mortality is reported to range from 10% to 35% (ACOG, 2018). Although maternal death is rare, DKA in pregnancy causes significant maternal morbidity (Dhanasekaran et al., 2022).

Many physiological adaptations occur during pregnancy that cause DKA so that the prevalence of DKA in pregnancy is higher than in non-pregnant women. Pregnancy is

associated with an increase in insulin antagonist hormones such as human placental lactogen, prolactin and estrogen which contribute to a decrease in insulin sensitivity by more than 50% in the third trimester (Dalfrà et al., 2016). The placenta secretes a variety of diabetogenic hormones that are responsible for insulin resistance. This condition will cause increased gluconeogenesis, glycogenolysis, and lipolysis. Lower glucose levels can also cause ketogenesis and lipolysis. Lipolysis will produce free fatty acids and ketones as alternative energy sources. In addition to these changes, the maternal capacity for buffering acid also decreases due to increased alveolar ventilation and decreased bicarbonate levels (Jaber et al., 2019).

There are various risk factors that cause DKA in pregnancy. Pregnant women who experience DKA often come from low socioeconomic status with poor compliance with examinations and suboptimal glycemic control before and during pregnancy (Dhanasekaran et al., 2022). Patients with high-risk pregnancies such as old age, preeclampsia, gestational hypertension, infection during pregnancy, multiple gestations, have comorbidities such as renal failure and congestive heart failure play an important role in the incidence of DKA in pregnant patient with DM (Barski et al., 2023; Wen et al., 2022). Unknown risk factors during pregnancy will increase the mortality rate. The interaction between these risk factors will certainly contribute to DKA and increase the burden of patient health care. This becomes even more complex if the patient has multiple risk factors because the management given will involve many aspects of treatment (Coetzee et al., 2023). The following is a case report showing a 39-year-old pregnant patient who experienced DKA with various risk factors during pregnancy. This report aims to discuss the management of DKA in pregnancy with multiple risk factors, from establishing diagnosis, appropriate management, to preventing the complications.

METHOD

This article is a case report study that provides diagnosis, clinical management, and patient follow-up care. Data from this case report were obtained through anamnesis, physical examination, and supporting examinations conducted at Wangaya Hospital, Bali. The data obtained were then analyzed qualitatively and presented in narrative form. This case report discusses a woman who experienced DKA during pregnancy with multiple risk factors. This case report will describe the risk factors found, analyze individual case, and is expected to provide new insights into clinical practice, especially regarding the problem of DKA in pregnancy. The results will be compared with relevant literatures to assess the appropriateness of the diagnosis, management, and the patient outcomes.

RESULT

CASE ILLUSTRATION

A 39-year-old woman, working as a housewife, came to the emergency room with complaints of shortness of breath that had been felt since 2 days ago. At that time, the patient was pregnant with her 4th child with a gestational age of 24 weeks 5 days. Shortness of breath was accompanied by fever, nausea, and vomiting several times. Complaints of chest pain, palpitations, and history of trauma were denied. The patient had no history of heart disease or diabetes mellitus. Family history of heart disease and diabetes mellitus was denied. The patient had never checked her pregnancy at all with a health worker. Her three previous pregnancies were carried out normally and assisted by a midwife without any comorbidities. The patient was in compost mentis but appeared weak with very shortness of breath. Vital signs upon admission to the ER showed blood pressure of 152/96 mmHg, heart rate of 138 beats/minute, respiratory rate of 33 beats/minute, temperature of 38.3ºC, and oxygen saturation of 98% on 3 l/min via nasal cannula. On physical examination, rhonchi were heard

in both lung fields with a Kussmaul breathing pattern. On obstetric examination, the height of the uterine fundus was found to be ½ center to the xyphoid process (26 cm) with a fetal heart rate of 143 beats/minute, no vaginal discharge, and no signs of uterine contractions.

Her laboratory investigations revealed: serum glucose level of 347 mg/dL with HbA1c of 10.4%. Complete blood count showed white blood cells of 19.10 x 10^3 /μL, hemoglobin of 13.2 g/dL, and platelets of 316 x 10^3 /μL. On initial blood gas analysis, arterial blood pH was 7.14, pCO_2 9 mmHg, pO_2 125 mmHg, serum bicarbonate level of 3, ABE of -25, SBC <5, and SO2 98%. On renal function tests, blood urea nitrogen of 26 mg/dL and serum creatinine of 1.4 mg/dL were found. Liver function tests showed no abnormalities with Serum Glutamic Oxaloacetic Transaminase (SGOT) of 9 μ/L and Serum Glutamic Pyruvate Transaminase (SGPT) of 6 μ/L. Initial electrolyte tests were also performed and found sodium values of 138 mmol/L, potassium 4.1 mmol/L, and chloride 104 mmol/L. Urine tests showed protein +3, glucose +2, ketones +4, urobilinogen +2, bilirubin +1, blood +1, bacteria +3. Triple elimination for pregnancy such as HbsAg, anti-HIV, and TPHA were non-reactive. Chest X-ray showed infiltrates suggestive of pneumonia. The patient was then diagnosed with diabetic ketoacidosis, severe preeclampsia, urinary tract infection, and pneumonia.

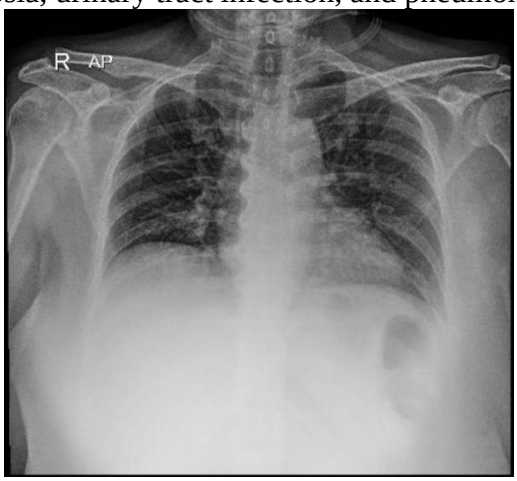

Figure 1. Chest X-ray results of a patient suggesting pneumonia.

This condition made the patient treated in the intensive care unit (ICU). This patient was treated together with obstetrician, anaesthesiologist, pulmonologist, and cardiologist. In the initial management, the patient was given 0.9% NaCl fluid resuscitation, blood sugar regulation with Aspart drip 6 units/hour, and sodium bicarbonate drip in 500 ml of 0.9% NaCl. The patient was given 40% MgSO4 at a rate of 0.5 grams/hour with a syringe pump, methyldopa 250 mg every 8 hours, nifedipine 10 mg every 8 hours PO if MAP>125 mmhg, paracetamol 1000 mg every 8 hours if fever occurs, ceftazidime 1 gr every 8 hours, combivent nebulization every 8 hours, and considering fetal life, dexamethasone 6 mg every 12 hours was given for lung maturation. Given the patient's poor initial condition, the patient was fitted with a CVC, nasogastric tube, and intubation. Blood sugar regulation has been carried out strictly according to the protocol. However, considering the side effects of dexamethasone administration, the target blood sugar achievement is difficult to achieve normal values. After periodic examinations, the patient had experienced hypokalaemia with a potassium level of 2.3 so that K+ replacement was given with a KCl drip of 50 meq in 500 cc of normal saline. Unfortunately, a few days later the fetal heart rate was not detected so that termination was performed and diagnosed with intrauterine fetal death (IUFD).

Insulin drip was continued with repeat glucose, electrolyte, and blood gas tests until the next day. Although through a complex process, after close monitoring, the patient's condition

gradually improved. Blood sugar regulation tended to be stable (blood sugar level lowered to 142 mg/dL). Final blood gas and electrolyte analysis showed pH 7.32, pCO_2 26 mmHg, $cHCO_3$ 13 mmol/l, SO_2 99%, sodium 146 mmol/l, potassium 3.4 mmol/l, and chloride 109 mmol/l. The patient's shortness of breath also improved and his consciousness began to be adequate so that the patient was then extubated and moved to a regular ward. The patient has shown improvement during treatment until insulin was changed to Glargine 1x6 IU and Aspart 3x3 IU subcutaneously. The patient was then recommended for outpatient treatment and to undergo routine check-ups at the endocrine polyclinic. The management of DKA during pregnancy can be adjusted to Figure 2.

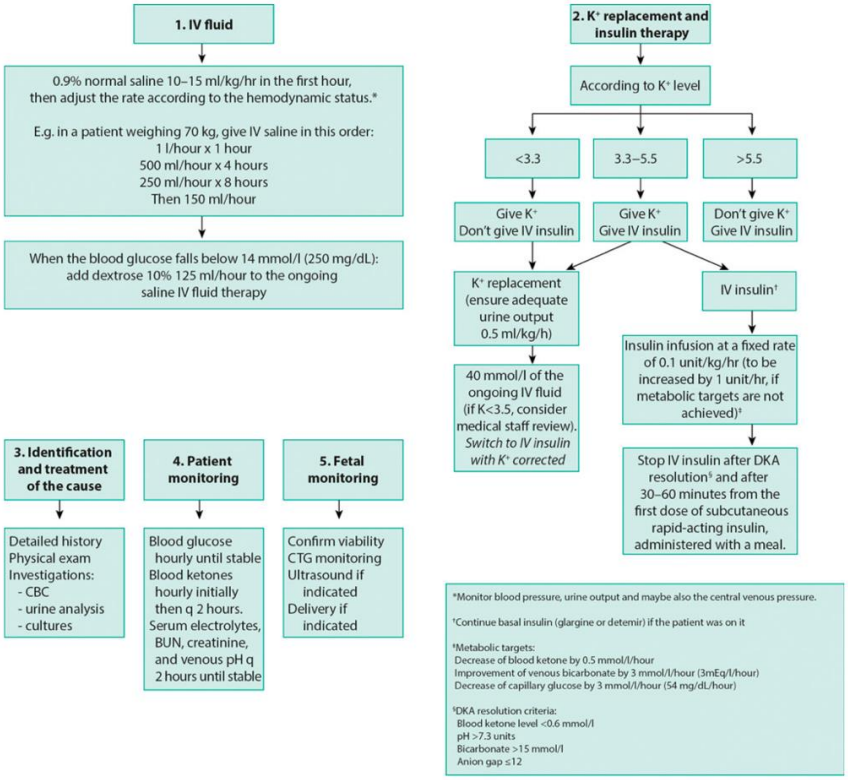

Figure 2. Algorithm for DKA management in pregnant patient (Mohan et al., 2017)

DISCUSSION

Diabetic ketoacidosis (DKA) is defined as a condition of hyperglycemia, hyperketonemia, and metabolic acidosis (Diguisto et al., 2022). Various adaptations can occur during pregnancy that can lead to DKA. Although the mechanism is not fully understood, changes in the metabolic environment in pregnancy can develop DKA. Pregnancy is associated with increased insulin antagonist hormones such as human placental lactogen, prolactin, and estrogen which contribute to more than 50% decrease in insulin sensitivity. Pregnancy is also associated with increased lipolysis and ketone body production even with relatively short periods of fasting. Increased minute alveolar ventilation also occurs in pregnant women which can cause respiratory alkalosis with increased renal bicarbonate excretion as compensation. The combination of all these processes predisposes to the development of DKA (Dhanasekaran et al., 2022).Clinically, the presentation of DKA in pregnancy can vary such as lethargy, shortness of breath, fatigue, headache, poor appetite, nausea, vomiting, and abdominal pain. This condition can be combined with dehydration and delayed gastric emptying. Kussmaul breathing (deep breathing pattern and hyperventilation in response to metabolic acidosis) is typical of DKA. Increased acetone due to ketonemia causes the patient's breath to have a fruity odor (Elendu et al., 2023). In this case, the patient presented with shortness of breath, fever, nausea and vomiting accompanied by Kussmaul breathing pattern.

Based on supporting examinations, a person is diagnosed with DKA when arterial pH <7.3 and/or bicarbonate levels <15 mmol/l with ketonemia >3 mmol/l or ketonuria >2 pluses on urine examination (Diguisto et al., 2022). In accordance with this case, the patient came with hyperglycemia with blood sugar 347 mg/dL accompanied by metabolic acidosis with blood gas analysis results of 7.14, pCO2 9 mmHg, pO2 125 mmHg, serum bicarbonate level of 3 and urine examination results with ketone +4. Based on clinical and laboratory criteria, the patient met the criteria for DKA.

The occurrence of DKA in pregnancy can be caused by various factors. In this case, the most important risk factor was patients who had never had pregnancy control so that many risk factors were missed. Studies showed that almost a quarter of pregnant women are only diagnosed with DM when they have already fallen into DKA. Low knowledge and awareness of carrying out pregnancy control, limited resources, and access to local health care make many pregnant women missed glucose homeostasis screening during pregnancy (Coetzee et al., 2023; Dhanasekaran et al., 2022). High HbA1c values in pregnant women with DKA indicate poor glycemic control. In newly diagnosed DM with poor glycemic control, the risk of DKA is highly likely to increase regardless of the identified precipitating cause (Coetzee et al., 2023). Reflecting on this case, the patient had an HbA1c level of 10.4% which indicates the patient may have poor glycemic control.Not only low pregnancy control, but this patient also has multiple gestations. Multiple gestations can be a risk for DKA. In this case, the patient is pregnant with her fourth child. Multiparous pregnancies cause cellular stress and aging in β cells which causes β cells to be unable to compensate for insulin resistance. The level of insulin resistance and adiposity will increase after multiparous pregnancies but the proliferation capacity of β cells to compensate for insulin resistance decreases, causing glucose intolerance (Moon et al., 2023).

Infection is another precipitating factor for DKA in DM. In this case, the patient experienced urinary tract infections and pneumonia as seen from urinalysis with increased leukocyte esterase and positive bacteria and chest X-ray showing infiltrates. A study by Coetzee found that 79% of pregnant patients with DM had urinary tract infections (Coetzee et al., 2023). This is also in line with a study by Shahid et al found that most of the precipitating factors for DKA were pneumonia (38.7%) and urinary tract infections (30.6%). Infection as a triggering factor can be caused by various factors including neutrophil dysfunction, humoral immune system disorders, and neuropathy. The presence of infection will also form an inflammatory environment with the production of various cytokines and oxidative stress that can worsen DKA (Shahid et al., 2020). In this case, the patient was given the broad-spectrum antibiotic ceftazidime 1 gr every 8 hours to treat the infection.The diagnosis of preeclampsia is made when there is an onset of hypertension after 20 weeks of gestation accompanied by proteinuria and/or significant end-organ dysfunction. Systolic blood pressure (SBP) ≥140 mmHg or diastolic blood pressure (DBP) ≥90 mmHg on two examinations four hours apart in previously normotensive individuals or a single examination with SBP ≥160 mmHg or DBP ≥110 mmHg can be categorized as preeclampsia. Severe preeclampsia is defined as the presence of proteinuria, oliguria, and disorders of various organs (Brown et al., 2018). Studies show that most pregnant patients with DM have hypertension with a percentage of 47% and as many as 46% of hypertensive patients experience fetal losses (Coetzee et al., 2023). Preeclampsia and DKA are closely related. Hyperglycemia can cause endothelial dysfunction which worsens hyperglycemia during pregnancy (Yang & Wu, 2022). A study by Lee et al postulated that preeclampsia is caused by endothelial injury. If this vascular dysfunction fails to recover, this condition can not only become recurrent preeclampsia but also DM (Lee et al., 2017). Reflecting to this case, the patient was diagnosed with severe preeclampsia which is characterized by high blood pressure in pregnancy above 20 weeks and organ involvement.

Urinalysis results also showed proteinuria which supports the diagnosis. The management given in this case is prevention of eclampsia and antihypertensive agents such as 40% MgSO4 at a rate of 0.5 grams/hour with a syringe pump, methyldopa 250 mg every 8 hours, nifedipine 10 mg every 8 hours PO if MAP>125 mmhg.

Recognition of conditions that trigger DKA in pregnant patient with DM is very important because delays in correction can worsen the prognosis and increase the risk of recurrence. Therefore, detailed history, physical examination, and supporting tests such as complete blood count (CBC), urine analysis, and culture can help determine the trigger (Mohan et al., 2017). In this case, there are clinical risk factors that are suspected of causing DKA, which were preeclampsia, urinary tract infection, and pneumonia. Management of triggers in this case includes antihypertensive agents, prevention of eclampsia, and antibiotics in patients according to indications.The key components for the management of DKA are volume replacement, infusion of insulin and correction of electrolytes, identification and treatment of the causes, maternal monitoring, and fetal monitoring. Fluid replacement should be initiated with isotonic fluids. IVFD is initiated at 10-15 ml/kg/hour in the first hour and thereafter the rate will be adjusted according to the patient's hemodynamic status guided by blood pressure, urine output, and central venous blood pressure in selected cases. Fluid therapy can increase fluid perfusion, cause hemodilution, and reduce stress hormone levels, thereby reducing hyperglycemia and improving the response to insulin therapy. However, this condition must be considered in patients with impaired renal and cardiac function (Kohler & Levy, 2014; Mohan et al., 2017). In this case, the patient has received fluid loading management with normal saline. Fluid adequacy is also monitored by central venous catheter which is also adjusted to the patient's hemodynamics.

The next management is insulin administration. Insulin therapy not only corrects hyperglycemia but also inhibits the ongoing production of keto acids. Insulin therapy should be started immediately in patients with potassium levels ≥ 3.3 mmol / l. However, insulin administration can be postponed if potassium is low until corrected to ≥ 3.3 mmol/l because insulin can push potassium into the intracellular space which causes hypokalemia and triggers cardiac arrhythmias. Regular intravenous insulin infusion can be started at a fixed rate of 0.1 units/kg/hour and is recommended not to exceed 15 units/hour at the beginning (Kitabchi et al., 2009). If metabolic targets such as blood ketones, bicarbonate, or glucose are not achieved, insulin infusion can be increased by 1 unit/hour. IV insulin can be stopped if blood sugar levels and DKA have improved and replaced with subcutaneous insulin (Kohler & Levy, 2014). Patients with serum potassium levels less than 5.5 mmol/l with good urine output (at least 0.5 ml/kg/hr) receive potassium chloride to maintain potassium levels because potassium begins to return to cells with IV fluids and ongoing insulin therapy (Sibai & Viteri, 2014). If serum potassium levels are ≥5.5 mmol/l then potassium chloride does not need to be added. However, if the level is 3.5 - 5.5 mmol/l potassium chloride can be given (Mohan et al., 2017). In accordance with this case, the patient has received insulin drip with strict blood sugar monitoring until it tends to be normal, so it is replaced with subcutaneous insulin. This patient also has hypokalemia, so she received KCl drip to replace low potassium levels.

Currently, bicarbonate administration is still controversial. Studies have shown that there is no evidence of the benefits of bicarbonate administration because it inhibits compensatory hyperventilation that releases carbon dioxide. As a result, there is an increase in partial pressure of CO2 (PCO2) which can reduce oxygen in the fetus (Mohan et al., 2017). Previously, the concept of bicarbonate administration was based on a decrease in bicarbonate levels which defined the severity of DKA (Kohler & Levy, 2014). In this case, the patient was given sodium bicarbonate drip in 500 ml of 0.9% NaCl because the patient showed

Kussmaul's breathing and the blood gas analysis pH was 7.14, pCO2 9 mmHg, pO2 125 mmHg, serum bicarbonate level of 3, ABE of -25, SBC <5, and SO2 98%. For further monitoring, the patient was then subjected to serial blood gas analysis to monitor the improvement of the condition.In maternal monitoring, glucose levels should be monitored hourly during insulin infusion. Blood ketones are also monitored hourly for the first 6 hours to ensure that ketones can decrease to at least 0.5 mmol/l. Studies suggest that glucose levels are checked hourly until stable, blood ketone levels are checked every 2 hours, while electrolytes, blood gas analysis, urea, and creatinine are checked every 2 hours until stable (Mohan et al., 2017). In pregnant women with diabetes and preterm labor, dexamethasone therapy can improve fetal lung maturation but also cause hyperglycemia. Steroid administration has been identified as a precipitant of hyperglycemia in pregnant women. Therefore, steroid administration must be under intensive glucose monitoring in pregnant patient with DM (Coetzee et al., 2023). A similar condition occurs in this case where steroid therapy is given for lung maturation but causes glycemic levels that are difficult to achieve. The last pillar is fetal monitoring. Fetal effects involve acidosis conditions that cause reduced uteroplacental perfusion. Electrolyte disturbances also not only cause arrhythmias in the mother but also in the fetus which can cause IUFD. Fetal viability, fetal heart rate monitoring, ultrasonography are needed to assess fetal life. The decision for delivery or termination is individual and based on the clinical status of the mother and fetus (Mohan et al., 2017).

Maternal-related death is a rare complication, but different from cases of fetal death which have a high prevalence of 10-35% (ACOG, 2018). In this case, the patient experienced IUFD at 24 weeks of gestation. The study found that the fetus is more sensitive to DKA in early pregnancy compared to older gestational ages. The high rate of IUFD during DKA indicates that DKA has a significant contribution. Acidosis, dehydration with reduced uteroplacental perfusion, and electrolyte imbalance are a combination of factors for fetal mortality. The more severe the level of acidosis experienced by the mother, the higher the risk of fetal death (Morrison et al., 2017). However, the patient's condition gradually improved with the management given and close monitoring. The patient's clinical status improved, the patient's blood sugar tended to be stable, and blood gas analysis became normal. Given the need for monitoring of both the mother and fetus, the high risk of IUFD, and the high prevalence of metabolic disorders, DKA in pregnancy is a disease to watch out for. Optimal management is needed to overcome DKA in pregnancy to achieve a good prognosis (Diguisto et al., 2022).

CONCLUSION

DKA in pregnant patient with various risk factors is a challenge for clinicians. Risk factors such as poor control compliance, multiple pregnancies, severe preeclampsia, and infection can be predisposing factors for DKA. Management such as volume replacement, infusion of insulin and correction of electrolytes, identification and treat the causes, maternal monitoring, and fetal monitoring are the keys to DKA management in pregnancy. Given the complications that can occur, patients with DKA require close monitoring and evaluation to achieve a good prognosis.

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