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ROLES (CRediT):

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Funding: Self-funded.

Conflict of interest: The authors declare that they have no conflict of interest.

Ethical considerations: The information was obtained from the patient's medical history, after obtaining her informed consent, duly signed and recorded in a form, thus guaranteeing the confidentiality of the data and respect for her autonomy.

Received: 5 March 2025

Accepted: 4 September 2025

Online publication: 27 October 2025

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Cite as: Rodríguez-Benites A, Sánchez-Landers M, Morgan A. Non-diabetic ketoacidosis due to starvation in a pregnant woman with dengue. *Rev Peru Ginecol Obstet.* 2025;71(2). DOI: <https://doi.org/10.31403/rpgv71i2783>

Non-diabetic ketoacidosis due to starvation in a pregnant woman with dengue

Cetoacidosis no diabética por inanición en gestante con dengue

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DOI: <https://doi.org/10.31403/rpgv71i2783>

ABSTRACT

Non-diabetic ketoacidosis in pregnant women is a rare complication, but it is potentially serious and often difficult to diagnose because blood glucose levels are normal. We present the case of a pregnant woman in her second trimester with dengue infection. After initial management, she developed metabolic acidosis and ketonuria due to oral intolerance secondary to the viral infection. With adequate fluid therapy, the patient's clinical condition improved, supporting the diagnosis.

Key words: ketoacidosis; starvation; pregnancy; Dengue.

RESUMEN

La cetoacidosis no diabética en gestantes es una complicación poco frecuente, pero es potencialmente grave y suele ser de difícil diagnóstico por presentar valores normales de glicemia. Se presenta el caso de una gestante en el segundo trimestre con infección por dengue. Tras el manejo inicial, presentó acidosis metabólica y cetonuria por intolerancia oral secundaria a la infección viral. Con fluidoterapia adecuada, la paciente mejoró su estado clínico, lo que respaldó el diagnóstico.

Palabras claves: cetoacidosis; inanición; gestación; Dengue.

INTRODUCTION

In recent years, the incidence of dengue cases in our region has increased significantly, affecting various population groups, including pregnant women⁽¹⁾. This group already has risk factors inherent to their condition, such as geographical, cultural, physiological, and genetic determinants, which predispose them to additional complications⁽²⁾. Infection with the dengue virus has been associated with an increased risk of maternal mortality, as well as other complications such as placental inflammation, low birth weight, premature birth, fetal death, and postpartum hemorrhage⁽³⁾.

During pregnancy, a physiological state of insulin resistance induced by placental hormones develops, which favors greater glucose availability for the fetus-placenta unit. This reduces maternal fasting glucose levels, leading to a condition known as "accelerated starvation." This response intensifies in stressful situations and usually appears from the second trimester onwards, increasing as pregnancy progresses. As a result, pregnant women become more susceptible to developing starvation ketoacidosis after relatively short periods of fasting, between 12 and 14 hours, even with normal glucose levels^(4,5).

This report analyzes the case of a pregnant woman in her second trimester diagnosed with dengue fever, whose clinical progression was complicated by the onset of starvation ketoacidosis, illustrating the interaction between the physiological state of pregnancy and viral infections.



CASE REPORT

A 19-year-old woman, 21 weeks pregnant according to ultrasound, from an area endemic for dengue, presented with symptoms of general malaise, headache, myalgia, arthralgia, retroocular pain, and fever. She also reported contact at home with family members recently diagnosed with dengue, so she was admitted to a health center in her community, where parenteral hydration with 0.9% saline solution was initiated and dengue infection was confirmed by a positive NS1 rapid test.

During her stay at the community health center, starting on the second day, she developed nausea, persistent vomiting, oral intolerance, and epistaxis, along with findings of thrombocytopenia, leukopenia, and hemoconcentration. On the fourth day, her fever subsided, although her gastrointestinal symptoms persisted. On the fifth day, she developed severe abdominal pain rated 10/10 on a visual analog scale, which led to her referral to a more complex hospital with a diagnosis of dengue with warning signs and laboratory findings of hemoglobin 8.98 g/dL, hematocrit 28.6%, and platelets 46,000/uL.

The patient was admitted to the Gynecology-Obstetrics ward of our hospital's Emergency Department at noon, with severe abdominal pain (VAS 10/10) and uncontrollable vomiting. Her actual body weight was 65 kg and her height was 145 cm. Her vital signs were: blood pressure 100/60 mmHg, heart rate of 93 beats per minute, respiratory rate of 31 breaths per minute, oxygen saturation of 98% in ambient air, temperature of 36.4°C, capillary refill time of less than 2 seconds, and urine output of more than 0.5 cc/kg/h. The obstetric ultrasound showed a 21-week pregnancy with a single, viable fetus, fetal heart rate of 152 beats per minute, and estimated fetal weight of 399 g. In addition, laboratory tests were requested: complete blood count, glucose, urea, creatinine, arterial blood gas analysis, and urinalysis. and fluid therapy was started with 0.9% saline solution at 10 mL/kg/h, following Technical Health Standard (NTS) No. 211-MINSA/DGIESP-2024, as she was classified within Group B2. According to this NTS, all pregnant women with suspected dengue should be evaluated for warning signs or shock. If she does not show warning signs, she is classified as Group B1; but if she shows one or more, she is classified as Group B2 (dengue with

warning signs) or Group C (severe dengue) and requires hospitalization for specific management. In Group B2, to which our patient belonged, hydration with crystalloid solution is administered at 10 mL/kg/h for one hour, adjusting according to response. For the calculation, the ideal body weight (IBW) is used, estimated according to the patient's height, which in our case was 145 cm.

The patient required two successive hydration boluses due to persistent pain. After the second bolus, the pain partially subsided, although she continued to experience tachypnea. In the ICU evaluation, the lung ultrasound showed multiple bilateral B-lines and mild pleural effusion; in addition, gallbladder edema was found. Fluid therapy was adjusted to 7 mL/kg/h for 4 hours, in accordance with the aforementioned NTS management flowchart. However, arterial blood gas analysis after 10 hours revealed metabolic acidosis with elevated anion gap and altered base excess.

Given food intolerance lasting more than 4 days, starvation ketoacidosis was suspected, confirmed by ketonuria (++) . Treatment was initiated with 5% dextrose with electrolytes, in addition to Ringer's Lactate, maintaining hydration at 7 mL/kg/h, then reduced to: 3-5 mL/kg/h for 2 to 4 hours and then 2 to 4 mL/kg/h for 24 to 48 hours. The patient showed clinical improvement, normalization of blood gas parameters, and progressive oral tolerance. She was discharged four days after admission to our hospital with favorable clinical and laboratory results (Table 1).

DISCUSSION

Ketoacidosis, defined by the presence of metabolic acidosis with elevated anion gap and ketonuria, is a serious metabolic disorder classically associated with diabetes mellitus. However, it can also occur in non-diabetic contexts such as alcoholism, prolonged starvation, pregnancy, and lactation. Regardless of its etiology, clinical manifestations such as nausea, vomiting, tachypnea, and abdominal pain are similar, which can make differential diagnosis difficult when biochemical profiles do not include obvious hyperglycemia⁽⁵⁾.

In the present case, we report a case of non-diabetic ketoacidosis in a pregnant woman with no history of comorbidities such as diabetes mellitus or alcoholism, triggered by a dengue infection and associated with prolonged fasting. This



clinical picture is unusual but relevant, especially in epidemiological contexts such as ours. In the medical literature, cases of non-diabetic ketoacidosis in pregnant women due to starvation have been described^(6,7), and there are also reports of diabetic ketoacidosis precipitated by dengue^(8,9,10); however, the combination of non-diabetic ketoacidosis due to starvation and dengue in pregnancy is rarely documented. According to our review, this would be one of the first reports of this association in pregnant women, highlighting its clinical importance.

The hypermetabolic state of pregnancy predisposes women to accelerated ketone body production, even after relatively short periods of fasting. This phenomenon is more pronounced from the second trimester onwards, when increased counterregulatory hormones such as cortisol, progesterone, estrogens, prolactin, and human placental lactogen induce insulin resistance. Compared to non-pregnant women, pregnant women have higher ketonemia after 12-14 hours of fasting^(5,6,7). In this context, dengue infection, which caused persistent vomiting for more than four days, precipitated exaggerated ketogenesis in our patient.

In addition, severe dengue can cause systemic inflammation and endothelial dysfunction, contributing to increased catabolism and greater production of ketone bodies. The presence of pleural effusion and gallbladder edema in this patient reflect the degree of inflammatory involvement, findings also described in similar reports of severe dengue. Therefore, dengue not only triggered involuntary fasting but also amplified the adverse metabolic response^(11,12).

In terms of management, the literature indicates that early administration of 5% dextrose is essential, as it stimulates endogenous insulin secretion and reduces lipolysis, which decreases ketone body production and corrects acidosis. In addition, fluid replacement with balanced solutions, such as normal saline or Ringer's lactate, helps restore intravascular volume and correct electrolyte imbalances⁽¹³⁾. In our patient, the timely use of Ringer's lactate and 5% dextrose reversed the metabolic imbalances, with clear clinical and laboratory improvement.

Compared to other published cases, ours shares the clinical presentation of fasting ketoacidosis^(6,7,14), but differs in the triggering infectious

etiology^(14,15). This case highlights the importance of including non-diabetic ketoacidosis in the differential diagnosis of pregnant women with persistent vomiting and viral infections such as dengue, especially in endemic areas. Furthermore, it reinforces the need for a multidisciplinary approach involving obstetricians-gynecologists, emergency physicians, intensivists, and infectious disease specialists. Monitoring parameters such as anion gap, blood gas analysis, and ketonuria should be a priority, as should the early identification of prolonged fasting.

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