Case Report

Dengue Hemorrhagic Fever Grade III with Diabetic Ketoacidosis: A Case Report

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A 16-year-old, previously healthy Thai girl presented with DHF grade III. Fifteen hours after the first episode of shock, she had received an excessive amount of crystalloid isotonic solution and 20 ml per kilograms of Dextran-40 however she still had persistently rapid pulse rate and high hematocrit but also had polyuria with more than 4 ml/kg/hr of urine output. She was re-evaluated. Clinical signs showed severe dehydration with some ascites without signs of pleural effusion. Blood gas revealed increased anion gap metabolic acidosis. The cause of polyuria and metabolic acidosis was identified with hyperglycemia, ketouria and glucosuria. Afterwards she was diagnosed and treated as DHF grade III and DKA. Besides insulin administration, fluid resuscitation was very crucial. Intravenous fluid rehydration was needed while the unnecessary extra-volume could cause massive plasma leakage and later on fluid overload. Volume replacement was adjusted to degree of dehydration when signs of volume overload were monitored closely. She was out of DKA at 14 hours after the start of insulin and the intravenous fluid was stopped at 27 hours (36 hours after the first episode of shock). The final diagnosis was DHF grade III, diabetes mellitus with DKA and hepatitis.

Keywords: *Dengue Hemorrhagic Fever, Diabetic ketoacidosis*

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Dengue virus infection is one of the most important mosquito-borne viral diseases in humans. About 50 million dengue virus infections and 25,000 deaths occur yearly[1] worldwide. Clinical manifestations include undifferentiated fever, Dengue Fever (DF) and Dengue Hemorrhagic Fever (DHF). There are 4 grades of DHF severity: grade I and II without shock; grade III shock and grade IV profound shock[2]. DHF grade III and IV are also called Dengue Shock Syndrome (DSS)[2]. The presented patient presented with shock caused not only by plasma leakage found in DHF but also osmotic diuresis found in diabetic ketoacidosis (DKA).

Case Report

A previously healthy, 16-year-old Thai girl from Samut Prakarn province presented with a 5-day history of fever without other symptoms and a 2-day history of bleeding per gum, vomiting and poor appetite. She had been taking only acetaminophen and oral electrolyte solution (ORS). On the day of admission, she had no fever but she felt very weak and had coffee ground vomiting once. Her body weight was 40 kilograms (kg) and height was 154 cm. Initial physical examination at a community hospital showed an afebrile girl without signs of pleural effusion, ascites or hepatomegaly. Her hematocrit (Hct) was 54%; white blood cell count (WBC) was 8,000/mcL with 49% neutrophils (N), 40% lymphocytes (L), 10% monocytes (M) and 1% eosinophil (E); platelet count was 10,000/mcL. Liver function test results included an albumin concentration of 3.4 g/dL, aspartate aminotransferase (AST) of 1,090 U/L and alanine aminotransferase (ALT) of 800 U/L. One hour after admission, she developed shock with a blood pressure (BP) of 130/110 mmHg, pulse rate (PR) of 190 beats/min and Hct of 56%. She was initially diagnosed as DHF grade III and received 10 milliliters per kilograms (ml/kg) of 5% Dextrose in Normal Saline intravenously. After the loading dose; BP was 130/100 mmHg; PR was 136 beats/min; and Hct was 54%.

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She was transferred to the dengue ward at the Queen Sirikit National Institute of Child Health (QSNICH) because she had a persistently rapid pulse rate (130-142 beats/min) and high Hct (54-56%) even though there was no fever and the BP was about 120/80-130/80 mmHg. Five hundred ml of Dextran-40 was given in one hour before referral in conjunction with 10% Calcium Gluconate, Vitamin K1 and 20 ml of diluted 7.5% Sodium Bicarbonate because carbon dioxide (from electrolyte results) was 10 mmol/L. Seven hours after shock, her total intravenous intake was 2,900 ml with 5% Dextrose in Normal Saline, Acetar and Dextran. The calculated amount of her maintenance fluid requirement plus 5% deficit was 3,900 ml for 24 hours after shock and the urine output, recorded from urinary catheter, was 1,200 ml (4.3 ml/kg/hr). The Hct after Dextran was 44%.

On admission at QSNICH, 9 hours after shock, she was afebrile and had good consciousness. PR was 140 beats/min, BP was 140/90 mmHg and respiratory rate (RR) was 38 breaths/min. Lung signs showed equal breath sounds, no crepitations or wheezing. Her abdomen was tender at the epigastric area, with the liver enlarged 1 cm below the right costal margin, with no splenomegaly. Meningeal signs were absent. Petechial rash was present at both arms and legs with a positive tourniquet test. Other physical findings were unremarkable.

Laboratory results showed Hct of 49%, WBC 11,250 /mcL with 57% N, 29% L, 6% M and 8% atypical lymphocytes (ATL); platelet count was 10,000 /mcL. Liver function tests included an albumin concentration of 2.54 g/dL, cholesterol of 133 mg/dL, AST of 941 U/L, ALT of 512 U/L, total bilirubin of 0.61 mg/dL and direct bilirubin of 0.22 mg/dL. Venous blood gas revealed pH 7.22, pCO2 15.5 mmHg, HCO_3- 6.1 mmol/L and base excess (BE) -18 mmol/L. Blood chemistries disclosed ionized calcium 1.33 mmol/L, BUN 13.17 mg/dL and creatinine 0.45 mg/dL. Blood sugar from glucose meter was 287 mg/dL. Pleural effusion and infiltration could not be noticed from a portable chest x-ray in supine position.

After six hours in the dengue ward (15 hours after shock), she vomited about 30 ml once. No diarrhea, fever or active bleeding was observed. For 6 hours her intake was 790 ml and urine was 1,410 ml, with total intake of 3,690 ml. Despite continuous administration of sodium bicarbonate and 5% Dextrose in Ringer Acetate (5% DAR), she still had a very rapid pulse rate (more than 150 beats/min), rising Hct up to 55% and persistent metabolic acidosis (pH 7.23, pCO2 13.7 mmHg, HCO_3 5.6 mmol/L and BE -19 mmol/L) even though she passed adequate urine (about 5.9 ml/kg/hr). The second dose of Dextran-40 was given at the same time as the patient was re-evaluated.

The patient had a past history of polyuria including nocturia, polydipsia and polyphagia for several months. Her grandmother and her mother developed diabetes mellitus (DM) which had been controlled with oral diabetic drugs at age of 50 and 30 years respectively. The patient did not take any toxic substances, alcohol or medication. Physical examination revealed signs of severe dehydration with dry lips, sunken eyeballs, and tachycardia (PR of 140 beats/min) with a BP of 130/90 mmHg. The patient's body mass index (BMI) was 17. Acanthosis nigricans was not present. The patient had tachypnea without signs of pleural effusion or pulmonary edema. Abdominal examination showed the liver enlarged 2 cm below the right costal margin and ascites. Additional investigations revealed blood sugar 390 mg/dL, sodium 127 with corrected sodium[3,4] 132.8, potassium 5.51, chloride 103 and carbondioxide 3.8 mmol/L with anion gap[3,4] of 26 mmol/L (formulations to calculate corrected sodium in hyperglycemia condition and anion gap are shown in Table 1). Urine analysis demonstrated positive urine sugar (3+) and positive urine ketone (4+) without any cells. Coagulogram results contained prothrombin time (PT) of 14.6 seconds (10.0-13.4) with INR of 1.3, partial thromboplastin time of 50.1 seconds (24.7-37.2), and thrombin time of 6.3 seconds (4.5-6.0). Only sinus tachycardia was present in the electrocardiogram. Erythrocyte sedimentation rate (ESR) was 3 mm/min. Serum ketone, C-peptide and autoimmune markers: anti-glutamic acid decarboxylase (anti-GAD), islet cell antigen 512 antibody (anti AI-2), anti-islet and anti-insulin autoantibodies were not sent due to some limitations.

Table 1. Formulations to calculate corrected sodium in hyperglycemia and anion gap[3]

	Formulation
Corrected sodium (Na) (mmol/L)	Na + 2 [(glucose mg/dl - 100) x 100]
Anion gap (mmol/L)	Na - (Cl + HCO3)

At this point, the patient was diagnosed as DHF grade III with diabetic ketoacidosis. Intravenous fluid was changed to Acetar solution without dextrose. After a consultation with a pediatric endocrinologist, continuous intravenous administration of regular insulin was started at 0.1 unit/kg/hr and fluid resuscitation was adjusted due to degree of dehydration and severity of DKA with close monitoring of signs of excessive plasma leakage. Acetar was given at 10 ml/kg/hr in 2 hours because of clinical severe dehydration, then BP was 120/90 mmHg and PR was 110 beats/min and the rate of fluid was gradually titrated down. Blood sugar by glucose meter, urine sugar, and ketone were monitored every hour while Hct, electrolytes and venous blood gas were monitored every 4 hours; vital signs and urine output were monitored hourly until stable then every 4 hours according to DKA[5-7] and DHF[2,8] management guidelines. Potassium chloride was added in intravenous fluid according to blood potassium level and only ORS with sodium 75 mEg/L was allowed orally. There was an attempt to limit total fluid intake at a volume of maintenance plus 5% deficit (3,900 ml) per day.

Eight hours after continuous regular insulin drip with maximal dose at 0.14 unit/kg/hr, the patient's blood sugar was 218 mg/dL. Intake was 2,100 ml and urine was 2,200 ml. The patient looked mildly dehydrated with slightly increased ascites. Then Acetar was switched to 5% DAR at maintenance rate and the rate of regular insulin was gradually decreased. At 14 hours after the beginning of regular insulin and 29 hours after shock, the patient was out of DKA with a blood sugar of 199 mg/dL, negative urine sugar and ketone, blood pH 7.43 and HCO_3 of 18.8 mmol/L. Intravenous fluid was stopped at 20 hours after the beginning of regular insulin (36 hours after shock). In total, the patient received 5,800 ml of intravenous fluid including 20 ml/kg of Dextran-40 within 36 hours. Laboratory results revealed BUN of 8.35 mg/dL, creatinine 0.28 mg/dL, AST and ALT of 503 and 398 U/L, respectively. Coagulogram was normal with an INR of 1.0. Repeated portable chest x-ray showed no effusion or infiltration.

Twenty-seven hours into the treatment, the continuous regular insulin was changed to intermittently subcutaneous regular insulin. Two days after that, insulin could be adjusted to subcutaneous intermediate insulin, Neutral Protamine Hagedorn (NPH) twice a day, and regular insulin when needed. Finally the patient received 24 units of NPH and 12 units of regular insulin in the morning before meals, 10 units of regular insulin in the evening before meals and 10 units of NPH at bed time.

The patient was in the hospital for 9 days with diagnosis of DHF grade III, diabetes mellitus with DKA and hepatitis. The patient was in the convalescent phase with stable vital signs and good appetite on the third day of admission. The later days of her hospital stay were for insulin adjustment, self-injection training and learning about diet control. Additional investigations before discharge showed hemoglobin (Hb) A1c of 11.2% and serum insulin of 2 mcU/L, while blood sugar was 390 mg/dL, with no bacterial growth in the blood and urine cultures, Hct of 38% and platelet count of 100,000/mcL. PCR for dengue virus was negative but dengue antibodies (using enzyme immunoassay, EIA), taken on the 6th day of illness, were positive with Dengue IgM of 48 and IgG of 184. One week later, the patient came for a follow-up with excellent clinical well-being. Her Hct was 39% and platelet count had increased to 433,000 /mcL. Liver enzymes decreased to normal as AST and ALT of 27 and 34 U/L and albumin was 4.32 g/dL.

Discussion

The patient presented with clinical symptoms and signs of DHF, according to the World Health Organization case definition for DHF[2] as shown in Table 2. These were a history of fever, bleeding tendencies; bleeding per gum, hematemesis and petechiae with positive tourniquet test, thrombocytopenia (platelet count of 10,000/mcL) and evidence of plasma leakage; hemoconcentration (Hct of 54%) and hypoalbuminemia (3.4 g/dL which decreased to 2.54 g/dL). The patient developed shock with narrow pulse pressure (BP of 130/110 mmHg), a very rapid pulse rate (190 beats/min) and higher Hct (56%). The initial diagnosis was DHF grade III. After an excessive amount of crystalloid intravenous fluid (2,400 ml in 6 hours), which was more than half of the volume of maintenance plus 5% deficit, was given, she still had a rapid pulse rate even though urine output was still more than 4 ml/kg/hr.

The recommended amount of fluid intake during the critical (leakage when platelet count < 100,000 /mcL) phase is maintenance plus 5% deficit[2,8,9] which should be divided in 24 hours in DSS. For example, in this 40-kg patient, maintenance plus 5% deficit equals 3,900 ml. Within 6 hours, the volume of fluid intake should not exceed 1,000 ml.

The patient's Hct was 54% so the first dose of Dextran-40 was given, then Hct dropped to 44%. But later on at 15 hours after shock the patient had received

Table 2. World Health Organization Case Definitions for DF/DHF/DSS[2]

Manifestations	Case Definitions
DF	Probable DF: - Fever of 2 to 7days' duration, with two or more of the following: Headache, retro-orbital pain, myalgia, arthralgia, rash, hemorrhagic manifestations, WBC < 5,000 /mcL - and supportive serology or occurrence at the same location and time as other confirmed cases of DF Confirmed cases: by isolation of the dengue virus, demonstration of the dengue virus antigen including by PCR or fourfold change in reciprocal IgM or IgG
DHF	All of the following must be present: 1. Fever or history of acute fever, lasting 2 to 7 days 2. Hemorrhagic manifestations in the form of at least one of the following: - A positive tourniquet test - Petechiae, ecchymosis, or purpura - Mucosal or gastrointestinal bleeding - Bleeding from injection site or other locations 3. Thrombocytopenia (≤ 100,000/mcL) 4. Evidence of plasma leakage as evidenced by one or more of the following: - A rise in the Hct (≥ 20% over baseline or average Hct for age, sex and population) - A drop in Hct following volume replacement treatment ≥ 20% of baseline - Signs of plasma leakage such as pleural effusion, ascites and hypoalbuminemia
DSS	DHF plus evidence of circulatory failure manifested by: - Rapid and weak pulse - Narrow pulse pressure(<20 mm Hg) or hypotension for age - Cold, clammy skin and altered mental status

3,990 ml of total fluid, equally to amount of maintenance plus 5% deficit, she still had rapid pulse rate and metabolic acidosis.

When DHF patients still have signs of impending shock, for example; rapid pulse rate (not related to degree of temperature) or shock and have already received an excessive amount of intravenous fluid, especially more than the amount of maintenance plus 5% deficit according to time, at least four parameters should be considered[8]. The first parameter is using Hct to determine the massive leakage with high Hct level or bleeding, especially concealed bleeding when the Hct drops or is not as high as the level of hemoconcentration (20% rising Hct from baseline). Second, blood gas should be sent to detect metabolic acidosis found in prolonged shock, bleeding and other causes. Third, hypoglycemia can be detected in unresponsive shock. The last parameter is blood chemistry including ionized calcium. The initial management in this case was 10 ml/kg in 1 hour of Dextran-40 because of the high Hct when the results of other investigations were pending.

The presented patient was re-evaluated by history-taking, physical examination and further investigations. The striking history was that the patient had a several-months history of polyuria including nocturia, polydipsia and polyphagia. The patient had a good urine output of more than 4 ml/kg/hr even though the patient was in unresponsive shock. Physical examination showed signs of severe dehydration without history of massive concurrent loss such as severe vomiting and diarrhea. Signs of severe dehydration should not be observed in DSS with plasma leakage without fluid loss outside the body and other distributive shock including septic shock. Polyuria without adequate fluid replacement can cause severe dehydration; so the cause of polyuria in the presented case should be evaluated.

The patient presented with polyuria and signs of severe dehydration together with hyperglycemia (390 mg/dL) and increased anion gap metabolic acidosis (pH 7.23, pCO2 13.7 mmHg, HCO_3 5.6 mmol/L and anion gap of 26 mmol/L). Urine examination found positive urine ketone and sugar. All of these results were

Table 3. Definition of DKA[3,4]

Criteria	ADA 2006[3]	ISPAD 2009[4]
1. Hyperglycemia	Blood glucose > 11 mmol/L (approximately 200 mg/dL)	Blood glucose > 11 mmol/L (approximately 200 mg/dL)
2. Acidosis	venous pH < 7.25 or arterial/ capillary pH < 7.3 and/or bicarbonate < 15 mmol/L	venous pH < 7.3 or bicarbonate < 15 mmol/L
3. Ketosis	Ketonemia and ketonuria	Ketonemia and ketonuria

ADA = American Diabetes Association, ISPAD = International Society for Pediatric and Adolescent Diabetes

compatible with diabetic ketoacidosis[3,4,6] as shown in Table 3. Other causes of increased anion gap metabolic acidosis should be excluded especially prolonged shock in DSS[8,10] due to inadequate or delayed fluid resuscitation of plasma leakage or delayed blood transfusion in case of bleeding; acute renal failure as DSS complication[8,9,11]; sepsis and toxic substances for example: salicylates, NSAID, metformin and alcohol[12,13]. A few can cause ketosis as in DKA for example, alcohol intoxication[13,14] and prolonged starvation[13] which can be ruled out in the presented patient by her history of illness.

Sepsis or septic shock was unlikely in the present case because her clinical manifestation was worse after defervescence; there were no symptoms and signs of localized infection and chest x-ray and urinalysis provided no evidence of infection. WBC count and differential count favored viral infection with lymphocytosis and presence of atypical lymphocytes without immature neutrophils. ESR was low (3 mm/hr), which is more common in dengue infection than in other viral or bacterial infections[15]. The patient's shock presented with high diastolic pressure, not the low diastolic BP seen in septic shock. Stable vital signs responded to fluid resuscitation and declining blood sugar level despite no antibiotics being given. Afterwards, blood and urine cultures disclosed no bacterial growth.

Acute renal failure was excluded by normal and no escalating tendency of BUN and creatinine on the same day of shock and later days after shock.

Prolonged shock caused from massive leakage was unlikely even though her initial shock could have been a result of plasma leakage. The patient had evidence of plasma leakage such as ascites which was later detected; hemoconcentration demonstrated by 47% rising Hct (with maximal Hct of 56% and minimum of 38%); hypoalbuminemia at the beginning and dropping from 3.4 to 2.54 g/dL within hours. Concealed bleeding was also less possible because of persistently high Hct (54-56%) and improvement of liver enzymes and BUN/ creatinine level which could worsen if the issue of inadequate tissue oxygenation, such as lack of red blood cells to carry oxygen, was left unsolved.

Later on, especially many hours after shock, unstable vital signs with a rapid pulse rate in the case could not be explained solely by a continuingly increasing amount of plasma leakage. First presentation of shock in DHF is the peak rate of the plasma leakage, after which the rate of leakage gradually decreases and usually stops at 24 hours after first episode of shock[8,9]. The present patient still had a rapid pulse rate at 15 hours after shock even though excessive amounts of crystalloid and 20 ml/kg of Dextran-40 were given and there was no further sign of massive leakage enough to cause shock shown on the physical examination or chest films.

Pleural effusion was not shown in the present case from physical examination or portable chest films but the supine position was less sensitive to detect right pleural effusion than right lateral decubitus. The patient might have presented with more leakage into abdominal space than pleural space. Pleural effusion is more common than ascites in DHF; however, detection of pleural effusion, when there is not a lot of effusion, may need a proper x-ray technique, or an ultrasound, or a follow-up film during early convalescent phase[16].

In addition to unstable vital signs with excessive crystalloid and colloid fluid administration, the important clues were polyuria and increased anion gap metabolic acidosis which was unresponsive to fluid and sodium bicarbonate replacement. Decrease of intravascular volume in this case occurred from two main causes: plasma leakage in DHF and osmotic diuresis from hyperglycemia.

After fluid resuscitation to replace fluid loss in urine and insulin to decrease blood sugar and prevent further gluconeogenesis from lipolysis and proteolysis,

the patient had more stable vital signs and improvement of metabolic acidosis. Acetar and 5% DAR were used because of their advantages above other isotonic crystalloid solution in DHF with its additional content especially potassium 4 mEq/L and acetate 28 mEq/L that can be changed to bicarbonate. Another benefit of Acetar above Ringer's lactate in the patients with elevated liver enzymes was that acetate can be changed to bicarbonate but lactate needs to be changed to acetate first in the liver. Her blood sugar level progressively decreased along with a decline in urine sugar and urine output, so the amount of intravenous fluid could be reduced and stopped at 20 hours since the start of insulin (36 hours after the first episode of shock).

The fluid replacement in the presented case was very crucial because the patient had signs of severe dehydration due to osmotic diuresis that need 10-20 ml/kg of fluid resuscitation, but at the same time she was in an active plasma leakage phase of DHF. When the loading dose of Acetar was started, the capillary permeability still increased but the rate of leakage decreased because it was already 15-16 hours after the first episode of shock. The unnecessary extra amount of fluid resuscitation could aggravate massive pleural effusion and ascites; later on fluid overload could compromise respiration and venous return and acute pulmonary edema when the patient was in the convalescent phase with fluid re-absorption. The amount of fluid resuscitation should be composed of the correction of dehydration and replacement of plasma leakage. In the presented case, the volume of fluid substitution should be able to mainly correct severe dehydration at the very first hours of the start of DKA management because the rate of leakage at this point was very minimal. After the severe and later moderate dehydration were improved, the volume of the fluid replacement should be equal to maintenance amount. When the patient was able to eat and drink well, the amount of oral intake should be subtracted from the planned total volume. During the time of fluid replacement, the signs of fluid overload had to be monitored including the signs of acute pulmonary edema which could occur during the convalescent phase of DHF (usually happened 36-48 hours after the first episode of shock)[9].

Finally the patient was diagnosed as DM which might have been happening for several months because Hb A1c was more than 6.5%[17] and this episode of DKA was precipitated by DHF grade III. It was difficult to classify the type of DM in the presented patient from the available data. Type 1 DM is more commonly presented with DKA because of low or absence of endogenous insulin. But DKA is not uncommon in type 2 DM with insulin resistance and relative insulin deficiency, especially in adolescents[18,19]. The patient presented with insidious onset of disease at puberty and history of DM in her family members which were compatible with type 2 DM. However, the patient was not obese and did not have acanthosis nigricans, which was a sign of insulin resistance[12,18]. Low levels of insulin and C-peptide can be found in both types. Autoantibody markers for example; anti-GAD, anti-AI2 can be found in 90% of type 1 DM patients even though one third of type 2 DM patients can have one of these markers. Therefore, close follow-up and additional investigations were required in this patient.

The presented patient was a good example of DHF occurring with other severe diseases. The patient's presentation was not usual DHF grade III because the patient also had signs of severe dehydration, polyuria despite sustainably rapid pulse rate and increased anion gap metabolic acidosis. Parameters, which can be found in unresponsive shock in dengue, should be evaluated in the same time of reassessment of other possible causes of individual unusual manifestation.

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Potential conflicts of interest

None.

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รายงานผู้ป่วยไข้เลือดออกช็อกระดับ 3 และมีภาวะร่างกายเป็นกรดร่วมกับคีโทน และน้ำตาลในเลือดสูงจากโรคเบาหวาน (Diabetic Ketoacidosis)

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ผู้ป่วยเด็กหญิงไทยอายุ 16 ปี ไม่มีโรคประจำตัวใดๆ มาด้วยไข้เลือดออกช็อกระดับ 3 หลังจากได้รับสารน้ำทางหลอดเลือดดำที่มีความเข้มข้นเท่ากับเลือดเป็นปริมาณมากรวมทั้ง Dextran-40 ปริมาณ 20 มิลลิลิตรต่อกิโลกรัม แล้วผู้ป่วยยังมีชีพจรเต้นเร็วผิดปกติ ความเข้มข้นของเลือดสูง แต่กลับมีปริมาณปัสสาวะมากกว่า 4 มิลลิลิตรต่อกิโลกรัมต่อชั่วโมง เมื่อประเมินผู้ป่วยใหม่พบว่าผู้ป่วยมีภาวะขาดน้ำรุนแรง มีน้ำในช่องท้องปริมาณไม่มาก โดยตรวจไม่พบอาการแสดงของน้ำในช่องปอด ผลเลือดแสดงภาวะเลือดเป็นกรดชนิด increased anion gap ตรวจเพิ่มเติมพบว่าผู้ป่วยมีภาวะน้ำตาลในเลือดสูง ร่วมกับตรวจพบคีโทนและน้ำตาลในปัสสาวะ ต่อมาผู้ป่วยได้รับการวินิจฉัยว่าเป็นภาวะร่างกายเป็นกรดร่วมกับคีโทน และน้ำตาลในเลือดสูงจากโรคเบาหวานชนิดที่หนึ่ง (Diabetic Ketoacidosis, DKA) ได้รับการรักษาด้วยอินซูลินทางหลอดเลือดดำ และได้รับสารน้ำทางหลอดเลือดดำเพื่อแก้ไขภาวะขาดน้ำอย่างรุนแรง ซึ่งการให้สารน้ำในผู้ป่วยรายนี้ต้องให้ด้วยความระมัดระวัง เนื่องจากผู้ป่วยยังอยู่ในระยะวิกฤตของไข้เลือดออก ซึ่งยังสามารถพบน้ำเหลืองรั่วออกนอกเส้นเลือดไปสู่ช่องปอดและช่องท้องได้ ถ้าสารน้ำที่ให้มีปริมาณมากเกินความต้องการอาจทำให้เกิดภาวะน้ำเกินตามมาในระยะพักฟื้นของไข้เลือดออกได้ ปริมาณสารน้ำที่ให้ต้องปรับตามระดับความรุนแรงของภาวะการขาดน้ำ โดยต้องเฝ้าระวังอาการ และอาการแสดงของภาวะน้ำเกินอย่างใกล้ชิด ผู้ป่วยรายนี้พ้นจากภาวะ DKA ที่ 14 ชั่วโมง หลังเริ่มอินซูลิน และหยุดให้สารน้ำทางหลอดเลือดดำได้ที่ 27 ชั่วโมง (36 ชั่วโมงหลังช็อก) ผู้ป่วยรายนี้มีอาการแสดงผิดออกไปจากผู้ป่วยไข้เลือดออกช็อกทั่วไป จึงจำเป็นต้องมีการทบทวนหาสาเหตุซึ่งอาจเกิดจากโรคไข้เลือดออกเองหรือเกิดจากสาเหตุอื่นๆ