

Coexistent dengue haemorrhagic fever and diabetic ketoacidosis: A therapeutic challenge

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Abstract

A 56-year-old Sri Lankan man presented with diabetic ketoacidosis (DKA) and he was in the critical phase of dengue haemorrhagic fever at the time of presentation. The concurrent presence of ketoacidosis and the leaking phase of dengue fever presents a challenge to a practising physician. He was successfully managed with careful fluid resuscitation taking into consideration the leaking phase of dengue fever and fluid depletion from diabetic ketoacidosis. The presence of diabetes increases the severity and the disease mortality in dengue fever. The metabolic disease-induced chronic inflammation and altered vascular endothelial integrity are thought to be the reasons for the increased mortality. Hyperglycemia results in polyuria which can mask oliguria and the depleted intravascular volume and shock status in dengue fever. Dengue fever can rarely precipitate diabetic ketoacidosis in diabetic patients. Using modified goal-directed fluid therapy according to the pathophysiology can avoid fluid overload and prolonged shock in complex situations.

Keywords: Diabetic ketoacidosis, dengue fever, dengue haemorrhagic shock

Background

Dengue is a mosquito-borne infection caused by the dengue virus. It is prevalent in Caribbean, South American, African, and Southeast Asian countries [1]. In Sri Lanka, it has become a major public health burden [2,3]. Sri Lanka has a significant burden of diabetes mellitus among its population [4]. Even though infection is a major precipitating factor for diabetic ketoacidosis (DKA), dengue fever is rarely known to cause DKA [5,6].

There are several potential interactions between dengue fever and DKA. The co-existence of both disease entities is a therapeutic challenge. Timely diagnosis and acute management with aggressive intravenous fluid administration, appropriate replacement of electrolytes, and administration of insulin are required to treat DKA [7]. In contrast, goal-directed careful fluid therapy is crucial in the management of dengue haemorrhagic fever. Hyperglycemia interferes with monitoring the patient's intravascular fluid status by resulting in polyuria even in volume-depleted or shock patients. Metabolic acidosis due to diabetic ketoacidosis can worsen the dengue shock state.

Case presentation

A 56-year-old man was admitted with polyuria, polydipsia, and postural giddiness. He was on aspirin and clopidogrel, having undergone primary percutaneous coronary intervention for inferior ST-elevation myocardial ischemia in 2017. He took losartan for hypertension and was on metformin for his type 2 diabetes mellitus. Further evaluation revealed a history of mild fever five days before admission which had settled at the time of presentation. He is a manual worker, nonsmoker and did not consume alcohol or illicit drugs.

On admission, the patient was conscious and rational, severely dehydrated, with cold peripheries and prolonged capillary refilling time. Blood pressure was 110/80 mmHg, pulse rate 100/min. Pulse volume was low. Lung fields were clear and air entry was equal bilaterally. The abdomen was non-tender, without evidence of ascites. His capillary blood sugar was high (> 600 mg/dl) with positive urine ketone bodies. Metabolic acidosis was evident in the venous blood gas analysis (Table 1).

Urgent fluid resuscitation was commenced as per diabetic ketoacidosis. A haematoma was observed at a site of venous cannulation and that led to the suspicion of the presence of a bleeding tendency or dengue fever. His regular dual antiplatelet and antihypertensives were withheld.

A full blood count revealed a platelet count of 4000/microliter and leukopenia. The full blood count results made dengue fever more likely. A bedside ultrasound scan of the abdomen showed free fluid in the hepatorenal pouch confirming the diagnosis of the critical phase of dengue haemorrhagic fever. Monitoring started assuming that the critical phase commenced six hours before admission. Packed cell volume (PCV) measurement before administration of fluid bolus was 60%. The PCV after administration of the first normal saline bolus (500 ml over one hour) was reduced to 58%. Another 500 ml normal saline bolus over one hour was given. After the second 500 ml bolus of normal saline, PCV was 55%, BP 115/80 mmHg, and pulse pressure was 35 mmHg. The rate of intravenous fluid was reduced to 350 ml over one hour and 250 ml over one hour in the next two hours. Intravenous insulin infusion (0.1 U/kg) continued with hourly capillary blood sugar measurements and electrolyte replacement. Hypokalemia was corrected with potassium chloride 40 mmol along with the normal saline infusions. Fluid therapy was modulated and reduced according to clinical parameters and PCV as per guidelines [3].

After total fluid resuscitation of 1690 ml, bilateral air entry was reduced in lung bases and finger-tip oxygen saturation was reduced to 92% on air. At that time, PCV was 50%, PR 110/min, BP 145/89 mmHg and pulse pressure 36 mmHg. A Dextran bolus (500 ml over one hour) was given, after which the air entry and oxygen saturation were improved, PCV reduced to 41%, tachycardia reduced to 100/min while blood pressure and pulse pressure remained normal. Fluid therapy was scaled down: 350 ml over one hour, 250 ml over one hour, 150 ml over one hour in the next three hours, and then continued as 100 ml/hour.

Insulin infusion was omitted once correction of acidosis was achieved and when bicarbonate was 13.6 mmol/l. He was established on a regular insulin regimen and frequent capillary blood sugar measurement was continued. Eventually, metabolic acidosis was corrected and ketone bodies were cleared.

He recovered from dengue haemorrhagic fever without complications. After recovery, the insulin regimen converted to oral hypoglycemic medication with gliclazide. Antihypertensives and antiplatelet were restarted. He was discharged home after arranging to follow up at the medical clinic.

Discussion

Diabetic ketoacidosis commonly presents with polyuria, polydipsia, nausea, vomiting, and abdominal pain. Orthostatic hypotension could be present at presentation due to intravascular volume depletion [7]. This patient presented with a full constellation of symptoms. The diagnosis of DKA was confirmed with a capillary blood sugar level of more than 600 mg/dl metabolic acidosis of pH 7.28, and positive urine ketone bodies.

The bleeding tendency was suspected because of the appearance of a haematoma during venous cannulation. As our patient gave a history of fever as well, dengue infection was suspected even before the laboratory investigations were available. Once DHF was confirmed goal-directed fluid therapy was instituted. Adequate fluid to restore the intravascular volume was given based on the trend in PCV. Dextran was used as per the guidelines. Blood pressure, pulse pressure, urine output, and PCV were monitored. Arterial blood gas, capillary blood glucose, and serum electrolytes were measured regularly and insulin infusion adjusted and potassium was replaced accordingly. It is important to avoid fluid overload during the management of the dengue leaking phase by avoiding overzealous intravenous fluid therapy. At the same time, it is vital to give adequate fluid when managing DKA to avoid persistent acidosis as it can lead to further complications. It is a therapeutic challenge to achieve the correct balance in fluid management when these two entities coexist. Close monitoring and judicious fluid management help to overcome this challenge.

Conclusion

This case report highlights the importance of considering the possibility of the coexistence of common diseases. It is vital to consider dengue infection when fever and bleeding tendency are noted. We emphasize the value of following the current guideline in managing fluid therapy in both these disease entities as it is paramount to achieve the correct fluid balance to avoid serious complications.

Author declaration

Author contributions: HWSP and KLRK led clinical management of the patient and reviewed the manuscript. WHM performed a literature survey, wrote and edited the manuscript. All authors read and approved the final version of the manuscript.

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Table 1: First venous blood gas analysis

Parameter	Value
pH	7.28
pCO ₂	22 mmHg
HCO ₃	10.3 mmol/l
Base excess	-16.4 mmol/l
Lactate	5.9 mmol/l
Potassium	2.6 mmol/l

Table 2: Daily platelet count chart

Day of febrile illness	D 5 6 pm	D 6 5 am	D 6 2 pm	D 6 6 pm	D 7 5 am	D 7 2 pm	D 8 5 am	D 10 5 am
Platelet count 10 ³ /μl	4	14	13	20	21	59	73	110