

# Hypertension Despite Dehydration in an Adolescent with Diabetic Ketoacidosis

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**ABSTRAK**

*Secara umum, informasi mengenai perubahan tekanan darah pada ketoasidosis diabetik remaja sangat langka. Tujuan artikel ini melaporkan kasus DKA parah pada seorang gadis remaja yang secara tak terduga mengalami hipertensi dari sebelumnya hipotensi.*

*Kasus seorang gadis berusia 17 tahun di unit gawat darurat anak dengan keluhan makan berlebihan selama 6 minggu, buang air kecil yang berlebihan selama 2 minggu, demam selama 1 minggu, muntah selama 4 hari, kesulitan bernafas selama satu hari dan tidak responsif menelepon selama 3 jam. Dia telah mengalami dehidrasi berat namun tidak mengalami hipotensi. Temuan laboratorium termasuk hiperglikemia (glukosa darah sewaktu 20,8 mmol/L; 347 mg/dl), asidosis (serum bikarbonat 5 mmol/L), ketonuria 2+; glikosuria 2+, dan berat jenis urine 1.015. Saat masuk, tekanan darah 100/60 mmHg tetapi semakin meningkat menjadi 140-180/80-100 mmHg pada hari ketiga saat masuk. Hipertensi yang signifikan dapat terjadi pada anak-anak dan remaja yang dirawat karena DKA parah meskipun ada dehidrasi. Oleh karena itu, dokter yang hadir harus waspada terhadap kemungkinan ini.*

**Key words:** ketoasidosis diabetik, dehidrasi, hipertensi.

**ABSTRACT**

*In general, information on blood pressure changes in diabetic ketoacidosis in paediatric population is very scarce. Our aim was to report a case of severe DKA in an adolescent girl who unexpectedly had hypertension rather than hypotension.*

*A 17-year-old girl presented in our Children's Emergency Unit with complaints of excessive eating for 6 weeks, excessive urination for 2 weeks, fever for 1 week, vomiting for 4 days, difficulty with breathing for one day and unresponsiveness to calls for 3 hours. She had moderated to severe dehydration but no hypotension. Laboratory findings included hyperglycaemia (random blood glucose 20.8 mmol/L; 347 mg/dl), acidosis (serum bicarbonate 5 mmol/L), ketonuria 2+; glycosuria 2+, and urine specific gravity of 1.015. At admission, the blood pressure was 100/60 mmHg but progressively rose to 140-180/80-100 mmHg by the third day from admission. A significant hypertension can occur in children and adolescents admitted for severe DKA despite the presence of dehydration. Therefore, the attending physician should be aware of this possibility.*

**Key words:** diabetic ketoacidosis, dehydration, hypertension.

## INTRODUCTION

Diabetic ketoacidosis (DKA) is a serious acute complication of diabetes mellitus. DKA is a state of hyperglycaemic dehydration and ketotic acidaemia.<sup>1</sup> Typically, it is characterized by a biochemical triad of hyperglycaemia, acidaemia/acidosis and ketonaemia/ketonuria. Dehydration from osmotic diuresis due glycosuria is a cardinal feature of DKA.<sup>1,2</sup> Theoretically, dehydration will lead to hypovolaemia and systemic hypotension. To the best of our knowledge there is no published report from Nigeria on blood pressure changes in children and adolescents with DKA. In fact, Deeter et al<sup>3</sup>, stated in their report that there was paucity of information on blood pressure in DKA in the paediatric population. The hyperglycaemia results in osmotic diuresis with the attendant loss of sodium, potassium and magnesium.<sup>1</sup> Measured hyponatraemia is common in DKA. A normal or high serum sodium measurement in the face of severe hyperglycaemia indicates hyperosmolality and dehydration.<sup>1</sup> Dehydration with the resultant hypotension may lead to decreased cerebral perfusion and cerebral ischaemia.<sup>4</sup> Thus, in DKA, both dehydration and cerebral oedema may co-exist. Blood pressure (BP) may be elevated due to cerebral oedema and increased intracranial pressure.<sup>3</sup> In severe cases, Cushing's triad (i.e. hypertension, bradycardia and irregular respiration) may be present.<sup>5</sup> Deeter et al<sup>3</sup>, in their study concluded that BP may not provide an accurate estimate of dehydration in paediatric DKA.<sup>3</sup>

The National Heart, Lung and Blood Institute (NHLBI) charts defined hypertension when either systolic or diastolic blood pressures is equal or greater than the 95<sup>th</sup> percentile for age, height and gender.<sup>6</sup> Although hypotension is not defined in NHLBI charts, it is traditionally defined as systolic BP less than 70 mmHg plus (2 x Age in years).<sup>7</sup> For descriptive purposes, cerebral oedema was defined by clinical evaluation which included alteration in mental status and vital signs that led attending physician (Consultant paediatrician) to administer hyperosmolar therapy (e.g. mannitol).<sup>3</sup>

The purpose of this paper is to report a case of severe DKA in an adolescent girl who unexpectedly developed hypertension rather than hypotension in association with dehydration.

## CASE ILLUSTRATION

Our patient was a 17-year-old secondary school girl who presented in our Children's Emergency Unit with complaints of excessive eating for 6 weeks, excessive urination for 2 weeks, fever for 1 week, vomiting for 4 days, difficulty with breathing for one day and unresponsiveness to calls for 3 hours. The patient was noticed to be losing weight despite increased appetite and consumption of food, even late at night. She had a positive history of polyuria and polydipsia but a negative history of enuresis. This was followed by fever and vomiting. At the onset of symptoms, she was taken to a Public Secondary Healthcare Hospital where she was treated for malaria on outpatient setting because of lack of bed space. She was later taken to another Public Secondary Healthcare Hospital where she was admitted and treated with intravenous fluid and some injections. Following worsening of symptoms, the patient was referred to University of Benin Teaching Hospital (UBTH) as a case of Sick cell anaemia in vaso-occlusive crisis with peptic ulcer disease as a differential diagnosis. There was no history of convulsion or irrational behaviour. She was not previously known to have diabetes mellitus or systemic hypertension. There was no family history of diabetes mellitus and hypertension. Physical examination revealed an acutely ill-looking adolescent girl with altered level of consciousness and a Glasgow coma score of 11/15. She was restless and moderately dehydrated. The pupils were of normal size and reactive to light. She had normal findings on fundoscopy. She had an acidotic respiratory pattern with a respiratory rate of 36 cycles/minute. The lung fields were clear on auscultation. Oxygen saturation was 99%. The pulse rate was 120 beats/minute and blood pressure was 100/60mmHg (percentile). Other cardiovascular examination findings were normal. Her body temperature was 37°C, weight 37kg (<5<sup>th</sup> percentile), length 158cm (10<sup>th</sup>-25<sup>th</sup> percentile) and BMI 15.6kg/m<sup>2</sup> (<5<sup>th</sup> percentile). Her point-of- admission laboratory findings are summarized in **Table 1**.

A diagnosis a ketoacidosis in a newly-diagnosed type-1 diabetes mellitus was made. The patient was commenced on 0.9% saline infusion at 20 ml/kg over the first one hour. The

**Table 1.** Summary of laboratory findings at the point of admission

Parameter	Result	Comments
Random blood glucose	20.8 mmol/L	Hyperglycaemia
Serum sodium	124	Hyponatraemia
Serum potassium	4.8	Normokalaemia
Serum chloride	90	Hypochloridaemia
Serum bicarbonate	5	Severe acidosis
Serum urea	31	Normal
Serum creatinine	0.7	Normal
Urine ketone	2+	Ketonuria
Urine glucose	2+	Glycosuria
Urine specific gravity	1.015	Elevated
Blood in urine	Negative	No haematuria
Urine protein	Negative	No proteinuria
Urine culture	Yielded no growth	Sterile
Blood culture	Yielded no growth	Sterile
Total white blood cell	21.6 x 10 <sup>3</sup> /μL	Leucocytosis

remaining fluid was to be given over the next 48 hours. She was commenced on continuous insulin infusion at 0.1 IU/kg/hr. As a policy, intravenous antibiotics (ceftriaxone) was administered. At point of admission the BP was 100/60mmHg (no hypotension). On the 3<sup>rd</sup> day of admission, she was still ill looking and dyspnoeic. With increase in BP and deterioration in level of consciousness, a diagnosis of DKA complicated by cerebral oedema was made. Intravenous mannitol was commenced but this intervention did not help. The blood pressure ranged between systolic 140 to 170 mmHg (percentile) and diastolic 80 to 100 mmHg (percentile). All the blood pressure measurements were performed with the patient lying in supine position, using mercury sphygmomanometer with appropriately sized arm cuffs and recorded in the patient's charts. She still had signs of moderate to severe dehydration co-existing with hypertension. The patient was subsequently transferred to the Intensive Care Unit of the hospital. Her clinical condition continued to deteriorate until her demise on the 5<sup>th</sup> day of admission.

## DISCUSSION

At the point of admission, our patient did not have hypotension despite the presence of

moderate to severe dehydration. Her blood pressure reading was within normal limits. DKA, being a hypovolaemic state is expected to be associated with hypotension rather than hypertension. Unexpectedly, on the third day of admission our patient developed hypertension which persisted till her demise, despite efforts to control the cerebral oedema with mannitol administration. A similar observation had been reported previously in a study involving children below 18 years old conducted at Seattle Children's hospital, USA.<sup>3</sup> DKA may result in both dehydration and cerebral oedema. However, these two pathologic processes may have opposing effects on blood pressure.<sup>3</sup> Several mechanisms have been postulated to explain the occurrence of hypertension despite dehydration in patients with DKA. Cerebral oedema may lead to increase in intracranial pressure (ICP) and subsequent increase in systemic BP aimed at maintaining cerebral perfusion.<sup>5</sup> Another mechanism for hypertension in DKA is stress response via increase in catecholamine levels following small increases in cerebral oedema and ICP.<sup>8</sup> In addition, the levels of other counter-regulatory hormones (such as glucagon, cortisol and growth hormone) and proinflammatory cytokines have been shown to be elevated in DKA. These chemical substances may have effect on BP acutely.<sup>9,10</sup> Hyperosmolality leads to release of anti-diuretic hormone which increases BP via V2 receptors.<sup>11</sup> Over-activity of the rennin-angiotensin system as well as arginine vasopressin (known to be highly activated in DKA) is also a possible cause of the observed hypertension.<sup>8,11,12</sup>

There was no specific therapy given for the hypertension. This was because of the uncertainty regarding the pathophysiological process involved in the development of cerebral oedema in DKA. If cerebral ischaemia is involved as the major causal pathway leading to cerebral oedema, then hypertension may be a physiological response to maintaining cerebral perfusion and would be desirable and protective.<sup>3</sup> On the other hand, if cerebral hyperaemia is the main pathophysiological process, then treating the hypertension may be warranted as a therapy to limit vasogenic cerebral oedema.<sup>13,14</sup> All

of these reflect paucity of information on the subject. There is, therefore, a need for further studies to assess the role and aetiopathogenesis of hypertension in paediatric DKA. Such knowledge will aid management of cases with DKA and hypertension.

## CONCLUSION

Greater awareness and perhaps, more frequent measurement of blood pressure is justified throughout the period of hospital admission and during outpatient follow-up visits.

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