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Case Report

Diffused Cerebral Microhemorrhage in a 15-Year-Old Boy with Ketoacidosis due to Undiagnosed Type I Diabetes Mellitus: A Case Report

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Abstract:

Diabetes mellitus is a chronic endocrine disorder characterized by impaired regulation of blood glucose metabolism, leading to elevated blood sugar levels. This condition can result in a wide range of acute and chronic complications, significantly impacting patients' quality of life and overall health. Among the most severe acute complications are hyperosmolar hyperglycemic state (HHS) and diabetic ketoacidosis (DKA), both of which are life-threatening if not promptly treated. DKA, in particular, is more commonly associated with type 1 diabetes mellitus and is caused by a severe insulin deficiency, leading to metabolic acidosis, hyperglycemia, and the accumulation of ketones. Neurological complications, such as cerebral edema and cerebrovascular events, are also well-documented consequences of DKA, particularly in pediatric patients. However, intracerebral microhemorrhages, which are small bleeds within the brain tissue, have not been previously reported in the literature as a complication of juvenile DKA. This case report describes a 15-yearold boy who was newly diagnosed with type 1 diabetes mellitus and subsequently developed an intracerebral microhemorrhage following an episode of DKA. The report highlights the clinical presentation, diagnostic imaging findings, and management of this rare complication. Radiological evidence, including magnetic resonance imaging (MRI), revealed the presence of microhemorrhages, providing valuable insights into the potential neurological risks associated with DKA in pediatric patients. This case underscores the importance of vigilant monitoring for atypical complications in juvenile diabetes, particularly during acute metabolic crises like DKA. Further research is needed to understand the underlying mechanisms and risk factors for such rare neurological events in diabetic patients.

Key words: diabetic ketoacidosis; microhemorrhage; microbleeding; radiologic findings; juvenile

Introduction

Type 1 diabetes mellitus (DM1) is one of the most prevalent chronic conditions affecting children. The global incidence of DM1 is rising steadily, with an increase of 3-5% each year. A significant complication associated with DM1 is pediatric diabetic ketoacidosis (DKA), which occurs in up to 30% of young patients with the condition and is considered a leading cause of mortality in this group [1]. According to guidelines from the American Diabetes Association [2], the European Society for Pediatric Endocrinology, and the Pediatric Endocrine Society [3], DKA is characterized by high blood sugar levels (greater than 200 mg/dl or about 11 mM) and either a venous pH below 7.3 or bicarbonate levels under 15 mM [4, 5]. One of the most serious complications of DKA is cerebral edema, which continues to be a topic of active research and discussion. Other rare complications can include coagulopathy,

rhabdomyolysis, and issues affecting the lungs and gastrointestinal tract [1]. In this report, we discuss a case involving a 15-year-old boy who developed intracerebral microhemorrhage as a result of juvenile DKA.

Case report

A 15-year-old boy who had previously been healthy presented with polyuria, polydipsia, and anorexia. The patient had presented nausea and vomiting from 15 days prior to hospital admission. At the admission time, hyperglycemia, nausea and vomiting, tachypnea, and decreased consciousness level were noted. The plasma pH was found to be 6.83 which showed an acidosis. The patient's vital signs and venous blood gas results are presented in Table 1.

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Ketonuria and glucosuria were noted in the results of the urinalysis. The patient with the diagnosis of DKA was transferred to the pediatric intensive care unit. After admission, appropriate fluid resuscitation, and treatment with regular insulin were initiated. His DKA was resolved after 3 days after the admission. The blood sugar was thoroughly controlled within the range between 200 and 300 mg/dl.

After 10 days of admission, the patient was subjected to functional endoscopic sinus surgery (FESS) due to the conjecture of mucormycosis infection.

Case Report

A previously healthy 15-year-old boy has been come to the hospital with symptoms of increased urination, excessive thirst, and a lack of appetite.

For the past 15 days, he had also been experiencing nausea and vomiting. Upon admission, he exhibited signs of hyperglycemia, along with nausea, vomiting, rapid breathing, and a decreased level of consciousness. Blood tests revealed a plasma pH of 6.83, indicating acidosis. His vital signs and venous blood gas results are detailed in Table 1.

The urinalysis showed the presence of ketones and glucose, confirming the diagnosis of DKA. He was promptly transferred to the pediatric intensive care unit, where he received appropriate fluid resuscitation and regular insulin treatment. After three days, his DKA was resolved, and his blood sugar levels were stabilized within the range of 200 to 300 mg/dl. Ten days after being admitted, the patient underwent functional endoscopic sinus surgery (FESS) due to concerns about a possible mucormycosis infection.

	Nausea and vomiting	
Clinical manifestation on admission	Tachycardia	
	Hyperglycemia	
	Drowsiness	
	Decreased consciousness level (GCS 4-5)	
Vital signs	Blood pressure (mmHg)	Systolic: 143
		Diastolic: 94
	Heart rate (beat.min ⁻¹)	105
	Respiratory rate (breath.min ⁻¹)	55
	O_2 saturation (%)	100
Venous blood test	pH	6.83
	P CO ₂ (mmHg)	13
	P O ₂ (mmHg)	49.2
	HCO ₃	2.1

Table 1: Clinical manifestation on admission, vital signs, and venous blood gas test results obtained on admission

Radiologic representations

Brain computed tomography (CT) and magnetic resonance imaging (MRI) were performed for further investigations regarding

unconsciousness. Initial CT and MRI revealed a diffused micro-bleeding in the cerebrum. Figure 1 presents the patient's CTs and MRIs on the first and fourth days after admission.



Figure 1: Radiological representations of the case; (A) Hyposignal change in axial cutoff T2* sequence of the subcortical white matter of both cerebral hemispheres is indicative of microbleeding; (B) Severe mucosal thickening in all paranasal sinus associated with irregularity and destruction of the medial walls of maxillary sinuses is suggestive of mucormycosis sinusitis.; (C) A round lesion with peripheral rim enhancement in the right

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frontal lobe associated with surrounding vasogenic edema in post-contrast axial brain CT was observed.; (D) Hyposignal change in subcortical white matter in axial cutoff T1 sequence is indicative of intracerebral hemorrhage.

Discussion

Neurological complications that may be encountered in those with DM can be classified into central and peripheral neuropathies. Encephalopathy, myelopathy, acute neuropsychological disorders on the background of decompensation of metabolism, and TIA/stroke as the result of impairments to cerebral circulation have all been defined previously [6].

Moreover, DKA, a state of severe insulin deficiency, resulting in hypoglycemia and ketonemia, is a well-known complication of type I DM, which carries a high risk of morbidity and mortality.

Major neurological complications of DKA including cerebrovascular accident (CVA), brain edema, and less common hemorrhage in white matter [7, 8], have been observed in 3-10/1000 cases and may result in an adverse outcome [9].

Brain edema is an uncommon, but fatal complication of severe DKA and its treatment. Several metabolic factors including ketone bodies, immuneinflammatory responses, and oxidative-nitrosative stress have been postulated to be responsible for the alteration of transcellular permeability and disruption of the blood-brain barrier, thus resulting in brain edema [10]. Less common and perhaps underestimated neurologic complication is the risk of infarction and hemorrhage during an episode of DKA. It is estimated that acute ischemia and hemorrhage are observed in up to 10% of those with neurological manifestations [11, 12]. Abnormalities in the coagulation cascade, increased platelet activity, and vasoconstriction may all have an additive role [10].

On the other hand, during an episode of DKA, the risk of hemorrhage is theoretically increased by endothelial disruption, caused by a proinflammatory state and oxidative injury [13-15].

Concomitant DIC sometimes has been reported together with DKA, which may further predispose to a hemorrhagic event due to the consumption of coagulation factors. In one radiologic study, 4 out of 23 children with DKA had subarachnoid or intraventricular hemorrhages. Three patients had both hemorrhage and cerebral edema. [8] Another case report reveals intraparenchymal hemorrhage secondary to cerebral vein thrombosis [16], however, to the best of our knowledge, the presence of microhemorrhage has not been previously reported in DKA. Cerebral microbleeds are defined as small foci of accumulated hemosiderincontaining macrophages, with a maximum total size of 5-10 mm [17]. These have been described in adults with an increasing frequency in the elderly, hypertensive encephalopathy, and cerebral amyloid angiopathy [17]. Cerebral microbleeds in our case, in the setting of DM I/DKA could probably be explained by the presence of microemboli and/or the inflammatory state that accompanies DKA, resulting in diffuse endothelial injury [10]. As CMB is associated with an increased risk of hemorrhagic and ischemic infarction, the detection of microhemorrhages could be of clinical significance. This is best done by implication of conventional T2*-weighted MRI or susceptibility-weighted imaging (SWI). These imaging techniques facilitate and enhance the depiction of microhemorrhages, which could have been otherwise overlooked in routine MRI sequences, especially when they are small.

Conclusion

In diabetic patients who are clinically suspicious of the presence of neurologic complications, neuroimaging is advisable. Brain CT and MRI may reveal brain edema, ischemic infarction, or hemorrhage in such cases. The application of optimized MR imaging techniques significantly improves the detection of possible intracranial hemorrhage. Early diagnosis is of the utmost value in patient management and may reduce associated morbidity and mortality.

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Conflict of interest disclosure

The authors have no conflict of interest to declare regarding this case report.

Authors contribution

Pooya Iranpour: Conceptualization and supervision, reviewing and proofreading; Mansooreh Jalilpour and Arash Teimourian: Data curation, formal analyses; Moein Masjedi: Original draft preparation, editing, reviewing and proofreading.

Ethics approval statement

Not applicable (All personal information related to the patient was kept confident)

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Not applicable

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