Posterior reversible encephalopathy syndrome complicating diabetic ketoacidosis

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Introduction

Posterior reversible encephalopathy syndrome (PRES) was first described by Hinchey et al. in 1996[1]. The most common clinical features are headache, altered sensorium and seizures[2]. According to our knowledge there is only a single reported case of PRES associated with diabetes ketoacidosis (DKA) in a child[3].

Case report

A previously healthy 12 year old boy presented to the emergency department with pain in abdomen and vomiting for 7 days. On examination, he had normal sensorium and maintained saturation in room air. He had tachypnoea, tachycardia and a normal blood pressure (BP). He was severely dehydrated but the neurological examination was normal. His blood gases showed severe metabolic acidosis (pH=6.95, bicarbonate 7, base deficit 21, pCO_2 23). He was diagnosed to have severe DKA (blood glucose 372mg/dl, ketones ++ in urine). The child was managed with intravenous (IV) fluids and insulin infusion according to protocol. Monitoring continued with capillary blood glucose, arterial blood gas (ABG) and serum electrolytes. On day 2, severe hypokalaemia was noted (K=2.1 mmol/l), which was corrected as per standard protocol, but other electrolytes were within normal range. Over the next 48 hours, the child remained irritable and confused with episodes of tachyarrhythmia. His BP and urine output remained normal throughout. After 48 hours of treatment, the child improved and oral feeds and subcutaneous insulin were started.

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On day 4, the child started complaining of blurring of vision. Pupillary response to light, funduscopic examination and eye movements were all normal. Computed tomography (CT) scan of the brain, done to rule out cerebral oedema, was normal. His vision improved with no headache and the child was discharged after 10 days on subcutaneous insulin. The same day the child was brought back to the emergency department in status epilepticus, with up-rolling of eyes, tongue biting, stiffening of body, altered sensorium and involuntary passage of urine. In the emergency department, blood glucose was 295 mg/dl, ABG was normal, and there was a trace of ketones in the urine. His BP was recorded at 140/90 mm Hg. Convulsions subsided with antiepileptic drugs which were continued. Next day he had two further episodes of generalized tonic-clonic seizures. He was put on maintenance valproate, with no further seizures. EEG was consistent with diffuse encephalopathy. But confusion and irritability persisted. CT scan of brain showed bilateral parieto-occipital oedema. Magnetic resonance imaging (MRI) with FLAIR was consistent with the diagnosis of PRES (Figure 1). Venous sinus thrombosis was excluded by MRI venography.

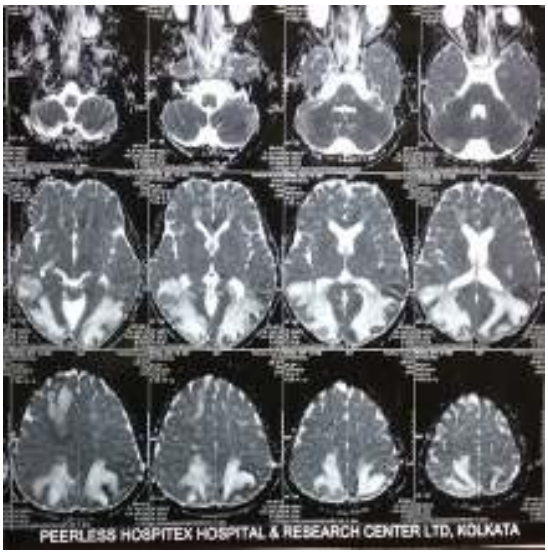

Figure 1: ***Magnetic resonance imaging of brain showing bilateral parieto-occipital distribution***

On day 2 of admission, several episodes of hypertension were recorded (BP up to 160/110 mm Hg) which were treated with labetalol infusion. Due to the appearance of fever spikes, relevant investigations were done. Except for leucocytosis and increased CRP, all other results were within normal limits. Lumbar puncture was done and the cerebrospinal fluid (CSF) study was normal. Investigations were done to rule out secondary causes of hypertension like autoimmune diseases and vasculitis [ANA, p-ANCA, c-ANCA – non reactive, c_3, c_4-normal]. Renal Doppler study ruled out renal cause for the hypertension. Gradually fever subsided and BP stabilized with antihypertensives. His sensorium improved after 7 days. He continued to improve and made a full recovery. Repeat CT scan showed resolving oedema after 3 weeks. Repeat MRI is planned in 6 months. At discharge, his BP was 100/68 mmHg. He was discharged on insulin, valproate and antihypertensives.

Discussion

In the only reported case of PRES associated with DKA in the literature, a 17 year old girl developed seizures, encephalopathy and cortical blindness due to PRES, after resolution of her DKA[3]. In this patient, hypertension was identified as the likely trigger for PRES[3]. Abrupt arterial hypertension is a known trigger for PRES[4]. In our patient, too, PRES occurred after resolution of the DKA and sudden onset of hypertension was the probable trigger for PRES. Typical MRI changes in the brain in PRES comprise bilateral hyperintensities predominantly in the parieto-occipital region[4]. This was present in our patient. The clinical outcome of PRES is excellent, recovery occurring within a few days although the resolution of the MRI abnormalities takes much longer[4]. Our patient showed clinical improvement within a week and the repeat MRI will be done in about 6 months. PRES is an unusual but nevertheless treatable complication of DKA. Any child with DKA with late deterioration, with seizures and altered sensorium merits exclusion of PRES, preferably by MRI brain.

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References

1. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, et al. A reversible posterior leukoencephalopathy syndrome. *New England Journal of Medicine* 1996; **334**: 494-500. https://doi.org/10.1056/NEJM199602223340803 PMid: 8559202

2. Incecik F, Hergüner MO, Altunbasak S, Erbey F, Leblebisatan G. Evaluation of nine children with reversible posterior encephalopathy syndrome. *Neurology India* 2009; **57**(4):475–8 https://doi.org/10.4103/0028-3886.55605 PMid: 19770551

3. Jones R, Redler K, Witherick J, Fuller G, Mahajan T, Wakerley BR. Posterior reversible encephalopathy syndrome complicating diabetic ketoacidosis; an important treatable complication. *Pediatric Diabetes* 2016 [Epub ahead of print] https://doi.org/10.1111/pedi.12362

4. Roth C, Ferbert A. The posterior reversible encephalopathy syndrome: What's certain, what's new? *Practical Neurology* 2011; **11**(3): 136-44. https://doi.org/10.1136/practneurol-2011-000010 PMid: 21551107