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Case Report

Unusual manifestation of the central hypothyroidism

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ABSTRACT

The central hypothyroidism occurs in one in 16,000–30,000 infants. The common presentations include lethargy, sluggishness, hoarse cry, and feeding. Seizure and apnea are a rare manifestations. Herein, we describe the case of an infant with the central hypothyroidism who presented with seizures.

Keywords: Hypothyroidism, Seizures, Infant

INTRODUCTION

The central hypothyroidism can occur in any condition associated with developmental defects of pituitary or hypothalamus. The central hypothyroidism occurs in one in 16,000–30,000 infants.[1] Deficiency of thyroid-stimulating hormone (TSH) may be caused by mutation in the genes coding for transcription factor essential for pituitary development or thyrotrope cell stimulation. Clinical manifestations are often subtle at birth due to placental transfer of T4. Common presentations include lethargy, sluggishness, hoarse cry, feeding problems, constipation, macroglossia, large fontanelle, umbilical hernia, hypotonia, delayed bone age, dry skin, and hypothermia. Congenital hypothyroidism can rarely present with seizures[2-4] and apnea.[5] Herein, we report a case of an infant who presented with seizures, secondary to the central hypothyroidism.

CASE REPORT

A 2½-month-old female child with normal antenatal and perinatal history presented with complaints of noisy breathing since birth and cough since 4 days. At admission, infant had an episode of convulsion in the form of uprolling of eyeballs and posturing of all four limbs associated with bluish discoloration of the body. Child also had an apneic spell associated with cyanosis and bradycardia, during course of hospital stay for which child was intubated. Child had multiple episodes of convulsions on ventilator and which settled with anticonvulsants.

On examination, child had coarse facies and large protruding tongue [Figure 1]. No evidence of external congenital defect. The other systemic examination was unremarkable. Laboratory investigations revealed CBC- and chest-X-ray as normal. Metabolic work up including GRBS (98 mg/dl), serum calcium (7.9 mg/dl), magnesium (1.8 mg/dl), and serum electrolytes (Na+136mmol/l, K+5.5mmol/lt) was within normal limits. Echocardiography was normal. EEG showed abnormal generalized electrical activity. CT brain showed no abnormalities [Figure 2].

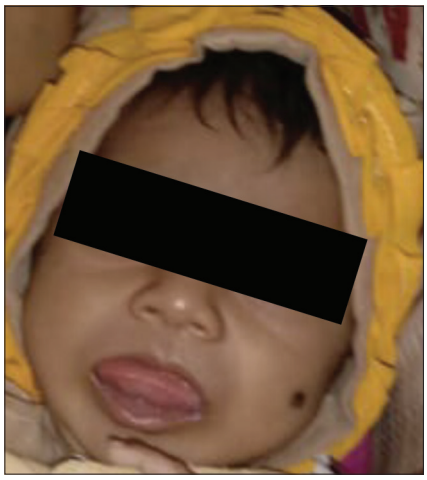

Figure 1: Case of central hypothyroidism in the infant showing coarse facies and large protruding tongue.

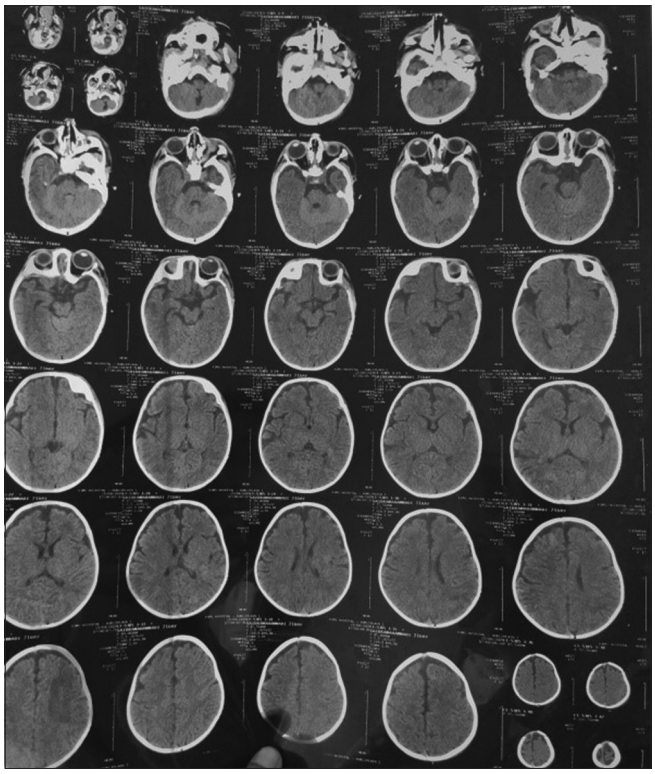

Figure 2: CT brain of the infant showing no significant neuroparenchymal abnormality.

As the child had coarse facies and large tongue, the infant was evaluated for hypothyroidism, TFT was sent after seizures were controlled, and was found to have reduced TSH (2.7 microunits/ml), with normal levels of free T3 (2.86 pg/ml), and free T4 (0.9 ng/dl) suggestive of the central hypothyroidism. However, workup for other pituitary hormones was not done. The child was started on l-thyroxine (8 µg/kg).Child improved with the treatment and was on mechanical ventilator for 6 days, there were no further episodes of convulsions. The child was discharged after 15 days of hospital stay with l-thyroxine. After 3 months during follow-up repeat free T4 levels (1.4 ng/dl) done was normal, no signs of thyrotoxicosis and the child is doing well.

DISCUSSION

Till date, only four case reports of congenital hypothyroidism presenting with seizures have been reported worldwide, of which one was from India.[4] In our case, the child presented with convulsions but during hospital stay, had apnea which improved with l-thyroxine therapy. The infant reported here had hypothyroidism that presented in the infantile period with seizures unresponsive to conventional therapy. Decreased levels of TSH and low/normal levels of T4 are confirmatory for this disorder. In our child, the central hypothyroidism was attributed as the cause for seizures because as testing for, infectious etiology and metabolic causes were normal and response to thyroxine.

The exact mechanism is not known; however, the proposed mechanism as per several animal studies has shown that congenital hypothyroidism has been associated with excessive or abnormal synchronous neuronal activity in the neocortex and hippocampus indicating the pathophysiological process leading to seizures.[6] Moreover, neurotransmitter regulation, synaptogensis, neurogenesis, neuronal migration, axon, and dendrite formation are all dependent on thyroxine.[3]

CONCLUSION

Although hypothyroidism presenting as seizure is a rare entity, the high index of suspicion is required when unresponsive to conventional management.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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