LETTER TO EDITOR (VIEWERS CHOICE)

COMMENT ON CONGENITAL HYPOTHYROIDISM LEADING TO ACUTE KIDNEY INJURY WITH HYPERNATREMIC DEHYDRATION: A LETTER TO EDITOR

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To the Editor,

In the present journal, a case report of Congenital Hypothyroidism Leading to Acute Kidney Injury with Hypernatremic Dehydration was published. (1) We read it with great interest. There are certain things which need clarification. First and foremost is it is not congenital hypothyroidism, which is directly implicated in the pathogenesis of Acute Kidney Injury (AKI) and dehydration. The author stated that the baby was having poor feeding and that is the cause of dehydration and AKI. Any child with lethargy and poor feeding can have dehydration so to state hypothyroidism as a cause of hypernatremia will not be true. Second; author mentioned that the baby had muffled heart sounds, but didn’t mention whether there was pericardial effusion or not; which is a well-known entity in hypothyroidism. (2) Third; author mentioned that child was initially rehydrated with two boluses 60cc/kg of 0.9% normal saline; what is the rationale for that. According to guidelines of fluid resuscitation in newborns boluses of 10 ml/kg saline upto 60 ml/kg should be given unless perfusion improves or hepatomegaly develops. (3) Fourth; author mentioned that ultrasound abdomen revealed bilateral medullary nephrocalcinosis (secondary to dehydration); probably it was increased in the attenuation of the renal medulla secondary to dehydration, which is commonly known as “dense renal medulla” sign which is well described in the literature. (4,5) Dense renal medulla is defined as the increased attention of the medulla as compared to the renal cortex. It is seen in conditions which increase urine osmolality like dehydration, hypernatremia and high-protein diet. Medullary nephrocalcinosis is one of the differential diagnosis. The disappearance of this hyperdensity following adequate hydration clinches the diagnosis. So; whether we repeated ultrasonography in this baby or not?

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References :

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A 1 year old female child born of non-consanguineous marriage presented with bluish discoloration of skin and mucosa since 6th months of life. Echocardiography done at 6th months of life was normal. Child had global developmental delay with a developmental quotient of 25%. She was the 3rd child born to G3P2L2 mother. Mother had history of intrauterine death at 6th months of first pregnancy. On examination, the child had central cyanosis and microcephaly. On Central nervous system examination, there was generalized hypertonia and brisk deep tendon reflexes. Other systems were normal. Pulse oximeter showed an oxygen saturation (So2) of 76% at room air which increased to 83% on administration of oxygen. Venous blood drawn was dark chocolate brown in colour (Figure 1). Chest x-ray and repeat echocardiography were normal. Arterial blood gas analysis revealed a pH of 7.35, pO2 252, So2 98%. The simultaneous pulse oximetry showed a saturation of 74%. MRI brain was normal. Methemoglobin levels were 24.7% by biochemical method. In view of global developmental delay, generalized hypertonia, saturation gap, dark colored venous blood and high methemoglobin levels a diagnosis of congenital methemoglobinemia type II was made. Child was started on high dose ascorbic acid following which cyanosis reduced clinically.

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