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?

Funding : None

Conflict of Interest : None

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DOI : 10.7199/ped.oncall.2017.22