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RESEARCH ARTICLE

ATYPICAL PRESENTATION OF UTI INFANCY: A REPORT OF 2 CASES

Khalid A.M Rahama[1], Mahmoud Al Hussain[2], Rikaz Bizzarri[3] and Eyas A. Mukhtar[4]

1. SSR Pediatrics Department, HH, DH, UAE.
2. Consultant Pediatrics Department, HH, DH, UAE.
3. Head of Pediatrics Department, HH, DH, UAE.
4. SSR Pediatrics Department, HH, DH, UAE.

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Abstract

We reporttow male infant with UTI and hydronephrosis who presented with profound hyponatremia and hyperkalemiadue to transient type 1 pseudohypoaldosteronism, which responded rapidly to intravenous normal saline and antibiotics. In infants with renal anomalies,it is important to highlight the association of type 1 PHA and UTI since prompt and appropriate treatment rapidly corrects the associated electrolytes abnormalities.

Introduction:-

UTI is the most common bacterial infection in childhood [1] and up to 30% of infants and children experience recurrent infections during the first 6–12 month after initial UTI [2]. In very young infants, symptoms of UTI differ in many ways from those in older infants and children. The prevalence of UTI is higher in the first year of age, with a male predominance. Most infections are caused by Escherichia coli, although in the first year of life Klebsiella pneumoniae, Enterobacter spp, Enterococcus spp, and Pseudomonas are more frequent than later in life, and there is a higher risk of urosepsis compared with adulthood [3,4,5] .

The incidence of UTIs depends on age and sex. In the first year of life, UTIs are more common in boys (3.7%) than in girls (2%). This is even more pronounced in febrile infants in the first 2 month of life, with an incidence of 5% in girls and 20.3% in uncircumcised boys, as demonstrated in one prospective study of >1000 patients using urine specimens obtained by catheterisation [4] . Later, the incidence changes, and about 3% of prepubertal girls and 1% of prepubertal boys are diagnosed with a UTI [3,4,5].Most children receive antibiotics and recover without any complications. UTI often presents with fever, vomiting and poor feeding in infants; however it can present in other ways like failure to thrive, electrolytes disturbances.

Severe hyponatraemia and hyperkalaemia is a medical emergency in infancy and usually points to adrenal failure, typically congenital adrenal hyperplasia salt-wasting (CAH)[6], congenital adrenal hypoplasia, however, alternative diagnoses involving inadequate mineralocorticoid secretion or action must be considered. The presence of UTI and renal anomalies should alert to the possibility of type 1 pseudohypoaldosteronism[7,8,9,10,11,12,13, 14,15]. UAE Neonatal screening program was started in 1995 then expanded where CAH screening was established in 2007, the total incidence of classical CAH detected through newborn screening in the UAE was 1in 9 030[19].

Corresponding Author:- Khalid A.M Rahama
Address:- SSR Pediatrics Department, HH, DH, UAE.

Methods:-

This was retrospective review the medical records of 2 infants admitted to our hospital and seen by our pediatric specialist and consultant between January to December 2014. All information was obtained from patient notes during our evaluations of each patient and complemented with laboratory, imaging, and follow-up data obtained from the patient records and electronic medical record.

Case report

Patient 1 was a7 month-old uncircumcised, previously healthy male, mother had GDM, with an uncomplicated birth history initially seen by his primary physician for failure to thrive (table 1), poor feeding and solid food refusal. The infant had no, fever GIT, urinary ,cardiorespiratory symptoms or skin rash ,neonatal screening was normal, the infant was active ,thin ,not cyanosed ,clubbed or dysmorphic, systemic examination was normal. No family history of renal or endocrine diseases. Initial urinalysis revealed pyuria, but considered contamination ,subsequently his serum electrolytes obtained and revealed hyponatremia and hyperkalemia, and he was treated with normal saline and his repeat urinalysis revealed pyuria treated with cefuroxime ; the urine eventually grew E coli .His serum sodium on admission was 118 mmol/L with a serum potassium 5.8 mmol/L which were corrected with saline and antibiotic ,urinary sodium after the treatment was less than 10, 17 OH progesterone was normal (table 2) . Renal sonogram revealed unilateral hydronephrosis and hydroureter, MCUG showed no VUR or posterior urethral valve. Due to technical difficulties serum aldosterone and rennin were not processed. In addition to treatment for her urinary tract infection, the patient was sent home oncotrimoxazole prophylaxis.

Table 1:- Growth parameters.

Date	27/2/14(birth)	5/3/14	5/5/14	2/7/14	5/11/14	12/11/14	10/12/4
Weight	3.8	4	4	7.1	6.3	6.2	7.9
Height	52	57	57	64	66	66	68
Head circum	35.5	36.5	36.5	42	42	42	43

His electrolytes remained normal on follow up. One month later, he developed E coli UTI with no electrolytes disturbance a dynamic renal scan showed equal function of both kidneys, with no evidence of obstruction.

Table 2:- The electrolytes.

Date	12/11/14	13/11/14	14/11/14	18/11/14
Na	118	124	129	133
K	5.8	3.9	3.6	4.5
CL	84	94	94	100
Urea	25	8	5	9
AG	16	11	12	7
CO2	16.4	20.4	22.9	25.9
creatinine	0.1	0.1	0.1	0.1

Patient 2 was a 48 day male infant with uneventful pregnancy and birth history seen due to vomiting, loose motion, fever and fussiness for one week, just before circumcision and became obvious after that. The infant had no urinary, cardiorespiratory symptoms or skin rash, neonatal screening was normal, the infant was dehydrated, less active, thin, not cyanosed, or dysmorphic, systemic examination was normal with normal male genitilia. No family of renal or endocrine diseases. At our facility the serum sodium was 113 mmol/L and serum potassium 7.5 mmol/L. Initial the infant treated with normal saline and urinalysis showed pyuria. Then he transferred to our ward with the presumptive diagnosis of UTI, started on intravenous saline and cefuroxime. Congenital adrenal hyperplasia was excluded as the 17- hydroxyprogesterone level was normal; at that point, urinalysis revealed pyuria, and urine culture was positive for Klebsiela pneumoniae, cefuroxime was continued. Renal sonography showed right hydronephrosis,voiding cystourethrogram demonstrated no vesicoureteral reflux, and dynamic renal study right hydronephrosis with PUJ obstruction with good function. Serum sodium and potassium improved after 12hour and normalized after 2 days of intravenous fluids and antibiotic therapy and without steroid. The diagnosis of pseudohypoaldosteronism was suspected and the serum aldosterone level was not obtained and his electrolytes remained normal. The patient was discharged from the hospital on trimethoprim prophylaxis. He was admitted twice one due to RSV bronchiolitis, and the other due pneumonia and wheezes and further UTI episode.

Table 1:- The electrolytes.

Date	10.8.14 morning	10.8.14evening	12.8.14
Na	113	123	134
K	7.5	4.5	4
Cl	79	89	96
CO2	19	19	24
Urea	81	44	12
AG	15	15	14
Creatinine	0.4	-	-

Discussion:-

One of our patient presented failure to thrive and electrolytes derangement while the other infant presented with vomiting, loose stool, weight loss and dehydration with electrolytes derangement. This electrolytes abnormalities was surprising, the extremely low sodium was not expected. Pseudohypoaldosteronism (PHA) was reached as a cause for this after more common causes were considered and eliminated by history, normalization of electrolytes and normal 17OHP. PHA is relatively rare, but may be under-recognized and sometime confused with CAH. The classification of PHA includes PHA Type 1 (subdivided into primary and transient, or secondary forms) and PHA Type 2. Primary PHA type 1 can be autosomal recessive or dominant, with the recessive form presenting early and having a severer clinical course, requiring lifelong sodium supplementation[18]. The autosomal dominant form may be asymptomatic, or present later with salt-wasting and hyperkalaemia; sodium supplementation requirement declines with age. They are characterized by mutations in the mineralocorticoid receptor or in the genes encoding subunits of the epithelial sodium channels, leading to renal or generalized resistance to aldosterone. Transient PHA is probably the cause of the hyponatraemia and hyperkalaemia in our infants, who were subsequently found to have UTI associated with hydronephrosis. Transient PHA has also been reported following small bowel resection, and with calcineurin immunosuppression (for example, cyclosporine and tacrolimus).

PHA Type 2 (also known as familial hyperkalaemic hypertension) is a rare autosomal dominant condition. Serum aldosterone levels are normal despite high potassium, therefore suggesting a degree of aldosterone resistance.

There are several reports relating PHA with urinary tract abnormalities in children. Several other case reports covering the newborn period and in infancy showed association of urinary tract malformations and UTI with PHA [7,8,9,10,11,12,13, 14,15]

Obtaining and processing a serum aldosterone level can be difficult because it is usually sent to a reference laboratory. Family history, laboratory studies, and the presence of urinary tract malformations may help to differentiate primary genetic PHA type1 from secondary or transient PHA type 1.[7,8,9,10,11,12,13, 14,15] The transient PHA can be differentiated from genetic causes by its duration which is transient and also by rapid correction of electrolyte imbalance with the treatment of UTI. This condition is almost exclusively seen in infancy as the diet is relatively low in sodium. In face of increased urinary sodium loss, infants are unable to compensate by increasing salt intake compared to older children.

The inference from our cases study is that diagnosis of transient PHA needs to be considered in any infant presenting with hyponatraemia, hyperkalemia andweight loss. One infant was not febrile at the time of presentation, so this condition needs to be considered in any afebrile patient with hyponatremia[15]

Conclusion:-

The acute clinical and biochemical presentation of transienttype 1 PHA is identical to adrenal failure and may easily beconfused with CAH, but these may be differentiated bycareful history and lab study and was associated with UTI.

Declarations

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