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Original Research Paper

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INFANT WITH METABOLIC ALKALOSIS: PSEUDO-BARTTER'S SYNDROME DUE TO FUROSEMIDE.

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ABSTRACT Metabolic alkalosis is an uncommon acid-base disorder in which serum bicarbonate concentration is increased. Two most imporatnt causes of metabolic alkalosis are emesis and diuretic use. Recently, metabolic mimicry of Bartter's Syndrome by vomitting, diarrhea, laxatives and diuretic abuse has been reported. We had female infant in our nephrology department who developed pseudo-bartter's syndrome as the result of surreptitious use of furosemide. In metabolic studies patient exhibited abnormalities similar to those reported in Bartter's syndrome: hypokalemic metabolic alkalosis. Indomethacin therapy resulted in marked improvement in general condition of these children.

KEYWORDS : Furosemide toxicity, Bartter's syndrome, metabolic alkalosis, failure to thrive

INTRODUCTION:

Metabolic alkalosis occurs when a primary pathophysiologic process leads to the net accumulation of base within or the net loss of acid from the extracellular fluid (ECF); typically, the intracellular compartment becomes more acidic in potassium-depletion alkalosis.(1)

Without treatment, severe metabolic alkalosis may result in significant adverse consequences, including impaired perfusion, decreased respiratory drive, cardiac arrhythmia, seizures and death. Identifying the underlying pathophysiology is essential to the management of this disorder.

A case of metabolic alkalosis was eventually diagnosed as Furosemide toxicity mimicking Bartter's Syndrome.

Acquired Bartter-like syndrome (BLS), characterized by hypokalemic metabolic alkalosis, hypomagnesemia, hypocalcemia, and normal kidney function, can be induced by diuretics or antibiotics such as capreomycin, viomycin, amphotericin B, cyclosporine, cisplatin, and aminoglycosides. BLS is a very rare condition and only anecdotal cases generally in adults were reported.(2-4)

Recently, we had a female infant who exhibited symptoms and signs compatible with Bartter's syndrome. A later survey disclosed that she had been taking furosemide surreptitiously. This report is an out- line of the clinical course of the patient and the results of metabolic studies. The data seem to support the hypothesis that the inhibition of Na+ reabsorption (by furosemide in this case) causes most of the metabolic disturbances observed in Bartter's syndrome. Bartter's syndrome is a rare genetic disorder characterized by hypokalemia, hyperreninism, hyperaldosteronism, normotension, juxta-glomerular cell hyperplasia, and resistance to the pressor effect of exogenous angiotensin(5). It is a hereditary condition which is transmitted as autosomal recessive (Bartter types 1–4) or dominant traits (Bartter type 5).(6)

CASE REPORT:

A two and half month old female infant born out of non consanguineous marriage was brought to our department with complaints of refusal to feed for one week. There was history of poor weight gain, with poor feeding for one month. Patient also had complaints of vomiting after every feed for one month. Child vomitus contained milk which was non-blood stained and non-bilious. No history of urinary complaints. Child had attained developmental milestones appropriate for the age.

Patient is known case of ASD with VSD with failure to thrive and was on furosemide for same.

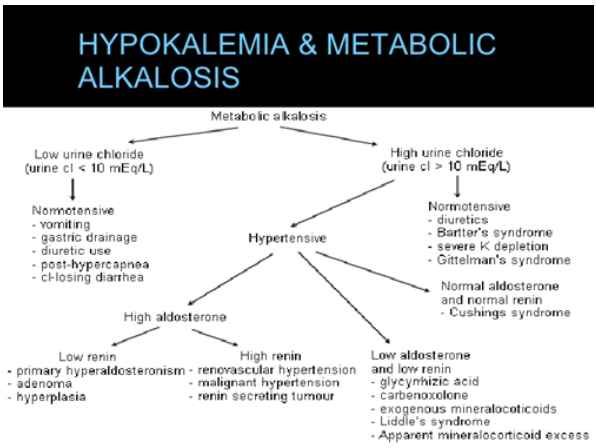
On examination , child was abefrile, dehydrated with a respiratory rate of 44 breaths per minute while sleeping. Pulses were well felt with a rate of 120 beats per minute. Oxygen saturation was normal. Neither anaemia nor jaundice was observed. No lymph node enlarged and no edema on the legs and face was found. Urine output monitoring was done, which showed polyuria. Systemic examination was unremarkable except syslotic murmur.

On laboratory investigation, blood urea was 19.23 mg/dl, serum creatinine was 0.67 mg/dl, sodium 119.7 mmol/L, potassium 2.0 mmol/L, Serum chloride was on lower side(91.5mEq/L), ionic calcium on higher side being 1.63 mmol/L. Patient's laboratory investigation revealed hyponatremia, hypokalemia, and hypocholremia. Serum calcium and magesium were within normal limits. Urinary calcium was 10.65 and low urinary chloride being 9.0. USG abdomen was normal.

In view of tachypneic, arterial blood gas was ordered. It showed:

metabolic alkalosis, hypokalemia , hypochloremia and hyponatremia . Disproportionate urinary wasting of sodium, potassium, and chloride were seen. In view of Hypokalemic metabolic alkalosis and prior history of furosemide intake, diagnosis of Bartter's like syndrome was made.

Indomethacin therapy was treated in dose of 0.5mg/kg/day which resulted in marked improvement in general condition of these children. Therapy with NSAIDs leads to marked improvement in the general well being. With continued treatment, child became better and started accepting feeds well. Serum electrolytes normalized soon. Three weeks later Indomethacin was stopped.



DISCUSSION:

Metabolic alkalosis has an extensive differential diagnosis (7). It is classified according to three mechanisms: (i) loss of hydrogen, further classified into gastrointestinal loss (vomiting, nasogastric suction), renal loss (diuretic use, mineralocorticoid excess, chronic hypercapnia, hypercalcaemia, Bartter and Gitelman Syndromes) or due to hydrogen shift; (ii) retention of bicarbonate, as occurs in massive blood transfusions and (iii) contraction alkalosis, characterized by loss of fluid containing Cl– and no, or little, HCO3– (diuretics).

In approach to look for cause of metabolic alkalosis in a child, common causes like emesis, diuretic use or excess base administration should be asked for. This is followed by estimation urinary chloride levels. If urine chloride levels are less than 10mEq/L , the cause can be emesis, repeated nasogastric suctioning, diuretic use, chloride losing diarrhea, chloride-deficient formula, cystic fibrosis or post hypercapnic state. If urine chloride level is more than 10mEq/L, it is suggestive of chloride- resisitant metabolic alkalosis. Measurment of blood pressure at this point is crucial in clinching the diagnosis.(8) Algorithm for approach to matabolic alkalosis is depicted in Table 1.

In the present case, the cardinal signs were severe metabolic alkalosis, profound hypokalaemia and failure to thrive.Initial laboratory workup showed that urinary chloride concentration was decreased, suggesting that a renal mechanism was not involved In our case, many of the features of bartter's were present. Thus, the furosemide intake could lead to the full constellation of metabolic abnormalities resembling Bartter's syndrome.

Careful subsequent inquiry revealed that she had furosemide administration. Although the distinction between diuretic abuse and Bartter's syndrome could be extremely difficult if patient denied, it is important to consider that the discontinuation of the drugs is associated with reversal of most abnormalities of the disorder. Thus, careful supervision and measurment of urinary output of the drug should be necessary to diffrentiate such patients from Bartter's Syndrome.

CONCLUSION:

In conclusion, surreptitious diuretic usage has to be carefully excluded in patient with normal blood pressure together with laboratory data consisted with Bartter's syndrome. Simple screening test for the drugs in urine should be necessary.

	ABG BEFORE TREATMENT	ABG AFTER TREATMENT
pH	7.520	7.415
pCO2	48.9	39
pO2	57	32
BEecf	17	0
HCO3	39.9	25
TCO2	41	26
SO2	91%	63%
Na+	133	138
K+	3.3	4.4
iCa2+	1.33	1.46
Hct	27%	26%
Hb	9.2g/dl	8.8g/dl

REFRENCES:

1. Tannen RL: Effect of potassium on renal acidification and acid-base homeostasis. Semin Nephrol7: 263 -273, 1987
2. Chen YS, Fang HC, Chou KJ, Gentamicin-induced Bartter-like syndrome. Am J Kidney Dis. 2009;54(6):1158–1161.
3. Ben Salem C, Hmouda H, Bouraoui K. Drug-induced hypokalemia. Curr Drug Saf. 2009;4(1):55–61.
4. Chrispal A, Boorugu H, Prabhakar AT, Moses V. Amikacin-induced type 5 Bartter-like syndrome with severe hypocalcemia. J Postgrad Med. 2009;55(3):208–210.
5. BarterFC, PronoveP^GillJR, et al: Hyperplasia of the juxtaglomerular complexes with hyperaldosteronism and hypokalemic alkalosis. AmerJ Med 33: 811, 1962.
6. Reinalter SC, Jeck N, Peters M, Seyberth HW. Pharmacotyping of hypokalaemic salt-losing tubular disorders. Acta Physiol Scand. 2004;181(4):513–521.
7. Lin SH, Lin YF, Chen DT, et al. Laboratory tests to determine the cause of hypokalemia and paralysis, Arch Intern Med , 2004, vol. 164 (pg. 1561-1566)
8. Gavhane J, Markanda K, Dogra S, Jafri N. Severe Metabolic Alkalosis in an Infant: Bartter Syndrome. MGM J Med Sci2017;4(1):43-45.