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AN UNEXPLAINED CASE OF HYPOKALEMIA IN PREGNANCY -BATTERS SYNDROME SUPERIMPOSED WITH GESTATIONAL HYPERTENSION

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ABSTRACT The renal tubular defect which is batters syndrome ,is one of the variant in renal tubular disorders Both clinical and biochemical features are heterogeneous, ranging from the incidental finding in an asymptomatic patient to marked clinical features of hypokalaemia. Inheritance is likely to be an autosomal recessive.we here present a case of batters syndrome and its clinical presentation in pregnancy and its outcome and complications ,where in the requirement of oral potassium supplements and the course in pregnancy is completely discussed in detail.

KEYWORDS : hypokalemia, batters syndrome.

INTRODUCTION:

Bartter syndrome was first described in the medical literature in the 1960s by Dr. Frederic Bartter. This syndrome can be variably classified as a renal tubulopathy (because certain small tubes within the kidneys are affected), a salt-wasting disorder (because affected individuals excrete excess amounts of salt), a salt-losing tubulopathy, and a channelopathy (because the ion channels in the kidneys are affected),The term antenatal (before birth) Bartter syndrome refers to those cases who present before birth and is typically associated with types 1, 2, 4a and 4b. These disorders were sometimes also called hyperprostaglandin E syndromes .Bartter syndrome type 3 is sometimes also referred to as classic Bartter syndrome. Gitelman syndrome, which has considerable clinical overlap with Bartter syndrome, especially type 3, is sometimes grouped with the Bartter syndromes.(1)

Understanding the normal physiology of renal system in pregnancy will give a better idea how the batters syndrome patient will present during the Time of pregnancy and we can estimate the complications regarding it. There is increase in the renal blood flow thereby glomerular filtration rate increases, alteration in the resorption of solutes, increase in the total body sodium and potassium and increase in the total plasma volume, now what happens in batters syndrome mutations in the thick ascending loop encoding the transporters involved in salt reabsorption type I BS caused by mutations in NKCC2 (SLC12A1); type II BS by mutations in ROMK (KCNJ1); type III BS by mutations in CLC-Kb (CLCNKB); type IV a BS by mutations in barttin (BSND)and type IV b BS by mutations in CLC-Ka and CLC-Kb (CLCNKA and CLCNKB9(2).Overall there's is defective in the NACL transport and increase in H+ and k+ ions so there will be metabolic alkalosis with hypokalemia, because of the volume contraction there will be stimulation of the RAS and increase in the aldosterone there by further increase in the potassium excretion and development of hypertension and its maternal and neonatal complications .

Case Report :

A 30 years old ,primigravida was initially booked at infertility clinic ,she conceived through IVF 2nd cycle , after confirmation they started on Inj. low molecular weight heparin and she was a known case of gestational diabetes mellitus diagnosed at 22 weeks and started on insulin ,hypothyroid since conception on Tab. thyronorm 50 mcg OD and initially at 20 weeks patient admitted in view of hypoglycaemia induced with insulin and hypokalemia ,admission CBG-45mg/dl corrected with 25 % dextrose and serum potassium - 1.9 mg/dl, multiple doses of I.V KCL correction along with Mgso4 2 GMs was given on the subsequent days in the hospital stay and asked to continue syp. kcl 10 ml TDS and for GDM ,medical nutrition therapy was advised and discharged . Patient was admitted at 36 weeks again with the serum potassium - 3.1 mg/dl so started with 20 meq correction and continue syp kcl 10 ml.patient was in poor follow up . At 37 NST showed non reassuring pattern. So patient was taken up for emergency LSCS on 1.10.2021, Intra operatively Patient had B.P of 180/100mmhg. In immediate post op period patient had features of acute pulmonary edema so patient was started on inj. Labetalol infusion and shifted to icu for monitoring and observed .because of her recurrent hypokalemia medicine opinion obtained .advised to do spot postassium and serum osmolality, spot potassium value 200 mEq in 24 hour urine sample . Serum osmolality 300 mOSm/kg . patient started on Tab. nifidipine 20 mg 1-0-1 ,tab labetalol 100mg 1-0-1. B.P was under control with these doses.Patient was monitored, medicine opinion obtained. antihypertensive continued and patient has been thoroughly explained about the condition and need of frequent follow up.

DISCUSSION :

Diagnosing the patient worth recurrent hypokalemia and frequent administration of potassium supplements ,which pointing to the diagnosing of renal tubular disorders .

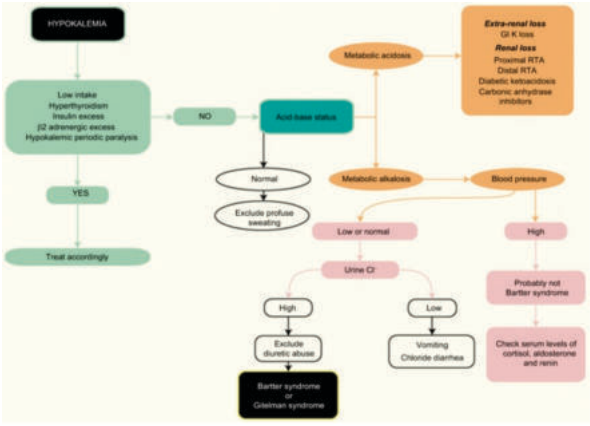

Figure 1 ,showing clinical presentation and how to diagnose it.(2)

Transient presentation of antenatal BS In a French cohort, *MAGE-D2* mutations explained 9% of antenatal BS cases.In the same cohort, severe polyhydramnios occurred in all pregnancies and most patients had a weight and/or length above the 90th percentile. Plasma chloride is higher in patients with *MAGE-D2* mutations compared with other antenatal BS and, together with birth weight percentile, contribute to differentiate the transient form. Genetic screening for *MAGE-D2* mutations should be routinely

performed in patients with suspected antenatal BS(3). There was two case reports of batters syndrome complicating pregnancy in which ,all were encountered hypertension and multiple hypokalaemia correction during the time of pregnancy , it also states that patient may be presented with fetal growth restriction and oligohydraminos are the most prominent clinical features (4) where as in Antenatal batters syndrome ,the presenting feature will be polyhydraminos .

There is a wide spectrum of clinical and bio- chemical presentation. It can range from asymptomatic with an incidental finding of biochemical derangement, to marked muscle weakness, polydipsia, polyuria, tetany, fits and failure to thrive in children. Clinical features are mostly related to hypokalaemia. Muscle weakness was the main symptom in this patient, without other serious complication. The cardinal features are marked hypokalaemia alkalosis with urinary potassium and chloride wasting, and normal blood pressure. Despite such an alarming low level of se- rum potassium, these patients often remained well and asymptomatic, as this chronic problem may be well tolerated.(5)

The effects of maternal Bartter's syndrome on fetal and neonatal outcome are still uncertain.

Obstetric complications such as preterm labor, intrauterine growth restriction had also been reported. There may be a theoretical risk of electrolytes disturbance in neonate. There has been a report on amniotic fluid analysis of affected fetus, which showed that the aldosterone level was significantly increased; while the renin level was not raised as expected .(6) in the previous studies it has been noted that The high cord blood renin levels we re- ported was unlikely to be of fetal origin, and could be derived from high maternal levels (7).

The outcome and complications of batters syndrome in pregnancy has been the poorly studied topic so far. Being the physiological changes it is reasonable to expect an increase in demand for oral potassium supplements antenatally in patients with Bartter's syndrome, as their renal potassium loss would be further aggravated. Such aggravation, however, tends to plateau off early in the second trimester, in correlation to with the early plateauing of renal glomerular filtration. In normal pregnancy, there will be a physiological gradual retention of potassium.(9)The tendency of potassium retention has been ascribed to the effect of raised progesterone in pregnancy. Theoretically, this should have some improvement in the renal loss of potassium in patients with Bartter's syndrome (8)

When the management of batters syndrome mainly depends upon the oral potassium supplements, and regular assessment of renal function.The lack of antenatal complications such as poly hydramnios will point against the possibility of the fetus inheriting this syndrome. The increasing demand of potassium supplement during the second trimester of pregnancy warrants careful dosage adjustment and monitoring. It may be reasonable to suggest that the effect of pregnancy on maternal Bartter's syndrome itself is reversible, as renal potassium loss tends to return to pre-pregnant levels shortly after delivery, and thus the need for oral potassium supplementation should also be reduced accordingly in the postnatal period.the use of potassium-sparing diuretics, such as spironolactone, eplerenone, or amiloride, should help to raise serum potassium and reverse metabolic alkalosis (2, 10) but all these drugs use in pregnancy is still under study .

CONCLUSION :

Batters syndrome in pregnancy is poorly studied and the maternal and neonatal complications is still uncertain .The most common presenting feature is hypo kalemia and the treatment is mainly demand of oral potassium supplements . Antenatal batters syndrome in prenatal diagnosis is challenging because of the major poly hydramnios leading to premature birth and the risk of severe newborn dehydration, requiring management in intensive care unit.

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