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# **Anesthetic Management Of A Patient With Bartter's Syndrome Undergoing Surgery For Bilateral Intracapsular Fracture Of Femur**

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Abstract: Bartter's syndrome is an unusual (estimated incidence is 1.2 per million people) but important congenital form of secondary hyperaldosteronism; due to abnormalities in renal handling of electrolytes. It is associated with hypertrophy and hyperplasia of the juxtaglomerular cells, normal blood pressure, and hypokalemic alkalosis withoutedema. We present a 23-yearold male with Bartter's syndrome underwent bilateral dynamic hip screw fixation surgery for bilateral intracapsular fracture of femur. The anesthetic management of Bartter's syndrome should be relevant to the pathophysiology of the syndrome. Therefore, it should be directed toward maintaining cardiovascular stability, control of associated fluid, electrolyte and acid-base derangements, and the prevention of renal damage.

Keywords: Bartter's syndrome, dynamic hip screw fixation surgery, intracapsular fracture of femur.

### I. Introduction

Bartter's syndrome is a heterogenous entity with at least 2 subsets: Hypokalemic alkalosis with hypercalciuria (true Bartter's syndrome) and hypokalemic alkalosis with hypercalciuria (Gitelman's syndrome). It can also present in utero (antenatal Bartter's syndrome) with resulting prematurity or polyhydramnios.[1] The pathogenesis of Bartter's syndrome is obscure. The primary (autosomal recessive) defect lies in the active chloride reabsorption in the loop of Henle.[1] There is loss of excessive amounts of sodium and potassium in the urine, which leads to hypovolemia and secondary hyperaldosteronism.[2] True Bartter patients usually present above 5 years with signs of vascular volume depletion, polyuria, and polydipsia, whereas Gitelman's syndrome patients typically present at older ages without overt hypovolemia as failure to thrive.[3] Other features include the following: Impaired urinary concentrating ability and hyperactive renin-angiotensin system (plasma renin increased, lack of effect of angiotensin on blood pressure, renal potassium wasting, increased renal prostaglandin production, and occasionally hypomagnesemia.[2]

#### 2.CASE REPORT

A 23 year old male patient weighing 28kg resident of jammikunta Telangana came with bilateral hip pain. On X ray diagnosed to have bilateral intracapsular fracture of femur.surgery plan was dynamic Hip screw fixation for both hips .He is a known case of bartter syndrome with single kidney diagnosed at the age of 2 yrs and he is on regular consultation with pediatric nephrologist.since 21 years he is on regular supplementation with potassium syrups, calcium tablet, vitamin D3 tablet and magnesium tablets.

On admission to the hospital (1 days preoperatively), the patient's physical examination was normal, and blood pressure was 90/60 mmHg. The electrocardiogram demonstrated only nonspecific ST-T changes and T wave inversion. Laboratory data shown as follows sodium, potassium, and chloride were 130, 2.7 mmol/L, and 96mmol/L, respectively .ABG Showed alkalosis with normocarbia and normal pa02 levels. Calcium (2.67 mmol/L), corrected calcium (2.7 mmol/l) and magnesium (0.8 mmol/L)and phosphorus levels were all within normal. Urea and creatinine were slightly elevated indicating underlying renal disease for which nephrologist was consulted and suggested for correction.potassium 40 meg in thrice daily and half normal saline (0.45%) fluid was suggested for correcting hypovolemia. Usg abdomen showed CKD grade 2 changes with single kidney. Complete blood picture showed HB 13 mg/dl platelet count 2.6 lakhs/mm3 .ESR was normal.B+VE blood group and clotting time and bleeding time normal.viral markers HIV ,HbsAg,HCV normal.chest x ray no significant changes.urine output was high and for which fluid is replaced accordingly.

On the day of surgery potassium 3.1mmol/l sodium 133 mmol/l chloride 102 mmol/l and slightly increased creatinine levels (1.5mg%) which was normal for single kidney patient.

After placing two wide bore canula patient shifted to operation theatre.plan of anaesthesia is to give graded epidural block while maintaining hemodynamic stability and replace ongoing potassium loss and hypovolemia with half normal saline.

Under strict asceptic precautions patient back area cleaned and draped. L3-L4 intervertebral space identified and infiltrated with local anaesthetic lignocaine .epid epidural space identified with loss of resistance syringe at 2cm with tuhoy needle. 20 gauge catheter

introduced through needle and fixed at 8cm. Test dose of 3 cc lignocaine with adrenaline 1 in 2 lakh dilution given which is negative that means intravascular and intrathecal migration of catheter ruled out.

5cc of bupivacaine 0.5% given first then another 5 cc after 5 minutes .....like wise another 5cc after 5 min.T10 level achieved within 20 min after first dose.

Scrubbing and drapping started first right side and then left side done in a single setting surgery duration was approximately 3 hrs. During surgery 2 top ups of 5 cc bupivacaine 0.5% given to maintain level above T10 dermatome. Total one litre of half normal saline fluid given with 30 meq potassium added to fluid.10 cc of calcium gluconate given over 10 min slowly.1gram magnesium added to 100 ml saline and given over 20 min intraoperatively.

Blood loss around 200 ml and urine output maintained more than 100ml per hour for which fluid replacement done. Intraop Intraoperatively hemodynamic stability maintained and not even one episode of hypotension was recorded as we given top ups gradedly according to level of analgesia and blood pressure.

Post operatively we started intravenous potassium replacement on day 1 and on day 2 onwards potassium syrups, calcium and Vitamin D3 tablets ,magnesium tablets started .our target was to maintain potassium levels above 3 meg/l.visual analogue scale used for pain score and given epidural top ups accordingly .0.1% bupivaicaine was used for epidural top ups.

Patient discharged happily on post operative day 5 as he was doing well

His potassium levels are above 3meq/l and creatinine 0.6mg% on day of discharge.

#### 3.DISCUSSION

Anesthetic management of a patient with Bartter's syndrome presents many challenges to anesthesiologist. In the light of pathophysiology of Bartter's syndrome, cardiovascular instability, perioperative electrolyte and acid-base disturbances, and renal dysfunction are the most concerned.

The stability of cardiovascular system is important in such patients.[2] Major hemodynamic problems were not encountered in patients with Bartter's syndrome, [4-6]. But here in this case patient is hypovolumeic and maintained adequate hydration and tried not to have any hypotension episodes. This have been achieved with graded epidural anaesthesia.

Perioperative electrolyte management is one utmost important in there cases as they are prone to hypokalemic alkalosis, hypocalcemia and hypomagnesemia.we have chosen to give aggressively potassium intravenously before and during surgery till day 1 post operatively so as to maintain our targets of 3 meg/l.we performed daily serum electrolytes checkup so as to increase or decrease potassium requirements accordingly. Then from day 2 we shifted to oral forms of potassium syrups calcium and magnesium tablets.

#### 4.CONCLUSION:

Although anesthesia management in patients with Bartter's syndrome requires judicious attention with respect to maintaining of cardiovascular stability, control of plasma potassium level and the prevention of renal damage, it should be individually tailored in accordance to the surgical procedure and patient's clinical condition.

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