PP141 RENAL TUBULAR ACIDOSIS IN PREGNANCY

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INTRODUCTION:

Renal tubular acidosis (RTA) in pregnancy is uncommonly encountered.^{1,2} RTA is usually associated with an inability of the renal tubules to excrete hydrogen ions and/or a defect in the reabsorption of bicarbonate. This results in significant systemic acidosis, hypokalaemia, hypocalcaemia, and hypercalciuria. Glomerular function is usually preserved.³ Rhabdomyolysis occurrence in pregnancy is rare however pregnancy may predispose individuals to rhabdomyolysis due to hypokalemia.^{2,4}

CASE REPORT:

A 33-years-old lady, previously healthy, who started the prenatal care uneventfully until 30 weeks, developed easily fatigue, weakness proximal part of upper limb and lower limb. No other symptoms such as fever, dysuria, contraction pain, prevaginal bleeding, vomiting or diarrhoea. Examination revealed a blood pressure 131/84, heart rate 88, sugar 6.5 mmol. Having weakness over proximal upper limb. White cells count 12.9, haemoglobin 13.3, platelet 314, sodium 138, potassium 2.0, creatine kinase 1795, magnesium 1.17, calcium 2.21. Urinalysis unremarkable. Venous blood gases pH of 7.43, bicarbonate of 19.8 and base deficit of 6.2. She started on intravenous and oral potassium correction together with intravenous magnesium sulphate. Subsequently, potassium and creatine kinase normalised to 4.2 and 14 and the symptoms resolved.

DISCUSSION:

Renal tubular acidosis in pregnancy is a very rare disorder.^{1,3} Most cases are either inherited or secondary to maternal disease or ingestion of toxic chemicals, mainly toluene, and less commonly with maternal diseases such as systemic lupus erythematosus (SLE), or chronic hepatitis.³

RTA has been divided into four different types. Distal RTA or type I is due to an inability of the distal tubule to excrete hydrogen ions. Proximal RTA or type II is due to an inability of the proximal tubule to reabsorb bicarbonate. Type III is a combination of both, while type IV is associated with diffuse distal tubular dysfunction. Types I and II are more common.³

Pregnancy is a high-risk state for predilection to hypokalaemia-induced rhabdomyolysis. Potassium plays a major role in regulating skeletal muscle blood flow. Local elevation in the potassium concentration during muscle activity causes vasodilatation, which enhances regional blood flow. This cellular release of potassium is impaired in potassium depleted state leading to relative muscle ischemia manifesting as muscle cramps and in severe hypokalaemia as muscle necrosis and rhabdomyolysis.¹

Pregnancy is known to worsen RTA. During pregnancy there is a mild physiological respiratory alkalosis, which, however, is not enough to upset the hyperfiltration in the kidneys that is associated with the increase in blood volume. This hyperfiltration results in an increased loss of some electrolytes and thus the net requirement of potassium and bicarbonate is significantly increased. Among the potential complications of RTA are rickets in children and osteocalcin in adults. Patients can also develop hypercalciuria with formation of kidney stones and secondary hyperparathyroidism. Hypokalaemia can result in severe muscle weakness, rhabdomyolysis, and even cardiac arrest.³

The majority of patients (86%) presented with preterm labour and others manifesting with muscle pain, cramps and weakness.^{3,4} Treatments included hydration, correction of the metabolic acidosis with alkali therapy, and potassium supplementation.^{2,4}

CONCLUSION:

When encounter pregnant patient with uneventful illness antenatally, we have to consider RTA in pregnancy if the patient presented with muscle weakness, cramping or contraction pain and associated with hypokalaemia, hypocalcaemia and metabolic acidosis, some patient associated with increase creatine kinase. The treatment is to correct electrolytes, metabolic acidosis and hydration. The symptoms will recover after normalisation of electrolyte and usually delivered healthy normal baby.