

SUMMARY

Renal impairment in the neonates usually occurs following complicated labor and delivery with perinatal hypoxia, and it is usually in form of tubular insult.

The clinical presentations of renal impairment in neonates is often subtle, lack of recognition of its occurrence has made this especially true when renal impairment follows perinatal anoxia.

In this work 24 neonates (13 males + 11 females), who suffered perinatal asphyxia and needed admission to the neonatal intensive care unit, were studied for occurrence of acute renal impairment.

12 normal infants were included in the study as controls.

Mean patients serum urea was $(72.94 \pm 43.24 \text{ mg/dl})$ which was not significantly different than that of controls $(58.32 \pm 52.91 \text{ mg/dl})$ which may be due to small number of the patients.

Mean serum creatinine of patients $(1.7 \pm .88 \text{ mg/dl})$ was significantly higher than that of the controls $(0.7 \pm 0.33 \text{ mg/dl})$.

Mean serum Na in patients $(141.6 \pm 2.49 \text{ meq/l})$ was not significantly in value but lower than controls $(142.81 \pm 2.48 \text{ meq/l})$.

The possible development of inappropriate secretion of antidiuretic hormone in perinatal hypoxia often results in hyponatremia which is not recognized in our patients may be due to small number of patients.

The present study revealed mild hyperkalemia in asphyxiated newborn as compared to healthy controls in mean.

Mean serum K in patients $(4.98 \pm .66 \text{ meq/l})$ was not significantly higher than that of the controls $(4.1 \pm .51 \text{ meq/l})$.

Mean serum CL in patients (110.0 ± 3.50 meq/l) did not differ significantly from that of the controls (110.36 ± 5.08 meq/l).

Routine urinalysis of cases with perinatal hypoxia revealed proteinuria in 41.7% in patients.

Many epithelial cells were found in 53.5% of the cases with perinatal asphyxia.

Based on the findings of the pathological sediment in the urine it is postulated that renal lesions are common as a result of perinatal asphyxia with tubular insult.

Mean urine analysis for R.B.Cs of patients (6.33 RBCs/HPF ± 4.02 RBCs/HPF) which was significantly higher than that of controls (4.16 RBCs/HPF ± 2.69 RBCs/HPF).

Mean urine analysis for pus cells of patients (2.84 pus cells/HPF ± 1.59 pus cells/HPF) which was not significantly different than that of controls (3.08 pus cells/HPF ± 3.87 pus cells/HPF).

Mean urinary B₂ microglobulin concentration in patients (218.30 ng/ml ± 100.93 ng/ml) was significantly higher than that of controls (144.316 ng/ml ± 126.3 ng/ml) which is specific for tubular renal insult in renal impairment.

Mean urinary urea in patients was (7.727 ± 3.16 meq/l) which was not significant in relation to controls 96.80 ± 2.907 meq/l).

Mean urinary creatinine in patients was (1.30 ± 1.4 meq/l) which was not significantly different than controls (1.22 ± 1.09 meq/l).

Based on the findings of the pathological laboratory and sediment in the urine it is postulated that renal lesions are in form of tubular insults as a result of perinatal asphyxia.

Among the clinical symptoms of the studied newborns, renal impairment in the form of tubular insult was detected more significantly in cases with asphyxiated newborn.

CONCLUSION AND RECOMMENDATIONS

The results of the present study suggested that renal impairment in form of tubular insult should be anticipated in any hypoxic newborn infant.

An early assessment of serum creatinine and B₂ microglobulin concentration levels in urine should be performed in every newborn who sustained - perinatal anoxia.

Elevated serum creatinine and urinary B₂ microglobulin concentration were detected in cases of perinatal asphyxia.

It is essential that fluid restriction should be done on the first day or two of life to avoid fluid over load.

Appearance and composition of urine should be determined routinely in all hypoxic infants. Presence of epithelial cells, RBCs, albumin is indicative of perinatal hypoxic renal insult.

Perinatal hypoxia is an important differential diagnosis in cases presenting with pyuria in the newborn period.

During resuscitation, oxygenation and correction of acidosis should be prompt.

Prevention and early detection of perinatal anoxia are the two cornerstones on which successful therapy of renal impairment is based.

The clinician should be aware that any infant experiencing severe perinatal anoxia and any infant requiring admission to a neonatal intensive care center is at risk for the development of renal insult in the form of tubular insult.