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ATRIAL NATRIURETIC PEPTIDE AND ALDOSTERONE IN SICK PREMATURE NEONATES*

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SUMMARY: Paç FA, Yiğitoğlu MR, Kalaycı AG, Adam B. (Department of Pediatrics and Biochemistry, Atatürk University Faculty of Medicine, Erzurum, Turkey). Atrial natriuretic peptide and aldosterone in sick premature neonates. Turk J Pediatr 34: 153-156, 1992.

Plasma atrial natriuretic peptide (ANP) and aldosterone concentrations were investigated in 25 sick premature neonates. Group A consisted of 11 premature neonates with sepsis and group B of 14 neonates with hyperbilirubinemia. In both groups, ANP and aldosterone levels were found to be higher than in the controls. Group A concentrations of ANP and aldosterone were also higher than in group B. *Key words: aldosterone, atrial natriuretic peptide, premature sick neonate.*

Atrial natriuretic peptide (ANP) affects kidney function by altering renal hemodynamics, increasing excretion of sodium and water[1] and inhibiting renin and aldosterone secretion[2]. ANP is a hormone involved in the regulation of sodium and body fluid homeostasis[3,4]. Plasma aldosterone concentration is relatively high in human newborn infants[5,6]. In the newborn, the homeostatic function of the kidney may be limited[7].

The present study was designed to determine ANP and aldosterone levels in 25 sick premature neonates.

Material and Methods

The study population comprised 25 infants, 14 boys and 11 girls, who were the products of uncomplicated pregnancies that were delivered vaginally. No congenital defects were present. Apgar scores at one minute were >7. Eleven of the infants were included in the study because of sepsis with positive blood cultures (Group A), and 14 were born in our hospital and had hyperbilirubinemia with a total bilirubin level exceeding 12 mg/dl (Group B). Gestational ages ranged from 32 to 37 weeks and birth weights from 1350 to 2410 g (1650 ± 245 g). All studies were performed between 7-12 days of life. The infants were all breast fed. Blood and urine samples were obtained before infusion and drug therapy.

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Urine was collected in plastic bags for 12 to 24 hours. Arterial blood samples were drawn from femoral arteries. EDTA-Plasma contained 400 KIU/ml of aprotinin (Trasylol®) which was added to measure the ANP level. Plasma ANP levels were determined using the RIA method (Incstar Corp., Stillwater, Minnesota, CANADA Cat. no: 22750). Plasma aldosterone levels were also determined using the RIA method (Diagnostic Products Corp., Coat-A-Count, USA Cat no: TKAL1). The plasma ANP and aldosterone levels were determined in twenty healthy neonates with similar characteristics, who were used as controls.

Creatine clearance (Ccr) and fractional excretion of sodium (FENa) were calculated from the formula shown below:

$$Ccr\ (ml/min/kg) = (Ucr \times V)/Scr$$

$$FENa\ (\%) = [(UNa \times SNa)/Ucr \times Scr)] \times 100$$

The data were expressed as a mean ± SEM. The Student's *t*-test was used for statistical analysis.

Results

Body weight, plasma sodium concentration, creatine clearance, fractional sodium excretion, plasma aldosterone and ANP concentrations of the groups are shown in Table I.

TABLE I: Plasma ANP and Aldosterone Levels in Sick Neonates and in Controls

Group	Birth Weight (g)	Serum Na (mEq/L)	Ccr ml/min/kg	FENa (%)	P-Aldosterone pg/ml	p-ANP pg/ml
A (n = 11) (sepsis)	2150 ± 115	141 ± 3.9	2.2 ± 0.8	0.37 ± 0.28	1870 ± 398	105 ± 48
B (n = 14) (hyperbilirubinemia)	1870 ± 372[c]	139.3 ± 2.7[b]	1.85 ± 0.6[b]	0.42 ± 0.20[b]	985 ± 445[a,c]	53 ± 32[a,b]
C (n = 20) (Control)	–	–	–	–	382 ± 107	29 ± 8.4

a: $p<0.001$ as compared with controls
b,c: $p>0.05$ and $p<0.001$ as compared with other patient groups, respectively.

The children with sepsis had the highest p-ANP and aldosterone values. In addition, the children with hyperbilirubinemia had higher p-ANP and aldosterone levels than the controls.

Discussion

ANP may provide a sensitive and important hormonal system for the control of sodium balance, even in premature neonates[8]. It is observed that premature infants with a birth weight of 1500g have markedly elevated plasma concentrations of ANP during the early postnatal period[8]. Hypersecretion of human ANP in

respiratory distress syndrome (RDS) and meconium aspiration syndrome (MAS) may play an important part in initiating spontaneous diuresis in sick neonates[9]. ANP inhibits renin and aldosterone secretion[2], but plasma aldosterone concentration is relatively high in newborn infants[5].

In our study, we found no differences between groups A and B in serum sodium concentrations, Ccr, and FENa (%). But plasma aldosterone concentrations in groups A and B were higher than in the controls ($p<0.001$). In addition, aldosterone concentrations in group A were higher than those in group B ($p<0.001$). Plasma concentrations of ANP in the sick premature neonates were higher than in the controls ($p<0.001$). Plasma ANP levels in the sepsis group were higher than in the hyperbilirubinemia group. In contrast to reports in the literature, we found that aldosterone levels did not decrease with an increase in ANP levels.

Kojima et al[10] found that plasma aldosterone concentrations were especially higher in younger premature infants and that ANP levels were higher in this age group when compared with healthy adults. High plasma aldosterone concentrations possibly act to enhance the reabsorptive capacity of the distal nephron for sodium intake and ANP may be released to control the extracellular space in premature infants[10].

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