

incompletely understood. The principal determinant of extracellular fluid volume is sodium and it has been calculated that normal pregnancy is associated with the net retention of some 900 mmol (3- 4 mmol /L) of sodium. Net sodium retention during pregnancy appears in some ways paradoxical in that there is a marked increment in factors which are known to enhance natriuresis⁽⁵⁾. These include glomerular filtration rate and circulating concentrations of progesterone and atrial natriuretic peptide. One noteworthy factor opposing this change is the very substantial increase in plasma aldosterone concentrations⁽⁵⁾.

It is obvious that a significant proportion of the retained sodium must be sequestered within the fetal compartment (including placenta, membranes and amniotic fluid) and it is noteworthy that the mother plasma sodium concentration decreases slightly, implying that factors other than sodium retention may also be responsible for the water retention of normal pregnancy⁽⁴⁾. Substantial alterations have been described in intracellular water and electrolyte concentrations and it is possible that these relate to changes in cell metabolism⁴. Failure to achieve these adaptational changes has been associated with intrauterine growth restriction and hypertensive disorders in pregnancy⁽⁴⁾.

Nitric oxide (nitrogen monoxide) plays an important role in a wide range of physiologic processes⁽⁶⁾. NO influences renal vascular tone and blood pressure (BP), glomerular and medullary hemodynamics, and extracellular fluid volume⁽³⁾. This renoprotective effect was supported by several genetic and experimental studies⁽³⁾. Nitric oxide synthase is particularly important in the function of human kidney. It plays a role in the

maintenance of normal vascular and renal function⁽⁷⁾. Not surprisingly, renal signs and symptoms from inhibiting NOS are similar to those seen in preeclampsia⁽⁷⁾. There may be a similar nitric oxide generation and sodium ion relationship in the endothelial cells of the small intestine and the tubules of the kidney cells in that when they are stressed by sodium entry, the exchange of sodium for calcium activates calcium dependent NOS⁽⁸⁾. A link between tubular absorption of sodium ions and NO generation has been shown in both in vivo and in vitro preparations⁽⁷⁾.

Subjects & Methods

A-Subjects

The study was a cross-sectional, case-control study conducted on 60 patients with preeclampsia (PE) attending the Obstetric Consultant-Clinic, Antenatal Clinic, and Labor Ward at Al-Kadhimiya Teaching Hospital, for re-evaluation of newly diagnosed PE, or for delivery.

The diagnosis of PE was based on clinical criteria that were hypertension (absolute BP of 140/90 mmHg twice over 4 hr without prior comparison)^(1, 2) and proteinuria (21.5 mg of urinary protein per μ mol creatinine)⁽⁹⁾.

The exclusion criteria used for cases and controls were gestational or chronic hypertension, diabetes mellitus, renal disease, multifetal gestation, intrauterine fetal death, and pregnancy less than 20 weeks of gestation.

Depending on the gestational age, the 60 patients were divided into two groups:

1. Preeclamptics in the second trimester (**G1**): They were 30 with age range from 18 to 37 years (mean age \pm SD = 26.1 ± 6.4 year) and gestational age range from 20 to 28 weeks (mean gestational age \pm SD = 26.3 ± 1.5 week).
2. Preeclamptics in the third trimester