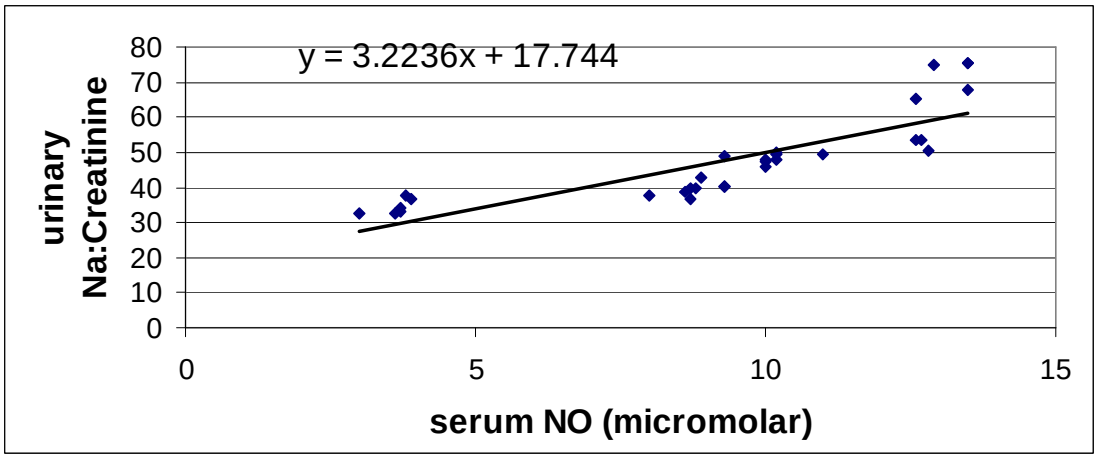

Figure 4: Correlation between serum NO & Na excretion in G4: third trimester pregnant controls (n=30, r = 0.8; P < 0.001).

Discussion

Nitric oxide mediates many functions of endothelium, including vasodilatation and inhibition of platelet aggregation [12]. Preeclampsia may be associated with nitric oxide deficiency [12], and the results of this study provide an evidence to support this hypothesis. As shown in Table 1. NO level in blood was similar in both healthy pregnants groups; it was unchanged during physiological pregnancy. During preeclampsia, the NO was decreased compared to the control level. This suggests that during preeclampsia the low activity of endothelial NO-synthases and redox-dependent transformation of NO in peroxynitrite provoke a decrease in the blood nitric oxide level [13], these results are comparable to those of Meher & Duly [12], Khetsuriani et al. [14], Choi et al.[13], Nishikawa & Miyamoto[15]

While serum Na^+ was significantly increased in normal pregnancy with advancing gestational age, it was insignificantly decreased in preeclamptics with advancing gestational age.

The observed significant low urinary excretion of sodium in the preeclamptic groups (Table 2) is comparable with Martniz et al. [16], who found that urinary excretion of sodium was lower in hypertensive than in normotensive gestation. But this finding can not be compared with the results of Halhali et al. [17], Kyey`nska et. al. [18], & Sigurdsson & Gengtss[19] who found normal range of urine Na excretion in their patients.

Preeclampsia is accompanied by amplification of the sodium retention that is a feature of a normal pregnancy [20]; which is associated with net retention of sodium with substantial alterations in intracellular water and electrolyte concentrations and possibly these are related to changes in cell membranes [21], which appear to be responsible for some pathological changes in preeclampsia. Some of the best documented alterations involve changes in the handling of sodium ion both on the systemic and intracellular levels [20, 22].

On intracellular level majority of studies support an increase in peripheral cell sodium concentration. This would suggest a defect in Na,K ATPase or sodium pump activity, leading to an increase cell sodium in vascular tissues that has been shown to enhance vascular sensitivity to vascular constricting agents or leading