

been established that high androgen level, primarily dependent on placental function, is a factor in the etiopathogenesis of preeclampsia^(5, 6). Nitric oxide (nitrogen monoxide) plays an important role in a wide range of physiologic processes⁽⁷⁾. A major mediator of endothelial function, NO, regulates vasodilatory and antithrombotic actions in the vasculature⁽⁷⁾. Impaired NO bioactivity has been postulated as an important pathogenic factor in preeclampsia⁽⁷⁾. Endothelium-dependent arterial vasodilation has been shown to be reduced and vascular impedance to be increased in preeclampsia compared with normal pregnancy⁽⁷⁾. Postpregnancy, women with a history of preeclampsia (3 months postpartum or later) have significantly reduced endothelium-dependent vasodilation compared with women with a history of normal pregnancy⁽⁷⁾. Also, NO is mainly expressed in Leydig cells where it regulates the concentration of testosterone by acting in an autocrine/paracrine fashion. In fact, NO is involved in testicular testosterone synthesis causing a significant decrease of androgen production⁽⁸⁾.

The present study was undertaken to elucidate the role of sex steroid (testosterone) on endothelial dysfunction in preeclampsia.

Subjects & Methods

A-Patients: The study was a cross-sectional, case-control study conducted on sixty 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 mmol creatinine)⁽⁹⁾.

The exclusion criteria, which were 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 patients were divided into two groups:

1. Preeclamptics in the second trimester (G1):

Included thirty Preeclamptics in their second trimester of pregnancy. Age range was from 18 to 37 years (mean age \pm SD = 26.1 \pm 6.4 year). The gestational age range was from 20 to 28 weeks (mean gestational age \pm SD = 26.3 \pm 1.5 week).

2. Preeclamptics in the third trimester (G2):

Included thirty preeclamptics in their third trimester of pregnancy. Age range was from 18 to 40 years (mean age \pm SD = 25.1 \pm 6.9 years). Gestational age ranged from 29 to 40 weeks (mean gestational age \pm SD = 35.6 \pm 1.6 week).

Controls: Sixty apparently healthy pregnant attending the Antenatal clinic, and Labor Ward at Al-Kadhimiya Teaching Hospital, for re-evaluation of their pregnancy, or for delivery. The control groups were comparable to the preeclamptic groups regarding the age, gestational age, Depending on the gestational age, the apparently healthy pregnant were divided into two groups:

3. Control pregnant in the second trimester (G3):

They were thirty apparently healthy pregnant in the second trimester of pregnancy. Age range was from 15 to 38 years (mean age \pm SD = 24.6 \pm 4.5 year).