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BRIEF COMMUNICATION

C-reactive protein during normal pregnancy and preeclampsia

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KEYWORDS

C-reactive protein; Pregnancy; Preeclampsia

Preeclampsia (PE) is a common (~7% of all pregnancies) disorder of human pregnancy in which the normal hemodynamic response is compromised. Creactive protein (CRP) was higher in women with established PE compared to normal pregnant (NP) women [1], and no differences were found between NP and PE if CRP was measured in early pregnancy [2,3]. However, recently it was reported that elevated CRP appears to be an independent predictor of PE [4]. Therefore, the purpose of this

Between April/01 and November/02 a prospective study was conducted, approved by the Bioethics Committee-Biomedical Center-Central University of Ecuador. There were 278 healthy pregnant women included, primigravidae, <25 years old, and attending to the "Hospital Gineco Obstetrico Isidro Ayora" in Quito, Ecuador.

From week 16 to 36 all women had a careful obstetrical control and blood sample withdrawal every four weeks and then every two weeks up to

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Table 1 Characteristic of women studied			
	Normal pregnancy (n = 183)	Preeclampsia (n = 24)	р
Age (years)	21.2 ± 3.1	$\textbf{21.1} \pm \textbf{2.9}$	NS
SBP (mm Hg)	105.6 ± 10.9	143.2 ± 6.3	< 0.0001
DBP (mm Hg)	67.4 ± 9.0	$\textbf{95.0} \pm \textbf{5.8}$	< 0.0001
Proteinuria (mg/24 h)	_	570 ± 190.2	
Delivery (weeks)	$\textbf{38.6} \pm \textbf{2.2}$	$\textbf{36.8} \pm \textbf{2.7}$	0.0004
Newborn weight (g)	2941 ± 455	2737 ± 297	0.03

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study was to investigate the variations of CRP in NP women and those complicated with PE.

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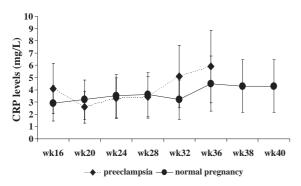


Figure 1 Evolution of C-reactive protein levels in women with normal pregnancy and those who developed preeclampsia.

delivery. High sensitivity latex C-reactive protein was measured by immunoturbidimetry (Roche Diagnostics, Switzerland), and PE was defined as a blood pressure >140/90 mm Hg and proteinuria >300 mg/dl.

Two hundred and seven women completed the study and 24 developed PE (11.6%; Table 1).

CRP in NP women increased from week 16 to 28 (2.9 \pm 2.5 vs. 3.6 \pm 2.3 mg/l, p = 0.003), and dropped at week 32 (3.2 \pm 2.2 mg/l, p = 0.05). Then CRP increased up to delivery (4.3 \pm 2.5 mg/l, p < 0.0001; Fig. 1).

In women who developed PE, CRP was lower at week 20 compared to 16 $(2.6\pm2.8 \text{ vs. } 4.1\pm3.8 \text{ mg/l}, p=0.06)$, but then it recovered at week 24 $(3.3\pm2.0 \text{ mg/l}, pNS)$. Later CRP was increasing until week 32 $(5.1\pm2.5 \text{ mg/l}, pNS)$, and up to delivery $(5.9\pm2.2 \text{ mg/l}; p=0.02)$.

CRP was different between PE and NP at week 32 (5.1 \pm 2.5 vs. 3.2 \pm 2.2 mg/l, p = 0.0007), and delivery (5.9 \pm 2.2 vs. 4.3 \pm 2.5 mg/l, p = 0.001), but not at week 16 (4.1 \pm 3.8 vs. 2.9 \pm 2.5 mg/l, p = 0.07).

Using the average value of CRP in NP at week 16 (2.9 mg/l) as a cut-off point, we did not find any risk for developing PE (RR=1.09, IC 95% 0.9—1.2). This follow up study demonstrates that women who developed PE increased CRP until delivery more than those with NP. However, CRP measured at week 16 is not a predictor for development of PE.

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