Table 2. Serum Metabolic Parameters at Baseline and 12 Weeks Following Eprosartan Treatment

	Baseline*	12 weeks*	P
SBP (mmHg)	154 ± 10	141 ± 12	<0.001
DBP (mmHg)	96 ± 8	84 ± 9	<0.001
TC (mg/dL)	212 ± 32	219 ± 33	NS
Triglycerides (mg/dL)	115 ± 50	113 ± 39	NS
HDL-C (mg/dL)	53 ± 8	55 ± 10	NS
LDL-C (mg/dL)	134 ± 24	142 ± 28	NS
ApoA1 (mg/dL)	145 ± 20	153 ± 25	NS
ApoB (mg/dL)	86 ± 22	95 ± 20	NS
ApoE (mg/dL)	3.8 ± 0.7	3.9 ± 1.4	NS
Lp(a) (mg/dL)	10 (8-72)	10 (8-11)	NS
Glucose (mg/dL)	94 ± 12	96 ± 13	NS
Insulin (mU/L)	8.8 ± 3.4	9.9 ± 5.5	NS
HOMA index	2.2 ± 0.9	2.6 ± 1.5	NS
AST (U/L)	21 ± 4.6	18 ± 4.6	0.04
ALT (U/L)	24 ± 8	19 ± 8	0.05
Creatinine (mg/dL)	0.86 ± 0.11	0.87 ± 0.12	NS
Uric acid (mg/dL)	4.5 ± 1.5	4.7 ± 1.4	NS
FE uric acid (%)	13 ± 6	10 ± 3	NS
Fibrinogen (mg/dL)	343 ± 85	347 ± 98	NS
PAI-1 (U/L)	3.4 ± 2.6	3.0 ± 1.3	NS
tPA (ng/mL)	7.1 ± 3.2	8.6 ± 4.8	NS
A2-antiplasmin (%)	100 ± 9	101 ± 17	NS
8-epiPGF2a (pg/mL)	61 ± 20	67 ± 24	NS
Lag time (min)	145 ± 54	180 ± 58	0.001
PON1 (U/L)	60 (20-175)	59 (27-201)	NS
Lp-PLA$_2$ (nmol/mL/min)	29 ± 8	34 ± 16	NS

SBP: systolic blood pressure; DBP: diastolic blood pressure; TC: total cholesterol; LDL-C: low density lipoprotein cholesterol; HDL-C: high density lipoprotein cholesterol; ApoA1: apolipoprotein A1; ApoB: apolipoprotein B; ApoE: apolipoprotein E; Lp(a): lipoprotein (a); HOMA: homeostasis model assessment index; AST: aspartate aminotransferase; ALT: alanine aminotransferase; FE: fractional excretion; PAI-1: platelet activator inhibitor-1; tPA: tissue plasminogen activator; 8-epiPGF2a: 8-isoprostanes; PON1: paraoxonase 1; Lp-PLA$_2$: lipoprotein-associated phospholipase A$_2$.
*Mean values ± standard deviation or median with min/max range.
**NS: not significant.

increase of the lag time for LDL oxidation has been previously reported in patients with mild to moderate essential hypertension, and was attributed to the modulation of NADH/NADPH oxidase, which counteracts superoxide production [16].

Eprosartan had no effect on lipid profile, as well as on PON1 and Lp-PLA$_2$ that are determinants of lipoprotein function and therefore are involved in atherosclerosis. The neutral effect of eprosartan on lipid profile has been previously reported [17], and was also evident in diabetic patients [18]. PON1 and Lp-PLA$_2$ are enzymes related to the inflammatory mechanisms that take part in atherogenesis. PON1 is an esterase that is exclusively associated with HDL in plasma and catalyses the hydrolysis of phospholipid hydroperoxides and cholesteryl ester hydroperoxides, which are formed during LDL oxidation. Thus, PON1 may play an important role in the anti-atherogenic activity of HDL. In contrast, Lp-PLA$_2$, an enzyme mainly associated with LDL, is currently related to the inflammatory mechanisms of the atherogenic process. Lp-PLA$_2$ exhibits a Ca^{+2}-independent phospholipase A$_2$ activity and catalyses the hydrolysis of the ester bond at the sn-2 position of the proinflammatory phos-