because normal arterial waveforms were found in three cases of proved adnexal torsion [3].

In favor of detorsion, the series of cases reviewed by Oelsner et al. [1] confirmed normal-size ovaries with follicular development after a mean follow-up time of 4.07 years. It seems that unless the mass contains unexpected tissues, such as in a dermoid cyst, peritoneal mechanisms may restore functionality. One difficulty of the conservative approach is that the time-course of the recovery process is unknown. This is important because the possibility of complications associated with expectant management [4,5] raises the question of whether recovery should be announced by some early signs, such as re-vascularization detectable by Doppler, progressive reduction in tumor size, or others. The absence of this information in the literature limits the option of a reliable prognosis.

The woman in our case had a necrotic ovary. Earlier suspicion would, most probably, have prompted quicker intervention and mitigated the necrotic impact. The detection of the cystic mass, although taken as residual from ovarian stimulation, should have activated adnexal torsion in the differential diagnosis. We can only speculate whether the recovery of color, as reported by surgeons performing the initial intervention, was a false indicator of viability.

Finally, the dilemma either leaving or excising the tumor is the other relevant point of the case. The magnitude of risk determined by this devitalized mass cannot be established. This uncertainty was key in the decision of the patient to ask for surgical ablation.

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Antepartum and postpartum maternal plasma levels of E-selectin in pre-eclampsia, gestational proteinuria and gestational hypertension

Dear Editors,

Pathophysiologic features of pre-eclampsia (PE) suggest that generalized endothelial cell damage and dysfunction are the major features of the disease. E-selectin is a cell adhesion molecule and its increased expression indicates endothelial cell activation [1]. In this pilot study, we have investigated alterations of maternal antepartum and postpartum plasma levels of soluble(s) E-selectin in normotensive pregnant women and women with PE, gestational hypertension (GH) and gestational proteinuria (GP). We further sought to determine whether changes in plasma levels of sE-selectin from antepartum to postpartum would correlate with the postpartum regression of PE.

A total of 34 pregnant women were included in the present study: 12 with PE (seven with severe PE and five with mild PE), 10 with GH (five with severe GH and five with mild GH), five with GP and seven controls. sE-selectin levels were assessed in maternal plasma at three periods: before delivery, 3–6 days after delivery and 12-14 weeks postpartum. Cases complicated by chronic hypertension, chronic renal disease, autoimmune disorders, inflammatory conditions, diabetes mellitus, or taking drugs other than vitamins were not included in the study. Control patients were matched with those with PE, GH and GP for maternal age, gestational age at delivery and gestational age at blood sampling.

Statistical analysis was performed using SAS for Windows version 9.1. Although the sample sizes were relatively small, comparisons among the various groups were performed using analysis of variance (ANOVA) with the Bonferroni correction method for post hoc analysis, since the Kolmogorov–Smirnov test for normality revealed no major deviations from normality. Mixed model analysis for repeated measurements was performed to assess the evaluation of sE-selectin levels at specified times (before delivery, after delivery and 12–14 weeks postpartum).

We found that women with severe PE and GP had significantly higher plasma sE-selectin levels as compared with controls in all three periods of sampling. In the GH group, especially in women with severe GH, sE-selectin levels were also higher than in the control group, but this trend did not reach statistical significance. During the study, there was a significant reduction in the plasma sE-selectin levels of the control group from the time before delivery until 12–14 weeks postpartum, whereas in the PE, GH and GP groups there was an increase which did not reach statistical significance (Table 1).

Although there are many studies on the serum levels of E-selectin in pre-eclamptic women [1,2], data are lacking about E-selectin in pregnant women with GP, which is believed to be a mild form of PE but has not been yet studied in depth [3,4]. Furthermore, maternal plasma levels of E-selectin have never been measured postpartum, when, as expected, the harmful effects of PE are minimal or absent. These two points are the foundation of our study.

The decision to include women with GP in our study was taken after the observation that this group had worse perinatal outcomes compared with normal pregnancies (more neonates with intrauterine growth retardation). It is interesting that sE-selectin levels of women with GP were significantly higher as compared with controls, contrarily to the sE-selectin levels of hypertensive women. That may imply that women with GP are more likely to progress to PE than women with isolated hypertension, suggesting that both clinical presentations probably share a common underlying pathophysiologic mechanism [4]. We have also observed that while high blood pressure in the PE or GH group persisted for 20–30 days

Table 1
Plasma sE-selectin levels of women with normal pregnancies (NP), preeclampsia (PE), gestational hypertension (GH), gestational proteinuria (GP) in three periods of sampling.

Category	sE-selectin levels (ng/ml) before delivery		sE-selectin levels (ng/ml) after delivery	sE-selectin levels (ng/ml) 12–14 weeks postpartum
	N	Mean ± SD	Mean ± SD	Mean ± SD
NP	7	4.91 ± 1.79[†,#]	4.03 ± 2.01	3.33 ± 1.40
Severe PE	7	11.74 ± 2.99[*]	9.92 ± 4.95[*]	13.69 ± 6.32[*]
Mild PE	5	7.51 ± 3.49	6.19 ± 3.78	9.60 ± 4.68
Severe GH	5	8.63 ± 1.76	7.71 ± 1.51	10.92 ± 2.11
Mild GH	5	6.61 ± 3.31	6.16 ± 5.07	9.36 ± 3.93
GP	5	11.20 ± 4.95[*]	8.79 ± 3.45[*]	11.13 ± 3.98[*]

Data are presented as mean ± SD.
Comparisons among the various groups were performed using analysis of variance (ANOVA) with the Bonferroni correction method for post hoc analysis.
Differences of means of sE-selectin levels between specified periods of time (before delivery, 3–6 days after delivery and 12–14 weeks postpartum) were performed with Mixed model analysis for repeated measurements.
[†] $p = 0.03$ before delivery vs. after delivery.
[#] $p < 0.001$ before delivery vs. 12–14 weeks postpartum.
[*] $p < 0.05$ vs. normal pregnancies.

after delivery, proteinuria was more persistent in the PE or GP group and in some cases receded only 8–10 weeks after delivery. However, neither hypertension nor proteinuria was observed in any of the study groups 12–14 weeks postpartum.

The other interesting aspect of our study was that no reduction, but rather a trend for higher plasma levels of sE-selectin, was noticed 12–14 weeks after delivery in women of all pre-eclamptic groups (PE, GH and GP), while a significant reduction was observed in normal pregnancies. This result firstly implies that endothelial dysfunction remains even after the 12th week postpartum, by which time, according to the definitions of the disease, PE is expected to resolve. Secondly, the trend for higher sE-selectin levels at the 12th week postpartum implies that the endothelial stimulation, which has been triggered during the pre-eclamptic pregnancy, progresses into endothelial dysfunction, a procedure that cannot be stopped even if the initial trigger, in our case PE, has been resolved. The endothelial dysfunction that persists might be the underlying mechanism that links PE with later cardiovascular disease (CVD) [5]. The increased levels of sE-selectin might reflect an increased sensitivity of the previously pre-eclamptic woman to develop CVD in the future, especially when other risk factors co-exist, such as obesity, abnormal lipid metabolism or insulin resistance.

We acknowledge that our conclusions are limited by the small numbers in our studies.

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