

# Letters to the Editor

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## Pelvic congestion/chronic pain dynamics

Dear Sir,

We read the article by Soysal *et al.* which was focusing on pelvic congestion (PC)/chronic pain (CPP) dynamics with interest (Soysal *et al.*, 2001). While reading the article, we were confused by the presentation of the methods, results and the final conclusion. We do agree with the concept and follow-up of the study. If the purpose was to identify PC by venography and all the patients had laparoscopy, what was the sensitivity/specificity of venography among their 148 CPP patients? Furthermore, why were asymptomatic patients used as controls, since they have their internal controls among symptomatic patients (without PC)? Was that group used to test the diagnostic efficacy of venography? Besides, dealing with vein stability and the possible effect of ovarian steroidogenesis on the aetiology of PC, the control group should have been age, body mass index and hormonally matched to avoid selection bias (Taskin *et al.*, 1996; Ciardullo *et al.*, 2000). Thus, endogenous hormonal status of the studied patients had to be compared.

Although the therapy arm was randomized, a placebo group or a cross-over design would be better in outlining the associated social/psychological variables/biases, despite extensive testing performed, which were indeed subjective. What was the power of the study? The limited study group impairs the validity of their conclusion. What was the power for such a statistically significant conclusion given in Table IV?

How were the hypoestrogenic side-effects of the GnRH agonist prevented? What were the effects of vasomotor reaction, mood changes and coital problems due to GnRH agonist on the tests, or how have the authors dealt with these confounding factors that may ameliorate their initial evaluation following therapy?

Most confusing is the continuing effect of GnRH agonist beyond 12 months, which needs to be explained. If estrogens were a possible cause affecting venous distensibility, congestion, and mediatory factors, (such as vascular endothelial growth factor and prostaglandins), causing pain in PC, how did the beneficial clinical effects of GnRH agonist persist despite normal estrogen status (Wang, 1993; Bausero *et al.*, 2000; Ceballos *et al.*, 2000; Ciardullo *et al.*, 2000)? Was an unknown variable, confounding factor or placebo effect missed, or a was psychosocial effect overlooked?

Moreover, as reported by Gangar *et al.* GnRH agonist/hormone replacement therapy (HRT) given for 4 months was not effective despite hypoestrogenism (Gangar *et al.*, 1993). In the present study, GnRH agonist given for 6 months without HRT revealed a prolonged effect for 12 months, despite the return of ovarian function. How can this 2 months difference end up with such a significant improvement? If the HRT makes the difference, then how did not much more elevated estradiol levels following GnRH agonist compared with HRT alter the efficacy?

Overall, the authors appear to believe that the estrogen status and/or sensitivity of pelvic veins is the underlying problem in PC (Foong *et al.*, 1992). However, neither their results or their discussion support how GnRH agonist-induced effects are maintained clinically, despite the normalized ovarian function. Moreover, how GnRH agonist therapy may be offered cost-effectively in the long-term remains elusive. What is the take home point from the study? Do they recommend adnexectomy and/or hysterectomy to the patients who did improve following GnRH agonist or intermittent GnRH agonist-only therapy (since HRT given to prevent GnRH agonist side-effects, impaired the clinical efficacy despite hypoestrogenism)?

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