

LETTERS TO THE EDITOR

Serum electrophoretic patterns in normal and preeclamptic pregnancies

To the Editor; The pathophysiology of preeclampsia is characterized by intense vasospasm of peripheral arterioles, and the initiating event in the development of this vasospastic hypertension is unknown, giving rise to a variety of theories about its etiology (1-3). This study was performed to search for a possible humoral factor which might be involved in the pathogenesis of preeclampsia, by using cellulose acetate zone electrophoresis technique. Electrophoresis was applied to sera from third-trimester preeclamptics (n=20), and third-trimester healthy pregnant.

The mean (\pm SD) serum protein bands in the healthy pregnant and preeclamptic subjects were 53.36% \pm 4.91 and 52.16% \pm 5.87 for albumin; 6.67% \pm 1.67 and 6.78 % \pm 1.29 for CM-globulin; 11.97% \pm 3.23 and 11.81% \pm 2.09 for α_2 -globulin; 12.66% \pm 2.12 and 13.19% \pm 2.68 for β -globulin; and 9.08% \pm 1.81 and 9.37% \pm 2.82 for γ -globulin, respectively. The mean values of all major protein bands in two groups did not differ from each other, all p values being >0.05. Moreover, we couldn't demonstrate any different electrophoretic band in the sera of healthy pregnant, or preeclamptic subjects.

When the literature was reviewed, one report was available determining the urinary protein profile in normal and hypertensive pregnant, by using gel electrophoresis. In this study, a protein band was detected in healthy pregnant, which was significantly, reduced or completely absent in 83% of hypertensive patients (4). When our results are evaluated, we cannot claim the disappearance of a different protein band in the sera of preeclamptics when compared to sera of healthy pregnant, since such an electrophoretic band was undetectable also in the samples of normal pregnant.

We conclude that there isn't any abnormal electrophoretic pattern in the sera of preeclamptics, to suggest a possible role of an electrophoretically detectable humoral factor in the pathogenesis of this hypertensive state.

Sallı ÇETİNER*

Gülay LOĞOĞLU*

İ.Hakkı DÜNDAR*

F.Tuncay ÖZGÜNEN*

*Central Lab. Depts. of *Physiology and
*Obst. & Gyn. Medical School of
Çukurova University, ADANA

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Treatment of oligoasthenospermia with arginine, indomethacin and kallikrein

To the Editor; Male infertility has multiple causes and similarly has varying treatments. Idiopathic oligoasthenospermia, designating those patients with an abnormality in the sperm count, motility and/or morphology is the puzzle of the treatment. In recent years kininogens, aminoacids and nonsteroidal antiinflammatory drugs gained attention in clinical practice (1-4). We studied the sperm quality and the fertilizing capacity of 40 oligoasthenospermic patients -with a sperm count of 1 million to 40 millions/ml and motility < 65 per cent- brought about by treatment with arginine, indomethacin and kallikrein. The patients were between 25-39 years old with a duration of infertility from 1.5 to 11 years (average 5.2 \pm 2.4 years). They tolerated the drugs quite well, except two people who complained about gastric symptoms due to indomethacin.

During the treatment with arginine, decrease in motility was observed in 2 subjects. No change occurred in another two, while improvement in motility was noted in the others (73.3%). Unsatisfying results were reported with 1 to 2 g. daily. But significant improvement in more than 30 per cent of the patients were achieved when larger doses (10 to 30 g. daily) were used (5). We used 10 g. daily. The amino acid L-arginine hydrochloride increases the metabolism and spermatogenesis, and also as a phosphoric acid, it interacts in the synthesis of adenosine triphosphate (ATP), a source of energy for spermatozoa motility. Arginine increased both the ratio of motile spermatozoa\$ ' the

grade of the motility. Motility increase was noted in 11 of our patients (73.3%). But these men were still in the range of oligo-and asthenospermia. In spite of these slight improvements, 3 pregnancies (20%) were reported in a short period. It would have been more realistic to treat with arginine if lower arginine levels were determined in the seminal fluid.

Indomethacin treatment had no effect in 3 patients, and worsened the sperm motility of the other 2. In 10 cases (66.6%) the sperm motility improved. Barkay et al. (1) reported stronger effects on fertility with 75 mg. indomethacin daily, compared to higher and lower doses, and described a therapeutic window. Increase in the sperm count with the same dose was very little in our study. Indomethacin probably acts by preventing the deleterious effect of prostaglandins on spermatogenesis and also by bringing about an increase in seminal fluid cAMP, which in turn increases sperm motility and fertilizing capacity (4,6). Only 1 pregnancy (6.6%) occurred in the indomethacin treated group. Our poor result was probably due to the short period of treatment (12 weeks).

There was no improvement in motility in 3 people treated with kallikrein, and decrease in 3. Improvement was achieved in 4 of them (40%). Kallikreins are enzymes that stimulate the release of kinins which are biologically active polypeptides. In addition to their various effects they also improve the sperm motility (7). Müftüoğlu et al. (8) reported significant improvement with kallikrein combinations. On the contrary, Göğüs et al, (3) noted improvement only in 7.44% of their patients treated with kallikrein, which they thought was due to the low dose (100 ku./day). The high price and the difficulty of finding the drug in our area were the handicaps of this study, which led us to use it in a

few people and in lower doses 100 ku./day). In spite of all these, we can conclude that kallikrein has not got any superiority to indomethacin or arginine.

Sabahattin AYDIN

Osman İNCİ

Bülent ALAĞÖL

**Department of Urology,
Medical School of
Trakya University, EDİRNE**

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