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 CIPROTERONE ACETATE (CPA) AND SPIRONOLACTONE (SL) :EFFECTS OF SHORT TERM ADMINISTRATION IN HIRSUTE WOMEN. V.Toscano, D.Casilli, M.Maroder, M.V.Adamo, R.Balducci\* and F.Sciarra. Istituto di V Clinica Medica, Università di Roma "La Sapienza" e Istituto di Clinica Pediatrica, II Università di Roma.

The effect of CPA (50 mg/die) and SL (50 mg/die) administered from the 5th to the 14th day of 2 different menstrual cycles were evaluated in 97 hirsute females, divided in 5 groups according to the diagnosis :peripheral or idiopathic hirsutism, polycystic ovary syndrome, late onset partial 21- $\beta$ -hydroxylase or 3 $\beta$ -ol-dehydrogenase deficiency. Androgens were evaluated by RIA after celite microcolumn chromatography, before treatment and in the last day of antiandrogen administration. CPA decreased plasma androgens in all cases, particularly 3 $\alpha$ -androstenediol (4% decrease in the 5 groups ranging between 45 and 60), followed by dihydrotestosterone (30-45), androstenedione (27-34), dehydroepiandrosterone sulfate (23-41) and testosterone (6-30). The response to short term administration of SL was variable, plasma androgens decreasing in some cases and remaining unchanged in others, independently from the diagnosis. CPA therefore is able to reduce not only androgens of ovarian/adrenal origin, but also to inhibit their utilization at skin level, as shown by the decrease in 3 $\alpha$ -androstenediol, which may be considered a useful parameter to evaluate the antiandrogenic effect. On the contrary, no definitive conclusions can be drawn after SL treatment, probably because of the low dosage and the short term of administration of the drug, showing a weaker antiandrogenic effect.

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 EFFECT OF CYPROTERONE ACETATE ON PROLACTINEMIA OF TREATED HIRSUTE WOMEN  
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Cyproterone acetate (CPA), an antiandrogen used for the treatment of early puberty, has been reported to induce hyperprolactinemia (Graf KJ and al. Acta Endocrinol 1978 suppl.215:96). Separately, dihydrotestosterone (DHT) decreases PRL release in rat anterior pituitary cells in culture (Guiguere and al. Endocrinology 1982,111:857), and 19 Nortestosterone derivative exhibits in vivo an anti-PRL effect (Sitruk Ware and al. J Clin Endocrinol Metab 1985,60:575).

In order to determine the effect of CPA in PRL secretion, we have measured basal and TRH stimulated PRL levels before and after 6 to 12 months of CPA treatment (50mg x 21 days per cycle), associated with percutaneous natural 17 $\beta$ -estradiol in 40 hirsute women.

A dramatic decrease of testosterone (T),  $\Delta_4$ -androstenedione (A) levels has been observed: (T)  $0.43 \pm 0.13$  to  $0.24 \pm 0.08$  (A)  $1.70 \pm 0.64$  to  $0.99 \pm 0.42$  ng/ml (mean + SD). Alternatively no change in basal PRL levels, neither in  $\Delta$  PRL under TRH has been noted under CPA treatment : (PRL  $15.17 \pm 15.64$  to  $10.34 \pm 10.19$  ng/ml ;  $\Delta$  PRL under TRH  $6.08 \pm 4.68$  to  $8.10 \pm 7$ ).

CPA has been proposed as a contraceptive agent (Kuttenn and al. Elsevier Science Publishers BV Endocrinology 1984,1078). Our results suggest that it can be used in women with hyperprolactinemia in whom estroprogestatifs contraceptifs agents are contraindicated. This can be of interest when nonsteroids contraceptifs agents are also contraindicated.