Impaired Prolactin Stimulation by Estrogens in Man

In their excellent review on prolactin in the last issue, Del Pozo and Brownell (1) note the stimulatory effect of estrogens on prolactin secretion. Indeed, it has been pointed out by several investigators (2,3) that estrogen treatment in humans induces a significant increase in prolactin levels within a few days. We would like to report an observation (4) in which huge amounts of endogenous estradiol (415 pg/ml) failed to stimulate prolactin secretion. An adolescent boy was referred to our clinic for evaluation of a pubertal gynecomastia. On the basis of clinical and laboratory data, diagnosis of a feminizing adrenocortical tumor was made; it was confirmed by pathological findings. Basal and dynamic studies on prolactin secretion are shown in table I.

Table I. Plasma prolactin (ng/ml)

<table>
<thead>
<tr>
<th></th>
<th>Basal</th>
<th>TRH stimulation test (peak value)</th>
<th>Phenothiazine stimulation test (peak value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient</td>
<td>5-9</td>
<td>5 ± 3</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>25+5</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>15 ± 5</td>
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From these data, it is obvious that high levels of circulating estradiol failed to modify prolactin secretion in an adolescent boy. This might reflect a difference between males and females in the positive feedback relationship of estrogens and prolactin, though it could also be due to an abnormal estrogen molecule, a minor pathological condition in the hypothala-mo-pituitary axis or a depletion of prolactin cells.

Letter to the Editor

338

Bibliographie


Chalres Sultan, MD, Bernard Descomps, MD, André Crastes de Paulet, MD and Roger Jean, MD, Department of Pediatrics and Biochemistry, St. Charles Hospital, F-34000 Montpellier (France)
Re: Sultan etal.: Impaired Prolactin Stimulation by Estrogens in Man
This is a very interesting observation since the possible effect of endogenous hyper-estrogenism on the pituitary galactotropes of a male subject has not yet been reported. However, the PRL elevation currently found in male subjects on estrogen intake is lacking here, despite plasma levels of steroid above 400 pg/ml. This would represent a high ovulatory peak in a normally cycling woman. This of course raises the question of the biological activity of the circulating estrogen, but is difficult to assess in the light of the scant information given. The presence of gynecomastia may favor such activity. However, this condition is not uncommon in adolescent boys. Is there any histological evidence of estrogenization of the mammary tissue? Furthermore, in the presence of high circulating estrogens, enough hypothalamic steroid receptors will have been induced to produce a disturbance in LH release (increased spiking?) and certainly central mechanisms will respond to clomiphene blockade, etc., theoretically. Is estrogen binding to protein increased?
It is assumed that testicular atrophy and tubular hyalinization are present, since these are typical features of hyperestrogenemia in the male.
E. del Pozo, MD