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تأثير هرمون الثيروكسين على تجمعات خلايا الليدج ونسبة هرمون التستوستيرون في دم الأرانب

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تم تعيين مستوى هرمون التستوستيرون في الدم في ثلاث مجموعات من الأرانب الذكور التي عولجت بثلاث مستويات من هرمون الثيروكسين (٥ر٦ ، ١٣ ، ١٩ر٥ مجم/كجم من وزن الجسم) . وقد أدى الثيروكسين الى زيادة عدد خلايا ليدج . وزيادة عدد خلايا ليدج مصحوبة بزيادة حيويتها ونشاطها وقيم ذلك عن طريق النسبه الخلويه السرتوليه للدورة الخلويه المنويه .

وكانت هناك علاقة عكسيه مابين جرعات الثيروكسين ونسبة هرمون التستوستيرون في الدم . وقد افترض أن خلايا ليدج مسئوله عن هرمون التستوستيرون الذى يفرز داخل الخصيه .

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EFFECT OF THYROXINE ON LEYDIG CELL CLUSTERS AND LEVEL OF BLOOD TESTOSTERONE IN RABBIT

(With One Table)

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SUMMARY

Determination of blood testosterone level in three groups of male rabbits treated with three dose levels of thyroxine 6.5, 13 and 19.5 mg/Kg body weight was performed. The thyroxine caused Leydig cell hyperplasia. The hyperplastic Leydig cells were evaluated to be biologically active by high Sertoli cell ratio of the spermatogenic cell cycle. Although the testosterone level of the blood was decreased and uncorrelated with thyroxine injection, Leydig cells were proposed to secrete intratesticular testosterone.

INTRODUCTION

Hyperplasia of Leydig cell under the effect of thyroxine injection combined with F.S.H. were demonestrated by EL-SHERRY, EL NAGGAR and NASSAR (1980) in the testicles of rabbits under summer stress. The hyperplastic cells were suggested to be non functioning because spermiogenesis was not corrected. NASSAR, FAHMI, ABDEL-ELKADER and MABROUK (1985) had recorded physiological hyperplasia of Leydig cells of normal rabbits under the effect of thyroxine adminstration. The Leydig cells was suggested to be functioning as judged by the increased Sertoli cell ratio of spermiogenesis. MAQSOOD (1951) had treated infertile rabbit by injection of thyroxine. The poor sexual desire in untreated rabbits seemed to be due interference with production of the male sex hormones by the interstitial cells of the testis. Interstitial cell showed atrophic changes. With moderate dose of thyroxine, sexual desire was improved and spermiogenesis was stimulated .

The question raised is wether hyperplastic Leydig cells in response to thyroxine therapy are secreting testosterone or non functioning ?.

The aim of this work is to measure testosterone level in the blood of rabbits subjected to three level doses of thyroxine and correlate it with the degree of Leydig cell hyperplasia.

MATERIAL and METHOD

Three doses of thyroxine 6.5, 13 and 19.5 mg/Kg body weight were given orally to three groups of Baladi rabbits 11/2 : 2 Kg body weight. Each group consisted of four rabbits. Another four rabbits were used as control. The animals were slaughtered after 12 days. Their blood was collected. Testosterone was measured in blood serum according to the method of VERMEULEN and VERDONCK (1976).

RESULTS and DISCUSSION

Testosterone level; determined in the blood of the three groups of rabbits injected by 3 doses of thyroxine; significantly decreased than the control. In the first group (6.5 mg/Kg body weight thyroxine) the testosterone decreased from 3.19 ng/ml (control level) to 0.87 ng/ml. For the second dose of thyroxine (13 mg/Kg), the testosterone level in the blood was decreased from 3.19 ng/ml (control level) to 1.38 ng/ml testosterone. So it is clear, although there is significant decrease in blood level of testosterone under the effect of thyroxine administration but was not in correlation with the dose of thyroxine. The third dose of thyroxine (19.5 mg/Kg) was accompanied by significant decrease in plasma testosterone from 3.19 ng/ml (control level) to 0.16 ng/ml.

Thyroxine injection resulted in hyperplasia of Leydig cells. The increased number of Leydig cells was in correlation with the dose level of thryoxine. The mean number of Leydig cell was for the first dose 21.9 ; 36.3 for the second dose and 42.9 for the third dose.

The main function of Leydig cell is the secretion of testosterone, as Leydig cells are the primary source of the intratesticular testosterone and circulating testosterone (RONALD, SWERDLOFF and DAVID, 1981).

Many workers use testosterone level in the blood as an index for biological activity of Leydig cells. If the present data is evaluated by this index, the hyperplastic Leydig cells would be non active. As the level of testosterone was lowered in the three doses; but this is not true.

The Sertoli cell ratio evaluation of the testicles of the three groups demonestrated high spermiogenesis correlated with the dose level of thyroxine (NASSAR,FAHMI,ABD EL-KADER and MABROUK, 1985). The Sertoli cell ratio of spermatids was 23.9 with thyroxine dose (6.5 mg/Kg) and 27.4 with the second dose (13.0 mg/Kg) and 32.9 with third dose (19.5 mg/Kg). Normal spermiogenesis is a function of testosterone. High intratesticular levels of testosterone are necessary for initiation of spermiogenesis (GERE and RICHARD, 1981). The activity of hyperplastic Leydig cell is proved by increased rate of spermiogenesis. The contradiction between low level of blood testosterone and hyperplasia of Leydig cell can be explained; that spermigoenesis depends on intratesticular testosterone rather than blood testosterone. GERE and RICHARD (1981) had demonestrated that semineferous tubules adjacent to the androgen producing tumour undergo germinal maturation while the contralateral testis remain unstimulated despite virilizing perepheral serum concentration of testosterone.

MASAHIKO, HIROYUKI, HOWARD and PHILIP (1978) stated that Leydig cell clusters providea direct index of steroid biosynthetic activity in the testis and reflect intratesticular testosterone levels.

So it is clear, that blood testosterone level is not a reflection of biological activity of Leydig cells. CARRICK and COX (1977) stated that testosterone appears to be the principal androgen secreted by the testis. However, very low concentrations of testosterone were found in blood from the internal spermatic vein. Although the accessory organs were well developed and spermatogenesis was preceeding, suggesting that other androgens may be secreted extratesticular.

The decreased testosterone level in blood with throxine administration may be due to increased metabolic activity of other testosterone target tissues which will pound this hormone.

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TABLE (1):

Testosterone in (ng/ml serum) from control and thyroxine (6.5, 13 and 19.5 mg/Kg body weight) treated rabbits.

Dose	Case number	1	2	3	4	mean
Control	ngm/ml	3.94	3.13	3.8	1.9	3.19 ± 0.5
6.5 mg/ml	ngm/ml	0.91	2.16	0.18	0.24	0.87* ± 0.5
	%	71.5	32.3	94.4	91.5	72.7
13 mg/ml	ngm/ml	0.75	1.85	0.43	2.5	1.38* ± 0.5
	%	76.5	42.0	86.5	21.6	56.7
19.5 mg/ml	ngm/ml	0 very low	0.36	0.1	0.18	0.16** ± 0.1
	%	-	88.7	96.9	94.4	95.0

* : (P/ 0.05).
** : (P/ 0.01).