A case report: a hCG secreting testicular seminoma discovered during routine infertility evalution

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ÖZET

Rutin infertilite değerlendirilmesi sırasında tanı konulan Human Koryonik Gonadotropin sekrete eden testiküler seminoma olgusu sunulmuştur. Sperm sayı ve motilitesi normal olmasına rağmen, yıkama sonrası sperm swim-up sonuçları normalden oldukça düşük idi. Bu değerler orşiektomiden sonra normale döndü.

Anahtar Kelimeler: Seminoma, infertilite, hormon.

SUMMARY

We present a human chorionic gonadotropin producing testicular seminoma case which was discovered during infertility evaluation. Although sperm count and motility were normal, postwash sperm swim-up results were poor that returned to normal after orchiectomy.

Key Words: Seminoma, infertility, hormon, surgery, swim-up.

INTRODUCTION

Sperm analysis of patients with testicular cancer are commonly severely oligozoospermic. Only 30% to 40 % of the patients have the sperm densities above 10 million/ml before orchiectomy or any additional therapy(1-3).

Histopathologic studies show impaired spermatogenesis in ipsilateral and contralateral testis with unilateral testicular tumors(4,5). Carcinoma in situ in contralateral testis are also shown in 5% of the testicular tumor patients(4). Autoimmun factors, increased scrotal temperature, direct compression are the other probable causes(3,5,6). Some congenital abnormalities (undescended testis, gonadal dysgenesis) may have tendency both to testicular tumors and infertility.

Germinal tumors of the testis can cause endocrine and exocrine changes in some of patients (1,3,7). In a group of testicular tumor causes hormonal changes are the main sources of infertility. We present a hormonally active testicular seminoma with infertility who has elevated beta-human chorionic gonadotropin (hCG) level and normal

sperm count and motility but poor quality sperm swim up.

CASE REPORT

Twentynine year old male presented 2 years history of infertility. His wife was found to be normal. Physical examination revealed a normal right testis. The left testis was significantly bigger than its mate. Gynecomastia was not present. Semen analysis with swim-up showed an excellent density of 120 million/ml, 50% motility and normal morphology of 56%. More significantly his postwash swim-up density was poor quality and only 250.000 sperms were able to swim up to primary fraction(Table 1).

In routine endocrine assay, estradiol(E2) and testosteron(T) levels were higher than normal. FSH and LH levels were below the normal limits. Endocrine assay was repeated and similar profile was found (Table 2). Testicular ultrasound was performed, the right testis and epidydimis were found normal. On the left side heterogenous hypoechoic mass replacing the whole testis was demonstrated with increased color flow worrisome for neoplasm. Serum beta-hCG was higher than normal 139 mIU/mI

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(upper limits of normal 27.8). Serum alpha- fetoprotein and lactic acid dehydrogensase levels were in normal limits. No metastasis was detected. Left radical orchiectomy was carried out. Histopathology showed classic seminoma with trophoblastic cells. His swim-up improved after orchiectomy with normal sperm count and motility (Table 1). Hormonal profile of the patient returned to normal including beta-hCG(Table 2).

DISCUSSION

Ten to twentyeight percent of the testicular seminoma cases have elevated beta-hCG levels (7). It was shown that elevated hCG increases estradiol levels resulting in supression of basal gonadotropin levels in patients with testicular seminomas(1,3,7). These hormonal changes cause gynecomastia or

oligoasthenospermia(1,3,4,6). During evaluation of male infertility, elevated E2 levels with lower than normal gonadotropin levels may be detected(1,3). This situation may remind the physician of leydig cell tumor of testis. We did not take into account Leydig cell tumor because of elevated hCG (8).

hCG secreting testicular tumor tissue can act as a normal trophoblastic tissue and can produce estrogens (9). Elevated hCG also stimulates Leydig cells and can cause increased production of estrogens and androgens with Leydig cell hyperplasia (7) In these cases elevation in estrogen levels may be higher than testesteron levels(3). FSH is supressed by elevated estrogens.

In this case elevation in E2 and testosterone levels due to hCG secreting testicular tumor sup-

Table 1. Pre- and postoperative semen parameters of the case.

	Dunne	Doctor	Ranges
	Preop.	Postop.	naliges
Volum (ml)	2.4	3.4	1.5 - 4.0
Density (million/ml)	120	80	>20
Motility (%)	50*	55*	>30
Normal morphology (%)	56	46	>30
Postwash swim-up density (million/ml)	0.25**	2.5**	>1

forward progression > 50%

Table 2. Pre- and postoperative hormon profile of the case.

of the Company starting to the man of this set by a 65 Boltzman was a sum of the control of the set	Preop.	Postop.	Ranges
Estradiol (pg/ml)	73	22	6-54
FSH (mIU/ml)	<0.3	4.8	0.9-15
LH (mIU/ml)	0.1	7.6	1.3-12.9
Prolactin (ng/ml)	16.4	14.6	0.0-21.0
Total Testosterone (pmol/L)	161.0	76.0	32.0-96.0
Alpha-fetoprotein	2.5		0.00-8.90
Beta-HCG (m'U/ml)	139	2.5	<27.8

^{**} forward progression > 90%

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ressed the FSH and LH levels. Although we detected slightly elevated testosterone levels, this is not a rule for hCG secreting tumors. In a study, 30 of the 42 patients had elevated beta-hCG levels. This group showed normal testosterone levels and there were no significant difference from the hCG negative group(1). Some studies revealed decreased FSH levels with decreased Sertoli cell function along with diminished sperm count (3,4). In this case, despite decreased FSH level, sperm count and motility were found in normal ranges. However, postwash swim-up clearly showed impairment. This result may indicate reduced sperm function.

Significant medical pathologies can be discovered during evaluation of male infertility. In a study six testicular tumors were found amongst 1236 patient evaluated for male infertility(10). We incidentally diagnosed a hormonally active testicular tumor during routine evalution. Gonadal dysgenesis may be an etiologic factor for both inferility and testicular seminoma (1,3,7).

Urologists should keep the possibility of a testicular tumor in mind during routine infertility evaluations, especially in patients with abnormal hormon profiles.

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