

Clinical characteristic of men with testicular lesions: single center experience

Elżbieta Oszukowska¹, Waldemar Różański², Marek Wrona², Piotr Lipiński², Marek Lipiński², Magdalena Jakubiak-Wielganowicz³, Krzysztof Kula¹, Jolanta Słowikowska-Hilczar¹, Marek Sosnowski⁴

¹Department of Andrology and Reproductive Endocrinology, Medical University of Łódź, Poland

²II Clinic of Urology, Medical University, Łódź, Poland

³Pathology Unit, Department of Oncology, Medical University, Łódź, Poland

⁴I Clinic of Urology, Medical University, Łódź, Poland

KEY WORDS

testis ► germ cell tumors ► AFP ► beta-hCG ► CIS/TIN

ABSTRACT

Introduction. Testicular lesions are an indication for testis removal; 95% are testicular germ cells tumors (TGCT). The aim of the work was to present the conditions leading to castration and the clinical stages of the disease.

Material and method. Case histories and subsequently treated patients were retrospectively analyzed. The type and size of the testicular lesion as well as pT and clinical stage (in cases of TGCT) were analyzed.

Results. Non-seminomas (NSGCT) were found in 51.9%, seminomas (SEM) in 35.8%, and non-TGCT lesions in 12.3% of cases. Bilateral TGCT was found in 4.3%. The mean TGCT diameter was 41.4 mm. The incidence of tumors <20 mm was significantly higher in SEM than in NSGCT. Tumor size in SEM stage I (34.6 ± 21.9 mm) was significantly smaller than in stage II (57.5 ± 28.8 ; $p < 0.05$). The percentage of subjects with nodal enlargement and higher stage of disease was significantly higher in NSGCT than in SEM. The incidence of men in stage I of disease was 52.1% in all; 75.9% in SEM and 35.7% in NSGCT.

Conclusions. High incidence of bilateral TGCT indicates the need for careful follow-up after unilateral TGCT treatment. Large tumor diameter and high percentage of subjects with higher stage of disease may suggest a diagnostic delay and/or low awareness of testicular cancer among young men, particularly those with NSGCT.

INTRODUCTION

Testicular tumors in 95% of cases are germ cells in origin (testicular germ cell tumors – TGCT) and about 1–3% originate from Leydig or Sertoli cells. In the remaining cases, lymphomas or other benign lesions are found. TGCT are the most frequent malignant tumors among young men between 15 and 40 years of age. The incidence of TGCT increases from puberty and achieves the highest level at about 32 years of the age [1]. An increasing incidence of TGCT has been described over the last several decades [1–6]. Seminoma (SEM) develops in older men (30–45 years), while non-seminoma (NSGCT) usually occurs in younger men between 20 and 40 years of age [1, 4–6].

NSGCT cells may produce alpha-fetoprotein (AFP) and/or beta subunit of human chorionic gonadotropin (β -hCG). In turn, in men with SEM, levels of AFP are most often within normal range, while increased levels of β -hCG may be observed in about 30% of patients [7]. Measurement of blood levels of AFP and β -hCG is used for diagnostics and monitoring treatment. TGCT in a short time may give metastases. The clinical stage of disease according to the modified TNM classification [7] or the American Joint Committee on Cancer (AJCC) classification of clinical stages [8] may depend on tumor size and diagnostic delay. Huyghe et al. showed that diagnostic delay correlated with disease stage for NSGCT and had impact on the 5-year survival rate in the NSGCT group [9].

TGCT may be accompanied by *carcinoma in situ* (CIS) called also *testicular intraepithelial neoplasia* (TIN) [7, 10]. It is believed that CIS/TIN cells originate from altered fetal germ cells (gonocytes) and undergo clonal expansion, probably under the influence of gonadotropins and testosterone giving rise to the overt TGCT [11–14].

Leydig cell tumors are usually benign and have a small diameter. They may secrete high amounts of testosterone and estradiol and produce signs of hyperestrogenism with gynecomastia. Gynecomastia may also accompany NSGCT, especially in cases with high levels of β -hCG.

The aim of the work was a presentation of causes leading to the castration. In subjects with TGCT tumor size, histological types were compared with clinical stage of disease and outcome of treatment.

MATERIAL AND METHODS

Case histories of subjects treated due to testicular lesions in the II Clinic of Urology of the Medical University of Łódź from 2001 to 2006 were reviewed. The type and size of the lesion was analyzed in histopathological reports. In cases of TGCT, the vascular invasion, infiltration of neighboring structures, and pathological T stage were analyzed. The presence of enlarged retroperitoneal lymph nodes or distant metastases was assessed. Blood levels of β -hCG and AFP before hemi-castration were compared with the type of TGCT and clinical stage.

Statistical analysis was performed by Statistica 7.1 (StatSoft, Cracow). Non-parametric tests (U Mann-Whitney, Chi-square, and r-Pearson) were used (p value < 0.05 was considered as statistically significant).

RESULTS

Over the period of 5 years, hemi-castration was performed in 79 subjects while bilateral orchidectomy was done in one man;

altogether 81 testes were removed. All men were between 15 and 70 years old.

In 10 cases (12.3% of all), tumors not derived from germ cells were found, in 3 lymphoma and in 7 benign lesions like: leydigoma – 1, testicular abscess imitated testicular tumor – 2, epidermal cyst – 2, lipoma surrounding testis – 1 and tuberculosis of the testis with active inflammation process – 1. The subjects were between 20 and 59 years old.

In 71 gonads (87.7% of all) TGCT were found. The subjects with TGCT were between 15 to 52 years old (mean 30.6 ± 9.4) and 82.9% of them were between 20 to 44 years of age.

SEM and NSGCT were recognized in 29 (35.8%) and in 42 (51.9%) gonads, respectively. Men with SEM were from 24 to 52 years old, mean 36.0 ± 8.5 years and they were significantly older by about 10 years than men with NSGCT (26.8 ± 8.2 ; 15–50; $p < 0.01$). Twenty-three (79.3%) men with SEM were older than 25 and younger than 44 years of age. The other 30 (73.1%) men with NSGCT were younger than 29 years of age and 5 of these (11.9%) were younger than 19 (Fig. 1).

Mean tumor size in the whole group was 41.4 ± 25.2 mm and did not differ between groups (Table 1). The incidence of tumors with diameter below 20 mm was 11.2% in all and was significantly higher in SEM (21.4%) than in NSGCT (5.6%, $p = 0.037$). Large tumors with diameter greater than 50 mm were more often found in NSGCT. The occurrence of tumor occupation of whole testis and multifocal lesions did not differ between groups. The incidence of vascular invasion, infiltration of rete testis, epididymis, or testicular tunics were significantly higher in NSGCT than in SEM, thus the incidence of pT2 stage was significantly higher in NSGCT than in SEM (Table 1).

β -hCG and AFP levels were within normal range in SEM patients except one subject, who only had a slightly elevated β -hCG level (Table 2). Elevated levels β -hCG or AFP were observed in 85.7% subjects with NSGCT. Subjects with NSGCT and metastases had significantly higher β -hCG blood levels, but not AFP, in comparison to those without metastases. In subjects with NSGCT containing embryonal carcinoma without enlarged retroperitoneal lymph nodes or distant metastases the positive correlation between AFP blood level and tumor diameter in the testis was found ($r = 0.58$; $p = 0.045$).

Stage I of disease according to AJCC was established in 37 subjects (52.1% of all) (Table 3). The incidence of stage I in the SEM group was significantly higher than in NSGCT. In NSGCT, the frequency of enlarged retroperitoneal nodes was significantly higher (57.1%) than in SEM (24.1%, $p = 0.006$). In one case of SEM (3.4%) and 6 of NSGCT (14.3%), uni- or bilateral hydronephrosis was found because of nodal enlargement. Only in NSGCT, distant

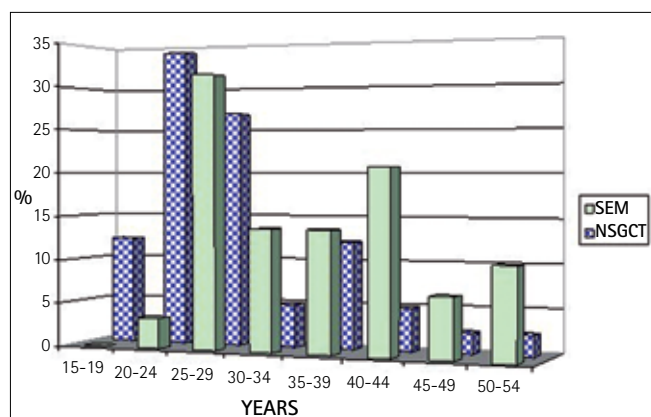


Fig. 1. Percentage of subjects with seminoma (SEM) or nonseminomatous (NSGCT) tumors in 5-year age groups.

Table 1. Tumor size, incidence of multifocal lesions, vascular invasion, and pathological T2 stage in men with seminoma (SEM) or nonseminomatous germ cell tumor (NSGCT).

	SEM (n = 29)	NSGCT (n = 42)	p
Tumor diameter (mm) mean $\pm$ SD range	39.4 $\pm$ 25.3 7 – 110	44.0 $\pm$ 26.5 15 – 110	NS
Tumor diameter <20 mm	6 (21.4%)	2 (5.6%)	0.037
Tumor diameter >50 mm	5 (17.9%)	9 (25.0%)	NS
Occupation of whole testis	11 (37.9%)	21 (50.0%)	NS
Multifocal lesions	6 (20.7%)	7 (16.7%)	NS
Vascular invasion	4 (13.8%)	16 (38.1%)	0.049
Rete testis, epididymis or testicular tunics infiltration	2 (6.9%)	16 (38.1%)	0.0071
pT2 stage	5 (17.2%)	23 (54.8%)	0.0034

Table 2. Blood levels of AFP and β -hCG before hemicastration in subjects with seminoma (SEM) or nonseminomatous germ cell tumor (NSGCT) with and without nodal (N) or distant (M) metastases.

	SEM (n = 29)	NSGCT (n = 42)	NSGCT N(-) M(-) (n = 15)	NSGCT N(+) or M(+) (n = 27)	Normal range
β -hCG (mIU/ml) median range	1.3 0.0 – 32.5	32.13 ** 0.1 – 361 037	6.9 0.1 – 292	280.0 ^a 0.1 – 361 037	0 – 2.6
AFP (U/ml) median range	1.7 0.0 – 6.0	99.27 ** 0.9 – 14 241	19.8 2.0 – 456	110.4 0.9 – 14 241	0 – 6.0

** $p < 0.001$ vs SEM; ^a $p < 0.05$ vs NSGCT N(-)M(-) (U Mann-Whitney test).

metastases (6 cases, 19%) and stage III of disease were observed (Table 3).

Testicular tumor size in men with SEM stage I was significantly smaller (34.6 ± 21.9 mm) than in those with stage II (57.5 ± 28.8 ; $p < 0.05$). Similarly, in NSGCT, the mean size of tumor in men with stage I (35.1 ± 25.9 mm) was lower than in those with metastases (47.0 ± 24.7 mm), but not significantly.

Both gonads were removed in 3 men (4.3% of all studied) because of bilateral TGCT. Two of them aged 28 and 43 years presented SEM after 3 and 4 years (respectively) from hemi-castration because of NSGCT. In a 33 year old patient, both testes were removed in one session because of bilateral multifocal TGCT; right testis revealed embryonal carcinoma and the left was diseased with multifocal seminoma and teratoma.

Mean follow-up after treatment was 67 mos. (range 90–36 mos.). Seventeen patients from the SEM group received retroperitoneal lymph node irradiation, 7 were treated with cisplatin-based chemotherapy, and 5 were entered into a surveillance group. At this time twenty-seven patients with SEM are alive; medical records of 2 subjects were incomplete.

All 42 subjects with NSGCT were treated with cisplatin-based chemotherapy. Six patients had surgical resection of residual retroperitoneal masses after chemotherapy. In 2 cases, subjects had relapse treated by high-dose chemotherapy with good result.

Table 3. Clinical stage according to AJCC in patients with seminoma (SEM) or nonseminomatous germ cell tumor (NSGCT).

		SEM n (%)		NSGCT n (%)		P	all n (%)
I	IA	18 (62.1%)	22 (75.9%)	2 (4.8%)	15 (35.7%)	0.002	37 (52.1%)
	IB	3 (10.3%)		4 (9.5%)			
	IS	1 (3.4%)		9 (21.4%)			
II	IIA	4 (13.8%)	7 (24.1%)	8 (19.8%)	17 (40.5%)	NS	24 (33.8%)
	IIB	2 (6.9%)		5 (11.9%)			
	IIC	1 (3.4%)		4 (9.5%)			
III	IIIA	0	0	3 (7.1%)	10 (23.8%)	0.013	10 (14.1%)
	IIIB	0		4 (9.5%)			
	IIIC	0		3 (7.1%)			

Twenty-eight patients are alive, in 9 cases medical records are incomplete, and 5 patients had died of their TGCT disease (11.9%).

In 8 subjects from SEM and 14 from NSGCT, blood levels of FSH, LH, testosterone, and estradiol before hemi-castration were estimated. Reference ranges for FSH were 1.5–12.4 mIU/ml, for LH 1.7–8.6 mIU/ml, for testosterone 12–35 nmol/l, and for estradiol 45–150 pmol/l. In NSGCT, levels of FSH (median 0.4, range 0.0–6.2 mIU/ml) and LH (1.0, 0.0–7.6 mIU/ml) were significantly lower than in SEM (9.1, 1.9–19.4, $p < 0.05$; 5.0, 2.1–8.0, $p < 0.05$, respectively), while testosterone (26.0, 12.8–44.1 nmol/l) and estradiol (164.0, 112–2453 pmol/l) were higher than in SEM (18.4, 10.4–26.0; 122.6, 42.8–125.4, respectively).

DISCUSSION

Mean tumor size in all patients (41.4 mm) was larger than described by other authors [15, 16]. Bhardwa et al. showed that in London the mean tumor size at the time of diagnosis was 2.5 cm, which had declined from 4 cm in the last two decades [15]. Additionally, the proportion of tumors less than 20 mm was 23%. This is a higher value than in our study (11.3%). Long duration of disease before referral may be one cause of large tumor size and metastatic disease. Vasudev et al. conducted a study evaluating changes in referral time and patients' awareness among men over the past 18 years [17]. They showed that the median time between first symptoms noticed by men and their first visit to GP decreased from 5 to 2 weeks, but 16% of men waited still longer than 2 months. They also showed increased awareness of testicular cancer among young men. Huyghe et al. showed an impact of duration of diagnostic delay on stage of disease and survival in subjects with NSGCT [9]. Their results support our observation, that larger tumor size probably due to longer duration of disease before referral and definite treatment may be a reason of higher disease stage at the diagnosis. Urologists should promote programs to enhance awareness and knowledge of testis cancer. This is more important when higher stages of disease are observed in very young patients with NSGCT. Some subjects with NSGCT with high tumor markers and metastases may be resistant to chemotherapy, so they benefit from earlier diagnosis [18].

Majority of patients with TGCT were 20–29 years old. The incidence of NSGCT was higher than SEM and similar distribution described by others excludes any selection bias during the study [19]. The youngest men were in the NSGCT group; 56% of them were younger than 24 years of age and 15.6% were younger than 19. This very early development of TGCT may suggest that increased levels of gonadotropin and testosterone associated with puberty are involved in TGCT development. In fact Slowikowska-Hilczner et al. showed a positive correlation between number of CIS cells and blood FSH and LH levels in children with gonadal dysgenesis before puberty [13]. It has been recently shown that impaired

testicular organogenesis predisposes to formation of CIS/TIN and also overt TGCT [14, 20, 21].

Our study showed a worse clinical stage of disease in the NSGCT group. pT stage is one of the risk factors for relapse. The main cause increasing pT stage was vascular invasion according to Lackner et al. and in our material the incidence of vascular invasion was significantly higher in the NSGCT than in SEM group (Table 1) [19]. According to AJCC classification all pT stages may be included into stage I, while vascular invasion was recognized as the risk factor for relapse in the clinical stage I NSGCT with positive predictive value of 52.7% according to Leibovitch et al. [22]. The percentage of subjects with stage I disease in the NSGCT group was significantly lower than in SEM, and additionally subjects with stage III of disease were observed only in NSGCT group. The proportion of patients with stage I disease in our study (52.1%) was lower than described by Bhardwa et al [15].

Mean tumor size did not differ between groups, but we noticed that the patients with SEM in stage I of disease had significantly smaller tumor diameter than those with stage II. Warde et al. showed that patients with SEM stage I and tumor size greater than 4 cm were more likely to relapse than those with smaller tumors [23]. Subjects with NSGCT stage I disease also had smaller tumor diameter than those in stage II, but not significantly. Beck et al. showed that primary tumor size was not predictive for retroperitoneal metastases in patients with NSGCT [16]. They suggested that volume of individual histologic type may be more useful. Leibovitch et al. showed that the volume of embryonal carcinoma in primary tumors less than 2 ml decreases the risk of retroperitoneal metastases [22].

Our study showed that an important problem in therapy of men with TGCT is the risk of TGCT development in the second testis. The incidence of bilateral TGCT in our material (4.3%) was similar to the one described previously by Sosnowski et al. in the same region, but higher than reported by other authors (1.9–2%) [24–27]. Sometimes in subjects with bilateral TGCT an organ preserving testicular tumor resection is proposed [28]. It may be considered when the size of the tumor is less than 20 mm. Multiple biopsies from the bed of the tumor, to assess CIS/TIN and local postoperative irradiation, are necessary to avoid local recurrence. Heidenreich et al. did not observe local relapse in 46 patients with associated CIS/TIN who had undergone organ preserving tumor resection and local irradiation [28]. CIS/TIN is a precursor of TGCT and early detection of CIS/TIN in the contralateral testis and effective treatment by irradiation may preserve the gonad as a source of endogenous testosterone, but the indications to perform contralateral testicular biopsy are still debated [7, 24, 29]. Adversaries stress the rare occurrence of CIS/TIN in the contralateral testis (5%) and low stage of disease at the time of second tumor diagnosis among subjects with metachronous TGCT [27, 30]. On the other hand, the incidence of second TGCT among patients who received adjuvant chemotherapy or those who received chemotherapy for metastases did not decrease compared to those in surveillance [26]. Moreover, early detection and treatment of CIS/TIN is believed to be a cause of declining morbidity from TGCT among men from Denmark [6]. Taking two or three specimens from the testis increases the detection rate of TIN/CIS up to 8.1%, particularly among men with normal testicular volume [31]. It seems that contralateral testis biopsy may be proposed to young patients with small testicular volume (<12 ml), a history of cryptorchidism, or poor semen parameters who haven't yet had a child [7]. If patients seek fatherhood, cryopreservation of semen or sperm extraction combined with cryopreservation may be performed prior to further treatment [32].

In the majority of cases with testicular lesion, TGCT was found. Non-germ cell tumor was found in 12.3% of subjects. Benign

epidermal cyst and lymphomas were found the most frequently. Epidermal cysts make up about 50% of benign testicular lesions. It is however important to differentiate a cyst from a mature teratoma. Epidermal cysts may be treated by partial resection of the testis [33].

CONCLUSIONS

High incidence of bilateral TGCT indicates the necessity for careful follow-up after unilateral TGCT treatment. Benefits of contralateral testicular biopsy should also be taken into consideration.

Large mean tumor diameter and high percentage of subjects with stage II or III disease may suggest a diagnostic delay and/or low awareness of testicular cancer among young men, particularly those with NSGCT.

Acknowledgments:

Supported by Medical University of Łódź fund nr. 502-11-427 and 503-1089-2/3.

REFERENCES

- Moller H: *Trends in incidence of testicular cancer and prostate cancer in Denmark*. Hum Reprod 2001; 16: 1007-1011.
- Bergstrom R, Adami HO, Mohnher M et al: *Increase in testicular cancer incidence in six European countries: a birth cohort phenomenon*. J Natl Cancer Inst 1996; 88: 727-733.
- Huyghe E MT, Thonneau P: *Increasing incidence of testicular cancer worldwide: a review*. J Urol 2003; 170 (1): 5-11.
- Huyghe E, Plante P, Thonneau PF: *Testicular cancer variations in time and space in Europe*. Eur Urol 2007; 51: 621-628.
- Richiardi L, Bellocco R, Adami HO et al: *Testicular cancer incidence in eight northern European countries: secular and recent trends*. Cancer Epidemiol Biomarkers Prev 2004; 13: 2157-2166.
- Jacobsen R, Moller H, Thoresen SO et al: *Trends in testicular cancer incidence in the Nordic countries, focusing on the recent decrease in Denmark*. Int J Androl 2006; 29: 199-204.
- Albers P, Albrecht W, Algaba F et al: *Guidelines on Testicular Cancer*. European Association of Urology, 2010; pp. 4-50; www.uroweb.org
- Greene FL, Page DL, Fleming ID et al (eds): *AJCC Cancer Staging Manual*. 6th edition. New-York, Springer-Verlag, 2002; Part IX, pp. 317-322.
- Huyghe E, Muller A, Miesusset R et al: *Impact of diagnostic delay in testis cancer: results of a large population-based study*. Eur Urol 2007; 52: 1710-1716.
- Oosterhuis JW, Kersemaekers AM, Jacobsen GK et al: *Morphology of testicular parenchyma adjacent to germ cell tumors. An interim report*. Apmis 2003; 111: 32-40; discussion 41-32.
- Reuter VE: *Origins and molecular biology of testicular germ cell tumors*. Mod Pathol 2005; 18 Suppl 2: S51-60.
- Slowikowska-Hilczer J, Walczak-Jędrzejowska R, Kula K: *Immunohistochemical diagnosis of preinvasive germ cell cancer of the testis*. Folia Histochem Cytobiol 2001; 32: 67-72.
- Slowikowska J, Szarras-Czapnik M, Walczak-Jędrzejowska R: *Gonadotropins and preinvasive testicular germ cell carcinoma*. Ped Pol 2002; LXXVII: 1051-1056.
- Slowikowska-Hilczer J, Romer TE, Kula K: *Neoplastic potential of germ cells in relation to disturbances of gonadal organogenesis and changes in karyotype*. J Androl 2003; 24: 270-278.
- Bhardwa JM, Powles T, Berney D et al: *Assessing the size and stage of testicular germ cell tumors: 1984-2003*. BJU Int 2005; 96: 819-821.
- Beck SD, Foster RS, Bihle R, Donohue JP: *Significance of primary tumor size and preorchietomy serum tumor marker level in predicting pathologic stage at retroperitoneal lymph node dissection in clinical Stage A nonseminomatous germ cell tumors*. Urology 2007; 69: 557-559.
- Vasudev NS, Joffe JK, Cooke C et al: *Delay in the diagnosis of testicular tumors - changes over the past 18 years*. Br J Gen Pract 2004; 54: 595-597.
- Bosl GJ, Motzer RJ: *Testicular germ-cell cancer*. N Engl J Med 1997; 337: 242-253.
- Lackner JE, Koller A, Mazal PR: *The changing distribution of pT stage in testicular germ cell tumors from 1976 to 2005: a single-centre analysis of histopathological reports*. BJU Int 2006; 98: 747-750.
- Skakkebaek NE, Holm M, Hoei-Hansen C et al: *Association between testicular dysgenesis syndrome (TDS) and testicular neoplasia: evidence from 20 adult patients with signs of maldevelopment of the testis*. Apmis 2003; 111: 1-9; discussion 9-11.
- Slowikowska-Hilczer J, Szarras-Czapnik M, Kula K: *Testicular pathology in 46 XY dysgenetic male pseudohermaphroditism: an approach to pathogenesis of testis cancer*. J Androl 2001; 22: 781-92.
- Leibovitch I, Foster RS, Kopecky KK et al: *Identification of clinical stage A nonseminomatous testis cancer patients at extremely low risk for metastatic disease: a combined approach using quantitative immunohistochemical, histopathologic, and radiologic assessment*. J Clin Oncol 1998; 16: 261-268.
- Warde P, Specht L, Horwich A et al: *Prognostic factors for relapse in stage I seminoma managed by surveillance: a pooled analysis*. J Clin Oncol 2002; 20: 4448-4452.
- Sosnowski M, Kula K: *Diagnosis and treatment of carcinoma in situ within contralateral testis of men with unilateral testicular germinal cancer*. Urol Pol 1997; 50: 1: 9-16.
- Sosnowski M, Wilkosz J, Salagierski M: *Bilateral testicular tumors*. Urol Pol 2000; 53 (3-4): 331-338.
- Oliver RT, Ong J, Berney D et al: *Testis conserving chemotherapy in germ cell cancer: its potential to increase understanding of the biology and treatment of carcinoma-in-situ*. Apmis 2003; 111: 86-91; discussion 91-82.
- Tekin A, Aygun YC, Aki FT, Ozen H: *Bilateral germ cell cancer of the testis: a report of 11 patients with a long-term follow-up*. BJU Int 2000; 85: 864-868.
- Heidenreich A, Weissbach L, Holtl W et al: *Organ sparing surgery for malignant germ cell tumor of the testis*. J Urol 2001; 166: 2101-2165.
- Petersen PM, Daugaard G, Rorth M, Skakkebaek NE: *Endocrine function in patients treated for carcinoma in situ in the testis with irradiation*. Apmis 2003; 111: 93-98; discussion 98-99.
- Albers P, Goll A, Bierhoff E et al: *Clinical course and histopathologic risk factor assessment in patients with bilateral testicular germ cell tumors*. Urology 1999; 54: 714-718.
- Kliesch S, Thomaidis T, Schutte B et al: *Update on the diagnostic safety for detection of testicular intraepithelial neoplasia (TIN)*. Apmis 2003; 111: 70-74; discussion 75.
- Kliesch S, Bergmann M, Hertle L, et al: *Semen parameters and testicular pathology in men with testicular cancer and contralateral carcinoma in situ or bilateral testicular malignancies*. Hum Reprod 1997; 12: 2830-2835.
- Zajączkowski T, Straube W: *Epidermoid cyst of the testis*. Urol Pol 2001; 54: 22-24.

Correspondence

Elżbieta Oszukowska
Medical University of Łódź
Department of Andrology and Reproductive Endocrinology
5, Sterling Street
92-425 Łódź, Poland
phone: +48 42 633 07 05
elzbieta.oszukowska@umed.lodz.pl