ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
Index Copernicus Value: 49. 23
PubMed - National Library of Medicine - ID: 101731606

International Journal of Medical Science and Innovative Research (IJMSIR)
IJMSIR : A Medical Publication Hub
Available Online at: www.ijmsir.com
Volume – 4, Issue – 1, January - 2019, Page No. : 66 - 77

Testicular Seminomas: Evaluation of 34 Cases with Histopathological Parametres

[1]Dr.Duygu Ayaz

[1]Department of Pathology, Turkish Ministry of Health - Tepecik Training and Research Hospital, İzmir, Turkey

Corresponding Author: Dr. Duygu Ayaz, Department of Pathology, Turkish Ministry of Health - Tepecik Training and Research Hospital, İzmir, Turkey

Type of Publication: Original Research Paper

Conflicts of Interest: Nil

Abstract

Introduction: Testicular tumors are rarely seen, accounting for 1-2% of malignant tumors in males and 13-23% of male urogenital system tumors. They are usually seen between the ages of 15-45. Germ cell neoplasia *in situ* (GCNIS) is considered to be the precursor of all testicular germ cell tumors except spermatocytic seminoma. Seminoma is the most common germ cell tumor of the testis. Pure seminoma constitutes approximately 50% of all testicular germ cell tumors. Seminomatous germ cell tumors consist of seminoma and spermatocytic seminoma according to the 2004 World Health Organization classification. In addition, the sincytiotrophoblastic giant cell variant of the seminoma and the sarcomatous component variant of the spermatocytic seminoma are included in the classification.

Materıal and method: H & E stained slides of 34 cases of seminoma diagnosed in the Department of Pathology of Izmir Training and Research Hospital between 1989-1999 were re-examined and classified according to 2004 World Health Organization classification. Histopathological parameters of the cases were reviewed. PAS and PAS diastase stains were applied to evaluate the glycogen content of the tumor in all cases. In addition, PAS was applied to investigate the presence of ITGCNU in slides containing non-tumoral testicular tissue and the diagnostic usefulness of PAS stain was evaluated.

Results: Thirty-four cases of seminoma included in our study were evaluated as seminoma (n = 25; 73.5%), a variant of seminoma containing syncytiotrophoblastic cells (n=8; 23.5%), and spermatocytic seminoma (n=1. 3%). The mean age of the patients was 40.1 (16-72) years. The mean tumor size was 6.4 cm (1.1-15 cm). GCNIS was observed in samples obtained from non-tumoral testicular tissue of 13 (52%) of 25 patients

Conclusion: The histopathological features of the tumors in our series are largely consistent with the literature. In all cases with presumptive diagnosis of GCNIS based on H & E stained sections was also confirmed by PAS staining.

Keywords : Germ-cell neoplasia in situ, seminoma, testis.

Introduction

Testicular tumors are rarely seen and constitute 1-2% of malignant tumors in males and 13 -23% of male urogenital tumors [1] They are usually seen between the ages of 15-45. They are rarely seen in the elderly, but peak in adolescents. In recent years, the incidence of

testicular tumors worldwide is increasing day by day [2-4].Conditions associated with testicular germ cell tumors are cryptorchidism, pre-existence of testicular germ cell tumors, intersex syndromes and oligospermic infertility [5]. Germ cell neoplasia in situ (GCNIS) is considered to be the precursor of all testicular germ cell tumors except spermatocytic seminoma [6]. When the patients with GCNIS have been followed up, development of invasive germ cell tumor was seen within 5 years [7,8].Seminoma is the most common germ cell tumor of the testis. The pure seminoma constitutes approximately 50% of all germ cell tumors of the testis. The median ages at the onset of the disease for seminomatous, and non-seminomatous germ cell tumors are 40, and 50 years, respectively [9]. Clinically, most patients have a painless mass in the ipsilateral testicle [2].Seminomatous germ cell tumors consist of seminoma and spermatocytic seminoma according to the 2004 World Health Organization classification. Also, syncytiotrophoblastic giant cell variant of seminoma and sarcomatous component variant of spermatocytic seminoma have been included in the classification [10].

Material and methods

H & E stained slides of 34 cases with seminoma diagnosed in the Department of Pathology of Izmir Training, and Research Hospital between 1989-1999 were re-examined and classified according to 2004 World Health Organization classification. In each case, the age of the patient, tumor size and the location of the tumor in the testis were noted.Histopathological parameters of the cases were re-examined. Cystic changes and edema in the tumor, interstitial development pattern in the tumor periphery, germ cell neoplasia in situ (GCNIS), development of intratubular tumor, spermatogenesis and Leydig cell hyperplasia in the normal testicular tissue adjacent to the tumor, evidence of invasion in the capsule, spermatic cord, epididymis, rete testis, vein and nerve were investigated.PAS and PAS diastase dyes were applied to evaluate the glycogen content of the tumor in all cases. In addition, PAS staining was used to investigate the presence of ITGCNU in sections containing non-tumoral testicular tissue, and diagnostic role of PAS was evaluated.

Results

When the 10-year archive of our department was retrospectively analyzed, 73.5% (n = 25) of 34 cases with seminoma were evaluated as seminoma, including syncytiotrophoblastic cell variant of seminoma (23.5%:n = 8), and spermatocytic seminoma 3% (n = 1). The median age of the patients was 40.1 (16-72) years. Age distribution of the patients is shown in Table 1.Clinical information of only 20 patients was accessible, and 1 patient had been followed up for infertility. Two patients had hydrocele in the ipsilateral testis. None of the patients had a history of cryptorchidism.The tumor was located on the right in 22, and left testis in 12 patients. The median tumor size was 6.4 cm (1.1-15 cm).In 6 cases, the tumor completely infiltrated the testis and any macroscopic healthy testicular tissue was not observed. All of these cases were seminoma type and the median tumor size was 7.5 cm. Histopathological parameters according to histological types of the tumors and their incidence rates are shown in Tables 2,3,4 and 5.In only 2 out of 8 cases evaluated as syncytiotrophoblastic cell variants, large numbers of syncytiotrophoblastic giant cells constituted groups. In the other 6 cases, small number of giant cells were scattered within the tumor. Syncytiotrophoblastic giant cells were adjacent to the blood vessels and areas of bleeding. (Figure 1)Lymphocytic infiltration in testicular tumors was graded as 1+ in 9, 2+ in 16, and 3+ in 8 cases. In the case with spermatocytic seminoma, lymphocytic infiltration

was rarely observed in focal areas and was considered as (-).Lymphoid follicles were observed in 18 cases with seminoma, and 5 of them were variants of seminomas containing syncytiotrophoblastic giant cells. Lymphoid follicles were not seen in the case with spermatocytic seminoma.Necrosis was observed in all but 6 cases. Necrosis was evaluated as Grade 1+ in 6, Grade 2+ in 6, and Grade 3+ in 18 cases. Necrosis was not observed in a case of spermatocytic seminoma. The median size of the tumor in cases with diffuse necrosis (Grade 3+) was 6 cm and those with Grade 1+ necrosis the median tumor size was 5.8 cm. An inverse relationship between density of necrosis and lymphocytic infiltration was detected. (Table 6).Mitosis was counted by reviewing 50 large magnification areas in all cases. Average number of mitoses per HPM (10X) in cases with seminoma (n=7: range, 1-30), its syncytiotrophoblastic giant cell variant (n=5; range 1-30), and spermatocytic seminoma (n=4) were calculated as indicated. In only 2 of 10 cases average number of mitoses (n=30) were significantly higher than the number of mitosis in all cases.Minimal edema and cystic changes were detected in 3 cases with seminoma. In spermatocytic seminoma, as a prominent feature edema was observed.The interstitial pattern of development is described when normal spermatogenesis is seen in the tumor periphery or intact seminiferous tubuli containing only Sertoli cells are surrounded by the cells of seminoma. Interstitial development pattern was observed in 17 (68%) of 25 cases in which the intact testis was observed in the periphery of the tumor. These cases consisted of seminomas (n=14), syncytiotrophoblastic cell variant of seminoma (n=2), and spermatocytic seminoma (n=1).In our study, in samples obtained from the area adjacent to the tumor in 13 cases, findings consistent with germ cell neoplasia in situ (GCNIS) were observed in seminiferous tubuli (Figure 2). However, since the tumor completely infiltrated the testis in 6 cases, and in 3 cases sampling could not be performed from the normal testicular tissue, the presence of germ cell neoplasia in situ was investigated in only 25 cases (24 seminomas, 1 spermatocytic seminoma). GCNIS was seen in only 13 cases with seminoma, while GCNIS was not seen in case of spermatocytic seminoma.Intratubular development pattern where dilated seminiferous tubuli in tumoral tissue, and its periphery were completely filled with tumor cells was observed in 6 cases which consisted of 5 cases with seminomas, and one case of spermatocytic seminoma.Granulomatous structures consisting of groups of histiocytes were observed in a total of 11 (32%) patients, and categorized based of histiocytic density as Grades 3+ (n=4), 2+ (n=1), 1 + (n=6) in respective number of cases. Among them 8 cases of seminoma, and 3 cases of trophoblastic cell variant of the seminoma were detected. Few foreign body type giant cells associated with granulomas were remarkable.Spermatogenetic activity was observed in the samples obtained from non-tumoral testicular tissue in 11 (44%) of 25 patients having intact testis tissue. Of these 11 cases 9 of them had seminomas, and 2 had syncytiotrophoblastic cell variant of seminoma.In other cases, seminiferous tubules were completely hyalinized or contained only-Sertoli cells.Capsular invasion was observed in 21 (61.7%) cases. Cases with completely devoid of capsule or those with capsules infiltrated partially with tumor cells were included in this group. Average size of tumors In the group consisting of 14 cases with seminomas with capsule invasion, 6 cases with syncytiotrophoblastic cell variant of seminoma, and one case with spermatocytic seminoma median tumor size was 6.7 cm . Paratesticular soft tissue was infiltrated by tumor in one of these cases.In one of the cases (3%),

tumor thrombus was observed in a vessel in the sample obtained from surgical margin of the spermatic cord. In two cases, tumor was not observed at the surgical margin, but tumor was encountered at other levels of spermatic cord.Epididymal invasion was detected in 18 (53%) cases including 14 cases with seminoma and 4 cases with syncytioprophoblastic cell variant of seminoma with an overall average size of 6.8 cm. In the cases in which the epididymis was invaded but the rete testis was not observed, the rete testis was considered as infitrated with a tumor. In 4 cases, rete testis involvement not accompanied by epididymal invasion was observed. In two cases, pagetoid spread was observed under the epithelium of rete testis. Vascular invasion was detected in 30 (88.2%) cases, including 22 cases with seminoma, 7 cases with syncytioprophoblastic cell variant of seminoma and the remaining case was spermatocytic seminoma. Nerve invasion was associated with vascular invasion in 5 cases.Nuclear pleomorphism was evaluated as Grade 1+ in 1 (3%), Grade 2+ in 27 (79.4%) and 3+ in 6 (17.6%) patients. Nuclear pleomorphism was prominent (3+) in indicated number of cases with seminoma (n=4) , syncytioprophoblastic cell variant of seminoma (n=1), and spermatocytic seminoma (n=1) (Figure 3,4). In 2 cases where nuclear pleomorphism was prominent, very high number of mitosis were detected. Pleomorphism was evaluated as 2+ in 84% of the cases with seminoma.The sections of all specimens obtained from tumors and nontumoral testis tissue were histochemically stained with . PAS (Periodic-Acid Schift), Diffuse, and granular PAS positivities were detected in cells.. Based on the evaluation of the intensity of staining were of Grades 1+ (n=11), 2 + (n=10) and 3+ (n=12) (Figure 5). Cytoplasm of tumor cells in a case of spermatocytic seminoma was not stained with PAS dye..In seminiferous tubuli where GCNIS was observed, cytoplasms of the cells with large hyperchromatic nuclei were not stained with H & E , however when stained with PAS dye they demonstrated diffuse, or granular eosinophilic appearance. . In these tubuli, PAS staining was not observed in the Sertoli cells displaced towards the lumen, and in normal germ cells in adjacent tubuli (Figure 6).

Conclusion

Atypical germinal epithelium, detected in intact seminiferous tubules in tissue samples adjacent to the tumor in the testis removed due to the germ cell tumor has been considered to be the precursor lesion of the tumor for many years. It was first described by Skakkebaek in 1972. Instead of the terms as testicular intraepithelial neoplasia, carcinoma in situ or intratubular germ cell neoplasia type, which describe the same histological structure, the term germ cell GHNIS is now used and it has been associated with all germ cell tumors except spermatocytic seminoma [11,12].Its frequent occurrence in contralateral testis patients with cryptorchidism and infertility in whom incidence rates of testicular tumors are higher relative to normal population, and in patients with testicular tumors such supports this assertion [13]. In our study, the areas of GCHIS in tissue adjacent to the tumor were investigated in cases with testicular seminoma and the diagnostic usefulness of PAS stain, which is replaced by PLAP immunohistochemical staining method , was reviewed. In addition, histological types of seminoma were compared in the light of histopathological parametersAs is reported seminoma which is the most common testicular germ cell tumor is seen in patients 40 years of age . However spermatocytic seminoma is seen at an average age of 50-60 years [14]. The median age of the patients in our study was found to be 40.1 years, and the patient with spermatocytic seminoma was diagnosed when he was 30

years old.It has been stated that in seminomas the right testis is affected slightly more frequently than the left at a ratio of 5: 4 (15). This difference was quite evident in our study. (1.9: 1)Any significant difference between the types of seminoma in terms of tumor size was not cited in the literature. Eble et al., indicated that though the tumor size is quite variable in cases with spermatocytic seminoma, usually tumor reaches large dimensions [16]. In our study, the tumor of spermatocytic seminoma type, constituted the largest tumor among our cases with a size of 15 cm,Syncytiotrophoblastic cells are present in 10-20% of seminomas [17]. In our study, these cells were found in 24% of the cases with seminoma.While spermatocytic seminoma has been accepted as a variant of the seminoma in recent years, it is now defined as a clinicopathological entity different from seminoma and other germ cell tumors with its morphological and clinical features [16]. This tumor, first described by Masson in 1946, accounts for 1-2% of testicular germ cell tumors. It is seen 20 times less frequently than seminoma [14,18]. When we retrospectively analyzed the 10-year archive in our department, we found 1 spermatocytic seminoma versus 33 seminomas.The characteristic histological appearance of the seminomas consists of cell layers separated by fibrous trabeculae with different amounts of lymphocytic infiltrates. Fibrous tissue bands can be very prominent or may separate tumor nodules in the form of thin connective tissue fibers [19].In seminomas lymphocytic infiltration of medium intensity (Grade 2+) was more frequently encountered. Lymphoid follicles were found to be associated with lymphocytic infiltration in 53% of the cases.. Lymphocytic infiltration is not seen or observed very rarely in spermatocytic seminomas [16]. In our case with spermatocytic seminoma, it was noted that the lymphocytes were very small in quantity, and the tumor was divided into nodules with thick hyalinized connective tissue trabeculae.In the seminoma, cells are characteristically, uniform, large, polyhedral or round shaped with distinct cell borders. The cells have a clear or granular cytoplasm with a centrally localized hyperchromatic nucleus [15]. Occasional seminomas show areas with dense cytoplasm, indistinct cytoplasmic boundaries, few nucleated cells and prominent cellular atypia [9]. The cellular properties in the spermatocytic seminoma are very different from the seminoma.A population of polymorphic cells consisting of small cells with hyperchromatic nuclei, medium-sized cells and giant cells that are often multinucleated are characteristic features of spermatocytic seminoma. In cases with spermatocytic seminoma, the absence of GCNIS pattern in the seminiferous tubules in the surrounding tissue suggests that the cell origin is quiet different from the seminoma [16]. In our case of spermatocytic seminoma, a population of polymorphic cells with three cell types was observed and dominancy of medium-sized cells was noticed.In our study, cell pleomorphism was evaluated in the seminomas, and in the majority of cases (79.4%), moderate pleomorphism (2+) was noted. The intensity of PAS positivity in seminoma cells was not associated with pleomorphism.The mean number of mitosis was significantly higher in 2 of the 5 seminoma cases in which nuclear pleomorphism was evident. In the past, anaplastic seminoma classification was performed for seminomas with increased number of mitosis and nuclear pleomorphism. However, today, the prognosis of these patients has been proven to be no different from those with seminomas with decreased number of mitosis and the term anaplastic seminoma has been abandoned [20]. Tickoo et al. [21] described the term “atypical seminoma” based on nuclear pleomorphism, crowding and decreased lymphocytic infiltration. These are usually

advanced stage tumors.The amount of necrosis in seminomas varies considerably. Necrosis can be limited to small cell groups or can be seen in the form of large coagulative necrosis. In our study, 3% of the large areas of necrosis evaluated as Grade 3+ was detected in 47% of the cases. The average tumor size (7 cm) in cases with diffuse necrosis was found to be greater than the average tumor size (5.8 cm) in patients with small areas of necrosis. Although spermatocytic seminoma was the largest tumor in all cases, limited necrotic changes were observed in small cell groups only in some areas.Edema can be seen in seminomas. Edematous stroma is very prominent in the spermatocytic seminoma, and on large areas, solid layers are replaced by pseudoglandular patterns and cell nests [19, 22]. In our study, although edema and cystic changes were detected as a rare feature, in spermatocytic seminoma it strikes us as a prominent characteristic..Interstitial development pattern is a prevalently detected finding of seminomas [19]. In our study, this pattern was found in 47% of the samples, and it was especially determined in the periphery of the tumor. Intact seminiferous tubules between tumor cells usually contain only Sertoli cells.Capsular (tunica albuginea) and epididymal invasion have been reported in 8-10% of cases with seminoma in various publications [15,22]. In our study, capsular, and epididymal invasion was detected in 61.7% and 53% of the samples, respectively. The high rates of invasion were attributed to the late-term referral of the patients and their advanced disease stage. Vascular invasion was detected in 88.2% of the cases, which was not associated with types of seminoma and tumor size.The incidence of GCNIS accompanying testicular germ cell tumors ranges between 10 tand 72% in different series. [1,23] Usually patchy areas of GCNIS are found in the testis. The surrounding seminiferous tubules may have a normal appearance. Even spermatogenesis can be seen [19,22]. Diagnosis of GCNIS is facilitated by the application of special dyes. Atypical germ cells with clear cytoplasma contain glycogen. In all cases PAS diastase material is found. However, since glycogen can be found in nonneoplastic spermatogonia and Sertoli cells, it has been suggested that this finding is nonspecific and immunohistochemical staining with PLAP would yield more useful results [19]. However, in a study by Osamu et al., atypical germ cells that were missed in H & E sections in 2 of 59 patients, were stained with PAS and PLAP dyes, and it was indicated that normal germ cells did not contain moderate or large amounts of medium- or large-sized coarse granular glycogen [24].In our study, GCNIS was observed in 13 (25%) of 25 patients whose nontumoral testicular tissue was sampled. This lower rate in comparison to literature was thought to be related to inadequate sampling in our study. Significant thickening of the lesioned basal membranes of seminiferous tubuli was noted, while some tubules were completely hyalinized and contained several atypical cells in their lumens. In 7 patients with ITGCNU, the surrounding seminiferous tubules showed varying degrees of atrophy and hyalinization. In 2 cases, the pagetoid spread of atypical germ cells into rete testis was observed.In all cases with presumptive GCNIS based on the evaluation of H & E stained slides, diagnosis was supported by PAS staining. The large cytoplasm of the atypical germ cells was negatively stained with H & E. However staining with PAS revealed the presence of densely diffused and granular cytoplasm. Since Sertoli cells and spermatogonias were not stained, they were easily diagnosed. When PAS diastase stain was applied on the same sections, staining disappeared. In contrast to that reported in some publications, PAS stain was specific and useful in the diagnosis of GCNIS when

confirmed with H & E stained tissue sections.The relationship between infertility and testicular germ cell tumor is known. In several studies, GCNIS was encountered in testicular biopsies of 0.39-1% of infertile men. Bilateral testicular biopsy was performed in 17 infertile men with GCNIS, and bilateral involvement was seen in 3-5% of all patients. Tumor in the contralateral testis has been detected in 3-5% of the patients with germ-cell testicular tumors [25].In pathology reports, the determination of normal spermatogenesis in non-tumoral testicular tissue may suggest a relatively reduced risk for tumor development in contralateral testis. The detection of mature sperm in the epididymis has been reported to be an easy way to support the presence of spermatogenesis in the testis [26]. In our study, spermatogenetic activity was observed in 11 (32%) cases. However, in all of these cases, the thickening pattern of the basal membrane of seminiferous tubuli was observed at different rates. In 9 of the remaining 23 cases, seminiferous tubuli were completely hyalinized. The findings were thought to be possibly related to the compression effect of the tumor.

References

1. Khan O, Proheroe A. Testis cancer. Postgrad Med J 2007; 83: 624-632.
2. Bahrami A, Ro JY, Ayata AG. An Overview of testicular germ cell tumours. Archives of Pathology&Laboratory Medicine 2007; 131(8): 1267-1280.
3. Bergström R, Adami HO, Möhner M, Zatonski W, Storm H, Ekbom A et al. Increase in testicular cancer incidence in six European countries: a birth cohort phenomenon. J Natl Cancer Inst 1996; 88: 727-733.
4. Bozkurt KK, Başpınar Ş, Akdeniz R, Bircan S, Koşar A. Testis tümörleri: 5 yıllık olgu serisi. Med J SDU 2014; 21(3): 88-92.
5. Lanson Y. Epidemiology of testicular cancers. Prog Clin Biol Res 1985; 203: 155-159.
6. Coffin CM, Ewing s, Dehner LP. Frequecy of intratubuler germ cell neoplasia with invasive testicular germ cell tumours. Arch Pathol Lb Med 1985; 109(6): 555-559.
7. Yörükoğlu K. Testis tümörlerinde prognozu belirleyen histopatolojik parametreler. Üroonkoloji Bülteni 2011; 3: 91-94.
8. Bredael JJ, Vugrin D, Whitmore WF. Autopsy findings in 154 patients with germ cell tumors of the testis. Cancer 1982; 50: 548-551.
9. Ulbright TM, Emerson RE. Neoplasm of the testis. In: Bostwick DG, Cheng L, editors. Urologic Surgical Pathology. 2nd Ed. Elsevier; 2008.p.758-861.
10. Woodward PJ, Heidenreich A, Looijenga LHJ, Oosterhuis JW, McLeod DG, Moller H et al. Germ cell tumours. In:Eble JN, Sauter g, Epstein JI, Sesterhenn IA, editors. Pathology&Genetics Tumours of the Urinary System and Male Genital Organs . Lyon IARCPress;2004.p.218-249.
11. Skakkebaek NE. Possible carcinoma in situ of the testis. Lancet 1972; 2: 516-7.v
12. In Brierley JD, Gospodarowicz MK, Wittekind C. The TNM classification of malignant tumours, 8th ed. Oxford: Wiley Blackwell, 2017.
13. Dieckmann KP,Skakkebaek NE. Carcinoma in situ of the testis: review of biological and clinical features. Int J Cancer 1999; 83: 815-822.
14. Jacobsen GK, Barlebo H, Olsen J,et al. Testicular germ cell tumours in Denmark 1976-1980. Pathology of 1058 consecutive cases. Acta Radiol Oncol 1984; 23: 239-247.
15. Mostofi FK, Price EB; Tumors of testis. In: Atlas of Tumor Pathology, Tumors of the Male Genital

System. Second series, Fascicle eight, Armed Forces Instıtude of Pathology 1973: 21-39.

16. Eble JN: Spermatocytic seminoma. Human Pathology 1994; 25(10); 1035-42.
17. Von Hochstetter AR, Sigg C, Saremaslani P, et al. The significance of giant cells in human testicular seminomas. A clinicopathological study. Virchows Arch (A)1985; 407: 309-322.
18. Talerman A. Spermatocytic seminoma: clinicopathological study of 22 cases. Cancer 1980; 45 : 2169-2176.
19. Ulbright TM: Germ cell neoplasm of testis: The American Journal of Surgical Pathology 1993; 17(1): 1075-1091.
20. Suzuki T, Sasano H, Aoki H, et al. Immunohistochemical comparison between anaplastic seminoma and atipycal seminoma. Acta Pathol Jpn 1993 ; 43: 751-757.
21. Tickoo SK, Hutchinson B, Bacik J, et al. Testicular seminoma: a clinicopathologic an immunohistochemical study of 105 cases with special reference to seminomas with atypical features. Int J Surg Pathol 2002 ; 20: 23-32.
22. Ulbright TM, Roth LM: Testicular ve paratesticular neoplasm. In: Sternberg SS, Antonioli DA, Carter d, Mills SE eds. Diagnosis Surgical Pathology. Second edition, New York, Roven Press. 1994: 1886-99.
23. Che M, Tamboli P, Ro JY, et al. Bilateral testicular germ cell tumors twenty-year experience at M.D Anderson Cancer Center. Cancer 2002;95: 1228-1233.
24. Osamu K, Shigetoshı I, Kensuke B, Hısamı I: Identification of testicular atypical germ cells by an immunohistochemical technique for Plasental Alkaline Phosphatase. Cancer 198; 60: 1325-30.
25. Klein FA, Melamed MR, Whitmore WF: Intratubuler malign germ cells (carcinoma in situ) accompanying invaziv testicular germ cell tumors. Journal of Urology 1985; 133. 413-5.
26. Brodsky GL: Pathology of testicular germ cell tumors. Hematol-Oncol-Clin-North- Am 1991; 5(6): 1095-126.

List of Tables and Figures

Table 1. Distribution of the ages of cases according to decades

Age	*n*	%
10-19	1	2.9%
20-29	5	14.7%
30-39	15	44.1%
40-49	7	20.6%
50-59	2	5.9%
60-69	2	5.9%
70-79	2	5.9%

Table 2. Histopathological parametres, and their incidence rates in seminomas -1

Tumor type	Median size	Lymphocytic infiltration			Necrosis				Cystic change	Interstisiel pattern
		1+	2+	3+	1+	2+	3+	yok		
Seminoma n=25	10.5 cm	6 (24%)	12 (%42)	6 (24%)	5 (20%)	5 (20%)	11 (44%)	4 (16%)	2 (%8)	13 (52%)
Seminoma SSTC variant n=8	7.2 cm	3 (37.5%)	4 (50%)	1 (12.5%)	1(12.5%	1 (12.5%)	5(62.5%)	1 (12.5%)	1 (12.5%)	2 (25%)

Table 3 Histopathological parametres and their incidence rates in seminomas -2

Tumor type	Granuloma	Capsular invasion	Spermatic cord surgical margin	Epidymal invasion	Invasion of rete testis	Vascular iinvasion	Neural invasionu	Pleomorphism		
								1+	2+	3+
Seminom n=25	9(36%)	14(56%)	1(4%)	14	17	22	4	1	20	4
Seminoma SSTC variant n=8	2(25%)	6(75%)	-	4	6	7	1	-	7	1

Table 4. Histopathological parametres in spermatocytic seminomas - 1

Tumor type	Size	Lymphocytic infiltration	Necrosis	Cystic changes	Interstitial pattern	GCNIS
Spermatocytic seminoma n=1	15 cm	–	–	+++	+	–

Table 5. Histopathological parametres in spermatocytic seminomas, and their incidence rates - 2

Tumor type	Capsular invasion	Spermatic cord surgical margin	epididymal invasion	İnvasion of rete testis	Vascular invasion	Neural invasion	pleomorphism
Spermatocytic seminoma n=1	+	–	–	–	+	–	+++

Table 6 Relationship between tumor necrosisdensity and lymphocytic infiltration.

		lymphocytic infiltration		
		1+	2+	3+
NECROSIS	1+	1	2	3
	2+	-	4	2
	3+	6	7	3

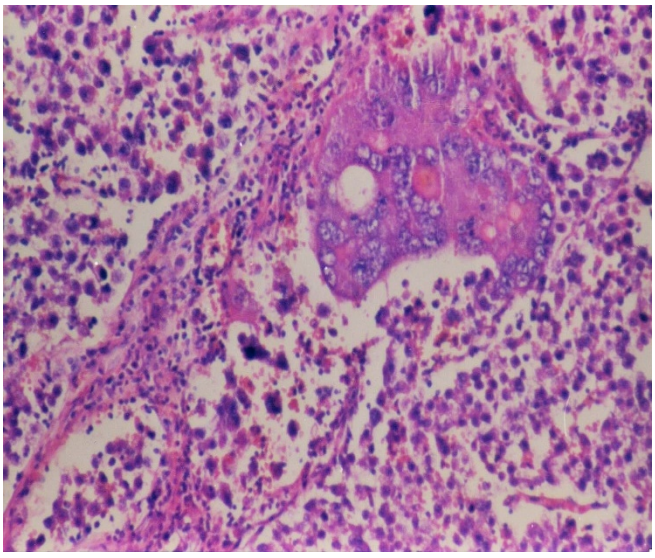

Figure 1. Syncytiotrophoblastic giant cell H&E 150X

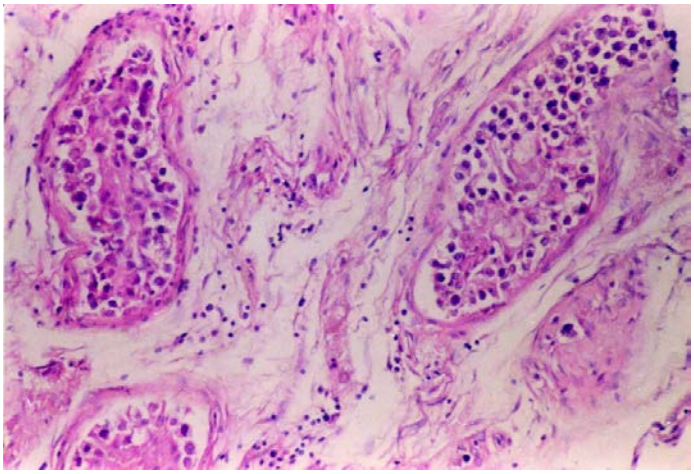

Figure 2. Germ-cell neoplasia in situ H&E 150X

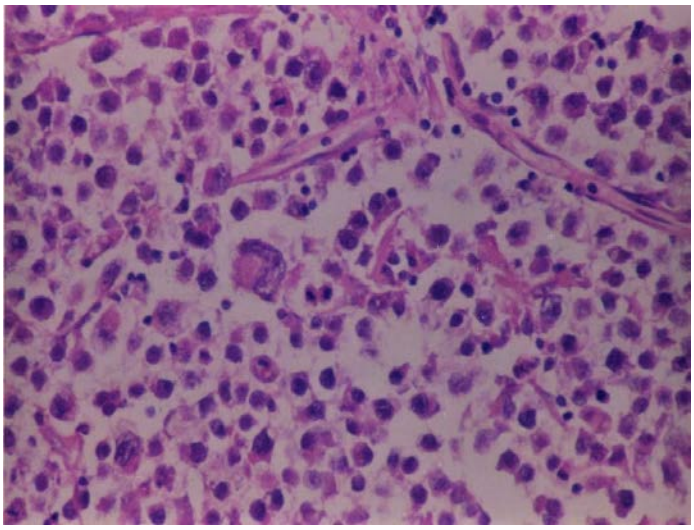

Figure 3. Atypical mitosis in seminoma H&E 300X

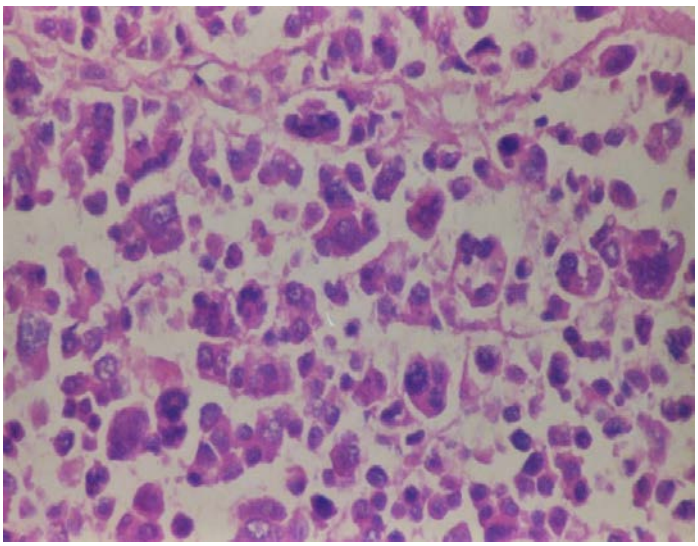

Figure 4. Cellular pleomorphism in spermatocytic seminoma H&E 300X

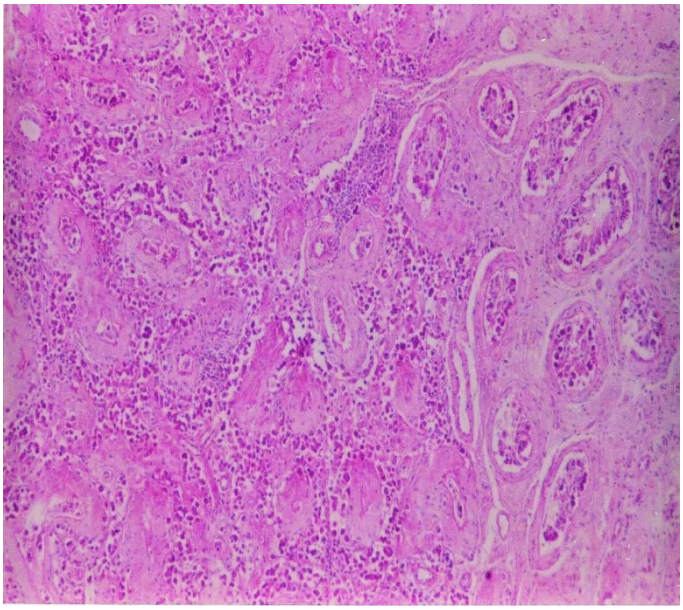

Figure 5. PAS positivity in tumor cells 300X

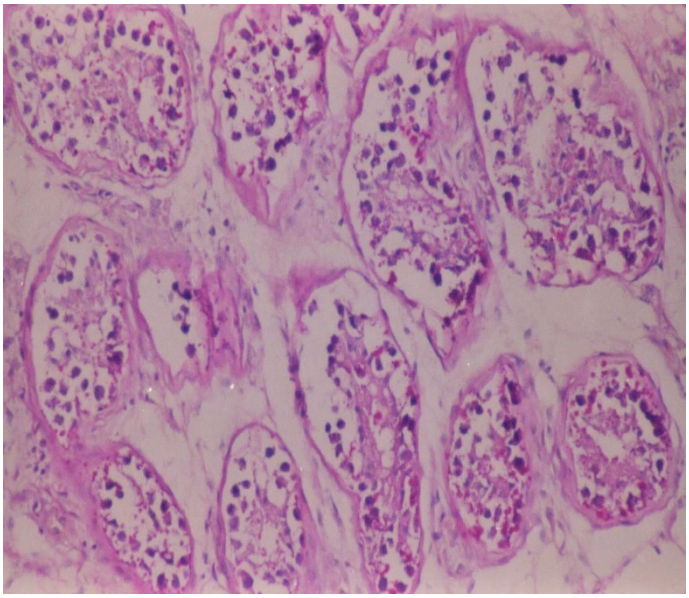

Figure 6. PAS positivity in seminoma and GCNIS 300X