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TESTICULAR GERMINAL TUMOURS IN ADULTS (ABOUT 08 CASES)

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ABSTRACT

The tumors of the testis are rare tumors, accounting for approximately 0.5-2% of men malignancies, of which germinal tumors account for more than 90%. Despite their low incidence, testicular germinal tumors take its importance from the youngage of patients and its high cure rate. **Materials and methods:** Regarding 8 cases of germinal testicular tumors, we conducted a retrospective study over the period of 8-years from January 2012 to January 2020, within the oncology department of the Moulay Ismail military hospital in Meknes. The data were collected on pre-established operating sheets, using the department's register andthe medical record for each patient. **Results:** The analysis of these 8 patients revealed the following results. The annual frequency was 1 new case per year. The average age of our patients was 33 years, with a range from 29 to 39 years. Only one patient had an antecedent of cryptorchidism, and the notion of a familial antecedent of testicular tumor was reported only in one patient. The most frequent clinical symptom is the large bursa, observed in 75% of patients. A positive diagnosis was made by orchiectomy in 100% of cases. NSGCT was the most common histological type with 62.5%. Our patients were classified as stage Iin 50% of cases, stage II in 0% of cases, stage III in 50% of cases. According to the IGCCG prognostic classification, 75% had a good prognosis, 25% had an intermediate prognosis and no cases had a poor prognosis. Only one patient did not receive chemotherapy; the BEP protocol was the most commonly used first-line regimen in 62.5% of our patients. After a median follow-up of 4 years, a PFS rate and OS rate of 87.5% each, all histologies combined, was noted. In seminomas, PFS and OS were 66.7% each. In NSGCT, PFS and OS were 100% each. **Conclusion:** Based on this study and the review of the literature, we can conclude that germinal testicular tumors remain rare tumors and that the epidemiological and clinical tables were close to those in the literature. In general, our study confirmed the good prognosis of GCT.

INTRODUCTION

Testicular tumours are rare (0.5 to 2% of malignant tumours in men of all ages).[1] It is essentially a disease of young men. Testicular germ cell tumours (GCTs) include pure seminomatous germ cell tumours (VSGCs) and non-seminomatous germ cell tumours (NSGCs). TGS account for around 60% of TGs. These are pure seminomas with no other tumour component.

TGNSs have varying percentages of different tumour components, including:

- Embryonal Carcinoma
- Teratoma
- Yolk Sac Tumour
- Choriocarcinoma.

However, the age of onset varies according to the histological type; it is earlier for TGNS (third decade) than for TGS (fourth decade).[1]

Incidence has risen sharply over the last 25 years, with remarkable geographical variation across the world.[2]

All testicular tumours are cancer until proven otherwise. The diagnosis of germ cell tumours of the testis is based on clinical findings, combined with a work-up including the measurement of tumour markers (AFP, total hCG, etc.). LDH and imaging (scrotal ultrasound and CT scan thoracoabdomino-pelvic).

In terms of treatment, orchiectomy and anatomopathological study of the operative lap allow a definitive diagnosis to be made.

Testicular germ cell tumours have an excellent prognosis, provided adequate treatment is given. The cure rate, for all stages combined, currently exceeds 90%.

This work is a retrospective study based on the analysis of 8 cases of testicular germ cell tumours in the oncology department of the Moulay Ismail military hospital in Meknes, over an 8-year period from 2012 to 2020, with the aim of comparing the epidemiological, clinical, paraclinical, therapeutic and prognostic data with those

in the literature.

MATERIALS AND METHODS

Our study took place in the oncology department of the Moulay Ismail military hospital in Meknes.

1. Type of study

Our study was retrospective.

2. Study period

Our study covered an 8-year period from January 2012 to January 2020.

3. Data collection procedures

The data was collected on pre-established data sheets, using the department's register and the medical file for each patient.

Variables studied

The following variables were collected from all participants

- Clinical data: age, history, infertility, duration of evolution, circumstances of discovery, general condition, physical signs, etc.
- Biological data: tumour markers (HCG, AFP, LDH) .
- Radiological data: testicular ultrasound, C A T scan, cerebral CAT scan.
- Data relating to the tumour: histological type, distant extension, stage, prognosis, TNM classification.
- Treatment: surgery, chemotherapy, radiotherapy.
- Follow-up and progress: Response, survival rate.

4. AIM OF STUDY

The aim of this study is to attempt to compare the various epidemiological, clinical, paraclinical, therapeutic and prognostic data from this series of patients with those in the literature.

RESULTS

I. Epidemiological profile

1. Frequency

Over the 8-year period from January 2012 to January 2020, 8 cases of testicular germ cell tumours were recorded.

Their annual frequency was 1 new case per year.

2. Age

The average age of our patients is 33, with extremes of 29 and 39.

The age for seminomas is 34.6 years (29-39 years), and for non- seminomatous germ cell tumours (NSGCTs) 32 years (29-36).

Table 1: Age by histological type.

HISTOLOGICAL TYPE	AVERAGE AGE	EXTREME
Seminoma	34,6	29-39
TGNS	32.4	29-36

The breakdown by 10-year age group showed a peak of 75% between the ages of 30 and 39.

Table 2: Breakdown by 10-year age group.

AGE	NUMBER OF CASES	%
20 - 29 years old	2	25%
30 - 39 years old	6	75%
>40 years	0	0%

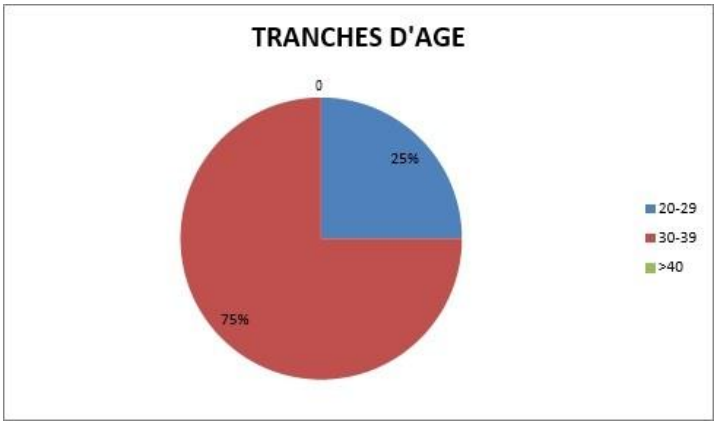

Figure 1: Breakdown by 10-year age group.

3. The residence

5 patients came from urban areas, and 3 from rural areas, giving approximate frequencies for geographical origin (Graph 2).

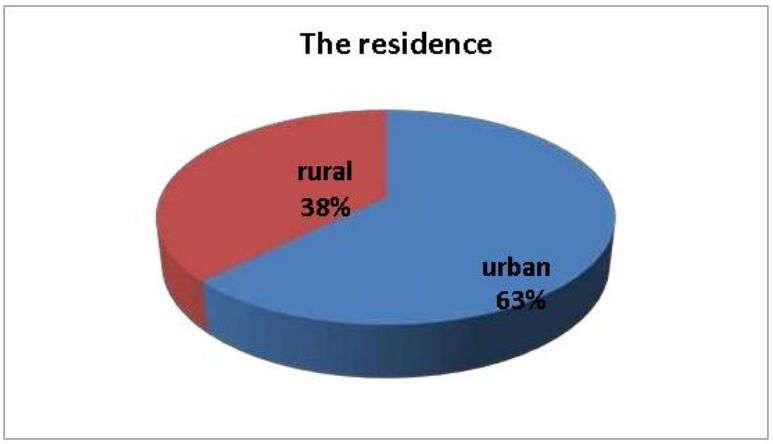

Figure 2: Breakdown by place of residence.

II. Background

1. Employees

Only one patient had a history of cryptorchidism. The rest of the patients had no particular history.

2. Family

A family history of testicular tumour was reported in only one patient.

III. Clinical profile

1. Revealing symptoms

The large bursa was the first sign observed in 6 patients (75%). The intrascrotal mass was the revealing sign in 2 cases, i.e. 25%.

Testicular pain was the revealing sign in only one case, associated with a large bursa, i.e. 12.50%.

2. Clinical examination

a. General condition

In our series of studies, the data on general condition, assessed according to the WHO scale, were as follows:
5 patients had a WHO of 0 3 patients had a WHO of 1 No patient had an OMS of 2 No patient had an OMS of 3 No patient had an OMS of 4.

b. Testicular examination

Our study of the series revealed the following data

- A painful testicle on palpation was noted in only 1 case.
- No patient presented with scrotal invasion.
- The tumour was unilateral in 100% of cases.

c. Examination of lymph nodes

Inguinal adenopathy was present in 2 cases, i.e. 25% of cases.

III. Anatomopathological profile

1. Positive diagnosis

The positive diagnosis was made histologically, on the specimen. orchiectomy in the 8 patients, which was also a therapeutic procedure.

2. Histology

TGNS was the most frequent histological type, found in 5 cases (62.5%), and pure seminomas were found in 3 cases (37.5%).

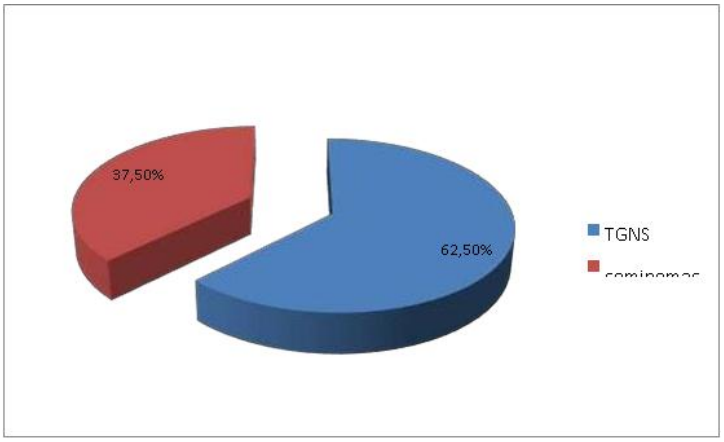

Figure 3: Distribution according to histological type.

Table 3: Breakdown of cases by histological type.

Histological type		Number of case	%
Seminome	Pure	3	37,5%

TGNS	A only component	Carcinoma Embryonic	1	12,5%
	Mixed component		4	50%

IV. Radiological assessment

1. Local

Scrotal ultrasound: Five patients had a scrotal ultrasound examination prior to orchidectomy.

2. Remote

a. Thoracic-abdominal-pelvic computed tomography

CAT scans were carried out as part of the extension w o r k - u p in all patients in our series. It was normal in 4 cases.

It was in favour of metastases in 4 cases

- Visceral metastases in 4 cases: 3 cases of pulmonary metastases, and 1 case of pulmonary and right urethral metastases at the same time.
- Lymph nodes in 2 cases.

b. Cerebral computed tomography

Brain CT scans were performed in 4 cases

- It was normal in all 4 cases.

V. Biological check-up

Tumour markers were performed in 7 patients

- The αFP assay was elevated in 3 cases.
- The βHCG level was elevated in 2cases
- LDH levels were elevated in 2 cases.

Table 4: Tumour markers.

Markers tumor	Number of cases	%
Normal	4	50%
Disturbed	3	37,5%
No facts	1	12,5%

VI. Classification into stages

At the end of the clinical, biological, histological and radiological assessments, we were able to classify our patients according to the AJCC classification.

- 4 patients were classified as stage I (50%)
- No patients classified as stage II (0%)
- 4 patients were classified as stage III (50%).

The most frequent stages in our series were stage I and III, found in 50% of cases each.

Table 5: Breakdown of cases by AJCC stage of disease.

STADE	TGS	TGNS	TOTAL
STAGE I	2(66.7%)	2(40%)	4(50%)
STAGE II	0(0%)	0(0%)	0
STAGE III	1(33.3%)	3(60%)	4(50%)

The most frequent stage of TGS was stage I, with a percentage of 66.7%.

The most frequent stage in cases of TGNS was stage III, with a percentage of 60%.

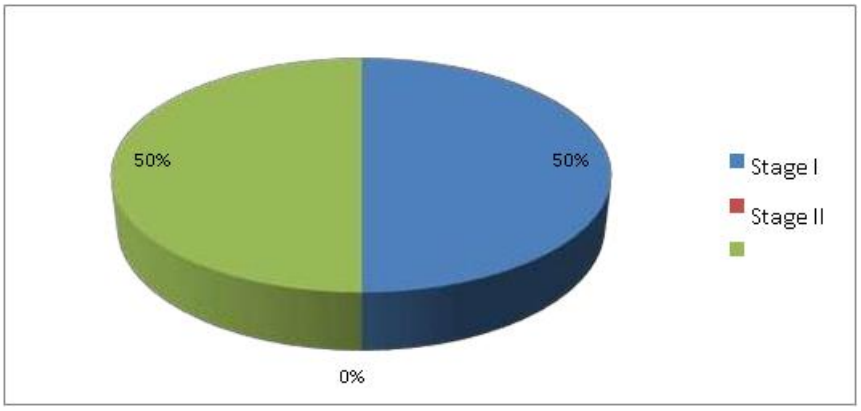

Figure 4: Breakdown of cases according to stage.

VII. Prognostic classification of IGCCCG

The results of the distribution of patients according to the IGCCCG prognostic classification were as follows

- 3 patients had a good prognosis (75%).
- Only 1 patient had an intermediate prognosis (25%).
- No patient had a poor prognosis (0%).

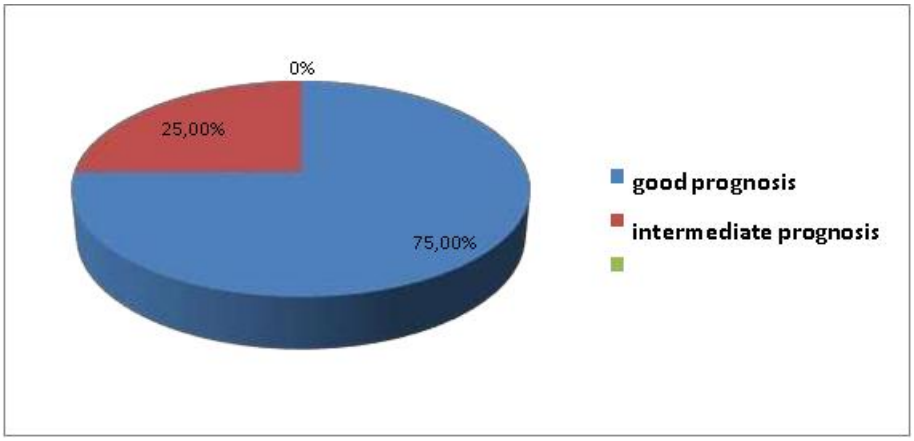

Figure 5: Distribution according to IGCCCG prognostic classification.

In the TGS group: 0% of patients were classified as having a good prognosis, 100% were classified as having an intermediate prognosis. In the TGNS group: 100% of patients were classified as having a good prognosis, 0% were classified as having an intermediate prognosis, and 0% were classified as having a poor prognosis.

Table 11: Distribution of prognostic groups according to histology.

Histology	Good prognosis	Prognosis intermediate	Bad prognosis
TGS	0%	100%	
TGNS	100%	0%	0%

VIII. Therapeutic methods

1. Surgery

All patients in our series underwent orchiectomy via the upper approach.
Lymph node dissection was only mentioned in one case.

2. Chemotherapy

Adjuvant chemotherapy was prescribed for 7 patients in our study

- 2 patients received a single course of carboplatin AUC 7
- Only 1 patient received 2 courses of BEP
- 3 patients received 3 courses of BEP
- Only 1 patient received 1 course of BEP

Toxicities of chemotherapy

In our series, 7 patients underwent sperm cryopreservation.

- 1 case of acute respiratory distress syndrome.
- 3 cases of febrile neutropenia.
- 3 cases of mucositis.
- 2 cases of thrombocytopenia.
- 2 cases of alopecia.
- 2 cases of vomiting.

3. Radiotherapy

N o n e of the patients in our series received radiotherapy for curative or palliative purposes.

Table 7: Prognosis.

Histology	SG at 5 years	5-year SSP
TGS+TGNS	87.5%	87.5%
TGS	66.7%	66.7%
TGNS	100%	100%

IX. Response to treatment

1. Biological response

3 patients had a biological response defined by a reduction in tumour marker levels after chemotherapy compared with pre-operative values.

2. Remission

Complete remission was achieved in 7 patients.

X. Course and prognosis

Our study series is marked by 1 death in a seminoma after 2 courses of BEP, following acute respiratory distress syndrome due to chemotherapy toxicity.

Generally speaking

After a median follow-up of 4 years, a PFS and OS rate of 87.5% was noted for all histologies combined.
In seminomas: PFS and OS were 66.7% each. In TGNS: PFS and OS were 100% each.

DISCUSSION

I. Epidemiology

1. Frequency

Testicular cancer accounts for 1 to 1.5% of all male cancers.[3,4]

The fact that we collected only 8 cases of testicular germ cell tumours over a period of 8 years testifies to the rarity of this tumour, which has also been reported in the literature.

The annual incidence rate was 1 new case per year.

Our results differ from those of G.C.W. Howard et al[5], who found 703 cases of germ cell tumours of the testis in a retrospective study over a period of 14 years from 1988 to 2002, i.e. an annual frequency of 50.2 new cases per year in England.

2. Age

In our series

- The median age of our patients was 33, with extremes of 29 and 39. This is in line with the literature.
- The median age of patients with seminomas was 34.6 years (29 - 39 years), that of patients with non-seminomatous germ cell tumours was 32 years (29 - 36 years), In the literature, peak incidence of non-seminomatous tumours is around age 25, whereas peak incidence of seminoma is around age 35.[6]

In the retrospective study carried out by the Spanish tumour group.

In a study of 1490 patients followed for TGT between January 1994 and April 2001 (J.R Germà et al.), the median age of patients with TGS was 33 years (18 - 81), that of patients with TGNS was 26 years (18 - 74).[7]

In the retrospective study carried out at the Mohamed V Military Hospital in Rabat[8], the median age of patients with TGS was 37 years (30-49), and that of patients with TGNS was 27.6 years (22-34).

Table 8: Correlation between age and histological type.

Histology	J.R Germà et al.[7]	Mohamed V Military Hospital Rabat[8]	Our series
TGS	33 years old (18 - 81)	37 years (30 - 49)	34.6 years (29 - 39 years)
TGNS	Age 26 (18 - 74)	27.6 years (22 - 34)	32 years (29 - 36 years)

The median age of onset of TGS in our series is similar to that in the literature.

Compared to the literature and the studies by J.R Germà et al. and At the Mohamed V Military Hospital in Rabat, the median age at onset of TGNS in our series was 6 years younger.

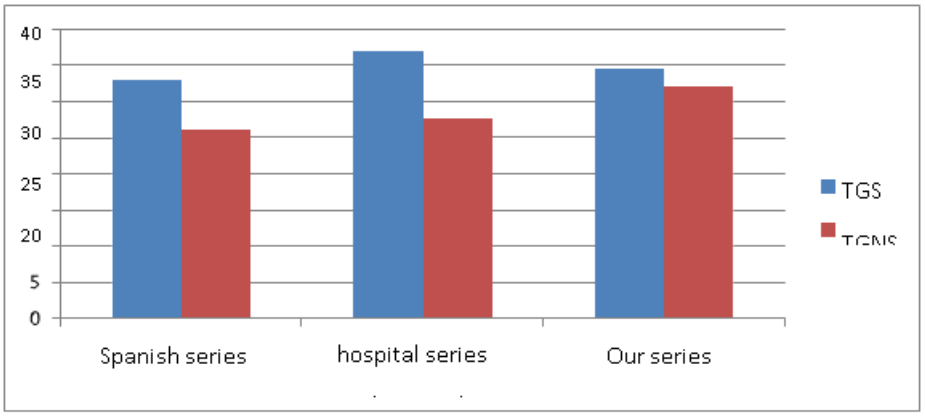

Figure 6: Median age at onset of TGT according to histological type.

3. Ethnic and geographical characteristics

This tumour is four times more common in Caucasians, and is more likely to be found in urban or socially privileged environments in rich, developed countries.[8]

In several studies, the disease is more common in people from higher social classes.[9]

In our study, germ cell testicular tumours were more frequent in urban areas than in rural areas, with a percentage of 62%.

4. Risk factors

A number of urological conditions may be associated with testicular cancer, apart from genetic factors, which seem to be heavily involved.

Recently, studies have suggested the influence of early

factors in an individual's life, during pregnancy or around birth.

These include endogenous and exogenous hormonal exposure in utero[10], low birth weight[11] and the presence of neonatal jaundice.[12]

Klinefelter's syndrome[13] and infertility[14] are frequent associations. - History of cryptorchidism (relative risk 5 to 10 times). In our study, two risk factors were identified for germ cell testicular tumours. These were essentially a history of cryptorchidism and a family history of cancers of the testicular gland testicle Cryptorchidi.

It is the main risk factor associated with the development of testicular cancer[15], and its association with testicular cancer was the subject of a general review by E. Fontaine at the AFU 2002 congress. The relative risk of developing cancer varies[16], ranging from 2.5 to 18%. Some studies have shown that delayed orchidopexy considerably increases the risk of TGT.[120]

In our series, cryptorchidism occurred in 12.5% of cases, which is a result close to that reported by many other authors.[16]

Family history of testicular cancer

A Swedish study of over 4,500 patients found a relative risk of 3.8 in the case of a paternal history and 7.6 in the case of testicular cancer in a brother.[17]

In our series, a family history of testicular tumour was reported in 12.5% of cases.

II. Clinical data

1. Circumstances of discovery

In our study, the most frequent revealing symptom of TGT was the appearance of a large bursa, observed in 6 cases, i.e. 75% of cases.

This result is consistent with that of J.R Germà et al.[7]

I. Anatomopathological data

In the literature, seminomas represent approximately 40% of TGTs and TGNS represent approximately 60% of TGTs.[18]

These results are similar to those of our series, where TGNS represented the majority histology with 62.5% and TGS 37.5% of TGT.

These results are identical to those reported by J.R Germa et al (Spanish series)[7], and different to those reported by G.C.W. Howard et al (English series)[8] and other English, American and Canadian series.[19,20,21]

Table 9: Distribution according to histological type (comparison with our series).

Histology	Our series N=8	English series N=703	Spanish series N=1490
TGS	3 (37.5%)	446 (63,4%)	533 (35,8%)
TGNS	5 (62.5%)	257 (36,6%)	957 (64,2%)

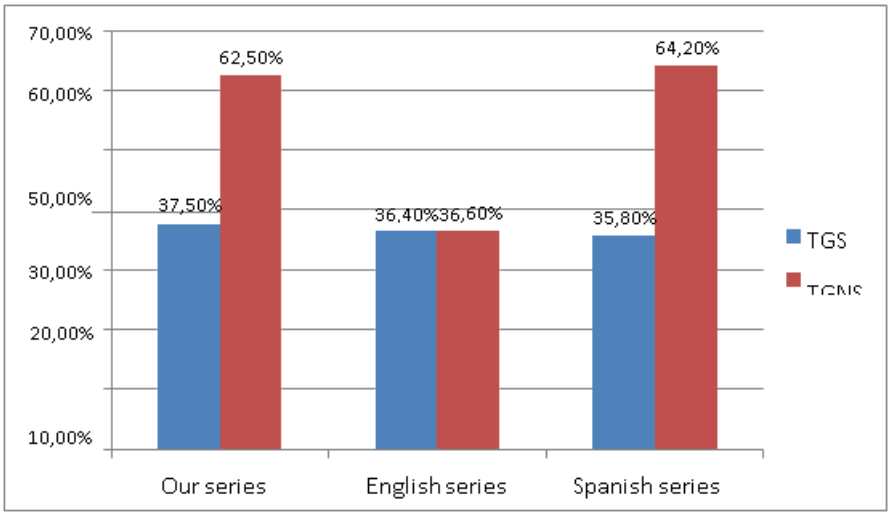

Figure 7: Distribution by histological type (comparison with our series).

IV. Radiological examination

The systematically recommended minimum requirement.[22]

1. Scrotal ultrasound

Ultrasound has a sensitivity of almost 100% for detecting testicular tumours.[20] The tumour appears as a h y p o e c h o i c nodule, more or less homogeneous in relation to the rest of the normal testicular parenchyma, sometimes with cystic components or calcifications.[23]

The main purpose of ultrasound is to determine the intra-testicular location of a palpable tumour and to differentiate cysts from solid tumours.[24]

In our series, 5 patients (62.5%) underwent scrotal ultrasound examination.

2. Thoracic-abdominal-pelvic computed tomography (TAP-CT)

This is the reference examination for lung and liver metastases, and retroperitoneal and mediastinal adenopathies. It will determine their number, size, location and appearance.

Abdominal-pelvic CT has a sensitivity of 70-80% for retroperitoneal lymph node assessment. Its sensitivity and negative predictive value increase when the limit of positivity for lymph node metastatic disease is set at 8 mm.

In our study, the initial and follow-up work-up included an abdominal- pelvic CT scan in all patients.

V. Biological check-up

A marker is very rarely a means of diagnosis or screening. The interest of markers in testicular tumours lies in assessing the response to treatment and detecting relapse.[24] Tumour marker assays should therefore be performed postoperatively.

Three markers must be systematically measured.[25]

1. The αfoeto-protein (αFP)

Its normal value is less than 20 ng/ml.[25]

In adult pathology, it rises in a few liver diseases and in 50 to 70% of non-seminomatous tumours: embryonal carcinoma, teratocarcinoma, vitelline tumour.[26] It is not never secreted by the seminoma.[27]

In our study, αFP was elevated in 3 cases.

2. Chorionic gonadotrophic hormone (HCG)

It is secreted in germ cell tumours when it contains syncitiotrophoblastic cells. High secretion is clinically accompanied by gynaecomastia.[26] HCG (normal is 1 to 1.5 ng/ml depending on the laboratory) is secreted mainly by choriocarcinomas and embryonal carcinomas and by 10 to 15% of seminomas.[25]

In our study, βHCG levels were elevated in 2 cases.

3. Lactic dehydrogenase (L.D.H.)

LDH is increased in 28% of stage I seminomas and in 90% of cases where there are metastases.[28] Its level is proportional to the tumour mass.[26]

In our study, LDH was elevated in2cases Overall, in our study, the marker assay was

- Disturbed in 3 cases, i.e. 37.5%.
- Normal in 4 cases, i.e. 50%.
- Not done in only 1 case, i.e. 12.5%.

VI. Classification

1. In stadiums

The distribution of stages in our series is similar to that reported in the literature[7], with seminomas diagnosed as more frequently in stage I than in advanced stages.

On the other hand, TGNS were diagnosed more frequently in the advanced stages than in the localised stages, whereas they were diagnosed more frequently in stage I than in the advanced stages in certain series, notably those of J.R Germà et al. (Spanish) and G.C.W. Howard et al.

Our series

STADE	TGS	TGNS	TOTAL
STAGE I	2(66.7%)	2(40%)	4(50%)
STAGE II	0(0%)	0(0%)	0
STAGE III	1(33.3%)	3(60%)	4(50%)

Spanish series[7]

STADE	TGS	TGNS	TOTAL
STAGE I	410 (77%)	496 (51,9%)	906 (60,8%)
STAGE II	103 (19,3%)	252 (26,3%)	355 (23,9%)
STAGE III	20 (3,5%)	209 (21,8%)	229 (15,4%)

English series[5]

STADE	TGS	TGNS	TOTAL
STAGE I	386 (86%)	155 (60,3%)	541 (77%)
STAGE II	50 (11,2%)	60 (23,3%)	110 (15,6%)
STAGE III	10 (2,2%)	42 (16,4%)	52 7,4%)

2. Prognostic classification of IGCCCG

The distribution of patients according to the prognostic classification of IGCCCG showed that all cases of seminoma in our series were classified in the intermediate prognosis category (100%): a different result to that of the series by J.R Germà et al[7], and that the proportions of TGNS classified in the good prognosis, intermediate prognosis and poor prognosis categories were 100%, 0% and 0% respectively: a result not very similar to that recorded in the Spanish[7] and English[5] series in which the good prognosis category predominated (table 10).

Table 10: IGCCCG prognostic classification for each series.

	Our series	Spanish series	English series
TGS stage II-III	N= 1	N=123	
Good prognosis	0(0%)	118 (96%)	-
Prognosis intermediate	1(100%)	5 (4%)	-
TGNS stage II-III (and IM+ for the [ ] and [ ] series) old)	N=3	N= 523	N=123
Good prognosis	3(100%)	329 (62,9%)	79 (64,2%)
Prognosis intermediate	0(0%)	98 (18,7%)	26 (21,1%)
Poor prognosis	0(0%)	96 (18,4%)	18 (14,7%)

VII. Therapeutic data

1. Surgery

1.1. High approach orchiectomy

The surgical procedure is governed by strict rules[29]

- Sperm preservation should be offered systematically before orchiectomy for at least one of the samples. The other samples can be taken after orchiectomy before any additional treatment is started.[30] It has medicolegal value.
- The patient is informed of the principles and risks of the operation by the Urologist, who will mention the possibility of implanting a testicular prosthesis during the same operation or at a later date.[29]
- The possibility of adjuvant treatments (radiotherapy or chemotherapy) is systematically raised.[26]
- The approach is the classic kelotomy[30], similar to that of an inguinal cure hernia[29] this is the only approach that allows primary vascular control.[30]

In our series, all our patients underwent inguinal orchiectomy before starting chemotherapy, which is in line with international recommendations.

1.2. Lymph node removal

In testicular tumours, surgery of the retroperitoneum is a major therapeutic step. It can take place at the beginning of the history of the tumour. treatment (staging curage), or after chemotherapy for a residual lesion (residual mass curage).[31]

In our series, only 1 patient underwent lymph node dissection.

2. Chemotherapy

Germ cell tumours are very sensitive to chemotherapy[32] Chemotherapy alone can be curative even in the metastatic stages.

The first combination consisted of vinblastine and bleomycin, soon followed by cisplatin: the PVB protocol resulted in a cure rate of over 50%.[32]

Subsequently, etoposide replaced vinblastine to form the current standard BEP. The study comparing PVB and BEP showed that BEP was less toxic than PVB, just as effective in forms with a good prognosis, and more effective in forms with a poor prognosis in terms of progression-free survival and overall survival, with 78% survival at 2 years compared with 48% for PVB.[33]

In our study, the latest chemotherapy treatment recommendations for TGT were followed
Adjuvant chemotherapy was prescribed for 7 patients in our study
TGS patients

- The 2 patients with stage I seminoma were treated with a single course of carboplatin AUC 7 (monitoring and radiotherapy were not adopted).

A cycle of carboplatin AUC7 is not inferior to radiotherapy (20 Gy, lumbo-aortic), in terms of recurrence rate (5% vs. 4%), time to recurrence and specific survival with a median follow-up of 5 years.[34]

- A patient with stage III seminoma was treated with 2 BEPs.

TGNS patients

- 1 patient with stage I NSTE was treated with 1 BEP. Being classified in the high-risk group by the presence of vascular emboli, the choice between monitoring and chemotherapy was in favour of chemotherapy.

Alexandre[35] has identified 2 prognostic factors i n stage I NSTE: the presence of vascular invasion and the presence of a mature teratoma.

After surveillance alone, they reported a 12-year recurrence rate of 0% when there was no vascular invasion but mature teratoma. This rate was 29% when there was vascular invasion and mature teratoma, or when there was neither. Finally, the rate was 61% when there was vascular invasion but no mature teratoma. For many authors, 'prophylactic' chemotherapy is therefore justified in NST with vascular invasion.

We have opted for the same attitude in our study.

- In the 3 patients with stage III NSGT, all with a good prognosis, the recommended protocol was 3 BEPs.

For the TGNS group with a good prognosis: the aim is to

reduce toxicity.

Various randomised studies have analysed the role of the number of cycles of chemotherapy and bleomycin have concluded that three cycles of the BEP combination is the standard. It is essential that this protocol is adhered to, as any deviation risks compromising its efficacy. Four cycles of EP is an option if bleomycin is contraindicated in a study which showed that 4 EP=3BEP.

Carboplatin is less effective than cisplatin, with 25% failure rates reported in studies, so it should not be used.[36,34]

A total of 87.5% of patients in our series received adjuvant chemotherapy. Despite the presence of lymphonodal and/or pulmonary metastases at the time of diagnosis in 4 patients (50%), this chemotherapy resulted in a long-term recurrence-free survival rate of 85.7%, confirming the good results reported in the literature.[37,38,39,40]

3. Monitoring

In our study, monitoring alone was proposed in only 1 patient with stage I NSGT, given the absence of risk factors. This low representation is due to the fact that early stages are most often monitored by urologists.

Surveillance of stage I TGNS without lymphovascular invasion shows a recurrence rate of only 12%, corresponding to the natural history of low-risk stage I TGNS.[27]

4. Radiotherapy

None of the patients in our series received radiotherapy for curative or palliative purposes.

Table 11: The different treatments received by patients in our series.

Treatment	TGS		TGNS	
	Stage I (n=2)	StageII-III (n=1)	Stage I (n=2)	Stage II-III (N=3)
Monitoring	-	-	1	-
Chemotherapy				
- Carboplatin AUC 7	2	-	-	-
- 1 BEP	-	-	1	-
- 2 BEP	-	1	-	-
- 3 BEP	-	-	-	3
Radiotherapy	-	-	-	-

VIII. Course and prognosis

Our series of studies was marked by
1 death following acute respiratory distress syndrome in a seminoma treated with 2 courses of BEP.

- **Generally speaking**

After a median follow-up of 4 years, a PFS and OS rate of 87.5% was noted for all histologies combined.

- **In seminomas**

SSP and SG were 66.7% each.

- **In TGNS**

SSP and SG were 100% each.

Our series confirms the good prognosis of TGT reported in the literature, with an 87.5% OS rate.

For TGS, our results are inferior to those reported by G.C.W. Howard et al[5] who recorded 97.7% OS.

For TGNS, our results are similar to those reported in the same English study[5], which recorded a 90% OS.

The limited number of patients in our series made it impossible to calculate survival rates for each stage and prognostic group in the two disease groups (TGS and TGNS) in order to compare them with other studies.

IX. Sperm preservation[2]

Sperm abnormalities are common (20%) in patients with testicular cancer. Chemotherapy and radiotherapy further impair fertility in these patients. Sperm cryopreservation is recommended and has medico- legal value. It can be carried out before or after orchiectomy, but before any chemotherapy.

In our series, all our patients were able to benefit from sperm conservation.

CONCLUSION

Testicular tumours are a subject that seems far from having revealed all its secrets. They most often arise from the germ lines of the testicle. They affect young men (aged 15-35) at a time in their lives when they have major family and professional commitments.

There is still a long delay between the discovery of a scrotal swelling and the first consultation. This delay in diagnosis is due to the fact that the patient does not always understand the seriousness of the lesion or does not dare to talk to their doctor about it.

To diagnose a testicular mass, ultrasound is the key first-line examination, complementing the clinical examination. The diagnosis of cancer and the degree of local involvement are determined by surgical removal (inguinal orchiectomy). A thoracoabdominopelvic CT

scan is used to assess the extent of lymph node and visceral involvement.

Paradoxically, TGNS represents the most frequent histological variety in our study. Locoregional extension and the distinction between seminomatous tumours and other germ cell tumours are important criteria for subsequent therapeutic strategy. Seminoma occupies a special place among testicular germ cell tumours because of its radiosensitivity. Although radiotherapy remains the preferred treatment for localised stage I tumours, surveillance and chemotherapy may now, in certain cases, represent a therapeutic alternative. The Chemotherapy is currently the reference treatment for more advanced stages.

Non-seminomatous germ cell tumours are characterised by their high chemosensitivity, which has improved their prognosis, particularly with the use of cisplatin. In some cases, early diagnosis means that treatment can be limited to simple orchiectomy. Chemotherapy for metastatic stages, with the number of courses depending on the prognostic category, is particularly effective. Surgery for residual masses is usually necessary after this treatment.

Finally, the results of this study provide sufficient confirmation of the data in the literature, particularly in terms of a less unfortunate prognosis and high sensitivity to chemotherapy, with a high rate of disease control and prolonged survival, even in the metastatic phase.

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