available at www.sciencedirect.com
journal homepage: www.europeanurology.com

European Association of Urology

EAU Guidelines – Editor's choice

European Association of Urology Guidelines on Testicular Cancer: Summary of the 2026 Guidelines

Anna Patrikidou[a,*], *Walter Cazzaniga*[b], *Christoph Oing*[c], *Daniel Berney*[d], *Joao Lobo*[e,f], *Joost Boormans*[g], *Christian Fankhauser*[h], *Stefanie Fischer*[i], *Ricardo Leao*[j], *Carmen Gravina*[k], *Domenico Di Nardo*[l], *Robert Cornes*[m], *Florian Janisch*[n], *Carla Bezuidenhout*[o], *Hege Sagstuen Haugnes*[p,q], *Alexandros Papachristofilou*[r], *Torgrim Tandstad*[s], *Axel Heidenreich*[t]

[a] *Genitourinary Oncology Group, Department of Medical Oncology and Drug Development Department, Gustave Roussy Cancer Campus, Villejuif, France;* [b] *Department of Urology, The Royal Marsden NHS Foundation Trust, London, UK;* [c] *Translational and Clinical Research Institute, Newcastle University, Newcastle upon Tyne, UK and The Northern Centre for Cancer Care, Freeman Hospital, The Newcastle upon Tyne Hospitals NHS Foundation Trust, Newcastle upon Tyne, UK;* [d] *Centre of Cancer Biomarkers and Biotherapeutics, Barts Cancer Institute, Charterhouse Square, Queen Mary University of London, London, UK;* [e] *Department of Pathology and Cancer Biology and Epigenetics Group-Research Centre, Portuguese Oncology Intituto of Porto (IPO Porto), Porto, Portugal;* [f] *Department of Pathology and Molecular Immunology, Institute of Biomedical Sciencces Abel Salazar, Univeristy of Porto (ICBAS-UP), Porto, Portugal;* [g] *Department of Urology, Erasmus University Medical Centre – Cancer Institute, Rotterdam, the Netherlands;* [h] *Department of Urology, Luzerner Kantonsspital, Luzern, Switzerland;* [i] *Department of Medical Oncology and Hematology; Health Ostschweiz/Cantonal Hospital St. Gallen, St. Gallen, Switzerland;* [j] *CUF Hospitals, iCBR, Faculty of Medicine, University of Coimbra, Coimbra, Portugal;* [k] *Department of Urology, Sant'Andrea Hospital-Sapienza University, Rome, Italy;* [l] *Patient Representative, European Association of Urology Guidelines Office, Arnhem, the Netherlands;* [m] *Orchid Male Cancer, London, UK;* [n] *Department of Urology, University Medical Center Hamburg-Eppendorf, Hamburg, Germany;* [o] *European Association of Urology Guidelines Office, Arnhem, the Netherlands;* [p] *Department of Oncology, University Hospital of North Norway, Tromsoe, Norway;* [q] *Department of Clinical Medicine, UIT-The Arctic Uniersity, Tromsoe, Norway;* [r] *Department of Radiation Oncology, University Hospital Basel, Basel, Switzerland;* [s] *Department of Oncology, The Cancer Clinic, St. Olav's University Hospital, Trondheim, Norway;* [t] *Department of Urology, Uro-Oncology, Robot-Assisted and Specialized Urologic Surgery, University Hospital Cologne, Germany*

Article info

Article history:
Accepted March 20, 2026

***Editor in Chief*:**
Alberto Briganti

Keywords:
EAU guidelines
European Association of Urology
Follow-up
Germ cell tumor

EU ACME
https://mcq.eu-acme.org/login

Please visit https://mcq.eu-acme.org/login to answer questions on-line. The EU-ACME credits will then be attributed automatically.

Abstract

Background and objective: The European Association of Urology (EAU) produces an annual guidelines document based on the most recent evidence for the diagnosis, treatment, and follow-up of testicular cancer (TC). To summarize the 2026 version of the EAU Guidelines on TC and highlight the main changes compared to the previous version.
Methods: A multidisciplinary team of clinicians with specific expertise in the disease (urologists, medical oncologists, radiation oncologists, and pathologists) reviewed the results of a comprehensive appraisal of the published literature on the topic since the last Guidelines update paper in 2023.
Key findings and limitations: Recommendations based on the highest available level of evidence are presented across TC stages, histologies and prognostic categories regarding diagnosis, primary management, detection and treatment of relapse and survivorship care. Areas of lack of strong recommendation consensus are highlighted.

* Corresponding author. Genito-urinary Oncology Group, Gustave Roussy, France.
E-mail address: anna.partikidou@gustaveroussy.fr (A. Patrikidou).

https://doi.org/10.1016/j.eururo.2026.03.021

Metastatic disease
Chemotherapy
Retroperitoneal lymph node dissection
Risk-adapted treatment
Testis cancer

Conclusions and clinical implications: The 2026 version of the EAU Guidelines on TC collates the highest available scientific evidence to standardize the management of patients with TC.

1. Introduction

Testicular cancer (TC) (90–95% corresponding to testicular germ cell tumors of the testis [TGCT]) accounts for 1% of adult neoplasms and 5% of urological tumors [1], but it is the most frequent cancer of adolescent and young adult males. Its incidence has increased in recent decades, particularly in Western countries [1–3]. The cure rates remain high with >95% long-term survival across all stages of disease. The outcomes of treatments are closely linked to the expertise of the treating center as demonstrated in both localized and advanced disease, where minimizing treatment-related long-term toxicities and side effects is critical amid a growing population of TGCT survivors [4,5].

International guidelines, such as the European Association of Urology (EAU) Guidelines, disseminate the most recent evidence on TGCT diagnosis and management and provide clinical practice recommendations.

2. Methods

The EAU Guidelines document was compiled by a multidisciplinary team of TGCT expert clinicians (urologists, medical oncologists, radiation oncologists, and pathologists) following a full appraisal of published literature on the topic since the last Guidelines update publication in 2023 [5].

The full version of the 2026 guidelines is available on the EAU website (http://uroweb.org/ guideline/testicular-cancer/). References have been assessed regarding their level of scientific evidence, and guideline recommendations have been graded according to a system modified from the Oxford Centre for Evidence-Based Medicine levels of evidence.

3. Evidence Synthesis

3.1. Diagnosis and primary management of TC

3.1.1. Clinical assessment, imaging, and serum tumor markers

The most common presentation is characterized by a painless testicular mass or as an incidental finding during an ultrasound (US) of the testicles. High-frequency (>10 MHz) testicular Doppler US is the initial examination of choice to confirm the presence of a testicular mass [6]. The clinical examination should encompass not only the testicles but also the abdomen, chest, and supraclavicular area [7]. Subsequently, the staging should be completed with a contrast-enhanced computed tomography (CT) scan covering the chest (and supraclavicular fossa), abdomen, and pelvis. Magnetic resonance imaging (MRI) for the abdomen and pelvis together with noncontrast CT of the chest is an alternative if available and if contraindications to iodine-based contrast media are present.

Cerebral imaging, with or without spinal imaging is recommended in GCT patients who have either extensive supra-diaphragmatic disease (eg, lung metastases), human chorionic gonadotropin (hCG) values >5000 UI/L, a poor-prognosis International Germ Cell Cancer Collaborative Group (IGCCCG) tumor, or clinical symptoms concerning for brain metastasis.

Serum alpha-fetoprotein (AFP), hCG, and lactate dehydrogenase (LDH) should be tested before and after the orchidectomy (see Table 2 for definition of stage IS tumors). These markers are useful in supporting the diagnosis and monitoring treatment response, but have a low sensitivity, as normal levels do not exclude the presence of disease. A number of studies suggest higher discriminatory accuracy for micro-RNA (miRNAs) (particularly miR-371a-3p) [8–10] compared to conventional GCT markers in diagnosis, clinical staging, treatment monitoring, and prediction of residual or recurrent viable disease (except for pure teratoma). However, miR-371a-3p assessment still lacks standardization, the evidence is still principally retrospective, is not broadly accessible and is more costly. Prospective, biomarker-driven interventional studies are currently ongoing to better define the role of miR-371a-3p in clinical decision-making. At present, miRNA assessment should be considered investigational and not a replacement for standard tumor markers outside of clinical trials.

3.1.2. Radical orchidectomy and Testis-sparing surgery

Radical inguinal orchidectomy with division of the spermatic cord at the internal inguinal ring continues to be the initial treatment modality for testicular masses suspicious for malignancy is suspected [11]. Testis-sparing surgery (TSS) should be considered when feasible, particularly in synchronous bilateral tumors or tumors affecting a single remaining testis [12]. If TSS reveals a TGCT, adjuvant radiotherapy with 18–20 Gy of the remaining testicular tissue is recommended to eradicate remaining germ cell neoplasia in situ (GCNIS); in this case, the relapse rate is low with <2% local relapse and <5% systemic relapses [13].

TGCT patients with a small volume (<12 ml) of contralateral testicle or a history of cryptorchidism have a higher risk of developing contralateral metachronous disease from GCNIS. In these patients, the risks and benefits of contralateral testicular biopsy should be discussed [14].

If a biopsy confirms GCNIS, the risk of developing TC is 50% at 5 yr [15]. Management options in case of normal contralateral testicle include radical orchidectomy or close observation. In a solitary testis with GCNIS, testicular radiotherapy with 18–20 Gy in 2 Gy-fractions reduces the risk of

TGCT development, but also commonly leads to infertility [16].

Prior to any therapeutic intervention, all patients should be offered semen cryopreservation.

3.2. Staging and prognosis

The 2025 Tumor, Node, Metastasis (TNM) classification of the International Union Against Cancer (UICC) is recommended to determine the anatomical extent of the disease. The updated UICC 9th Edition has been included in the full text document [17]; (Table 1 and Table 2).

For patients with metastatic disease, the IGCCCG prognostic classification is used to guide outcome prediction and treatment decision-making [18–20]. The original 1997 IGCCCG classification [18] has been validated and amended in 2021 [19,20] based on a retrospective analysis of around 12,000 metastatic TGCT patients and the results are shown in Table 3, which highlighted improved outcomes especially for intermediate- and poor-prognosis disease. However, the updated classification did not provide treatment recommendations as is based on retrospective data. Specifically for metastatic seminomatous GCT (SGCT), the update demonstrated that an LDH elevation >2.5 times the upper limit of normal (ULN) within the good prognosis group is associated with outcomes similar to the intermediate prognosis group, exhibiting a lower 3-yr progression-free survival (PFS) rate (80% and 75% for the training and validation sets respectively and 79% for the intermediate prognosis disease), compared to the low-LDH good prognosis patients (92% and 93% for the training and validation sets, respectively) [20]. Hence, the question arises regarding the best management option for good prognosis high-LDH patients. In this regard, the global SEMITrends Survey [21], organized by the EAU Testicular Cancer Guidelines panel, demonstrated that only a minority of clinicians internationally (18%) adapt the treatment to reflect the higher risk of relapse for these patients, although this percentage increases in high-volume centers to up to 50%. Importantly,

Table 1 – TNM classification for testicular cancer (adapted from UICC, 2025, 9th edn)

pT – Primary Tumor[a]			
pTX	Primary tumor cannot be assessed[a]		
pT0	No evidence of primary tumor (e.g., histological scar in testis)		
pTis	Intratubular germ cell neoplasia (carcinoma *in situ*)[b]		
pT1	Tumor limited to testis, including the rete testis, but without vascular/lymphatic invasion or invasion of the epididymis; tumor may invade tunica albuginea but not tunica vaginalis[c]		
pT2	Tumor limited to testis with vascular/lymphatic invasion, or invading hilar soft tissue or epididymis or tumor extending through tunica albuginea with involvement of tunica vaginalis		
pT3	Tumor invades spermatic cord with or without vascular/lymphatic invasion[d]		
pT4	Tumor invades scrotum with or without vascular/lymphatic invasion		
N – Regional Lymph Nodes – Clinical			
cNX	Regional lymph nodes cannot be assessed		
cN0	No regional lymph node metastasis		
cN1	Metastasis with a lymph node mass 2 cm or less in greatest dimension or multiple lymph nodes, none more than 2 cm in greatest dimension		
cN2	Metastasis with a lymph node mass more than 2 cm but not more than 5 cm in greatest dimension; or multiple lymph nodes, any one mass more than 2 cm but not more than 5 cm in greatest dimension		
cN3	Metastasis with a lymph node mass more than 5 cm in greatest dimension		
Pn – Regional Lymph Nodes – Pathological			
pNX	Regional lymph nodes cannot be assessed		
pN0	No regional lymph node metastasis		
pN1	Metastasis with a lymph node mass 2 cm or less in greatest dimension and five or fewer positive nodes, none more than 2 cm in greatest dimension		
pN2	Metastasis with a lymph node mass more than 2 cm but not more than 5 cm in greatest dimension; or more than five nodes positive, none more than 5 cm; or evidence of extranodal extension of tumor		
pN3	Metastasis with a lymph node mass more than 5 cm in greatest dimension		
M – Distant Metastasis			
M0	No distant metastasis		
M1	Distant metastasis[d]		
M1a	Non-regional lymph node(s) or lung metastasis		
M1b	Distant metastasis other than non-regional lymph nodes and lung		
S – Serum Tumor Markers (Pre-chemotherapy)			
SX	Serum marker studies not available or not performed		
S0	Serum marker study levels within normal limits		
	LDH	**hCG (mIU/ml)**	**AFP (ng/ml)**
S1	<1.5 x N and	<5000 and	<1000
S2	1.5–10 x N or	5000–50,000 or	1000–10,000
S3	>10 x N or	>50,000 or	>10,000

N indicates the upper limit of normal.

LDH = lactate dehydrogenase; hCG = human chorionic gonadotrophin; AFP = alpha-fetoprotein.

[a] Except for pTis and pT4, where radical orchidectomy is not always necessary for classification purposes, the extent of the primary tumor is classified after radical orchidectomy; see pT. In other circumstances, Tx is used if no radical orchidectomy has been performed.

[b] Germ cell neoplasia in situ (GNCIS), per the World Health Organization (WHO) Classification of Tumors: Urinary and Male Genital Tumors, 5th edition, 2022

[c] AJCC 8th edition subdivides T1 pure seminoma by T1a and T1b depending on size no greater than 3 cm or greater than 3 cm in greatest dimension.

[d] AJCC 8th edition notes that the discontinuous involvement of the spermatic cord is considered as pM1.

Table 2 – Prognostic groups for testicular cancer (UICC, 2025, 9th edn.) (15)

Stage grouping	T	N	M	S
Stage 0	pTis	N0	M0	S0
Stage I	pT1-T4	N0	M0	SX
Stage IA	pT1	N0	M0	S0
Stage IB	pT2–pT4	N0	M0	S0
Stage IS	Any pT/TX	N0	M0	S1-3
Stage II	Any pT/TX	N1-N3	M0	SX
Stage IIA	Any pT/TX	N1	M0	S0
	Any pT/TX	N1	M0	S1
Stage IIB	Any pT/TX	N2	M0	S0
	Any pT/TX	N2	M0	S1
Stage IIC	Any pT/TX	N3	M0	S0
	Any pT/TX	N3	M0	S1
Stage III	Any pT/TX	Any N	M1a	SX
Stage IIIA	Any pT/TX	Any N	M1a	S0
	Any pT/TX	Any N	M1a	S1
Stage IIIB	Any pT/TX	N1-N3	M0	S2
	Any pT/TX	Any N	M1a	S2
Stage IIIC	Any pT/TX	N1-N3	M0	S3
	Any pT/TX	Any N	M1a	S3
	Any pT/TX	Any N	M1b	Any S

Stage IA: Primary tumors limited to the testis and epididymis without evidence of vascular or lymphatic invasion by tumor cells on histology, no sign of metastases on clinical examination or imaging, and post-orchidectomy serum tumor marker levels within normal limits. Marker decline in patients with Clinical Stage I (CS I) disease should be assessed until normalization occurs on two consecutive measurements.
Stage IB: More locally invasive primary tumor, but no sign of metastatic disease.
Stage IS: Following orchiectomy tumor markers increase, remain persistently elevated, or fail to decline as expected by half-lives indicating the presence of subclinical metastatic disease. The presence of a second GCT in the contralateral testis should also be excluded.

there is no prospective evidence to support treating these patients as per intermediate prognosis disease, highlighting the need for prospective randomized evidence to support guidelines on optimal treatment in this population.

Important changes have also been introduced regarding relapse risk stratification for stage I SGCT. A multi-institutional retrospective series including 1016 patients [22] and an additional validation cohort of 285 patients identified primary testicular tumor size (size <2, 2–5, and >5 cm), rete testis invasion (RTI; pagetoid and stromal invasion) and lymphovascular invasion (LVI) to be associated with risk of relapse. This resulted in the development of a three-tier risk stratification with increasing 5-yr cumulative probability of relapse (8% vs 20% vs 44%). In addition, a large prospective series from Denmark including 924 patients with central pathology review [23] identified invasion of the testicular hilum (rete testis and hilar soft tissue), LVI, and elevated hCG and LDH prior to orchiectomy (but not tumor size) as independent predictors of relapse. In this study a six-group stratification system was developed with 5-yr cumulative probability of relapse ranging from 6% to 62% in patient with all risk factors.

For clinical stage I nonseminomatous germ cell tumor (NSGCT), LVI was strongly associated with the risk of relapse. A recent Danish study also identified the presence of embryonal carcinoma with the probability of relapse ranging from <5% to >85% [24].

3.3. Management of clinical stage I germ cell tumors

3.3.1. Clinical stage I seminomatous germ cell tumor (SGCT) (Table 4)

In unselected patients, up to 20% of patients with clinical stage I SGCT will experience relapse after orchidectomy alone [5]. Individual patients have different risks of relapse within 5 yr of follow-up according to a combination of risk factors (see section 3.2) [22,23], and therefore, the decision regarding active surveillance (AS) versus potential adjuvant treatment should be considered after a thorough discussion with the patient. Nevertheless, AS is the preferred approach as approximately 80% of patients are cured by orchiectomy alone [25].

3.3.1.1. Surveillance. Given the low risk of relapse after orchidectomy alone and an estimated cancer-specific survival (CSS) rate of >99% regardless of the postsurgical treatment strategy, AS is the preferred adjuvant strategy for almost all patients. AS entails a protocol of regular cross-sectional imaging, serum tumor markers testing and clinical assessment for the early identification of relapses [26]. Abdominopelvic CT and MRI scanning is similarly effective to safely identify recurrences, according to prospective randomized trial. Therefore, MRI should be privileged as it avoids radiation exposure. Further, the frequency of scans does not impact patient survival, but three instead of seven scans over 5 yr can lead to a higher clinical stage at the time the recurrence is detected [26]. Patient compliance is crucial for an AS management strategy [27,28].

3.3.1.2. Adjuvant chemotherapy. Adjuvant chemotherapy with one course of carboplatin (AUC7) is an established alternative to AS, which reduces the risk of recurrence to around 5%, but represents overtreatment for up to 80% of patients. Patients without risk factors have no essential benefit from adjuvant chemotherapy, while for patients with risk factors (tumor size >4 cm and/or RTI), the recurrence risk is reduced from 15% to 9% according to a prospective Scandinavian series of 897 patients [29]. Consequently, whenever adjuvant carboplatin is considered, the above-

Table 3 – Prognostic-based staging system for metastatic germ cell cancer (IGCCCG)(16,17,18)[a]

Good-prognosis group	
Non-seminoma	*All of the following criteria:*
5-year PFS 90%	• Testis/retro-peritoneal primary
5-year OS 96%	• No non-pulmonary visceral metastases
	• AFP <1,000 ng/ml
	• hCG <5,000 IU/L (1,000 ng/ml)
	• LDH <1.5 x ULN
Seminoma	*All of the following criteria:*
5-year PFS 89%	• Any primary site
5-year OS 95%	• No non-pulmonary visceral metastases
	• Normal AFP
	• Any hCG
	• Any LDH
Intermediate-prognosis group	
Non-seminoma	*Any of the following criteria:*
5-year PFS 78%	• Testis/retro-peritoneal primary
5-year survival 89%	• No non-pulmonary visceral metastases
	• AFP 1,000–10,000 ng/ml or
	• hCG 5,000–50,000 IU/L or
	• LDH 1.5–10 x ULN
Seminoma	*All of the following criteria:*
5-year PFS 79%	• Any primary site
5-year survival 88%	• Non-pulmonary visceral metastases
	• Normal AFP
	• Any hCG
	• Any LDH
Poor-prognosis group	
Non-seminoma	*Any of the following criteria:*
5-year PFS 54%	• Mediastinal primary
5-year survival 67%	• Non-pulmonary visceral metastases
	• AFP >10,000 ng/ml or
	• hCG >50,000 IU/L (10,000 ng/ml) or
	• LDH >10 x ULN
Seminoma	No patients classified as "poor-prognosis"

AFP = alpha-fetoprotein; hCG = human chorionic gonadotrophin; LDH = lactate dehydrogenase; OS = overall survival; PFS = progression-free survival; ULN = upper limit of normal.

[b]The updated IGCCCG classification showed that good-prognosis metastatic seminoma with LDH>2.5 ULN have a worse prognosis than other good-prognosis patients, similar to the intermediate prognosis group.

[a] Pre-chemotherapy serum tumor markers should be assessed immediately prior to the administration of chemotherapy (same day).

mentioned risk factors should be taken into account. However, the application of adjuvant carboplatin may limit de-intensified treatment options in case of low-volume recurrence (see section 3.4.1).

3.3.1.3. Adjuvant radiotherapy. Adjuvant radiotherapy (RT) to the ipsilateral para-aortic/para-caval and common iliac strip with a cumulative dose of 20 Gy has traditionally been used for CS I seminoma patients and had shown similar activity to carboplatin in a randomized trial [30]. However, due to long-term side effects of radiotherapy, especially radiation-induced nongerm cell malignancies in the irradiated field, RT is now generally obsolete. It may only be reserved for highly selected patients, who are unsuitable for any systemic chemotherapy.

3.3.1.4. Risk-adapted treatment. Using primary tumor size and rete testis stromal infiltration to depict higher risk patients may offer some benefit from adjuvant chemotherapy with a reduced risk of relapse (Table 4). The predictive value of the new, more granular risk assessment models (see chapter 3.2); however, remains to be defined, to date.

3.3.2. Clinical stage I non-seminomatous germ cell tumour (NSGCT) (Table 5)

Overall, approximately 70% of CSI NSGCTs are cured with orchiectomy alone. LVI plays a crucial role in defining the risk of relapse (15% without LVI and 50% in those with LVI), and it is the best validated risk factor for CSI NSGCT.

3.3.2.1. Surveillance. The largest reports of surveillance indicate a cumulative relapse risk in about 30% of CS I NSGCT (5-yr conditional risk of relapse 42% and 17% for high- and low-risk CS I NSGCT, respectively). Most relapses (92%) occur within the first 2 yr of follow-up [26,31].

3.3.2.2. Retroperitoneal lymph node dissection (RPLND). The role of adjuvant primary retroperitoneal lymph node dissection (RPLND) in men with CS I NSGCTs has decreased in view of the high CSS rates of surveillance, low relapse rates following adjuvant chemotherapy, and the lower reproducibility of primary RPLND on a large scale.

The few indications in stage I NSGCT disease include men with testicular predominant teratoma with somatic malignant component, or patients who are not willing or suitable to undergo chemotherapy in case of recurrence, in particular for LVI-positive disease.

The development of nerve-sparing dissection and minimally invasive surgical approaches have reduced the morbidity of primary RPLND, however this should be performed only by experienced surgeons in a specialist, high-volume center to minimize the risk of in-field relapses.

Table 4 – Recommendation for the management of Stage I Seminoma

Recommendations	Strength rating
Inform patients about all available management options, including surveillance or adjuvant therapy after orchidectomy, as well as treatment-specific recurrence rates and acute and long-term side effects.	Strong
Offer surveillance as the preferred management option if resources are available and the patient is compliant.	Strong
Offer one dose of carboplatin at area under curve 7 if adjuvant chemotherapy is considered.	Strong
Do not perform adjuvant treatment in patients at very low risk of recurrence (no risk factors).	Strong
Do not routinely perform adjuvant radiotherapy.	Strong
Adjuvant radiotherapy should be reserved only for highly selected patients.	Strong

Table 5 – Recommendation for the management of Stage I Non-seminomatous germ cell tumor

Recommendations	Strength rating
Inform patients about all management options after orchidectomy: surveillance, adjuvant chemotherapy, and retroperitoneal lymph node dissection, including treatment-specific recurrence rates as well as acute and long-term side effects.	Strong
Offer surveillance or risk-adapted treatment based on lymphovascular invasion.	Strong
Discuss one course of cisplatin, etoposide, bleomycin as an adjuvant treatment alternative in patients with stage I non-seminomatous germ cell tumor if patients are not willing to undergo or comply with surveillance.	Strong

Table 6 – Guidelines for the treatment of metastatic testicular germ cell tumors

Recommendations	Strength rating
Treat low-volume non-seminomatous germ cell tumor (NSGCT) stage IIA/B with elevated markers like metastatic good- or intermediate-prognosis risk group IGCCCG with three or four cycles of cisplatin, etoposide, bleomycin (BEP).	Strong
Nerve-sparing retroperitoneal lymph node dissection when performed by an experienced surgeon in a specialized center is the recommended initial treatment in clinical stage (CS) IIA NSGCT disease without elevated tumor markers.	Weak
Repeat staging after six weeks before making a final decision on further management should be considered in patients with small volume (CS IIA <2 cm) marker-negative NSGCT.	Weak
Treat metastatic NSGCT (stage >IIC) with an intermediate prognosis with four cycles of standard BEP.	Strong
Treat metastatic NSGCT with a poor prognosis and favorable marker decline with four cycles of BEP.	Strong
Assess tumor marker decline after one cycle of standard chemotherapy in metastatic NSGCT with a poor-prognosis. With unfavorable decline, consider chemotherapy intensification.	Weak
Perform surgical resection of visible (>1 cm in longest diameter) residual masses after chemotherapy for NSGCT when serum levels of tumor markers are normal or normalizing.	Strong
Offer cisplatin chemotherapy according to IGCCCG prognosis groups, or alternatively radiotherapy to seminoma patients with stage II A/B and, inform the patient of potential long-term side effects of both treatment options.	Weak
Treat seminoma stage IIC and higher, with primary chemotherapy according to IGCCCG classification (BEP x 3 in good-prognosis and BEP x 4 in intermediate prognosis).	Strong

If RPLND is done for CSI NSCT and documents a PS II disease, both AS and adjuvant chemotherapy, usually comprising two cycles of bleomycine-etoposide-cisplatin (BEP) chemotherapy although the regimen is to be discussed for each individual case (except for cases of postpubertal teratoma only, for which there is no indication for chemotherapy), are standard option to be discussed with each individual. Recent publications support the safety of surveillance alone in PS II disease following RPLND, as 75–80% are relapse-free at 2 and 5 yr [32–34].

3.3.2.3. Adjuvant chemotherapy. Adjuvant chemotherapy has been evaluated with both one and two cycles of BEP in CS I NSGCT.

Results from series including one or two cycles of chemotherapy have identified similar relapse rates (2.7% with two cycles of BEP and approximately 3% with one cycle of BEP) [35], as such BEPx1 is the standard of care if chemotherapy is considered.

A retrospective analysis indicated that about one-third of relapses after adjuvant chemotherapy were late, and that the outcome may be slightly worse compared to patients presenting with de novo metastatic disease [36]. The very long-term (>20 yr) side effects of adjuvant chemotherapy, particularly cardiovascular events and secondary malignancies, are yet to be fully defined and this should be considered during decision-making.

3.3.2.4. Risk-adapted treatment. LVI is the strongest predictive factor for relapse and should be carefully outlined to the patient to assist informed treatment decision-making. Those patients with LVI presence in their tumors should have their high risk of relapse (up to 50%) highlighted and adjuvant chemotherapy with BEP x 1 ought to be considered as the preferred option, given the significant reduction in the risk of relapse that adjuvant chemotherapy conveys (Table 5; Fig. 1).

3.4. Metastatic germ cell tumors (Table 6)

3.4.1. Stage IIA/B SGCT

Historically, retroperitoneal radiotherapy to the ipsilateral para-aortic/para-caval and common iliac field with 30 Gy or 36 Gy was the previous gold standard for stage II A/B seminoma, leading to relapse rates of 9–24% and 5-yr relapse-free survival rates of 92% and 90% for CS IIA and CS IIB, respectively [37]. Today, cisplatin-based chemotherapy with BEPx3 or EP (etoposide, cisplatin) x4 in case of good prognosis disease [38] and BEP x4 for intermediate prognosis disease (or VIP x4 in case of contraindication to bleomycin) is considered the standard of care.

The high cure rates and the known possible long-term sequelae of cisplatin-based combination chemotherapy have led to the clinical assessment of de-escalation strategies in recent years. Chemotherapy, chemo-radiation, and surgical approaches have demonstrated promising results in phase 2 trials, while randomized comparisons to standard chemotherapy are lacking and are challenging to obtain for this rare clinical setting [39–41].

The phase 2 SEMITEP trial [42], assessed chemotherapy de-escalation in state II-III patients with low-LDH (<1.5 xULN) guided by metabolic response on FDG-PET/CT after

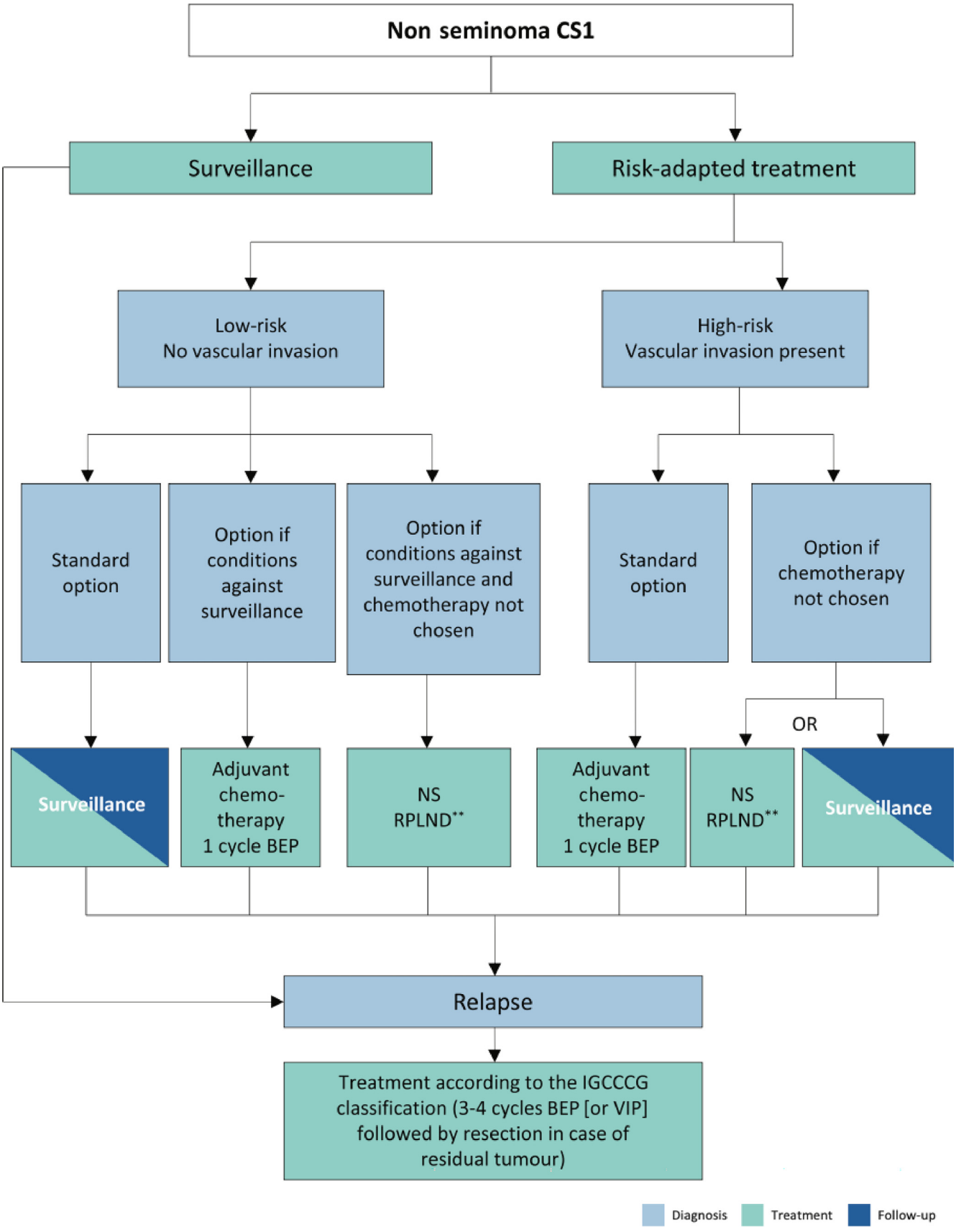

Fig. 1 – Risk-adapted treatment in patients with clinical stage I NSGCT.*.* Discuss all treatment options with individual patients, to allow them to make an informed decision as to their further care. ** In case of PS II, the rate of recurrence is higher, and chemotherapy can be discussed (max. two cycles). # Primary retroperitoneal lymph node dissection should be advised in men with postpubertal teratoma with somatic malignant component. BEP = cisplatin, etoposide, bleomycin; CS = clinical stage; IGCCCG = International Germ Cell Cancer Collaborative; Group; NS = nerve-sparing; RLNPD = retroperitoneal lymph node dissection; VIP = etoposide, cisplatin, ifosfamide.

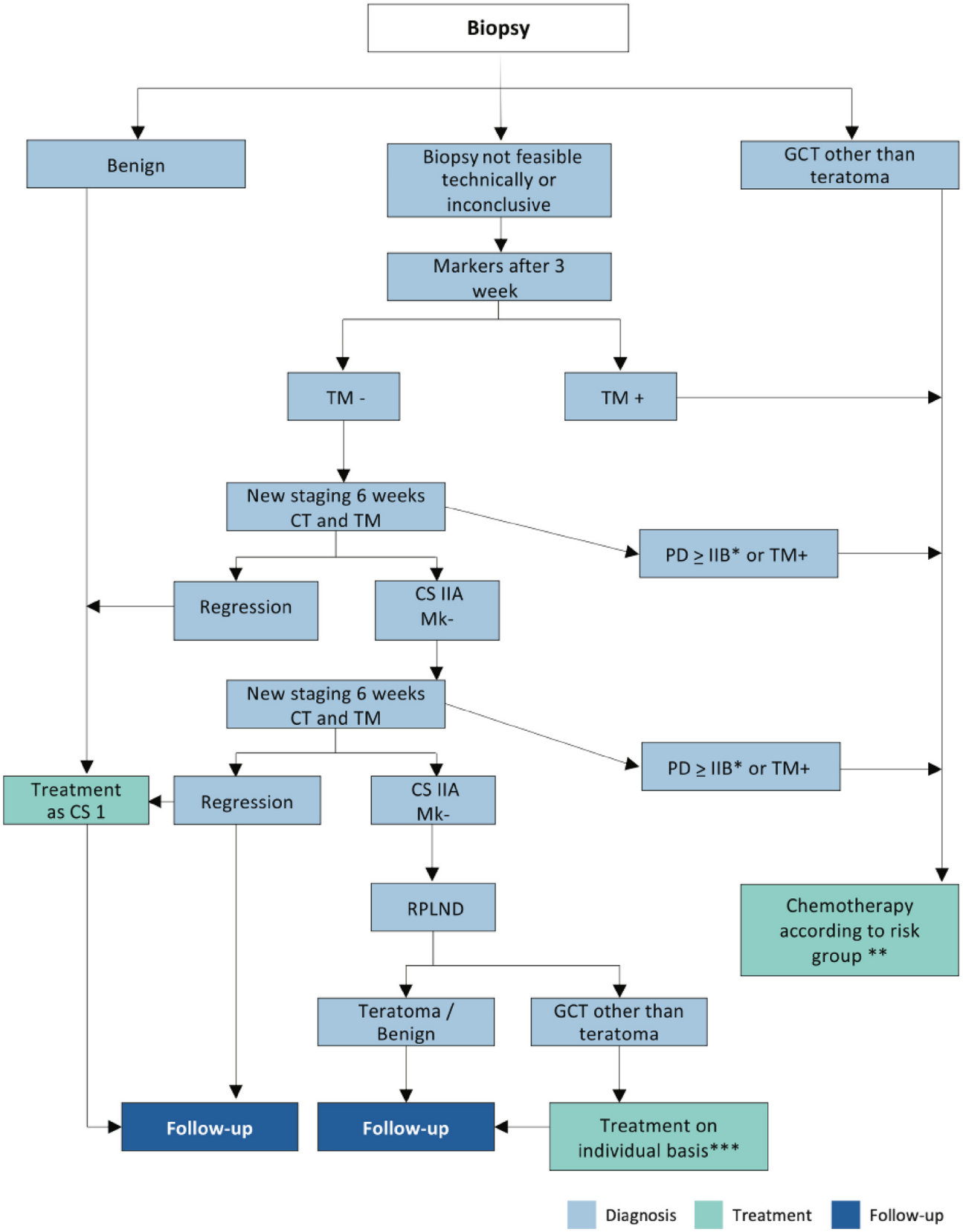

Fig. 2 – Flowchart NSGCT CS IIA with normal tumor markers at diagnosis/staging. CS = clinical stage; CT = computed tomography; GCT = germ cell tumor; GCC = germ cell cancer; RPLND = retroperitoneal lymph node dissection; PD = progressive disease; TM = tumor markers. [a] With marker negative PD > IIB RPLND may be considered if radiological features of teratoma. [b] Most will be good prognostic group (BEP x3 or EP x4)—see Appendix 3 https://uroweb.org/guidelines/testicular-cancer/publications-appendices. [c] In case of PS II A/B patient can be followed-up or receive adjuvant chemotherapy (maximum of 2 cycles).

two initial cycles of EP chemotherapy. Patients with complete metabolic response after EP x2 received only one subsequent cycle of carboplatin AUC7, while patients with residual metabolic activity completed the standard of EP × 4. Although nonrandomized, the study showed comparable 3-yr PFS rate of 90% and 91% for the EP and carboplatin groups respectively, with a 2-yr overal survival (OS) of 100% for both groups.

The SAKK 01/10 trial [43] tested a combination therapy of one cycle of carboplatin AUC7 followed by involved-node (small-volume) radiotherapy (30 Gy in 15 fractions for stage IIA and 36 Gy in 18 fractions for stage IIB). This approach has shown a 3-yr PFS rate of 93.7% and 10-yr PFS of 92.8% with reasonable acute and long-term toxicity, although the trial narrowly missed its primary endpoint of a 3-yr PFS rate of 95%.

Lastly, primary RPLND (with or without adjuvant chemotherapy) has also been assessed in phase 2 trials and prospective institutional series. The reported 2-yr recurrence rates range between 5–30% (compared to <10% with chemotherapy or radiotherapy) [38], surgery-related complications Clavien-Dindo ≥3a are uncommon (5–12%) and antegrade ejaculation was preserved in 90–97% of cases according to different studies [39–41,44,45]. If primary RPLND is considered for men with low-volume, marker-negative CS II seminoma, it ought to be performed by surgeons with extensive experience in specialized TC centers.

Overall, while promising, de-escalation strategies (chemotherapy, radiotherapy, or surgical approaches) should currently only be considered in suitably selected patients and preferably within experienced centers or prospective clinical trials.

3.4.2. *Stage IIA/IIB NSGCT*

Initial surveillance with early re-evaluation should be considered in patients with normal markers and equivocal lymph nodes (<2 cm) (Fig. 2). If the lesion progresses or fails to resolve or if tumor markers increase, the disease should be regarded and treated as CS II.

Patients with elevated tumor markers and radiological stage IIA/B at diagnosis or relapse should be treated with chemotherapy according to IGCCCG risk group. Most patients will have a good prognosis disease, for whom BEP x3 is the standard regimen, while EP x4 is considered if contraindication to bleomycin exist.

Nerve-sparing RPLND is a valid upfront management option for CS IIA NSGCT with normal (or normalized) tumor makers and confirmed lymphadenopathy. Patients may be down staged to PS I in up to 20% of cases and require no further treatment. Patients with postpubertal teratoma alone would equally receive the appropriate treatment and avoid unnecessary chemotherapy [46] (Fig. 2).

Marker-positive stage IIA disease ought to be treated as per IGCCCG prognostic classification (see section 3.4.3).

3.4.3. *Advanced metastatic disease (stage IIC and III)*

The standard regimen in good prognosis SGCT is three cycles of BEP chemotherapy. Alternatively, EP × 4 may be used, especially when bleomycin is contraindicated. Particular attention should be reserved to those patients within this category who have high level of LDH (>2.5 ULN), corresponding to approximately 20% of good prognosis metastatic SGCT [20], whose tumors have a worse prognosis compared to low-LDH tumors, posing the realistic, still unanswered question of the optimal treatment for these patients and whether treatment should be intensified [21]. Prospective randomized data to support clinical recommendations in this setting are currently lacking.

The standard regimen in good prognosis NSGCT is BEP ×3 cycles. Chemotherapy should be given without dose reductions or delays at 21-d intervals whenever possible. This includes patients showing prolonged cytopenias. Uncontrolled septicemia, however, may require dose delays.

For patients with intermediate prognosis SGCT or NSGCT, BEP x4 is the standard regimen. If contraindications against bleomycin exist, VIP x4 has shown similar efficacy but with more pronounced hemato-toxicity [47,48].

In poor-prognosis NSGCT risk group the historical standard regimen is four cycles of BEP. Serum tumor marker decline kinetics are the only prospectively confirmed predictor for response to cisplatin-based chemotherapy in metastatic germ cell tumor patients. Patients showing inadequate (unfavorable) marker decline have a higher risk of treatment failure [49]. A dose-dense regimen (consisting of two cycles of paclitaxel-BEP-oxaliplatin and two further cycles of cisplatin/bleomycin/ifosfamide) in cases of unfavorable marker decline after the first cycle of BEP (corresponding to approximately 80% of poor prognosis NGCT) has shown statistically superior PFS outcomes (5-yr PFS of 58.9% vs 46.7%, HR: 0.65), and numerically superior OS (10% absolute difference at 5 yr), without significant long-term toxicity and with decreased subsequent need for salvage high-dose chemotherapy. Given the above, such a marker-based decline approach should be considered in this context [50,51].

3.4.4. *Re-staging and treatment evaluation*

Tumor markers should be assessed immediately prior to the start of each cycle of chemotherapy. When four cycles of chemotherapy are administered, a mid-treatment re-staging imaging is performed.

If markers have normalized and masses with features of postpubertal teratoma show early progression, early surgical resection before the end of chemotherapy should be considered.

Table 7 – Summary of changes 2026 guidelines

The literature for the complete document has been updated.
A new section on treatment de-escalation strategies for early metastatic seminoma has been included. This covers new evidence on role of RPLND, chemo-radiotherapy, and de-escalated chemotherapy with updated summary of evidence and recommendation.
The section regarding risk factors for stage I seminoma has been updated.
The section regarding treatment for metastatic disease has been restructured and integrated. A new section of the treatment of bone metastasis has been included.
A new section regarding insertion of testicular prosthesis has been added to the text.
The section on rare adult and para-testicular tumors has been updated and restructured.

3.4.5. Residual tumour resection

3.4.5.1. *SGCT.* A residual mass of seminoma should initially be monitored with imaging and tumor markers. Postchemotherapy residual masses of up to 3 cm represent necrosis in 97–100% and FDG-PET is not recommended, therefore these should be monitored with iodine contrast-enhanced CT scan (or MRI in case of contraindication). As FDG-PET has a reliable negative predictive value (>90%) in patients with residual masses >3 cm in largest diameter, this should be considered, not earlier than 6–8 wk after the end of chemotherapy to provide more accurate information on disease viability. The positive-predictive value instead varies between 23–69%, so that PET-tracer avidity alone is not enough to confirm persistent disease and to trigger further treatment. For PET-avid but volumetrically stable residual disease without marker increase, a repeat FGD/PET-CT scan after 6–8 wk is recommended to assess possible progression and initiate further investigation or treatment [52,53].

Surgical resection of residual seminoma masses can be extremely challenging, therefore in the limited cases when RPLND is indicated, this should be performed in specialist high-volume centers by an expert surgeon.

3.4.5.2. *NSGCT.* In this setting, resection is mandatory for all patients with a residual mass >1 cm at the transaxial long axis on cross-sectional contrast-enhanced CT imaging [54]. To date, there are no reliable prediction tools to assess the histology within residual masses following first-line chemotherapy (7% of residual masses contain active cancer, 33% postpubertal teratoma, and 40% necrotic-fibrotic tissue only) [55].

Surgery, when indicated, should be performed within 6–8 wk after the last chemotherapy cycle. The role of surgery with residual retroperitoneal lesions <1 cm following first-line chemotherapy is uncertain. However, following any further line of systemic treatment for relapsed disease, all visible residual masses including those <1 cm need to be resected if technically feasible due to the greater risk of chemo-resistant viable disease within any visible mass.

For retroperitoneal residual mass resection, bilateral nerve-sparing RPLND is the standard option. Ipsilateral template resection avoids contralateral nerve dissection and may be considered for patients with a residual mass diameter <5 cm [56], and unilateral lymph node metastases on pre- and postchemotherapy CT scans (mapping studies indicate the potential risk of contralateral disease with this approach is low at around 1–3%). Laparoscopic or robotic RPLND may yield comparable outcomes to open procedures in selected cases with low-volume residual disease and when undertaken by highly experienced surgeons at GCT specialist centers.

Postchemotherapy surgery is demanding and often requires ad hoc vascular interventions (eg, vena cava or aortic prosthesis). Patients referred to specialized centers capable of interdisciplinary surgery show a significant reduction in perioperative morbidity and mortality.

3.5. Quality of life and long-term toxicities after cure of TC

TC generally has an excellent response to treatment and consequent overall prognosis. Given the young age at diagnosis of this patient cohort (between 18 and 40 yr), it is important to discuss and screen for treatment associated toxicities.

While in CS I disease the side effects are minimal and mostly associated with adjuvant chemotherapy, after completion of systemic chemotherapy for more advanced disease numerous side effects affecting nearly all organ systems, and psycho-social aspects including fatigue, have been described.

Adverse health outcomes (AHOs) are more commonly found in patients that received chemotherapy than those cured by surgery alone. During follow-up, patients should be screened and treated for known cardiovascular risk factors such as hypertension, hyperlipidemia, and testosterone deficiency. Further, modifiable risk factors such as smoking, dietary style or exercise contribute to AHOs like hypertension, noise exposure to hearing impairment or smoking to Raynaud phenomenon. When curative treatment is completed, a written cancer survivorship plan addressing late toxic effects, lifestyle recommendations and recurrence risk is indicated. Structured survivorship programs should be integrated into long-term follow-up, including cardiovascular and endocrine assessment, fertility counseling, secondary malignancy screening, and lifestyle support.

4. Conslusions

The current paper summarises the most up-to-date evidence on the management of testicular cancer as preseanted in the 2026 Guidelines of the European Association of Urology. Changes and issues of special interest are summarised in Table 7.

Author contributions: Anna Patrikidou had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study concept and design: Patrikidou, Cazzaniga.

Acquisition of data: Patrikidou, Cazzaniga, Oing, Berney, Lobo, Boormans, Fankhauser, Fischer, Leao, Gravina, Di Nardo, Cornes, Jain, Janisch, Haugnes, Papachristofilou, Tandstad, Heidenreich.

Analysis and interpretation of data: Patrikidou, Cazzaniga, Oing, Berney, Lobo, Boormans, Fankhauser, Fischer, Leao, Gravina, Di Nardo, Cornes, Jain, Janisch, Haugnes, Papachristofilou, Tandstad, Heidenreich.

Drafting of the manuscript: Patrikidou, Cazzaniga.

Critical revision of the manuscript for important intellectual content: Patrikidou, Cazzaniga, Oing, Berney, Lobo, Boormans, Fankhauser, Fischer, Leao, Gravina, Di Nardo, Cornes, Jain, Janisch, Haugnes, Papachristofilou, Tandstad, Heidenreich.

Statistical analysis: None.

Obtaining funding: None.

Administrative, technical, or material support: Bezuidenhout.

Supervision: Heidenreich.

Other (specify): None.

Financial disclosures: Anna Patrikidou certifies that all conflicts of interest, including specific financial interests and relationships and affiliations relevant to the subject matter or materials discussed in the manuscript (eg, employment/affiliation, grants or funding, consultancies, honoraria, stock ownership or options, expert testimony, royalties, or patents filed, received, or pending), are the following: None.

***Funding/Support and role of the sponsor*:** None.

References

1 Park JS, Kim J, Elghiaty A, Ham WS. Recent global trends in testicular cancer incidence and mortality. Med (Baltim) 2018;97:e12390.
2 Nigam M, Aschebrook-Kilfoy B, Shikanov S, Eggener S. Increasing incidence of testicular cancer in the United States and Europe between 1992 and 2009. World J Urol 2015;33:623–31.
3 Gurney JK, Florio AA, Znaor A, et al. International trends in the incidence of testicular cancer: lessons from 35 years and 41 countries. Eur Urol 2019;76:615–23.
4 Albers P, Albrecht W, Algaba F, et al. Guidelines on testicular cancer: 2015 update. Eur Urol 2015;68:1054–68.
5 Patrikidou A, Cazzaniga W, Berney D, et al. European Association of Urology guidelines on testicular cancer: 2023 update. Eur Urol 2023;84:289–301.
6 Angulo JC, González J, Rodríguez N, et al. Clinicopathological study of regressed testicular tumors (apparent extragonadal germ cell neoplasms). J Urol 2009;182:2303–10.
7 Germà-Lluch JR, Garcia del Muro X, Maroto P, et al. Clinical pattern and therapeutic results achieved in 1490 patients with germ-cell tumors of the testis: the experience of the Spanish Germ-Cell Cancer Group (GG). Eur Urol 2002;42:553–62, discussion 62–3.
8 Dieckmann KP, Dumlupinar C, Radtke A, et al. Associations of serum levels of microRNA-371a-3p (M371) with risk factors for progression in nonseminomatous testicular germ cell tumors clinical stage 1. World J Urol 2022;40:317–26.
9 Leão R, Albersen M, Looijenga LHJ, et al. Circulating microRNAs, the next-generation serum biomarkers in testicular germ cell tumors: a systematic review. Eur Urol 2021;80:456–66.
10 Belge G, Grobelny F, Radtke A, et al. Serum levels of microRNA-371a-3p are not elevated in testicular tumors of non-germ cell origin. J Cancer Res Clin Oncol 2021;147:435–43.
11 Patel HD, Gupta M, Cheaib JG, et al. Testis-sparing surgery and scrotal violation for testicular masses suspicious for malignancy: a systematic review and meta-analysis. Urol Oncol 2020;38:344–53.
12 Nason GJ, Aditya I, Leao R, et al. Partial orchiectomy: the Princess Margaret cancer center experience. Urol Oncol 2020;38:605e.19–24.
13 Paffenholz P, Held L, Loosen SH, Pfister D, Heidenreich A. Testis sparing surgery for benign testicular masses: diagnostics and therapeutic approaches. J Urol 2018;200:353–60.
14 Dieckmann KP, Kulejewski M, Pichlmeier U, Loy V. Diagnosis of contralateral testicular intraepithelial neoplasia (TIN) in patients with testicular germ cell cancer: systematic two-site biopsies are more sensitive than a single random biopsy. Eur Urol 2007;51:175–83, discussion 83–5.
15 Hoei-Hansen CE, Rajpert-De Meyts E, Daugaard G, Skakkebaek NE. Carcinoma in situ testis, the progenitor of testicular germ cell tumors: a clinical review. Ann Oncol 2005;16:863–8.
16 Petersen PM, Giwercman A, Daugaard G, et al. Effect of graded testicular doses of radiotherapy in patients treated for carcinoma-in-situ in the testis. J Clin Oncol 2002;20:1537–43.
17 Brierley JD, Giuliani M, O'Sullivan B, Rous B, Van Eycken E. The TNM Classification of Malignant Tumours ed. 9 2025;2025, https://www.uicc.org/news-and-updates/25-7-announcements/9th-edition-uicc-tnm-classification-malignant-tumours-now-available.
18 Mead GM. International Germ Cell Consensus Classification: A prognostic factor-based staging system for metastatic germ cell cancers. International Germ Cell Cancer Collaborative Group. J Clin Oncol 1997;15:594–603.
19 Gillessen S, Sauvé N, Collette L, et al. Predicting outcomes in men with metastatic nonseminomatous germ cell tumors (NSGCT): results from the IGCCCG update consortium. J Clin Oncol 2021;39:1563–74.
20 Beyer J, Collette L, Sauvé N, et al. Survival and new prognosticators in metastatic seminoma: results from the IGCCCG-update consortium. J Clin Oncol 2021;39:1553–62.
21 Patrikidou A, Oing C, Markellos C, et al. Redefining good-prognosis seminoma: implications for clinical practice of the updated International Germ Cell Cancer Collaborative Group classification and results from the SEMITrends survey. Eur Urol Oncol 2025;8:1248–52.
22 Boormans JL, Sylvester R, Anson-Cartwright L, et al. Prognostic factor risk groups for clinical Stage I seminoma: an individual patient data analysis by the European Association of Urology testicular cancer guidelines panel and guidelines office. Eur Urol Oncol 2024;7:537–43.
23 Wagner T, Toft BG, Lauritsen J, et al. Prognostic factors for relapse in patients with clinical stage I testicular seminoma: A nationwide, population-based cohort study. J Clin Oncol 2024;42:81–9.
24 Wagner T, Toft BG, Lauritsen J, et al. Prognostic factors for relapse in patients with clinical stage I testicular non-seminoma: A nationwide, population-based cohort study. Eur J Cancer 2024;202:114025.
25 Huang MM, Su ZT, Cheaib JG, et al. Cost-effectiveness analysis of non-risk-adapted active surveillance for postorchiectomy management of clinical Stage I seminoma. Eur Urol Focus 2021;7:1409–17.
26 Joffe JK, Cafferty FH, Murphy L, et al. Imaging modality and frequency in surveillance of Stage I seminoma testicular cancer: results from a randomized, Phase III, noninferiority trial (TRISST). J Clin Oncol 2022;40:2468–78.
27 Groll RJ, Warde P, Jewett MA. A comprehensive systematic review of testicular germ cell tumor surveillance. Crit Rev Oncol Hematol 2007;64:182–97.
28 Ruf CG, Schmidt S, Kliesch S, et al. Testicular germ cell tumours' clinical stage I: comparison of surveillance with adjuvant treatment strategies regarding recurrence rates and overall survival-a systematic review. World J Urol 2022;40:2889–900.
29 Tandstad T, Ståhl O, Dahl O, et al. Treatment of stage I seminoma, with one course of adjuvant carboplatin or surveillance, risk-adapted recommendations implementing patient autonomy: a report from the Swedish and Norwegian Testicular Cancer Group (SWENOTECA). Ann Oncol 2016;27:1299–304.
30 Oliver RTD, Mason MD, Mead GM, et al. Radiotherapy versus single-dose carboplatin in adjuvant treatment of stage I seminoma: a randomized trial. Lancet 2005;366:293–300.
31 Kollmannsberger C, Tandstad T, Bedard PL, et al. Patterns of relapse in patients with clinical stage I testicular cancer managed with active surveillance. J Clin Oncol 2015;33:51–7.
32 Nicolai N, Miceli R, Necchi A, et al. Retroperitoneal lymph node dissection with no adjuvant chemotherapy in clinical stage I nonseminomatous germ cell tumors: long-term outcome and analysis of risk factors of recurrence. Eur Urol 2010;58:912–8.
33 Nicolai N, Tarabelloni N, Gasperoni F, et al. Laparoscopic retroperitoneal lymph node dissection for clinical Stage I nonseminomatous germ cell tumors of the testis: safety and efficacy analyses at a high volume center. J Urol 2018;199:741–7.
34 Al-Ahmadie HA, Carver BS, Cronin AM, et al. Primary retroperitoneal lymph node dissection in low-stage testicular germ cell tumors: a detailed pathologic study with clinical outcome analysis with special emphasis on patients who did not receive adjuvant therapy. Urology 2013;82:1341–6.
35 Tandstad T, Dahl O, Cohn-Cedermark G, et al. Risk-adapted treatment in clinical stage I nonseminomatous germ cell testicular cancer: the SWENOTECA management program. J Clin Oncol 2009;27:2122–8.
36 Fischer S, Tandstad T, Cohn-Cedermark G, et al. Outcome of men with relapses after adjuvant bleomycin, etoposide, and cisplatin for clinical Stage I nonseminoma. J Clin Oncol 2020;38:1322–31.
37 Classen J, Schmidberger H, Meisner C, et al. Radiotherapy for stages IIA/B testicular seminoma: final report of a prospective multicenter clinical trial. J Clin Oncol 2003;21:1101–6.
38 Giannatempo P, Greco T, Mariani L, et al. Radiotherapy or chemotherapy for clinical stage IIA and IIB seminoma: a systematic

review and meta-analysis of patient outcomes. Ann Oncol 2015;26:657–68.
39 Daneshmand S, Cary C, Masterson T, et al. Surgery in early metastatic seminoma: A Phase II trial of retroperitoneal lymph node dissection for testicular seminoma with limited retroperitoneal lymphadenopathy. J Clin Oncol 2023;41:3009–18.
40 Hiester A, Che Y, Lusch A, et al. Phase 2 single-arm trial of primary retroperitoneal lymph node dissection in patients with seminomatous testicular germ cell tumors with clinical stage IIA/B (PRIMETEST). Eur Urol 2023;84:25–31.
41 Heidenreich A, Paffenholz P, Hartmann F, Seelemeyer F, Pfister D. Retroperitoneal lymph node dissection in clinical stage IIA/B metastatic seminoma: results of the COlogne trial of retroperitoneal lymphadenectomy in metastatic seminoma (COTRIMS). Eur Urol Oncol 2024;7:122–7.
42 Loriot Y, Texier M, Culine S, et al. The GETUG SEMITEP trial: de-escalating chemotherapy in good-prognosis seminoma based on fluorodeoxyglucose positron emission tomography/computed tomography. Eur Urol 2022;82:172–9.
43 Papachristofilou A, Bedke J, Hayoz S, et al. Single-dose carboplatin followed by involved-node radiotherapy for stage IIA and stage IIB seminoma (SAKK 01/10): a single-arm, multicentre, phase 2 trial. Lancet Oncol 2022;23:1441–50.
44 Thor A, Negaard HFS, Grenabo Bergdahl A, et al. Primary retroperitoneal lymph node dissection as treatment for low-volume metastatic seminoma in a population-based cohort: the Swedish Norwegian Testicular Cancer Group experience. Eur Urol Open Sci 2024;65:13–9.
45 Matulewicz RS, Benfante N, Funt SA, et al. Primary retroperitoneal lymph node dissection for seminoma metastatic to the retroperitoneum. J Urol 2024;211:80–9.
46 Neuenschwander A, Lonati C, Antonelli L, et al. Treatment outcomes for men with clinical Stage II nonseminomatous germ cell tumors treated with primary retroperitoneal lymph node dissection: A systematic review. Eur Urol Focus 2023;9:541–6.
47 de Wit R, Stoter G, Sleijfer DT, et al. Four cycles of BEP vs four cycles of VIP in patients with intermediate-prognosis metastatic testicular non-seminoma: a randomized study of the EORTC Genitourinary Tract Cancer Cooperative Group. European Organization for Research and Treatment of Cancer. Br J Cancer 1998;78:828–32.
48 Nichols CR, Catalano PJ, Crawford ED, Vogelzang NJ, Einhorn LH, Loehrer PJ. Randomized comparison of cisplatin and etoposide and either bleomycin or ifosfamide in treatment of advanced disseminated germ cell tumors: an Eastern Cooperative Oncology Group, Southwest Oncology Group, and Cancer and Leukemia Group B Study. J Clin Oncol 1998;16:1287–93.
49 Fizazi K, Culine S, Kramar A, et al. Early predicted time to normalization of tumor markers predicts outcome in poor-prognosis nonseminomatous germ cell tumors. J Clin Oncol 2004;22:3868–76.
50 Fizazi K, Pagliaro L, Laplanche A, et al. Personalised chemotherapy based on tumor marker decline in poor prognosis germ-cell tumors (GETUG 13): a phase 3, multicentre, randomized trial. Lancet Oncol 2014;15:1442–50.
51 Fizazi K, Le Teuff G, Fléchon A, et al. Personalized chemotherapy on the basis of tumor marker decline in poor-prognosis germ-cell tumors: updated analysis of the GETUG-13 Phase III trial. J Clin Oncol 2024;42:3270–6.
52 Bachner M, Loriot Y, Gross-Goupil M, et al. 2-^{18}fluoro-deoxy-D-glucose positron emission tomography (FDG-PET) for postchemotherapy seminoma residual lesions: a retrospective validation of the SEMPET trial. Ann Oncol 2012;23:59–64.
53 Cathomas R, Klingbiel D, Bernard B, et al. Questioning the value of fluorodeoxyglucose positron emission tomography for residual lesions after chemotherapy for metastatic seminoma: results of an international global germ cell cancer group registry. J Clin Oncol 2018, JCO1800210.
54 Heidenreich A, Pfister D. Retroperitoneal lymphadenectomy and resection for testicular cancer: an update on best practice. Ther Adv Urol 2012;4:187–205.
55 Conduit C, Hong W, Martin F, et al. A meta-analysis of clinicopathologic features that predict necrosis or fibrosis at post-chemotherapy retroperitoneal lymph node dissection in individuals receiving treatment for non-seminoma germ cell tumors. Front Oncol 2022;12:931509.
56 Heidenreich A, Pfister D, Witthuhn R, Thüer D, Albers P. Postchemotherapy retroperitoneal lymph node dissection in advanced testicular cancer: radical or modified template resection. Eur Urol 2009;55:217–24.