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Management of Stage I–II Testicular Germ Cell Tumours: Current Treatment Paradigm and Future Perspectives

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Introduction

Testicular cancer is the most common solid tumour in males aged 15–44 years, with an estimated 1,300 new cases in Canada in 2024.[1] Over 90% are germ cell tumours (GCTs) originating from spermatocyte precursors, with most arising in the testes.[2] Risk factors include cryptorchidism, gonadal dysgenesis, genetic syndromes such as Klinefelter syndrome, family history, and possibly cannabis use.[2]

This review summarizes the evidence-based diagnosis and management strategies of clinical stage I (CSI) and II (CSII) testicular GCTs used at the Princess Margaret Cancer Centre (PM), highlighting potential clinical pitfalls and future directions.[3]

Workup, Diagnosis, and Staging

Most testicular GCTs present as a palpable testicular mass. Radical orchiectomy serves both diagnostic and therapeutic purposes. Pre- and post-orchiectomy tumour markers (TMs) and computed tomography (CT) chest abdomen pelvis should be obtained.[4]

Staging Classification

Testicular GCTs are staged using the 8th edition of the American Joint Committee on Cancer (AJCC) TNM staging (**Table 1**), which considers primary tumour extent, lymph node involvement, metastatic sites, and post-orchiectomy serum TM levels (S).[5] A potential pitfall is using pre-orchiectomy TMs for staging, which can lead to over- or under-treatment.

Histologic Classification and Tumour Markers

GCTs are classified as pure seminomas (45%) or non-seminomas (NSGCTs, 55%) based on histology to guide prognosis and management.[4] NSGCTs include embryonal carcinoma (EC), yolk sac tumour, choriocarcinoma, and teratoma.[6]

Pure seminomas do not secrete alpha-fetoprotein (AFP) but can produce some human chorionic gonadotrophin in the blood (B-HCG) with syncytiotrophoblast differentiation. Rising AFP should be treated as NSGCT, even if pathology reports pure seminoma. Choriocarcinomas secrete B-HCG, yolk sac tumours secrete AFP, while EC may result in modest elevations of both. Teratomas are generally marker-negative but may secrete some AFP. Due to its low specificity,[7] LDH alone should not guide treatment decisions for early-stage GCT.[4]

A potential clinical pitfall is basing treatment decisions on persistent low-level TM elevations, without considering false positives. AFP may be elevated in the 10–15 ng/mL range due to heterophile antibodies, liver dysfunction, or hereditary factors.[8,9] B-HCG may show false positives due to hypogonadism, heterophile antibodies, marijuana use, or certain medications.[10] A consistent rise in TMs helps differentiate active GCT from false positives.

Management of Stage I-II Seminoma

With appropriate management, patients with CSI-CSII seminoma have long-term survival rates approaching 100% (**Table 2**), highlighting the need to minimize overtreatment and unnecessary toxicity. At PM, patients are managed in the Multidisciplinary Testes Clinic, which involves experienced uro-oncologists, radiation, and medical oncologists.

CSI Seminoma

Approximately 85% of patients with CSI seminoma are cured with orchiectomy alone.[11] Historically, rete testes invasion (RTI) and a primary tumour size of ≥4 cm were considered risk factors for relapse,[12] although not consistently validated.[11] A recent multicentre study refined risk stratification using lymphovascular invasion (LVI) and three categories of tumour size (<2 cm, >2–5 cm, or >5 cm). Five-year relapse rates were 8% in very low-risk, 20% in low-risk, and 44% in high-risk disease.[13] Only 2.3% of patients had high-risk disease.

Another Danish nationwide study identified elevated preorchiectomy b-HCG, LDH, testicular hilum invasion and LVI as independent risk factors, with five-year relapse rates ranging from 6% (no-risk factors) to 62% (all four risk factors), although only 10% of patients had 3 or 4 risk factors. These contemporary models require further external validation.[14]

Active Surveillance for CSI Seminoma

Active surveillance is preferred for patients with CSI seminoma.[4] Most relapses (95%) occur in retroperitoneal lymph nodes (RPLNs), mainly within the first two years (73%).[15] Virtually all patients who relapse can be cured with subsequent radiation or chemotherapy (**Table 2**).

Our published surveillance protocol includes physical examination, including the contralateral testicle, bloodwork, and low-dose CT scans without intravenous (IV) contrast. CTs of the abdomen and pelvis are obtained every 6 months until year 3, CT abdomen only at years 4, 5, 7, and 9, and chest X-ray at year 9 (see **Table 3**).[16]

Use of magnetic resonance imaging (MRI) versus contrast-enhanced CT and fewer (three total) versus more (seven) scans were explored in the Phase III TRISST trial.[17] MRI was non-inferior, but fewer scans resulted in numerically higher, though not statistically significant, rates of Stage ≥IIC relapse (2.8% versus 0.3%) requiring chemotherapy. At PM, we use non-contrast low-dose CT to minimize radiation exposure, eliminate IV access, and shorten imaging time, with excellent outcomes.[18] Due to the numerically higher advanced relapse rate with three scans, and limited accessibility of MRI, our surveillance protocol remains unchanged.

Adjuvant Radiotherapy for CSI Seminoma

Adjuvant radiotherapy (20 Gy in 10 fractions or 25 Gy in 20 fractions to the para-aortic lymph nodes with or without ipsilateral pelvic lymph nodes) reduces the relapse risk for CSI seminoma from 15-20% to 5%.[19-21] However, most patients are cured with orchiectomy alone. Given the high likelihood of cure upon relapse (with radiotherapy or chemotherapy), adjuvant radiotherapy carries risk of overtreatment for most patients and is therefore not the favoured approach. Toxicities include fatigue, nausea, vomiting, peptic ulcer disease, infertility, cardiovascular disease,[22-24]

TNM Staging	Unit	Value		
Primary Tumour (pT)	pTX	Primary tumour cannot be assessed		
	pT0	No evidence of primary tumour		
	pTis	Germ cell neoplasia *in situ*		
	pT1	Tumour limited to testis (including rete testis invasion) without lymphovascular invasion		
	pT1a*	Tumour <3 cm in size		
	pT1b*	Tumour ≥3 cm in size		
	pT2	Tumour limited to the testis (including rete testis invasion) with lymphovascular invasion OR Tumour invading hilar soft tissue or epididymis or penetrating visceral mesothelial layer covering the external surface of tunica albuginea with or without lymphovascular invasion		
	pT3	Tumour directly invades spermatic cord soft tissue with or without lymphovascular invasion		
	pT4	Tumour invades scrotum with or without lymphovascular invasion		
	NX	Regional lymph node cannot be assessed		
Regional Lymph Nodes (pN and cN)	pN0	No regional lymph node metastasis	cN0	No regional lymph node metastasis
	pN1	Metastasis with a lymph node mass ≤2 cm in greatest dimension and ≤5 positive nodes, none >2 cm in greatest dimension	cN1	Metastasis with a lymph node mass ≤2 cm in greatest dimension OR multiple lymph nodes, none >2 cm in greatest dimension
	pN2	Metastasis with a lymph node mass >2 cm but not >5 cm in greatest dimension; or >5 positive nodes, none >5 cm in size; or evidence of extranodal extension of tumour	cN2	Metastasis with a lymph node mass >2 cm but ≤5 cm in greatest dimension OR multiple lymph nodes, any one mass >2 cm but ≤5 cm in greatest dimension
	pN3	Metastasis with a lymph node mass >5 cm in greatest dimension	cN3	Metastasis with a lymph node mass >5 cm in greatest dimension
Distant Metastasis (M)	M1	Distant metastases present		
	M1a	Non-retroperitoneal nodal or pulmonary metastases		
	M1b	Non-pulmonary visceral metastases		
	SX	Serum marker studies not available or performed		
Serum tumour markers (S)	S0	Marker study levels within normal limits		
	S1	LDH <1.5 × normal and HCG (IU/L) <5,000 and AFP (ug/L) <1,000		
	S2	LDH 1.5–10 × normal or HCG (IU/L) 5,000–50,000 or AFP (ug/L) 1,000–10,000		
	S3	LDH >10 × normal or HCG (IU/L) >50,000 or AFP (ug/L) >10,000		

Stage Grouping				
Stage	**Tumour**	**Node**	**Metastasis**	**Serum Factor**
0	pTis	N0	M0	S0
I	pT1-4	N0	M0	SX
IA	pT1	N0	M0	S0
IB	pT2	N0	M0	S0
	pT3	N0	M0	S0
	pT4	N0	M0	S0
IS	Any T	N0	M0	S1-3
II	Any T	N1-3	M0	SX
IIA	Any T	N1	M0	S0
	Any T	N1	M0	S1
IIB	Any T	N2	M0	S0
	Any T	N2	M0	S1
IIC	Any T	N3	M0	S0
	Any T	N3	M0	S1
III	Any T	Any N	M1	SX
IIIA	Any T	Any N	M1a	S0
	Any T	Any N	M1a	S1
IIIB	Any T	N1-3	M0	S2
	Any T	Any N	M1a	S2
IIIC	Any T	N1-3	M0	S3
	Any T	Any N	M1a	S3
	Any T	Any N	M1b	Any S

Table 1. TNM Staging of Testicular GCTs; *adapted from Hamilton et al., CUAJ 2022.*[4]

***** T1a and T1b subclassification applies only to pure seminoma.

Abbreviations: AFP: alpha-fetoprotein, **HCG:** human chorionic gonadotrophin, **LDH:** lactate dehydrogenase.

Histology	Stage	Treatment Modality	RFS	OS
Seminoma	CSI	**Active Surveillance**	**85%**	**100%**
		Adjuvant Radiotherapy	95%	100%
		Adjuvant Chemotherapy (carboplatin)	90.7–97.8%	100%
	CSIIA	**Radiotherapy**	**95%**	**100%**
		Primary RPLND	70-89%	100%[1]
		Chemotherapy	93%	100%
	CSIIB	**Radiotherapy**	**88%**	**100%**
		Primary RPLND	70-89%	100%[1]
		Chemotherapy	**95%**	**100%**
NSGCT	CSI	**Active Surveillance**	**75%**	**100%**
		Adjuvant Radiotherapy	NR	NR
		Adjuvant RPLND	91.60%	100%
		Adjuvant Chemotherapy (BEP)	>95%	100%
	CSIIA	**Primary RPLND**	**80%[2]**	**100%**
		Chemotherapy	>95%	100%
	CSIIB	Primary RPLND	80%[2]	100%
		Chemotherapy	**98%**	**100%**
Seminoma and NSGCT	CSIIC	**Chemotherapy**	**95%**	**96%**

Table 2. Efficacy Outcomes for CSI-CSIIC Testicular GCTs by Treatment Modality; *courtesy of Deepro Chowdhury, MD, FRCPC, Rachel Glicksman, MD, MSC, FRCPC, Robert J. Hamilton, MD, MPH, FRCSC, and Maria Jiang, MD, MSc, FRCPC.*

Preferred treatment approaches at PM are bolded.

[1] Median follow-up 22–32 months only
[2] >90% of patients with S0 disease

Abbreviations: BEP: bleomycin, etoposide, and cisplatin, **CS:** clinical stage, **GCT:** germ cell tumour, **NR:** Not recommended, **NSGCT:** nonseminomatous germ cell tumour, **OS:** overall survival, **RFS:** relapse-free survival, **RPLND:** retroperitoneal lymph node dissection

Time Post Orchiectomy	Month 1	Month 2	Month 3	Month 4	Month 5	Month 6	Month 7	Month 8	Month 9	Month 10	Month 11	Month 12
Year 1						**CT A&P***						**CT A&P** **CXR**** serum LH, FSH, free & total testosterone
Year 2						**CT A&P**						**CT A&P** **CXR** serum LH, FSH, free & total testosterone
Year 3						**CT A&P**						**CT A&P** **CXR** serum LH, FSH, free & total testosterone
Year 4												**CT Abdo*** ONLY** serum LH, FSH, free & total testosterone
Year 5												**CT Abdo ONLY** serum LH, FSH, free & total testosterone
Year 7												**CT Abdo ONLY** serum LH, FSH, free & total testosterone
Year 9												**CT Abdo ONLY** serum LH, FSH, free & total testosterone

Table 3. Princess Margaret Cancer Centre Surveillance Protocol for CSI Seminoma; *courtesy of Deepro Chowdhury, MD, FRCPC, Rachel Glicksman, MD, MSC, FRCPC, Robert J. Hamilton, MD, MPH, FRCSC, and Maria Jiang, MD, MSc, FRCPC.*

CT A&P* (Abdomen &Pelvis) first 3 years
CXR** (Chest X-Ray)
CT Abdo*** (Abdomen) only after 3 years

and secondary malignancies (standardized incidence ratio [SIR]: 1.62, 95% confidence interval [CI]: 1.43–1.83).[25] Additionally, continued surveillance of the abdomen and pelvis (if not treating ipsilateral pelvis) remains necessary.

Adjuvant Chemotherapy for CSI Seminoma

One cycle of adjuvant carboplatin (AUC 7) reduces relapse risk in CSI seminoma comparably to adjuvant radiotherapy (see **Table 2**). The MRC TE19/EORTC 30982 study showed long-term relapse-free survival (RFS) for both modalities, with a lower risk of contralateral testicular relapse in the carboplatin group (relapse rate in contralateral testicle 0.2% versus 1.2% in favour of adjuvant carboplatin).[26] The SWENOTECA study reported relapse rates of 15.5% with surveillance versus 9.3% with adjuvant carboplatin in patients with one or more risk factors.[27] Despite a risk-adapted strategy, adjuvant carboplatin likely leads to overtreatment and unnecessary toxicity, including fatigue, myelosuppression, infection, nausea, and vomiting. Secondary malignancies (SIR: 0.96, 95% CI: 0.26–2.45) and rarely cardiovascular disease (SIR: 1.44, 95% CI: 0.39–3.69).[28] Therefore it is not the preferred approach at PM. Relapses after adjuvant carboplatin may also have more aggressive tumour biology.[29]

CSII Seminoma

Up to 30% of CSIIA seminomas have benign (pN0) RPLNs, which may regress spontaneously.[30] Short-interval imaging (6–8 weeks) and TM reassessment can help avoid this pitfall.[4,31] Treatment should proceed only if metastatic RPLNs are unequivocal (enlarging, rising TMs or confirmed on biopsy).[6]

Definitive Radiotherapy for CSIIA or CSIIB Seminoma

Radiotherapy delivered to the para-aortic lymph nodes and ipsilateral pelvic lymph nodes known as the “dog-leg” or “modified dog-leg” approach using 3-dimensional conformal radiotherapy (20–25 Gy to the entire volume with a boost to gross disease to a total dose of 30–36 Gy) is preferred for CSIIA/B seminoma due to lower toxicity compared to chemotherapy, and excellent long-term outcomes (**Table 2**).[32] National Comprehensive Cancer Network (NCCN) guidelines suggest a 3 cm cut-off in the trans-axial axis,[33] but recent data suggest low relapse rates with radiation even with RPLNs >5 cm in select patients.[32] Treatment decisions require multidisciplinary input, and consideration of patient preferences.

Most relapses following radiotherapy (>95%) are out-of-field,[34] and can be successfully treated with 3 cycles of chemotherapy, achieving 10-year overall survival (OS) rates of 91%.[34] However, when both radiotherapy and chemotherapy are used, the risk of long-term toxicities, particularly secondary malignancies, is likely higher.[35]

Chemotherapy for CSIIA or CSIIB Seminoma

Relapse rates after chemotherapy are numerically lower (8-14%) than radiotherapy (11–21%), particularly in CSIIB disease (5% vs. 12%).[36] Given its more unfavourable toxicity profile, chemotherapy is typically reserved for patients with rising TMs or bulky CSIIB disease, the definition of which is not standardized.

The standard chemotherapy regimen is three cycles of bleomycin, etoposide, and cisplatin (BEP) or four cycles of etoposide-cisplatin (EP), if the patient is unable to tolerate bleomycin.[6] Sperm banking should be offered prior to chemotherapy.[37] Acute toxicities include fatigue, alopecia, nausea, vomiting, myelosuppression, neutropenic infections, renal dysfunction, skin toxicity, and venous thromboembolism. Bleomycin-induced lung injury occurs in up to 10% of patients and can rarely be fatal.[38] Risk factors include smoking history, pre-existing lung disease, age >50 years, and renal impairment.[39] Hearing loss and peripheral neuropathy can occur in 20–40% of patients.[39] Other long-term risks include tinnitus, chronic kidney disease, infertility, secondary malignancy, cardiovascular disease, Raynaud’s phenomenon, and avascular necrosis of the hip.

The SAKK 01/10 Phase II trial recently evaluated the combination of one cycle of carboplatin (AUC 7) followed by radiotherapy, and reported 3-year progression-free survival (PFS) of 93.7%, which did not meet the pre-specified 95% target.[40] Given these data, along with concerns for increased long-term toxicity with combination therapy, this approach is not adopted at PM.

Primary RPLND for CSIIA and CSIIB Seminoma

Three Phase II studies, SEMS (n=55), PRIMETEST (n=33), and COTRIMS (n=30), have evaluated retroperitoneal lymph node dissection (RPLND), mainly open surgery, in marker-negative CSIIA and CSIIB seminoma.[41-43] With a median

follow-up of 22–32 months, two-year relapse-free survival (RFS) ranged from 70% to 89%, with most recurrences occurring out-of-field (>90%). The PRIMETEST trial was terminated early due to high relapse rates (30%)[42]; however, all relapses were successfully salvaged with additional surgery or chemotherapy.

Across all three studies, grade >3 short-term complications were observed in 3.6–13% of patients, and included paralytic ileus, chylous ascites, lymphoceles requiring drainage, and pulmonary emboli. Rates of anejaculation were ≤10%, and other long-term surgical complications were rare.[41] The American Urology Association (AUA) now recognizes RPLND as an option for select patients with RPLNs ≤3 cm who wish to avoid chemotherapy or radiotherapy.[43] However, higher relapse rates than standard of care, variations in patient selection, and surgical technique across centres remain concerns. PM has not adopted this approach outside of a clinical trial setting, pending data from larger studies with longer follow-up, consistent with the European Urological Association (EAU) recommendations.[31] The THERATEST trial (NCT06309745) is ongoing and compares RPLND to radiotherapy in seminoma with RPLN <3 cm.

Clinical Stage IIC (CSIIC) Seminoma

CSIIC seminoma is treated with chemotherapy according to the International Germ Cell Cancer Collaborative Group (IGCCCG) risk classification.[45] Post-chemotherapy residual masses ≥3 cm may contain viable tumour in up to 30% of cases based on historical data.[46] Fludeoxyglucose positron emission tomography (FDG-PET) has been studied in this setting[47] but is used infrequently due to its low positive predictive value.[4]

Management of Stage I-II NSGCT

CSI NSGCT

Approximately 75% CSI NSGCT are cured with orchiectomy alone.[48] Risk factors include lymphovascular invasion (LVI) (30%) and EC-predominant disease (50%).[49] About 75% of relapses occur in RPLNs.[50] Data from the Danish Testicular Cancer Registry recently showed hilar soft tissue invasion, tumor size (log 2), LVI and EC (absent vs <50% vs ≥50%) were independent risk factors for relapse, with 5-year relapse risk ranging from <5% (no risk factors) to >85% (all 4 risk factors).[51] External validation is warranted.

Active Surveillance for CSI NSGCT

Active surveillance is the preferred approach for CSI NSGCT4 (the PM surveillance protocol is included in **Table 4**).[48] Even in high-risk cases, 50% are cured with orchiectomy alone.[50] Most relapses (90%) occur within the first two years,[15] and are effectively treated with primary RPLND or chemotherapy.[50,52]

Adjuvant Chemotherapy for High-Risk CSI NSGCT

Some centres offer a risk-adapted approach using one cycle of adjuvant BEP for CSI NSGCT with LVI and/or predominant EC histology, reducing relapse risk to <5%.[53] Two cycles of adjuvant chemotherapy yield similar RFS but with increased toxicity and are generally not recommended.[31] Up to 37% of relapses after adjuvant BEP occur beyond 2 years, underscoring the need for long-term surveillance.[54]

The decision to use adjuvant chemotherapy should balance its efficacy in reducing relapse (and risk of requiring 3 cycles of chemotherapy subsequently) against the chance of avoiding chemotherapy and its potential toxicities altogether.[55] There is also some concern that relapses following adjuvant chemotherapy may be more treatment-resistant,[54] although data is limited.

RPLND for High-Risk CSI NSGCT

A German Phase III study compared RPLND with one cycle of adjuvant BEP. Following RPLND, 18% of patients received additional adjuvant BEP. The 12-year relapse rate was higher with RPLND (8.4%) than with adjuvant BEP (1.6%).[56] Only about one-third of patients had pathologic nodal involvement at the time of surgery, highlighting the significant risk of over-treatment.[57] RPLND is rarely used at PM in this context.

CSII NSGCT

Patients with CSIIA disease should undergo short-interval CT scans (6–8 weeks) and serial TMs to differentiate benign lymphadenopathy (20–30%), teratoma, and viable GCT.[30] If TMs remain negative, shrinking lesions are likely benign and can be observed. Persistent, slow-growing cystic lesions may indicate teratoma, warranting RPLND.

Time Post Orchiectomy	Month 1	Month 2	Month 3	Month 4	Month 5	Month 6	Month 7	Month 8	Month 9	Month 10	Month 11	Month 12
Year 1	Markers*	Markers		Markers CT A&P*** CT Thorax		Markers		Markers CT A&P CT Thorax		Markers		**Markers** **CT A&P** **CT Thorax** Serum LH, FSH, free & total testosterone
Year 2		Markers		Markers		Markers		Markers		Markers		**Markers** **CT A&P** **CT Thorax** Serum LH, FSH, free & total testosterone
Year 3				Markers				Markers				**Markers** Serum LH, FSH, free & total testosterone
Year 4						Markers						**Markers** Serum LH, FSH, free & total testosterone
Year 5												**Markers** **CT A&P** **CT Thorax** Serum LH, FSH, free & total testosterone
Transition to primary care after 5 years. No ongoing imaging/labs required. Physical surveillance of remaining testis.												

Table 4. Princess Margaret Cancer Centre Surveillance Protocol for CSI NSGCT; *courtesy of Deepro Chowdhury, MD, FRCPC, Rachel Glicksman, MD, MSC, FRCPC, Robert J. Hamilton, MD, MPH, FRCSC, and Maria Jiang, MD, MSc, FRCPC.*

Markers* (HCG, AFP, LDH)
CT A&P*** (CT Scan of Abdomen and Pelvis)

Primary RPLND for CSIIA and CSIIB NSGCT

For marker-negative CSIIA NSGCT, primary RPLND is associated with a higher relapse rate (20%) than chemotherapy (<5%); however, it avoids chemotherapy toxicities[58] and is the preferred approach. Relapses post-RPLND are mostly out-of-field when performed at experienced centres,[4] and are highly curable with chemotherapy.

For CSIIB NSGCT, relapse rates after primary RPLND reach 50%,[59] and chemotherapy is often preferred. No standardized criteria exist for selecting primary RPLND versus chemotherapy[60] but TM levels, lymph node (LN) size and distribution may help guide decision making. Patients with an unequivocal rise in TM or rapidly progressing disease should receive chemotherapy.

After RPLND, two cycles of adjuvant chemotherapy can reduce relapse rates (e.g., N2 disease from ≥50% to <5%).[61] However, this undermines the goal of primary RPLND, which is achieving cure without chemotherapy.

Chemotherapy for CSIIA, CSIIB, and CSIIC NSGCT

For CSIIA and CSIIB NSGCT, chemotherapy has lower relapse rates (<5%) than RPLND at the cost of increased short- and long-term toxicity.[58] CSIIC disease, regardless of TM status, should be treated with chemotherapy (see **Table 2**).[62]

Post-chemotherapy, residual masses over 1 cm warrant RPLND to remove any teratoma (to avoid growing teratoma syndrome or somatic transformation) or viable chemo-resistant GCT.[4]

Future Directions

Molecular biomarkers such as micro RNAs (miRNAs), particularly miR371,[63] have shown excellent sensitivity (80–100%) and specificity (90–100%),[64] and may be particularly valuable for detecting marker-negative GCTs.[65]

For metastatic NSGCT treated with chemotherapy, miR371 has shown prognostic value[66-68] and promising ability to assess residual masses (negative predictive value of 100% in NSGCT <3 cm).[66] However, miR371 has limited ability to detect teratoma[69]; with miR375 co-testing this may be improved.[70] Further prospective studies, such as the ongoing SWOG S1823 trial (NCT04435756), are needed. Circulating tumour DNA (ctDNA)[71] also shows potential utility in detecting molecular residual disease post-treatment.[72]

Conclusions

CSI and CSII testicular GCTs are highly curable. Management strategies are tailored to histology, tumour burden, stage, and patient preferences, with an emphasis on minimizing treatment toxicities. Multidisciplinary evaluation is essential, and treatment at experienced centres optimizes outcomes. Ongoing research, including RPLND in CSIIA-CSIIB seminoma and biomarkers, such as miRNA and ctDNA, may enable personalized treatment strategies pending confirmatory data.

For other surveillance protocols including post primary RPLND, radiation or chemotherapy, please refer to page 20–26 **here**.

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