SECTION 1: INTRODUCTION

Testicular germ cell cancer (TGCC) accounts for only 1%–2% of tumours in men overall, but is the most commonly diagnosed malignancy in young men [1]. The incidence of TGCC varies by ethnic origin, with the highest rates reported in developed countries and the lowest in developing countries [2]. The European Society for Medical Oncology (ESMO) Clinical Practice Guideline (CPG) provides high-level guidance on optimal strategies for the diagnosis, treatment and follow-up of patients with TGCC [3, 4]. However, some issues relating to the optimal management of patients with TGCC remain controversial and warrant further discussion and clarification. Accurate diagnosis of stage and type of testicular cancer is also a concern since testicular cancers are one of the most diverse areas of human pathology and pathologists may see few tumours in a year [5-8].

Regarding the treatment of TGCC, the optimal use of adjuvant chemotherapy in stage I disease remains an area of controversy. Defined strategies to accurately identify those patients who require adjuvant chemotherapy could therefore protect low-risk patients from the toxicities associated with over-treatment. The optimal treatment approach for stage IIA and IIB seminoma and non-seminoma is also a matter of debate and is discussed in this manuscript. Other areas which are currently only supported by marginally higher levels of evidence, but nonetheless often require treatment decisions in clinical practice, are issues of post-chemotherapy surgery, salvage chemotherapy and salvage and/or desperation surgery. Finally, given the excellent prognosis of most patients with TGCC, high quality follow-up care and survivorship care plan recommendations are crucial. Indeed, the long-term global health-related quality of life (HRQoL) of testicular germ cell cancer survivors (TGCCSs) is
similar to that of the general population [9], although chronic side effects can adversely affect HRQoL, particularly after chemotherapy [10, 11]. However, the optimal follow-up of TGCCSs has not yet been defined and is an unmet need.

Collectively, these and other topics represent points in the care pathway where a consistent approach between physicians is lacking. Given these unresolved and complex issues, the aim of this consensus conference was to produce multidisciplinary evidence-based guidelines on selected clinically relevant questions that complement the existing ESMO CPG where possible and facilitate an optimal and consistent approach to the diagnosis, treatment and follow-up of patients with testicular cancer.

SECTION 2: METHODS

Leading up to the consensus conference, all five working group chairs developed clinically relevant questions surrounding their given subject area, which were subsequently discussed with their group members and modified as needed. Key literature relevant to the subject areas and questions were then reviewed by each working group prior to the consensus conference in order to draft preliminary recommendations. No systematic literature search was undertaken. During the conference, preliminary recommendations were discussed and prepared for voting by the five working groups in parallel breakout sessions. The level of evidence and strength of each recommendation proposed by the group was defined based on the ‘Infectious Diseases Society of America-United States Public Health Service Grading System’, as shown in Table 1 [12]. Recommendations from all working groups were then presented to the full expert panel for deliberation and amendment, as needed. Finally, a vote was carried out to establish the level of agreement among the expert panel. Members of the panel were given the opportunity to abstain from the voting process, to allow for cases where
they felt they did not have enough expertise in the area to agree or disagree, or if they had any conflict of interest which could influence their vote.

Results from this consensus conference, including all agreed recommendations and a summary of evidence supporting each recommendation, are described in this article. A summary of all recommendations is included in supplementary Table S1, available at Annals of Oncology online.

The draft manuscript was reviewed by two representatives of patient advocacy groups from France (Olivier Jerome, President of CERHOM, Villejuif, France) and Norway (Hans Sverre Hansen-Gaard, TGCCS, Oslo, Norway). The final manuscript was reviewed and approved by all ESMO consensus panel members.

SECTION 3: RESULTS

Diagnostic work-up and patient assessment

5. Old and new biomarkers

Assessment of the serum biomarkers α-fetoprotein (AFP), beta-human chorionic gonadotropin (β-hCG) and lactate dehydrogenase is a prerequisite for the staging of TGCC, monitoring of treatment outcomes and early detection of TGCC relapse [13, 14]. Drawbacks of these classical biomarkers include having a diagnostic sensitivity of only 60%–80% and a wide variation in marker expression levels in different histologic subgroups and clinical stages [15, 16]. Potential new biomarkers include the microRNAs miR-371-3 and miR-302/367 cluster, which are present in TGCC tissue [17, 18] and are also known to circulate in serum [19, 20]. These microRNAs can be measured using the quantitative polymerase chain reaction method. Currently, results from four pilot studies have suggested that serum levels of
miR-302/367 and miR-371-3 are promising biomarkers of TGCC [19, 21-23]. More recently, a prospective study in Germany in 166 patients with TGCC and 106 healthy controls has indicated that miR-371a-3p may be a highly useful marker, featuring a sensitivity of 86.3% (95% confidence interval [CI]: 79.7-90.4) and a specificity of 92.5% (95% CI: 89.0 -95.9) [24]. Serum levels of miR-371a-3p were significantly higher in patients with metastatic disease than in those with localised disease. In addition, serum levels of miR-371a-3p correlated with tumour size in stage I disease and decreased to normal after completion of treatment. Increasing serum levels of miR-371-3p were associated with treatment failure, and high levels were observed in patients with disease relapse. Importantly, teratoma and germ cell neoplasia in situ (GCNIS) do not appear to express these particular microRNAs, as shown in two recent studies [24, 25]. Thus, miR-371a-3p outperforms the classical biomarkers and represents a highly sensitive and specific new biomarker for TGCC. While this marker deserves attention by clinicians managing patients with TGCC, particularly given that a serum diagnostic test for miR-371a-3p is expected to be introduced soon into clinical practice [26], issues around laboratory standardisation and availability of the test must be resolved before this new biomarker can be recommended for routine clinical use.

Post-chemotherapy surgery, salvage chemotherapy, salvage and desperation surgery, and special topics

23. When is post-chemotherapy retroperitoneal lymph-node dissection (PC-RPLND) indicated?

The most important consideration for post-chemotherapy surgery is whether a complete resection of residual radiological lesions is possible. Patients do not usually benefit from debulking or incomplete resections. Removal of the residual mass only (lumpectomy) is
associated with a risk of incomplete resections and should not be performed. Post-chemotherapy surgery should therefore only be performed at high-volume centres with multidisciplinary teams who perform this procedure regularly. Patients with residual lesions after chemotherapy should be referred to these centres [27, 28].

A bilateral open PC-RPLND remains the standard of care, based on mapping studies of nodal deposits and retrospective studies [29-33]. The field and extent of surgery should be based on the pre-chemotherapy pattern of metastases, and a nerve-sparing technique is recommended whenever possible. In patients presenting with infra-hilar nodal metastatic disease, the bilateral resection template, when indicated, should include infra-hilar, pre-caval and para-caval nodes medial to the right ureter, and retro-caval, inter-aorto-caval, pre-aortic, retro-aortic and para-aortic nodes medial to the left ureter, as well as the ipsilateral iliac nodes. In patients presenting with nodal metastatic disease outside the classical template, all sites outside the template should be included in the resection. This particularly applies to patients with supra-hilar and pelvic disease.

A more limited dissection, defined as a ‘unilateral’ template, may be an alternative to a full bilateral resection. Eligible patients include those with resectable residual lesions <5 cm in the maximum axial diameter within the planned template, and those with residual retroperitoneal nodal disease within the pre-chemotherapy primary landing site of the tumour-bearing testis. [34-37]. Minimally-invasive laparoscopic RPLND should only be performed in high-volume, multidisciplinary testicular cancer centres with additional laparoscopic expertise.

Adjunctive resections in addition to PC-RPLND are required in up to 20% of patients, and may include nephrectomy, vascular resection and/or other intra-abdominal visceral resections [38-40]. The aim of these procedures is complete resection of all residual disease. Where this does not appear feasible due to multi-focality or anatomical difficulty, incomplete resections
may not be beneficial. The combination of thoracic and retroperitoneal resections is relatively common. The timing and sequence of combined resections should be based on the location of the highest volume of residual disease [41]; usually, the first site of resection is in the retroperitoneum. The histology of residual disease in different organs may be discordant [42-44]. Therefore, in the presence of resectable disease in the retroperitoneum and thorax, lesions in the thorax should also be resected.

In patients with bilateral thoracic disease, the initial resection should be unilateral. A discordance rate of up to 20% has been reported [45]. Decisions for contralateral pulmonary resections are complex and should be based on the number of lesions, their size and location. Surgery for liver lesions may involve wedge resection or full lobectomy and may be performed at the time of RPLND or as a separate procedure [46, 47].

Patients with necrosis or complete resection of differentiated teratoma require no further treatment. The benefit of adjuvant treatment with two cycles of cisplatin-based chemotherapy in patients with an International Germ Cell Consensus Classification Group classification of ‘intermediate risk’ or ‘poor risk’ at initial presentation, those with >10% viable tumour in the resection specimen, and/or in patients with incomplete resection, has recently been questioned as the value of complete resection of residual masses is more relevant for improving outcome than any adjuvant chemotherapy [48, 49].

A small number of patients will experience radiological progression during chemotherapy despite tumour marker decline or normalisation (so called ‘growing teratoma’). If possible, chemotherapy should be completed as planned followed by resection of all radiological lesions [50]. Salvage chemotherapy is not indicated in patients with ‘growing teratoma’.

Late relapses are defined as evidence of new lesions, or sequentially increasing serum tumour markers (AFP or HCG), more than 2 years after ≥3 cycles of cisplatin-based chemotherapy.
Viable cancer and/or somatic-type malignant transformation that do not respond well to chemotherapy are more frequent in late relapse than in early relapses [51-53]. Available evidence emphasises the central role of surgery in these patients [54, 55]. There is currently no evidence to show that chemotherapy, either before or after complete resection, improves the overall outcome. However, conventional-dose chemotherapy and high-dose chemotherapy have both been associated with long-term remissions in a small proportion of patients with unresectable late relapses [56, 57].

**Survivorship and follow-up schemes**

26. **How can post-therapeutic psychosocial issues be minimised, and HRQoL protected?**

**HRQoL: emotional and psychosocial issues.**

Long term global HRQoL is similar between TGCCSs and the general population, regardless of the applied treatment [9]. However, chronic side effects, particularly after chemotherapy (including peripheral neuropathy, Raynaud’s syndrome, hearing loss and chronic fatigue [CF]), impact negatively on global, physical and mental HRQoL [10, 11].

Patients with a ‘helpless-hopeless’ coping style and limited social support experience poorer mental HRQoL, anxiety and depression [58]. In comparison with the general population, long-term TGCCSs express higher levels of anxiety; young age and certain socio-economic factors (including unemployment, low educational level and alcohol problems) can increase anxiety and stress, which in turn reduce HRQoL [59]. Some patients experience fear of recurrence in the long term, especially those with a medium educational level, traumatic cancer-related stress symptoms and neurotic personality [60]. Although there is currently no evidence of testicular cancer leading to subsequent unemployment or reduced work
engagement, poorer health and reduced work ability related to physical and psychological symptoms after cancer treatment is reported for a subgroup of patients [61, 62].

**Quality of life and post-therapeutic psychosocial issues.**

TGCCSs are more likely to have impaired sexuality (ejaculation and erectile disorders, reduced sexual interest and enjoyment) compared with healthy men of the same age [63-65]. Ejaculation impairment is usually caused by damage to sympathetic nerves after RPLND and may reduce sexual satisfaction [65]. Overall sexual problems are associated with older age, lack of a partner, high anxiety and change in body image [64, 65].

Self-reported cognitive complaints are common among TGCCSs and are linked with CF (i.e. fatigue above a certain level after a median observation time, as defined by the fatigue scale used) and emotional distress [66]. Recent studies have also identified objective cognitive impairments (mainly in verbal learning, memory and processing speed) after treatment, with younger age and a higher number of chemotherapy cycles associated with a greater incidence of overall decline in cognitive function [67, 68].

Most patients with testicular cancer have at least one supportive care need, including physical care, lifestyle programme support, attitude towards self-management (including psychological support) and eHealth [69, 70]. Recent survivorship care plans among cancer survivors have generally not demonstrated improvements in HRQoL, satisfaction or distress [71]. Nevertheless, healthcare professionals should inform patients about the potential late negative effects of treatment, and endeavour to identify psychological distress early. A healthy lifestyle should always be promoted. Future research will examine the potential benefit of TGCCS-specific patient care plans.
27. How should fatigue be identified, prevented and treated?

**Chronic fatigue**

CF has been described as one of the most common and distressing adverse effects of cancer and its treatment [72]. CF should be regularly assessed using validated questionnaires [73]. Commonly used fatigue questionnaires include the Fatigue Questionnaire (FQ) [74], Functional Assessment of Cancer Therapy - Anaemia and Fatigue (FACT-An) [75] and the EORTC Quality of Life Questionnaire (QLQ C30) (fatigue subscale) [76].

CF (i.e. fatigue above a certain level after a median observation time as defined by the fatigue scale used) is more common in TGCCSs (16%) than in the general male population (10%) [77-79]. The prevalence of CF increases with age in the general male population, from 9.6% to 12.2% in the age cohorts 40–49 and 50–59 years, respectively [79], with a substantial increase in CF from 12 to 19 years after treatment combined with biochemical hypogonadism [80]. Moderate or high physical activity appears to have a preventive and therapeutic effect [80]. CF has been mitigated by cognitive behavioural therapy and mindfulness-based cancer recovery [81]. Healthcare professionals should strive to prevent CF through early detection of fatigue and lifestyle interventions throughout treatment and follow-up of co-morbid conditions. Testosterone substitution may be considered. CF may dramatically impair HRQoL and work ability, and the disturbing increase in CF among TGCCSs and its association with partly treatable side effects underlines the importance of continued long-term assessments of TGCCSs.
28. How can the risk of ototoxicity and neurotoxicity be minimised?

**Ototoxicity.**

Ototoxicity and neurotoxicity are both important toxicities related to cisplatin treatment as well as ageing, and may substantially impair HRQoL [82]. After treatment of metastatic disease with standard-dose bleomycin/etoposide/cisplatin (BEP) regimens, 20%–25% of patients report long-term hearing impairment and tinnitus [83, 84]. When objectively measured by audiograms covering frequencies up to 12 kHz, and without any comparison with age-matched controls, only 20% of patients have normal audiograms [85]. However, daily life hearing ability is associated with findings on audiograms up to only 6–8 kHz [86]. The cumulative dose of cisplatin has consistently been shown to be a risk factor for ototoxicity, and scheduled administration with cisplatin 100 mg/m$^2$ as 20 mg/m$^2$/day over 5 days, as opposed to 50 mg/m$^2$/day over just 2 days, reduces the risk of hearing impairment and tinnitus [85, 87, 88]. Cisplatin-induced ototoxicity may become an increasing problem with increasing age-related hearing loss (premature presbycusis). Various genetic polymorphisms have been associated with an increased risk of ototoxicity [89-93], but these findings have not influenced clinical practice. Other possible risk factors include severe noise exposure prior to treatment, co-treatment with other ototoxic agents (such as aminoglycosides) and abnormal renal function [94, 95]. Drugs to prevent ototoxicity, or therapy to relieve symptoms, have not yet been identified.

**Neurotoxicity.**

Self-reported chemotherapy-induced peripheral sensory neuropathy (CIPN) has been reported in 5% of patients after one cycle of BEP [96], and in 25%–35% of patients with germ cell cancer treated with three to four cycles of BEP [87]. The risk of neuropathy increases with cumulative cisplatin doses exceeding 300 mg/m$^2$, and almost every patient receiving doses
higher than 500–600 mg/m² will experience neurotoxicity [97]. Although patient-reported symptoms are often partly reversible, not least due to patients’ adjustment to the problem (‘response shift’), they persist in 20%–25% of patients after 2 years of follow-up [83]. The risk of CIPN has been associated with polymorphisms in glutathione S-transferases and excision repair cross-complementation group 1 protein (ERCC1) [98-100], and long-term neurotoxicity has been associated with residual serum platinum levels [101]. However, these findings have not led to new management strategies.

Various neuroprotective therapies have been tested [102]. Vitamin E has shown some effect [103, 104] but results could not be replicated in larger studies [105]. Promising results were achieved with amifostine, but as this drug has acute side effects and may also reduce the anticancer potency of chemotherapy, it is not routinely used [106]. In one study, treatment with duloxetine was associated with positive effects on long-term CIPN; however, the majority of patients in this study were experiencing oxaliplatin-induced CIPN [107]. Other potentially therapeutic agents include tricyclic antidepressants and anticonvulsants [108].

Thus, although symptomatic ototoxicity and neurotoxicity are currently unpreventable complications of cisplatin-based chemotherapy, they should generally not influence curative treatment. Nevertheless, patients should be informed about the risk of long-term ototoxicity and neurotoxicity prior to treatment.

29. Which TGCCSs should be offered testosterone replacement therapy?

Leydig cell dysfunction and testosterone.

Primary biochemical hypogonadism (low testosterone and high luteinising hormone [LH] levels) is prevalent in 5%–13% of patients after orchiectomy, increasing to 11%–27% after subsequent chemotherapy [109-112]. Furthermore, mean levels of LH are higher in
chemotherapy-treated patients than in stage I patients after orchiectomy only, while mean testosterone levels are either comparable or decreased, suggesting compensated (high LH, normal testosterone) or uncompensated (high LH, low testosterone) chemotherapy-induced damage to Leydig cells [109-112].

Sprauten et al. demonstrated that TGCCSs had lower testosterone and higher LH and follicle-stimulating hormone levels than healthy controls of a similar age at a median of 11 and 18 years after orchiectomy [113]. Importantly, the proportion of biochemically hypogonadal TGCCSs seemed to increase between the 11-year follow-up and the 18-year follow-up [113].

Symptoms of hypogonadism include decreased sexual function (often including loss of morning and spontaneous erections), a more sedate lifestyle and decreased bone health [114]. High body weight and the metabolic syndrome also seem to be related to testosterone levels in TGCCSs, but it is unclear whether obesity and the metabolic syndrome develop as a result of hypogonadism or vice versa [114].

Potential benefits of testosterone replacement therapy in young men with subclinical biochemical hypogonadism, or only mildly decreased testosterone levels, are uncertain. Howell et al. evaluated testosterone replacement in a randomised placebo-controlled trial among survivors of haematological malignancies with testosterone levels of <20 nmol/L (i.e. in the lower half of the normal range) [115]. They demonstrated a significant reduction in fatigue and low-density lipoprotein cholesterol, but no change in bone mineral density or other lipids, in the testosterone replacement group compared with the placebo group. Studies of testosterone supplementation in TGCCSs are ongoing.

In conclusion, the effect size of testosterone replacement in TGCCSs with low or low-to-normal testosterone levels remains unclear. Whether the effects of testosterone replacement therapy are sustained during long-term use, and whether the beneficial effects outweigh any
negative effects, are also unknown and warrant further investigation. It is also unclear if testosterone replacement therapy is a valuable treatment strategy in the management of obesity and the metabolic syndrome in TGCCSs. The current recommendation is that TGCCSs with repeatedly low testosterone levels and clinical symptoms of hypogonadism should be offered testosterone replacement therapy for a trial period of 3–6 months.

30. How can the risk of cardiovascular disease (CVD) be reduced in TGCCSs?

CVD, in particular coronary artery disease, is one of the most serious late effects after treatment for testicular cancer. Most studies have shown a 2–3-fold increase in risk for CVD in men previously treated with cisplatin-based chemotherapy or radiotherapy, compared with men treated with surgery only or the general population [116-118]. The risk is increased beyond ten years of follow-up, and risk prediction tools such as Framingham or SCORE, applied among all TGCCSs, have failed to identify high-risk individuals, likely due to their limited follow-up period (only 5 or 10 years) [119].

The absolute CVD risk 20 years after treatment is 6%–10%, with a particularly high risk (20%) after combined chemotherapy and radiotherapy [118]. The elevated risk of CVD in TGCCSs is thought to be primarily mediated by increases in CVD risk factors such as hypertension, obesity, hypercholesterolaemia, diabetes, smoking and physical inactivity [120]. The clustering of CVD risk factors into the metabolic syndrome [121] is a possible link between cytotoxic treatment and later development of CVD [122]. Low testosterone levels, which are relatively common in TGCCSs, are related to increased risks for the metabolic syndrome and CVD [120]. In addition, platinum is detectable in serum up to 20 years after treatment [123], and circulating platinum may continuously damage the endothelium, resulting in an accelerated atherosclerotic process [124].
Healthcare providers should focus on the prevention of CVD from the start of cytotoxic treatment and throughout follow-up. Early and repeated counselling about the importance of a healthy lifestyle including smoking cessation, keeping a healthy diet and being physically active, play an important role in reducing the potential CVD risk among TGCCSs. Lifelong check-ups for CVD risk factors should be performed every 2 years, including measurements of blood pressure, weight, sex hormones, lipids and glucose [125]. Hypertension, hypercholesterolaemia, diabetes and hypogonadism should be treated. All patients with testicular cancer should have a survivorship care plan in place, including tools for acquiring and maintaining a healthy lifestyle.

31. How can the risk of a second cancer and its consequences be reduced in TGCCSs?

Second non-germ cell cancer.

A significantly increased risk of a second cancer (relative risk ~1.5–2.1) represents one of the most feared long-term adverse effects after treatment for testicular cancer [126]. Before the introduction of cisplatin-based chemotherapy, most second cancers were localised below the diaphragm (pancreas, ventricle, bladder), within or close to the radiation fields [127, 128]. A significant dose-relationship has been demonstrated between the target radiation dose and incidence of a second cancer [129, 130]. The combination of radiotherapy and older chemotherapy regimens also increase the risk of a second cancer [131]. The increased risk of a second solid malignancy becomes measurable 10–15 years after diagnosis and remains elevated for at least 35 years after initial treatment [131].

Following the gradual decline of radiotherapy as a treatment modality for testicular cancer since the mid-1970s, the pattern of second cancer development has changed. Leukaemia (mainly acute myeloid leukaemia) is most often diagnosed within the first 10 years after
cisplatin-based chemotherapy and is associated with the cumulative dose of etoposide administered [132, 133]. The few published studies that have looked at the long-term incidence of solid tumours in patients treated with cisplatin-based chemotherapy indicate an increased risk of urological cancer and probably thyroid and lung cancer [128, 134]. However, larger studies are needed to clarify the risk of a second cancer in relation to treatment received for the primary testicular cancer. The prognosis of patients with post-testicular cancer second non-germ cell cancers is similar to that of patients with the same non-germ cell cancers as their first lifetime malignancy [135].

**Second germ cell testicular cancer.**

Between 2% and 5% of patients with testicular cancer develop a germ cell tumour in the contralateral testicle, most frequently on the basis of GCNIS [136-138]. It is not clear whether early histological demonstration and treatment (most often radiotherapy) of this pre-invasive stage is of overall clinical benefit for the individual patient [139].

Four or more cycles of cisplatin-based chemotherapy delayed or prevented the development of an invasive testicular germ cell cancer, halving the rate at 5 years [140, 141].
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