



Transplantation and Cellular Therapy

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Survivorship

International Recommendations for Screening and Preventative Practices for Long-Term Survivors of Transplantation and Cellular Therapy: A 2023 Update

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As hematopoietic cell transplantation (HCT) and cellular therapy expand to new indications and international access improves, the number of HCTs performed annually continues to rise. Parallel improvements in HCT techniques and supportive care entails more patients surviving long term, creating further emphasis on survivorship needs. Survivors are at risk for developing late complications secondary to pretransplantation, peritransplantation, and post-transplantation exposures and other underlying risk factors. Guidelines for screening and preventive practices for HCT survivors were originally published in 2006 and then updated in 2012. An international group of experts was convened to review the contemporary literature and update the recommendations while considering the changing practices of HCT and cellular therapy. This review provides updated pediatric and adult survivorship guidelines for HCT and cellular therapy. The contributory role of chronic graft-versus-host disease (cGVHD) to the development of late effects is discussed, but cGVHD management is not covered in detail. These guidelines emphasize the special needs of patients with distinct underlying HCT indications or comorbidities (eg, hemoglobinopathies, older adults) but do not replace more detailed group-, disease-, or condition-specific guidelines. Although these recommendations should be applicable to the vast majority of HCT recipients, resource constraints may limit their implementation in some settings.

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INTRODUCTION

Hematopoietic cell transplantation (HCT) is a potentially lifesaving treatment for many diseases. With expansion to new indications, better international access, and improved outcomes, the population of long-term HCT survivors is rapidly growing [1–6]. However, survivors face serious long-term medical issues, psychosocial challenges that often impact quality of life (QoL), and decreased life expectancy [7–10]. Consequently, prevention and recognition of late effects, followed by prompt intervention, are crucial to improving long-term outcomes in survivors. Additionally, there is an urgent need to better understand the biology and patient experience of HCT's late effects, as well as the ideal health care delivery infrastructure for managing this growing population [11–17].

Previous guidelines for long-term survivors of HCT were produced as collaborative efforts by multiple societies in 2006 and 2012 [18–23]. To update guidelines and provide further geographic

diversity, we convened a working group of experts from multiple international organizations as well as patient advocates (Figure 1). We set out to design these recommendations to adapt to evolving treatment paradigms in transplantation and cellular therapy. The topics are organized by organ system or complication type. Tables with recommendations have been created by organ system, and supplementary tables that include more detailed information are also provided. Owing to the overall scope of the topic, several appendices are included.

It should be noted that many late effects may take years or even decades to manifest, and recommendations generally can be followed in perpetuity (unless specified otherwise); however, for some clinical situations, eventual discontinuation of screening may be reasonable. Additionally, although these recommendations can be helpful at a population level, patient-specific risk factors also should be considered, and these recommendations are not meant to replace the judgment or



Figure 1. International societies represented. American Society for Transplantation and Cellular Therapy (ASTCT), Asia-Pacific Blood and Marrow Transplantation Group (APBMT), Australia and New Zealand Transplant and Cellular Therapies (ANZTCT), Children's Oncology Group (COG), Cell Therapy Transplant Canada (CTTC), Center for International Blood and Marrow Transplantation Research (CIBMTR), East Mediterranean Blood and Marrow Transplantation Group (EMBMT), European Society for Blood and Marrow Transplantation (EBMT), Latin American Bone Marrow Transplantation Group (LABMT), Pediatric Transplantation and Cellular Therapy Consortium (PTCTC), and Sociedade Brasileira de Transplante de Medula Ossea (SBTMO).

advice of clinicians caring for individual patients. The recommendations in this document rely heavily on expert consensus given the lack of prospective randomized trials for screening, prevention, or treatment [5]. Survivorship care may take many forms, and these recommendations may be adapted as appropriate. Furthermore, currently available data are derived largely from North American and European centers and might not be generalizable to all populations, and resource constraints may limit guideline implementation, especially in certain geographic regions. Therefore, consideration of local data-driven guidance is valuable for survivor care.

METHODOLOGY

First, a core group of 7 participants (S.J.R., N.S.B., B.K.H., C.D., K.S.B., N.S.M., and R.P.) reviewed the 2012 guidelines and suggested new topics for inclusion, areas to emphasize, and changes in formatting. These suggestions were reviewed with the larger group of 30 participants, additional changes were suggested and implemented, and the overall format was agreed upon. Subgroups of 2 to 4 members reviewed the relevant literature to draft topic section content and recommendations; all participants had the opportunity to provide feedback. All participants were then surveyed to determine agreement with each screening, prevention, and treatment recommendation. The recommendation was adopted if $\geq 85\%$ agreement occurred but was further edited before a second round of voting if agreement was $< 85\%$. If $> 50\%$ consensus ultimately could not be

reached, the recommendation was abandoned. Specific recommendations were then categorized similarly to the National Comprehensive Cancer Network (NCCN) approach; all were classified as either 2A or 2B, indicating a lower level of evidence with uniform consensus (2A; $\geq 85\%$) or consensus (2B; $> 50\%$ to $< 85\%$) [24].

HEMATOPOIETIC COMPLICATIONS

Common hematopoietic complications include autoimmune cytopenias (AICs), clonal hematopoiesis of indeterminate potential (CHIP), iron overload, and venous thromboembolism (VTE). Mixed chimerism also may present challenges for some patients (Table 1, Supplementary Table S1). AICs can occur weeks to years after allogeneic HCT, with risk factors including younger age, nonmalignant disease, umbilical cord blood graft source, unrelated or haploidentical donor, conditioning with antithymocyte globulin (ATG) or alemtuzumab, absence of total body irradiation (TBI), presence of GVHD, and cytomegalovirus (CMV) reactivation [25–27]. Cytopenias should prompt an etiologic investigation, especially to exclude autoimmunity or therapy-related myeloid neoplasms (tMN; see Subsequent Malignant Neoplasms); treatment is based on underlying etiologies.

The clonal expansion of hematopoietic progenitors in CHIP appears to be due to age-associated somatic mutation without an overt hematologic malignancy [28–30]. CHIP is associated with the development of subsequent hematologic malignancies and cardiovascular disease in the general

Table 1
Hematologic Complications

Recommendation	Grade	Ref	Comments
• CBC at most routine clinical visits for at least 10 years post-HCT, and as needed	2A		
• Hemoglobinopathy: chimerism at least every 3 months in year 1 post-HCT and every 6 months in year 2. Further chimerism based on previous results	2A		
• Regular monitoring of serum ferritin until normalized	2A	[33,362,363]	• Serum ferritin is a good, albeit nonspecific, initial screening test for iron overload.
• Iron quantification by MRI more accurately evaluates iron overload than serum ferritin; recommended to assess liver and cardiac iron levels and follow progress after phlebotomies and/or iron chelation.	2A	[33,362,363]	• Risk based on prior transfusion history or underlying HCT indication (eg, hemoglobinopathy, Diamond-Blackfan anemia, high-risk leukemia, neuroblastoma)
• Post-HCT phlebotomy is the treatment of choice for significant iron overload.	2A	[33,364,365]	• Iron chelation may be considered in patients ineligible for phlebotomy; prolonged or combined modality treatment may be necessary. • In females, resumption of menstruation (either naturally or via cyclical hormone replacement regimens) may reduce iron burden.
• VTE prophylaxis is indicated in patients with multiple myeloma receiving immunomodulatory imide drugs (eg, lenalidomide) and chemotherapy and/or dexamethasone after HCT.	2B	[366,367]	
• Before initiating anticoagulation, the risk of bleeding should be assessed.	2A		
• Inherited bone marrow failure syndromes require specialized follow up with a multidisciplinary specialist team.	2A		• Hematologist familiar with the underlying condition, HCT physician, other subspecialty providers

CBC: complete blood count.

Key references and further reading: [26,34,35,315]

population [28–30], as well as inferior overall survival, increased risk of tMN, and higher rates of cGVHD in the HCT setting [31,32]. Currently available data are too sparse to allow for specific recommendations regarding CHIP-associated late effects monitoring; however, all survivors should undergo an individualized cardiovascular disease risk assessment (see Cardiovascular Disease), and CHIP may be considered among those risk factors.

Iron overload from pre-HCT transfusion burden is common, and patients with disorders associated with ineffective erythropoiesis are at particular risk [33]. Post-HCT iron overload is associated with infections, chronic liver disease, pituitary dysfunction, glucose dysregulation, and cardiomyopathy

[33]. Survivors of both autologous and allogeneic HCT are at increased risk for VTE [34–37], and long-term HCT survivors with a history of VTE have greater nonrelapse mortality [38]. Risk factors include indwelling catheters, acute GVHD (aGVHD) or cGVHD, infections, prolonged immobilization, HCT for malignancy, endothelial damage from conditioning, and prior history of VTE [34–36,39,40]. Patients receiving immunomodulatory drugs for myeloma also are at higher risk.

IMMUNITY AND INFECTIONS

HCT survivors are at risk for developing infections and autoimmune diseases post-HCT; however, significant gaps still remain in our knowledge

Table 2
Immunity and Infections

Recommendation	Grade	Ref	Comments
<ul style="list-style-type: none"> All survivors should be offered a full vaccination program according to published guidelines considering patient age and country recommendations. Inactivated vaccines may begin 3 to 6 months after HCT. Includes patients with GVHD and/or on IST given higher risk of infection. Vaccinate patients with asplenia or functional asplenia for <i>S. pneumoniae</i> and <i>N. meningitidis</i> (MCV4, group B); educate on sepsis/ fever management; consider antimicrobial prophylaxis. 	2A	[63–66,368]	<ul style="list-style-type: none"> We recognize the potential limitation of early vaccination in some groups of patients with impaired immunity and the potential value of monitoring immune responses in these patients to guide ongoing management. Splenic presence and function (ie, SCD and autoinfarction, high-dose splenic irradiation), lymphocyte function (ie, hypogammaglobulinemia), and overall immune status should be considered in overall vaccine timing/prioritization.
<ul style="list-style-type: none"> Routine use of IVIg in adults generally not recommended. Supplemental IVIg may be considered for selected HCT recipients with IgG levels <400 mg/dL 	2B	[57,72–79]	<ul style="list-style-type: none"> No demonstrated survival advantage in unselected adult patients Consider for those with IgG levels <400 mg/dL and recurrent sino-pulmonary infections or those receiving anti-B cell or CAR T-Cell therapy or patients with very low IgG levels (<200 mg/dL)
<ul style="list-style-type: none"> For patients with IEI, pay careful attention to immune reconstitution and mixed chimerism. Assess lymphocyte subsets, mitogen proliferation, immunoglobulin levels and antibody responses every 3–6 months until normalized and as needed 	2A		
<ul style="list-style-type: none"> Patients with MM are at increased risk for infection; specific anti-myeloma medications warrant specific antimicrobial prophylaxis 	2A		

CAR, chimeric antigen receptor; IEI, inborn error of immunity; MCV4, quadrivalent meningitis vaccine; MM, multiple myeloma; SCD, sickle cell disease.

Key references and further reading: [50–56,58,63,66,76]

of immune dysfunction as a late effect of HCT [16]. Infection is a significant cause of late mortality after allogeneic HCT, even in individuals without cGVHD [7,8,41]. Late CMV infections are seen most frequently in patients with early CMV disease, cGVHD, and late immune manipulation (eg, lymphocyte infusions). Other viral infections that may lead to significant hospitalization, morbidity, and mortality include varicella zoster virus (VZV), influenza, and coronavirus disease 2019 (COVID-19) [42,43]. Survivors also are at risk for Epstein-Barr virus (EBV), post-transplantation lymphoproliferative disorder (PTLD) (see Subsequent Malignant Neoplasms), and hepatitis B and C viruses (see Gastrointestinal Complications). Several risk factors affect the incidence of late fungal infections, including cGVHD with ongoing immunosuppression, history of relapse, age, underlying disease,

type of conditioning (especially with TBI), umbilical cord blood graft source, and the use of T cell depletion (Table 2, Supplementary Table S2) [16,44–46]. *Pneumocystis jirovecii* pneumonia is rare unless there is nonadherence with prescribed prophylaxis, although when it does occur, the mortality rate is high [47–49]. Finally, bacterial infections pose a risk for long-term survivors with asplenia, cGVHD, a central line, unvaccinated status, or other risk factors.

General guidelines for prophylaxis and treatment of HCT-associated infections (both early and late) and advice for safe living are beyond the scope of these recommendations and have been reviewed elsewhere [50–59]. Dietary restrictions and guidelines on returning to work or school practices also lack uniform consensus [60–62]. Nonetheless, chronically immune suppressed

patients should be aware of infection risks and how to recognize signs and symptoms of infection and instructed to adhere to antimicrobial prophylaxis and vaccination guidelines.

Vaccine-preventable late infections are 3 times more common in ≥ 2 -year HCT cancer survivors compared to non-HCT cancer survivors and > 30 times more common compared to the general population [45]. All survivors should be offered a full vaccination program according to published guidelines, taking into account patient age and country recommendations [63–66], as detailed in reports from the Infectious Disease Society of America and the 2017 European Conference on Infections in Leukemia (Appendix 1) [63,67,68]. At the time of this publication, COVID-19 vaccination is recommended at 100 days post-HCT, although immunogenicity remains variable, and this is a rapidly changing area of study [69–71].

The routine use of i.v. immune globulin (IVIG) for hypogammaglobulinemia in adults generally is not recommended, given the lack of survival advantage or infection prevention with routine use in unselected patients after HCT [72], as well as potential side effects. Certain populations may benefit from IVIG supplementation, however [57,72–79].

OCULAR COMPLICATIONS

Ocular complications after HCT can be broadly divided into GVHD-related and non-GVHD-related, although overlap often exists (Table 3, Supplementary Table S3) [80–86]. Lacrimal gland dysfunction is the most common feature of ocular GVHD. Conjunctival involvement in GVHD is rare in children but more frequent in adults. Dry eyes also can be seen with radiation effects, chemotherapy, lid dysfunction, medications, and meibomian gland dysfunction. Post-HCT cataract formation has been associated with glucocorticoid treatment for GVHD, busulfan conditioning, and TBI [86]. Glaucoma may be a late complication of TBI, although systemic or topical corticosteroid therapy for cGVHD also can elevate intraocular pressure in susceptible patients, with children at greater risk. Infectious complications include viral bacterial, fungal, and toxoplasma. Retinal hemorrhage and detachment are rare but can be associated with CMV retinitis or the neovascularization seen in ischemic retinopathy.

ORAL AND DENTAL COMPLICATIONS

Oral and dental complications may result from cGVHD, chemotherapy, and irradiation therapy (Table 3, Supplementary Table S4). Oral cGVHD is

common and can involve the mucosa, salivary glands (xerostomia), oral and lingual muscles, taste buds, and gingiva. Patients may report oral pain, dryness, odynophagia, dysphagia, and sensitivity to normally tolerated flavors [80,87,88]. Gingivitis due to cGVHD may further limit toothbrushing. Presence of cGVHD is also a risk factor for squamous cell cancer (see Subsequent Malignant Neoplasms) [89–95]. Late complications also include increased dental demineralization and caries, teeth staining, gingival enlargement, symptomatic acute periodontal infection, and asymptomatic chronic periodontal infection [96]. Salivary gland dysfunction predisposes to caries, oral herpes simplex and candidiasis, mechanical and epithelial injuries, and impaired tooth mineralization [97]. Protracted xerostomia also may occur in patients without cGVHD due to chemotherapy or radiation or as medication side effect. Chemoradiotherapy exposure may disturb dental development in 50% to 80% of children; younger age at HCT and receipt of TBI are important risk factors [96,98]. TBI also may lead to mandibular underdevelopment and mandibular joint anomalies. Routine dental care is imperative for optimal oral health. Frequent self and professional oral examinations are the mainstays of early diagnosis of oral cancer. Patients should report lesions that do not heal, localized pain, leukoplakia, or other mucosal changes.

RESPIRATORY COMPLICATIONS

Late pulmonary complications include idiopathic pneumonia syndrome (IPS), pulmonary fibrosis, bronchiolitis obliterans syndrome (BOS), and cryptogenic organizing pneumonia (COP) (Table 4, Supplementary Table S5) [22]. IPS usually develops within the first 120 days post-HCT, although later cases can occur. IPS increases the risk of transplantation-related mortality and is thought to be multifactorial, with risk factors including allogeneic HCT, chest radiation or TBI, certain chemotherapies, increasing age, and GVHD [99–101]. Pulmonary fibrosis may occur late after HCT and is generally characterized by pre- or post-HCT lung injury with specific risk factors including radiation, bleomycin, busulfan, carmustine, smoking, history of acute lung injury, and post-transplantation CMV pneumonitis.

BOS, considered a lung manifestation of cGVHD, is often diagnosed within the first 2 years post-HCT [102–105]. Patients may be asymptomatic initially, making diagnosis difficult without screening. cGVHD is the most important risk factor for developing BOS; other risk factors include aGVHD, lung

Table 3
Ocular, Oral and Dental Complications

Recommendation	Grade	Ref	Comments
<ul style="list-style-type: none"> • Question about eye concerns at each follow-up visit. 	2A	[84,85,369]	<ul style="list-style-type: none"> • Advise to report dryness, light sensitivity, excessive tearing, foreign body sensation, pain, redness, swelling, mucoid aggregates, vision changes.
<ul style="list-style-type: none"> • Inform about risk of premature cataracts for TBI recipients 	2A		<ul style="list-style-type: none"> • Busulfan and glucocorticoids are also risk factors.
<ul style="list-style-type: none"> • Ocular exam recommended at onset of any cGVHD, dry eye symptoms, changes in vision or other eye symptoms. 	2A	[84,369]	<ul style="list-style-type: none"> • The NIH Consensus Development Project recommends consultation with an eye specialist every 3 months during the first year post-HCT and then at longer intervals thereafter, with recommendations on best practice eye examination; currently unknown if this approach reduces long-term ocular morbidity.
<ul style="list-style-type: none"> • Monitoring of IOP important in patients receiving any form of glucocorticoids. 	2A		<ul style="list-style-type: none"> • IOP may be elevated with topical glucocorticoids, particularly in children.
<ul style="list-style-type: none"> • Hurler syndrome: ongoing ophthalmologic assessment for glaucoma, cataracts, progression of corneal clouding. 	2A		
<ul style="list-style-type: none"> • Evaluation by a dentist or oral medicine specialist at 6 months, 1 year, and annually thereafter may be beneficial. • More frequent dental consultations and screening (eg, every 6 months) for those at a high risk for oral cancer (eg, FA, radiation to head/ neck, refractory cGVHD). • Oral/ radiologic tooth development assessment for children. 	2A		<ul style="list-style-type: none"> • Inform regarding preventive oral health, routine dental hygiene, and risks/ symptoms of oral cancer.
<ul style="list-style-type: none"> • At each survivorship visit screen for cGVHD by history and exam, ask about high-risk habits; head/neck/ oral exam. 	2A	[370]	<ul style="list-style-type: none"> • Symptoms: dry mouth, difficulty swallowing, pain in the oral cavity.
<ul style="list-style-type: none"> • Council to avoid smoking, vaping, and chewing tobacco, decrease regular intake of sugar containing beverages, avoid intraoral piercing, pursue post-HCT HPV vaccination. 	2A	[66,342]	
<ul style="list-style-type: none"> • Patients with xerostomia should receive meticulous oral hygiene, undertake preventive measures for dental/ periodontal disease, and aggressive treatment of oral infections. Trauma to oral mucosa should be avoided. 	2A		<ul style="list-style-type: none"> • Mouthguards may be useful. • Avoid medications that can cause or exacerbate xerostomia.

FA, Fanconi anemia; HPV, human papilloma virus; IOP, intraocular pressure.

Key references and further reading: [84,85,88,95,96,371]

toxic medications, ABO incompatibility, peripheral blood grafts, and early post-HCT viral infections [102,106]. Early detection and treatment of BOS impacts outcomes. The use of National Institutes of Health (NIH) diagnostic criteria usually establishes the diagnosis of BOS without the need for lung

biopsy [80]. Cryptogenic organizing pneumonia, previously known as bronchiolitis obliterans organizing pneumonia (BOOP), typically presents at <1 year post-HCT with fever, cough, and dyspnea; a chest computed tomography scan shows solitary or multifocal pulmonary infiltrates, whereas

Table 4
Respiratory Complications

Recommendation	Grade	Ref	Comments
• Assessment for pulmonary symptoms (cough, wheezing, dyspnea) and physical examination at each visit	2A		
• Expert consensus recommends PFT screening every 3 months for the first year after HCT, every six months for the second year, and then annually for 5 years after HCT (or until final adult height in children, whichever occurs later)	2B	[103–105,369,372–377]	• Spirometry and hemoglobin corrected DLCO • Age <6 usually unable to perform PFTs; alternative screening with pulse oximetry, multiple breath washout testing, parametric response mapping by CT may be considered if available, but this requires further study.
• PFTs at initial diagnosis of any cGVHD , then at least spirometry every 3 to 6 months until discontinuation of all systemic IST for cGVHD.	2A	[369,375–377]	• Survivors, especially those with cGVHD, can develop moderate to severe PFT abnormalities before becoming symptomatic.
• PFTs recommended at regular intervals following HCT for patients with DKC ; referral to a pulmonologist if abnormalities are noted.	2A	[378]	
• CT chest imaging (high resolution where available) recommended in symptomatic individuals/ abnormal PFTs.	2A		
• Consider pulmonology consultation for those with symptomatic pulmonary dysfunction or new asymptomatic PFT abnormalities, especially irreversible air flow obstruction	2A		
• Recommend vaccinations against respiratory pathogens (eg, pneumococcus) and avoidance of tobacco, smoking, vaping .	2A		

DKC, dyskeratosis congenita; DLCO, diffusion capacity of the lungs for carbon monoxide); PFT, pulmonary function testing. Key references and further reading: [101,102,369,372]

pulmonary function tests classically show a restrictive pattern [107,108]. cGVHD is a risk factor, along with drug toxicity, radiation, HLA mismatch, donor-recipient sex mismatch, and use of peripheral blood grafts [108–110]. The diagnoses of IPS, COP, and BOS can be confirmed through lung biopsy; however, less invasive means are typically sufficient. IPS and COP are diagnoses of exclusion and typically require bronchoalveolar lavage to rule out infection.

CARDIAC AND VASCULAR COMPLICATIONS

Cardiovascular complications that occur frequently and contribute to late mortality include hypertension (see Renal and Urinary Complications), dyslipidemia, congestive heart failure, arrhythmias, valvular heart disease, premature

coronary artery disease, stroke (see Neurologic and Cognitive Complications), CHIP (see Hematopoietic Complications), and peripheral vascular disease (Table 5, Supplementary Table S6) [17,22,111–120]. Metabolic syndrome (MetS) includes parameters that together increase the risk for diabetes and cardiovascular disease and is associated with increased all-cause mortality. The prevalence of MetS among HCT survivors is 31% to 53%, higher than that seen in background populations [121–123]. Diagnostic criteria include the presence of visceral obesity, elevated blood pressure, hyperglycemia or insulin resistance, and high triglyceride or low high-density lipoprotein levels [124].

Allogeneic HCT recipients have higher rates of abdominal obesity, lipid disorders, and impaired

Table 5
Cardiac and Vascular Complications

Recommendation	Grade	Ref	Comments
<ul style="list-style-type: none"> • Evaluate cardiovascular risk on personal/family history, lifestyle, autologous vs allogeneic HCT, cumulative anthracycline dose, history of chest radiotherapy, TBI, chronic CNI use, sirolimus, or glucocorticoids. Further surveillance, prevention and treatment should be tailored according to those individual risks 	2A	[379,380]	<ul style="list-style-type: none"> • Healthy lifestyle is advised: normal weight, eating fruits and vegetables, tobacco abstinence, frequent physical activity; a minimum standard would be to follow USPTF and NHLBI general population guidelines.
<ul style="list-style-type: none"> • Measurement of blood pressure, weight, and BMI at each survivorship visit 	2A		
<ul style="list-style-type: none"> • For patients with or at very high risk for cardiovascular disease, consultation with a cardiologist • Patients with abnormal imaging studies or concerning symptoms should be referred for cardiology consultation 	2A		<ul style="list-style-type: none"> • Preferably familiar in late effects of cancer and HCT
<ul style="list-style-type: none"> • Start lipid assessment for adults at 6 months post-HCT and then annually if low risk for cardiovascular disease; screen high-risk patients or on glucocorticoids, CNI or sirolimus, every 3–6 months 	2A		<ul style="list-style-type: none"> • Pediatric survivors: less frequent monitoring reasonable
<ul style="list-style-type: none"> • Start HbA1c screening for insulin resistance/diabetes at 3–6 months post-HCT, then annually if low risk; high risk: every 6 months 	2A		<ul style="list-style-type: none"> • Pediatric survivors: less frequent monitoring reasonable
<ul style="list-style-type: none"> • Children exposed to CRT and/or TBI, and survivors treated with abdominal radiation and/or TBI are considered high risk for mets 	2A		
<ul style="list-style-type: none"> • Multimodal weight loss treatment for obese adults 	2A		<ul style="list-style-type: none"> • With geographically appropriate, population-based definitions for abnormal BMI or abdominal circumference, when available
<ul style="list-style-type: none"> • HTN management should follow general population guidance 	2A	[381]	
<ul style="list-style-type: none"> • Dyslipidemia management should follow the AHA/ ACC recommendations or local guidelines 	2A	[382]	<ul style="list-style-type: none"> • Consultation with a pharmacist to review potential drug interactions especially if on systemic IST
<ul style="list-style-type: none"> • Cardiomyopathy pediatric survivors: radiation and anthracycline chemotherapy are significant risk factors; serial screening with ECHO and ECG per COG guidelines 	2A	[383,384]	<ul style="list-style-type: none"> • COG Guidelines (http://survivorshipguidelines.org/) • Pre-HCT chemotherapy, HCT condition and clinical course should all be included in assessment of risk
<ul style="list-style-type: none"> • Cardiomyopathy adult survivors: For those receiving anthracyclines, ECHO may be considered within a year of therapy completion for those with high cumulative anthracycline exposure (≥ 250 mg/m²), or those with lower exposure and additional heart failure risk factors. • Adults who never received anthracyclines ECHO and ECG may not be routinely required, however a low threshold to request these studies based on shared decision making in high-risk groups 	2A	[383,385]	<ul style="list-style-type: none"> • Ongoing surveillance for adults should be based on individual risk factors

(continued)

Table 5 (Continued)

Recommendation	Grade	Ref	Comments
• Hurler Syndrome: post-HCT cardiopulmonary assessment	2A		• Valvular dysplasia, regurgitation, stenosis, and progression of pre-existing pulmonary disease may worsen
• Amyloidosis: close monitoring of cardiac function longer-term	2A		• Cardiac issues may persist long-term

AHA/ACC, American Heart Association/ American College of Cardiology; BMI, body mass index; CHIP, clonal hematopoiesis of indeterminate pathology; COG, Children's Oncology Group; CRT, cranial radiation; ECHO, echocardiogram; ECG, electrocardiogram; HTN, hypertension; MetS, metabolic syndrome; NHLBI, National Heart, Lung, and Blood Institute; USPTF, United States Preventive Service Task Force.

Doxorubicin equivalent: daunorubicin, .5; epirubicin, .67; idarubicin, 5; mitoxantrone, 10 (may consider 4:1). [383]

Key references and further reading: [17,115,117,118,127,384]

glucose metabolism compared with autologous HCT recipients, possibly related to the use of glucocorticoids, sirolimus, or calcineurin inhibitors (CNIs); alloreactivity; or cranial radiation or TBI [122,125–131]. TBI has been associated with hyperglycemia, diabetes, and dyslipidemia, and pancreatic irradiation can lead to diabetes; GVHD also has been associated with hypertension [129,132–134]. Ischemic events are more frequent after allogeneic HCT than after autologous HCT [135]. Hypertension, diabetes, dyslipidemia, smoking, sedentarism, and obesity have been identified as important additive risk factors [115,135,136]. Cardiomyopathy and heart failure are strongly related to anthracycline exposure (particularly at cumulative doses ≥ 250 mg/m²), hypertension, history of chest irradiation, diabetes, and age, whereas data on the associations with TBI and conditioning intensity are conflicting [115,117,137]. Because genetic polymorphisms play a role in anthracycline-related cardiomyopathy, there is no clear “safe” dose of anthracyclines [138]. It is important to note that the incidence of cardiovascular events increases with greater time since HCT [135], and the presence or absence of cardiovascular disease or echocardiographic findings soon after HCT does not necessarily predict the risk of long-term cardiac toxicities.

GASTROINTESTINAL COMPLICATIONS

Long-term gastrointestinal complications of HCT involve luminal and solid organs (Table 6, Supplementary Table S7). cGVHD of the gastrointestinal tract may lead to dysphagia, esophageal webs, strictures, and stenosis [139]. Previous radiation therapy (eg, mediastinal for Hodgkin lymphoma) also may increase the risk for esophageal strictures [140]. Esophageal cGVHD and/or targeted radiation or TBI are risk factors for

esophageal cancer (see Subsequent Malignant Neoplasms) [95,140,141]. Likewise, luminal strictures may occur in patients with a history of severe gastrointestinal GVHD, abdominal surgery, or abdominal radiation and may be considered in the differential diagnosis of intermittent abdominal pain or small bowel obstruction [142]. Abdominopelvic radiation also increases the risk for colon cancer in childhood cancer survivors (see Subsequent Malignant Neoplasms) [143–145].

Hepatic complications may be multifactorial and potentially associated with GVHD, infections, medication, underlying acquired or genetic liver disease, and iron overload (see Hematopoietic Complications). Focal nodular hyperplasia is a common incidental radiologic finding that may be associated with oral contraceptive use, younger age at HCT, and abdominal radiation and occurs frequently in children with neuroblastoma [146–149]. Although malignant transformation is uncommon [147], consultation with a hepatologist may be warranted in cases with lesion growth or diagnostic uncertainty [149]. The prevalence of chronic hepatitis B virus (HBV) infection varies widely depending on patient age and geographic location. HBV antibody titers might not be detectible owing to immunosuppression and should not be relied on entirely. Pre-HCT chronic HBV infection (surface antigen-positive) or resolved HBV infection (core antibody-positive but surface antigen-negative) may result in fulminant post-HCT hepatitis [150,151]. Survivors with chronic HBV infection need regular monitoring to assess viral load, liver status, and need for antiviral therapy and are usually referred to a hepatologist. Hepatitis C virus (HCV) infection usually results in chronic hepatitis, presenting as asymptomatic alanine aminotransferase (ALT) elevation at 2 to 4 months post-HCT when immunosuppressive

Table 6
Gastrointestinal, Renal, and Urinary Complications

Recommendation	Grade	Ref	Comments
<ul style="list-style-type: none"> Elicit symptoms of GI cGVHD at follow-up visits. 	2A		<ul style="list-style-type: none"> Positive review of systems should warrant further workup.
<ul style="list-style-type: none"> Check total bilirubin, alkaline phosphatase, and ALT at least every 1-2 months during the first year; then at least yearly in allogeneic HCT recipients without risk factors for liver disorders. 	2A		<ul style="list-style-type: none"> Patients with chronic hepatitis, or those tapering IST for cGVHD typically require more frequent monitoring.
<ul style="list-style-type: none"> DKC: ongoing liver function evaluation and referral to a hepatologist if abnormalities noted. 	2A		
<ul style="list-style-type: none"> HBV antiviral prophylaxis for patients with chronic HBV regardless of HBV DNA levels and continue ≥ 1 year after withdrawal of IST. Monitor patients with chronic HBV infection at least every 2 months with HBV DNA. 	2A	[151,386]	<ul style="list-style-type: none"> Monthly monitoring of HBV DNA and ALT should be done after the withdrawal of antiviral treatment. For patients with chronic HBV infection, monitoring of HBV DNA should be lifelong.
<ul style="list-style-type: none"> Patients with resolved HBV infection who received allogeneic HCT, HBV DNA (monitoring at least every 2 months) guided preemptive antiviral therapy. 	2A	[387,388]	<ul style="list-style-type: none"> Consider prophylactic anti-HBV therapy for patients at centers without resources to perform HBV DNA monitoring. Continue monitoring of HBV DNA or prophylactic anti-HBV therapy for ≥ 3 years after discontinuation of IST.
<ul style="list-style-type: none"> Antiviral prophylaxis for those receiving grafts from donors with chronic HBV infection. Preemptive approach (based on viral monitoring) in those receiving grafts from donors with resolved HBV infection. 	2B		
<ul style="list-style-type: none"> Offer antiviral therapy if available (and not contraindicated) to HCV-infected survivors. Evaluate for progressive liver disease every 6-12 months with bilirubin, albumin, PT/INR, and CBC. 	2A	[389]	<ul style="list-style-type: none"> In general, refer to a hepatologist for counseling, monitoring, and treatment considerations. Evaluate and counsel for fibrosis/ cirrhosis risk factors (obesity, ethanol, medications, hepatotoxic herbal supplements).
<ul style="list-style-type: none"> In chronic HCV infection, routine monitoring of quantitative serum HCV RNA not recommended except at baseline, or if unexplained serum ALT elevation, or during HCV antiviral therapy. 	2A	[152,389]	
<ul style="list-style-type: none"> Monitor patients with cirrhosis for esophageal varices, hepatocellular carcinoma, and other sequelae with an experienced hepatologist. 	2A	[390]	<ul style="list-style-type: none"> Screening for HCC in HCT recipients with cirrhosis may follow general guidelines available from the AASLD. Liver transplantation may be considered in any HCT survivor with hepatic decompensation or early HCC.
<ul style="list-style-type: none"> Evaluate renal function with UA, urine protein-to-Cr ratio, BUN, and Cr at 6 months, 1 year, and \geq yearly thereafter 	2A		<ul style="list-style-type: none"> MM and amyloidosis: renal function should be monitored more frequently.
<ul style="list-style-type: none"> For patients with progressive CKD, avoid nephrotoxins and refer to a nephrologist early. 	2A		
<ul style="list-style-type: none"> Monitor blood pressure at every clinic visit; persistent hypertension should prompt intervention and/or referral. 	2A	[112]	<ul style="list-style-type: none"> Lifestyle modification including weight reduction, dietary sodium restriction, regular physical activity generally first interventional step, followed by antihypertensive medications.

AASLD, American Association for the Study of Liver Diseases; ALT, alanine aminotransferase; BUN, blood urea nitrogen; Cr, creatinine; GI, gastrointestinal; HCC, hepatocellular carcinoma; PT/INR, prothrombin time/international normalized ratio; UA, urinalysis.

Key references and further reading: [65,151,153,156,159,386,389]

therapy (IST) is tapered. Chronic HCV infection may cause liver-related mortality only rarely in the first 10 years but is the leading cause of post-HCT cirrhosis [112,152,153].

Although uncommon, pancreatic complications are related mainly to biliary stone passage and rarely to tacrolimus-associated pancreatic damage [154]. Pancreatic exocrine insufficiency occasionally presents with steatorrhea and weight loss despite adequate caloric intake. Its pathogenesis is speculative but thought to involve pancreatic atrophy from prior damage, possibly GVHD-associated; response to a trial of enzyme supplementation may be diagnostic [155].

RENAL AND URINARY COMPLICATIONS

Renal and urinary complications after HCT include chronic kidney disease (CKD), transplantation associated-thrombotic microangiopathy (TA-TMA), nephrotic syndrome (NS), and hypertension. CKD can be caused by a number of pre-, peri-, and post-HCT exposures, with risk factors including previous acute kidney injury, GVHD, older age at HCT, baseline renal insufficiency, hypertension, and TBI [22,156]. Most cases of CKD are multifactorial owing to an accumulation of peritransplantation events and/or risk factors (Table 6, Supplementary Table S8). The cumulative incidence of CKD varies from 7% to 48%, and it may develop between 6 months and 10 years post-HCT, with ~4% of cases progressing to end-stage renal disease [156]. A progressive glomerular filtration rate decline is associated with increasingly higher risk for mortality [157]. TA-TMA is a well-recognized complication, but diagnosis is often been delayed and confounded [158–160]. TA-TMA occurs most frequently early post-HCT but may occur late after HCT, often in association with cGVHD [158–161]. Elevated lactate dehydrogenase, rising urine protein-to-creatinine ratio, and hypertension are the earliest markers of TA-TMA and should prompt clinicians to pursue further workup [112,156]. NS is usually characterized by proteinuria, hypoalbuminemia, and/or edema, occurring most often after IST is tapered for GVHD (at 6 to 12 months post-HCT) [17,156]. As many as 70% of patients develop hypertension at <2 years post-HCT [17,156]. Known risk factors include CNI therapy, acute kidney injury, TBI, autologous HCT, obesity, and diabetes [156]. Effective antihypertensive therapy is important for reducing cardiovascular disease risk (see Cardiac and Vascular Complications) and progression of CKD [316].

ENDOCRINE COMPLICATIONS

HCT survivors are at risk of developing growth impairment, gonadal insufficiency and infertility, thyroid dysfunction, and adrenal insufficiency. Growth can be impacted by multiple factors, including treatment exposures and post-HCT complications (Table 7, Supplementary Table S9) [162]. The reported incidence of post-HCT growth hormone deficiency due to hypothalamic-pituitary injuries varies from 20% to 85% [163–166]. Cranial irradiation (particularly ≥ 18 Gy) and TBI are established risk factors; the final impact on growth depends on age at exposure, patient sex, and dose of and time since radiation therapy [163,167–172]. Nutritional deficiencies also may impact growth and development. The prevalence of gonadal dysfunction exceeds 90% in some studies and can manifest as delayed pubertal development or otherwise as gonadal insufficiency and infertility (see Sexual Health, Fertility, and Pregnancy) [65]. If untreated, it may lead to sexual dysfunction, low bone mineral density, cardiovascular disease, and poor QoL [173]. TBI, cranial or gonadal irradiation, alkylating agents, and platinum chemotherapy are risk factors; age at exposure also may impact the risk. Thyroid dysfunction is the most common post-HCT endocrinopathy, with reported prevalence ranging from 10% to 47% [174–177]. Hypothyroidism is diagnosed at a median of 4 years post-HCT, although the risk persists for longer [175]. Risk factors for thyroid dysfunction are younger age, head and neck radiation, high-dose TBI, busulfan and cyclophosphamide conditioning, and prolonged cGVHD [175,176,178–180]. Primary adrenal insufficiency is uncommon; most adrenal insufficiency is secondary owing to prolonged glucocorticoid treatment, which suppresses the hypothalamic-pituitary-adrenal axis [181].

SEXUAL HEALTH, FERTILITY, AND PREGNANCY

Sexual health, fertility, and pregnancy concerns of HCT survivors are summarized in Table 8 and Supplementary Table S10. Approximately one-third of survivors report the inability to perform sexually, inability to derive pleasure from sex, and/or little or no interest in sex [182,183]. Women are more likely than men to report being sexually inactive in the preceding year (39% versus 27%) and among those sexually active, to report low sexual function (64% versus 32%) [184]. Factors associated with being sexually inactive include older age, <4 years of college education, low clinical performance status, and not being in a committed relationship. Additional

Table 7
Endocrine Complications

Recommendation	Grade	Ref	Comments
<ul style="list-style-type: none"> In children, height, weight, and body mass index should be monitored. Patients with suspected growth velocity abnormality should be evaluated by a pediatric endocrinologist. 	2A	[391]	<ul style="list-style-type: none"> Males without signs of puberty by age 14 years or with failure of pubertal progression or abnormal growth velocity: early morning testosterone, FSH, and LH levels and/or endocrinology consultation. Females without signs of puberty by age 13 years or with pubertal progression failure, abnormal menstrual patterns, or menopausal symptoms: FSH and estradiol levels and/or endocrinology/ gynecology consultation.
<ul style="list-style-type: none"> Monitor children for onset and progression of puberty with annual Tanner staging until sexually mature. 	2A		<ul style="list-style-type: none"> Monitoring frequency should increase as survivors approach and undergo puberty, or if puberty appears delayed.
<ul style="list-style-type: none"> Assessment of menarche/ menstrual history and menopausal symptoms at least annually. 	2A		
<ul style="list-style-type: none"> Adult survivors should have gonadal assessment at least at 1 year post-HCT. Gonadal dysfunction should be assessed by appropriate subspecialties for potential HRT. 	2A		<ul style="list-style-type: none"> Subsequent frequency according to clinical need.
<ul style="list-style-type: none"> Serum TSH and free T4 levels should be checked at 1 year post-HCT or sooner in case of symptoms and annually thereafter. 	2A		<ul style="list-style-type: none"> Patients with exposure to CNS radiation are at risk for central hypothyroidism, and as such TSH alone is unreliable; hypothyroidism can be identified by a low free T4 plus low TSH with a history of hypothalamic-pituitary insult.
<ul style="list-style-type: none"> Patients on long-term systemic glucocorticoids may benefit from ACTH stimulation testing during or after glucocorticoid withdrawal, particularly if symptoms of adrenal insufficiency develop. 	2A		<ul style="list-style-type: none"> Patients on topical and inhaled steroids also are at risk for adrenal insufficiency. Patients should carry notification of adrenal insufficiency to alert emergency medical providers.

HRT, hormone replacement therapy; RT, radiation therapy; TSH, thyroid-stimulating hormone.

Key references and further reading: [189,392]

factors for men include nonmyeloablative conditioning and not being employed or in school. Lower sexual function also has been associated with TBI in men and cGVHD in both men and women [182–187].

HCT is associated with infertility owing to pretransplantation and transplantation-related treatment exposures and late effects [188,189]. Female sex, pre-HCT cytotoxic therapy, myeloablative conditioning, and germ cell tumor diagnosis have been associated with lower fertility post-HCT [190]. Underlying conditions also may impact fertility (eg, Fanconi anemia). In females, the degree of ovarian damage is related to the dose and type of exposure (eg, myeloablative conditioning, radiation), as well as to ovarian reserve, which is

dependent on age and previous treatment. The use of alkylating agents has the highest age-adjusted odds ratio of ovarian failure [189,190]. The TBI dose that is potentially sterilizing appears to decrease with increasing age [190,191]. In males, chemoradiotherapy can impair spermatogenesis, but the testosterone level generally remains normal because Leydig cells are relatively resistant. Lower testosterone can be seen when affected by GVHD, particularly in the setting of chronic glucocorticoid exposure [192]. For women, embryo and oocyte cryopreservation remains the preferred method of fertility preservation. Ovarian tissue cryopreservation is becoming increasingly feasible and remains the sole option for prepubertal patients [193]. Despite

Table 8

Sexual Health, Fertility, and Pregnancy

Recommendation	Grade	Ref	Comments
<ul style="list-style-type: none"> • Discuss sexual function with patients at 3–6 months post-HCT and annually thereafter. 	2A	[393–396]	<ul style="list-style-type: none"> • Potentially modifiable symptoms: genital cGVHD, ED, vaginal dryness/ dyspareunia, relationship quality, treatment related distress, depression. <p>Screening/ managing sexual dysfunction includes individualized multimodal approach addressing medical and psychosocial (including partner) needs.</p>
<ul style="list-style-type: none"> • Timely assessments of hypogonadism and/ or urogenital GVHD. 	2A	[393]	<ul style="list-style-type: none"> • GYN/GU referrals to address reversible risk factors relevant to sexual dysfunction. • POI is treated with HRT if not otherwise contraindicated. • Refer women with vaginal dryness ± cGVHD to GYN/GU; may benefit from lubricants, topical estrogens, dilator therapies. • Consider ED therapies, testosterone replacement in males.
<ul style="list-style-type: none"> • Patients desiring pregnancy should meet with a fertility specialist to best understand current fertility potential and what methods (ie, IVF, donor oocyte, donor sperm) may be considered if applicable. • Semen analysis or assessment of ovarian function for those contemplating future parenthood when age appropriate. 	2A	[393]	<ul style="list-style-type: none"> • Reassess fertility potential at intervals; partial or full fertility potential may recover for some patients.
<ul style="list-style-type: none"> • Contraception advised if fertility potential unknown, wishing to avoid parenthood. • Counseling regarding safer sex practices/ contraception should be discussed, even if infertile, to prevent sexually transmitted infections. 	2A		
<ul style="list-style-type: none"> • Women with gonadal recovery should be advised about the risks of POI. 	2A		
<ul style="list-style-type: none"> • Pregnant patients should be followed by MFHM (high-risk obstetrics). 	2A	[393]	<ul style="list-style-type: none"> • May require review by an anesthetist before delivery.
<ul style="list-style-type: none"> • Pregnant women with systolic dysfunction/ significant cardiac risk factors to be followed by cardiologist with expertise in cardiac late effects. 	2A	[196,393]	
<ul style="list-style-type: none"> • Counsel women who received radiation to the uterus (including TBI) and desire pregnancy on risk of uterine factor infertility, other pregnancy risks. 	2A		
<ul style="list-style-type: none"> • Offer genetic counseling before pregnancy to those undergoing HCT for genetic disorder or cancer predisposition syndrome 	2A		

ED, erectile dysfunction; GYN/GU, gynecology/urology; HRT, hormone replacement therapy; IVF, in vitro fertilization; MFHM: maternal-fetal medicine; POI, premature ovarian insufficiency.

Key references and further reading: [192,393,394,397]

success in animal models, the clinical value of gonadotropin-releasing hormone agonists to preserve ovarian function during chemotherapy remains uncertain [194]. Sperm cryopreservation is an established fertility preservation option for postpubertal males. In prepubertal males, the sole option is testicular tissue cryopreservation; although animal models are encouraging, to date there have been no reports of reimplanted testicular tissue leading to human live births [189].

Uterine radiotherapy exposures may lead to adverse reproductive outcomes [195]. Increased rates of infertility, miscarriage, preterm labor, intrauterine growth restriction, and low birth weight have been described, particularly when conception occurred within 1 year of radiotherapy [189,196]. However, in women who have not received radiation, the miscarriage rate is comparable to that of the background population without a significant increase in congenital malformations or genetic abnormalities [197,198]. Similarly, reported pregnancies and deliveries from partners of male recipients usually have been uncomplicated [199].

MUSCLE AND CONNECTIVE TISSUE COMPLICATIONS

Muscle and connective tissue complications after HCT are often associated with cGVHD and its treatment and include glucocorticoid-induced myopathy, fasciitis/deep sclerosis, polymyositis, and myasthenia gravis (Table 9, Supplementary Table S11) [200–206]. Steroid myopathy typically presents with proximal muscle weakness, difficulty rising from a squatting position, and atrophy of these muscle groups. Although an improvement in strength may occur by 2 to 3 weeks after steroid reduction, complete resolution can take longer. The use of fluorinated glucocorticoids (eg, dexamethasone) is associated with a higher risk of myopathy compared with nonfluorinated glucocorticoids (eg, prednisolone) [207]. Although there is significant variability in individual susceptibility to myopathy, ≤ 10 mg/day of prednisone or equivalent is unlikely to result in myopathy, but ≥ 40 mg/day for ≥ 1 month usually causes weakness [207].

Fasciitis and polymyositis are cGVHD manifestations [206,208,209]. Fasciitis may cause tightness or restricted range of motion on a photographic range of motion scale [208], with various combinations of visibly tight tendons in volar forearms/palms and palpable deep tissue sclerosis with overlying hyperpigmentation and “groove” signs. Along with cGVHD treatment,

patients with fasciitis may benefit from a multidisciplinary rehabilitation program to control edema and preserve range of motion. Polymyositis usually presents with moderate to severe proximal muscle weakness and myalgia [202]. Myasthenia gravis is a rare cGVHD complication that may be due to donor-derived antibodies against recipient acetylcholine receptors and manifests similar to classical myasthenia gravis, with most cases occurring >2 years post-HCT [200,210].

SKELETAL COMPLICATIONS

Skeletal complications following HCT include abnormal bone density and avascular necrosis (AVN). Low bone mineral density (BMD) or osteopenia is a common complication that if untreated may lead to osteoporosis and increased risk of bone fragility fractures (Table 9, Supplementary Table S12). The prevalence of low BMD is up to 75% among allogeneic HCT survivors and 65% in autologous HCT survivors [211]. Low BMD can be seen as early as 1 month post-HCT and often persists beyond 3 years post-transplantation [212–219]. Patient-related risk factors include extremes of age at HCT, female sex, low body weight or body mass index, inadequate calcium or vitamin D intake, physical inactivity, renal dysfunction, and hypogonadism. Disease-related risk factors include myeloma, hemophagocytic lymphohistiocytosis, hemoglobinopathies, and pre-HCT chemotherapy exposures [220]. Finally, HCT-related risk factors include TBI or craniospinal irradiation, GVHD, and prolonged IST including CNIs and glucocorticoids [212–224].

The cumulative incidence of AVN is 3% to 10% at 5 years post-HCT [211,225], and time of onset may range from 6 months to 10 years post-HCT [225]. Patients with a history of acute lymphoblastic leukemia or sickle cell disease may have AVN pre-HCT [226]. AVN most commonly affects the femoral head, although knees, ankles, elbows, vertebrae, and multiple concurrent joints often may be implicated [227]. The incidence of AVN is greater in females and with more intensive conditioning regimens (especially TBI), moderate to severe cGVHD, prior aGVHD, higher glucocorticoid exposures, and adolescents and young adults (AYA), in whom rapid bone growth occurs [224,226–231].

DERMATOLOGIC COMPLICATIONS

Cutaneous complications may occur in up to 70% of survivors [22] and is most commonly due to cGVHD but also may result from infections, subsequent neoplasms, or anti-infective and

Table 9

Musculoskeletal, Connective Tissue, Skeletal, and Dermatologic Complications

Recommendation	Grade	Ref	Comments
<ul style="list-style-type: none"> Routinely evaluate patients on glucocorticoid treatment for glucocorticoid-induced myopathies Patients with/ at risk for steroid myopathy should engage in physical activity and physical therapy 	2A		<ul style="list-style-type: none"> Observe patient rising from a squatting position Physiatry referral may be helpful. Low resistance exercise to prevent/ slow loss of muscle mass
<ul style="list-style-type: none"> cGVHD-associated polymyositis, statin toxicity, or myasthenia gravis should be included in differential diagnosis of myalgia/ weakness if persistent or progressive, 	2A		<ul style="list-style-type: none"> CPK, aldolase, anti-acetylcholine antibodies are a reasonable next step. If negative, muscle MRI, EMG or muscle biopsy may be considered
<ul style="list-style-type: none"> Encourage patients with cGVHD to perform self-assessment of ROM and perform ROM evaluation at each clinic visit 	2A		<ul style="list-style-type: none"> Ideally with medical photos for subsequent comparison
<ul style="list-style-type: none"> Standard risk patients: DEXA at 1-year post-HCT. If abnormal, repeat every 1-2 years, sooner if ongoing risks, or response assessment. High risk patients pre-HCT, or post-HCT high-dose glucocorticoids: Obtain DEXA at 3 months post-HCT. 	2B	[211,398–400]	<ul style="list-style-type: none"> <5 years old, lumbar spine BMD may be measured. DEXA hip measurements less reliable for age <13 FRAX and vertebral fracture assessment (VFA) may help evaluation/ management
<ul style="list-style-type: none"> Optimize Ca and vitamin D intake through diet, supplementation. Recommend regular, weight-bearing exercise. 	2A		<ul style="list-style-type: none"> Vitamin D may be measured regularly in those at deficiency risk.
<ul style="list-style-type: none"> Consider bisphosphonates in high-risk patients, significant abnormalities on DEXA or FRAX assessments, or fragility fractures. 	2A	[211,401]	<ul style="list-style-type: none"> Bisphosphonate choice made with consideration of the patient's presentation, renal function, and respective adverse events.
<ul style="list-style-type: none"> For patients with MM, supportive management with use of bisphosphonates for at least 2 years. 	2A	[211,402]	<ul style="list-style-type: none"> Such as zoledronic acid and pamidronate
<ul style="list-style-type: none"> Hormone replacement therapy should be discussed for patients with hypogonadism if age-appropriate, and not otherwise contraindicated (see Endocrine complications). 	2A		
<ul style="list-style-type: none"> Routine imaging screening for asymptomatic AVN not indicated. Maintain a high index of suspicion for AVN in patients with prior AVN, radiation exposure or prolonged glucocorticoids. Those with joint symptoms should be evaluated promptly 	2A	[211]	<ul style="list-style-type: none"> Symptomatic patients: non-contrast MRI is the most sensitive way to confirm and stage AVN; Once diagnosis of AVN prompt referral to orthopedic specialist recommended.
<ul style="list-style-type: none"> Following HCT for Hurler Syndrome, neurologic screening for spinal canal narrowing and carpal tunnel syndrome should be considered 	2A	[403,404]	
<ul style="list-style-type: none"> Inform patients about risk of dermatological complications, particularly related to cGVHD, medications, and radiation 	2B		<ul style="list-style-type: none"> Advise to seek medical attention for non-healing skin lesions, skin tightening or other changes.

(continued)

Table 9 (Continued)

Recommendation	Grade	Ref	Comments
<ul style="list-style-type: none"> • Undergo regular skin self-examination; refer to a dermatologist for further evaluation of suspicious lesions. • Frequency/extent of examination tailored to individual risk factors: prior or present GVHD, sun exposure and radiation history, voriconazole exposure, family history, and history of skin cancer 	2A	[57,80,232]	<ul style="list-style-type: none"> • Minimum of every 6-12 months for malignancy screening and general examination, including for cutaneous cGVHD based on NIH-criteria. • Regular skin exam involves exposure of all body areas and includes manual palpation to detect sclerosis
<ul style="list-style-type: none"> • Advise to avoid direct sun exposure without appropriate protection: proper clothing, hats, applying UVA/UVB sunscreen to exposed areas 	2A		<ul style="list-style-type: none"> • Particularly important for patients on immunosuppression, voriconazole, with a history of TBI or skin cGVHD

Key References and Further Reading: [57,80,207,211,224,232–234,405–407]

Abbreviations: AVN: Avascular Necrosis, BMD: Bone Mineral Density, Ca: Calcium, CPK: Creatine Phosphokinase, DEXA: Dual Energy X-ray Absorptiometry, EMG: Electromyography, FRAX: Fracture Risk Assessment Tool, GVHD: Graft versus Host Disease, MRI: Magnetic Resonance Imaging, MM: Multiple Myeloma, ROM: range of motion, TBI: Total Body Irradiation, UVA: Ultraviolet A, UVB: Ultraviolet B

immunosuppressive drugs (Table 9, Supplementary Table S13). Excellent overviews of diagnosis and management of cutaneous cGVHD are available elsewhere [57,80,232–234], and the risk of skin cancers is reviewed below (see Subsequent Malignant Neoplasms). Health care providers should recognize potential cutaneous side effects of glucocorticoids (eg, easy bruising, loss of skin integrity) and cyclosporine (eg, hirsutism, malignancy); they should monitor for cutaneous atrophy in survivors on high-potency topical glucocorticoids and recommend low-potency glucocorticoids (eg, hydrocortisone 1% to 2.5%) only in high-risk areas, such as the face [80,233,234].

NEUROLOGIC AND COGNITIVE COMPLICATIONS

Late neurologic dysfunction after HCT may affect the central nervous system (CNS) and peripheral nervous system and are more frequent after allogeneic HCT (Table 10, Supplementary Table S14) [235–237]. Potential causes include infection or immunosuppression (with CNIs), neurotoxic chemotherapy (eg, methotrexate, cytarabine, busulfan, thiotepa) and other medications, TA-TMA, cranial radiation or TBI, radiation-induced vasculitis, underlying disease (eg, cerebrovascular disease, sickle cell disease, adenosine deaminase deficiency), CNS relapse of the original disease, PTLD, subsequent neoplasms (local effects or paraneoplastic syndromes) and, finally by exclusion, cGVHD [238].

Survivors of TBI or cranial irradiation are at increased risk for secondary brain tumors (see Subsequent Malignant Neoplasms). Radiation,

cGVHD, and glucocorticoid treatment are major risk factors for stroke [239–243]. For asymptomatic recipients of CNS radiation (particularly at higher doses), brain magnetic resonance imaging (MRI)/magnetic resonance angiography (MRA) screening for vasculitis may be considered during shared decision making but consensus on its utility is lacking [244]. Likewise, in recipients of high-dose neck radiation, carotid ultrasound screening can be considered during shared decision making.

Hearing loss secondary to radiation, platinum agents, and other drugs or complications may develop and potentially lead to learning impairment [86,245,246]. Adenosine deaminase deficiency, osteopetrosis, lysosomal storage diseases, and leukodystrophies are also associated with hearing disabilities.

Encephalopathy, aphasia, hemiparesis, seizures, apraxia, and tremors can occur in patients who have received intrathecal chemotherapy, cranial radiation, monoclonal antibodies, or tyrosine kinase inhibitors. Progressive multifocal leukoencephalopathy (PML) is caused by JC polyomavirus and has been associated with alemtuzumab, ATG, or rituximab therapy [247–252]. Opportunistic bacterial, toxoplasmosis, and viral infections (eg CMV, HHV-6) can lead to serious morbidity [253,254]. Neurologic complications may impact cognitive function, affecting memory, concentration, speech and language skills, spatial abilities, and executive function [255–258]. Major risk factors include female sex, younger age at HCT, extensive cGVHD, use of narcotics, glucocorticoids, antidepressants, sedatives, and TBI in

Table 10
Neurological, Cognitive Complications, Psychosocial Health, and Quality of Life

Recommendation	Grade	Ref	Comments
<ul style="list-style-type: none"> Clinical assessment for symptoms or signs of peripheral and CNS dysfunction at 6-12 months after HCT and at least yearly thereafter 	2A		<ul style="list-style-type: none"> Earlier and more frequent evaluation to be considered in high-risk patients. Careful history/ examination/ review of systems/ medication history and assessment of time of onset of neurological signs and symptoms during survivorship visits.
<ul style="list-style-type: none"> Patients with exposures to head and neck irradiation, platinum chemotherapy, aminoglycosides, or an inherited condition associated with hearing disability, audiologic evaluation within 1st year post-HCT. Counsel patients about hearing loss prevention and to seek assessment for new symptoms. 	2A	[408]	<ul style="list-style-type: none"> Follow-up evaluations as clinically warranted. Hearing loss may be present in ADA deficiency prior to HCT and require developmental support, regular otolaryngology, and audiologic assessment post-HCT
<ul style="list-style-type: none"> Perform Childhood cognitive developmental milestones \geq annually. Neurocognitive testing and educational/vocational progress assessment in pediatric survivors 	2A		<ul style="list-style-type: none"> Strongly consider before returning to work/school, major changes in school (ie, moving from elementary to middle school), or changes in school performance
<ul style="list-style-type: none"> Query adults annually for cognitive function changes which may be subtle. Neuropsychological testing and imaging should be considered in cases of reported functional impairment. 	2A	[302]	<ul style="list-style-type: none"> Inquire about difficulties multitasking, attention, remembering things or whether thinking feels slow. Exclude reversible causes of cognitive decline: depression, fatigue, insomnia, or medication toxicity. SCD: offer neurocognitive testing, if available.
<ul style="list-style-type: none"> Perform CGA pre-HCT and at 6 months and 1 year post-HCT. 			<ul style="list-style-type: none"> To identify patients more likely to benefit from enhanced toxicity risk prediction and aid treatment decision making.
<ul style="list-style-type: none"> At each survivorship visit, review current symptom patterns, distress, medications, comorbidities, and physical activity. 	2A		<ul style="list-style-type: none"> Minimum: days +100, +180, and +365, then annually.
<ul style="list-style-type: none"> Discuss medication adherence and potential at each visit. 	2A		
<ul style="list-style-type: none"> Set incremental goals for healthy diet, activity, weight management. Encourage adequate sleep and age-appropriate preventative measures. Assess those with significant physical, visual, or auditory disabilities for appropriate support services and medical equipment needs. 	2A		<ul style="list-style-type: none"> ie, vegetables, fruits, whole grains, low in excess sugars, dried foods, red/processed meat, and dietary supplements.
<ul style="list-style-type: none"> General screening of patient mental health, with standardized questionnaires (eg, NCCN distress thermometer, survivorship questionnaire). 	2A	[301,302]	<ul style="list-style-type: none"> No gold standard for screening mental health after HCT; take care to not overburden patients with PRO tools. To guide clinical investigations or behavioral or psychological support, particularly if multiple somatic complaints, new GVHD, major life events, or treatment changes.
<ul style="list-style-type: none"> Regularly inquire to level of spousal/caregiver psychological adjustment, family functioning, educational, vocational activities, and financial toxicity. 	2A		<ul style="list-style-type: none"> Appropriate referral if necessary. Offer peer support and return to work/school programs
<ul style="list-style-type: none"> For AYA, provide transition of care education and plans. 	2A		

CGA, comprehensive geriatric assessment; PRO, patient-reported outcome.
Key references and further reading: [238,255–258,267,299,301]

children [12,257]. Other potential risk factors include blinatumomab, intrathecal chemotherapy, posterior reversible encephalopathy syndrome, TA-TMA, and history of immune effector cell-associated neurotoxicity syndrome.

In addition to CNS complications, survivors less commonly develop immune mediated manifestations of peripheral nervous system, such as polymyositis, myasthenia gravis (*see muscle and connective tissue complications*) and chronic inflammatory demyelinating polyneuropathy, usually developing in association with tapering IST [259–264]. Guillain-Barre-like syndrome with peripheral neuropathy and chronic demyelinating polyneuropathy related to GVHD have also been reported [265–267].

PSYCHOSOCIAL HEALTH AND QUALITY OF LIFE

Understanding patient perspectives on health related QoL is an integral part of survivorship care (Table 10, Supplementary Table S15). Physical function limitations can adversely affect a survivor's ability to carry out daily tasks [12]. Approximately 10% of survivors report somatic distress >10 years post-HCT, with cGVHD, glucocorticoid exposure, and depression being risk-factors [12,268,269]. As many as 68% of patients report fatigue which is one of the most consistent symptoms negatively impacting QoL [270]. Risk factors for fatigue include female gender, cGVHD, younger age, and chronic pain [12,271]. Similarly, up to half of survivors report sleep disturbance that does not seem to improve over time, and has been associated with female gender, older age, divorced status, unemployment, depression, distress, and autologous HCT [12]. Pain is reported among ~25% of survivors, often associated with musculoskeletal symptoms [12,237,269].

Anxiety and post-traumatic stress disorder (PTSD) affect 5–10% of long-term survivors [268,272], whereas depression seems to gradually increase over time, affecting 10–30% [5,273]. PTSD has been associated with GVHD and prolonged hospitalizations [272]. Anxiety has been associated with female gender, poor reported health status, lower household income, lower education, prednisone exposure and cGVHD [268,273]. Depression is more frequent in male patients, those with poor functional status, lower household income, less education, TBI, prednisone exposure and cGVHD [268,273,274].

While lower social function scores have been reported in survivors compared to controls, others have reported excellent support from family and friends [255,274,275]. 60–80% of survivors are

able to resume social roles, such as returning to work and school, but up to one third of survivors report worrying about being able to maintain employment [274,276–284]. Financial burden of HCT is a major concern for >20% of North American survivors and is relevant in other locations as well [255,285–288]. AYAs seem to report lower social well-being and more difficulties establishing themselves in the labor market [281,287,289]. Pre-HCT lack of employment, less education, medical disability, late-effects, fatigue, pain, mental distress, GVHD, relapse and having a manual job have been associated with lower chances of post-HCT employment [279,281–284,287,289–295]. Financial difficulties are associated with worse physical and mental functioning, adverse medical outcome, and increased severity of GVHD [268,292,296–298].

There is no standard way of addressing perceived health status in HCT survivors, but many standardized patient-reported outcome questionnaires have been utilized, with accumulating evidence supporting the NIH-supported Patient-Reported Outcomes Measurement Information System (PROMIS) [299,300]. Additionally, though not specifically validated in HCT, the NCCN distress thermometer can be used to triage patient concerns [301,302]. For fatigue, NCCN survivorship guidelines may be helpful [302]. Sleep disorders can be investigated by detailed history and assessment of symptoms and potential interventions include review of medications, reviewing sleep/wake timing, physical activity, caffeine and other substance use and providing coping strategies such as relaxation and meditation techniques. For pain, appropriate mitigation strategies include non-pharmacologic interventions (eg, massage, physical therapy, acupuncture) and/or analgesics, with nonopioids prioritized [302]. Family members (including siblings) and informal caregivers can be even more affected by mental health issues than patients themselves, and their own QoL cannot be inferred from the patients' results; referral to appropriate providers may be indicated [303–306].

It is essential that survivors be able to rely on their standard support system and also recognize they may get additional help from interacting with survivors with similar experiences. Online peer mentor programs are available (Appendix 2). Return to work programs can be instrumental, but practices vary widely (Appendix 3) [307,308]. Patients should be encouraged to evaluate their working/educational goals and identify barriers, working with human resources through their

employer, occupational/vocational therapists, social workers, and financial counselors [302].

SUBSEQUENT MALIGNANT NEOPLASMS

HCT survivors have a 4- to 11-fold increased risk of developing subsequent malignant neoplasms (SMNs) compared with the general population [93,309]. Among allogeneic HCT recipients, the incidence of SMNs increases from 3.5% at 10 years to 12.8% at 15 years post-HCT [91,94,309]. SMNs can be categorized as hematologic tumors (tMNs, PTLD) or solid tumors (Table 11, Supplementary Table S16).

The overall incidence of tMNs is estimated to be 4% at 7 years post-HCT, with a median occurrence at 2.5 years post-HCT [310]. Recipients of pre-HCT alkylating agents (particularly etoposide or cyclophosphamide), and possibly post-HCT cyclophosphamide are at higher risk [311,312]. Survivors have a higher risk of developing tMN after autologous HCT, but tMN rarely can arise from donor hematopoiesis in allogeneic HCT recipients [313,314]. Patients who were conditioned with TBI, received ≥ 3 lines of chemotherapy, were poor stem cell mobilizers, or received lenalidomide maintenance for myeloma may be at higher risk for tMN [315–318]. The cumulative incidence of PTLD is 1% at 10 years after HCT and is associated with greater donor-recipient HLA disparity, T cell depletion, and GVHD [319]. Patients with primary immune deficiency also are at higher risk of developing lymphomas. Recommendations on prevention and treatment of PTLD have been developed by the sixth European Conference on Infections in Leukemia and are described in detail elsewhere [320].

The incidence of solid cancers increases from approximately 2% at 10 years to 3% to 5% at 15 years post-HCT, varying widely based on exposure, family/genetic history, age at HCT, and time since HCT [95,321,322]. Younger age at HCT, TBI, female sex, and cGVHD are risk factors for thyroid cancer in survivors [179,323]. Survivors have a 7- to 16-fold higher risk for oral cancer compared to the general population; this risk is particularly increased in survivors with cGVHD and/or Fanconi anemia [90,91,94,95,324–326]. Genital cancer risk is also increased in recipients of reduced-intensity conditioning and limited field radiation and in patients with cGVHD and/or a diagnosis of Fanconi anemia [89,91,327]. Allogeneic HCT recipients are at an increased risk for gastrointestinal malignancies, with one report citing an standardized incidence ratio in survivors at 5 to 10 years post-HCT of 74.0 for cancer of the esophagus, 46.6 for cancer of the oral cavity, and 2.3 for colon

cancer [328]. Patients exposed to TBI are at risk of colorectal cancer, and children exposed to TBI can develop polyps at an early age [144,329]. Patients with inflammatory bowel disease are at very high risk of developing colon cancer, although it remains to be seen whether similar issues occur in patients with gastrointestinal GVHD. Colonoscopy has been shown to be cost-effective in patients who received abdominal radiation exposure [143]. Recipients of chest irradiation (eg, Hodgkin lymphoma) are at increased risk for breast cancer [330,331]. Recipients of TBI without other radiation are also at increased risk [332]. Although rare, breast cancer in male childhood cancer survivors may be related to radiation therapy [333]. Anthracycline exposure, endogenous hormones and hormone replacement, and family history also should be considered when determining a patient's overall risk [334,335].

The 20-year post-HCT cumulative incidence is 6.5% for basal cell carcinoma and 3.4% for cutaneous squamous cell carcinoma [336]. The risk of melanoma is also significantly increased [337]. Areas of previously irradiated skin are the most vulnerable to developing carcinomas, further exacerbated by a history of excessive sun exposure and chronic skin GVHD, which itself can be triggered or aggravated by sun exposure. The role of IST in precipitating skin cancer is another a concern. Screening and prevention of skin malignancies mimic the guidance offered for skin cGVHD (see Dermatologic Complications).

Survivors also are at higher risk for CNS tumors and sarcoma. Meningioma is the most frequent CNS tumor, although more aggressive histologies may occur [338,339,340]. Heightened awareness for symptoms should be emphasized in patients after cranial irradiation or TBI. Survivors are at risk for secondary bone cancers, with a standardized incidence ratio of 8.5 to 13 [341,342]. Risk factors may include underlying cancer predisposition syndromes (eg, Li-Fraumeni syndrome, Diamond-Blackfan anemia) and radiation therapy. Clinicians should maintain a high level of suspicion in patients who present with relevant symptoms [342].

SPECIAL POPULATIONS

Although there are many commonalities in risk factors and chronic health conditions in all HCT survivors, certain populations are at elevated risk for late effects due to their underlying disease, treatment regimen, or age-related late effects and warrant long-term follow-up with multidisciplinary population-specific teams. Appendix 4 outlines considerations for special populations,

Table 11
Subsequent Malignant Neoplasms

Recommendation	Grade	Ref	Comments
<ul style="list-style-type: none"> • Counsel about SMN risk and encourage recommended screening. 	2A		<ul style="list-style-type: none"> • Based on treatment exposures, time from exposure, cGVHD history, and other modifying factors.
<ul style="list-style-type: none"> • Encourage avoidance of high-risk behaviors, unhealthy diet. 	2A		<ul style="list-style-type: none"> • eg, tobacco and vaping, passive tobacco exposure, alcohol abuse; high-fat/low-fiber diet.
<ul style="list-style-type: none"> • If risk not otherwise modified by HCT history, screening as regionally indicated and clinically based on other underlying medical, family, and genetic history. 	2A		
<ul style="list-style-type: none"> • Patients at risk for tMN: annual CBC until at least 10 years post-HCT. 	2B		<ul style="list-style-type: none"> • Consider more frequent screening in those with greater risk (ie, chemotherapy exposures, age; autologous HCT). • Attention to unexplained cytopenias, macrocytosis, or cellular atypia.
<ul style="list-style-type: none"> • Prevention of PTLD includes EBV-DNA screening in high-risk patients, preemptive treatment with rituximab. 	2B		<ul style="list-style-type: none"> • eg, ATG, alemtuzumab, ex vivo T cell depletion. • Evaluation includes lymph node palpation and review of B symptoms (fever, drenching night sweats, $\geq 10\%$ weight loss over 6 months).
<ul style="list-style-type: none"> • Oral exam should be part of annual survivor exam. • Advise reporting non-healing oral lesions, leukoplakia, localized pain, changes in mucosal color/ texture. • HPV vaccination according to country-specific general population recommendations, unless otherwise contraindicated 	2A	[58,94,95,342,409]	<ul style="list-style-type: none"> • Consider every 6 months screening in patients at high risk for oral cancer (oral cGVHD, tobacco use, FA). • HPV's role in mucocutaneous/ genital cancers post-HCT unclear. • Endoscopic screening for esophageal cancer may be considered in high-risk patients (cGVHD receiving prolonged immunosuppression, symptoms of gastroesophageal reflux, dysphagia).
<ul style="list-style-type: none"> • Cervical cancer screening based on age and risk factors. 	2A	[325]	<ul style="list-style-type: none"> • FA: annual pap smear, gynecologic screening starting in adolescence.
<ul style="list-style-type: none"> • Pediatric survivors <age 45 years and RT exposed: CRC screening colonoscopy or multitarget stool DNA screening at 5 years post-RT exposure or age 30 years, whichever occurs later. • Adult survivors: as part of shared decision making, consider exposed to abdominal/ pelvic RT or TBI who underwent HCT at ≥ 25 years of age, colonoscopy beginning 5 years post-RT exposure and no later than age 45 years 	2B	[342,410,411]	<ul style="list-style-type: none"> • Screen patients without RT exposure according to general population guidelines for their geographic region, individual risk factors, and family history. • Screen patients with colon CPS (eg, hereditary nonpolyposis colorectal cancer, FAP, DBA) based on underlying CPS. • Risk of subsequent CRC in those exposed to TBI likely dose-dependent; lower doses may not increase the risk of CRC to level that risk/benefit of early colonoscopy warranted.

(continued)

Table 11 (Continued)

Recommendation	Grade	Ref	Comments
<ul style="list-style-type: none"> Female recipients of TBI or chest RT should begin breast cancer screening with mammogram and breast MRI (if available) at age 25, or 8 years post-RT, whichever occurs later, but no later than age 40. In high-risk individuals, discussion with appropriate experts on risk reduction strategies (ie, prophylactic mastectomy, prophylactic medical therapies, lifestyle). 	2A	[330–335,412,413,342]	<ul style="list-style-type: none"> Female HCT survivors without risk factors should participate in regular annual mammogram/ clinical breast exam according to general population guidelines for geographical region and individual risk factors including family history. Counsel on additional breast cancer risk factors (ie, BRCA1/2, family history).
<ul style="list-style-type: none"> Patients should have an annual thyroid exam and review of potential symptoms of thyroid cancer. As part of shared decision making, consider ultrasound screening in patients who received thyroid RT. 	2A	[323,414]	<ul style="list-style-type: none"> Routine use of thyroid ultrasound may not provide additional benefit; some groups recommended. Screen patients with inherited thyroid CPS (eg, multiple endocrine neoplasia, FAP) based on underlying condition.
<ul style="list-style-type: none"> As part of shared decision making, consider meningioma screening in patients who received CNS RT. 	2B	[338,339,340]	
<ul style="list-style-type: none"> FA survivors with mutations in <i>BRCA2</i> (<i>FANCD1</i>) or <i>PALB2</i> (<i>FANCN</i>) require screening for specific solid cancers. 	2A	[325,415–418]	

CPS, cancer predisposition syndrome; CRC, colorectal cancer; DBA, Diamond-Blackfan anemia; HPV, human papilloma virus; SMN, subsequent malignant neoplasm.

Key references and further reading: [65,320,332,342,409,414,419]

including hemoglobinopathies, marrow failure syndromes, inborn errors of immunity, enzymopathies, metabolic and other disorders, autoimmune disorders, myeloma, amyloidosis, infants, AYAs, and older adults (ie, geriatric population). [Appendix 5](#) provides a nonexhaustive list of additional references that may be helpful in managing the late effects of these specific populations, along with specific recommendations for these populations.

MODELS OF SURVIVORSHIP CARE DELIVERY

As survivorship care has become an integral part of cancer treatment, guidelines from the Foundation for the Accreditation of Cellular Therapy and the Joint Accreditation Committee of International Society for Cell and Gene Therapy and European Society for Blood and Marrow Transplantation (FACT-JACIE) mandate the monitoring and treatment of late effects after HCT, including the transition from pediatric care to adult care providers [343]. Survivorship care is being increasingly recognized by patients as a basic need [288,344,345], and evidence for the added value of such practice is emerging. Indeed,

recipients of HCT performed at centers with survivorship programs have improved survival [346]. However, at present in the United States, only 45% of programs have an HCT-specific long-term follow-up clinic [347]. Several different models of late effects follow-up have been proposed [4,11,347]; however, barriers to the establishment of survivorship clinics include lack of expertise, logistical challenges, and financial and reimbursement issues [347]. The implementation of programs and specific guidelines remains a challenge, and ensuring that survivors receive the recommended guidance is crucial [348–350].

Patients indicate a preference for holistic care, ideally with continued direct contact with the transplant center, particularly when they have developed GVHD, but this can be challenging when they live remote from their HCT team [288,344,345]. Survivorship care plans have been shown to decrease cancer-related distress and improve mental health in long-term survivors, although they do not exclusively replace comprehensive survivorship care [351]. Models to overcome barriers using telehealth, mobile apps, and wearable devices are currently being tested

[352–356]. However, technology use and uptake may be challenging, and patients often value human contact [288,345,357,358]. In summary, a specific optimal survivorship care model has not been determined, and different models continue to evolve based on HCT program and system strengths and abilities.

FUTURE DIRECTIONS

As the transplantation and cellular therapy field continues to evolve and expand, so will the need for survivorship care. The long-term impact of novel small molecule cancer therapies and immunotherapies have not yet been well studied but have mechanisms of action generally distinct from conventional chemotherapy; some have idiosyncratic side effects particularly relevant to specific populations (eg, acute endocrine complications from certain immunotherapies), but long-term impacts are unclear [359]. Multiple chimeric antigen receptor T cell therapies are available, and indications for these and additional cellular therapy products are expanding rapidly. Cellular therapies may be associated with acute and subacute postinfusion complications mostly from altered immune function (eg, cytokine release syndrome, immune effector cell-associated neurotoxicity syndrome) or from the elimination of cells expressing common antigens with the target cells (eg, B cell aplasia, CD19⁺ cell therapy) [77]. Gene therapies using an autologous HCT platform have moved to late-stage clinical trial testing, with some products now approved by regulatory agencies. Most clinical trial protocols require prolonged follow-up and are typically geared toward monitoring the underlying disease, specific genomic modifications, and surveillance for clonal hematopoiesis/tMN [360,361]. Although these trial-mediated outcomes will provide crucial information for the long-term care of these patients, routine post-HCT care—immune reconstitution and revaccination, monitoring for late-effects of conditioning chemotherapy—remains equally important. Given that many participants in these clinical trials have traveled great distances to enroll, often internationally, ensuring ongoing local follow-up and partnership with the research team is critical. Further, as these therapies become more established it will become increasingly important to provide data and encourage provider awareness of the long-term issues. All these novel approaches are likely to impact the range of late effects in future HCT and cellular therapy survivors, and further investigation is of the utmost importance.

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SUPPLEMENTARY MATERIALS

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