

## Hematologic Malignancy Education for Stem Cell Transplantation Nurses

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**P**atient presentations and treatment protocols in the stem cell transplantation (SCT) practice setting are constantly evolving, multiple clinical trials are ongoing, state-of-the-art advanced care changes rapidly, and care of the patient before, during, and following transplantation is complex. Nursing practice in this population requires a specialized knowledge of individual protocols coupled with a broad understanding of disease presentation and treatment for the variety of diagnoses for which patients undergo SCT. The sheer volume of information related to the care of patients undergoing SCT is challenging for the most experienced nurses and, particularly, for new oncology nurses. The complexity and diversity of the SCT patient population and their care parallels the increasing generational, educational, and professional backgrounds of nursing staff, all of which necessitate the provision of consistent and evidence-based education for safe and effective patient care.

Both new oncology nurses and those unfamiliar with complex treatment protocols must quickly integrate a breadth of knowledge along with an advanced set of clinical skills specific to SCT. In addition, increasing subspecialization may require novice nurses to quickly become experts, particularly in the oncology practice setting, where the numerous dose-intensive conditioning regimens administered before transplantation are complicated and require knowledge of individual protocols and medications for specific cancer diagnoses.

To provide SCT nurses, particularly those new to the practice setting, with contemporary evidence-based information on the prominent underlying hematologic malignancies for which individuals undergo SCT, as well as the complications and oncologic emergencies for which these populations are at increased risk, an educational reference chart was developed by the first author of this article.

### Background and Significance

Nurses' lack of knowledge on how to locate accurate and reliable information, lack of access to the tools necessary to search for evidence-based information, and insufficient time to resolve these shortcomings are frequent barriers to meeting the demands of evidence-based practice (Pravikoff, Tanner, & Pierce, 2005). Pravikoff et al. (2005) reported that most RNs recognize the need for evidence-based information in their practice, but the majority admitted to obtaining that information from a peer or colleague rather than from a printed or electronic resource, largely because of nurses' trust in their colleagues.

Nurses can access information in many ways. For example, some nurses may be inclined to search the Internet for information, which can provide unreliable, non-evidence-based data. In Pravikoff et al. (2005), 73% (n = 741) of nurse respondents stated they sometimes, frequently, or always use the Internet to obtain clinical information. In the acute care practice setting, where new graduate nurses composed more than 10% of a typical hospital or health-care system staff, this instinctual use of the Internet for clinical resources may, in part, account for the fact that only 10% of hospital and health system executives felt that newly graduated nurses were completely prepared to provide safe care (Berkow, Virkstis, Stewart, & Conway, 2009). This is particularly true for SCT and cellular therapies, services that address the needs of patients from a variety of oncologic backgrounds.

In many ways, SCT nurses are isolated from the specialty areas from which their patient populations are derived in the context of a large, subspecialized National Cancer Institute (NCI)-designated comprehensive cancer care center. An increasing number of patients with hematologic malignancies, such as leukemia, lymphoma, and myeloma, undergo SCT as a treatment option. Therefore, an

evidence-based working knowledge of these malignancies is essential for SCT nurses to effectively understand and care for patients undergoing transplantation to treat these disease presentations. Although a wealth of resources are available to nurses at the authors' institution, most require accessing databases, which can be time consuming.

### Educational Chart Development

To address the information needs of an SCT unit nursing staff of almost 150 individuals within the authors' institution, 23% of whom were newly graduated or within the first two years of their nursing career, the first author developed a comprehensive quick-reference chart (see Table 1) regarding the prominent underlying hematologic malignancies for which individuals undergo SCT, as well as the complications and oncologic emergencies for which these populations are at increased risk.

Prior to the development of the chart, an educational needs assessment was provided to the nursing staff of this 52-bed unit to determine their learning needs related to primary hematologic diagnoses and oncologic emergencies. The assessment also queried nurses as to the preferences for the formatting of such information. The assessment results were overwhelmingly in favor of an easily accessible quick reference for the purposes of education in the clinical setting. Using this feedback as a guide, the first author then identified the most common hematologic diseases for which SCT is used as treatment. Primary information was obtained based on a search of the literature to ensure evidence-based content regarding oncology and SCT and to establish background on the hematologic diagnoses most commonly treated with SCT. In addition, a review of the most common complications and oncologic emergencies associated with these hematologic malignancies and their management was conducted. With the help of a

medical librarian, a literature search was conducted using the electronic databases PubMed and CINAHL®, which yielded the articles referenced in the chart (Rimkus, 2009; van Besien, 2009; Yarbrow, Gobel, & Wujcik, 2011). Content of the chart was then cross referenced with information from the NCI, American Cancer Society, and the National Comprehensive Cancer Network to ensure content consistency and validity. Once compiled, the chart was reviewed by advanced practice nurses at the University of Texas MD Anderson Cancer Center who specialize in leukemia, lymphoma, myeloma, and SCT to ensure content validity with current practice.

## Implementation

On approval of the content, the chart was constructed with a color-coordinated system to allow for quick reference and ease of use. Beginning in February 2009, the chart was provided to the SCT nursing staff in unit-based pod reports, and a 3 x 4-foot laminated poster was placed on each of the four nursing pods that constituted the unit, making the information easily accessible to all nursing staff. In addition, the chart is included in nursing orientation binders for nurses who are newly employed on the unit.

For quality improvement purposes, a postimplementation survey was conducted with the SCT nurses to assess the chart's benefits, shortcomings, and ease of use, and to determine whether it addressed the items reported in the educational needs assessment and improved nursing knowledge and continuity of information delivery to patients transitioning from other services. The postimplementation questionnaire included questions related to the chart's usefulness as a reference in educating patients and the chart's ease of use and accessibility, to which nurses' responses were overwhelmingly favorable.

## Accomplishments

The hematologic malignancies educational chart was developed and completed through the effort of staff nurses whose primary objective was to provide accurate information in an easy-to-use and readily accessible format for health-care clinicians caring for patients undergoing SCT. The driving force for this project was to address the educational needs of a diverse nursing staff caring for a patient population with increasingly complex needs and, in doing so,

to enhance patient education and safe practice.

Feedback from nursing staff as well as from physicians and multidisciplinary members of the transplantation service at MD Anderson has been positive and supportive. In addition, the chart has been distributed to other units at the institution that are involved in hematology and transplantation, and it has been an invaluable tool in providing information to off-service units receiving patients from these populations. When presented at the 2010 American Society for Blood and Marrow Transplantation (ASBMT) annual conference, the chart received positive feedback from other institutions interested in using it as a resource.

## Challenges

The development of the hematologic malignancies educational chart did not come without challenges, such as the format in which it can be made available and the ability to keep the chart's information current. Because of the wealth of data contained in the chart, it can be comfortably read only in a large poster form, making it difficult to create an easily accessible pocket-sized guide, as some nurses have requested. In the meantime, a large-print spreadsheet version will be placed in the orientation binder for new patient care providers in the SCT unit. Funding is being sought to accommodate this request as well as to provide access to the chart for staff in other hospital subspecialties caring for SCT patients. In addition, the information on this educational chart must be continually updated with evidence-based information that reflects current practice, a particularly challenging task given the rapid evolution of cancer treatment, interventions, and management. To address this challenge, a formal plan for revisions is being established to ensure that the chart is regularly reviewed and updated by content specialists, as well as through an updated literature search.

## Implications for Practice

Patients who undergo SCT receive highly complex treatment regimens and are at great risk for life-threatening complications. Knowledge of evidence-based treatments for specific hematologic cancers and of potential complications is imperative for nurses to be able to accurately identify the sequelae of complications (Rimkus, 2009). Quality evidence-based nurse education also contributes to knowledgeable patient education and

evidence-based care. The hematologic malignancy educational chart addresses many of the major concerns of nurses in having a standardized, evidence-based source of quickly referenced information related to their patient population. The chart provides carefully reviewed, accurate information concerning patients' diseases and prepares nurses for possible emergent situations arising from them. This tool is applicable to both inpatient and outpatient transplantation settings, is useful in solid tumor oncology and other medical practice settings that receive overflow patients from SCT units, and may be used in any context in which patients with cancer are cared for, even beyond the oncology setting.

*The authors gratefully acknowledge Holly Benson, RN, OCN®, for her contribution and implementation of the disease chart, Lyn Greenberg, APN, for her expertise in the area of leukemia, and Geri LoBiondo-Wood, PhD, RN, FAAN, and Judith K. Payne, PhD, RN, AOCN®, for their valuable insight.*

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Digital Object Identifier: 10.1188/11.ONF.401-402

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**Table 1. Stem Cell Transplantation Nurses' Hematology Reference Chart**

Diagnosis	Etiology and Risk Factors	Pathophysiology	Clinical Manifestations	Treatment and Management
<b>Common Primary Diagnoses in the Stem Cell Transplantation Population</b>				
Acute lymphocytic leukemia	<ul style="list-style-type: none"> <li>EBV, human T-cell leukemia virus (HTLV-1), exposure to radiation and chemicals, and chromosomal abnormalities</li> </ul>	<ul style="list-style-type: none"> <li>Malignant disease of lymphoid progenitors; originates in the marrow, thymus, and lymph nodes</li> <li>L1: childhood (pre- B and T cell); 85% of cases are children</li> <li>L2: adult type (pre- B and T cell)</li> <li>L3: Burkett type (B cell)</li> <li>Diagnosis with marrow blasts is greater than or equal to 20%.</li> </ul>	<ul style="list-style-type: none"> <li>Recurrent infections, pancytopenia, fever, fatigue, weight loss, swollen and bleeding gums, slow-healing abrasions, petechiae and bruising, prolonged bleeding time, bone pain, organ infiltration (abdominal pain), and swollen lymph nodes</li> </ul>	<ul style="list-style-type: none"> <li>Hyper-CVAD, methotrexate and cytarabine, CNS prophylaxis (Ara-C or methotrexate intrathecal)</li> <li>Add rituximab if CD 20 positive (Burkett)</li> <li>Add imatinib if Philadelphia-chromosome-positive</li> <li>Bone marrow transplantation recommended following first remission</li> </ul>
Acute myelogenous leukemia and acute non-lymphocytic leukemia	<ul style="list-style-type: none"> <li>Etiology unclear</li> <li>Genetics</li> <li>Down syndrome</li> <li>Correlation with benzene, chemotherapy, radiation, and tobacco exposure</li> <li>Peak age is in the 50s.</li> </ul>	<ul style="list-style-type: none"> <li>Disease of the pluripotent myeloid stem cell; chromosomal abnormalities of undifferentiated stem cells</li> <li>Early stage: depletion of normal myeloid cells (pancytopenia)</li> <li>Later stage: Lack of apoptosis in malignant blast cells leads to accumulation in marrow, blood, and organs.</li> <li>Blast cells unable to fight infection; more than 30% blasts in marrow is diagnostic.</li> </ul>	<ul style="list-style-type: none"> <li>Recurrent infections: susceptible to cytomegalovirus, <i>Pneumocystis carinii</i> pneumonia, herpes simplex virus, and vancomycin resistant enterococcus</li> <li>Pancytopenia, fever, fatigue, weight loss, swollen or bleeding gums, slow-healing abrasions, petechiae and bruising, prolonged bleeding time, bone pain, organ infiltration (abdominal pain), and swollen lymph nodes</li> </ul>	<ul style="list-style-type: none"> <li>Cytarabine plus daunorubicin; cytarabine plus idarubicin; cytarabine, daunorubicin, and thioguanine; and mitoxantrone plus etoposide</li> <li>For CNS, cytarabine or methotrexate</li> <li>Autologous or allogeneic bone marrow transplantation</li> <li>Blood product support (red blood cells, platelets, and WBCs), broad spectrum antimicrobial, and antifungals</li> </ul>
Acute promyelocytic leukemia	<ul style="list-style-type: none"> <li>Subtype of acute myelogenous leukemia</li> </ul>	<ul style="list-style-type: none"> <li>Myeloid cell line; pre- B and T cell</li> </ul>	<ul style="list-style-type: none"> <li>Recurrent infections, pancytopenia, fever, fatigue, weight loss, swollen or bleeding gums, slow-healing abrasions, petechiae and bruising, prolonged bleeding, bone pain, organ infiltration, swollen lymph nodes, and bleeding (disseminated intravascular coagulation, fibrinolysis, and proteolysis)</li> </ul>	<ul style="list-style-type: none"> <li>Arsenic trioxide (the most common treatment), all-transretinoic acid, interleukin-2, and anthracycline-based chemotherapy regimens</li> <li>Autologous or allogeneic stem cell transplantation</li> </ul>
Burkitt lymphoma (non-Hodgkin lymphoma)	<ul style="list-style-type: none"> <li>Median survival is weeks, if untreated.</li> <li>Related to EBV</li> </ul>	<ul style="list-style-type: none"> <li>Very aggressive and fast growing; involvement with bone marrow, meninges, CNS, and blood</li> <li>Risk for tumor lysis; CNS is a common site of relapse.</li> <li>Potentially curable with aggressive therapy</li> </ul>	<ul style="list-style-type: none"> <li>Commonly presents at extranodal sites; typically present with rapidly growing, bulky disease.</li> <li>Elevated serum uric acid and LDH levels are commonly found.</li> </ul>	<ul style="list-style-type: none"> <li>CODOX-M</li> <li>Hyper-CVAD alternating with methotrexate and cytarabine</li> <li>High risk: CODOX-M with ifosfamide, etoposide, and cytarabine</li> <li>Rituximab and hyper-CVAD alternating with methotrexate and cytarabine</li> </ul>

(Continued on the next page)

CNS—central nervous system; CODOX-M—cytoxan, vincristine, doxorubicin, and high-dose methotrexate; CVC—central venous catheter; EBV—Epstein-Barr virus; hyper-CVAD—cyclophosphamide, vincristine, doxorubicin, and dexamethasone; LDH—lactic dehydrogenase; R-CHOP—rituxan, cyclophosphamide, doxorubicin, vincristine, and prednisone; R-ESHAP—rituxan, etoposide, methylprednisolone, cytarabine, and cisplatin; WBC—white blood cell

**Table 1. Stem Cell Transplantation Nurses' Hematology Reference Chart (Continued)**

Diagnosis	Etiology and Risk Factors	Pathophysiology	Clinical Manifestations	Treatment and Management
<b>Common Primary Diagnoses in the Stem Cell Transplantation Population (Continued)</b>				
Chronic lymphocytic leukemia	<ul style="list-style-type: none"><li>• No known occupational or environmental risk factors</li><li>• Genetic link: high familial risk</li><li>• Most common adult leukemia</li></ul>	<ul style="list-style-type: none"><li>• Progressive accumulation of nonproliferating, morphologically mature but immunologically less mature lymphocytes</li></ul>	<ul style="list-style-type: none"><li>• 40% asymptomatic at diagnosis</li><li>• B or constitutional symptoms</li><li>• Fatigue; weakness; malaise; bacterial infections; herpes zoster; exaggerated skin reaction to insect bites or bee sting; and enlarged spleen (less frequently hepatomegaly), abdominal discomfort, and early satiety</li></ul>	<ul style="list-style-type: none"><li>• FCR (fludarabine, cyclophosphamide, and rituxan), FC (fludarabine and cyclophosphamide), CFAR (cyclophosphamide, fludarabine, alemtuzumab, and rituximab (campath—found to “clean up” minimal residual disease), and stem cell transplantation</li></ul>
Chronic myelogenous leukemia	<ul style="list-style-type: none"><li>• Also called chronic granulocytic leukemia</li><li>• Chromosomal abnormalities</li><li>• No known specific cause except exposure to ionizing radiation</li></ul>	<ul style="list-style-type: none"><li>• Disorder of the myeloid stem cells characterized by marked splenomegaly and increased production of granulocytes</li><li>• About 90% of patients are Philadelphia-chromosome-positive.</li><li>• Mature-appearing dysfunctional cells in three phases: chronic, accelerated, and blast phase; blast crisis presents like acute leukemia, granulocytes replacing marrow</li><li>• Average age of diagnosis is in 30s–40s; most diagnoses are in the chronic phase.</li></ul>	<ul style="list-style-type: none"><li>• Initial signs and symptoms: massive splenomegaly, fatigue, malaise, headache, weakness, weight loss, bone or joint pain, excessive (night) sweats, fever, abdominal pain (left upper quadrant), early satiety, vague abdominal fullness, hepatosplenomegaly, lymphadenopathy, easy bruising, petechia, elevated basophils and neutrophils, blasts present, high B<sub>12</sub> levels, and low alkaline phosphatase</li><li>• Chronic phase: high WBC count (10–150 times the normal level).</li><li>• Blastic phase presents with hypercatabolism, pancytopenia, and infection.</li></ul>	<ul style="list-style-type: none"><li>• Interferon alpha, imatinib, dasatinib, nilotinib, and stem cell transplantation (last resort, mostly allogeneic)</li></ul>
Diffuse large B-cell lymphoma (non-Hodgkin lymphoma)	<ul style="list-style-type: none"><li>• Median age is 64 years; affects more men than women</li><li>• Overall survival is 50% at five years.</li></ul>	<ul style="list-style-type: none"><li>• Most common type of non-Hodgkin lymphoma</li><li>• Encompasses a diffuse group of large, neoplastic B lymphocytes with large nuclei.</li><li>• Destruction of the normal architecture of the involved lymph node occurs in a diffuse pattern.</li><li>• Frequently express B-cell markers (CD19, CD20, CD22, CD79a)</li></ul>	<ul style="list-style-type: none"><li>• Disseminates rapidly</li><li>• “B” symptoms (unexplained fever, night sweats, and unexplained weight loss)</li><li>• Anemic</li><li>• Most present with nodal and extra nodal (gastrointestinal, skin, sinus, and stomach).</li><li>• Potentially curable</li><li>• Presents either as primary lymph node disease or at extranodal sites</li></ul>	<ul style="list-style-type: none"><li>• Stage I: R-CHOP six times with radiation therapy</li><li>• Stage I/II: R-CHOP 6–8 times plus radiation therapy</li><li>• Stage III/IV: clinical trial of R-CHOP and Hyper-CVAD every 14 days or 6–8 courses (rituximab eight times)</li><li>• Second line: rituxan, ifosphamide, etoposide, and carboplatin (RICE), R-ESHAP, salvage (dexamethasone, cisplatin, and cytarabine [DHAP]), and carmustine, etoposide, cytarabine, and melphalan (BEAM)</li></ul>

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Diagnosis	Etiology and Risk Factors	Pathophysiology	Clinical Manifestations	Treatment and Management
<b>Common Primary Diagnoses in the Stem Cell Transplantation Population (Continued)</b>				
Follicular lymphoma (non-Hodgkin lymphoma)	<ul style="list-style-type: none"> <li>Accounts for 35% of all non-Hodgkin lymphoma cases</li> <li>Median age is 60; median survival from diagnosis is 10 years.</li> </ul>	<ul style="list-style-type: none"> <li>Neoplasm of follicle-center (germinal-center) B cells</li> </ul>	<ul style="list-style-type: none"> <li>B-cell symptoms: unexplained fever, night sweats, and unexplained weight loss</li> <li>Lymphadenopathy (cervical, supraclavicular, and maxillary)</li> </ul>	<ul style="list-style-type: none"> <li>Treatment usually not curative. Responds well initially, but high incidence of recurrence</li> <li>Watch and wait, radiation therapy alone, chemotherapy, or chemotherapy with radiation therapy</li> <li>High risk: CODOX-M/VAC</li> <li>Rituximab and hyper-CVAD alternating with methotrexate and cytarabine</li> </ul>
Hodgkin lymphoma	<ul style="list-style-type: none"> <li>Affects more men than women</li> <li>Genetics, EBV, HIV, herpes virus 6</li> <li>Occupational (wood-working, chemical exposures)</li> </ul>	<ul style="list-style-type: none"> <li>B-cell origin (Reed-Sternberg cells)</li> <li>Nodular sclerosis (grades 1 and 2), lymphocyte-rich, mixed cellularity, lymphocyte depleted</li> </ul>	<ul style="list-style-type: none"> <li>Most present with peripheral lymphadenopathy above the diaphragm.</li> <li>B cell symptoms: unexplained fever, night sweats, unexplained weight loss (greater than 10%)</li> <li>Pruritis</li> <li>Lymphadenopathy (cervical, supraclavicular, and maxillary)</li> <li>Fatigue; pain with alcohol consumption</li> </ul>	<ul style="list-style-type: none"> <li>Staging dependent</li> <li>Radiation therapy</li> <li>Chemotherapy: doxorubicin, bleomycin, vinblastine, and dacarbazine (ABVD)</li> <li>Mechlorethamine, vincristine, procarbazine, and prednisone (MOPP)</li> <li>Stem cell transplantation</li> </ul>
Mantle cell (non-Hodgkin lymphoma)	<ul style="list-style-type: none"> <li>Median age is 60 years; median survival is 3–5 years.</li> <li>Aggressive, cure is rare.</li> </ul>	<ul style="list-style-type: none"> <li>B-cell neoplasm</li> <li>Strong surface immunoglobulin M</li> <li>Monoclonal lymphoid proliferation destroys the architecture of the lymph node.</li> <li>A nodular, diffuse, or mantle zone growth pattern is observed.</li> </ul>	<ul style="list-style-type: none"> <li>Hepatomegaly and splenomegaly are common findings.</li> <li>Most common extranodal sites reported include the gastrointestinal tract and Waldeyer ring found on the tonsils.</li> <li>Anemic</li> <li>May include gastrointestinal tract, marrow, blood, liver, brain, and cerebrospinal fluid</li> </ul>	<ul style="list-style-type: none"> <li>First line: hyper-CVAD</li> <li>Second line: fludarabine, cytoxan, and rituximab (FCR)</li> <li>Pentostatin, cytoxan, and rituximab (PCR)</li> <li>Bortezomib</li> </ul>
Mycosis fungoides mature T-cell lymphoma (non-Hodgkin lymphoma)	<ul style="list-style-type: none"> <li>Accounts for 0.5%–3% of non-Hodgkin lymphoma cases</li> <li>Median age is 55–60 years; affects more men than women</li> </ul>	<ul style="list-style-type: none"> <li>T-cell lymphocytes become cancerous and affect the skin.</li> <li>Infiltration of the epidermis and dermis with T cells</li> </ul>	<ul style="list-style-type: none"> <li>Skin lesions, initially seen on the trunk</li> <li>May involve lymph nodes, liver, spleen, lungs, and blood</li> <li>Bone marrow involvement is rare.</li> </ul>	<ul style="list-style-type: none"> <li>Steroids and chemotherapy applied to the skin in early phase</li> <li>Treatment usually is palliative.</li> </ul>

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<b>Common Primary Diagnoses in the Stem Cell Transplantation Population (Continued)</b>				
Myelodysplastic syndrome (MDS)	<ul style="list-style-type: none"><li>Unknown</li></ul>	<ul style="list-style-type: none"><li>Group of hematologic disorders with an increased risk of transformation to acute myelogenous leukemia; occur as result of altered stem cells</li><li>Chromosome abnormalities are present at the level of stem cell in 50% of patients with primary MDS and 75% of patients with therapy-related MDS.</li><li>80%–90% of patients older than age 50</li><li>Median survival is 28 months.</li></ul>	<ul style="list-style-type: none"><li>Characterized by one or more peripheral blood cytopenias secondary to bone marrow dysfunction</li><li>Anemia, bleeding, easy bruising, and fatigue</li></ul>	<ul style="list-style-type: none"><li>Bone marrow transplantation (successful if marrow comes from human leukocyte antigen donor and if the patient is younger than age 55)—only curative therapy</li><li>High-dose chemotherapy for ablation can induce remission in 40%–60% of patients.</li></ul>
Multiple myeloma (MM)	<ul style="list-style-type: none"><li>Unknown</li><li>Monoclonal gammopathy of undetermined significance (MGUS): possible precursor</li><li>Abnormal level of serum protein that does not cause tumors</li></ul>	<ul style="list-style-type: none"><li>Cancer of plasma or B cell</li><li>Serum or urine protein (monoclonal immunoglobulin M protein)</li><li>No cure; relapse inevitable</li><li>Plasmablastic cells (plasma stem cell) colonize the bone marrow.</li><li>Research suggests correlation with deletion of chromosome 13 in MM.</li><li>Bone destroyed by osteolytic action of malignant plasma cells</li></ul>	<ul style="list-style-type: none"><li>Bone pain, anemia, and recurrent infections (related to impaired T cells)</li><li>Lytic bone lesions: risk for fractures, spinal cord compression, and loss of weight bearing ability</li><li>High-protein serum (M protein)</li><li>Pallor: Most common physical finding</li><li>Liver or spleen palpable in many patients</li><li>Mental status changes related to hypercalcemia, hyperviscosity syndrome, or renal insufficiency</li><li>Limitations in range of motion</li><li>Hypercalcemia, elevated blood urea nitrogen, creatinine, and uremia</li></ul>	<ul style="list-style-type: none"><li>Bortezomib, thalidomide, and lenalidomide</li></ul> <p><b>Primary:</b></p> <ul style="list-style-type: none"><li>Melphalan and prednisone</li><li>Vincristine, carmustine, melphalan, cytoxan, and prednisone</li><li>Vincristine, carmustine, doxorubicin, and prednisone</li><li>Vincristine, doxorubicin, and dexamethasone</li></ul> <p><b>Maintenance therapy:</b></p> <ul style="list-style-type: none"><li>Steroids and interferon</li><li>Radiation therapy: limited, can further deplete bone marrow reserve; used for palliative therapy</li><li>Autologous transplantation</li></ul>
Richter syndrome	<ul style="list-style-type: none"><li>Occurs in 1 of 20 patients with chronic lymphocytic leukemia</li></ul>	<ul style="list-style-type: none"><li>Rare, progresses rapidly, aggressive</li><li>Related to EBV</li><li>Richter syndrome transforms into high-grade non-Hodgkin lymphoma, acute leukemia, prolymphocytic leukemia, and Hodgkin leukemia</li><li>Median survival is five to eight months.</li></ul>	<ul style="list-style-type: none"><li>Rapid onset lymphadenopathy</li><li>Bulky retroperitoneal adenopathy</li><li>Fever, night sweats, and weight loss; massive hepatosplenomegaly; clinical deterioration</li><li>Extranodal involvement (skin, CNS, gastrointestinal tract, eye, testes, lung, and kidney)</li></ul>	<ul style="list-style-type: none"><li>Chemotherapy: lenalidomide, lumiliximab, clofarabine, and dasatinib</li><li>Monoclonal antibodies and biologic therapy</li><li>Allogeneic stem cell transplantation</li><li>Radiotherapy</li></ul>

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Diagnosis	Etiology and Risk Factors	Pathophysiology	Clinical Manifestations	Treatment and Management
<b>Common Primary Diagnoses in the Stem Cell Transplantation Population (Continued)</b>				
T-cell lymphoma	<ul style="list-style-type: none"> <li>• Median age: 61</li> <li>• Cutaneous T cell: skin involvement and mycosis fungoides</li> <li>• Viral cause: human T-cell lymphotropic virus type 1, EBV</li> <li>• Genetics, radiation, and immunosuppression</li> </ul>	–	<ul style="list-style-type: none"> <li>• Lymphadenopathy, "B" symptoms (unexplained fever, night sweats, and unexplained weight loss), and fatigue</li> </ul>	<ul style="list-style-type: none"> <li>• Hyper-CVAD plus etoposide, prednisone, vincristine, cyclophosphamide, and doxorubicin (EPOCH)</li> <li>• Relapsed: transplantation, DHAP, R-ESHAP, and ifosfamide, etoposide, and carboplatin (ICE)</li> <li>• Nontransplantation: velcade, gemcitabine, and ontak</li> </ul>
<b>Tips</b>				
<ul style="list-style-type: none"> <li>• Thorough assessment: at beginning of shift and any time throughout shift as necessary</li> <li>• Status changes rapidly; therefore, assessment is critical.</li> <li>• Infection control is essential: masks, gloves, handwashing, and sanitation. All patients are covered with broad spectrum antimicrobial, antifungal, and antiviral pharmacotherapeutics.</li> <li>• Blood product support (often on a daily basis)</li> <li>• Check IV compatibilities. Patients receive numerous IV medications. Assess compatibility to avoid blocking a central line from precipitation.</li> <li>• Tacrolimus (a 24-hour continuous infusion); do not turn off unless ordered to by an doctor. Daily dose is important, calculate with rate to check that dosing is correct.</li> <li>• Psychological issues include coping, stress and role changes, hair loss, body changes, end-of-life worries, and financial strain.</li> <li>• Symptom management: nausea and vomiting, diarrhea, gastroesophageal reflux disease, pain, cramping, rash, fatigue, and edema.</li> <li>• Be an advocate for your patients.</li> </ul>				
<b>Hematology Oncologic Complications and Emergencies</b>				
Cerebellar syndrome	<ul style="list-style-type: none"> <li>• Treatment with radiation, high-dose cytarabine, 5-fluorouracil (5-FU), and capecitabine (rare)</li> </ul>	<ul style="list-style-type: none"> <li>• Drug crosses blood-brain barrier; degenerative changes in cerebellum</li> <li>• Inhibits function of neurons by blocking a critical deoxynucleoside</li> </ul>	<ul style="list-style-type: none"> <li>• Slurred speech, pauses between words or parts of words, slowed speech; unsteady gait, stumbling when walking or loss of balance; jerky movements when reaching for an object; difficulty writing with a pen or pencil; and double vision</li> </ul>	<ul style="list-style-type: none"> <li>• Corticosteroids and plasma exchange</li> <li>• Reduce dose or discontinue the aggravating agent.</li> </ul>
Diffuse alveolar hemorrhage	<ul style="list-style-type: none"> <li>• Diffuse alveolar damage, Goodpasture syndrome, mitral stenosis, and pulmonary veno-occlusive disease</li> </ul>	<ul style="list-style-type: none"> <li>• Most often occurs within two weeks of transplantation; although there are multiple pathologies, can be characterized by hemorrhage in the alveolar spaces with inflammation and with or without necrosis, depending on the exact cause.</li> <li>• Damage to alveolar capillaries, alveolar capillary basement membranes, and alveolar walls</li> </ul>	<ul style="list-style-type: none"> <li>• Hypoxemia, dyspnea, cough, fever, hemoptysis, and anemia</li> </ul>	<ul style="list-style-type: none"> <li>• High-dose corticosteroids</li> </ul>
<i>(Continued on the next page)</i>				

CNS—central nervous system; CODOX-M—cytoxan, vincristine, doxorubicin, and high-dose methotrexate; CVC—central venous catheter; EBV—Epstein-Barr virus; hyper-CVAD—cyclophosphamide, vincristine, doxorubicin, and dexamethasone; LDH—lactic dehydrogenase; R-CHOP—rituxan, cyclophosphamide, doxorubicin, vincristine, and prednisone; R-ESHAP—rituxan, etoposide, methylprednisolone, cytarabine, and cisplatin; WBC—white blood cell

**Table 1. Stem Cell Transplantation Nurses' Hematology Reference Chart (Continued)**

Diagnosis	Etiology and Risk Factors	Pathophysiology	Clinical Manifestations	Treatment and Management
<b>Hematology Oncologic Complications and Emergencies (Continued)</b>				
Disseminated intravascular coagulation	<ul style="list-style-type: none"><li>Risk factors: cancer, infection, sepsis (most common cause), trauma, shock, burns, presence of liver disease, and recent blood transfusion or hemolytic transfusion reaction</li></ul>	<ul style="list-style-type: none"><li>The inappropriate, accelerated, and systematic activation of coagulation cascade, resulting in simultaneous thrombosis and hemorrhage</li><li>Overstimulation of clotting cascade; increased fibrinolysis (bleeding); infection, malignancy (AML, acute promyelocytic leukemia, and adenocarcinomas)</li><li>Trauma can trigger clotting cascade; thrombosis is accelerated, fibrin clots deposited in microcirculation.</li><li>Consumption of clotting factors: decreased ability for replacement (decreased clotting factors)</li><li>Fibrinolysis initiated: fibrinolytic split products produced and accumulate (anticoagulant effect)</li><li>Acute: bleeding occurs simultaneously from at least three sites; frequently associated with shock, respiratory failure, or renal failure. Chronic: usually manifests with minimal bleeding and frequently seen in malignancies</li></ul>	<ul style="list-style-type: none"><li>Petechiae, ecchymosis, purpura, pallor, and bleeding from sites of invasive procedures; acral cyanosis (tips of fingers, toes, nose, and ears cyanotic from wedged clots); abdominal distention; decreased bowel sounds; positive guaiac stool to frank blood stool; hematuria and decreased urinary output; increased heart rate; decreased blood pressure; diminished peripheral pulses; cool or clammy skin; dyspnea; increased respiratory rate; desaturation; rales or rhonchi; restlessness; hemoptysis; confusion; lethargy; projectile vomiting; joint pain and stiffness; and positive Homan's sign</li><li>Clinical indicators: elevated D-dimer assay, elevated fibrin degradation products titer, decreased platelet count, decreased fibrinogen, prolonged prothrombin time, prolonged activated partial thromboplastin time, prolonged international normalization ratio, and schistocytes in peripheral smear</li></ul>	<ul style="list-style-type: none"><li>Treat underlying condition (i.e., chemotherapy for malignancy; antibiotics for infection).</li><li>Replace coagulation factors and platelets (cryopreservation, fresh frozen plasma, and red blood cells).</li><li>Disrupt clot formation; disrupt breakdown of fibrin clot (aminocaproic acid).</li><li>Apply direct pressure or ice packs to sites of active bleeding; elevate sites of active bleeding if possible; apply pressure dressings and sandbags to sites of active bleeding; and apply thrombin-soaked gauze to sites of active bleeding if possible (nose bleeds, CVC lines).</li><li>Antithrombin III: inhibits procoagulants and fibrinolytic process</li></ul>
Graft-versus-host disease	<ul style="list-style-type: none"><li>Most common long-term complication</li><li>Risk factors: degree of mismatch, age of donor, number of T cells, and cytokine storm</li></ul>	<ul style="list-style-type: none"><li>Can affect the skin, liver, gastrointestinal tract, oral mucosa, muscles, vagina, nerves, kidneys, lungs, and marrow function</li></ul>	<ul style="list-style-type: none"><li>Acute: day 7–100; chronic: past day 100</li><li>Skin: maculopapular erythematous rash on trunk, palm, soles, and ears. Liver: increase in liver enzymes (bilirubin and alkaline phosphatase), right-upper quadrant pain, hepatomegaly, and jaundice. Gut: green watery diarrhea, abdominal cramping, paralytic ileus, anorexia, and nausea and vomiting</li></ul>	<ul style="list-style-type: none"><li>Prophylaxis: tacrolimus with low-dose methotrexate</li><li>Treatment: cyclosporine and prednisone; mycophenolate mofetil, tacrolimus, thalidomide, azathioprine, infliximab, and daclizumab; photopheresis</li></ul>
Hemorrhagic cystitis	<ul style="list-style-type: none"><li>Chemical toxicity (cyclophosphamide, busulfan, etoposide), radiation (total body irradiation), and viral infection</li></ul>	<ul style="list-style-type: none"><li>Diffuse inflammation of the bladder</li></ul>	<ul style="list-style-type: none"><li>Pain and/or feeling of fullness in the pelvic area, dysuria, hematuria, and hemorrhage</li></ul>	<ul style="list-style-type: none"><li>Administer diuretics (e.g., furosemide); maintain urine alkalinity; implement continuous bladder irrigation.</li></ul>

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CNS—central nervous system; CODOX-M—cytoxan, vincristine, doxorubicin, and high-dose methotrexate; CVC—central venous catheter; EBV—Epstein-Barr virus; hyper-CVAD—cyclophosphamide, vincristine, doxorubicin, and dexamethasone; LDH—lactic dehydrogenase; R-CHOP—rituxan, cyclophosphamide, doxorubicin, vincristine, and prednisone; R-ESHAP—rituxan, etoposide, methylprednisolone, cytarabine, and cisplatin; WBC—white blood cell

**Table 1. Stem Cell Transplantation Nurses' Hematology Reference Chart (Continued)**

Diagnosis	Etiology and Risk Factors	Pathophysiology	Clinical Manifestations	Treatment and Management
<b>Hematology Oncologic Complications and Emergencies (Continued)</b>				
Hemorrhagic cystitis	<ul style="list-style-type: none"> <li>Chemical toxicity (cyclophosphamide, busulfan, etoposide), radiation (total body irradiation), and viral infection</li> </ul>	<ul style="list-style-type: none"> <li>Diffuse inflammation of the bladder</li> </ul>	<ul style="list-style-type: none"> <li>Pain and/or feeling of fullness in the pelvic area, dysuria, hematuria, and hemorrhage</li> </ul>	<ul style="list-style-type: none"> <li>Administer diuretics (e.g., furosemide); maintain urine alkalinity; implement continuous bladder irrigation.</li> </ul>
Hyperviscosity syndrome	<ul style="list-style-type: none"> <li>Waldenström macroglobulinemia, multiple myeloma (particularly immunoglobulins A and G3), polycythemia (increased red blood cell count), and leukemia (more WBCs, particularly in acute leukemic blast crises)</li> </ul>	<ul style="list-style-type: none"> <li>Increased blood viscosity usually resulting from increased circulating serum immunoglobulins</li> <li>Associated with plasma cell dyscrasias (large size of the excess immunoglobulin M paraproteins)</li> </ul>	<ul style="list-style-type: none"> <li>Usual radiation treatment triad: mucosal bleeding, visual changes, and neurologic symptoms</li> <li>Mucosal bleeding occurs from prolonged bleeding time caused by myeloma proteins interfering with platelet function (spontaneous gum bleeding, epistaxis, rectal bleeding, menorrhagia, and persistent bleeding after minor procedures); visual changes range from blurred vision to vision loss; neurologic symptoms are frequent and varied (result from increase viscosity of the blood and decrease cerebral blood flow). Referred to as the Bing-Neal syndrome (vertigo, hearing loss, paresthesias, ataxia, headaches, seizures, with somnolence progressing to stupor or coma)</li> </ul>	<ul style="list-style-type: none"> <li>Plasmapheresis is the treatment of choice because it removes circulating paraproteins. The serum viscosity decreases and symptoms improve.</li> <li>While arranging for plasmapheresis, treat hemorrhage, congestive heart failure, and metabolic imbalances with standard therapies.</li> <li>Treat underlying multiple myeloma.</li> </ul>
Increased intracranial pressure	<ul style="list-style-type: none"> <li>Tumor, increased cerebrospinal fluid volume, obstruction to blood flow, and brain edema</li> </ul>	<ul style="list-style-type: none"> <li>Pressure in the cranium, brain tissue, and cerebrospinal fluid, thus exerting pressure on the brain's intracranial blood circulation vessels</li> </ul>	<ul style="list-style-type: none"> <li>Headache; nausea and vomiting (projectile); papilledema; focal effects, including weakness and mental disturbance; seizures; gait disorder; visual and language disturbance; impaired cognition; hemiparesis; sensory loss (unilateral); ataxia; aphasia; and vital sign change (widening pulse pressure [decreased heart rate, decreased respiratory rate or irregular])</li> </ul>	<ul style="list-style-type: none"> <li>Radiation, steroids, intrathecal chemotherapy (if increased intracranial pressure is caused by carcinomatous meningitis), and surgical decompression (only sensitive cases of resistant or relapsing cerebral metastasis)</li> </ul>

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Table 1. Stem Cell Transplantation Nurses' Hematology Reference Chart (Continued)

Diagnosis	Etiology and Risk Factors	Pathophysiology	Clinical Manifestations	Treatment and Management
Hematology Oncologic Complications and Emergencies (Continued)				
Leukostasis	<ul style="list-style-type: none"><li>• Individuals with extremely high numbers of circulating blasts</li><li>• Acute lymphocytic leukemia (occurs most often)</li></ul>	<ul style="list-style-type: none"><li>• Leukemic blasts accumulate and invade vessel walls, causing rupture and bleeding.</li><li>• Because of the extensive capillary network and limited vascular space of the brain, intracerebral hemorrhage is most common and lethal.</li><li>• Effects seen primarily in brain, CNS, and lungs when the WBC count is more than 100,000 cells.</li></ul>	<ul style="list-style-type: none"><li>• Pulmonary leukostasis: dyspnea, tachypnea, cough, chest discomfort, progressive hypoxemia, acidemia, bilateral infiltrates, pulmonary edema, and bilateral infiltrates on chest x-ray</li><li>• CNS leukostasis: headache, dizziness, tinnitus, ataxia, lethargy, disorientation, stupor, somnolence, visual changes, and seizures</li></ul>	<ul style="list-style-type: none"><li>• Immediate leukopheresis</li><li>• Initiate chemotherapy after pheresis is completed; watch for tumor lysis syndrome (at the start of tumor lysis syndrome precautions, increase IV fluids).</li><li>• Cranial irradiation</li><li>• Early detection of patients at risk (WBC count of more than 50,000 cells) and reducing the number of circulating cells are imperative.</li></ul>
Sepsis or infection	<ul style="list-style-type: none"><li>• Profound neutropenia; skin and mucosal barrier disruption; immunosuppressives (steroids); pretreatment infections; microorganism colonization; underlying hematologic or lymphoid malignancy; and presence of indwelling CVC</li></ul>	<ul style="list-style-type: none"><li>• Systemic inflammatory response in the presence of infection; WBC count may be greater or less than normal</li><li>• Severe sepsis: state of organ dysfunction, hypoperfusion, and/or hypotension</li><li>• Septic shock: marked by sepsis-induced hypotension despite reasonable fluid resuscitation or sepsis that requires vasopressors or inotropic agents to maintain normal blood pressure; presence of multiple organ dysfunction syndrome</li></ul>	<ul style="list-style-type: none"><li>• Manifested by two or more of the following: temperature of more than 38°C or less than 36°C; heart rate of more than 90 beats per minute; respiratory rate of more than 20 breaths per minute or partial pressure of carbon dioxide in the arterial blood less than 32 mmHg; and WBC count of more than 12,000 cells, less than 4,000 cells, or more than 10% immature (band) forms.</li></ul>	<ul style="list-style-type: none"><li>• Gram-positive coverage (with CVCs) or gram-negative coverage (particularly with interruption of bowel mucosa)</li><li>• Prophylactic and therapeutic: third and fourth generation cephalosporin, nafcillin; primaxin, merrem, quinolones, aminoglycoside, vancomycin (check serum levels for increased creatinine when using aminoglycoside and vancomycin)</li><li>• Medical management: blood cultures from peripheral blood and CVC; culture other sites as needed (stool for <i>Clostridium difficile</i>, vancomycin-resistant enterococcus); nasal washings or throat swab for respiratory syncytial virus; chest x-ray; and give Tylenol® as needed (avoid in patients with busulfan second-degree liver failure).</li><li>• Change microbial coverage: bacterial, viral, or fungal coverage (implement immediately)</li></ul>
(Continued on the next page)				
CNS—central nervous system; CODOX-M—cytoxan, vincristine, doxorubicin, and high-dose methotrexate; CVC—central venous catheter; EBV—Epstein-Barr virus; hyper-CVAD—cyclophosphamide, vincristine, doxorubicin, and dexamethasone; LDH—lactic dehydrogenase; R-CHOP—rituxan, cyclophosphamide, doxorubicin, vincristine, and prednisone; R-ESHAP—rituxan, etoposide, methylprednisolone, cytarabine, and cisplatin; WBC—white blood cell				

**Table 1. Stem Cell Transplantation Nurses' Hematology Reference Chart (Continued)**

Diagnosis	Etiology and Risk Factors	Pathophysiology	Clinical Manifestations	Treatment and Management
<b>Hematology Oncologic Complications and Emergencies (Continued)</b>				
Spinal cord compression	<ul style="list-style-type: none"><li>• Vertebral fracture, tumor, abscess, and ruptured intervertebral disc; metastatic spinal lesions</li></ul>	<ul style="list-style-type: none"><li>• Compression of spinal cord or cauda equina, which results in decompensation of neurologic status</li><li>• Neurologic emergency caused by compression of the spinal cord itself or altered blood supply to the cord.</li><li>• Ninety-five percent of spinal cord compression is caused by metastatic decrease in epidural space or outside of spinal cord.</li></ul>	<ul style="list-style-type: none"><li>• Pain (first symptom), motor weakness, autonomic dysfunction, and sensory loss; increasing neck or back pain will be a presenting feature in 90% of cases.</li><li>• Physical findings: radicular signs, straight leg raise, positive Babinski reflex or sustained ankle clonus, loss of deep tendon reflexes, and loss of proprioception</li><li>• Early recognition and diagnosis is imperative</li><li>• Most critical prognostic factor in spinal cord compression is neurologic status at time of diagnosis (how much function, sensation, and bowel and bladder control patients will regain).</li></ul>	<ul style="list-style-type: none"><li>• Treatment will not reverse fixed paralysis of more than 48 hours.</li><li>• Radiation therapy is the most common treatment choice; surgical: kyphoplasty (relieves pain, increases height); surgical decompression (laminectomy or vertebral body resections [for patients with more than three months of expected life]; chemotherapy [used with sensitive cancers such as lymphoma and Hodgkin disease], patients who are not candidates for additional radiation therapy); and high-dose steroids (always used—reduces spinal cord edema and pain)</li><li>• Pain management: oral or parenteral opioids (do not forget bowel management), epidural blocks, gabapentin (trial), and steroids</li></ul>
Superior vena cava syndrome	<ul style="list-style-type: none"><li>• Compression of the superior vena cava from intrathoracic tumors; venous thrombosis from CVC; and radiation-induced fibrosis</li></ul>	<ul style="list-style-type: none"><li>• Mechanical obstruction of superior vena cava</li><li>• Occurs when the superior vena cava is occluded by external pressure, invaded by a neoplasm, or obstructed by a large thrombosis</li><li>• Venous return from the head and upper extremities to the heart is impaired.</li></ul>	<ul style="list-style-type: none"><li>• Upper body swelling and venous congestion, cerebral edema and increased intracranial pressure, laryngeal edema and tracheal obstruction, and decreased cardiac output</li><li>• Early signs: facial swelling when waking in the morning, redness or edema in conjunctivae and eyes, swelling of neck and arms, neck and thoracic vein distention, dyspnea (most common symptom), nonproductive cough, hoarseness, and cyanosis and stridor</li><li>• Late signs: brain edema (headache, irritability, visual disturbances, dizziness, changes in mental status, intracranial hypertension, dyspnea, and tachypnea)</li></ul>	<ul style="list-style-type: none"><li>• Diuretic therapy, steroids, oxygen therapy, and bronchodilators</li><li>• Goals of treatment: relief of symptoms and reduction of the obstruction; maintain airway and cardiac output.</li></ul>
Thrombotic thrombocytopenic purpura	<ul style="list-style-type: none"><li>• High-dose conditioning regimen; infection and severe acute graft-versus-host disease; immunosuppressive agents</li></ul>	<ul style="list-style-type: none"><li>• Microangiopathic hemolysis and platelet aggregation in which formation is unrelated to coagulation system activity (can cause ischemia and thrombocytopenia)</li></ul>	<ul style="list-style-type: none"><li>• Thrombocytopenia, hemolytic anemia, renal abnormalities, fever, neurologic abnormalities, and hypertension</li><li>• Laboratory values: rise in LDH, decrease in hemoglobin and haptoglobin</li></ul>	<ul style="list-style-type: none"><li>• Plasma exchange, transfusion of plasma, administration of antihypertensives, platelet transfusions, red blood cell replacements, replacement fluids, and monitor for seizure activity.</li></ul>

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Table 1. Stem Cell Transplantation Nurses' Hematology Reference Chart (Continued)				
Diagnosis	Etiology and Risk Factors	Pathophysiology	Clinical Manifestations	Treatment and Management
Hematology Oncologic Complications and Emergencies (Continued)				
Tumor lysis syndrome	<ul style="list-style-type: none"><li>• Malignancies: massive tumor burden and responsive to chemotherapy (high-grade lymphomas [e.g., non-Hodgkin lymphoma], acute lymphocytic leukemia, Hodgkin disease)—less common with solid tumors, renal dysfunction, and sepsis</li><li>• High risk: increased WBC count, LDH of more than 1,500 IU/L, blasts, splenomegaly, and lymphadenopathy</li></ul>	<ul style="list-style-type: none"><li>• Metabolic imbalance that occurs with rapid release of intracellular potassium, phosphorus, and nucleic acid into blood stream as a result of tumor cell kill</li></ul>	<ul style="list-style-type: none"><li>• Early signs: weakness, muscle cramps, nausea and vomiting, diarrhea, and lethargy</li><li>• Late signs: flaccid paralysis, bradycardia, hypotension, oliguria, anuria, edema, hematuria, crystalluria, azotemia, flank pain, and tetany</li><li>• High phosphorus, potassium, and uric acid; low calcium (from phosphorus binding to calcium to form calcium phosphorus salts), tetany, hypotension, altered mental status, electrocardiogram changes, and seizures</li><li>• Prothrombin time, partial prothrombin time, and D-dimer increased</li><li>• Hematocrit and hemoglobin, fibrinogen, and decreased platelet count</li></ul>	<ul style="list-style-type: none"><li>• Allopurinol (decrease uric acid); hydration and diuresis; aluminum hydroxide (decrease phosphorus); kayexalate (decrease potassium); urine alkalinization (prevent precipitation), sodium bicarbonate or sodium acetate; cardiac monitoring for dysrhythmias; fluid and electrolyte management; dialysis (if indicated for renal insufficiency); sodium bicarbonate for severe potassium of 6.5–7 mEq/L, give 1–2 ampules over 2–5 minutes; regular insulin of 5–10 units with dextrose 50% in water; and albuterol nebulizer 10–20 mg in 4 ml of normal saline nebulized over 10 minutes</li></ul>
Veno-occlusive disease	<ul style="list-style-type: none"><li>• Preexisting liver disease</li><li>• Second myeloblastic allogeneic stem cell transplantation</li><li>• Conditioning with busulfan, melphalan, cytoxan, and total body irradiation</li></ul>	<ul style="list-style-type: none"><li>• Complication of high-dose chemotherapy given before a bone marrow transplantation that is marked by weight gain from fluid retention, increased liver size, and raised levels of bilirubin in the blood</li></ul>	<ul style="list-style-type: none"><li>• Weight gain, fluid retention, increased abdominal circumference, hepatomegaly, right upper quadrant pain, acites, jaundice, and increase in total and direct bilirubin</li><li>• Onset of transfusion (refractory thrombocytopenia with no detectable cause [early sign])</li></ul>	<ul style="list-style-type: none"><li>• Prevention: heparin, actigall</li><li>• Treatment: defibrotide</li><li>• Supportive care: careful fluid and electrolyte balance, analgesics, platelet transfusions, correct clotting issues, and minimize exposure to hepatotoxic and nephrotoxic agents</li><li>• Meticulous input and output (careful fluid and electrolyte balance)</li><li>• Monitor weight daily; measure abdominal girth daily; monitor laboratory values and signs and symptoms of bleeding, mental status, and infection; and minimize exposure to hepatotoxic medications.</li></ul>
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