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Essential Review of Oncological Emergencies

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Innovations in oncology have expanded treatment eligibility, leading to a rise in cancer patients requiring critical care. This necessitates that all critical care clinicians possess a fundamental knowledge of prevalent oncological conditions and identify emergent scenarios requiring immediate action. This article will explore key oncological complications and their management approaches. **Key words:** *cancer, febrile neutropenia, hyperviscosity syndrome, leukostasis, oncological emergencies, oncology, pericardial tamponade, superior vena cava syndrome, tumor lysis syndrome*

BACKGROUND

Cancer remains a leading cause of mortality globally and in the United States.[1] Enhanced screening, public health initiatives, and advances in cancer therapies have collectively contributed to a steady decline in death rates from various cancers.[2] Concurrently, the increase in survival rates has amplified the demand for intensive care, with cancer patients now constituting about 15% of ICU admissions.[2] Consequently, a thorough grasp of the clinical complications arising from both the cancer itself and its treatment is crucial for all critical care clinicians. This review offers a concise overview of key oncological conditions and outlines the imperative immediate and follow-up care in the ICU.

FEBRILE NEUTROPENIA

Febrile neutropenia (FN), a frequent side effect of cytotoxic chemotherapy, often manifests as the sole indication of infection in oncology patients. It affects 10% to 50% of solid tumor patients and over 80% of those with hematologic malignancies during chemotherapy, leading to fever and neutropenia.[3] Despite this, only 20% to 30% of febrile episodes are linked to documented infections, leaving the majority of FN cases without an identified infectious cause.[4] Given the clinical variability in defining fever and neutropenia, clinical judgment becomes paramount in deciding the necessity of antibiotics for patients at risk of neutropenia, even when specific criteria are not met.[4] Fever is characterized by a single oral temperature of ≥38.3 °C or a sustained temperature of ≥38.0 °C for more than an hour.[4] Neutropenia is defined by an absolute neutrophil count of <500 cells/mm^3 or an anticipated decline to <500 cells/mm^3 within 48 hours.[4]

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Cytotoxic chemotherapy targets not only tumor cells but also bone marrow's myeloproliferative cells, leading to neutropenia.[5] This treatment also harms other rapidly dividing cells, such as those lining the gut mucosa, compromising the gastrointestinal tract's role as a barrier against bacterial colonization and increasing the risk of infection and sepsis from bacterial and fungal pathogens.[5] The most common infection sites in neutropenic patients are the intestines, lungs, and skin, with bacteremia affecting 10% to 25% of this group.[4] The lack of an inflammatory response in neutropenic patients often delays the appearance of localized infection signs, underscoring the need for prompt FN evaluation and treatment.[5]

In the 1960s and 1970s, the advent of cytotoxic chemotherapy saw a predominance of gram-negative pathogens. By the 1980s and 1990s, gram-positive organisms became prevalent due to the increased use of indwelling venous catheters, facilitating the colonization and entry of skin flora.[6,7] Coagulase-negative staphylococci emerge as the most frequent blood isolates. Fungal infections are uncommon, but yeasts, especially Candida, can lead to superficial infections or, via chemotherapy-induced mucositis, enter the bloodstream through the oral mucosa. Aspergillus, a mold, poses a significant risk of severe lung and sinus infections after prolonged neutropenia of 2 weeks or more.[4]

High-risk FN patients face poorer outcomes. Key recommendations from the revised 2010 Infectious Society Disease of America guidelines include[4] the following: (1) Hospitalize high-risk patients (expected prolonged neutropenia > 7 days) with significant comorbidities—such as hypotension, pneumonia, abdominal pain, or neurologic changes—for empiric therapy. (2) Conduct laboratory tests, including complete blood count with differential, serum creatinine, blood urea nitrogen, electrolytes, liver function tests, and at least 2 sets of blood cultures (one from each central venous catheter lumen and one from peripheral venipuncture). Culture other suspected infection sites and perform chest radiographs for respiratory symptoms. (3) Initiate monotherapy with an anti-Pseudomonas beta-lactam agent like piperacillin-tazobactam, cefepime, or carbapenem. Add aminoglycosides, fluoroquinolones, or vancomycin for complications such as hypotension or pneumonia, or if resistant pathogens are suspected. Reserve vancomycin for specific conditions like skin infections, pneumonia, hemodynamic instability, or suspected catheter-related infections. (4) Expand the antibiotic regimen to include coverage for resistant gram-negative and gram-positive bacteria, aerobic bacteria, and fungi if patients remain hemodynamically unstable after initial treatment. (5) Generally, avoid hematopoietic growth factors like G-CSF or GM-CSF for treating established FN and consult the primary oncology team for potential use.

Intensive care unit care

Sepsis and septic shock are major causes of ICU admissions and deaths among patients with hematologic malignancies and solid tumors, particularly those experiencing chemotherapy-induced FN.[8,9] The Surviving Sepsis Campaign (SSC) has formulated guidelines for managing sepsis, described as life-threatening organ dysfunction due to a dysregulated host response to infection.[10] FN serves as a critical early sign of potential infection and is deemed an emergency, with mortality rates ranging from 4% to 21%.[4] It is important to note that tools like the Sequential Organ Failure Assessment and the National Early Warning Score were not specifically designed for or validated in FN patients. Thus, applying SSC guidelines with an understanding of the unique risks faced by neutropenic patients is essential. Below, key points from the 2021 revised SSC guidelines for managing sepsis and septic shock are summarized[11]: (1) Administer antimicrobials promptly, ideally within 1 hour of diagnosing sepsis, with urgency varying based on sepsis likelihood and septic shock presence.(2)In cases of probable sepsis or shock, give antimicrobials immediately, ideally within 1 hour of recognition. (3) For possible sepsis without shock, assess rapidly for infection vs noninfectious causes; if

infection concerns persist following a brief investigative period, administer antimicrobials within 3 hours from initial sepsis recognition. (4) In sepsis-induced hypoperfusion or septic shock, it is recommend to administer at least 30 mL/kg of IV crystalloid within the first 3 hours of resuscitation.[12] (5) Opt for balanced crystalloids over normal saline for resuscitating adult sepsis or septic shock patients to avoid potential adverse effects like hyperchloremic metabolic acidosis.[13] (6) Norepinephrine is the preferred first-line vasopressor for adult septic shock patients. Add vasopressin if norepinephrine alone does not maintain adequate mean arterial pressure (MAP), followed by epinephrine if needed. Consider dobutamine or epinephrine alone for septic shock patients with cardiac dysfunction and persistent hypoperfusion despite adequate resuscitation and arterial blood pressure.[11] (7) Initiate vasopressors peripherally to restore MAP (>65 mmHg) in adults with septic shock, without waiting for central venous access. Peripheral administration is safe for short durations (<6 hours) when infused distally to the antecubital fossa.[14] (8) For adults with septic shock needing ongoing vasopressor therapy, suggest using IV corticosteroids (hydrocortisone at 200 mg/day).[15] (9) The SSC advises low tidal volume ventilation and limiting plateau pressure in sepsis-associated ARDS patients, recommending prone positioning for moderate-to-severe Acute respiratory distress syndrome and a low tidal volume strategy for all sepsis-induced respiratory failure cases.[11]

HYPERVISCOSITY SYNDROME

Hyperviscosity syndrome (HVS) represents an oncological emergency characterized by increased blood viscosity, which can be attributed to alterations in red blood cell (RBC) morphology or elevated serum concentrations of proteins, RBCs, white blood cells (WBCs), or platelets. The condition arises from various causes, including monoclonal and polyclonal serum disorders, as well as whole blood disorders stemming from both RBC and WBC anomalies. Notably, monoclonal disorders are common culprits, with WBC disorders such as chronic lymphocytic leukemia (CLL), chronic myeloid leukemia, and acute nonlymphocytic leukemia frequently implicated in oncological emergencies.[16]

The primary pathophysiological effect of HVS is the induction of tissue hypoxia and hypoperfusion due to elevated blood viscosity.[17] Patients with HVS typically present with a triad of symptoms: mucosal bleeding, visual disturbances, and neurological deficits. Mucosal bleeding may include bilateral epistaxis, gingivitis, gastrointestinal, or retinal bleeding. Visual symptoms, warranting immediate ophthalmoscopic evaluation, can range from retinal hemorrhages or thrombosis to papilledema and blurred vision. Neurologically, patients may experience somnolence or coma, cerebral hemorrhage, seizures, and ataxia.[16] The variability in symptoms among patients reflects the underlying cause and the severity of serum viscosity.

Diagnosis of HVS relies on viscosity measurement via viscometry, which is considered the gold standard, although this may not always be promptly available. In the interim, a comprehensive diagnostic approach includes a chemistry panel to identify electrolyte imbalances such as hypercalcemia, hyponatremia, and hyperproteinemia. A complete blood count may reveal significant leukocytosis due to malignancy, alongside possible anemia, and thrombocytopenia. Coagulation tests may indicate elevated Prothrombin time/Partial thromboplastin time levels. Imaging studies, such as chest X-rays, can detect interstitial disease related to increased plasma volume, and CT scans of the abdomen/pelvis might reveal organomegaly and lymphadenopathy. Serum and urine protein electrophoresis can aid in identifying the underlying cause of hyperviscosity. Additionally, it is crucial to exclude other potential causes of the symptom triad, which may involve a head CT for neurological abnormalities and further oncological assessment if the diagnosis remains unconfirmed.[18]

Intensive care unit care

Treatment of HVS aims to reduce blood viscosity and prevent circulatory damage, typically involving hydration, diuresis, and addressing the underlying cause or malignancy, with plasma exchange being a cornerstone intervention. Plasmapheresis, the primary modality for managing HVS symptoms, involves separating plasma from blood cells through centrifugation, subsequently replacing the removed plasma with a protein solution like albumin. Multiple plasmapheresis sessions might be necessary for clinical improvement. It is critical to recognize that plasmapheresis does not reduce tumor load, necessitating concurrent cancer-specific treatments, which vary according to the malignancy type and its root cause.[18] For patients without HVS symptoms or those in stable condition, starting cancer-directed therapy might preclude the need for plasmapheresis.

Furthermore, individuals with HVS, especially when associated with hyperleukocytosis, are predisposed to spontaneous tumor lysis syndrome (TLS), posing a risk of severe electrolyte imbalances. Such patients warrant vigilant monitoring and regular assessments of electrolyte levels in an intensive care setting.

TUMOR LYSIS SYNDROME

TLS is an oncologic emergency triggered by the rapid lysis of malignant cells, leading to the release of intracellular contents into the bloodstream. This process can be spontaneous or induced by therapy, and it often results in metabolic disturbances such as hyperkalemia, hyperphosphatemia, hypocalcemia, and hyperuricemia, which can cause acute kidney injury, life-threatening arrhythmias, and even death. TLS is most commonly seen after chemotherapy initiation, within 1 to 5 days, and is rare in the context of spontaneous tumor necrosis.[19] Conditions like Burkitt lymphoma, acute lymphoblastic leukemia, and other aggressive lymphomas are frequently associated with TLS, with chronic leukemias and solid tumors being less common. The risk of TLS increases in CLL patients following treatment with nucleoside agents like fludarabine or hormonal therapies such as glucocorticoids, letrozole, and tamoxifen. Monoclonal antibodies like rituximab and gemtizumab have also been linked to TLS.[19]

With the advent of targeted therapies for hematologic malignancies, the prevalence of TLS has been reevaluated. A review by Howard et al[20] analyzed the incidence of TLS in clinical trials involving monoclonal antibodies, tyrosine kinase inhibitors, proteasome inhibitors, chimeric antigen receptor T cells, and the proapoptotic agent lenalidomide. The highest TLS rates were observed in trials of venetoclax for CLL (8.3% and 8.9%) and in studies involving CAR T cells for B-cell malignancies and obinutuzumab for non-Hodgkin lymphoma, each reporting a 10% incidence. Acute leukemia trials showed TLS rates of 15% with dinaciclib and 42% and 53% with alvocidib, following cytarabine and mitoxantrone treatment, respectively.[20] Although TLS mitigation strategies were routinely applied in alvocidib and lenalidomide trials, their mention was either absent or vague for other agents, highlighting the ongoing risk of TLS with novel treatments and the critical need for early intervention and risk management strategies, which should be coordinated with the primary oncology team.

Hyperuricemia, often present at chemotherapy initiation, can lead to renal failure due to uric acid crystal deposition in the renal tubules. Hyperphosphatemia, stemming from tumor cell lysis, can induce a drop in serum calcium levels, causing neuromuscular irritability and tetany, while calcium phosphate deposition in the kidneys further contributes to renal damage. Massive cell destruction can also release potassium, leading to potentially fatal hyperkalemia, especially in patients with compromised renal function, by causing ventricular arrhythmias and sudden cardiac death.[19]

In patients with Burkitt's lymphoma, a large tumor burden and preexisting renal dysfunction signify a higher TLS risk. Elevated uric

acid levels and serum lactate dehydrogenase (LDH > 1500 U/L) are indicative of the total tumor burden and TLS likelihood. Elevated leukocyte and platelet counts can falsely increase potassium measurements due to cell lysis post-blood draw, thus plasma potassium levels are more reliable in such cases. For patients with baseline renal abnormalities, imaging like ultrasonography or CT is recommended to exclude obstructive uropathy, and urine output should be closely monitored.[19]

Intensive care unit care

Hydration

Aggressive hydration plays a crucial role in TLS prevention and treatment, facilitating uric acid and phosphate excretion by enhancing intravascular volume, glomerular filtration rate, and renal blood flow. While diuretics can help maintain optimal urine output, their use is not recommended in cases of obstructive uropathy or significant hypovolemia.[21]

Allopurinol

This medication prevents the conversion of xanthine and hypoxanthine to uric acid, a key strategy in TLS management. Allopurinol, a xanthine oxidase inhibitor, blocks purine metabolites from becoming uric acid. It is part of the initial TLS management for patients with intermediate risk, alongside hydration.[22] Rasburicase, a recombinant urate oxidase, offers an alternative when allopurinol and hydration fail to adequately reduce uric acid levels. It is particularly recommended for high-risk TLS patients as initial treatment. However, checking for glucose-6-phosphate deficiency is essential before rasburicase administration due to the risk of hemolysis from oxidative stress caused by its byproduct, hydrogen peroxide. While rasburicase acts swiftly, it may trigger hypersensitivity reactions, including bronchospasm, hypoxemia, and hypotension. Prophylactic use of rasburicase is advised for patients at high risk for TLS.[19] In cases of impending renal failure, early initiation of dialysis, particularly hemodialysis, should be considered.[19]

Table 1 Summarizes the Management Strategies for Electrolyte Imbalances Associated with TLS.[21,23]

SUPERIOR VENA CAVA SYNDROME

Superior vena cava (SVC) syndrome, primarily resulting from malignancies, leads to significant SVC obstruction via external compression, intravascular invasion, or thrombosis.[24] While device-induced SVC syndrome from central lines or cardiac devices and benign causes are documented, this discussion focuses on malignancy-related cases, comprising 70% to 90% of instances. Non–small cell lung cancer emerges as the chief malignancy linked to SVC syndrome, representing 50% of such cases, followed by small cell lung cancer, lymphomas, and cancers like thymomas or those with mediastinal metastases. Although SVC syndrome itself seldom leads to mortality, the median survival for patients with cancer-induced SVC syndrome is limited to 6 months.[25] Predominantly affecting men, SVC syndrome usually manifests in individuals aged 50 years and above.[24]

Patients typically present with facial and neck swelling, engorged neck and chest veins, alongside symptoms of dyspnea, cough, and headaches. Symptoms often intensify in a supine or forward-leaning stance, reflecting the venous pressure surge from the blocked SVC. The SVC, a vein accustomed to low-pressure flow, facilitates blood return from the upper body to the heart. An obstruction disrupts this flow, causing blood accumulation upstream and eliciting the hallmark symptoms.[24]

Diagnosis relies on clinical signs and imaging. Initial chest radiography might suggest a mass or central lung pathology but falls short of a definitive diagnosis. Contrast-enhanced CT scans provide comprehensive SVC visualization, revealing the extent of venous obstruction, collateral circulation, and

Table 1. Management Strategies for Electrolyte Derangements Associated With TLS

Electrolyte Abnormality	Management Strategy
Hyperphosphatemia	Avoid IV phosphate administration, administer phosphate binder
Moderate, ≥2.1 mmol/L	Dialysis, CAVH, CVVH, CAVHD, CVVHD
Severe	No therapy
Hypocalcemia, ≤1.75 mmoL/L	Calcium gluconate 50-100 mg/kg IV administered slowly with ECG monitoring
Asymptomatic	Avoid IV an oral potassium
Symptomatic	ECG and cardiac rhythm monitoring, Sodium polystyrene sulfonate
Hyperkalemia	Same as above, as well as the following:
Moderate and asymptomatic, ≥6.0 mmol/L	Calcium gluconate (100-200 mg/kg by slow IV infusion for life-threatening arrhythmias)
Severe (>7.0 mmol/L) and/or symptomatic	Regular insulin (0.1 U/kg IV) + D25 (2 mL/kg) IV
	Sodium bicarbonate (1-2 mEq/kg IV push) can be given to induce influx of potassium into cells; however, sodium bicarbonate and calcium should not be administered through the same line
	Dialysis
Renal dysfunction (uremia)	Fluid and electrolyte management
	Uric acid and phosphate management
	Renally adjust appropriate medications
	Dialysis (hemodialysis or peritoneal dialysis)
	Hemofiltration (CAVH, CVVH, CAVHD, or CVVHD)

Abbreviations: CAVH, continuous arterial-venous hemodialysis; CAVHD, continuous arterial-venous hemodialysis; CVVH, continuous veno-venous hemofiltration; CVVHD, continuous veno-venous hemodialysis; IV, intravenous.

distinguishing thrombotic from extrinsic causes. While venography offers precise obstruction localization, it is less accessible compared to other diagnostic tools. Ultrasonography, both bedside and formal, serves as an adjunctive tool to delineate thrombus reach. For patients precluded from contrast CT due to allergies or renal issues, MRI offers a suitable alternative in emergency departments.[24]

Intensive care unit care

SVC syndrome severity ranges from asymptomatic conditions to critical emergencies requiring intensive care, especially for patients with life-threatening symptoms such as elevated intracranial pressure or airway obstruction. In such cases, ICU admission is imperative for comprehensive monitoring and management. Emergency interventions may include SVC stenting for tumor-induced obstructions or thrombectomy and thrombolytic treatments for thrombotic blockages.[26] Supportive care is crucial, involving elevation of the patient's head and chest and provision of supplemental oxygen. The efficacy of diuretics and steroids in managing SVC syndrome symptoms remains inconclusive.[26]

Immediate initiation of curative treatment is paramount when feasible, tailored to the underlying malignancy and potentially encompassing chemotherapy, systemic treatments, and radiation therapy. In the

absence of curative options, palliative care becomes the focal point of management.

PERICARDIAL TAMPONADE

Pericardial tamponade presents a critical condition where fluid rapidly accumulates in the pericardial sac, compressing the heart and disrupting its function. Normally, the pericardial space harbors 15 to 20 ml of serous fluid to lubricate the heart's outer layer. This space can adapt to additional fluid if the increase is slow, allowing oncology patients to accommodate up to 1 liter without cardiac compromise. Critical tamponade develops when pericardial pressure surpasses that within the heart, hindering ventricular filling, reducing cardiac output, and potentially causing cardiogenic shock.[28,29]

Key indicators of pericardial tamponade encompass hypotension, tachycardia, raised jugular venous pressure, and subdued heart sounds, along with pulsus paradoxus and signs indicative of diminished cardiac output.[30] Diagnostic approaches for suspected tamponade include electrocardiogram, which might reveal tachycardia, low electrical signal, and alternating heart rhythm patterns. While CT and MRI can identify pericardial effusion, they may not confirm tamponade physiology.[29,30] Echocardiography and point-of-care ultrasound (POCUS), conducted by skilled practitioners, stand as the primary diagnostic tools, capable of detecting both pericardial effusion and tamponade physiology. Echocardiographic evidence of tamponade includes significant pericardial effusion and the inward collapse of the right-sided heart chambers during diastole.[28,29]

Intensive care unit care

In the ICU, managing pericardial tamponade involves initial supportive strategies such as aggressive fluid resuscitation and the administration of inotropes and vasopressors to ensure hemodynamic stability.[27-30] The primary treatment aims to eliminate the excess pericardial fluid, typically through pericardiocentesis or the creation of a pericardial window. For cases at risk of recurrent fluid accumulation, pericardiodesis may be employed to mitigate this risk. It is important to note that pericardiocentesis can lead to complications like bleeding, damage to the heart's inner layer, or injury to surrounding chest structures.[27-30] Furthermore, a notable but uncommon complication following substantial fluid drainage is pericardial decompression syndrome (PDS), characterized by a paradoxical decline in hemodynamic function post-fluid removal. The underlying cause of PDS may relate to either the revelation of preexisting cardiac issues or the emergence of new cardiac dysfunction due to the autonomic stress associated with tamponade.[30,31]

To summarize, pericardial tamponade is a critical condition marked by fluid buildup in the pericardial sac, compressing the heart and impairing its function. Diagnosis primarily employs echocardiography or POCUS, with ICU management focusing on stabilizing hemodynamics until the pericardial fluid can be safely removed.

CONCLUSION

With oncology patients comprising 15% of ICU admissions, it is crucial for health care providers to promptly identify and manage prevalent oncologic emergencies. This review, encompassing FN, HVS, SVC syndrome, TLS, and pericardial tamponade, underscores the importance of early detection and intervention in enhancing patient outcomes. As the landscape of oncologic care evolves with the advent of new treatments and the extension of options to a broader, more diverse patient population, including those with significant comorbidities and the elderly, continuous updates on the complications, and management strategies of such emergencies are vital for maximizing treatment efficacy, safety, and patient survival.

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