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Review Article

Oncologic emergencies: a review

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ABSTRACT

Cancer and its treatment include many medical emergencies. Hence taking care of these emergencies presents a challenge not only to the clinicians but also to the medical oncologists. Cancer patients may have complex medical problems in addition to cancer such as coronary heart diseases, diabetes mellitus, and respiratory diseases. Such patients require immediate medical assistance and emergency care facilities to improvise their health condition. The present review paper focuses on more commonly confronted emergencies in cancer patients and their related management.

Keywords: Hyperviscosity syndrome, Hemostatic emergencies, Oncologic emergencies, Tumor lysis syndrome, SIADH

INTRODUCTION

Cancer remains one of the leading causes of morbidity and mortality the world over. According to 1991 Indian census data about 609000 had been observed, this number had increased exponentially by the turn of the century and is ever increasing.[1]

With increasing life expectancy, the incidence of cancers is also on the rise. This in combination with decreasing mortality rates and longer periods of remission for all malignancies brings in more patients with emergencies due to their cancers or treatment related to their cancers.[2]

Cancer patients may often have other medical problems such as heart diseases, diabetes, chronic respiratory issues. Even for treating acute emergencies the approaches should be more similar to those without cancer. However, stage of the tumour, patient's response to current treatment and family support plays an integral part in planning an appropriate treatment plan.

An oncologic emergency could be described as any acute morbidity or life threatening event directly or indirectly related to a patients' tumor or its treatment.[3] These complications result from a cancer, a paraneoplastic syndrome or from treatment of the cancer that requires immediate medical attention.[4] Inpatient treatment is a must and very often intensive care is required. Differential diagnosis includes medical emergencies not related to patients' diagnosis of cancer.

Classification of oncologic emergencies

By their system of origin

This class includes metabolic and hematologic emergencies. The most common among metabolic effects are tumour lysis syndrome, syndrome of inappropriate secretion of antidiuretic hormone (SIADH) and hypercalcemia. Hyperviscosity syndrome, hyperleukocytosis syndrome, and hemostatic emergencies are more common among hematologic emergencies.

Tumor lysis syndrome (TLS)

TLS results from destruction of large number of rapidly multiplying cells as a result of cancer induction chemotherapy but can also be seen after treatment with radiotherapy, corticosteroids, and hormonal agents such as tamoxifen, biological agents such as Interferon or spontaneously in patients with high tumor burden. This syndrome is associated with severe electrolyte abnormalities, hyperuricemia, hyperkalemia, hyperphosphatemia, hypocalcemia, lactic acidosis and can lead to acute renal failure.[5]

No specific signs and symptoms are noted for this syndrome. Symptoms may advance in line with electrolyte disturbances that include muscle weakness, arrhythmias, neuromuscular irritability, seizures and sudden death.[6]

The main cause of mortality in these patients was arrhythmia associated with electrolyte disturbances, predominantly hyperkalemia and renal failure.[7] Hence, careful assessment, monitoring and treating related electrolyte disturbance is very essential to avoid serious complications of this syndrome.

Regular monitoring of electrolytes, BUN, creatinine, uric acid, phosphates and calcium levels forms the basis of therapy. Patients should be well hydrated (200-300ml/hr). Urinary flow should be increased by diuretics such as Mannitol. Sodium bicarbonate 100mEq/l should be given for urinary alkalization, Allopurinol 500 mgm/m^2 on day 1 to day 3 then reduced to 200mgm/m2 throughout cytoreductive therapy.[8] Calcium therapy and exchange resins for hyperkalemia may be considered. Calcitrol therapy may be considered for persistently low Calcium levels.[9]

SIADH

SIADH, characterized by hyponatremia that occurs due to the production of arginine vasopressin by the tumour cells. Hyponatremia is related with plasma hyposmolarity and high urinary osmolarity, together with a high level of elimination of urinary sodium without any change in plasma volume.[6] Other reasons may include use of some drugs like ACE inhibitors, antidepressants, and antimitotics such as cyclophosphamide, vincristine, cisplatin, melphalan and even some surgical procedures and due to some tumours like small-cell lung cancer.[10]

Most of the patients are asymptomatic. Early symptoms include anorexia, irritability, depression, muscle cramps, lethargy, weakness and behavioral changes. In severe conditions (plasma levels of <110mEq/l) depressed deep tendon reflexes, seizures and coma may appear.[11]

Management depends on the severity of symptoms. In mild cases, fluid restriction to about 0.5 to 1L of free water, along with increased intake of salt and protein, is usually sufficient. In severe cases serum sodium should be corrected with 3% saline cautiously. In long standing cases, serum sodium be corrected by no more than 8 to 10mmol/L in 24 hours, or less than 18mmol/L in the first 48 hours.[12] If specific therapy is not available in case of small-cell lung cancer, water restriction and demeclocycline should be considered.[6]

Hypercalcemia

Hypercalcemia is one of the most common emergencies noted in many of the cancers and may occur in up to 30% of all cancer patients.[13] Cancers such as advanced solid tumours of lung, breast, head and neck, renal, malignant lymphomas and myelomas produces hypercalcemia. The underlying mechanisms for the prevalence of hypercalcemia includes parathyroid hormone-related peptide (PTHrP), parathyroid hormone (PTH) over secretion, overproduction of vitamin D, or direct osteolytic effect of tumor on bone.[14] PTHrP-mediated hypercalcemia is the most common mechanism accounting for 80% of all cases particularly in squamous cell carcinoma. Hypercalcemia due to over production of Vitamin D is seen in about 15% cases. Particularly lymphomas secrete the active form of Vitamin D i.e. calcitriol which leads to increased bone resorption and gastrointestinal absorption of calcium, leading to hypercalcemia.[15]

The signs and symptoms of hypercalcemia in cancer patients are nonspecific and often associated with many co-morbid conditions in patients with advanced cancer. The initial symptoms include (serum Ca levels >2.6mmol/l) fatigue, malaise, anorexia, nausea, vomiting, confusion, bone pain, polydypsia, polyuria, constipation and weakness. If serum calcium rises above 3.5mmol/l, neurological symptoms are more prevalent and may lead to coma and death if proper measures are not taken.[6]

Initial management includes aggressive therapy with fluid replacement of 5-8 l of saline in first 24 hours in patients maintaining urine output of 3-4 l/day until chronic therapy becomes effective. Chronic therapy includes treatment with bisphosphonates along with hydration. Agents such as gallium nitrate which strongly inhibit bone reabsorption may be used rarely. In patients with seizures and arrhythmias, calcitonin can be used in first 12-24 hours but should not be used alone due to tachyphylaxis. Zoledronate (4mg) by intravenous infusion in half an hour, is found to be more effective in the treatment of severe, acute hypercalcemia. Glucocorticosteroids are used in the treatment of lymphomas.[16]

Hyperviscosity syndrome (HVS)

HVS occurs due to increased viscosity of the blood as result of excessive production of circulating immunoglobulins (Igs) predominantly Igm and also occur with IgA and IgG3 molecules. These proteins increase

the osmotic pressure and resistance to blood flow leading to microvascular congestion, decreased tissue perfusion and tissue damage.[17]

HVS is a common symptom seen in Waldenstrm's macroglobulinemia that accounts for 90% of the cases and myeloma for 5-10% of cases.[18]

The symptoms of HSV vary from one person to other and are non-specific in nature. Retinal changes are more common and are secondary to vascular changes. Other symptoms include bleeding and neurologic abnormalities. Hyponatremia and hypercalcemia are often present. Other clinical symptoms include pulmonary edema, congestive heart failure, ischemic acute tubular necrosis, multiorgan failure and sometimes death.[19,20]

Diagnosis is clinical and confirmed by determination of Serum viscosity. Normal range is 1.4-1.8, symptoms develop when above 4. Treatment of choice is plasmapheresis, where a plasma exchange (fresh frozen plasma) of 3-4 liters in 24 hours is recommended. Maintenance plasmapheresis 1-2 liters once or twice a week may be needed. Definitive cytoreductive therapy should be started as soon as possible.[21]

Hyperleukocytosis syndrome

End organ damage related to markedly elevated WBC counts causing leukemic infiltration and the effects of leukemic cells on the vasculature are the hallmarks of this syndrome. This is very common in up to 30% of adult acute myelogenous leukemias and is the mainstay of their mortality. Poor deformability of leukemic blasts results in sludging of the microvasculature. Increased consumption of oxygen by blast cells, affinity to the pulmonary epithelium and blast cell invasiveness cause local hypoxia.[22]

The symptoms of this syndrome are related to pulmonary system and CNS. Pulmonary symptoms can range from exertional dyspnea to severe respiratory distress. Neurolologic symptoms range from mild confusion to somnolence. The other manifestations of this syndrome includes congestive heart failure, priapism, and peripheral vascular occlusion.[23]

Management should include leukapheresis as chemotherapy causes cell lysis and may aggravate symptoms. Definitive antileukemic therapy can be given after initial therapy reduces the risk to the patient.

Bleeding

Malignant disease and its treatment significantly alter the hemostatic system. Commonest cause for hemorrhage is thrombocytopenia usually the result of chemotherapy or marrow involvement by a tumor. Other causes could be consumptive coagulopathy, immune mediated, infection and sequestration.[24] Bleeding due to platelet dysfunction associated with myeloproliferative disorders are mucosal in nature and may be life threatening when from the GI tract. Malignancies involving liver may cause defective or decreased synthesis of coagulation factors.

Solid tumors capable of inducing fibrinolytic activity include sarcomas, and tumors of the breast, thyroid, colon and stomach. Cytotoxic drugs may contribute to bleeding in patients with malignancies. Asparaginase for acute leukemia is known to be associated with events similar to DIC. Others are plicamycin, suramine, cyclosporine, mitomycin, cisplatin, carboplatin and bleomycin.[25]

The management is governed by the severity of bleeding and treatment of underlying malignancy. Replacement of platelets and coagulation factors may be warranted.

Thrombotic events

These occur in 5-10% of patients and often manifest as DVT, migratory thrombophlebitis, pulmonary embolism and non-bacterial endocarditis.[26]

Management should be focused on treatment of the acute event and reducing risk for subsequent events. Surgical intervention such as embolectomy may be required. Prophylactic low dose enteric coated aspirin or dipyridamole may be considered for susceptible patients.

By organs involved

Cardiac tamponade

Cardiac tamponade can develop with as little as 100-200 ml of fluid in the pericardial sac if accumulated rapidly. Tamponade results from pressure in the pericardial sac exceeding the cardiac filling pressure causing decreased cardiac output and hypotension. The mechanisms involved in the development of tamponade are obstruction of lymphatic drainage or excess fluid secretion from tumour nodules on pericardial spaces.[6]

Pericardial inflammation associated with thoracic radiation or chemotherapeutic agents results in thickening and scarring of the pericardium leading to tamponade with relatively small effusions.[27]

In already diagnosed metastatic cases, about 2/3rd of the patients are asyptomatic. Shortness of breath, chest pain, orthopnea and general weakness are common in symptomatic patients.

2D echocardiography is the best diagnostic tool for assessing pericardial effusion and hemodynamic impact. Pericardial drainage is the only emergent treatment. At the bed-side it is safest under ultrasound guidance. A catheter left in situ for continuous drainage is ideal as the effusion is very likely to recur.[28] Video-assisted thoracoscopic (VATS) pericardial window is another safe and highly effective surgical alternative.[29] Intrapericardial

administration of chemotherapeutics such as bleomycin, carboplatin, or mitomycin-C found to be safe in chemosensitive tumours.[30-32]

Superior vena cava syndrome

More than 90% of cases of superior vena cava syndrome (SVCS) are associated with malignancies, primarily lung cancer and Non-Hodgkin Lymphoma.[33] The thin walled superior vena cava is easily compressed with minimal pressure by extrinsic tumor mass or metastatic lymph nodes. Slow compression allows for development of a collateral system with azygos vein and the internal mammary vein. Rapid compression leads to acute onset of symptoms.

Facial swelling due to edema of the upper body, dilated veins across chest wall and upper extremities, dyspnea, hoarseness and dysphagia common signs of SVCS. Headache suggests raised ICP. Other neurologic symptoms include confusion, coma, brain stem herniation and death.[34]

Management includes reducing ICP, airway management and steroids to reduce edema. Radiation and chemotherapy directed at the underlying malignancy are useful.[34] The most definitive and rapid treatment is stenting of the SVC by interventional radiography in whom radiation and chemotherapy has failed.[35]

Brain metastases

Brain metastases is the most common type of intracranial tumour characterized by raise in intracranial pressure and is common complication in tumours of lung, breast and melanomas.[36] Local growth of these tumours causes mass effect on surrounding tissue which may increase ICP. Angiogenesis due to vascular endothelial growth factor may cause acute hemorrhage.

The common neurologic symptoms are subacute onset of headache, focal neurologic deficits, seizures, neurocognitive changes or combination of these. Other symptoms include headache, nausea, vomiting, weakness, ataxia and change in mental status.[17]

Contrast MRI in stable patients or computerised tomography (CT) scan in unstable patients are the sensitive and reliable diagnostic tools.[17] The first line treatment includes steps to reduce ICP with drugs and hyperventilation. Intravenous bolus dexamethasone injection at an initial dose of 16-40mg, followed then by 40-100mg per day.[37]

Shows its effects within hour and lasts for several days. Steriods are recommended to control vasogenic edema. Newer antiepileptics such as levetiracetam, lamotrigine and lacosamide are preferable as they lack enzyme induction and drug interaction with chemotherapeutic agents.[38] Treatment with radiation depends on degree of brain metastases.[17]

Spinal cord compression

Metastatic spinal cord compression (MSCC) may be an initial presentation of metastatic cancer, but it is much more common in established cases of lung, breast, prostate, and renal solid tumors.[6] It confers a grave prognosis with a median survival rate of six months.

In MSCC, damage to the spinal cord occurs by direct compression causing demylenation, axonal damage and secondary vascular complications. The most common clinical symptom is back pain in about 90% of cases. Others include are weakness, sensory deficits and autonomic dysfunction like urinary retention and constipation indicates more advanced Metastatic Spinal Cord Compression. Less than 10% of patients who are non-ambulatory for more than 24 hours regain the ability to walk.[39]

Magnetic resonance imaging (MRI) is the gold standard technique for diagnosing MSCC with a sensitivity of 93%, specificity of 97%, and overall accuracy of 95%.[40]

Fist line therapy for MSCC includes treatment with corticosteroids. They reduce vasogenic edema, inflammation and also exhibit tumoricidal effect on leukaemias and lymphomas. Emergent neurosurgical intervention for spinal decompression and possible stabilization may be required in severe cases. Radiation and chemotherapy will not be rapid enough to prevent irreversible cord damage.[39]

Airway obstruction

Malignant airway obstruction can arise from locally invasive tumors of the tongue, oropharynx, thyroid, esophagus, trachea, as well as metastatic disease of mediastinum and thoracic lymph nodes.[41] Primary bronchogenic carcinomas are assessed as common cause of malignant airway obstructions and almost 30% of the patients with primary lung cancers will experience airway obstruction in their course time. Prompt recognition and treatment improves the quality of life.[17] The common symptoms include are dyspnea, stridor, wheezing and hemoptysis.

In case of upper airway obstruction, diagnosis can be done by direct visualization via laryngoscopy or bronchoscopy should be done and in case of lower airway obstruction, chest X-ray or CT scan may be done.[6]

Management requires the airway to be established either through an endotracheal tube or by surgical access via a cricothyroidotomy or tracheostomy. Urgent placement of stents for extrinsic compressions or laser resection for luminal tumors are the preferred methods.[42]

Toxic lung injury

Many chemotherapeutic agents such as mitomycin, bleomycin, cyclophosphamide, and carmustine damage the lung tissue because of its large contact surface area and a metabolic site for the drugs.[43] Approximately 1-10% of the patients will be affected by taking one of these drugs. The primary mechanism of injury involved is by free radicals on the endothelium leading to necrosis of pneumocytes.[3] Radiation pneumonitis is an acute syndrome characterized by fever, cough, dyspnea and infiltrates on chest X-ray.

Management involves discontinuing the toxic agent and administrating corticosteroids primarily prednisolone. Patients with late radiation fibrosis respond poorly to steroids as it is a fixed lung injury. Hence, long term supplemental oxygen is often required.[44]

Urinary obstruction

Urinary obstruction occurs in patients with cervical or prostatic cancers. Occasionally metastases in the pelvis may produce urinary obstruction leading to bilateral hydronephrosis and renal failure. Cardinal symptoms include flank pain, sudden anuria and progressive rise in serum creatinine levels. Renal ultrasound is the best way of detecting the etiology. CT scan often helps in detecting the exact location of obstruction i.e. retroperitoneal or pelvic mass.

Management includes the placement of ureteric stents to relive obstruction. If this is not possible, percutaneous nephrostomy is an alternative approach. But after relief there is a disadvantage of incidence of polyuria, increasing the risk of dehydration and electrolyte disturbances. Hence care should be taken for replacement of fluid and electrolytes.[6]

Haemorrhagic cystitis

Management of cervical cancer by external pelvic radiation and brachytherapy may cause hemorrhagic cystitis, particularly if radiation is given after removal of uterus.[45] It is also observed in patients receiving high doses of chemotherapeutic agents such as ifosfamide or cyclophosphamide for longer periods. The incidence is quite high at 40%, despite prophylactic measures.

Common symptoms include dysuria, burning, frequency, gross hematuria, urgency and incontinence. Prophylaxis includes vigorous hydration, continuous bladder irrigation and administration of uroprotective agent mesna. Mesna should always be administered along with ifosfamide or cyclophosphamide to detoxify acrolein and its metabolite in urine thereby reduces the incidence of hemorrhagic cystitis.

Management includes clot evacuation, continuous bladder irrigation, systemic aminocaproic acid as the first line of treatment followed by fulguration. Intractable cases may require urinary diversion, internal iliac ligation, embolization or even cystectomy.[6]

Priapism

Acute, painful prolonged erection of the penis is associated with leukemias and other myeloproliferative disorders. Malignancy associated with hypercoagulable states may also be responsible. Treatment of the underlying cause is the best approach. Corporal shunting may be of some benefit.[46]

Gastrointestinal tract bleeding, perforation and obstruction

Lymphomas are the most likely tumors to directly cause bleeding in GI tract.[47] The perforations may be intrinsic or extrinsic viz. carcinoma cervix eroding into rectum. Management of bleeding includes surgery, use of vasopressin locally, laser ablation and arterial embolization may be useful. Sometimes perforations may require diversion colostomy.

Neutropenic enterocolitis

Chemotherapy induced or neutropenia caused by the disease process itself may present with abdominal distension, typhlitis, watery diarrhea and fever.[48] It is most commonly associated with hematological malignancies and aggressive chemotherapy for carcinoma breast.[3]

Therapy includes both medical and surgical management. Medical management consists of total parenteral nutrition for bowel rest, nasogastric suction, broad spectrum antibiotics and hematopoietic growth factor support. Surgery is reserved for when medical management fails or bleeding, perforation and abscess formation complicate it. Necrotic bowel is resected and bowel diversion performed.[44]

Miscellaneous

Venous thromboembolism and extravasation

Chemotherapeutic drugs like daunorubicin, doxorubicin, epirubicin, actinomycin and mitomycin are vesicants, which cause necrosis when extravasation occurs. Management involves elevation of the limb and cooling of the site with ice packs. Extravasation of vinca alkaloids needs to be managed by warm compress and hyluronidase injection locally. Immediate consultation with a plastic surgeon may be required for emergent debridement to minimize overall injury.[44].

CONCLUSION

As the overall incidence of cancer rises due to varied reasons, the life expectancy of the patients can be

increased with better modalities of treatment and better qualitative care. Emergency physicians and critical care specialist need to be well aware of these oncologic emergencies to tackle them better.

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