Basics of Cancer Care Workshop

“Oncology Emergencies”

Nursing Division,

Ministry of Health Malaysia

Learning Outcomes

By the end of this lesson, learners will be able to:

- Outline the oncology emergencies and managements

Oncology Emergencies

- Oncology emergencies are acute, life-threatening conditions caused either by the cancer itself or by complication of cancer treatment.
- Identifying these early is critical because the window for intervention is often very narrow.

Oncology Emergencies

Metabolic

- Hypercalcaemia
- Tumour Lysis Syndrome (TLS)
- SIADH

Hematological

- Neutropenic Sepsis
- Hemorrhage

Structural

- Cord Compression
- Superior Vena Cava Obstruction (SVCO)

Oncology Emergencies

Metabolic: Hypercalcaemia

Hypercalcaemia

- Hypercalcaemia of malignancy is a common finding in patients with advanced stage cancers.
- Hypercalcaemia is defined as an increase in the serum calcium level above the upper limit of normal for a given reference value used in a laboratory.

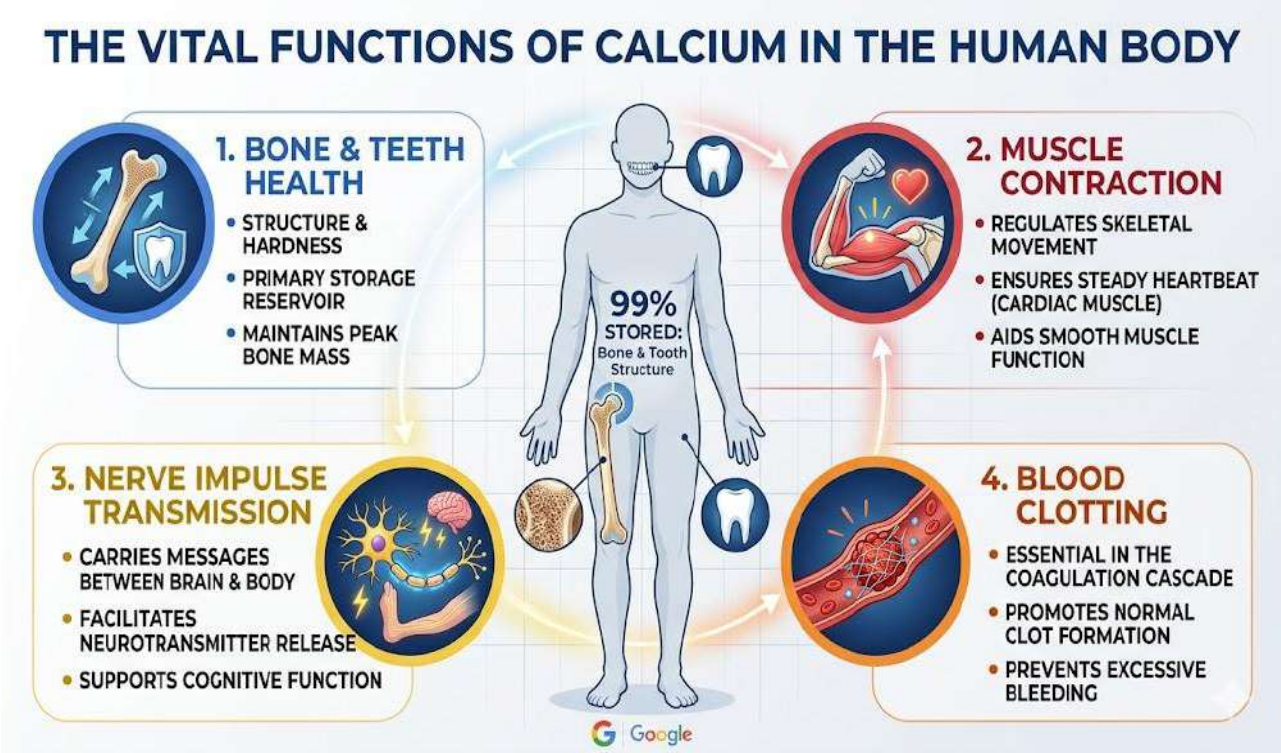


Figure 1. Function of calcium

How Can Hypercalcaemia Occur ?

1. Osteolytic- Metastasis
2. Humoral Hypercalcemia of Malignancy (HHM)

} **PTHrP related**

3. 1.25-Dihydroxyvitamin D

How Can Hypercalcaemia Occur ?

1 Osteolytic- Metastasis

- Bony metastases, which lead to the release of osteoclast-activating factors, account for 20% of hypercalcemia of malignancy cases.
- Tumour cells release PTHrP and cytokines, which stimulate osteoblasts to express RANKL. RANKL binds to RANK on osteoclast precursors, increasing osteoclast activity and bone resorption.
- They are commonly observed in patients with multiple myeloma and solid tumors, such as breast cancer, prostate and lung cancer.

1

Osteolytic-Metastasis

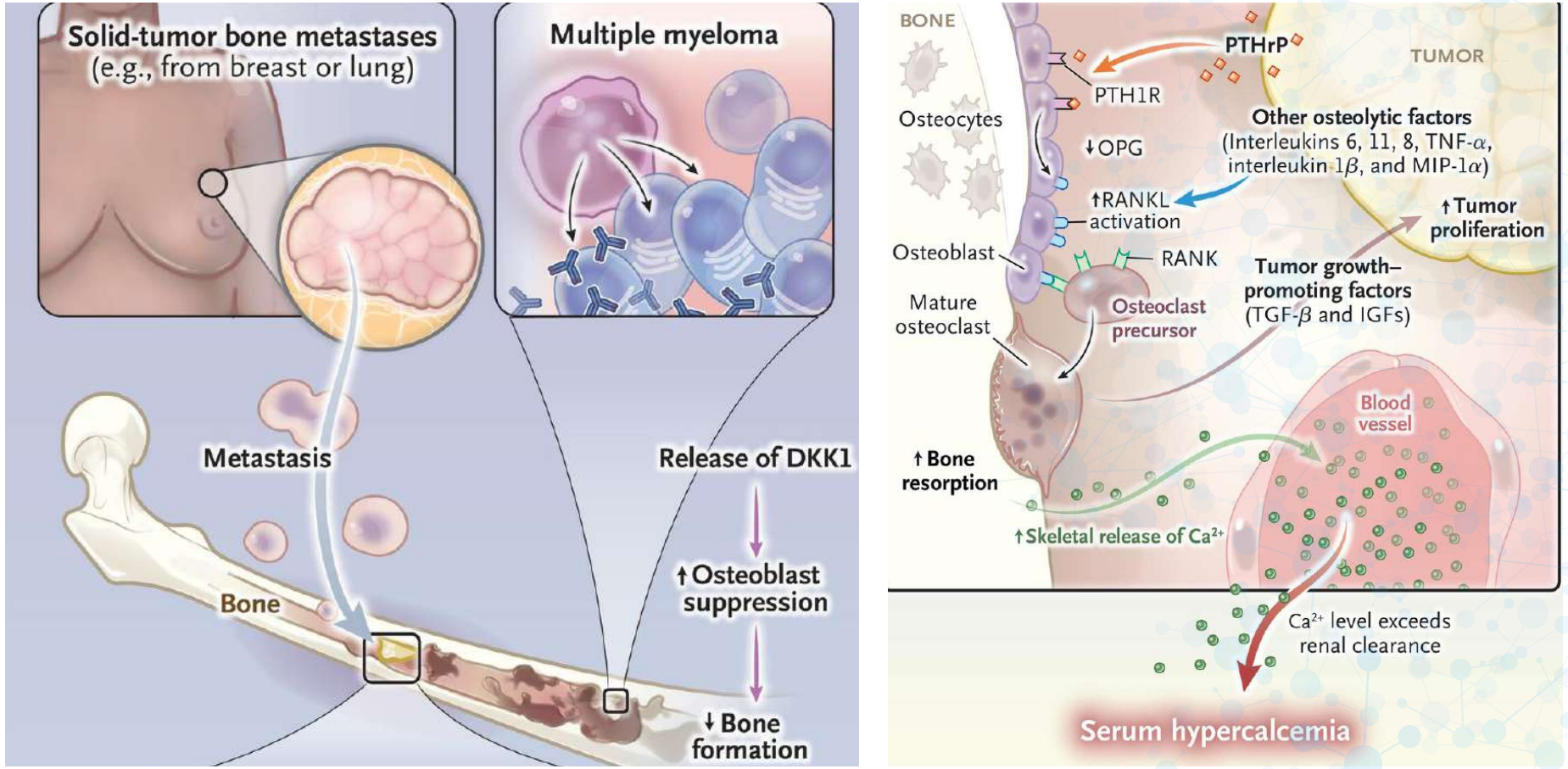


Figure 2. Physiology of Hypercalcaemia cause by osteoclast Metastatic

How Can Hypercalcaemia Occur ?

2 Humoral Hypercalcaemia of Malignancy (HHM)

- Excessive secretion of PTHrP is the most common cause of hypercalcemia of malignancy, accounting for about 80% of cases.
- This is usually seen in solid tumors and hematological malignancies.
- PTHrP binds to the same receptor as PTH, leading to bone resorption, increased phosphate excretion from the proximal tubules, and calcium reabsorption from the distal tubules in the kidneys.
- HHM should be considered when hypercalcaemia occurs without obvious bone metastases, especially with suppressed PTH and elevated PTHrP.

How Can Hypercalcaemia Occur ?

3 1.25-Dihydroxyvitamin D

- Elevated levels of 1.25-dihydroxyvitamin D (1.25-D) are the most frequent cause of hypercalcaemia in lymphoma.
- Vitamin D–mediated hypercalcemia in cancer occurs when cancer cells and surrounding macrophages produce an enzyme (1 alpha-hydroxylase) that converts inactive vitamin D into its active form, 1,25-dihydroxyvitamin D (calcitriol) which increases intestinal calcium absorption and may lead to hypercalcaemia.

How Can Hypercalcaemia Occur ?

1 Osteolytic- Metastasis

Tumour cells in bone release cytokines and PTHrP

↓

These factors stimulate osteoblasts to release RANKL with bind with receptor RANK

↓

Binding activate osteoclast activity cause bone resorption which release Ca to the blood

↓

High amount of calcium in blood = Hypercalcaemia

↑ **PTHrP** ↓ **PTH** ↓ **1,25 $(OH)_2$ D**

✓ **Bone Mets**

2 Humoral Hypercalcemia of Malignancy (HHM)

Tumor cell release PTHrP

↓

PTHrP mimic function of PTH which bind with receptor and activate osteoclast activity

↓

PTHrP increases renal calcium reabsorption and phosphate excretion; osteoclast activation causes bone resorption

↓

Reabsorption led to high amount of calcium in blood = Hypercalcaemia

↑ **PTHrP** ↓ **PTH** ↓ **1,25 $(OH)_2$ D**

3 1.25-Dihydroxyvitamin D

Tumor cell such as Lymphoma release cell enzyme

↓

Enzyme 1 alpha-hydroxylase convert an inactive vitamin D to active vitamin D

↓

High increase vitamin D in blood will increase absorption of calcium from intestine

↓

High amount of calcium in blood = Hypercalcaemia

↓ **PTH** ↑ **1,25 $(OH)_2$ D**

Incidence of Hypercalcaemia

10 - 30%

in cancer patients

(Kanduri et al., 2026)

Aggressive Lymphoma

Produce the active form of vitamin D resulting in increased renal calcium retention and bone reabsorption.

Squamous cell cancer
renal, ovarian, endometrial, and breast cancer

PTH-related peptide secretion - osteoclast activated & increased renal calcium reabsorption

Multiple Myeloma
Acute Leukemia

Investigation of Hypercalcaemia

Lab Parameter

Corrected calcium or ionised calcium, albumin, renal profile, phosphate, magnesium, PTH, PTHrP, vitamin D metabolites, and ECG if severe or symptomatic.

Imaging

- Imaging should be guided by clinical context and suspected malignancy or bone metastases; it is not required for every hypercalcaemia workup.
- MRI, CT Scan, Bone Scan, Pet Scan: To look for metastasis and bone Mets

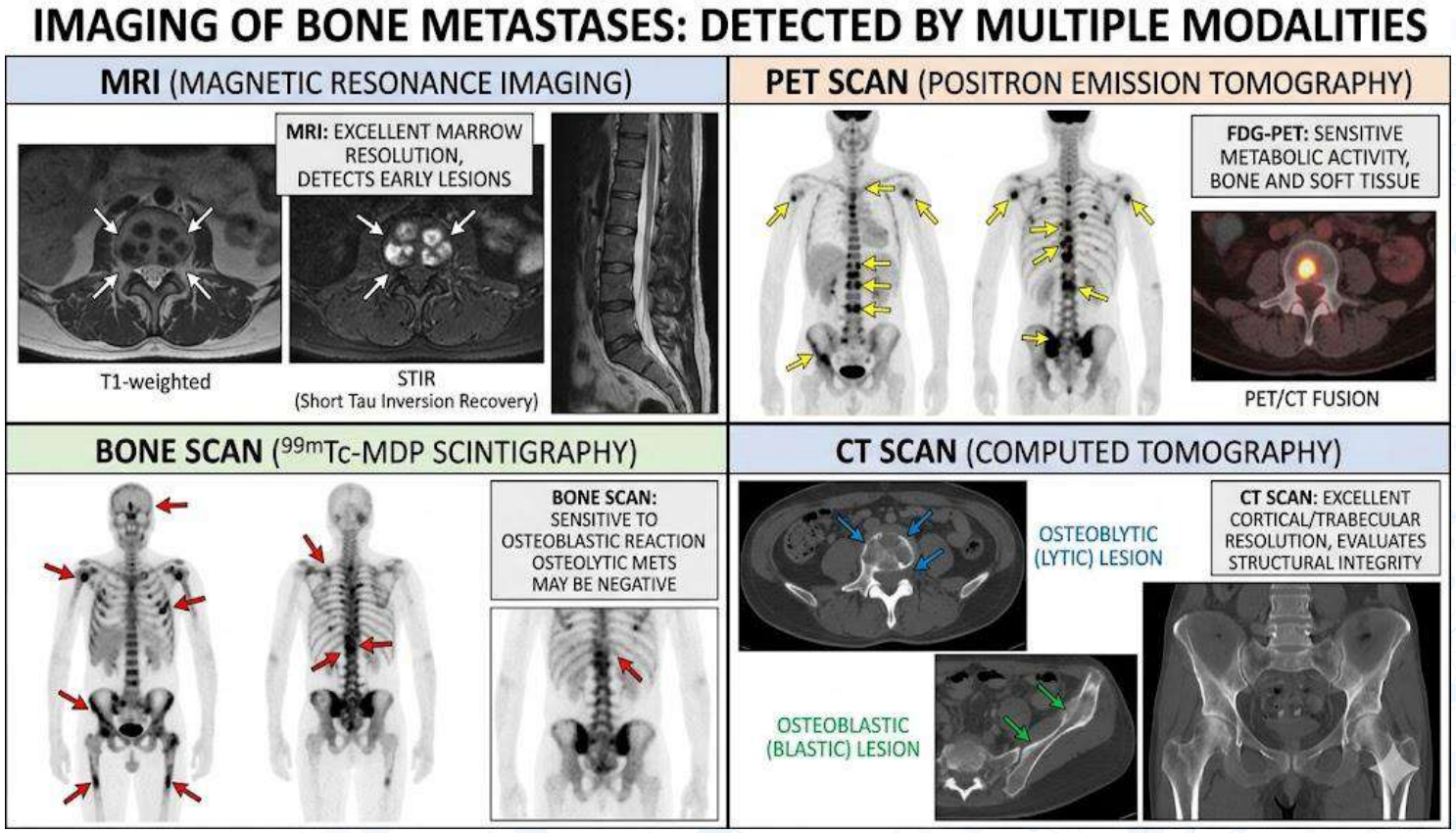
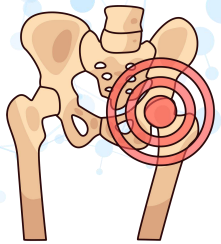


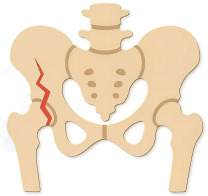
Figure 3. Image investigation for Hypercalcaemia

Clinical Manifestation of Hypercalcaemia

Bones

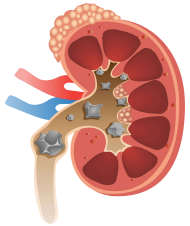


Bone pain

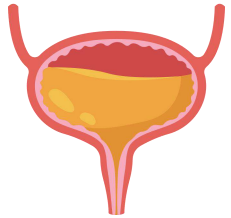


Pathological Fracture

Stones



Renal Stones



Polyuria

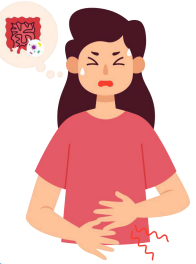


Dehydration

Groans



Nausea & Vomiting



Constipation



Abdominal Pain

Moans



Fatigue



Confusion



Stupor



Change in cardiac rhythm

Management & Intervention

Management

- Hydration
- Low calcium diet intake
- Medical therapy: Bisphosphonate, Denosumab, Steroid, Loop diuretics
- Cancer treatment
- Dialysis
- For severe symptomatic HCM, consider calcitonin with IV bisphosphonate or denosumab; limit calcitonin to 48–72 hours because of tachyphylaxis

Intervention

- Assess the patient for symptoms of hypercalcaemia such as nausea, fatigue, constipation, confusion and dehydration
- Encourage fluid intake to promote excretion of calcium from the body
- Encourage mobilization only if safe; assess fracture risk and falls risk, especially in bone metastases
- Record intake output to ensure patient hydration adequately and prevent fluid overload
- Monitor lab parameter
- Serve medication as prescribed such as biphosphanate, steroid, diuretics

Management & Intervention

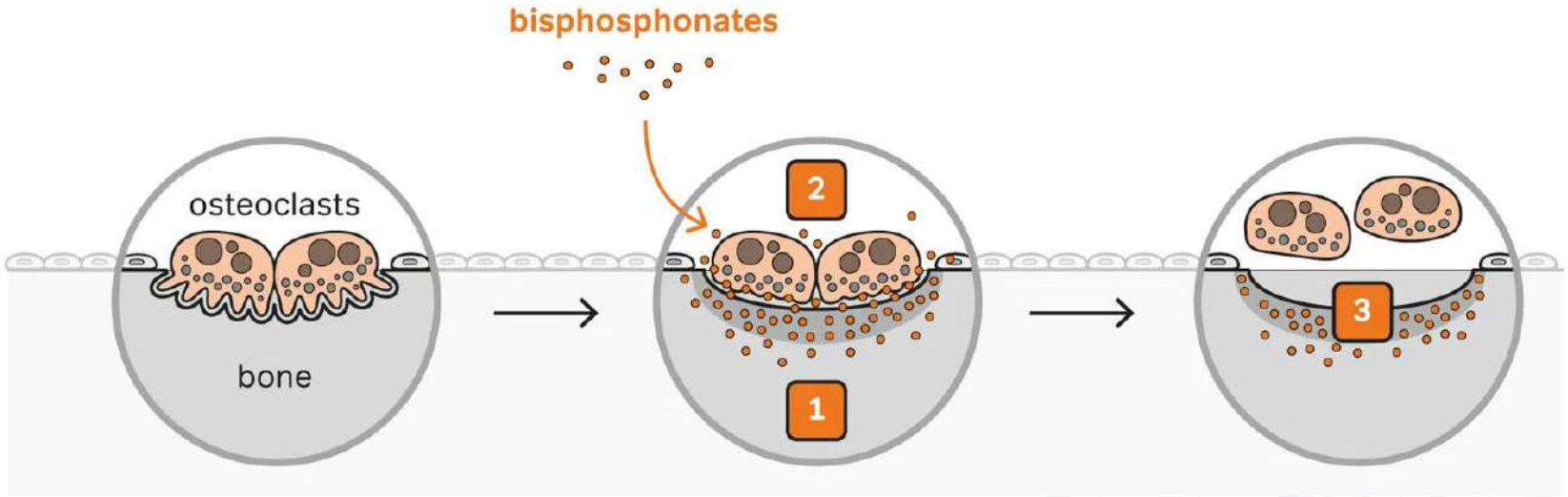


Figure 4. Mechanism Action of Biphosphonates on Bone

Oncology Emergencies

Metabolic: Tumour Lysis Syndrome (TLS)

Tumour Lysis Syndrome (TLS)

- Tumor Lysis Syndrome (TLS) is an oncologic emergency characterized by massive, rapid tumor cell death, either spontaneously or, more commonly, following the initiation of cytotoxic therapy

- This rapid lysis releases huge quantities of intracellular components, principally potassium, phosphorus, and nucleic acids into the bloodstream, quickly overwhelming the body's homeostatic and renal excretory mechanisms.

Pathophysiology of TLS

- Tumour Lysis Syndrome (TLS) develops when many malignant cells undergo rapid lysis, either spontaneously or after cytotoxic therapy (chemotherapy, radiotherapy, corticosteroids, or targeted therapy).
- The massive release of intracellular ions and metabolites into the bloodstream overwhelms the body's normal metabolic and excretory mechanisms, leading to metabolic derangements and multi-organ dysfunction.
- TLS progresses through cellular breakdown, metabolic derangements, renal injury and systemic complications.

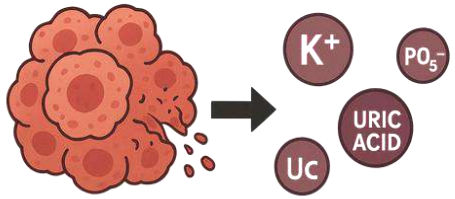
1. Cellular Breakdown
2. Metabolic impact
3. Renal pathophysiology
4. Systemic effects

Pathophysiology of TLS

1. Cellular Breakdown
2. Metabolic impact
3. Renal pathophysiology
4. Systemic effects

- Cancer cells, especially those with high turnover rates or tumour burden such as in leukemia or lymphoma contain large amounts of potassium, phosphate, and nucleic acids.
- When tumour cells rupture, the cell membrane disintegrates, releasing these intracellular substances into extracellular space. The sudden overload exceeds the renal excretory capacity, triggering metabolic imbalances.

Pathophysiology of TLS



1. Cellular Breakdown
2. Metabolic impact
3. Renal pathophysiology
4. Systemic effects

- This Metabolic phase, the killed cell will release intracellular ion such as potassium, phosphate, DNA & RNA to the blood.
- For phosphate, when renal have achieved its maximum level of release, phosphate in blood will bind with calcium and become calcium phosphate. Thus, free calcium in blood will automatically low – hyporcalcaemia.
- Hyperuricaemia begins with the release and breakdown of nucleic acids from DNA and RNA
  Nucleic acids → Hypoxanthine → Xanthine → Uric acid
  (This reaction is cataluzed bu xanthine oxidase).

Pathophysiology of TLS

1. Cellular Breakdown
2. Metabolic impact
3. Renal pathophysilogy
4. Impact on systemic system

Crystalline precipitation within renal tubules- Hyperuricaemia

- Obstruction → increased intratubular pressure → decreased glomerular filtration rate (GFR).
- Leads to acute kidney injury (AKI) and further accumulation of potassium and phosphate

Metabolic overload - Hyperphosphatemia

- Calcium phosphate deposition in renal tubules can cause tubular obstruction and inflammation further decrease renal perfusion and excretory function.
- Exacerbates hypocalcemia and renal failure.

Reduced Renal Perfusion

- Caused by dehydration, hypotension, or systemic inflammatory effects.
- Leads to decreased blood flow to the kidneys, reducing glomerular filtration rate (GFR).
- Results in worsened excretion of potassium, phosphate, and uric acid, aggravating acute kidney injury (AKI).

Pathophysiology of TLS

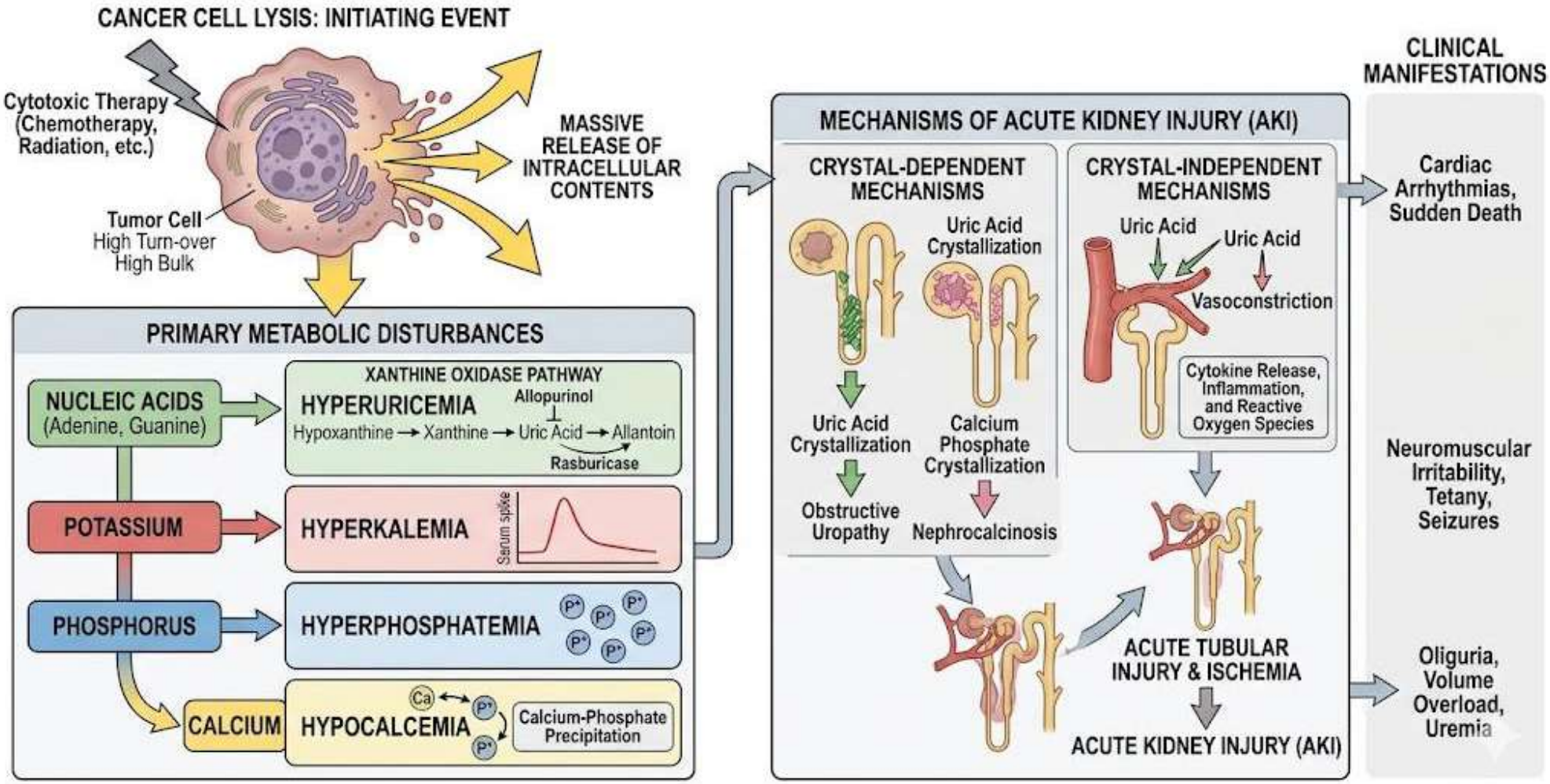


Figure 5. Summary of pathophysiology of Tumour Lysis Syndrome (TLS)

Risk Factors of TLS

Tumor Related

- High grade lymphoma
- Acute leukemia
- Bulky disease
- High chemosensitive tumours

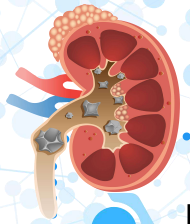
Patient Related

- High baseline LDH, uric acid, phosphate and potassium
- Pre-existing renal dysfunction
- Large tumor burden

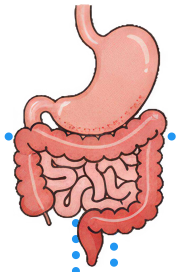
Treatment Related

- Chemotherapy
- Targeted therapy
- Biologic agents' radiation therapy

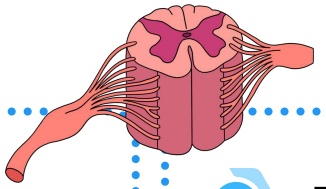
Clinical Manifestation of TLS



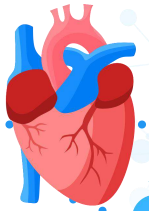
- Hematuria
- Flank Pain
- Edema
- Fluid overload
- Oliguria
- Metabolic acidosis



- Nausea
- Anorexia
- Diarrhea
- Vomiting



- Tetany
- Weakness
- Lethargy
- Paraesthesia
- Seizures



- Elevated T Waves
- Dysrythmias
- Arrhythmias
- Bradycardia
- Wide QRS

Investigation of TLS

- Lab Parameter
  - FBC, renal profile/creatinine, potassium, phosphate, calcium, uric acid, LDH, bicarbonate, and urine output monitoring.
- Electrocardiogram (ECG)

Tumor Lysis Syndrome: $Ca\downarrow$ $K^{+}\uparrow$ $PO_4\uparrow$ Uric Acid$\uparrow$ LDH$\uparrow$

Management of TLS

Prevention

- Identify patient with risk factor.
- Monitor lab parameter: Renal Profile, LDH, Uric Acid, Calcium, Phosphate.
- Hydration prior and post treatment.
- Use hydration plus allopurinol for many intermediate-risk patients; consider rasburicase for high-risk patients or established TLS according to local protocol.

Intervention

- Investigation: Lab Parameter and Electrocardiogram.
- Strict intake output charting.
- Encourage adequate oral fluid intake.
- Escalate early to oncology/ haematology, nephrology and ICU/ anaesthesia when clinically indicated.

Oncology Emergencies

Metabolic: Syndrome of Inappropriate Antidiuretic Hormone secretion (SIADH)

SIADH

- A metabolic emergency characterized by excessive, non-physiological release of Arginine Vasopressin (ADH), resulting in impaired water excretion.
- Hyponatremia with euvolemia. The body retains water, diluting serum sodium levels despite normal salt intake.
- SIADH is an important and common cause of euvolaemic hyponatraemia in cancer patients, particularly in SCLC. Severe drops in sodium can lead to life-threatening cerebral edema.
- Commonly associated with Small Cell Lung Cancer (SCLC). Up to 15% of SCLC patients develop clinically significant SIADH.

Pathophysiology of SIADH

Ectopic Antidiuretic Hormone (ADH)

- In cancer patients, tumor cells most notably SCLC act as autonomous endocrine organs. They synthesize and secrete ADH independent of plasma osmolality or volume status.

- This "inappropriate" secretion bypasses the normal physiological feedback loop of the hypothalamus-pituitary axis.

Pathophysiology of SIADH

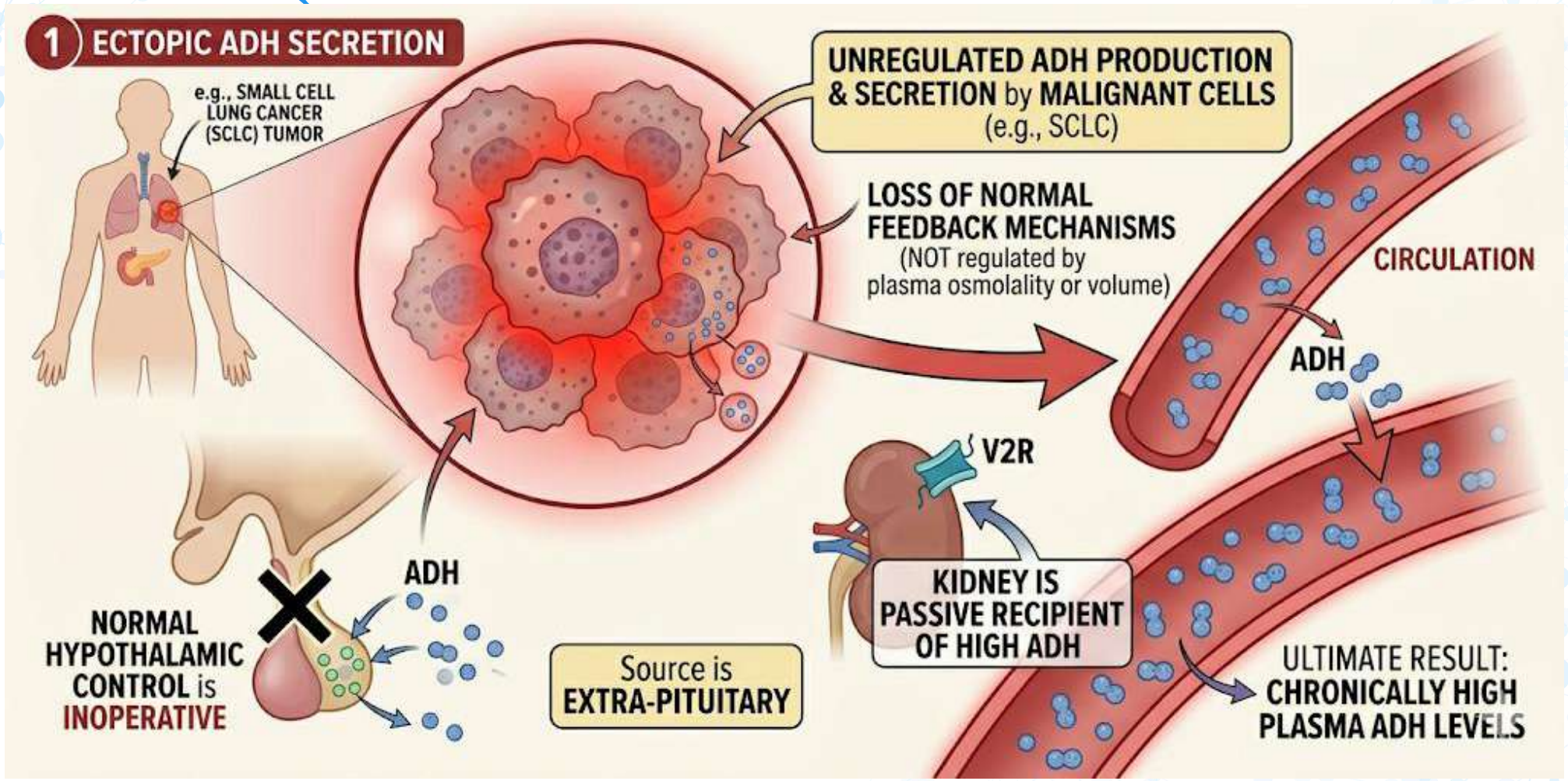


Figure 6. Pathophysiology of SIADH in ectopic ADH secretion

Pathophysiology of SIADH

Renal Action:

- Excessive ADH binds to V2 Receptors in the basolateral membrane of the renal collecting ducts.
- This triggers the translocation of Aquaporin-2 water channels to the luminal surface, massively increasing water reabsorption.

Investigation

Blood Parameter

- Sodium: Reference range 136 - 145 mmol/L
- Serum Osmolality: 275 - 295 mOsm/kg H2O
- Urine Sodium: < 20-40 mmol/L
- AM Cortisol: 166.0 - 507.0 nmol/L
- Thyroid Function Test:
  - Thyroid Stimulating Hormone: 0.270 - 4.200 mIU/L
  - Free Thyroxine: 11.900 - 21.600 pmol/L
- Urine Osmolality

Clinical Manifestation of SIADH

Mild: Asymptomatic

Medium:

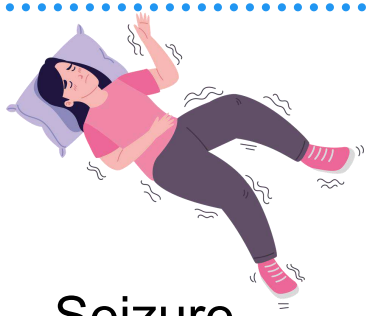

Malaise

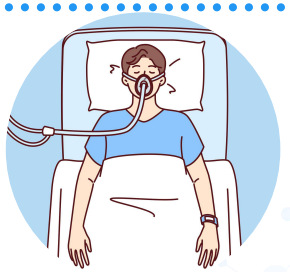

Nausea

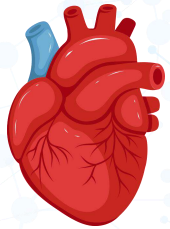

Vomiting


Headache

Severe:


Seizure


Coma


Cardiorespiratory arrest


Fluctuate GCS

Management of SIADH

- Restrict fluid 800 - 1000 mls /day.
- Monitor intake output chart, blood parameter.
- Stop medication that may cause hyponatremia.
- Increase sodium chloride intake through diet.
- Medication:
  - Oral Sodium Chloride 1-2g TDS.
  - Oral Vasopressin V2 Receptor Antagonist: Act on distal nephron which promote excretion of water without affecting excretion of electrolytes.
- Severe symptomatic hyponatremia, for example seizure or reduced consciousness, requires urgent medical review and controlled correction, often with hypertonic saline according to local protocol.

Nursing Intervention of SIADH

- Assess patient condition for altered mental status due to hyponatremia.
- Nursing safety: Safe environment to prevent fall and precaution for seizure.
- Monitor intake output chart, vital sign and blood parameter.
- Educate patient and family for fluid restriction, the importance of compliance.
- Served medication as prescribed by the doctor
  - Oral Sodium Chloride 1-2g TDS
  - Vaptans: If the patient is on Vasopressin antagonists (e.g., Tolvaptan), monitor for rapid diuresis and thirst.

Use vaptans only under specialist advice with close sodium monitoring because over-rapid correction can be harmful

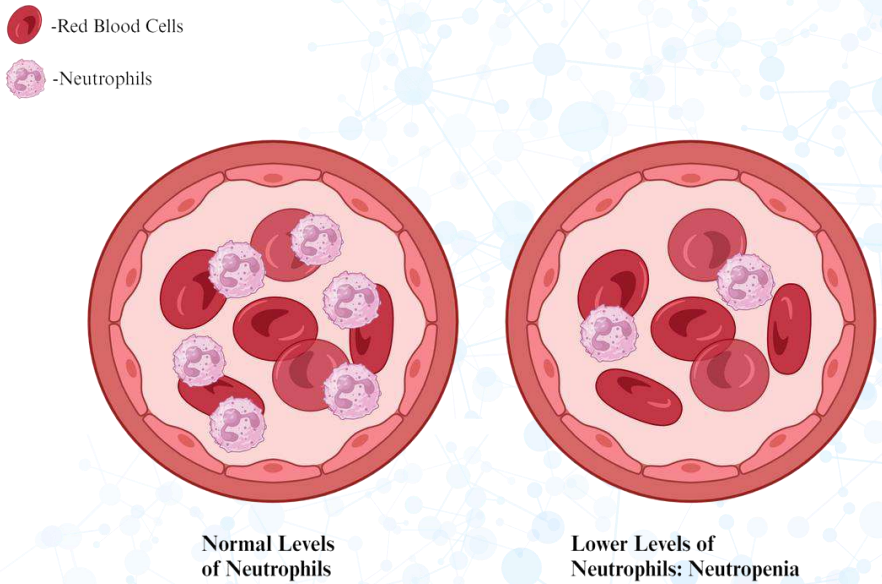


Oncology Emergencies

Hematology: Neutropenic Sepsis

Neutropenic Sepsis

- Neutropenic sepsis is suspected in a patient receiving anticancer treatment who is unwell or febrile with neutropenia.
- Neutrophil count ≤0.5 × 10⁹/L with fever >38°C or other signs of clinically significant sepsis; or ANC <1.0 × 10⁹/L with predicted fall.
- Neutrophil act as defense mechanism in protecting body from any infection to our body.
- Neutrophils constitute 40 to 70% of total circulating white blood cells.
- Mature neutrophils have a half-life of about 2 to 3 days. Timing varies by chemotherapy regimen, dose and patient factors (most of chemo nadir 7-14 days).



Pathophysiology of Neutropenic Sepsis

- Chemotherapy agents target rapidly dividing cells, including hematopoietic stem cells in the bone marrow.
- The resulting nadir (lowest cell count) typically occurs 7–14 days after treatment, leaving the body without its primary defense: neutrophils.
- Impaired immunity allows for the rapid translocation of endogenous flora into the bloodstream, triggering systemic inflammatory response syndrome (SIRS).

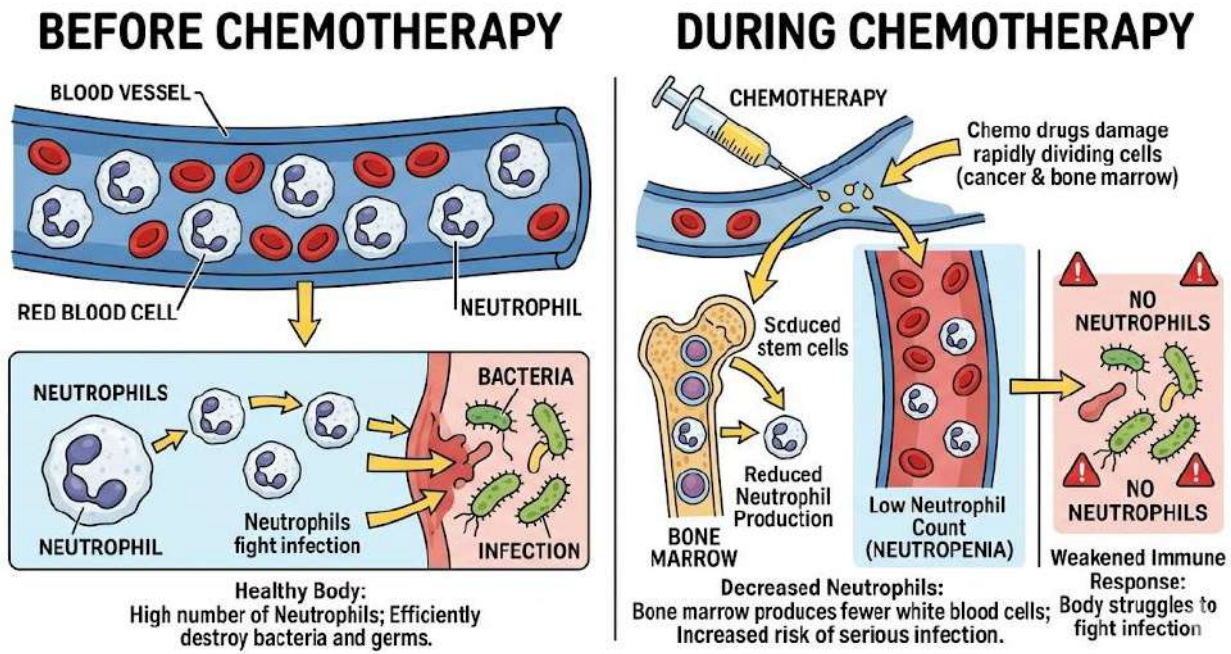


Figure 7. Pathophysiology of neutropenic sepsis

Risk Factor of Neutropenic Sepsis

Patient Related

- Older age, especially above 60 years, increases risk
- Malignancy Type:
  Hematology Cancer high risk compared to solid cancers
- Comorbid Status:
  Patient with renal problem, hematology disease or ECOG >2

Treatment Related

- Type of treatment:
  Combination treatment such as Chemo + Radiotherapy
- Indwelling devices
- Prior history of having neutropenia sepsis

Investigation for Neutropenic Sepsis

- FBC, renal and liver function, CRP, lactate and blood cultures; add cultures from central venous catheter if feasible and clinically appropriate

Full Blood Count (FBC)

	Flag	Result	Unit	Reference Range
White Blood Cell	L	2.26	10^9/L	4.00 - 10.00
Red Blood Cell				3.80 - 4.80
Haemoglobin	L	7.4	g/dL	12.0 - 15.0
Haematocrit	L	0.22	L/L	0.36 - 0.46
Mean Cell Volume		94.0	fL	83.0 - 101.0
Mean Cell Haemoglobin		31.6	pg	27.0 - 32.0
Mean Cell Haemoglobin Concentration		33.6	g/dL	31.5 - 34.5
Red cell Distribution Width - CV		13.4	%	11.6 - 14.0
Platelet		204	10^9/L	150 - 410
Mean Platelet Volume		10.3	fL	9.2 - 12.8
Neutrophil Absolute	L	0.34	10^9/L	2.00 - 7.00
Neutrophil %		15.1	%	40.0 - 80.0
Lymphocyte Absolute	L	0.87	10^9/L	1.00 - 3.00
Lymphocyte %		38.5	%	20.0 - 40.0
Monocyte Absolute	H	1.02	10^9/L	0.20 - 1.00
Monocyte %	H	45.1	%	2.0 - 10.0
Eosinophil Absolute		0.02	10^9/L	0.02 - 0.50
Eosinophil %	L	0.9	%	1.0 - 6.0
Basophil Absolute	L	0.01	10^9/L	0.02 - 0.10
Basophil %		0.4	%	< 2

Figure 8. Abnormal result of Full Blood Count (FBC)

Clinical Manifestation for Neutropenia Sepsis



Chills and rigor



Fever > 38'C



Diarhea



Nausea and vomiting



Respiratory symptoms



Cough and Sore Throat



Lethargic



Altered Mental Status



Hypotension

Management for Neutropenic Sepsis

- Treat as an acute medical emergency and give empiric IV antibiotics immediately; choice should follow local protocol

  Start IV Antibiotic Broad Spectrum within one hour:
  IV Tazosin/ IV Cefepime/ IV Meropenem

  GOLDEN HOUR

- Isolate patient in isolation room
- Daily FBC until TWC and Neutrophil in normal range
- Medication: S/C Granulocyte colony-stimulating factor (G-CSF)

  Stimulates the bone marrow to produce white blood cells (neutrophils) and stem cells, releasing them into the bloodstream to prevent infection, particularly after chemotherapy

- Refer hematology team for expert opinion if not response to treatment
- Treat underlying symptoms accordingly: Diarrhea, Shortness of Breath (SOB), respiratory symptoms and altered mental status



Health Education to Prevent Infection

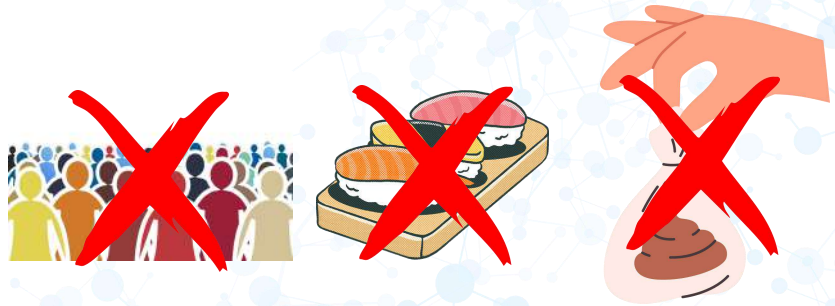
Do's



- Maintain hygiene:
  Wash hands, daily showering
- Eat fresh food, consume within 4 hours and drink boiled water
- Wear mask in crowded area
- Eat well balanced and clean food
- Take medication as prescribe
  eg: S/C GCSF @ Pegfilgrastim as prescribed for prophylaxis

Don'ts

- Share meal utensil
- Eat raw food such as sushi
- Handling pet waste and gardening
- Go to crowded place



Oncology Emergencies

Hematology - Haemorrhage

Introduction

- Clinically significant bleeding occurs in approximately **3.9% - 10%** of patients with advanced, incurable cancer. (Johnstone & Rich, 2018).
- In advanced cancer, major bleeding may occur as a terminal event, but it can also occur earlier and may be treatable depending on cause, site and performance status.
- Haemorrhage may manifest in a variety of ways, including hematemesis, hematochezia, melena, hemoptysis, hematuria, epistaxis, vaginal bleeding, or ulcerated skin lesions.

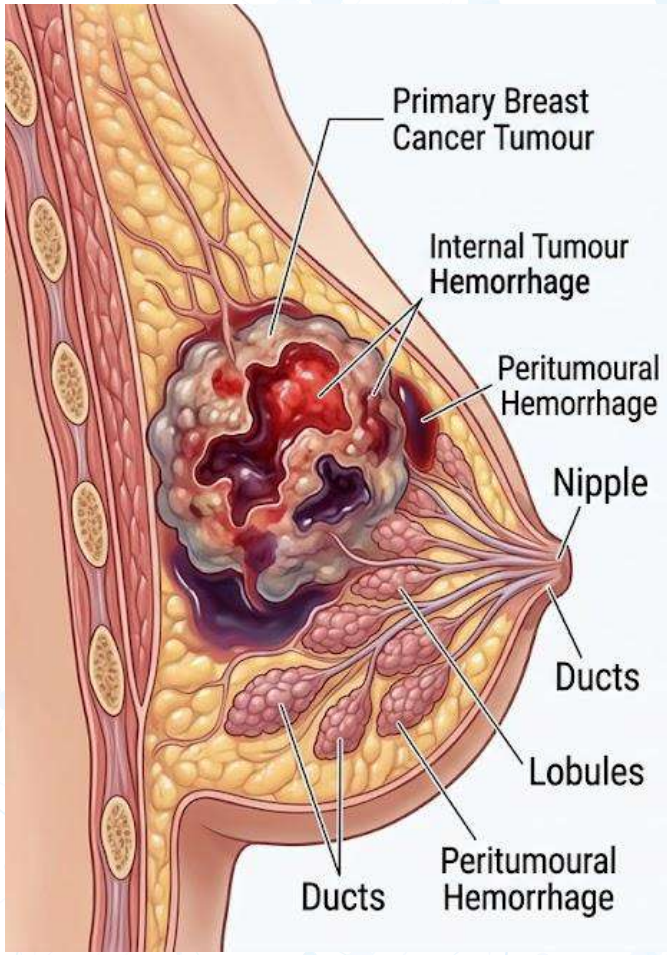


Figure 9. Tumour in Breast

Risk Factor of Haemorrhage

Direct Tumour Invasion

- Direct tumor invasion damaging blood vessels

Haematology Related

- Thrombocytopenia
  - Reduced platelet counts, often caused by bone marrow suppression from chemotherapy or radiation, inhibit clotting.
  - Bone Marrow Failure
  - DIC can cause both thrombosis and bleeding; present it separately as coagulopathy-related bleeding risk

Treatment Related

- Treatment-Related Complications
- **Anticoagulants**: Blood thinners increase bleeding risk.
- **Targeted Therapies**: Anti-angiogenic drugs (e.g., bevacizumab)
- **Surgery/Radiation**: Procedures can cause immediate or delayed bleeding.

Tumour Related



Head and Neck Cancer

- Carotid Blowout
- Rupture of the carotid artery due to tumor erosion or necrosis, often through a cutaneous ulcer.



Respiratory

- Hemoptysis
- Lung cancer
  - This can from blood-streaked sputum to massive, life-threatening hemorrhage.



Gastrointestinal

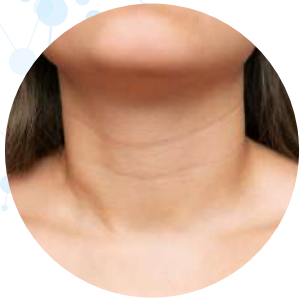
- Haematemesis / Malaena
- Upper GI bleeding



Urological

- Haematuria
- Clot retention occurs when blood clots obstruct the urethra or bladder neck, causing acute pain.

Risk Factor of Haemorrhage - Tumor Related



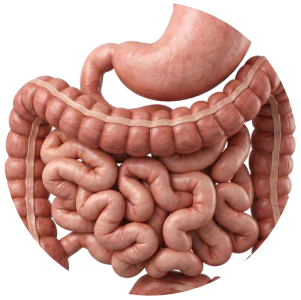
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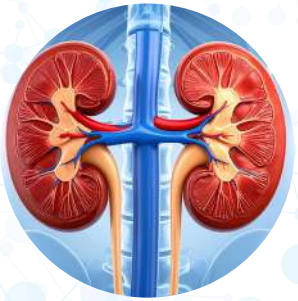
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Gastrointestinal

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Urology

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Management



Dressings, packing, and topical agents

- Hemostatic Gauze: Gauze impregnated with agents like Kaolin (QuikClot) or Chitosan (Hemcon) actively speeds up clotting.
- Topical Hemostatic Agents - Tranexamic Acid, Adrenaline (1:1000), Sucralfate Paste, Silver Nitrate Sticks, Surgical Hemostats: Materials like Gelfoam.
- Compression dressing



Radiotherapy

- Haemostatic radiotherapy can palliate tumour bleeding; onset and response vary by tumour site, dose, fractionation and bleeding mechanism.

Endoscopic procedures

- Identify and treat bleeding tumors in the visualized organs.

Transcutaneous Arterial Embolization

- Successful hemostasis is reached in **70–99% of patients** (Takeda et al., 2026)



Nursing Intervention

Immediate Life - Saving Actions

- Apply Direct Pressure: For external bleeding
- Positioning - to promote venous return to the heart and maintain cerebral perfusion.
- Airway and Oxygen

Fluid and Volume Resuscitation

- Establish IV Access - Insert two large-bore peripheral IV
- Fluid Replacement - to maintain blood pressure
- Blood Transfusion

Monitoring and Assessment

- Vital signs, urine output, mental status and laboratory parameter monitoring

Pharmacological

- Vasopressors may be required for shock after appropriate resuscitation

Oncology Emergencies

Structural: Spinal Cord Compression

Cord Compression

- Metastatic Spinal Cord Compression (MSCC) occurs when a malignant growth compresses the spinal cord

- Patients at risk include those with cancers that spread to the vertebrae, epidural space or spine, causing spinal cord or cauda equina compression

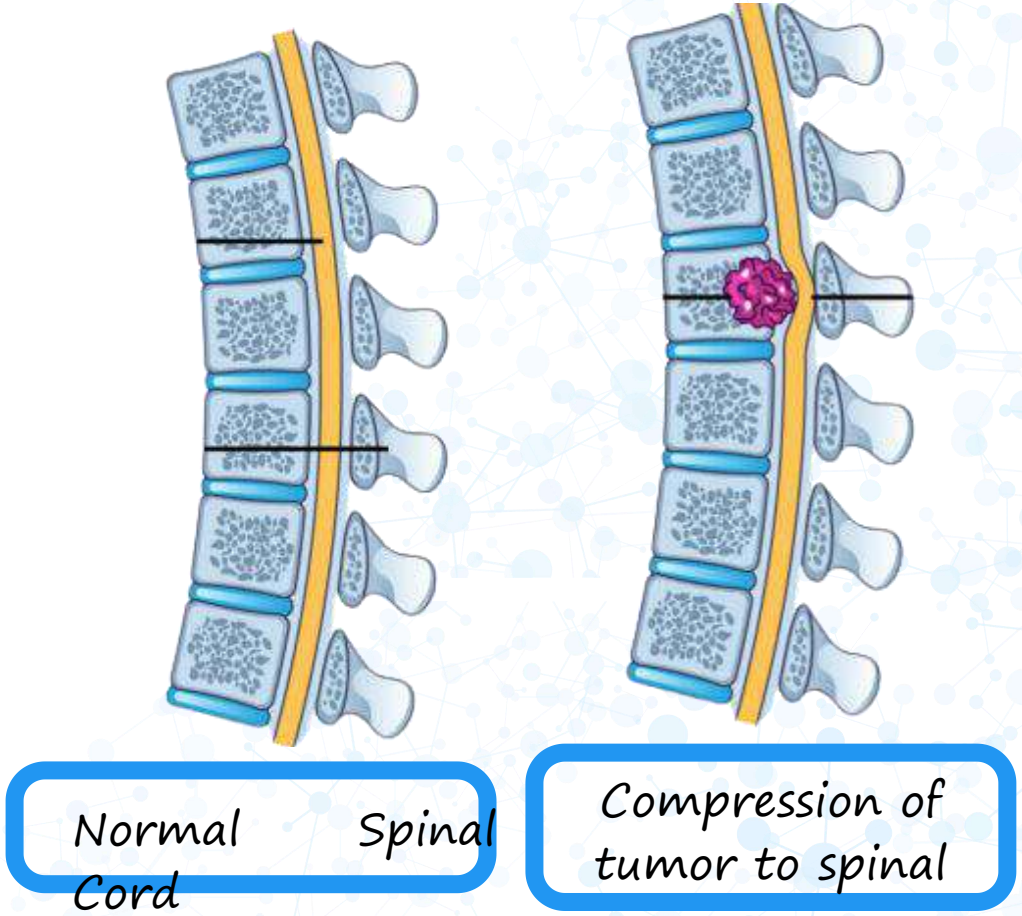


Figure 10. Differences between normal and abnormal spinal

Pathophysiology of MSCC

1. **Tumor Seeding:**
   Hematogenous spread usually targets the vertebral bodies (85%) (Bai et al., 2022).

2. **Epidural Expansion:**
   The tumor expands into the epidural space, compressing the venous plexus.

3. **Vasogenic Edema:**
   Venous congestion leads to edema in the spinal white matter.

4. **Ischemia:**
   Sustained pressure results in irreversible ischemic nerve damage.

Risk Factor of MSCC

1. Primary Malignancy: Breast Cancer, Lung Cancer, Prostate Cancer and Multiple Myeloma.

2. Bone Metastasis or previous bone lesion.

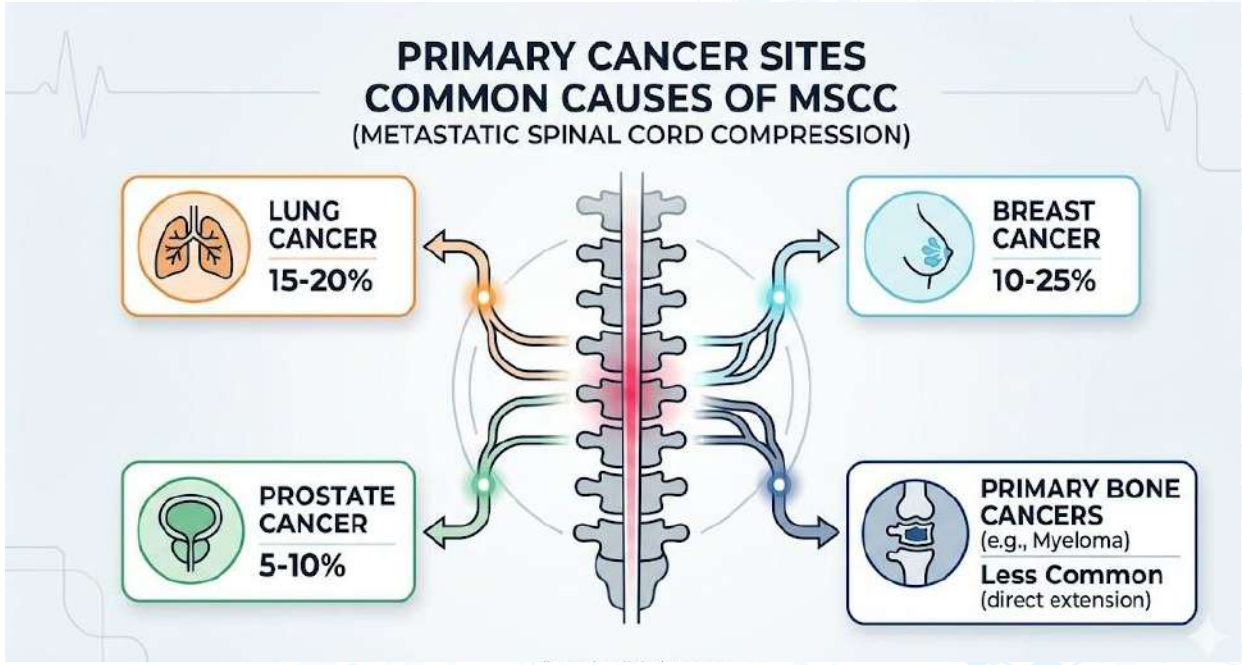


Figure 11. Risk Factor of Cord Compression

Investigation for MSCC

- **Magnetic Resonance Imaging (MRI):** MRI whole spine is the investigation of choice for suspected MSCC and should be done urgently, ideally within 24 hours.
- **Myelogram:** To detect presence of tumor or abnormalities of the spinal cord and vertebrae.
- **CT Scan:** To identify tumor mass, bone erosion or compression.
- **Bone Scan:** Identify metastasis to vertebral bodies.
- **X-ray Lumbar Sacral:** Avoid using plain X-ray to diagnose or rule out MSCC. Use MRI; CT if MRI contraindicated.

Clinical Manifestation for MSCC

95% with MSCC will complaint of **BACK PAIN**

(Boussios et al., 2018)

- **Motor Deficit:** Heavy legs, unsteady gait (ataxia), and progressive weakness. Often bilateral but can be asymmetrical.
- **Autonomic Loss:** Bladder, Bowel dysfunction and loss of anal tone are late signs and often indicate poor prognosis for recovery.
- **Sensory Loss:** Paresthesia, Numbness.

Management for MSCC

Medication:

- For suspected MSCC with neurological symptoms/signs, NICE recommends dexamethasone 8 mg BD orally while awaiting surgery or radiotherapy, with glucose/PPI monitoring.
- Pain Killer: Syrup Morphine 4 Hourly

Diagnostic Investigation:

- Xray Lumbar Sacral (baseline to detect any fracture)
- Arrange MRI whole spine as soon as possible, always within 24 hours when MSCC is suspected

Radiotherapy:

Palliative radiotherapy to reduce the compression and tumor size, improve motor function

Multi-Disciplinary Team (MDT):

Surgery type depends on spinal stability, prognosis and tumour factors.

Nursing Intervention

Prevention of fall

- Assist patient when ambulation.
- Use assistance device: wheelchair, walking frame etc.
- Locate personal belongings near to patient.
- Rail up both side when in bed.
- Avoid slippery slippers.
- Environment: organize things, no slippery mat, enough light for ambulation

Prevention of pressure injury

- 2 Hourly turning position.
- Ripple mattress.
- Change diaper every 3-4 hours.
- Maintain hygiene.
- High protein diet.

Continence Management

- Catherization if indicated.

Exercise

- Refer physiotherapy for assessment and plan for exercise.
- Assist patient with passive and active range of motion.

Venous Thromboembolism (VTE) to prevent Deep Vein Thrombosis:

- S/C Clexane
- Tab Rivaroxaban

Oncology Emergencies

Superior Vena Cava Obstruction (SVCO)

SVCO

- Superior vena cava (SVC) syndrome is a collection of clinical signs and symptoms resulting from partial or complete obstruction of blood flow through the SVC.
- The SVCO is caused by thrombosis, invasion or extrinsic compression involving the SVC or nearby structures.
- Results from compression, invasion or thrombosis of the SVC.

Pathophysiology of SVCO

- Superior Vena Cava Obstruction (SVCO) is caused by the external compression, direct invasion, or thrombosis of this thin-walled, low-pressure vessel.
- The resulting impairment of venous return from the upper body leads to a profound increase in hydrostatic pressure within the venous circuits of the head, neck, and upper extremities, manifesting clinically as Superior Vena Cava Syndrome (SVCS).
- Increased venous pressure and impaired blood flow from the head, neck, and upper extremities.

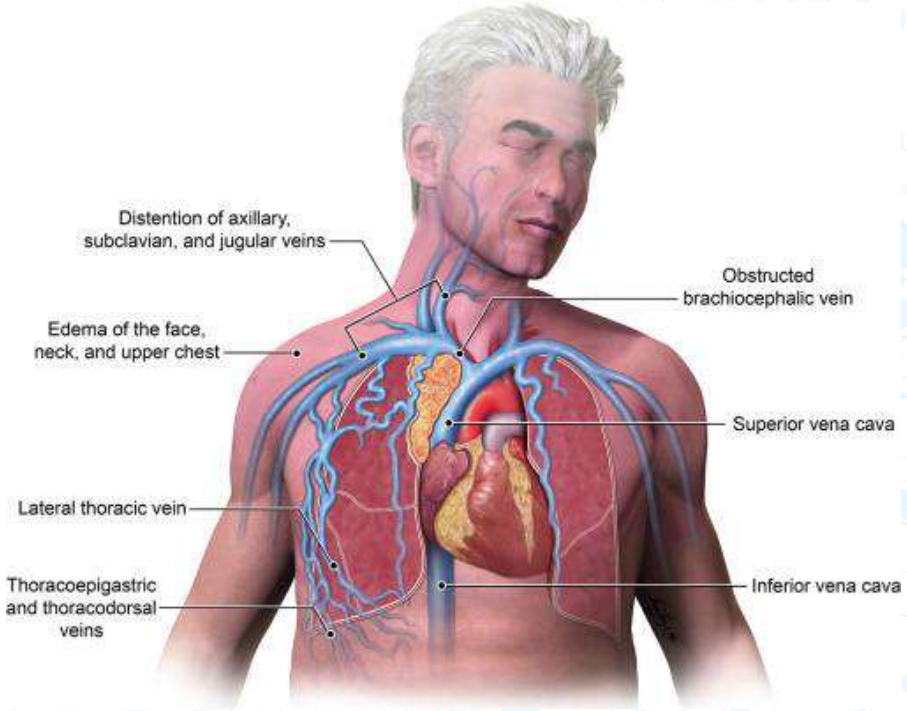


Figure 12. Overview Superior Vena Cava Obstruction

Risk Factors SVCO

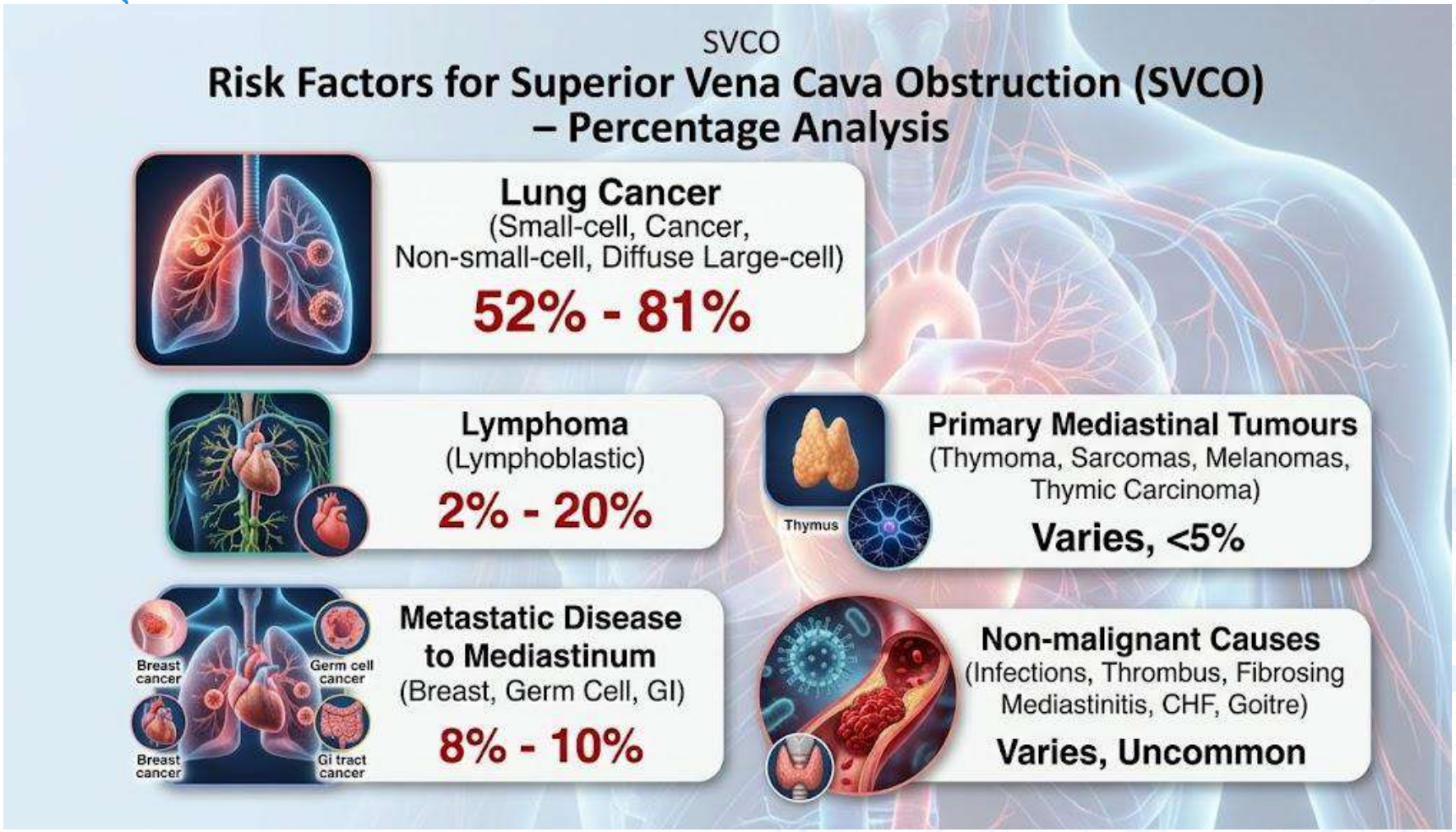


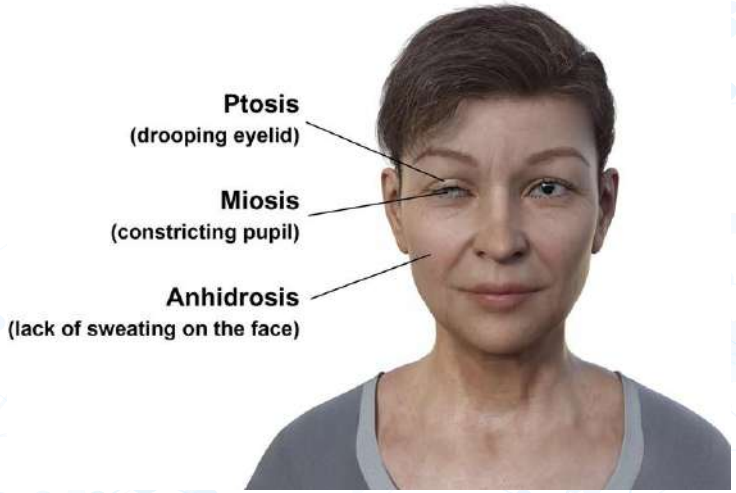
Figure 13. Risk Factor of Superior Vena Cava Obstruction (SVCO) (Patriarcheas et al., 2022)

Clinical Manifestation of SVCO

- Edema on the face, neck, upper thorax and breast and upper extremities.
- Respiratory compromise.
- Dilatation and prominence of the collateral vessels in upper thorax and neck.
- Compensatory tachycardia.



Telangiectasis



Horner's syndrome

Clinical Manifestation of SVCO

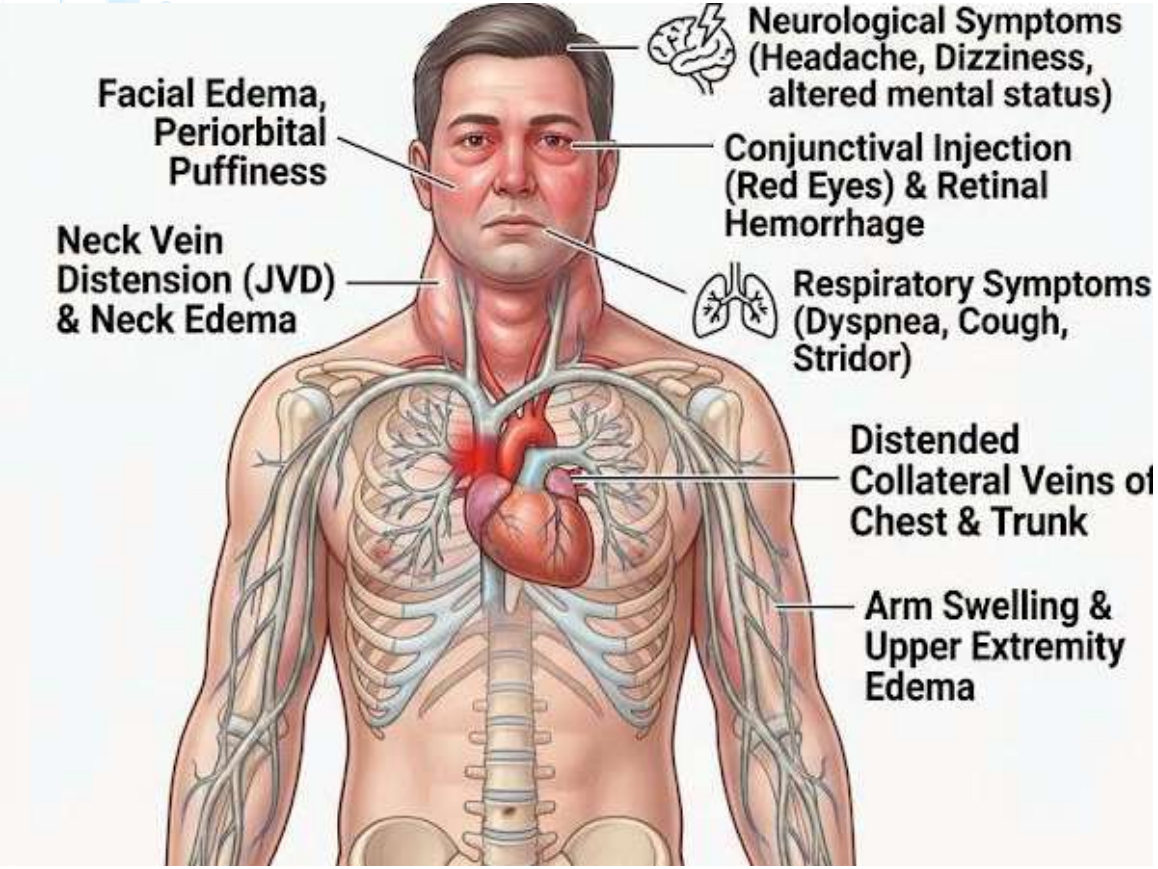


Figure 13. Clinical Manifestations of Superior Vena Cava Obstruction



Figure 14. Pemberton Sign

Investigation of SVCO

- Physical assessment
- CXR:
  - Superior mediastinal mass
  - Mediastinal widening
  - Pleural effusion
  - Hilar mass
  - Aortic nipple

CXR may show mediastinal widening or mass, but contrast-enhanced CT is usually more useful to identify level, cause, thrombus and collateral vessels

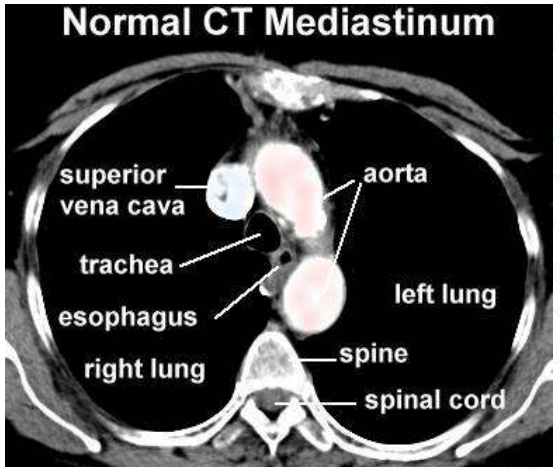


Figure 15. Image for normal SVC

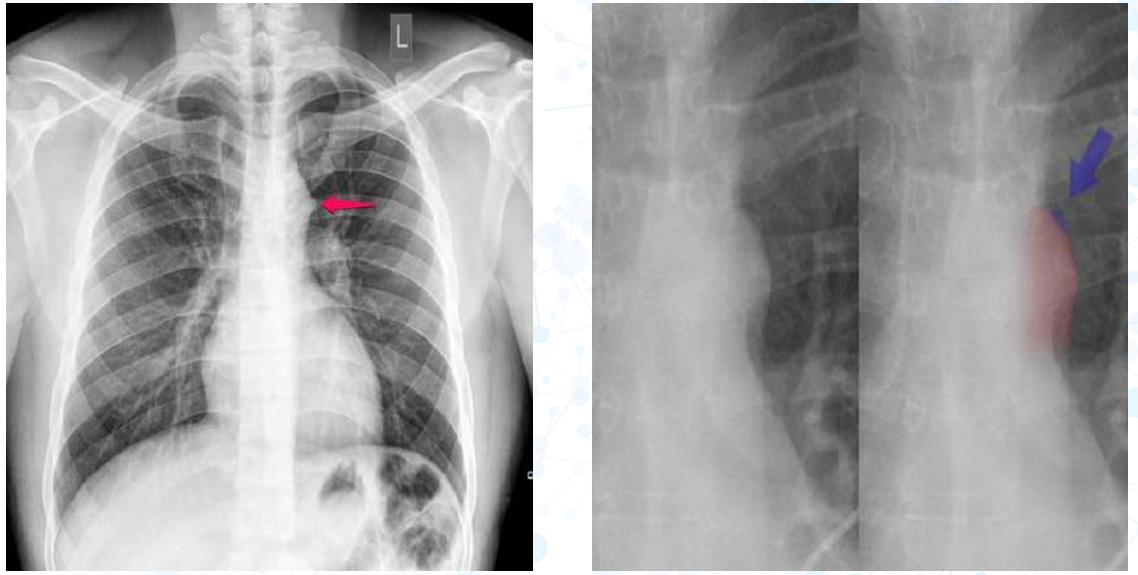


Figure 16. Image for abnormal SVC

Investigation of SVCO

- **MRI/ CT Scan Imaging**
  - Localise the level of SVCO and underlying pathology
  - Tumour mass size
  - Assess SVC diameter
  - Length of stenosis/obstruction
  - Evidence of SVCO thrombus and formation of collateral vessels

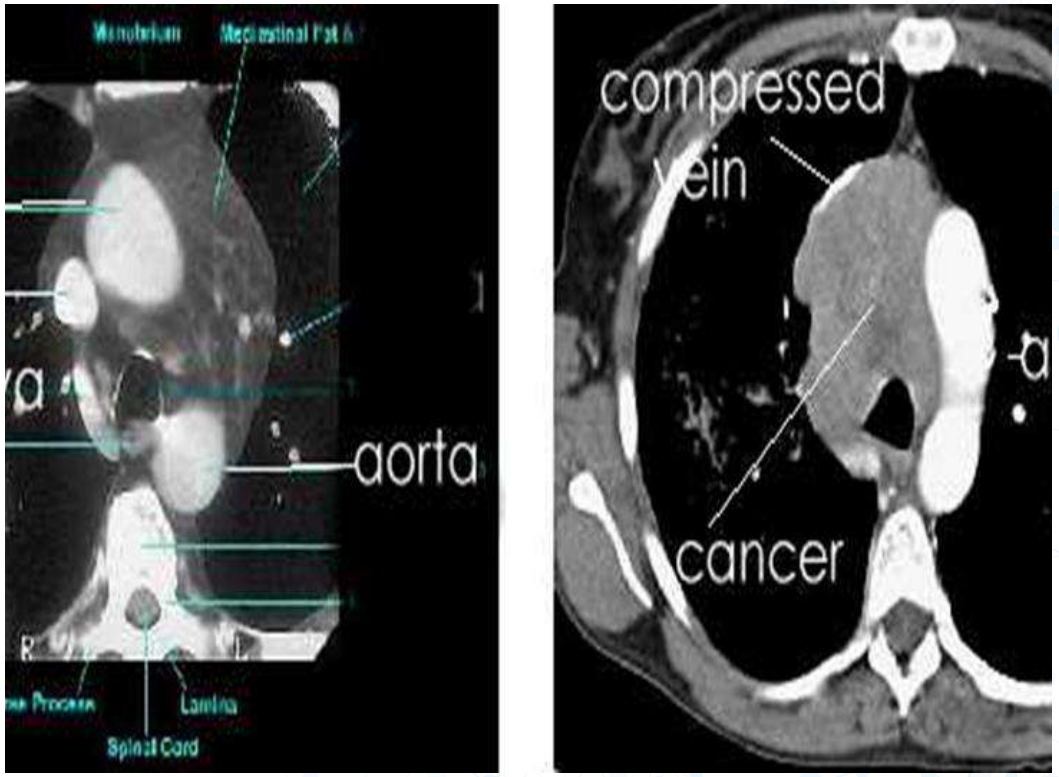


Figure 17. Imaging of SVCO through CT Scan

Management of SVCO



Supportive and Medical Care

- Elevation of the head
- Consider steroids for steroid-sensitive tumours or significant airway/ cerebral oedema. Obtain biopsy before starting corticosteroids whenever possible.
- Supplemental oxygen.



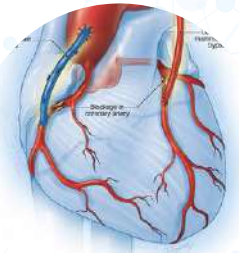
Radiotherapy

- Effective for symptoms relief.
- 75% of patients with malignancy-associated SVCO experience improvement within 3–7 days.



Chemotherapy

- Complete relief of SVCO in 80% NHL/SCLC, 40% in NSCLC.
- Effectively for chemotherapy sensitive cancers.

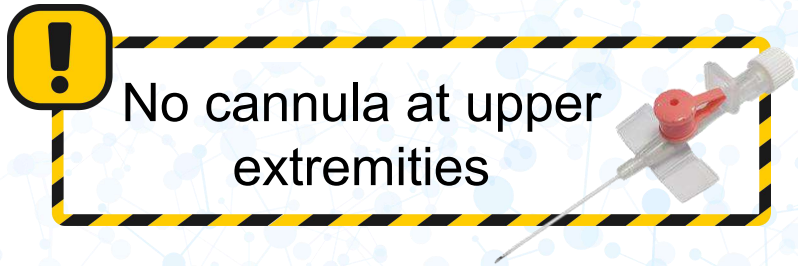


Surgery

- Bypass or resect tumours to decompress the SVC.
- Relieves SVCO - 95% of patients with lung cancer.
- For severe symptomatic malignant SVCO, endovascular stenting can provide rapid relief when feasible; chemotherapy or radiotherapy depends on tumour histology and clinical scenario.

Nursing Intervention SVCO

- Assess cardiac function
- Give oxygen if hypoxic or in respiratory distress; titrate to target oxygen saturation according to local protocol
- Position: Fowlers, Semi fowlers for chest expansion
- Restrict activity (CRIB): To reduce oxygen consumption
- Assist Activity Daily Living (ADL)
- Monitor vital sign/ mental status
- Administer medication as prescribed– Steroid, Anticoagulant
- Psychology support
- Monitor intake output



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THANK YOU