Contents

General principles

Preoperative

= increasing cause of mortality and morbidity

CLINICALLY

- assess in terms of physical state
- think about nutrition (either IV or enteral)

- GENERAL: cachexia, weight loss, immunosuppression, weakness
- LOCAL: SVC Obstruction, spinal cord compression,
- CVS: arrhythmias, radiation induced myocardial dysfunction, CCF, anthracycline induced cardiomyopathy, PVD, pericardial disease
- RESP: pulmonary dysfunction
- GU: renal failure (pre, intra and post)
- HAEM: neutropenia, immuosuppression, hospital acquired infections, anaemia, pancytopaenia, VTE
- GI: severe N+V (multifactorial), acute abdomen, fistulae, bleeding
- METABOLIC/endocrine: hypercalcaemia, hyponatraemia (SIADH), tumour lysis syndrome, DM, diabetes insipidus, hypopituitism, thyroid disorders, adrenal function
- CNS: pain, psychological distress and depression

INVESTIGATIONS

- fix electrolytes
- post chemo echo if agents cardiotoxic
- standard otherwise

MANAGEMENT

1. Surgery
2. Chemo (see next)
3. Radiation (quantify damage to normal tissues, neurological damage, lung fibrosis, radiation nephropathy, pericarditis and effusion, liver acute enlargement -> cirrhosis)

- IVF for dehydration
- replenish blood volume

Intraoperative

- no N2O -> increased synergy between chemotherapeutics and N2O (unpredictable)
- TIVA:
  - propofol safe and good anti-emetic
  - in vitro evidence volatile suppresses immune system
- opioid sparing anaesthetic:
  - multimodal esp NSAIDs
  - regional anaesthesia - low grade evidence. Also benefit of avoiding SNS drive
- NSAIDs - can reverse pro-oncogenic effects of opioids
- routine monitoring
- avoid ↑BSL
- hypothermia cares
- may require advanced airway securement (AFOI or awake tracheostomy)
- blood product transfusion only as indicated
- all IV hypnotics are safe and may provide some benefit interms of immunomodulation

Postoperative

- re-establish feeding early -> decreases complications
- adjust opioids to take into account of normal doses

Chemotherapy

Priorities

1. Assessment of disease process
2. Assessment of treatment

Common side effects

GENERAL

- nausea
- alopecia
- myelosuppression
- fatigue
- mucositis

SYSTEM BASED

RESP: pulmonary fibrosis
CVS: cardiomyopathy
GU: renal impairment
NEURO: peripheral neuropathy

Side effect management

- ondansetron
- dexamethasone

Chemotheraputics

1. Anthracyclines
2. Alkylating agents
3. Antibodies
4. Antimetabolites
5. Tumour antibiotics
6. Topoisomerase inhibitors
7. Tyrosine kinase inhibitors
8. Mitotic inhibitors
9. Mitotic stabilizers
10. Hormone agents

ANTHRACYCLINES (ie. doxorubicin, danorubicin)

- mechanism = DNA cleavage and intercalation with double strained DNA
- cardiomyopathy
- even in patients with normal heart function post chemo -> they are more susceptible to the myocardial depressant effects of anaesthetics
- risk factors of development of cardiomyopathy: very old, very young, mediastinal radiation, pre-existing heart disease, concurrent cyclophosphamide or mitomycin use.

ALKYLATING AGENTS (ie. cyclophosphamide, cisplatin)

- mechanism = alkylate DNA and interfere with mitosis
- may produce tumour lysis syndrome

- can cause multi-system problems: act like SIADH, interstitial pneumonitis, haemorrhage cystitis

ANTIMETABOLITES (ie. methothrexate, 5-FU)
- mechanism = they screw up DNA code during synthesis
- generally cause: myelosuppression and GI side effects

ANTIBODIES (ie. 'Herceptin' = trastuzumab, rituximab)
- Herceptin – used in Breast Cancer (can cause cardiomyopathy)
- Rituximab – used in non-hodgkin's lymphoma and RA (flu like side effects)

TUMOUR ANTIBIOTICS (ie. bleomycin)
- causes a pneumonitis (hypersensitivity reaction)
- mechanism: ? unknown ? superoxide anion generation + chemotaxis of neutrophils, ? superoxia -> overwhelming anti-oxidant ability of lung
- risk factors = increasing age, renal impairement, lung radiation, IV administration, O2 exposure, increased total cumulative dose
- can be treated with high dose steroids
- keep FiO2 < 0.3 if possible

TOPOISOMERASE INHIBITORS (ie. etoposide)
- mechanism = stop unwinding of DNA during replication
- side effects: myelosuppression, anorexia and nausea

TYROSINE KINASE INHIBITORS
- recent developments
- mechanism = switch of intracellular signaling
- side effects: hypertension, skin rash, GIT symptoms, myelosuppression, liver dysfunction

MITOTIC INHIBITORS (ie. vincristine)
- mechanism = inhibit the formation of microtubule complexes within cytoplasm -> arrest cell growth
- side effects: peripheral neuropathy, constipation

MITOTIC STABILISERS (ie. docetaxel, paclitaxel)
- mechanism = stabilize the microtubular assembly once formed and prevents mitosis proceeding
- side effects: myelosuppression, peripheral neuropathy

HORMONES (ie. corticosteroids, tamoxifen)
- mechanism = modify the hormonal environment
- corticosteroids -> tumour lysis
- tamoxifen -> breast ca
- LHRN analogues -> prostate cancer
- side effects: vary