Haematology:
The take home messages

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The whole of haematology in 20 slides

@bloodworkapp

Never Miss

Tumour lysis syndrome: Hyperphos, hypocalc, Oliguria

Prevent with allopurinol, rasburicase and generous IV fluids

Cord Compression: Back pain, known malignancy (or suspected), neurology

Suspect with low index of suspicion

Steroids (unless a new diagnosis), image, radiotherapy, neurosurgery

Thrombotic Thrombocytopenic Purpura

Anaemia, jaundice, thrombocytopenia, fever, renal fx, Neurological

IV lines and straight to pheresis centre for PLEX +/- chemotherapy

Acute transfusion reaction

Unwell on a transfusion -> STOP it, ?adrenaline needed

check BAG, BAND, BLOOD; involve HDU, Hydrate, inform lab, send samples,

Never Miss: the dangerous thrombocytopenias

Always ask for a blood film and check medication history

Are they bleeding? Or may they have a clot?

Could they have a bone marrow failure or immunological syndrome?

- Pregnancy-Associated
  - Liver function? Proteinuria? Hypertension?
  - Think HELLP / Eclampsia spectrum
- Heparin Induced Thrombocytopenia & Thrombosis (HITT)
- Thrombotic Thrombocytopenic Purpura (TTP)
  - Anaemia, jaundice, thrombocytopenia, fever, renal fx, Neurological
  - IV lines and straight to pheresis centre for PLEX +/- chemotherapy

Take Home: Microcytic Anaemia

Microcytic is IDA or thal trait

IDA is Diet or bleeding - Gynae or Gut?

Investigate the cause

Don't Transfuse if you don't have to

Ferritin is a guide not an absolute: CONTEXT!

SO WHAT IS THALASSEMIA?

ALPHA OR BETA? : EXCESS OF THE 'OTHER' GLOBULIN IS PATHOGENIC

TRAIT/MINOR, INTERMEDIA OR MAJOR: NUMBER OF COPIES LOST

HAEMOLYSIS & INEFFECTIVE & EXTRAMEDULLARY HAEMATOPOIESIS

SKELETAL DEFORMITY & ENDOCRINOPATHY

IRON OVERLOAD SYNDROME & VIRAL TRANSMISSION RISK

TRANSFUSION WITH AGGRESSIVE CHELATION IS MAINSTAY OF THERAPY

BONE MARROW TRANSPLANATION IS CURATIVE

TAKE HOME: Macrocytic Anaemia

Helpful Tests: B12, Folate, Retics, DAT, LDH, Haptoglobs

Acute: Haemolysis: Retics UP

Subacute: Folic acid ?pregnancy & Chronic: B12 ? P.A.: Retics DOWN

Alcohol? Drugs? Thyroid? Liver? Myelodysplastic syndrome?

rapid transfusion KILLS in pernicious anaemia

Replace B12 first THEN Folic Acid

WATCH for Hypokalemia; Retics rise at DAY 5

TAKE HOME: The Sickle Cell Syndrome

A lifelong, life-limiting, multisystemic disease

Treat without prejudice but **WITH** individual care plans

adequate **ANALGESIA**, 20 mins, 1 hr targets

YES! Bloods, Oxygen, Fluids (oral or IV), Spirometry

MAYBE! CXR, ANTIBIOTICS, TRANSFUSION

LONG-TERM:Hydroxyurea, Transfusion, Chelation &

Screening; ?Bone marrow Transplantation?

Take Home: The Sickle Cell Crises

Pain: Limb or Axial including Skull or Chest

Chest: Pain, Hypoxia, Pulmonary Infiltrates

Oxygen, Analgesia, Antibiotics & Spirometry

Ventilatory Support, Exchange Transfusion

Aplastic: Parvovirus (or drug); Transfuse & Support!

Sequestration: Liver (or spleen); Transfuse & Support!

Septic: Recognise Early, Treat, Re-assess

Haematological Malignancy

everyone needs:

a biopsy - Lymph node, bone marrow

FBC: Marrow failure often a complication

Chemistry: Tumor lysis, Calcium, Liver infiltration, Fitness for treatment

HIV and Hepatitis status check

Autoimmune and thyroid

Staging / Prognostication:

imaging:

Lymphoma: CT and 'functional' PET-CT

Myeloma: Skeletal survey, MRI spine

Molecular:

Immunophenotyping defines cell type

Cytogenetics determines prognosis

TAKE HOME: LYMPHOMAS

NON-HODGKIN: COMMON

USUALLY B CELL, HIGH GRADE OR LOW GRADE

- HIGH GRADE = **DLBCL**
- LOW GRADE = **FOLLICULAR LYMPHOMA**
- LOW GRADE + IGM PARAPROTEIN = **LPCLYMPHOMA**

SOMETIMES **T CELL** (10%) = **RASHES** AND **BAD NEWS**

BURKITT'S - RARE SUPER-HIGHGRADE, EBV

3 TYPES: **SPORADIC** (ELDERLY); **ENDEMIC** (AFRICA, JAW, KIDS); **HIV / IMMUNOSUPPRESSION ASSOCIATED**

TARGETED ANTIBODY: **RITUXIMAB** (CD20) FOR B CELL

HODGKIN: RARE (NLPHL RARER)

TEENS AND TWENTIES PLUS ELDERLY

REED STERNBERG CELLS; 30% EBV+

HISTOLOGICAL SUBTYPES X4 (USU NS OR MC)

TARGETED ANTIBODY: **BRENTUXIMAB** (CD30)

STAGED AND TREATED THE SAME!

BIOPSY, CT OR PET-CT: ANNE ARBOR STAGE

CHEMOTHERAPY MAINSTAY

RADIOTHERAPY FOR LOCALISED

BM TRANSPLANT FOR RELAPSE

TAKE HOME: Acute Leukaemias (AML/ALL)

Behave VERY like each other (contrast CHRONIC)

Present similarly: Bone marrow failure, infections/bleeding, leucostasis

Treated similarly: Chemotherapy +/- transplant; don't forget the CNS

Cytogenetics are all important prognostically:

t(15;17) GOOD, monosomy 3,5,7 BAD

SUSPECT: Bone marrow failure with 'Systemic Symptoms', +/- leucocytosis

TREAT: DISEASE Rx: CHEMOTHERAPY, Allogeneic Transplant

ADJUVENT Rx: Analgesia, Bisphosphonates, Anticoags, Antibios, RT

BEWARE!: Leucostasis, Tumor Lysis, Coagulopathy, Sepsis

TAKE HOME: Chronic Leukaemias

Behave NOTHING like each other (contrast ACUTE)

CLL: The commonest leukaemia!

All about too many mature **lymphocytes**

Often requires **NO treatment**

TREAT when the **symptoms** get bad:

Lumps, Cytopenias, 'B symptoms'

No magic treatment

Chemo + anti B cell (CD20) **Rituximab**

Novel agents: Ibrutinib, Idelalisib

CML: extremely rare!

All about too many mature **granulocytes**

Remember: **t(9;22) and BCR-ABL**

ALWAYS requires treatment

Has a **magic treatment** (**Imatinib** & sons)

Can TURN to **acute leuk** (beware new cytopenias)

And don't forget the MPDs: PCV, ET, MF

JAK2 (etc): Thrombosis & AML risk

Cytoreduce (venesect or HU) & an ASPIRIN!

TAKE HOME: MYELOMA

MULTISYSTEMIC MALIGNANCY: **CRAB** CRITERIA (+ INFECTIONS)

CALCIUM, RENAL, ANAEMIA, BONE (+INFECTION, THROMBUS, AMYLOID)

SUSPECT: ANAEMIA, BONE PAIN, FATIGUE, HIGH GLOBULINS, HYPERCALCEMIA

TREAT:

DISEASE Rx: CHEMO/RT, **NOVEL AGENTS**: VELCADE & IMIDS, AUTO BMT

ADJUVENT Rx: ANALGESIA, BISPHOSPHONATES, ANTICOAGS, ANTIBIOS

BEWARE!: CORD COMPRESSION, PATHOLOGICAL #, RENAL FAILURE, INFECTION

TAKE HOME: Bone Marrow Failure Syndromes

EXCLUDE **CONGENITAL** AND SECONDARY CAUSES: **NUTRITIONAL/VIRAL/TOXIN/RADIATION**

SUPPORT WITH **BLOOD** PRODUCTS AND **ANTIMICROBIAL** PROPHYLAXIS

Aplastic Anaemia:

SOMETIMES **CURABLE** DISEASE OF MID-AGE

AUTOIMMUNE MECHANISM

DOES NOT EVOLVE TO AML

TREATMENT:

IMMUNOSUPPRESSION (ATG/CSA) + BMT

Myelodysplastic syndrome:

GENERALLY **INCURABLE** DISEASE OF THE ELDERLY

NEOPLASTIC MECHANISM

FREQUENTLY EVOLVES TO **AML**

TREATMENT:

HYPOMETHYLATORS,

GROWTH FACTORS, CHEMO + **BMT**

SOMETIMES IMMUNOSUPPRESSION OR LENALIDOMIDE

Take Home: Iron Overload

Hyperferritinemia = acute phase, liver ... or iron overload

Gut is the main regulator through hepcidin and HFE

Causes: Transfusional, Ineffective Erythropoiesis or H.H.

Complications: Liver, Endocrine, Cardiac, Joint, Skin

Hereditary (HH): C282Y or H63D mutated HFE gene (& rarer others)

Treatment: venesect if H.H.

Iron Chelation if Ineffective Epoiesis/Transfusion

Take Home: Bleeding and Bruising

Platelets abnormal: Mucocutaneous bleeds: Bruises and purpura
Clotting proteins abnormal: Joint bleeds if congenital, EVERYWHERE if acquired (muscle, retroperitoneal, GI, Mucosal, cranial)

APTT abnormal?: It's heparin, Lupus or HAEMOPHILIA (acquired/congenital)
PT abnormal?: It's warfarin, Nutrition, liver or a RARE haemophilia
BOTH abnormal?: It's DIC, Liver... check fibrinogen / FDPs

BLEEDING HISTORY: structured bleeding assessment tool is better than labs
Significant Haemorrhage?: Consider **TRANEXAMIC ACID**

Take Home: Oral Anticoagulation

Warfarin still has its place:

- **Reversible**, Well tolerated, useful with **renal** impairment
- Always use for **valvular heart disease** (especially prosthetics)
- **Takes 3 days to work** (at least), but **reversible** (Vitamin K and PCC)

Novel agents are preferable for new patients or clinic non-attenders:

- **Rivaroxaban** most commonly (+**apixiban** if frail), also edoxaban
- **Dabigatran** sometimes used (but GI bleeds and MIs?)
- Act immediately but **irreversible** (for now... watch this space)

CHADS2Vasc and HASBLED scores help risk stratify atrial fibrillation

Take Home: Parenteral Anticoagulation

Unfractionated heparin rarely used

- except for **cardiology** and **renal impairment**
- **APTT monitoring** required because of **unpredictable pharmacokinetics**
- Risk of **Heparin-induced Thrombocytopenia**
- Can be reversed with **protamine** (or just switched off - short half-life)

Low molecular weight heparin (and 'ultra-low' Fondaparinux for ACS)

- Many brands, pretty much the same (dalte/enoxa/tinza-**parin**)
- Predictably RENALLY excreted hence easy dosing (weight-based)
- But **unlike UFH** - it's **irreversible** (mainly) and has a 10-20 hour half-life

Take Home: Blood Products

Plasma (FFP):

Corrects deficiencies of all clotting factors
No good for warfarin-induced deficiencies
Used in massive transfusion (>6 units)
Generally NOT for DIC!

Cryoprecipitate:

Fibrinogen concentrate
Good for dys/hypofibrin
Occasionally DIC and obstetric bleeds

Prothrombin Complex Concentrates:

LIFE THREATENING warfarin bleeds

Clotting factors

Once were 'super-concentrates'
Now RECOMBINANTS (less viral risk)
Factors VIIa, VIII, IX and VWF
Generally for haemophilia
Activated version: FEIBA
For haemophilia with inhibitors

Take Home: Proper Transfusion

Red Blood Cells:

Only for emergencies and congenital anaemias: Avoid if you can

Patient Blood management

Conservative vs Liberal Hb thresholds

Why use 2 when 1 will do?

Optimise iron (IV) +/- Epo

Fewer blood tests for patients

Salvage blood intraop

Treat anaemia cause before Hb too low

Platelets:

A scarce and precious resource: for emergencies only

Seek the cause of a new thrombocytopenia: look at patient, drug chart & blood film.

Excess use leads to **refractoriness**

1 unit usually adequate

Most invasive procedures require platelets >30-50; only neurosurgery >100

Remember to replace during massive transfusion

Good 'last minute' runthrough of clinical cases: Haematology: Clinical Cases Uncovered https://appsto.re/gb/4LUvz.

Want to be a haematologist? Follow these...

@bloodworkapp

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