

HAEMATOLOGY FOR CLINICAL FINALS

THE TAKE HOME MESSAGES

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The whole of haematology in under 20 slides



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

STERIODS (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, GYNAECOLOGY 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 RBCPOIESIS, EXTRAMEDULLARY HAEMATOPOIESIS

SKELETAL DEFORMITY & ENDOCRINOPATHY

IRON OVERLOAD SYNDROME & VIRAL TRANSMISSION RISK

TRANSFUSION WITH AGGRESSIVE CHELATION IS MAINSTAY OF THERAPY

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? THYROID? LIVER?

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

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)

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: CHRONIC LEUKAEMIAS

ARE NOTHING LIKE EACH OTHER (CONTRAST ACUTE)

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 INTO ANY ACUTE LEUKAEMIA (BEWARE NEW CYTOPENIAS)

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

TAKE HOME: ACUTE LEUKAEMIAS (AML/ALL)

ARE 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

ADJUVANT Rx: ANALGESIA, BISPHOSPHONATES, ANTICOAGS, ANTIBIOS

BEWARE!: LEUCOSTASIS, TUMOR LYSIS, COAGULOPATHY

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

MYELOYDYSPLASTIC SYNDROME:

GENERALLY INCURABLE DISEASE OF THE ELDERLY

NEOPLASTIC / MALIGNANT MECHANISM

FREQUENTLY EVOLVES TO AML

TREATMENT:

HYPOMETHYLATORS, GROWTH FACTORS, CHEMO + BMT

SOMETIMES IMMUNOSUPPRESSION OR LENALIDOMIDE

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

ADJUVANT Rx: ANALGESIA, BISPHOSPHONATES, ANTICOAGS, ANTIBIOS

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

TAKE HOME: IRON OVERLOAD

HYPERFERRITINEMIA = ACUTE PHASE, LIVER ... OR IRON OVERLOAD

GUT IS THE MAIN REGULATOR THROUGH HEPCIDIN AND HFE

TRANSFUSIONAL, INEFFECTIVE ERYTHROPOIESIS OR H.H.

LIVER, ENDOCRINE, CARDIAC, JOINT, SKIN

C282Y OR H63D MUTATED HFE GENE

VENESECT IF H.H.

IRON CHELATION IF INEFFECTIVE EPOIESIS/TRANSFUSION

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)

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

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