NHS CATS
Children's Acute Transport Service

Clinical Guidelines

Oncology Emergencies

1. Management of Tumour Lysis Syndrome

2. Management of Hyperleukocytosis

3. Anterior Mediastinal Mass

Document Control Information

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Document Version	Version 1	**Replaces Version**	N/A
First Introduced	2022	**Review Schedule**	2 Yearly
Active Date	2026	**Next Review**	2028
Applicable to	All CATS employees		

1. Management of Tumour Lysis Syndrome (TLS)

Introduction

- Life threatening condition from rapid cell lysis in haematological malignancies /lymphoproliferative (LPD) conditions.
- Causes hyperuricaemia, hyperphosphataemia, hyperkalaemia and hypocalcaemia, which may lead to acute renal failure.
- Usually occurs following initiation of treatment.
- Can occur prior to chemotherapy- for instance a child presenting with stridor (from undiagnosed mediastinal mass) may be treated as croup and receive dexamethasone.
- Aim of management is prevention- high fluid intake and allopurinol or rasburicase.
- TLS is still possible even in children that have had rasburicase.
- All children either at risk of or with established TLS should be discussed urgently with the haematology/oncology consultant on call and referred to CATS if they are likely to require PICU admission.

Recognition of Risk

High Risk	Intermediate Risk	Low Risk
Leukaemia WCC>100	Leukaemia WCC 20-100	Leukaemia WCC<20
Non Hodgkins Lymphoma (B/T cell)	Absence of large tumour bulk or renal impairment	Absence of large tumour bulk/renal impairment
Large tumour bulk		Non bulky NHL, LPD or PTLD, normal renal function
Post-transplant lymphoproliferative disease (PTLD)		
Evidence of renal impairment/involvement		

Prevention

- Discuss management plan with the haematology/oncology consultant on call.
- Biochemical abnormalities may not be apparent for 4-6 hours after starting chemotherapy.
- Please refer to Flowsheets 1&2 at end of guideline for full details
- Ideally document G6PD status prior to starting rasburicase (can cause haemolysis)

Treatment of established TLS

Call haematology/oncology consultant and CATS early - patient may require haemofiltration/dialysis and urgent PICU admission.

- Hyperhydration and rasburicase (allopurinol if not available) as per Flowsheet 2
- Full monitoring of O_2 sats, heart rate, BP, urinary output, and neurological status
- If evidence of cardiorespiratory compromise may need intubation
- ECG monitoring – look for evidence of peaked T waves, widened QRS (hyperkalaemia) and prolonged QT (hypocalcaemia)
- 4-6 hourly TLS bloods (Na, K, Ca, phosphate, urate, urea and creatinine)
- Regular (4 hourly) clinical review for evidence of hyperkalaemia or hypocalcaemia
- Hyperkalaemia can cause weakness and paralysis
- Hypocalcaemia can cause vomiting, seizures, spasms, cramps, altered mental state and tetany.

Stabilisation

Airway

- Intubate if airway needs protection due to deteriorating neurology, or if respiratory distress warrants.
- Discuss with CATS consultant prior to intubation if there is a suspicion of a mediastinal mass.
- Prepare for cardiorespiratory instability post intubation.

Breathing

- Use a cuffed ETT as pulmonary oedema will result in “stiff lungs” and may need relatively high peak inspiratory pressures.
- If intubated for pulmonary oedema use a high PEEP strategy
- CXR to confirm ETT position.

Circulation

- Cardiovascular compromise may occur due to *vascular compression* compromising cardiac output.
- Ensure the child is well filled.

- *Avoid potassium containing intravenous fluids. If severe hypokalaemia, discuss with haematology/oncology consultant.*
- Try repositioning them e.g. sitting up or positioning on their side. This will reduce the pressure of the mass off the great vessels.

In the event of arrhythmias

- Start treatment of hyperkalaemia without delay - see CATS hyperkalaemia guideline.

Transport

- In the setting of a mediastinal mass causing TLS, transport in a sitting position or prone may be necessary- see which position ventilation/haemodynamics are optimal.
- Prepare for CVS compromise due to electrolyte disturbance.
- Transport with defib pads attached.
- If transport is prolonged recheck potassium en-route.

Prepare

- Arrest doses of adrenaline
- Calcium gluconate
- Sodium bicarbonate
- Salbutamol
- Insulin dextrose

2. Management of Hyperleukocytosis

Introduction

High count leukaemia (leucocytosis) is generally defined as:

- WCC > $100x10^9$
- In monocytic AML (FAB type M5), high count is defined as > 50 – cells are large and aggregate and cause coagulopathy more readily.

These patients are at risk of death/serious complications as a result of:

- Hyperviscosity syndrome
- Coagulopathy
- Tumour Lysis Syndrome (TLS)

Management

Inform haematology consultant, local paediatric consultant, and plan prompt transfer from local hospital to GOSH.

If very high count (suspected AML) e.g. WCC > 50, especially AML M4/5 and APML:

- Liaise with haematology consultant and lab haematology SpR urgently.
- Haematology consultant to consider urgent transfer to PICU for exchange transfusion or leukapheresis (not recommended for APML)
- If consultant recommends urgent transfer to PICU, ensure prompt referral to CATS.
- Ensure local paediatric consultant is aware.

Manage TLS as per TLS guideline.

Airway and breathing

May need to be protected after cytotoxic therapy is started because of deterioration in respiratory status and neurology.

Use a cuffed ET tube as respiratory distress can occur from:

- Leukostasis
- Fluid overload (from hyperhydration)
- Pulmonary haemorrhage
- Infection

Prior to giving furosemide discuss with haematology-oncology consultant as it may worsen hyperviscosity.

Circulation

Avoid red cell transfusion- PRBCs are viscous with haematocrit 70%.

- PRBC transfusion may exacerbate leukostasis and cerebral infarction.
- Only transfuse after discussion with haematology-oncology consultant.
- If transfusing - administer no more than 5mls/kg over 4 hours.
- If a second transfusion is required, re-discuss with haematology/oncology consultant.
- Aim platelet count > 50 in the presence of active bleeding or coagulopathy.
- Coagulopathy - more common in AML than ALL but can occur in any leukaemia.
- *If active bleeding* - FFP (15ml/kg) and cryoprecipitate (5ml/kg) should be given according to the clotting parameters – discuss with consultant haematologist.
- If evidence of DIC, even if not bleeding, give FFP to correct the PT and APTT, and give cryoprecipitate to maintain the fibrinogen >1g/l.
- If not bleeding – with prolonged PT or APTT (>3 seconds above normal range) – discuss with consultant haematologist prior to taking any action.
- Recheck coagulation post transfusion.

Disability

Clinical signs of CNS leukostasis

- Abnormal neurology
- Raised intracranial pressure (high BP, low HR, irregular respiratory pattern, pupillary changes, change in GCS)
- May indicate infarction or haemorrhage.

Discuss urgently with local anaesthetic team (airway and neuroprotection).

Discuss with CATS and haematology consultants.

The patient will need urgent neuroimaging and the images should be image – linked to specialist centre on “blue light transfer”.

Transport

- May need high pressures to oxygenate.
- Use high PEEP in setting of pulmonary haemorrhage/oedema.
- If neurological concerns- neuroprotective measures

3. Management of Anterior Mediastinal Mass

These children are at very high risk of fatal decompensation.

Fatal complications can occur on presentation and/or during sedation/general anaesthesia from

- Airway compromise
- Loss of venous return
- Loss of outflow from heart

Presentation

Can be very variable:

- May be an incidental finding - asymptomatic.
- May present with increased work of breathing, noisy breathing, or “wheeze”.
- Positional dyspnoea, unable to lie flat.
- Cough, chest pain
- Weight loss, generally unwell, night sweats
- Can present in cardiac arrest.

Management

- Inform local paediatric consultant. Liaise with oncology consultant and urgent referral to CATS.
- Will need urgent transfer from local hospital to specialist centre.
- Investigations – should be kept to a minimum to avoid distressing the patient unnecessarily – this can worsen airway compromise.
- Aim to do a CXR (caution regarding position – these patients may not be able to lie flat), FBC and blood film, biochemistry, and coagulation – if possible.

Airway

The major risk is loss of airway and/or loss of cardiac output from vascular compression – check for:

- Stridor or wheeze
- Positional changes – may not like lying flat or prefer one side or even prone.
- Signs of SVC obstruction – facial or neck swelling or enlarged neck veins.
- Signs of pleural/pericardial effusion or tamponade- raised HR, narrow “pulse pressure” on non-invasive BP.

- Avoid sedation and general anaesthesia at local hospital – can lead to acute loss of airway/cardiac output.
- If the patient has airway compromise discuss management with CATS.
- These patients should only be intubated for life threatening airway compromise.
- Ensure senior anaesthetic and ENT support is available prior to intubation.
- If intubation is required use a reinforced and cuffed tube where possible.
- Intubate the patient in the position in which their breathing is most comfortable – often sitting up.

Breathing

- Will require high peak pressures and need to be kept sitting post intubation.
- If unable to ventilate consider lateral or prone positioning

Circulation

- Be prepared for significant cardiovascular compromise peri and post intubation.
- Have fluid boluses prepared as filling may help.
- If clinical evidence of Superior Vena Cava obstruction, avoid large volume fluid boluses or hyperhydration in upper limbs. (Risk of cerebral oedema). Give large volumes via IV access in lower limb instead.
- Have peripheral adrenaline infusion running prior to induction.

Transport considerations

- Ideally transport self-ventilating in the position of maximum comfort.
- Have multiple fluid boluses prepared and keep the patient well filled.
- Check electrolytes (especially K) immediately prior to and if possible, during transport as these children are at risk of TLS.
- Be prepared to treat hyperkalaemia en-route.

Prior to starting treatment/chemotherapy

TLS Flowchart 1: Prevention of Tumour Lysis Syndrome (TLS) – prior to starting treatment

At presentation – can be initiated at POSCUs

Low risk for TLS AND

Normal U&E & urine output
(eg WCC <20x10^9/L)

i) no need to hyperhydrate

ii) maintenance (oral with or without intravenous fluids combined total)

iii) Start Allopurinol 100mg/m^2/dose 8 hourly by mouth

iv) Prior to start of treatment, only need once daily bloods (TLS* & FBC).

*TLS bloods = urate, U&E, K, Ca, Phos

12 to 24 hours prior to starting treatment:

Start hyperhydration 0.45% saline/2.5% glucose
2.5 - 3 L/m^2/day, can be ↑ up to 4L/m^2/day if there is no evidence of fluid overload.

Ensure urine output >3ml/kg/hour.
Frusemide PRN.

Intermediate risk AND

Normal U&E & urine o/p
(eg WCC 20-100x10^9/L)

i) Consider hyperhydration 0.45% saline/2.5% glucose
2.5 - 3 L/m^2/day. Discuss with PTC

ii) Allopurinol (oral) 100mg/m^2/dose tds

iii) Prior to start of treatment, only need 12 to 24 hourly bloods (TLS* & FBC)

*TLS bloods = urate, U&E, K, Ca, Phos

12 to 24 hours prior to starting treatment:

If not already started, then start hyperhydration 2.5 - 3 L/m^2/day, can be ↑ up to 4L/m^2/day if there is no evidence of fluid overload.

Ensure urine output >3ml/kg/hour.
Frusemide PRN.

High risk for TLS

- High cell count leukaemia (>100 x10^9/L)
- Burkitt's type lymphoma (BNHL)
- Large tumour bulk
- Bulky T cell lymphoma (mediastinal mass)
- Bulky LPD/PTLD
- Evidence of renal infiltration with tumour
- Renal impairment with abnormal U&E
- Consider insertion of permacath during first GA

On presentation start following:

i) hyperhydration 0.45% saline/2.5% glucose 3 L/m^2/day, can be ↑ to 4L/m^2/day if there is no evidence of fluid overload. Caution with hyperhydration in children with very low Hb and high WCC count. (Risk of haemodilution and dropping Hb further). Discuss with PTC for plan of hyperhydration.

ii) Ensure urine output >3ml/kg/hour. Frusemide PRN. For patients at risk of leukostasis, discuss with PTC before giving furosemide. (Dehydration may worsen leukostasis)

iii) Start IV rasburicase** (ideally document G6PD status in patients who are at risk of G6PD deficiency before starting rasburicase)
200 microgram/kg/dose once daily.

iv) If rasburicase is unavailable at POSCU, start allopurinol, then switch to rasburicase on arrival to PTC.

iv) If TLS bloods normal: repeat 8 hourly with FBC

***Rasburicase can trigger haemolysis in G6PD deficiency.*

AFTER starting treatment/chemotherapy

TLS Flowchart 2: Prevention and treatment of Tumour Lysis Syndrome (TLS) – after starting treatment