UHL EMCHC, PICU & Children's Hospital

UHL guidelines on management of Kawasaki Disease

Staff relevant to:	**Clinical staff working within the UHL Children's Hospital and East Midlands Congenital Heart Centre.**
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Contents

1. Introduction and Who Guideline applies to

Kawasaki Disease (KD) is an acute self-limiting inflammatory disorder, affecting predominantly medium sized arteries, particularly coronaries; causing coronary artery aneurysms (CAA) in 15-25% of untreated patients; while 2-3% of untreated cases die as a result of coronary vasculitis.

KD is the commonest cause of acquired heart disease in children in developed countries and potentially important cause of long term cardiac disease in adult life with risk of thrombosis and/or stenosis of the coronary artery, which may result in coronary thrombosis and myocardial ischaemia or infarction in patients with KD related aneurysms (NHS patient safety alert stage1, 11th May 2016).

2. Epidemiology

Incidence in UK - 8.1/10,000 children; younger than 5 years, peak incidence at 18-24 months
Less common in patients aged less than 3 months or more than 5 years, but are at increased risk for coronary artery aneurysm (CAA) formation.

3. Clinical features and diagnosis

KD is a purely clinical diagnosis with combined criteria and absence of gold standard for diagnosis.
There is no diagnostic test for KD, thus the diagnosis rests on combination of clinical criteria and laboratory findings of raised inflammatory markers.
Fever of more than 38°C lasting at least five days without any other explanation is a mandatory criterion.

Complete KD

Fever persisting for at least 5 days without any other explanation PLUS 4 out of 5 of the following criteria:

1. Bilateral non-suppurative conjunctivitis
2. Cervical lymphadenopathy, often more than 1.5 cm
3. Polymorphous rash- nonspecific maculopapular eruption, usually extensive with involvement of trunk and extremities, no vesicles or crusts
4. Changes in lips or oral mucosa (red cracked lips; strawberry tongue; or diffuse erythema of oral mucosa

5. Changes in extremities:
   Initial stage: erythema and edema of the palms and soles;
   Convalescent stage: peeling of skin from finger and toe tips in week 2 or 3.

Incomplete KD (Atypical)

10 - 40% of cases have some but not all the above features. Diagnosis of these "**Incomplete KD**'' depends on high level of suspicion in children presenting with some of the KD features and evidence of systemic inflammation (such as- elevated CRP, ESR, and or leucocytosis).

One should start presumptive treatment as KD, particularly if febrile exanthematous illness persists longer than 4-5 days with no other explanation. KD may be diagnosed with fewer than 4 of the above criterions if coronary abnormalities are detected. (Seek an expert advice in such cases).

Refractory Kawasaki disease

20% of the cases have resistance to initial course of IVIG: Persistent or recurrent fever of any magnitude between 36 hours to about 2 weeks after the start of treatment in patients with KD is generally assumed to be the result of failure to abort the disease process.

Patients who have persistent or recurrent fever more than 24 hours after completion of the initial treatment should also be assessed for intercurrent infection, and the diagnosis of KD should be re-evaluated.

However, these patients should be retreated for presumed recrudescence of KD unless there is clear evidence of another explanation for fever, since numerous studies have confirmed an **association between prolonged fever and development of coronary artery abnormalities.** (Seek expert advice from rheumatologist and cardiologist)

• Fever within 36 hours of the start of intravenous IVIG does not warrant re-treatment, because it may represent a reaction to the medication or a slow response to therapy.

Severe disease & high risk of CAA – especially with the following features. (If in doubt, seek expert advice and refer to paediatric cardiologists)

- Already failed IVIG: Refractory KD.
- Severe cases: very young <12 month, those with markers of severe inflammation i.e. persistently elevated CRP despite IVIG, liver dysfunction, hypoalbuminaemia, anaemia.
- Features of Hemophagocytic lymphohistocytosis (HLH)/ Macrophage Activation Syndrome (MAS) or shock.
- Already have on going evolving coronary and/or peripheral aneurysm with ongoing inflammation.
- Kobayashi risk score more than or equal to 5 (please see appendix. table 1)

Additional features of KD

Irritability is characteristic. The **desquamation** may affect the genital area, a characteristic but late feature.

Other features may include: joint pain and swelling, vomiting, abdominal pain and diarrhoea, proteinuria, cough, rhinorrhoea, pneumonitis, CNS involvement with meningism and CSF pleocytosis, fits, meatal inflammation in boys, hydrops of gall bladder, erythema and induration of BCG scars, abnormal LFT and sterile pyuria, SIADH resulting in hyponatremia.

Vascular involvement

The main sites of clinically important vascular involvement are coronary arteries. CAA occur in 15-25% of untreated cases, with additional cardiac features including ECG abnormalities, myocarditis, endocarditis, pericarditis +/- effusion, valvular incompetence, heart failure and myocardial infarction.

Systemic arterial injury (major limb arteries, renal and other visceral vessels) occurs, but it is rarely seen in absence of CAA.

4. Differential diagnosis

• **The absence of any of above criteria and/or the absence of fever should suggest a diagnosis other than KD.**

• Of note, concurrent infections (both viral and bacterial) are common in patients with KD, found in up to 33 percent of children in one study. In any event, diagnosis of an infectious condition does not preclude a concurrent diagnosis of KD.

• **Common differential diagnosis** are viral exanthems (e.g. measles, adenovirus, enterovirus, echovirus, EBV, mycoplasma, CMV, parvovirus), Scarlet fever, Staphylococcal scalded skin syndrome, Stevens-Johnson syndrome, Juvenile idiopathic arthritis (JIA) (systemic onset), Toxic shock syndrome and drug hypersensitivity reactions. (Please see appendix table 2 for differentiating features)

5. Investigations and Laboratory findings

There is no specific diagnostic tests for KD

KD is invariably associated with an inflammatory process with raised ESR/PV, CRP and white cell counts. In the absence of systemic inflammatory response, KD is unlikely.

1. FBC- mild anaemia, neutrophil leucocytosis, thrombocytosis occurs towards the end of second week of illness and not helpful in early stage. Acute thrombocytopenia may be associated with poorer prognosis.
2. CRP raised >10 mg/dl. Raised ESR/PV
3. U&E - hyponatremia may be present.
4. LFT - Liver function may be deranged and some patients present with jaundice and elevation of serum transaminases. Hypoalbuminaemia is common.
5. Urine microscopy and culture - **sterile pyuria.**
6. **Other Investigations**: ASOT, throat swab, blood culture, coagulation screen, viral serology and Autoimmune screen (if indicated)
7. **Lumbar puncture** (if clinically indicated) - pleocytosis of cerebrospinal fluid
8. **ECG** - non- specific ST changes, prolong PR interval if myocarditis present.
9. **Chest X-ray**
10. **Echocardiogram (ECHO)** - should request on diagnosis (**discuss with on-call paediatric cardiology registrar- bleep 2528).**

Subsequent ECG and repeat ECHO (a minimum of 3 ECHO) should be performed in the first 6 weeks of illness and liaise with **Paediatric Cardiology Team (but do not delay therapy prior to obtaining ECHO)**

Early ECHO may reveal evidence of coronary vasculitis and confirm diagnosis but negative ECHO does not exclude the diagnosis.

6. Treatment

All patients need hospital admission and should be reviewed by a consultant before starting a definitive treatment.
Early recognition and treatment with IV Immunoglobulin (IVIG) and aspirin has been shown to reduce the occurrence of CAA in 80% of cases.
Treatment should not be delayed waiting for an ECHO.
Recent clinical trials and meta-analyses have demonstrated that the addition of corticosteroids to IVIG is beneficial for prevention of CAA in severe KD with high risk of IVIG resistance.

1) **IV Immunoglobulin: 2gms/kg single infusion over 12 hours** is the optimal dose. Most effective if given within 10 days of onset. **It may be beneficial in late presenting cases even given after 10 days have elapsed if signs of inflammation persist**. (Fever, elevated CRP)

    a. **(IVIG form (available on INSITE) must be completed and emailed to immunoglobulins.mailbox@uhl-tr.nhs.uk)**

2) **Aspirin: Anti-inflammatory dose of 30-50 mg/kg/D in 4 divided doses** is recommended during the acute phase of the illness. The dose should be reduced to anti-platelet dose of 3-5 mg/kg/D in single dose once fever and inflammation have subsided.

    Low dose Aspirin should be continued for minimum of 6 weeks but should be continued longer if CAA persists. For those on low dose aspirin, we also recommend avoiding the concomitant use of non-steroidal anti-inflammatory drugs (NSAIDs) as these interfere with the anti-platelet effect of low dose aspirin.

3) **Corticosteroids are recommended as part of primary treatment** in severe KD/ cases of high risk of IVIG resistance as described above (seek an expert advice from cardiology and or rheumatology if in doubt). There are two suggested steroids regimen and treating clinician can determine the suitable regimen for an individual patient.

    Methyl-prednisolone 0.8 mg/kg BD IV for 7 days or until CRP normalize; then convert to prednisolone 2mg/kg/day PO and wean off over the next 2-3 weeks.
    (OR)
    Methyl-prednisolone 10-30 mg/kg IV once a day for 3 days followed by prednisolone 2mg/kg/day PO until D7 or until CRP normalize, then wean over next 2-3 weeks.

4) **Consider gastro- protection:** Omeprazole/ Lansoprazole while on high dose aspirin and or steroids.

5) **Fluid balance** should be closely monitored.

6) If no disease defervescence occurs within 48 hours or disease recrudescence within 2 weeks, **second dose of IVIG** is recommended to be given at the same time as

commencing steroids if they have not already been commenced for severe KD. (Seek expert advice to consider if in doubt)

7) **Role of anti-tumor necrosis factor (anti- TNF)** - Infliximab should be considered in patients with IVIG resistant KD with the expert advice from Cardiology and Rheumatology.

7. Management of KD in convalescence phase

If CAA persists, anti- platelet therapy in the form of aspirin should be continued at 3-5 mg/kg/dose OD long term until CAA resolves (Duration as per advice by cardiology team).

Clopidogrel could be considered as an alternative anti-platelet.

In the presence of giant CAA (>8 mm), warfarin is recommended in addition to aspirin.

Heparin should be administered initially for 48 hours and stopped only when warfarin has been commenced and INR stable between 2-3

In small children less than one year with giant aneurysms Warfarin absorption and metabolism is erratic making INR control extremely difficult LMWH is recommended with Clopidogrel or aspirin, target factor Xa is around 1.0[1]
In acute giant aneurysms with Z score above 10; the 2017 AHA guideline recommendation is for triple therapy in the form of dual antiplatelet and warfarin or LMWH[1]

8. Follow up

All patients should be **followed up by general paediatrics** (SE of steroids if received should be vigilant - hypertension, behavioural changes, secondary infections, hyperglycaemia and bone necrosis).
Regular follow up by paediatric cardiology team will be needed, and tailored according to the degree of coronary involvement, recommendations regarding anti-platelet and anticoagulant therapy, physical activity, follow-up assessment, and the appropriate diagnostic procedures to evaluate cardiac disease are classified according to risk strata.

Immunisations with all vaccinations should be deferred for at least 3 months following an episode of KD treated with IVIG.

Cross sectional imaging is increasingly considered especially in severe disease within first 3-6 months of first diagnosis then tailored according to involvement.

NOTE: Aetiology, pathogenesis, genetics, long term cardiology complications and long-term investigations and management of cardiac complications are out of the scope of this guideline, however reference number 5 the **AHA guidelines 2017** in 72 pages has covered all aspects of the acute and chronic disease in a very comprehensive which makes it an essential and highly recommended for both Paediatric and adult clinicians dealing with the disease.

9. Education and Training

A) 1-2 Yearly updates for the Paediatric Medical staff during Paediatric Clinical meetings and Audit meetings

B) Access to Guidelines on INsite during day to day clinical management of Kawasaki's Patient

10. Monitoring Compliance

What will be measured to monitor compliance	How will compliance be monitored	Monitoring Lead	Frequency	Reporting arrangements
Overall Management and the use of IVIG	Regular Audit	Dr A Sridhar and Team	2-3 yearly audits	Clinical Governance and Audit teams

11. Supporting References

1. American Heart Association (AHA) and American Academy of Paediatrics (AAP): Recommendations
2. Brian W. McCrindle, Anne H. Rowley et.al. Diagnosis, treatment, and long-term management of Kawasaki Disease: A Scientific Statement for Health Professionals from American Heart Association. *Circulation* 2017;135: e927–e999.
3. Eleftheriou D, Levin M, Shingadia D, et al. Management of Kawasaki disease. Archives of Disease in Childhood 2014; 99:74-83
4. Kawasaki Disease: Clinical features and Diagnosis: UpToDate review October 2020
5. Kawasaki Disease: Initial Treatment and Prognosis:UpToDate review October 2020
6. Incomplete (Atypical) Kawasaki Disease: UpToDate review October 2020
7. NHS patient safety alert: Risk of death and serious harm from failure to recognise acute coronary syndromes in Kawasaki disease patients: NHS/PSA/W/2016/004 (11 May 2016)
8. NICE ng143 November 2019 (updated November 2021): Fever in under 5s: assessment and initial management
9. Recommendations for Quantification methods during Pediatric Echocardiography: ASE Guidelines. Published J Am Echocardiogr 2010; 23:465-9
10. Robert Sundel- Kawasaki Disease: Initial treatment and diagnosis: Up To Date Feb19 2016.
11. Sleeper L etal; J Pediatr 2011:158(5): 831-835; Evaluation of Kawasaki Disease Risk Scoring Systems for IVIG Resistance
12. Sridhar A : UHL Kawasaki Disease Guideline, June 2016, Nov 2020
13. **Sundel R et al: Initial diagnosis and treatment of Kawasaki disease in Children. UpToDate October 2023**

12. Key Words

Kawasaki Disease (KD), Immunoglobulin (IVIG), Coronary artery aneurysm (CAA)

The Trust recognises the diversity of the local community it serves. Our aim therefore is to provide a safe environment free from discrimination and treat all individuals fairly with dignity and appropriately according to their needs.
As part of its development, this policy and its impact on equality have been reviewed and no detriment was identified.

Contact & review details			
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Date	**Issue number**	**Reviewed by**	**Changes made**
December 2020	**V3**	**S Arani** **S Shebani**	Added reference to PIMS-TS guidance Section 3 – Clinical features & diagnosis Added Macrophage Activation Syndrome (MAS) Section 6 – Treatment Point 2: Specified ***Low Dose*** Aspirin should be continued for minimum 6 weeks Point 4: Removed ranitidine as gastro protection option Section 8 – Follow-up Removed recommendation for varicella zoster immunization in patients requiring long term aspirin for persistent CAA Updated references Re-formatted
November 2023	**V4**	**S Arani** **S Shebani**	Removed ref to **Paediatric Inflammatory Multisystem Syndrome – Temporally Associated with SARS-CoV-2 (PIMS-TS) UHL Childrens Guideline** D4 (2020) as this document now archived Section 6. Treatment IV immunoglobulin : 2g/kg amended to **IV Immunoglobulin: 2gms/kg** Updated references

Appendix

1) Table 1- Kobayashi scoring system for predicting IVIG resistance
2) Table 2- Differential diagnosis of Kawasaki disease
3) Table 3- Recommended clinical guideline for the management of Kawasaki disease in the UK
4) Recommendation for prevention of thrombosis during the acute illness AHA 2017 guidelines
5) Recommended Coronary artery views and position of measurements (ASE 2010 paediatric Echo Guidelines).
6) Table 4- Kawasaki disease: Audit check list: please complete the form
7) Immunoglobulin request form- available from Insite
8) Parents information leaflets- www.chfed.org.uk/info (please download, print out and give to parents)

Table 1- Kobayashi scoring system for predicting IVIG resistance

Na< or equal to 132 (2 points)
Illness < or equal to 4 days (2 points)
ALT > or equal to 100 U/L (1 point)
Platelets < or equal to 300* 109 /L (1 point)
CRP > or equal to 10 mg/dl (1 point)
Age < or equal to 12 months (1 point)
> or equal to 80% neutrophils (2 points)

High risk > or equal to 5 points

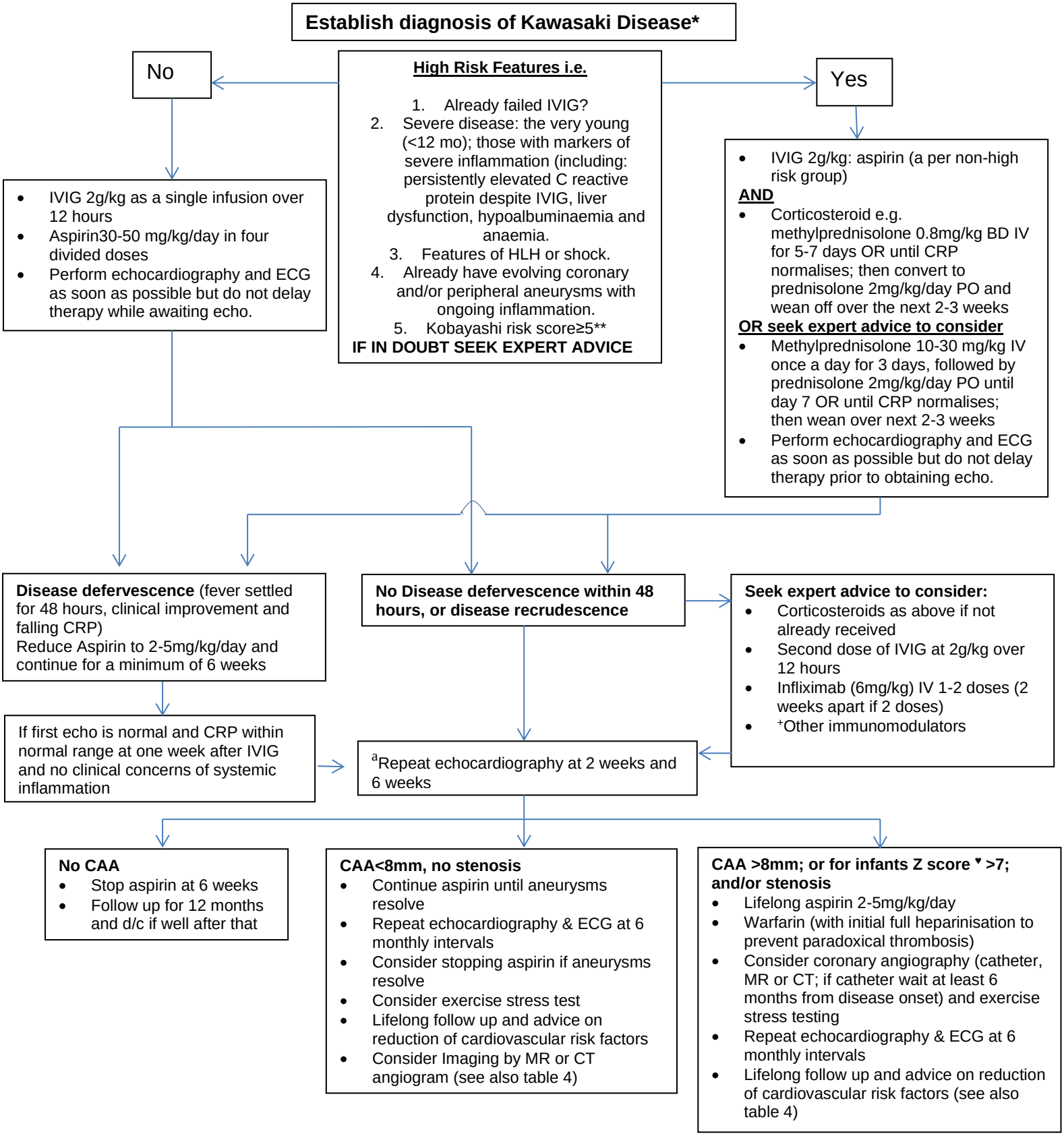
N.B: Kobayashi Risk scoring systems from Japan have good specificity but low sensitivity for predicting IVIG resistance and hence cannot be systematically adopted for patients of different ethnic groups living in other countries

Table 2- Differential diagnosis of Kawasaki disease

	Kawasaki disease	Toxic shock syndrome	Streptococcal scarlet fever	Steven-Johnson syndrome	Systemic onset Juvenile Idiopathic Arthritis
Age (years)	Usually<5	Usually >10	Usually 2-8	All ages	2-5
Fever (days)	Persistent	Usually<10	Variable, usually <10	Prolonged	Prolonged
Eyes	Non-exudative conjunctivitis, limbal sparing, anterior uveitis	Conjunctivitis	Normal	Exudative conjunctivitis, keratitis	Normal
Oral mucosa	Diffuse erythema, strawberry tongue	Erythematous	Pharyngitis, strawberry tongue	Erythema, ulceration, pseudomembrane formation	Normal
Peripheral extremities	Erythema of palms and soles, indurative edema, periungual desquamation	Swelling of hands and feet	Flaky desquamation	Normal	Arthritis
Rash	Erythematous, polymorphous, targeted or purpuric in 20%	Erythroderma	Papular erythroderma Pastia's lines, circumora;pallor	Target lesions	Transient salmon pink
Cervical lymph nodes	Non purulent swelling	Normal	Painful swelling	Normal	Diffuse adenopathy
Other	Arthritis	Mental status changes, coagulopathy, shock	Throat culture positive for group A Streptococcus	Arthralgia, associated herpes virus infection (30-75%)	Arthritis pericarditis
Characteristic lab results	Systemic inflammation, anaemia, transaminitis	Thrombocytopenia	Positive throat swab culture	Associated herpes virus infection	Systemic inflammation, anaemia

Table 3-Recommended clinical guideline for the management of Kawasaki disease in the UK[1] Eleftheriou D, et al. 2016

Please also see AHA 2017 guidelines that recommend use of Dual antiplatelet, LMWH and triple therapy**,** Appendix 4**:** Recommendation for prevention of thrombosis during the acute illness AHA 2017 guidelines Which we followed in fulminant cases with giant aneurysms in infants



*Treatment can be commenced before 5 days of fever if sepsis excluded; treatment should also be given if the presentation is > 10 days from fever onset if there are signs of persistent inflammation; **Kobayashi risk score ≥5 points Refer to paediatric cardiologist; Other specific interventions such as positron emission tomography (PET) scanning, addition of calcium channel blocker therapy, and coronary angioplasty at discretion of paediatric cardiologist. + Other immunomodulators may include ciclosporin. ♥For infants, Z score for internal coronary artery diameter >7 based on Montreal normative data: http://parameterz.blogspot.co.uk/2010/11/montreal-coronary-artery-z-scores.html.

Recommendation for prevention of thrombosis during the acute illness AHA 2017 guidelines:

Low dose Aspirin 3-5mg/kg/does should be given to patients without evidence3 of coronary artery changes until 4-6 weeks after the illness (Class I; Level of evidence C)
For Patients with rapidly expanding coronary artery aneurysms (CAA) or a maximum Z of ≥10, systemic anticoagulation with low molecular weight heparin (LMWH) or warfarin INR target 2.0-3.0 in addition to low does aspirin (Class IIa; level of evidence B) .
For patients at increased risk of thrombosis for example, with large or giant aneurysms (≥8 or Z score ≥10) and a recent history of coronary artery thrombosis, “triple therapy with aspirin and a second antiplatelet agent and anticoagulation with warfarin or LMWH may be considered (Class IIb; level of evidence C).
Ibuprofen and other non-steroidal inflammatory drugs with known or potential involvement of cyclooxygenase pathway may be harmful in patients taking Aspirin for its antiplatelet effects (Class III; level of evidence B)

Recommended Coronary artery views and position of measurements (ASE 2010 paediatric Echo Guidelines, Page 486 . Figure 16, please notice:
Measurements are inner to inner, 1mm distal to the CA orifice.
Measure at maximum filling in diastole, dual view (2D, and colour), low Niquest

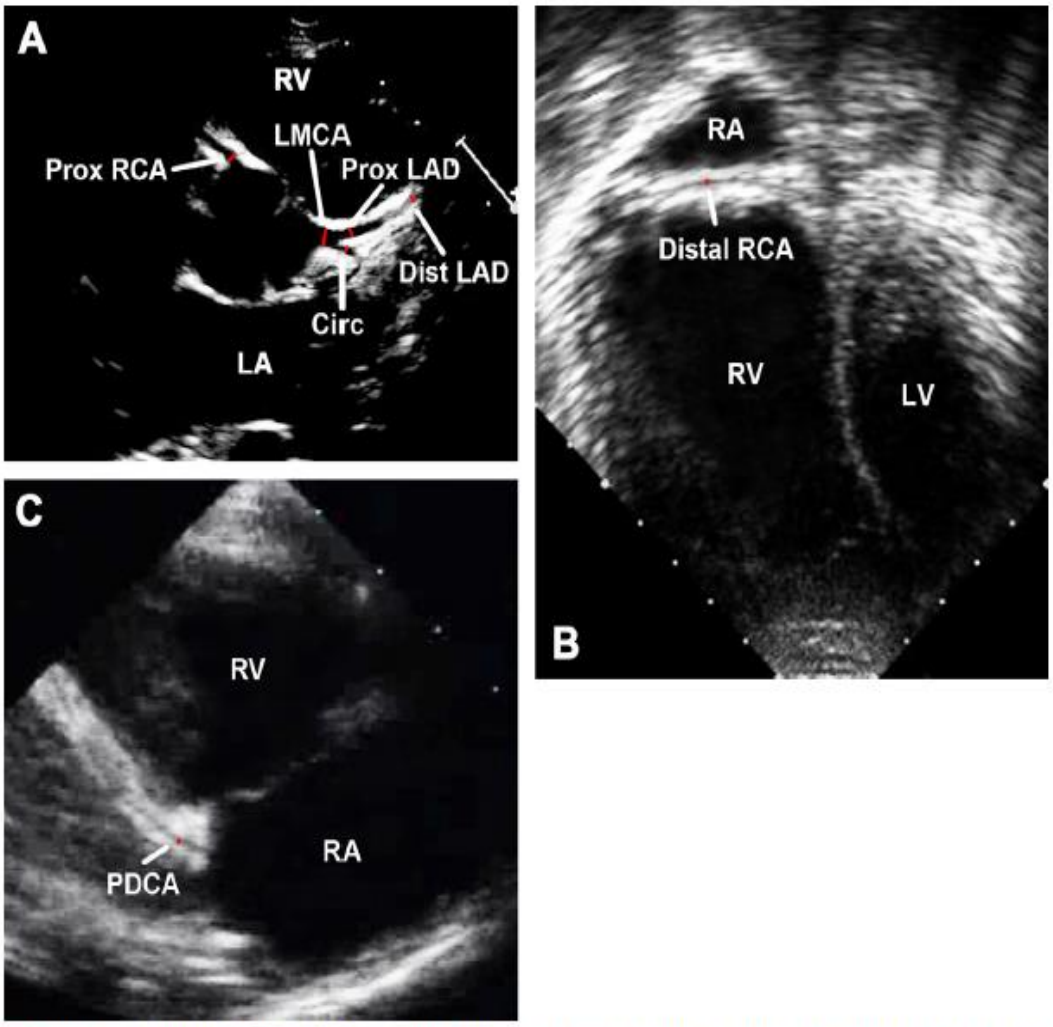


Figure 16 **(A)** Left main coronary artery (LMCA), proximal left anterior descending coronary artery (Prox LAD), distal left anterior descending coronary artery (Dist LAD), circumflex coronary artery (Circ), and proximal right coronary artery (Prox RCA) diameters in a parasternal short-axis view; **(B)** distal right coronary artery (Distal RCA) diameter in an apical 4-chamber view with posterior angulation; and **(C)** posterior descending coronary artery (PDCA) diameter in a parasternal long-axis view with posterior angulation. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

Macrophage Activation Syndrome: MAS

Hemophagocytic lymphohistiocytosis (HLH), is a potentially fatal hyperinflammatory syndrome characterized fever, hepatosplenomegaly, and cytopenias. HLH can be either primary, with a genetic aetiology, or secondary, associated with malignancies, autoimmune diseases, or infections.
Among rheumatic disorders, HLH occurs most frequently in systemic JIA and JSLE

HLH 2004 Diagnostic criteria

The diagnosis of HLH can be established if one of either 1 or 2 below is fulfilled:
1. A molecular diagnosis consistent with HLH
2. **Diagnostic criteria for HLH are fulfilled** (**five out of the eight criteria below**):
• Fever
• Splenomegaly
• Cytopenias (affecting ≥ 2 lineages in the peripheral blood): – Haemoglobin < 90 g/l (in infants < 4 weeks: haemoglobin < 100 g/l – Platelets < 100.000/ml – Neutrophils < 1000/ml
• **Hypertriglyceridemia** and/or **hypofibrinogenemia:** – Fasting triglycerides ≥ 265 mg/dl – Fibrinogen ≤ 1.5 g/L
• Haemophagocytosis in bone marrow or spleen or lymph nodes
• Low or absent NK-cell activity • Ferritin ≥ 500 µg/l
• Soluble CD25 ≥ 2400 U/L

Eur Rev Med Pharmacol Sci. 2012 Oct;16(10):1414-24.
Haemophagocytic syndrome in Rheumatic patients: A systematic review
Atteritano M1, David A, Bagnato G, Beninati C, Frisina A, Iaria C, Bagnato G, Cascio A.

Clinical Features and Diagnosis of Haemophagocytic Lymphohistiocytosis (HLH)- Macrophage Activation Syndrome- MAS: UpToDate review October 2020

If you suspect HLH- Please discuss with the Paediatric Haematologist and Rheumatologist teams

Table 4 - Kawasaki disease Audit check list

Hospital number: **Age at diagnosis:** **Gender:** **Ethnicity:**
Time difference between the onset of symptoms and diagnosis of Kawasaki disease: _____weeks _____days
Type of Kawasaki disease: Complete / Incomplete/ Refractory/ Severe
What criteria are documented (circle/tick the answers) 1) Bilateral non- exudative conjunctivitis 2) Mucositis 3) Cervical Lymphadenopathy 4) Polymorphic Rash 5) Extremity Changes (Erythema of palms and soles, Oedema of hands and feet)
Any other additional features mentioned below present (please circle/tick the answers) a. Extreme irritability b. Abdominal Pain c. Diarrhoea and vomiting d. Urethritis with sterile pyuria e. aseptic meningitis f. desquamation of genital area g. Raised platelets h. Raised CRP (>30) i. Hepatic dysfunction j. Arthritis or arthralgia k. Heart murmur l. cardiac arrhythmias m. gallbladder hydrous n. Reddening of BCG scar o. Heart failure
Document abnormal blood results:
Document the abnormal culture results (blood, csf, urine, throat and skin swab)
Document the treatment given 1. IV Gamma globulin Yes / No If No, reason _____ 2. Whether needed second dose of IV Gamma globulin: **Yes / No** 3. Aspirin: High dose or Low dose or High dose followed by low dose 4. Gastro prophylaxis given: Yes / No 5. Antibiotics: Yes / No Reason: _____ 6. Steroids given: Yes / No 7. Others example: heparin, anti-TNF
Responded well to IVIG: Yes / No If No, answer the following questions. If refractory to IV Gamma globulin, discussed with rheumatology: Yes / No What was the outcome: Treated in LRI / Transferred to other hospital If treated in LRI, what was treatment: Methylprednisolone/Oral Prednisolone/Infliximab/Other
ECG done during this admission: Yes / No If yes, result: Cardiology referral requested as: **Inpatient / Outpatient** Agreed to be seen by Cardiologist as: **Inpatient / Outpatient** Result of ECHO if done before discharge:
ECHO Report (To be completed for outpatient ECHO)
Total length of hospital stay: _____days