



**BLOOD MANAGEMENT
CLINICAL PRACTICE GUIDELINE**

Haemoglobinopathy screening and management in pregnancy

Scope (Staff): WNHS Obstetrics and Gynaecology Directorate staff

Scope (Area): Obstetrics and Gynaecology Directorate clinical areas

This document should be read in conjunction with this [Disclaimer](#)

Contents

Background	2
Effects of thalassaemia in pregnancy	4
Effects of sickle cell in pregnancy	5
Screening women for haemoglobin disorders.....	6
1. Screening.....	6
2. Women with NO identified risk factors for haemoglobinopathy	6
3. Women who are at risk of haemoglobinopathy	6
4. Obtaining partner haemoglobin studies.....	7
5. Assessing the fetal risk of clinically significant haemoglobinopathy	7
6. Women and current partners who have been screened in previous pregnancies	8
7. Partners who decline ‘paternal’ testing, partner no longer in contact with woman or unknown.....	8
8. Women referred to KEMH for haemoglobinopathy screening	9
9. Women who attend KEMH in early pregnancy / non-pregnant with suspected or confirmed haemoglobin disorder	9
Management of co-existing iron deficiency.....	9
Screening women for haemoglobin disorders: Flow chart	10
Clinically significant maternal haemoglobinopathy – MFM Service referral	11
Significant maternal haemoglobinopathies requiring immediate referral to MFM in pregnancy	11
No fetal risk of clinically significant haemoglobinopathy – Sign off by CNC PBM	11
References	13



Aim

- To identify women and their partners at risk of haemoglobinopathies
- To identify fetuses at risk of clinically significant haemoglobinopathies
- To provide a referral process for women to the Maternal Foetal Medicine (MFM) Service for specialised multidisciplinary obstetric management when there is a confirmed high-risk haemoglobinopathy in the women or fetus

Background

Normal haemoglobin contains an iron-containing haem molecule that combines with four globin chains.

The major globin chain in adults consists of:

- two alpha (α) chains, encoded by four α globin genes
- two beta (β) chains, encoded by two β globin genes.

Haemoglobinopathies occur as a result of changes in the structure or quantity of these globin genes. The resulting changes in the assembled haemoglobin molecule may result in haemolysis or impaired erythropoiesis.^{1,2}

Haemoglobinopathies are *autosomal recessive disorders*, indicating that patients must inherit abnormalities from both parents to develop clinically significant disease.

Global estimates suggest that approximately 7% of the world's population are carriers of haemoglobinopathy. They are becoming more prevalent within Australia due to changes in migration from endemic regions.³

Thalassaemia

Thalassaemia is caused by mutations in, or deletions of, the α or β genes resulting in decreased globin chain production. It is classified as alpha (α)-thalassaemia when there is impaired (α) chain synthesis, or beta (β)-thalassaemia when there is impaired β -chain synthesis.^{1,2}

Both forms of thalassaemia typically show

- a reduction in mean corpuscular volume (MCV) and
- a reduction in mean corpuscular haemoglobin (MCH)

with or without accompanying anaemia.

The severity of the disorder will depend on the number of abnormal genes present.

A single α gene deletion/dysfunction may have no impact on the patient or red cell indices, whereas deletion of all four α genes results in Bart's hydrops fetalis,^{1,2,3,4,5,6,7,8} commonly resulting in intrauterine death unless treated by intrauterine transfusion. Clinically, β -thalassaemia ranges from an asymptomatic carrier state to thalassaemia major, characterised by lifelong dependency on blood transfusions and associated complications including iron overload.^{4,5,6,7} See 'Effects of thalassaemia types' in this document.

Partner testing is required if a woman has a thalassaemia trait because co-inheritance with a second thalassaemic typically results in more significant clinical manifestations for the fetus.

^{1,2,3,4}

Variant haemoglobins

Variant haemoglobins are also caused by mutations in the α or β genes. In contrast to thalassaemia, resulting in reduced globin production, the mutations result in production of a structurally abnormal haemoglobin. There are a large number of variant haemoglobins with variable clinical impact. Sickle cell disease is a special case, discussed below. Haemoglobin E (HbE) is also common and has significant clinical implications for the fetus if co-inherited with β -thalassaemia trait.^{1,2,3,4,5,6,7}

Sickle cell disease

Sickle cell disease is characterised by production of an abnormal β -globin chain that forms haemoglobin S (HbS).

- Those with one abnormal β -globin chain have sickle cell trait.
- Those with sickle cell disease have inherited abnormalities from both parents.
- Sickle cells have a shortened lifespan, causing chronic haemolytic anaemia, and – under hypoxia and other conditions – may result in acute haemolytic / ischaemic episodes known as sickle cell crises.^{1,2,4,7} Maternal effects may include pain, infections, pulmonary complications, anaemia, pre-eclampsia and high rates of caesarean section. The fetus is at risk for spontaneous abortion, preterm birth, intrauterine growth restriction and perinatal death.^{1,2,3,4,5,6,7,8}

Geographical distribution of haemoglobin disorders

Historically, the prevalence of α - and β - thalassaemia was high in the Middle East, Mediterranean countries, South East Asia, Indian sub-continent and parts of Africa.

- The most common mutation is -a.3.7 (single gene deletion) which can show a significantly lower Hb and MCV, and higher RCC, showing microcytosis and mild anaemia
- A severe form of α -thalassaemia (α^0) is found in South East Asia and parts of the Mediterranean.^{1,2,4,5,6}
- Haemoglobin E (HbE) commonly occurs in the South East Asia and the Indian sub-continent.
- Sickle cell disease is prevalent in Africa.^{1,2,4,5,6} See 'Populations at risk of haemoglobin disorders' below.

With changing patterns of migration within Australia and throughout the world, it is important to assess a patient's ethnic background to assess if they are at risk of haemoglobinopathy.^{3,4,5,7} In addition, identifying the patient's ethnic background on the haemoglobinopathy request form also assists the laboratory in directing testing and interpreting the results.^{4,7}

Populations at risk of haemoglobin disorders

Population locations with associated high risk for haemoglobin disorders: 1,2,3,4,5,7

Thalassaemia	Sickle cell disease
<ul style="list-style-type: none"> African Caribbean South East Asian and Chinese Middle Eastern Pacific Islanders New Zealand Maori Southern Europe/Mediterranean Indian subcontinent Some northern Western Australian and Northern Territory and Torres Strait Australian indigenous communities. 	<ul style="list-style-type: none"> African Caribbean Middle Eastern Southern Europe/Mediterranean Indian subcontinent South American

Effects of thalassaemia in pregnancy^{1,2,3,4,5,6,7,8}

Type of thalassaemia	Genotype	Effect
α^+ thalassaemia trait (α thalassaemia minor)	One deleted α gene on one chromosome only (eg. $\alpha\alpha/\alpha^-$)	Normally asymptomatic. Slight decrease in MCV/MCH
α^+ thalassaemia trait (α thalassaemia minor)	Two deleted α genes, one on each chromosome (e.g. α^-/α^-)	Reduced MCV/MCH +/- mild anaemia
α^0 thalassaemia trait (α thalassaemia minor)	Deletion of two α genes on one chromosome (e.g. $\alpha\alpha^-/-$)	Reduced MCV/MCH +/- mild anaemia
Haemoglobin H disease	Total of three deleted α genes ($\alpha^-/-$)	Moderate microcytic anaemia, hepatosplenomegaly. May require transfusion
Haemoglobin Barts	Complete absence of all α genes ($-/-/-$)	Usually foetal hydrops and death in utero
β thalassaemia minor	One absent (β^0) or impaired (β^+) β gene on one chromosome only (e.g. β/β^0 or β/β^+)	Normally asymptomatic. Reduced MCV/MCH +/- anaemia.
β thalassaemia intermedia	Defined clinically. Various genetic defects (e.g. β^+/β^+ or β^0/β^+)	Variability in symptoms. Moderate microcytic anaemia; hepatosplenomegaly. May require transfusion.
β thalassaemia major	Two absent β genes (i.e. β^0/β^0)	Severe microcytic anaemia. Requires frequent blood transfusions.

Type of thalassaemia	Genotype	Effect
Haemoglobin E trait	One normal β gene on one chromosome and one abnormal β gene on the other chromosome (e.g. β/β^E)	Asymptomatic. Possible decreased MCV/MCH
Haemoglobin E disease	Two abnormal β genes (i.e. β^E/β^E)	Mild anaemia, reduced MCV/MCH
Haemoglobin E/ β thalassaemia	one absent/impaired β gene (β^0 or β^+) one abnormal β gene (e.g. β^E/β^0 or β^E/β^+)	Moderate to severe microcytic anaemia. Clinical symptoms vary – potentially thalassaemia intermedia or major.

NB. This list is a guide only. Advice should always be obtained from Haematology in the first instance, particularly in women with coinherited haemoglobinopathy (e.g. α thalassaemia and HbE)

Effects of sickle cell in pregnancy ^{1,2,3,4,5,6,7}

Sickle status	Genotype	Effect
Sickle cell trait	one normal β gene one abnormal β gene mutation (e.g. β/β^S)	Asymptomatic normally, normal RBC indices. Sickle cell formation can occur during high fever and significant hypoxia
Sickle cell disease/anaemia	Two abnormal β genes (e.g. β^S/β^S)	Mild to moderate chronic haemolytic anaemia, vaso-occlusion - brain, chest, bones, kidneys, spleen and placenta. Increased maternal and perinatal mortality

NB. This list is a guide only. Advice should always be obtained from Haematology in the first instance, particularly in women with coinherited haemoglobinopathy (e.g. α or β thalassaemia and HbS).

Screening women for haemoglobin disorders

Refer to [flow chart at the end of this guideline](#).

1. Screening

All antenatal women should be offered screening if they fall into these categories:

- Women from any of the geographical regions that put them at risk
- With a past history of unexplained anaemia
- With a family history of anaemia (unknown cause) or haemoglobinopathy
- With a **MCV ≤ 80 fL or MCH ≤ 27 pg**

2. Women with NO identified risk factors for haemoglobinopathy

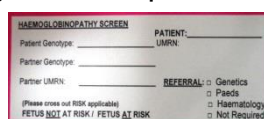
Assess the FBP (full blood picture) and iron studies at booking bloods.

- If FBP is normal, reassess FBP and iron studies at 28 weeks gestation
- Treat underlying iron deficiency if ferritin < 30 ug/L and reassess FBP once iron deficiency is corrected.
- Assess, identify and treat Vitamin B12 deficiency
- See WNHS Clinical Guideline: Obstetrics and Gynaecology: [Anaemia and Iron Deficiency: Management in Pregnancy and Postpartum](#)

3. Women who are at risk of haemoglobinopathy

- Assess ethnic background.
- Assess FBP and iron studies
- Determine if haemoglobinopathy screening has been performed previously (e.g. through General Practitioner (GP) or in previous pregnancy). If so, obtain a copy and file/upload the results. *If testing was performed outside Australia, or the results are not available, testing should be repeated.*
- If the woman or partner has been found to have a haemoglobinopathy, ascertain if the same partner is the father of the current pregnancy. In the case of a new partner, partner studies will be required (see section 4 'Obtaining partner haemoglobin studies' below).
- All known, suspected or being screened haemoglobinopathies need to be referred to the Clinical Nurse Consultant Patient Blood Management (CNC PBM), via e-referrals ('Haematology CNC PBM').
- The CNC PBM will then commence a Haemoglobinopathy Management Plan (MR036) and continue with the processes below.

The haemoglobinopathy screening sticker is placed on the MR004 and uploaded to DMR, once all is completed.



HAEMOGLOBINOPATHY SCREEN

Patient Genotype: _____ PATIENT: _____
Partner Genotype: _____ UMRN: _____
Partner UMRN: _____

REFERRAL: ☐ Genetics
☐ Paeds
☐ Haematology
☐ Not Required

Please cross out risk applicable
FETUS NOT AT RISK / FETUS AT RISK

- If testing has not been performed (or results are unavailable), provide counselling and with maternal consent arrange haemoglobinopathy studies **on the woman only**. Request “**FBP, iron studies and haemoglobinopathy studies**”. The request form should also include the woman’s ethnic background to guide interpretation.
- Women should be directed to have the testing undertaken at a PathWest Laboratory to facilitate full haemoglobinopathy screening including DNA studies.
- If the woman is attending a low risk midwives’ clinic and the haemoglobin studies are abnormal, the woman should be referred to the CNC PBM for follow-up. If the haemoglobinopathy studies results are normal the women may then continue antenatal care at the midwives’ clinic.
- If the woman’s results identify a haemoglobinopathy which places the foetus at risk, partner studies should be performed and arrangements for the woman to be reviewed in the obstetric clinic.
- If partner test results confirm a haemoglobinopathy which places the foetus at high-risk of a clinically significant haemoglobinopathy, arrange referral to MFM Service for multidisciplinary care. This may also include genetic counselling and haematologist involvement.

4. Obtaining partner haemoglobin studies

- Ideally partner screening is obtained preconception such that the parents can make an informed decision about risks prior to pregnancy. In the absence of this, maternal results are used to direct partner testing. Concurrent testing of the woman and the partner is only performed when specifically directed by the MFM Service, CNC PBM or Haematology Department.
- Provide counselling and with paternal consent arrange haemoglobinopathy studies on the partner. Request “**FBP, iron studies and haemoglobinopathy studies**”. The request form should be completed with the partner’s demographic details; a UMRN will be registered when he attends a PathWest laboratory for testing. In the clinical notes section include his ethnic background, the woman’s details and haemoglobin genotype (i.e. $\alpha\alpha/--$) and clearly identify “**Partner testing**”.
- Partners should be directed to have the testing undertaken at a PathWest Laboratory to facilitate full haemoglobinopathy screening.

5. Assessing the fetal risk of clinically significant haemoglobinopathy

- The risk to the fetus is dependent upon the maternal and paternal genotype. Assessment of the risk is undertaken by the CNC PBM and Haematology department. If the fetus is identified as **high risk** for haemoglobin disease by the CNC PBM and Haematology department, a e-referral is sent to MFM service

- Following review of the results by The MFM Service an appointment will be arranged for the woman to attend KEMH for assessment as soon as possible +/- referral to Genetic Services WA (GSWA) if required.
- An individual management plan will be developed and documented in the medical records between the MFM Service and the woman/partner. This may include;
 - invasive testing, specialised ultrasonography and family testing
 - a Neonatal Management Plan (NMP) to direct specific management and neonatal testing following delivery of the infant.
 - a copy of the Neonatal Management Plan will be forwarded to the Neonatal Department via the Neonatal Clerical Support team.
 - The NMP is usually completed by the CNC PBM but may also be done by the MFM team.
- If the fetus is identified as **not at risk** of a clinically significant haemoglobinopathy on review of the results by Haematology, the CNC PBM will complete the Management Plan and place the MR036 and sticker in the patient medical notes.

6. Women and current partners who have been screened in previous pregnancies

If a woman and her current partner have been screened previously within Australia and results are available for both high performance liquid chromatography (HPLC) and DNA analysis, no further testing is required.

Obtain a copy of the results and place in the medical records.

If the results are not available via the GP, original testing laboratory or within the patient's medical/laboratory records, re-testing is required.

All previously known and tested women must still be referred to the CNC PBM for the current pregnancy.

7. Partners who decline 'paternal' testing, partner no longer in contact with woman or unknown

In a woman with a confirmed haemoglobinopathy, screening is aimed to identify the fetus at risk, to enable informed choice regarding management of an affected infant. Partners should be encouraged to participate in screening. However, if after discussion the partner declines screening (or is not available for testing or is unknown) this should be clearly documented in the woman's medical records, and the process below followed:

- Complete an e-referral to haematology 'CNC PBM Haemoglobinopathy' and add additional details or results
- CNC PBM will liaise with Haematology, MFM and others services required and create a Neonatal Management Plan for screening the baby at birth

8. Women referred to KEMH for haemoglobinopathy screening

Women can be referred to KEMH for assessment of foetal risk when the woman has a confirmed or suspected haemoglobinopathy. The screening process is followed to ascertain the risk to the foetus but not all women will require care and delivery at KEMH.

The CNC PBM also liaises with GPs and screening can be facilitated this way.

Women with identified at risk pregnancies will generally remain under the care of KEMH for antenatal care, delivery and neonatal care.

9. Women who attend KEMH in early pregnancy / non-pregnant with suspected or confirmed haemoglobin disorder

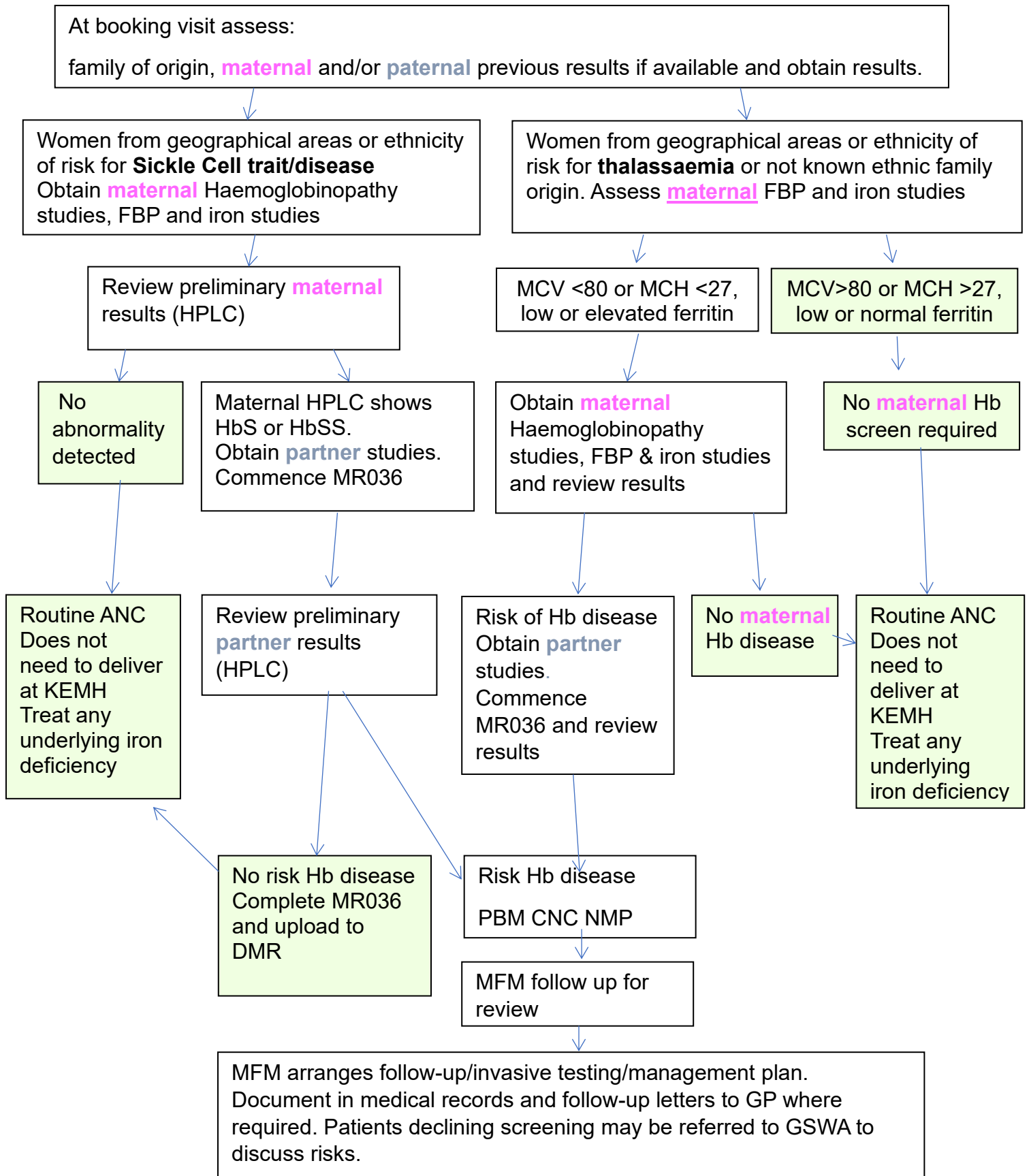
Women who attend KEMH (e.g. Emergency Centre, Outpatient clinics – exclusive of ANC – or Ward 6) with a suspected or confirmed haemoglobin disorder may require additional follow-up dependent on age and childbearing potential. If the woman is likely to become pregnant, then follow-up of results and partner testing (if appropriate), as above, is warranted. If the woman will no longer be attending KEMH for follow-up, the woman's GP should be contacted by letter with the results and instructed to undertake partner testing if the woman has childbearing potential.

Management of co-existing iron deficiency

It is important to monitor ferritin levels in pregnant women with a confirmed haemoglobinopathy disorder, at booking, 28 weeks and at 34 weeks gestation as a minimum. Iron deficiency should be treated as per current guidelines. See WNHS Clinical Guideline: [Anaemia and Iron Deficiency: Management in Pregnancy and Postpartum](#).

Women may present at booking with an elevated ferritin level and develop iron deficiency as pregnancy progresses. As haemoglobinopathy commonly causes microcytosis⁵, the most reliable marker to assess iron deficiency are trends in ferritin (and other parameters available on iron studies) and haemoglobin levels over time. **The administration of IV iron will not correct anaemia in iron replete women and should not be given.**

Screening women for haemoglobin disorders: Flow chart



Abbreviations: ANC- Antenatal clinic, FBP- full blood picture, GP- General Practitioner, GSWA- Genetic Services WA, Hb- haemoglobin, HPLC- high performance liquid chromatography, HbS- sickle cell trait, MCV- mean cell volume, MCH- mean cell haemoglobin, MFM- Maternal Foetal Medicine NMP – neonatal management plan

Clinically significant maternal haemoglobinopathy – MFM Service referral

Pregnant women with a clinically significant haemoglobinopathy should have their obstetric care managed through the MFM Service. A multidisciplinary approach, with collaboration between Obstetricians, Haematologists, Neonatologists, Geneticists and Pathologists (regarding laboratory testing) is preferred in these cases.

The following list identifies the women with confirmed significant haemoglobinopathy who require immediate referral to the MFM Department for ongoing care and management on confirmation of pregnancy.

Significant maternal haemoglobinopathies requiring immediate referral to MFM in pregnancy

- Maternal sickle cell anaemia and all other types of sickle cell disease (i.e. HbSS, HbSC, HbSD-Punjab)
- HbS/ β thalassaemia
- β thalassaemia intermedia, β thalassaemia major
- HbE/ β thalassaemia
- HbH (α -/-)
- Pregnancies with a 1 in 4 risk of significant haemoglobinopathy in the fetus

No fetal risk of clinically significant haemoglobinopathy – Sign off by CNC PBM

- The tables below identify situations where the CNC PBM at KEMH may acknowledge results without medical review.
- An asterisk (*) denotes that CNC sign off of 'no fetal risk' will only occur in the absence of other co-inherited haemoglobinopathy. This is also dependent on the number and type of α and/or β gene deletions / mutations in the case of maternal and paternal haemoglobinopathy.
- All other haemoglobinopathy must be reviewed by the MFM Service or Haematologist as they will have an identified or increased risk of haemoglobinopathy requiring multidisciplinary assessment.

Table 1: Maternal ALPHA globin disorders and paternal status

		Paternal status									
		Normal	α^+ thal trait (α^-/α^- or $\alpha\alpha/\alpha^-$)	α^0 thal trait ($\alpha\alpha/--$)	HbH disease ($\alpha^-/--$)	β thal trait	β thal int./maj	HbE trait	HbE disease HbE + β thal	HbS trait	HbS disease
Maternal status	Normal		CNC	CNC	CNC	CNC	CNC	CNC	CNC	CNC	CNC
	α^+ thal trait (α^-/α^- or $\alpha\alpha/\alpha^-$)	CNC	CNC*	MFM	MFM	CNC*	CNC*	CNC*	CNC*	CNC*	CNC*
	α^0 thal trait ($\alpha\alpha/--$)	CNC	MFM	MFM	MFM	CNC*	CNC*	CNC*	CNC*	CNC*	CNC*
	HbH disease ($\alpha^-/--$)	CNC	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM

Table 2: Maternal BETA globin disorders and paternal status

		Paternal status									
		Normal	α^+ thal trait (α^-/α^- or $\alpha\alpha/\alpha^-$)	α^0 thal trait ($\alpha\alpha/--$)	HbH disease	β thal trait	β thal int./maj	HbE trait	HbE disease HbE + β thal	HbS trait	HbS disease
Maternal status	Normal		CNC	CNC	CNC	CNC	CNC	CNC	CNC	CNC	CNC
	β thal trait	CNC	CNC*	CNC*	CNC*	MFM	MFM	MFM	MFM	MFM	MFM
	HbE trait	CNC	CNC*	CNC*	CNC*	MFM	MFM	MFM	MFM	MFM	MFM
	HbS trait	CNC	CNC*	CNC	CNC*	MFM	MFM	MFM	MFM	MFM	MFM
	β thal int./major	CNC	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM
	HbE disease	CNC	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM
	HbE/ β thal	CNC	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM
	HbS disease	CNC	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM

References

Bibliography









1. Weatherall D J. The thalassaemia's: Disorders of globin synthesis. In: Kaushansky K et al, Editors. Williams Haematology. 8th ed. China: McGraw Hill; 2010. p 675 – 707.
2. Bain B J. Haemoglobinopathy diagnosis. 2nd ed. Carlton: Blackwell; 2006.
3. Tan Y L & Kidson-Gerber G. Antenatal haemoglobinopathy screening in Australia. MJA, 2016; 204 (6): 226 - 230.
4. NHS Screening Programmes. Sickle Cell and Thalassaemia Handbook for Laboratories. London; NHS:2012
5. Welch E and Wright J. Inherited red cell disorders. In: Pavord S and Hunt B. Editors. The obstetric Haematology Manual. Cambridge: Cambridge University Press; 2010. P 28 – 44.
6. Leung T Y and Lao T T. Thalassaemia in pregnancy. BPOGYN. 2012; 26: 37- 51.
7. Ryan et al. Significant haemoglobinopathies; guidelines for screening and diagnosis. BJH. 2010; 149: 35 – 49.
8. Fucharoen S, Viprakasit V. Hb H disease: clinical course and disease modifiers. ASH Education Program Book. 2009 Jan 1;2009(1):26-34.

Related WNHS policies, guidelines and related forms

WNHS Clinical Guideline: Obstetrics and Gynaecology: [Anaemia and Iron Deficiency: Management in Pregnancy and Postpartum](#)

Forms

- MR004 Obstetric Special Instruction Sheet
- MR036 Referral for Haemoglobinopathy Screening

Keywords	Fetal risk, foetal risk, Hb disease, haematology trait, haemoglobinopathy screening in pregnancy		
Document owner:	Blood Management Department		
Author / Reviewer	Patient Blood Management - Haematology Consultant and CNC PBM		
Date first issued:	01/2010 (v1- in O&GD guidelines- see below)		
Reviewed dates:	Apr 2014(v2); Oct 2014(v3); Oct 2015(v4); Apr 2017(v5); Sept 2017(v6); Feb 2022(v7); Apr 2025(v8-moved to BM set))	Next review date:	Apr 2028
Endorsed by:	Blood Management Committee	Date:	08/04/2025
Noted by:	Maternity and Obstetrics Clinical Practice Outcomes Committee (guideline moving from OG to BM set)	Date:	16/04/2025
NSQHS standards applicable:	<div style="display: flex; justify-content: space-between;"> <div style="width: 48%;"> <input checked="" type="checkbox"/>  1: Clinical Governance <input checked="" type="checkbox"/>  2: Partnering with Consumers <input type="checkbox"/>  3: Preventing and Controlling Healthcare Associated Infection <input type="checkbox"/>  4: Medication Safety </div> <div style="width: 48%;"> <input type="checkbox"/>  5: Comprehensive Care <input checked="" type="checkbox"/>  6: Communicating for Safety <input checked="" type="checkbox"/>  7: Blood Management <input checked="" type="checkbox"/>  8: Recognising and Responding to Acute Deterioration </div> </div>		

Printed or personally saved electronic copies of this document are considered uncontrolled.

Access the current version from WNHS HealthPoint.

Version history

Number	Date	Summary
1	Jan 2010	First version. Previously titled 'B1.1.12 Haemoglobinopathies Screening and Referral'.
2-7	Prior to Apr 2025	<p>Previously within O&GD guideline set- Refer to O&GD Guideline Updates for details of previous versions. Note- In Apr 2017, amalgamated three individual documents on haemoglobinopathy management dating from Jan 2010 and title changed to 'Haemoglobinopathy Screening in Pregnancy'.</p> <p>Superseded:</p> <ol style="list-style-type: none"> 1. B1.1.12 Haemoglobinopathy Screening (dated Oct 2014) 2. Assessment of No fetal risk of Hb disease which may be signed off by the CNC Haematology (dated Sept 2016) <p>Contact OGD Guideline Coordinator for archived versions prior to 2025.</p>
8	Apr 2025	<ul style="list-style-type: none"> • Guideline reviewed- made more succinct, with updated formatting. • Title changed to 'Haemoglobinopathy Screening and Management in Pregnancy'. Guideline moved from O&G guideline set to Blood Management Committee ownership. • Page 3 under geographical distribution the first dot point "the most common mutation" has been added • Reference to the word 'black' has been removed and just the country or region included for populations at risk • Flow chart tidied up, and terminology maternal and partner used throughout

This document can be made available in alternative formats on request for a person with a disability.

© North Metropolitan Health Service 2025

Copyright to this material is vested in the State of Western Australia unless otherwise indicated. Apart from any fair dealing for the purposes of private study, research, criticism or review, as permitted under the provisions of the *Copyright Act 1968*, no part may be reproduced or re-used for any purposes whatsoever without written permission of the State of Western Australia.

www.nmhs.health.wa.gov.au