



CLINICAL PRACTICE GUIDELINE

Haemoglobinopathy Screening in Pregnancy

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

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Aims

1. To identify and screen women and their partners at risk of haemoglobin disorders.
2. To identify fetuses at risk of significant haemoglobin disease.
3. To offer genetic counselling to women and their partners identified as “high risk”, to enable informed choice surrounding decision making.
4. To explain haematological abnormalities including microcytic anaemia.
5. Provide referral mechanism for women to the Maternal Fetal Medicine for specialised multidisciplinary obstetric management when there is a confirmed haemoglobin disorder which carries risk for the woman or fetus.

1. Background information

Haemoglobinopathies are autosomal recessive disorders which imply that they must be inherited through both parents who may be carriers, or have the disorder themselves. Normal haemoglobin contains a haem molecule that combines with four globin chains; two of which are classified as alpha (α) chains, made up of four α globin genes and two as beta (β) chains, made up of two β globin genes. Haemoglobin diseases are a result of changes within the structure or quantity of these globin genes which causes an imbalance in the globin chains resulting in haemolysis and impaired erythropoiesis.^{1,2} Global estimates suggest that approximately 7% of the world's population are carriers of haemoglobinopathy; however they are becoming more prevalent within Australia due to changes in migration patterns.³

Thalassaemia

Thalassaemia is caused by mutations or deletions in the α or β genes. The red cell indices often show a reduction in mean corpuscular volume (MCV) and mean corpuscular haemoglobin (MCH) \pm anaemia. See table 2. It is classified as alpha (α)-thalassaemia when there is absent or decreased α -chain synthesis, or beta (β)-thalassaemia when there is absent or decreased β -chain synthesis.^{1,2} Partner testing is required if a woman has a thalassaemia trait because if her partner also has a confirmed carrier trait it will pose risk for the fetus of major thalassaemia.^{1,2,3,4} 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 and carries significant risk for the pregnant woman and offers very limited cure/treatment for the fetus/baby.^{1,2,3,4,5,6,7,8} Similarly β -thalassaemia ranges from an asymptomatic carrier state to thalassaemia major, which results in life long dependency on blood transfusions and associated complications including iron overload.^{4,5,6,7} There are a large number of β globin abnormalities, however Haemoglobin E (Hb E) has significant clinical implications for the fetus if the partner testing identifies β thalassaemia.^{1,2,3,4,5,6,7}

Sickle cell disease

Sickle cell disease occurs when the structure of the beta globin chain is abnormal.^{1,2} Defective genes produce abnormal haemoglobin beta chains resulting in Hb called HbS. Sickle cell disease occurs when the abnormal genes are inherited from both parents. Sickle cells have increased fragility and a shortened life span of 17 days causing chronic haemolytic anaemia which leads to episodes of ischaemia and pain known as sickle cell crises.^{1,2,4,7} Maternal effects may include pain, infections, pulmonary complications, anaemia, pre-eclampsia and caesarean section. The fetus is at risk for spontaneous abortion, pre-term birth, intra-uterine growth restriction and perinatal death.^{1,2,3,4,5,6,7,8}

2. 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. A severe form of α -thalassaemia (α^0) is found in South East Asia.^{1,2,4,5,6} Haemoglobin E (Hb E) commonly occurs in the South East Asia and the Indian sub-continent, Sickle cell disease is prevalent in tropical Africa.^{1,2,4,5,6} See table 1.

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

Populations at risk of Haemoglobin Disorders

Populations at risk for haemoglobin disorders include:

Table 1 Population locations with associated high risk of haemoglobin disorders
^{1,2,3,4,5,7}

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

3. Effect of Thalassaemia Types in Pregnancy

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

TYPE OF THALASSAEMIA	GENOTYPE	EFFECT
α thalassaemia trait α^+ (α thalassaemia minor)	One deleted α gene in one chromosome only e.g. $-\alpha/\alpha\alpha$	Asymptomatic normally. Slight decrease in MCV/MCH
α thalassaemia trait α^+ (α thalassaemia minor)	Two deleted α genes, one from each chromosome e.g. α^-/α^-	Mild anaemia, slight decrease in MCV/MCH
α thalassaemia trait α^0 (α thalassaemia minor)	Deletion of two α genes in one chromosome only e.g. $--/\alpha\alpha$	Mild anaemia, decrease in MCV/MCH
Haemoglobin H disease	Total of three deleted α genes, in both chromosomes $\alpha^-/--$	Moderate anaemia, microcytic anaemia, hepatosplenomegaly, may require transfusion
Haemoglobin Barts hydrops	Complete absence of all α genes in both chromosomes $--/--$	Usually death in utero, hydrops fetalis syndrome
β thalassaemia trait (β thalassaemia minor)	One absent (0) or defective ($^+$) β gene in one chromosome only e.g. β/β^0 or β/β^+	Asymptomatic normally. May have mild anaemia, normal/reduced MCV
β thalassaemia intermedia	Two defective β genes or one absent and one defective in each chromosome e.g. β^+/β^+ or β^0/β^+	Variability in symptoms. Moderate anaemia, hepatosplenomegaly
β thalassaemia major	Two absent β genes in both chromosomes β^0/β^0	Severe anaemia. Require frequent blood transfusions
Haemoglobin E trait (HbE)	One normal β gene in one chromosome and one abnormal E (β gene mutation) in the other chromosome e.g. β/β^E	Asymptomatic, possible microcytosis and decreased MCV/MCH
Haemoglobin E disease	Two abnormal E (β gene mutations), one in each chromosome β^E/β^E	Mild anaemia and microcytosis, decrease in MCV/MCH

TYPE OF THALASSAEMIA	GENOTYPE	EFFECT
Haemoglobin E/ β thalassaemia	Total one abnormal E (β gene mutation) and one absent (0) or defective ($^+$) β gene in both chromosomes e.g. β^E/β^0 or β^E/β^+	Variable symptoms, moderate to severe anaemia, greater decrease in MCV/MCH. Thalassaemia intermedia or major

NB. This list is a guide only; there are a wide number of varieties of gene mutations and deletions.. Guidance should always be obtained from Haematology in the first instance, particularly in women with coinheritance of haemoglobin disease. E.g. α thalassaemia and Hb E.

4. Effect of Sickle Cell in Pregnancy

Table 4 Effect of Sickle Cell in pregnancy^{1,2,3,4,5,6,7}

SICKLE STATUS	GENOTYPE	EFFECT
Sickle cell trait	One normal β gene in one chromosome and one abnormal S (β gene mutation) in the other chromosome e.g. β/β^S	Asymptomatic normally, normal RBC indices. Sickle cell formation can occur in during high fever and significant hypoxia
Sickle cell disease/anaemia	Two abnormal S (β gene mutations), one in each chromosome B^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; there are a wide number of varieties of gene mutations and deletions. Guidance should always be obtained from Haematology in the first instance, particularly in women with coinheritance of haemoglobin disease. E.g. β thalassaemia and Hb S.

5. Screening Women for Haemoglobin Disorders

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

5.1 Screening and Referral Process

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

- If the woman is of Black African/African Caribbean/African American, Black British origin (irrespective of red cell indices value)
- Past history of unexplained anaemia
- Family history of anaemia (unknown cause) or haemoglobinopathy

- If the family originates from a geographical location which puts them at risk of haemoglobin disorders (see section 2 above) and show a MCV \leq 80fL and MCH \leq 27 pg

5.2 Women with no identified risk factors for haemoglobinopathy

Assess the FBP (full blood picture) and ferritin levels (if done) at the booking visit.

- Normal FBP – reassess FBP at 28/40, when the diabetes screening is ordered
- Abnormal FBP i.e. MCV \leq 80fL and the MCH \leq 27pg check ferritin level
- Treat underlying iron deficiency if ferritin $<$ 30ug/L and reassess FBP once iron deficiency is corrected.

http://www.kemh.health.wa.gov.au/development/manuals/O&G_guidelines/section_b/2/b2.23.pdf

5.3 Women who are at risk of Haemoglobinopathy Disorders

1. Assess family of origin
2. Assess FBP and ferritin levels
3. Confirm if the woman had previous haemoglobinopathy studies undertaken before through General Practitioner or in previous pregnancies. If so, obtain a copy and file the results. If the testing was obtained outside Australia; or the results are not available then testing should be repeated.
4. If the woman and partner have been screened in previous pregnancies have an identified haemoglobin disorder, an assessment should be made at booking to ascertain if the same partner is the father of the current pregnancy. In the case of a new partner, then partner studies will be required (see section 5.4 below).
5. Assess if partner testing has been undertaken before undertaken before through General Practitioner or in previous pregnancies and confirm status by obtaining a copy of the results which should be clearly identified as “partner studies” and placed in the woman’s medical records.
6. If no testing undertaken or results available, provide counselling and with maternal consent arrange **haemoglobinopathy studies on the woman only**. Testing should be requested as “FBP, ferritin and haemoglobin studies”. The request form should also include the woman’s family of origin, to direct testing.
7. Women should be directed to have the testing undertaken at a PathWest Laboratory (as they undertake a full haemoglobinopathy screen including DNA analysis).
8. If ferritin levels are $<$ 30ug/L initiate treatment for iron deficiency. http://www.kemh.health.wa.gov.au/development/manuals/O&G_guidelines/sectionb/2/b2.23.pdf (IV iron should not be given until a blood sample has been obtained for haemoglobin studies, as the IV iron induced elevated ferritin levels will interfere with the results).
9. If the woman is attending a low risk midwives clinic and the haemoglobin studies are abnormal, the woman should be referred to the Team Obstetric clinic for follow-up and counselling about the results. If the haemoglobin

studies results are normal the women may then continue antenatal care at the midwives clinic.

10. If the woman's results identify a risk of a significant haemoglobinopathy which places the fetus at risk, partner studies should be urgently undertaken (See section 5.4 below). Arrange for the woman to be reviewed in the obstetric clinic.
11. If test results confirm a high risk haemoglobinopathy condition impacting on pregnancy arrange referral to Maternal Fetal Medicine for multidisciplinary care. This may also include genetic counselling, physician and haematologist involvement.

5.4 Obtaining Partner Haemoglobin Studies

1. Ideally partner screening is obtained preconception, so the parents can make an informed decision about the risks prior to pregnancy. If this has not been done, in order to optimise the screening process, then the maternal results are used to direct the partner testing. Partner testing is only initiated concurrently when you are specifically directed by the Maternal Fetal Medicine Service or Haematology Department.
2. Provide counselling and with maternal/paternal consent arrange haemoglobinopathy studies on the partner. Testing should be requested as "FBP, ferritin and haemoglobin studies". The request form should be completed with the partner's demographic details, and annotated with his family of origin (The partner will be registered with a UMRN when he attends a PathWest Laboratory for testing). In the clinical notes section include the woman's details and haemoglobin genotype (i.e. $\alpha\alpha$ /--) and clearly identify "**Partner testing**".
3. Partners should be directed to have the testing undertaken at a PathWest Laboratory (as they undertake a full haemoglobinopathy screen including DNA analysis).
4. When initiating partner screening, complete a MR036 'Referral for Haemoglobinopathy Screening' form, include a copy of the maternal results and forward to the Maternal Fetal Medicine (MFM) Service. This will alert MFM/Haematology paternal testing is being undertaken and an assessment of risk to the fetus will be made when the results are available (See section 5.5 below).

5.5 Assessing the Fetus at Risk

Partner testing will identify if the fetus is at risk, and is dependent upon the maternal and paternal genotype. Assessment of the risk is undertaken by the Fetal Medicine Service/Haematology Department only.

1. If the **fetus is identified as high risk for haemoglobin disease** on review of the results by Haematology, then the MFM Service should be contacted immediately and provided with a copy of the paternal results. A MR036 'Referral for Haemoglobinopathy Screening form' should have been completed when the paternal studies were requested.

2. MFM will review the results and MR036 and arrange for the woman to attend KEMH for assessment as soon as possible and referral to Genetic Services if required.
3. An individual management plan will be developed and documented in the medical records between MFM and the woman/partner. This may include invasive testing, specialised ultrasonography and family testing. A Neonatal Management Plan may also be developed 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.
4. The haemoglobinopathy screen sticker is completed by the MFM or Haematology Consultant and placed on the MR004.

5. If the **fetus is identified as not at risk for haemoglobin disease** on review of the results by Haematology. Haematology/ MFM will review the results and MR036.
6. The haemoglobinopathy screen sticker is completed by MFM or Haematology and placed on the MR004.

5.6 Women and current partners who have been screened in previous pregnancies

If women and their current partners have been screened previously within Australia and results are available for both high performance liquid chromatography (HPLC) and DNA analysis, then 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, then re-testing is required. See section 5.3, 5.4 and 5.5 above, for maternal and paternal testing as required. If the woman has previously confirmation of a haemoglobin disorder and presents with a new partner, partner testing should be initiated.

5.7 Partners who decline 'paternal' testing/partner no longer in contact with woman or unknown

In a woman with a confirmed haemoglobin disorder, screening is aimed to identify the fetus at risk and enable informed choice regarding management of an affected infant; and thus partners should be encouraged to participate in screening. However if after careful discussion the partner continues to decline screening then it should be clearly documented in the woman's medical records, and the process below followed:

1. Complete the MR036 'Referral for Haemoglobinopathy Screening' form, include a copy of the maternal results and forward to the Maternal Fetal

- Medicine (MFM) Service. Clearly identify on the form that the partner is declining screening/no longer available or unknown,
2. MFM/Haematology will review the results and the MR036. The fetus will be identified as at risk for haemoglobin disease. MFM may arrange for the woman to attend KEMH for assessment as soon as possible and referral to Genetic Services if required (dependent upon the maternal genotype).
 3. An individual management plan will be developed and documented in the medical records between MFM and the woman. This may include invasive testing, specialised ultrasonography and family testing. A Neonatal Management Plan will be developed 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.
 4. The haemoglobinopathy screen sticker is completed by MFM or Haematology placed on the MR004.

5.8 Women referred to KEMH for Haemoglobinopathy Screening

Women are frequently referred to KEMH for assessment of fetal risk in which the woman has a confirmed or suspected haemoglobin disorder. The screening process using steps 5.3 to 5.7 is followed to ascertain the risk to the fetus. Not all women will require delivery and care at KEMH, although women will be required to attend at least one hospital appointment for midwifery and medical review.

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

5.9 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, which will be dependent upon age and child bearing potential. If the woman is likely to become pregnant, then steps should be taken to follow up results obtain partner testing if appropriate (as identified above). If the woman will no longer be attending KEMH for follow-up, then the woman's GP should be contacted by letter with the results and instructed to undertake partner testing if the woman has child bearing potential.

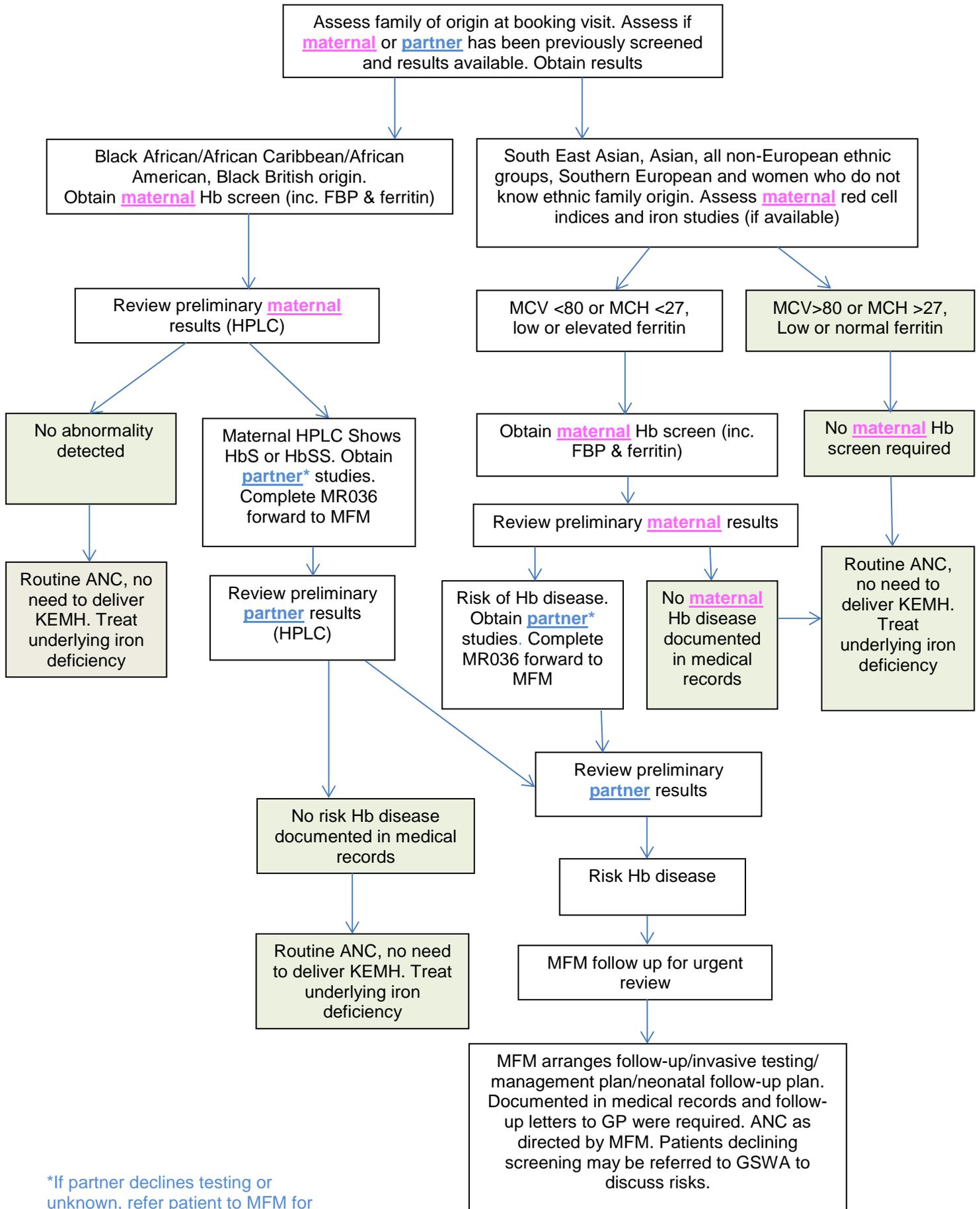
6. Management of Co-Existing Iron Deficiency

It is important to monitor ferritin levels in pregnant women with a confirmed haemoglobin disorder at booking and at 34/40 gestation and treat any underlying iron deficiency as per current guidelines.

http://www.kemh.health.wa.gov.au/development/manuals/O&G_guidelines/sectionb/2/b2.23.pdf

Women may often present at booking with an elevated ferritin level; and as the pregnancy progresses, they develop iron deficiency. As these women have an underlying microcytosis⁵, it can be difficult to evaluate changes in the red cell indices due to iron deficiency and treatment therein, thus the most reliable marker to assess iron deficiency are trends in ferritin 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



*If partner declines testing or unknown, refer patient to MFM for urgent review

Significant maternal haemoglobinopathies requiring referral and obstetric management by the Department of Fetal Medicine, King Edward Memorial Hospital

Pregnant women with confirmed significant haemoglobin disease should have their obstetric care managed through the Department of Maternal Fetal Medicine (MFM) where a multidisciplinary approach is used in collaboration with the Obstetricians, Haematologists, Neonatologists, Genetic Counsellors and Pathologists (Laboratory testing).

The following table identifies the women with confirmed significant haemoglobinopathy who require immediate referral to the Maternal Fetal Medicine 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. Hb SS, Hb SC, Hb SD ^{Punjab} , HbS/ β thalassaemia etc.
Maternal β thalassaemia intermedia, β thalassaemia major
Maternal Hb E/ β thalassaemia
Maternal Hb H (--/-a)
Pregnancies with a known 1: 4 risk of significant haemoglobinopathy in the fetus following review of results of maternal and paternal haemoglobinopathy screening*

*Pregnancies/fetus defined as not at risk do not require tertiary level obstetric care

No Fetal Risk of Fetal Hb Disease; sign of by the CNC Haematology

Aim

To provide the circumstances when the CNC Haematology may acknowledge results without medical review.

Key Points

- The tables below identifies the situations where the Clinical Nurse Consultant Haematology at KEMH may acknowledge results without medical review.
- * denotes that CNC sign off of “No Fetal Risk’ will only occur provided there are no other co inherited haemoglobin abnormalities and is dependent upon the number / type of α and or β gene deletions / mutations in the case of maternal and paternal α and or β thalassaemia traits
- All other Hb abnormalities not on this list must be reviewed by the Maternal Fetal Medicine team as they will have an identified or increased risk of Hb disease which requires multidisciplinary assessment

Table 1 Confirmed maternal alpha haemoglobin disorders and paternal status

		Paternal status									
		Not a carrier	α -thal trait α^+ (a-la-)or (aa-la-)	α -thal trait α^0 (aal-)	Hb H disease	β -thal trait	β -thal int./maj	Hb E trait	Hb E \pm β thal	Hb S trait	Hb S disease
Maternal Status	Not a carrier		CNC	CNC	CNC	CNC	CNC	CNC	CNC	CNC	CNC
	α -thal trait α^+ (a-la-)or (aa-la-)	CNC	CNC*	MFM	MFM	CNC*	CNC*	CNC*	CNC*	CNC*	CNC*
	α -thal trait α^0 (aal-)	CNC	MFM	MFM	MFM	CNC*	CNC*	CNC*	CNC*	CNC*	CNC*
	Hb H disease	CNC	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM

Table 2 Maternal beta haemoglobin disorders and paternal status

		Paternal status									
		Not a carrier	α -thal trait (a-la-)or (aa-la-)	α -thal trait (aal-)	Hb H disease	β -thal trait	β -thal int./maj	Hb E trait	Hb E \pm β thal	Hb S trait	Hb S disease
Maternal Status	Not a carrier		CNC	CNC	CNC	CNC	CNC	CNC	CNC	CNC	CNC
	β -thal trait	CNC	CNC*	CNC*	CNC*	MFM	MFM	MFM	MFM	MFM	MFM
	Hb E trait	CNC	CNC*	CNC*	CNC*	MFM	MFM	MFM	MFM	MFM	MFM
	Hb S trait	CNC	CNC*	CNC	CNC*	MFM	MFM	MFM	MFM	MFM	MFM
	β -thal inter./major	CNC	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM
	Hb E disease	CNC	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM
	Hb E/ β -thal	CNC	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM
	Hb S disease	CNC	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM	MFM

References and resources

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Related policies

Related WNHS policies, procedures and guidelines

Keywords:	Fetal risk, Hb disease, haematology trait, haemaglobinopathy screening in pregnancy		
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