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Related Trust Policies (to be read in conjunction with)	(Refer to the main body of the text) 04071 Standard Infection Prevention 04072 Hand Hygiene 06036 Guideline for Maternity Record Keeping including Documentation in Handheld Records 08014 Management of women requiring antenatal thromboprophylaxis

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2.0	Dr C. Anita-Rao		October 2013
2.1	Dr C. Anita-Rao	Clarification to points 17, 18 and Appendix D	20 November 2015
3.0	Miss Rao	Clarification to points - 3, 5, 9, 10, 11, Appendices D and G.	20 February 2017
3.1	Miss Rao and Dr Shetty		4 June 2017
4.0	Miss Rao	Full Review	16th January 2020

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1.0 Purpose

1.1 This guideline provides guidance in the assessment and management of hyperemesis gravidarum to provide a consistently high standard of care.

2.0 Equality and Diversity

2.1 Mid Essex Hospital Services NHS Trust is committed to the provision of a service that is fair, accessible and meets the needs of all individuals.
(Refer to Appendix 1)

3.0 Definition

3.1 Nausea and vomiting during pregnancy are extremely common, affecting up to 80% of pregnant women. Hyperemesis gravidarum is severe, intractable nausea and vomiting affecting 0.3 -3.6% of pregnancies. Recurrence rate of hyperemesis gravidarum ranges from 15.2% to 81%.

3.2 The onset of symptoms is usually early in the first trimester at around 4-6 weeks gestation and peaks at 9 weeks gestation. Vomiting abates in 90 % of cases by 20 weeks. They may persist beyond 20 weeks in 13% of cases.

3.3 Hyperemesis gravidarum (HG) is inconsistently defined. It is typically characterised by severe nausea and vomiting that causes dehydration and imbalance of fluid and electrolyte, disturbs nutritional intake and metabolism, causes physical and psychological debilitation with loss of >5% pre-pregnancy weight, and often necessitates hospital admission.

4.0 Pathophysiology

4.1 HG is poorly understood and has a complex multifactorial aetiology. HG appears to occur as a complex interaction of biological, psychological, and sociocultural factors.

4.2 **Human chorionic gonadotrophin:** Prospective studies reported that there was significantly higher level of serum hCG in hyperemesis patients than in controls.

4.3 It is postulated that hCG causes hyperemesis via stimulating effect on secretory process in the upper gastrointestinal tract.

4.4 **Oestrogen**: One study reported a positive association between nausea and vomiting and maternal serum E2 level.

4.5 It has been proposed that elevated maternal serum steroid hormone causes a decrease in intestinal motility and gastric emptying. This in turn alters gastrointestinal pH and encourages the development of subclinical helicobacter pylori infection.

4.6 **Thyroid hormone:** In early pregnancy physiological stimulation of thyroid gland occasionally lead to gestational transient thyrotoxicosis.

4.7 Biochemical hyperthyroidism (raised free thyroxine and/or suppressed thyroid stimulating hormone (TSH) may be found in 66% of hyperemesis gravidarum.

4.8 Abnormal thyroid function test do not require treatment with anti-thyroid drugs and resolve as the hyperemesis improves.

4.9 True thyrotoxicosis may present in early pregnancy. Discriminatory features to distinguish this from a gestational syndrome include the presence of tremor, exophthalmos, goitre with bruit and the presence of thyroid stimulating antibodies.

4.10 **Subclinical helicobacter pylori infection**: One study using histological examination of mucosal biopsy reported that 95% of all hyperemesis patients tested positive for subclinical helicobacter pylori infection compared with 50% in the control group.

4.11 **Psychosocial factors**: Various psychological stresses have been linked with hyperemesis, including emotional immaturity, strong mother-dependence, anxiety and tension related to pregnancy, and resentment towards her unwanted pregnancy.

4.12 However, more recent investigations argue that the psychological symptoms are the result of stress arising from physical burden of hyperemesis rather than a cause.

5.0 Diagnosis

5.1 Diagnosis of HG is made clinically after exclusion of other causes. Vomiting refractory to treatment and new symptoms appearing after 12 weeks should not be attributed to hyperemesis gravidarum.

5.2 **Clinical features:** Onset of symptoms usually occurs between 4 and 10 weeks.

Symptoms:	**Signs:**
Nausea	Dehydration
Vomiting	Weight loss (≥5% of pre-pregnancy)
Spitting	Ketonuria
Enhanced olfactory senses	Anaemia
Food and/or fluid intolerance	Tachycardia
Lethargy	

5.3 **Investigations**

5.3.1 Aim

- to establish the severity of hyperemesis and associated electrolyte derangement;
- to exclude other causes of nausea and vomiting;

5.3.2 Initial investigations:

- Urea and electrolytes;
- Liver function test and Amylase;
- Full blood count;
- Urinalysis and mid stream urine;
- ABG (in severe cases to rule out metabolic acidosis);
- Early ultrasound scan to refer on multiple or molar pregnancies which will increase the incidence of HG.

5.3.3 Additional investigations:

- Calcium;
- Blood glucose;
- Thyroid function test.

6.0 Differential Diagnosis

6.1 Coexisting pathology that should be considered in women with HG, if it is not responding to supportive treatment. (Refer to Appendix A).

7.0 Complications of HG

7.1 Maternal complications :

- Hypokalemia causes lethargy, skeletal muscle weakness and cardiac arrhythmia;
- Hyponatremia and central pontine myelinolysis ;
  (Refer to Appendix B)
- Wernickie's encephalopathy ;
  (Refer to Appendix C)
- Vitamin B6/B12 deficiency causes anaemia and peripheral neuropathy;
- Malnutrition ;
- Mallory-Weiss oesophageal tears ;
- Venous thromboembolism ;
- Psychological morbidity.

7.2 Fetal complications ;

- No increased risk of congenital malformations;
- Growth restriction;
- Wernicke's encephalopathy is associated with a 40% incidence of fetal death.

8.0 Management of HG

8.1 In a systematic review, seven RCT testing different methods of treatment in hyperemesis, there was no treatment shown to be of benefit.

8.2 Therefore, the management is based on:
(Refer to Appendix D)

- Correction of dehydration and electrolyte imbalance;
- Prophylaxis against recognised complications;
- Provision of symptomatic relief.

8.3 Admit to hospital if:

- Symptoms are severe despite 24 hours of medication;
- There is evidence of dehydration and the patient is ketotic;
- A lower threshold for admitting to hospital if the patient has co-existing condition (i.e. diabetes) which can be adversely affected by nausea and vomiting.

9.0 Fluid Therapy

9.1 Maintaining hydration is crucial in managing hyperemesis. Patients who are unable to tolerate oral fluid and who are ketotic should receive intravenous fluid and electrolyte replacement.

9.2 Sodium chloride 0.9% with 20-40mmol KCl per litre and Hartmann's solution are recommended.

9.3 Ready prepared bags of intravenous fluid, including potassium chloride (KCl) can be prescribed according to patient's serum potassium level. (Refer to Appendix D).

9.4 Sodium chloride 0.9% contains Na+ 150 mmol/l; Hartmann's solution contains Na^+ 131mmol/l and K^+ 5 mmol/l.

9.5 Dextrose containing fluid should be avoided except in women with diabetes as neither 5% dextrose nor with specific concentrations that it refers to e.g. dextrose 5% sodium chloride 0.45% contain sufficient sodium to correct the commonly associated hyponatraemia. Dextrose is contraindicated if no thiamine is given prior.

9.6 High concentration of dextrose may precipitate Wernicke's encephalopathy.

9.7 Double strength saline should be avoided, even in case of severe hyponatremia.

10.0 Vitamin Supplementation

10.1 Thiamine Hydrochloride oral 100mgs 12hrly.

10.2 Pabrinex 2 pairs of ampoules given intravenously diluted in 100ml 0.9% sodium chloride over 30-60 minutes t.d.s (if patient is unable to tolerate oral thiamine).

11.0 Antiemetic Therapy

11.1 There are substantial data from systematic reviews and cohort studies to support the safety of antiemetic in pregnancy and no sign of teratogenicity of drugs studied. The NICE guidelines recommend the use of antiemetic therapy in the treatment of nausea and vomiting in pregnancy.

11.2 They concluded that patients using antiemetics have a better pregnancy outcome than other patients. This may reflect better nutritional status. Antiemetics may be used liberally and safely in pregnancy.

11.3 Patients with severe hyperemesis may require regular parenteral doses of more than one antiemetic to control symptoms.

11.4 Pharmacological group of antiemetics.
(Refer to Appendix F)

11.5 Ondensetron can be used if hyperemesis is not responding to above antiemesis after discussion with obstetric registrar/consultant on call. Ondansetron 4 mg, 8 hourly orally, intramuscularly (IM) or intravenously (IV).

11.6 Side effects of antiemetics include drowsiness, particularly with phenothiazine and extra pyramidal effects and oculogyric crisis, particularly with metoclopramide and phenothiazines.

11.7 Metoclopramide is not suitable for younger patients (younger than 20 years of age) as acute dystonic reactions involving facial and skeletal muscle spasms and oculogyric crisis are common in younger people. Metoclopramide should be given for a maximum 5 days.

11.8 These reactions generally occur within few days of starting the treatment and subside within 24 hours of stopping metoclopramide. Procyclidine 5 -10 mg IV or IM will abort dystonic attack.

11.9 Prochlorperazine may cause drowsiness but is less sedating than cyclizine and promethiazine. The degree of sedation varies among individuals and depends on the dose given. The person should avoid driving or performing skilled tasks (e.g. driving).

12.0 Corticosteroid Treatment

12.1 Steroids should be used for intractable hyperemesis which is not responding to above management, after discussion with consultant.

12.2 Steroid should be accompanied by histamine receptor antagonist or proton pump inhibitor to counteract gastric effect of steroid.

12.3 Hydrocortisone 100 mg intravenously, twice daily (BD) for 48 hours until the patient is able to tolerate tablets.

12.4 It is followed by prednisolone 30 – 40 mg daily in a single or divided dose. The response is usually rapid.

12.5 Steroids should be discontinued if there is no effect after 2 days.

12.6 Suggested corticosteroid regime.
(Refer to Appendix H)

13.0 Thromboprophylaxis

13.1 Hyperemesis is associated with increased risk of venous thromboembolism (VTE) due to dehydration and immobilisation if patients are needed to be hospitalised. Many antenatal VTE events (including fatal) occurs in the first trimester.
(Refer to the guideline for the 'Management of patients requiring antenatal thromboprohylaxis'; register number 08014)

13.2 Enoxaparin should be given if the risk factor score for VTE is 3 or more.
(Refer to Appendix E)

14.0 Nutrition Advice

14.1 Nil by mouth or suck ice cubes for the first 24 hours until anti-emetics are effective.

14.2 Afterwards, small and frequent dry meals are usually tolerable. Drinking small amount of fluid regularly is important to maintain hydration.

14.3 Eating before getting out of bed and at times when nausea is less severe may reduce severity of nausea and vomiting.

14.4 Try sipping on herbal teas containing ginger, lemon or peppermint.

14.5 A recent trial showed that ginger was therapeutically equivalent to vitamin B6 for improving nausea, dry retching, and vomiting. It also reported no major adverse effect to ginger. All of the trials used at least 1gm of ginger per day as a treatment. The NICE guidelines recommended the use of ginger as an effective means of reducing symptoms of nausea and vomiting.

15.0 Acupuncture/ Acupressure

15.1 Acupressure is the application of pressure at acupuncture site without the use of needles. The Neiguan point or P6 is on the volar aspect of the wrist.

15.2 The NICE guidelines recommend the non pharmacological treatment of P6 (wrist) acupressure to be effective in reducing the symptoms of nausea and vomiting.

16.0 Management Options for Extremely Severe Hyperemesis Gravidarum

16.1 In cases that fail to respond to all of the above therapies:

- Enteral nutrition;
- Total parenteral nutrition (TPN);
- Termination of pregnancy.

17.0 Enteral Nutrition

17.1 The cost of enteral nutrition is considerably less than that of TPN and it is safer.

17.2 It is successful in patients with meal related nausea and vomiting only.

17.3 But enteral hyperalimentation may be poorly tolerated due to nausea and vomiting and may be even contraindicated because of the risk of aspiration.

17.4 To minimize the risk of aspiration, a nasojejunal feeding tube may be placed beyond the pylorus, but this necessitates radiation exposure for correct position of tube or insertion under endoscopic guidance. Patients should be allowed to make an informed decision given the risks, benefits and alternatives of any invasive procedure aimed at improving nutrition. Discussion with the gastroenterology and / or nutrition team is recommended.

17.5 Enteral nutrition is the management for hyperemesis in the first trimester and early second trimester.

18.0 Total Parenteral Nutrition (TPN)

18.1 It is very rarely necessary. Because of substantial metabolic, infectious and thrombotic risks, it should be regarded as a measure of last resort. Discussion with a tertiary nutrition centre should be considered

18.2 Because TPN involves the use of high concentration of glucose, thiamine supplementation is mandatory.

19.0 Recurrence Risk of Hyperemesis Gravidarum

19.1 The risk of recurrence in subsequent pregnancy is 15.2 % to 81%. The risk is reduced by a change of paternity.

20.0 Staffing and Training

20.1 All midwifery and obstetric staff must attend yearly mandatory training which includes skills and drills training.

20.2 All midwifery and obstetric staff are to ensure that their knowledge and skills are up-to-date in order to complete their portfolio for appraisal.

21.0 Infection Prevention

21.1 All staff should ensure that they follow Trust guidelines on infection prevention, using Aseptic Non-Touch Technique (ANTT) when carrying out this procedure.

22.0 Audit and Monitoring

22.1 Audit of compliance with this guideline will be considered on an annual audit basis in accordance with the Clinical Audit Strategy and Policy (register number 08076), the Corporate Clinical Audit and Quality Improvement Project Plan and the Maternity annual audit work plan; to encompass national and local audit and clinical governance identifying key harm themes. The Women's and Children's Clinical Audit Group will identify a lead for the audit.

22.2 The findings of the audit will be reported to and approved by the Multi-disciplinary Risk Management Group (MRMG) and an action plan with named leads and timescales will be developed to address any identified deficiencies. Performance against the action plan will be monitored by this group at subsequent meetings.

22.3 The audit report will be reported to the monthly Directorate Governance

Meeting (DGM) and significant concerns relating to compliance will be entered on the local Risk Assurance Framework.

22.4 Key findings and learning points from the audit will be submitted to the Clinical Governance Group within the integrated learning report.

22.5 Key findings and learning points will be disseminated to relevant staff.

23.0 Guideline Management

23.1 As an integral part of the knowledge, skills framework, staff are appraised annually to ensure competency in computer skills and the ability to access the current approved guidelines via the Trust's intranet site.

24.0 Communication

24.1 A quarterly 'maternity newsletter' is issued and available to all staff including an update on the latest 'guidelines' information such as a list of newly approved guidelines for staff to acknowledge and familiarise themselves with and practice accordingly.

24.2 Approved guidelines are published monthly in the Trust's staff newsletter that is sent via email to all staff.

25.0 References

National Institute for Clinical Excellence (2008) Antenatal Care: Routine care for the healthy pregnant women. NICE: London; March.

Review on hyperemesis gravidarum, Best Practice & Research Clinical Gastroenterology; Vol 21, No.5, pp. 755-769, 2007

James D. Paauw and Alan T. Davis; Hyperemesis in pregnancy; Maternal-Fetal Nutrition during Pregnancy and Lactation; 2010; P 138 – 140

C. Nelson-Piercy, C. Williamson, Gastrointestinal and hepatic disorder, Maternal Medicine, Medical Problems in Pregnancy. 2007, P 172 –1276

Nikos A Kametas, Catherine Nelson-Piercy; Hyperemesis gravidarum, gastrointestinal and liver disease in pregnancy; Obstetrics, Gynaecology and Reproductive Medicine; Volume 18:3; P 69 – 70, March 2008

CKS safe practical clinical answers; www.cks.nhs.uk

RCOG (2016) Green-top Guideline No. 69.

Appendix A

Differential Diagnosis

System	Diagnosis	Investigation/Initial Assessment
Genitourinary	UTI Uraemia Molar pregnancy	MSU (mid-stream specimen of urine) U&E's (urea and electrolytes) Ultrasound of the uterus
Gastrointestinal	Gastritis/ peptic ulcer Reflux/ oesophagitis Pancreatitis Bowel obstruction	Helicobacter pylori antibody empirical PPI therapy or endoscopy amylase, blood glucose, calcium plain supine abdominal
Endocrine	Addison's disease Hyperthyroidism Diabetes ketoacidosis	U&E, early morning cortisol Short Synacthen test with ACTH Signs and symptoms TFTs (thyroid function test), thyroid autoantibodies blood glucose urine dipstick for ketones
CNS	Intracranial tumours Vestibular disease	CNS examination Brain imaging CNS examination

Appendix B

Hyponatremia and Central Pontine Myelinolysis

- Hyponatremia (plasma sodium <120 mmol/l) may present with anorexia, headache, nausea, vomiting and lethargy

- More pronounced hyponatremia may lead to central pontine myelinolysis (osmotic demyeliniation)

- It is a rare complication of HG and may result from severe hyponatremia or from over rapid correction of hyponatremia

- It can cause pyramidal tract signs, spastic quadriparesis, pseudobulbar palsy and impaired consciousness. It may result in personality change, muscle cramps and weakness, confusion, ataxia, drowsiness, diminished reflexes, and convulsion

- Serious symptomatic hyponatremia is medical emergency and should be managed appropriately by skilled personnel, since the treatment may be potentially as dangerous as the condition itself

Appendix C

Wernicke's Encephalopathy

- It results from vitamin B1 deficiency and is precipitated by intravenous fluid containing high concentration of dextrose

- It is characterised by ophthalmoplegia (typically sixth nerve palsy, diploia), ataxia and confusion

- Diagnosis of Wernicke's encephalopathy is clinical and may be confirmed by the finding of a low red cell transketolase, a thiamine dependent enzyme

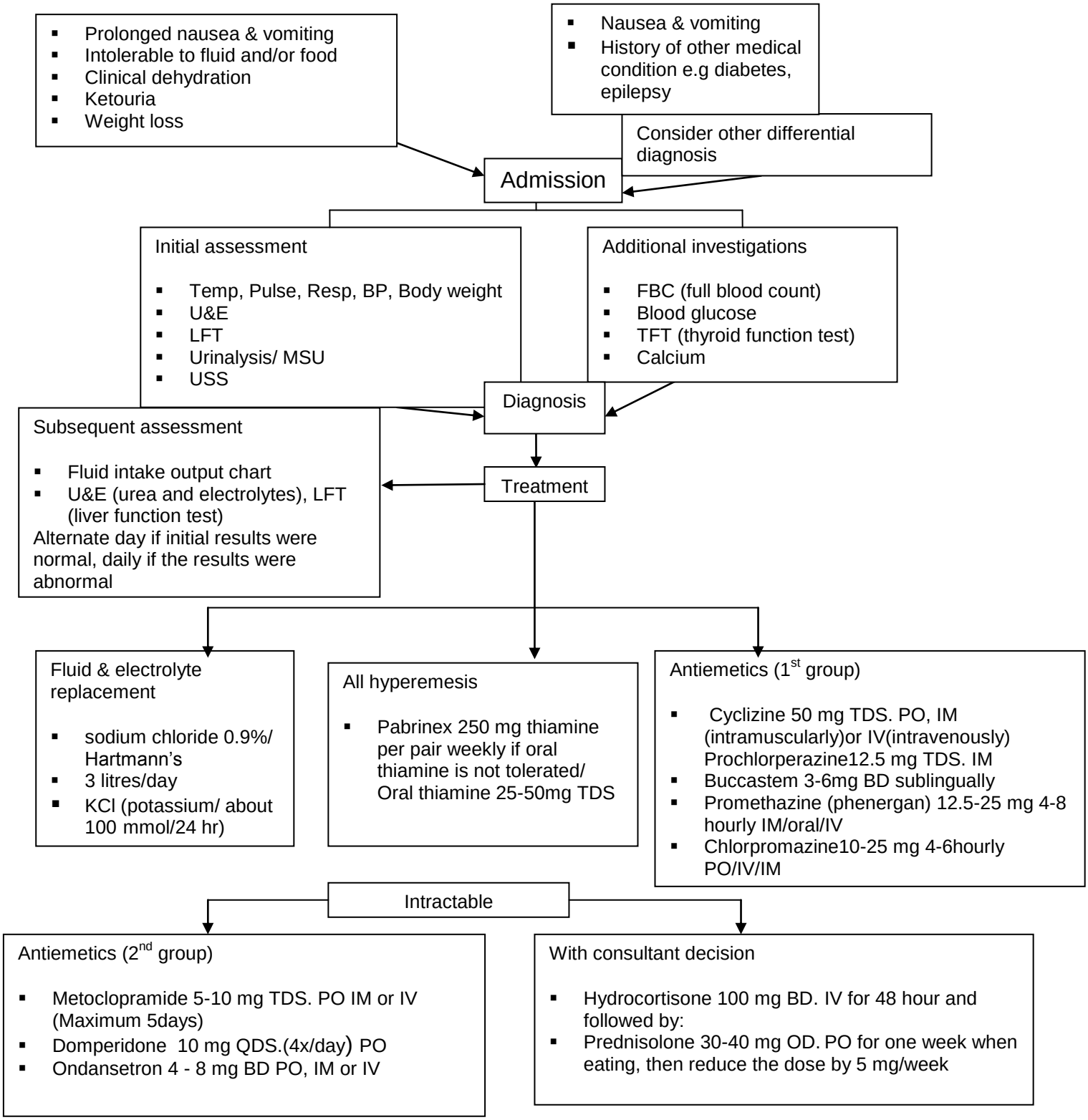
- MRI may reveal symmetrical lesion around the aqueduct and fourth ventricle, which resolve after treatment with thiamine

- Although thiamine replacement may improve the symptoms of Wernicke's encephalopathy, if Korsakoff psychosis develops manifest by retrograde amnesia, impaired ability to learn and confabulation, the recovery rate is only about 50%

- Wernicke's encephalopathy is associated with a 40% incidence of fetal death

Appendix D

Summary for Management of Hyperemesis Gravidarum



Other supportive treatment

- Diet & lifestyle (small frequent dry meal, learn to avoid certain scents which make the patient intolerable
- Ginger
- Acupressure/acupuncture

The options for severe hyperemesis who failed to response to above measures

- enteral nutrition to discuss with gastroenterologist
- parenteral nutrition (TPN) to discuss with gastroenterologist at tertiary level
- termination of pregnancy psychiatric opinion to be sort

Appendix E

Risk assessment for Venous Thromboembolism (VTE)

Pre-existing risk factors	Tick	Score
Previous recurrent VTE		3
Previous unprovoked or estrogen related		3
Previous VTE provoked		2
Family history of VTE		1
Known thrombiphilia		2
Medical comorbidity		2
Age (> 35 years)		1
Obesity		1 or 2 *
Parity (≥ 3)		1
Smoker		1
Gross varicose vein		1
Obstetric risk factors		1
Pre-eclampsia		1
Dehydration/ **Hyperemesis**/ OHSS		1
Multiple pregnancy or ART		1
Transient risk factors		
Current systemic infection		1
Immobility		1
Surgical procedure in pregnancy		2
Total score		

*Score 1 for BMI >30

*Score 2 for BMI >40

Appendix F

Pharmacological Group of Antiemetics

Phenothiazine	Prochlorperazine (stemetil/ buccastem) Chlorpromazine
Dopamine antagonists	Metoclopramide Domperidone (motilium)
5-HT3 (serotonin) antagonist	Ondansetron
Antihistamines (H1 receptor antagonist)	Cyclizine Promethazine (phenergan) Meclozine

Appendix G

Recommended Antiemetic Regime

Group One		**Dose**	**Route**
First line	Cyclizine	50 mg TDS.	PO, IM or IV
Second line	Prochlorperazine (Stemetil) Buccastem	12.5 mg TDS. 3-6 mg BD	IM sublingual
Third line	Promethazine (phenergan) Chlorpromazine	12.5 - 25 mg, 4-8hourly 10-25 mg 4 – 6 hourly	IM/oral PO/IV/IM
Group two:			
	Metoclopramide	5 – 10mg 8 hourly	PO/ IV/ IM (Max 5days)
	Domperidone	10mg 8 hourly	PO
	Ondansetrone	4-8mg 6-8hourly 8mg over 15mins, 12hourly	PO IV

Appendix H

Suggested Corticosteroid Regime

Hydrocortisone	100 mg B.D	IV for 48 hour and followed by
Prednisolone	30-40 mg daily Dose (mg) 30 25 20 15 10 5 Stop	Orally for one week when eating, then reduce the dose by 5 mg per week as follows: Duration (days) 7 7 7 7 7 7

Appendix 1: Preliminary Equality Analysis

This assessment relates to: Hyperemesis Gravidarum in Pregnancy/10099

A change in a service to patients		A change to an existing policy	**X**	A change to the way staff work	
A new policy		Something else (please give details)			

Questions	Answers
1. What are you proposing to change?	Full Review
2. Why are you making this change? (What will the change achieve?)	3 year review
3. Who benefits from this change and how?	Patients and clinicians
4. **Is anyone likely to suffer any negative impact as a result of this change?** If **no**, please record reasons here and sign and date this assessment. If **yes**, please complete a full EIA.	No
5. a) Will you be undertaking any consultation as part of this change? **b) If so, with whom?**	Refer to pages 1 and 2

Preliminary analysis completed by:

Name	Anita Rao	**Job Title**	Obstetrics and Gynaecology Consultant	**Date**	December 2019