Table 4

Maternal and fetal complications secondary to hyperemesis gravidarum

Maternal complications

Electrolyte imbalance

- Hyponatraemia: (lethargy, headache, confusion and seizures). Quick and exaggerated correction of hyponatraemia can lead to central pontine myelinolysis[6]
- Hypokalaemia (muscle weakness and cardiac arrhythmias). Measure potassium and do an ECG (inverted T wave, ST segment depression and prominent U wave). Measure magnesium levels to rule out deficiency as a contributing factor for hypokalaemia
- Hypochloraemia
- Hypercalcaemia or hypocalcaemia (Chvostek's or Trousseau's sign)

Nutritional deficiencies

- Vitamin B1 (thiamine) deficiency can lead to Wernicke's encephalopathy. It is important to note that dextrose-containing solutions can precipitate Wernicke's encephalopathy in thiamine-deficient states
- Vitamin B12 (cobalamin) and vitamin B6 (pyridoxine) deficiency can cause anaemia and peripheral neuropathies
- Folic acid

Metabolic disturbances

- Metabolic hypochloraemic alkalosis
- Transient biochemical thyrotoxicosis. Resolves as the hyperemesis gravidarum improves and treatment with antithyroid drugs is not usually indicated.[1,3] Present in up to 60% of women with hyperemesis gravidarum[20]
- Acute kidney injury
- Liver function tests may be abnormal in up to 40% of women with hyperemesis gravidarum, mostly transaminitis

Gastrointestinal complications

Oesophageal reflux disease, oesophagitis or gastritis, Mallory-Weiss tear of the oesophagus

Other

Depression,[21,22] anxiety and thromboembolism

Fetal complications[23]

It is suggested that the increased risk of fetal complications is probably due to the low pregnancy weight gain (< 7 kg during the pregnancy) and nutritional deficiencies. Complications include:

- Fetal growth restriction
- Prematurity
- Low birthweight
- Low 5 min Apgar scores
- Anomalies of the central nervous system and skeletal malformations