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Gastrointestinal and Liver Disease in Pregnancy

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primary biliary sclerosis
Budd–Chiari syndrome
Wilson disease
liver transplantation

This chapter on the gastrointestinal and hepatic systems in pregnancy focusses on those conditions that are frequent and troublesome (gastro-oesophageal reflux and constipation), distressing (hyperemesis gravidarum) or potentially fatal (obstetric cholestasis, acute fatty liver of pregnancy and HELLP (haemolysis, elevated liver enzymes, low platelets) syndrome). It also highlights the clinical challenge obstetricians may face in managing rare conditions such as the Budd–Chiari syndrome, liver transplantation, primary biliary cirrhosis and Wilson disease.

The clinical presentation of liver and gastrointestinal dysfunction in pregnancy is not specific, and certain 'abnormalities' may represent physiological changes of pregnancy. Diagnosis and management are often difficult because of atypical symptoms, a reluctance to use invasive investigations and concerns about the teratogenicity of the medications. The best available evidence to manage these conditions is discussed in the chapter.

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Gastrointestinal disease

Hyperemesis gravidarum

Nausea and vomiting are common in the first trimester of pregnancy and are usually self-limiting. It is estimated that 70–90% of pregnant women experience nausea and 50% have at least one episode of vomiting or retching. Most doctors and pregnant women are excessively cautious with anti-emetics during pregnancy especially in the first trimester and they are generally avoided, unless vomiting is very severe, due to the fear of teratogenicity.

Clinical features

Hyperemesis gravidarum (HG) occurs in around 0.1% of pregnancies and presents with severe and persistent nausea and vomiting leading to dehydration. Onset is usually in the first trimester (6–8 weeks' gestation) of pregnancy. In addition to nausea and vomiting, there may be ptyalism (inability to swallow saliva) and spitting. The persistent vomiting may also lead to postural hypotension, tachycardia, electrolyte disturbances, ketosis, muscle wasting and weight loss.

Pathogenesis

The pathophysiology of hyperemesis is poorly understood. It appears to have a complex metabolic background and a number of hormonal, mechanical and psychological factors have been implicated. Hormones such as human chorionic gonadotropin (HCG), low levels of prolactin and high levels of oestradiol have been implicated [1–3]. Recent reports have suggested an association between HG and *Helicobacter pylori* infection [4]. Some studies have shown increased concentrations of cell-free foetal deoxyribonucleic acid (DNA) and activation of natural killer and cytotoxic T-cells in women with hyperemesis [5,6]. A recent pilot study has shown a possible role of leptin and nesfatin-1 in the pathology of hyperemesis [7].

Diagnosis

HG is a diagnosis of exclusion with no single confirmatory test. Therefore, other causes of nausea and vomiting such as systemic infection, peptic ulceration, pancreatitis and, rarely, Addison's disease must be considered. Hyperemesis usually recurs (women with a history of HG, 15.2%, vs. without a history of HG, 0.7%) in subsequent pregnancies; hence, a previous history makes the diagnosis more likely [8].

Investigations

Laboratory investigations may reveal hyponatraemia, hypokalaemia, low urea, ketosis and a metabolic hypochloraemic alkalosis. Two-thirds of patients with HG may have abnormal thyroid function tests, which are more common in Asians than in Europeans [9,10]. These patients have raised free thyroxine (T4) and/or a suppressed thyroid-stimulating hormone (TSH) but are clinically euthyroid and do not require treatment.

Effect of HG on pregnancy

Maternal

Serious morbidity can result from inadequate or inappropriate treatment [11]. Death can result from aspiration of vomit or Wernicke's encephalopathy. The latter is due to vitamin B_1 (thiamine) deficiency and it has been reported in as many as 60% of HG patients [12].

Wernicke's encephalopathy is characterised by diplopia, abnormal ocular movements, ataxia and confusion. It may be precipitated by dextrose-containing fluids and total parenteral nutrition (TPN). The diagnosis may be confirmed by the finding of a low red-cell transketolase activity or a raised thiamine pyrophosphate effect. Magnetic resonance imaging (MRI) of the brain in acute Wernicke's encephalopathy may reveal symmetrical lesions around the aqueduct and the fourth ventricle. The recovery rate is only about 50%, if Korsakoff's psychosis has ensued.