Chapter 3

INTRAHEPATİC CHOLESTASIS OF PREGNANCY

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Intrahepatic cholestasis of pregnancy (ICP), characterised by pruritus and elevated serum bile acid concentrations, typically develops in the late second and/or third trimester and is associated with increased rates of adverse fetal outcomes. The etiology of cholestasis is poorly understood, and management is difficult due to limited data on diagnosis, treatment, and associated adverse outcomes. It is thought to result from the cholestatic effects of reproductive hormones and their metabolites in genetically susceptible women and resolves rapidly after delivery. The mechanisms by which fetal complications occur are also unclear.

INTRODUCTION

Cholestasis, translated from the Greek, describes the "stoppage of bile". This disturbance of bile flow can occur at any site in the biliary system and can be caused by extrahepatic mechanical obstruction, pathology of the intrahepatic biliary tree, or dysfunction of individual hepatocytes. Usually, the disorder does not result in a complete interruption of bile flow, and often only certain components of bile become pathologic. The clinical and biochemical manifestations of cholestasis may therefore vary. Conventionally, cholestatic liver diseases are first divided into those with extrahepatic and those with intrahepatic etiology. Cholestasis in pregnancy is an intrahepatic disorder that occurs only during pregnancy and regresses rapidly after delivery (1). ICP was originally described by Ahlfeld in 1883 as recurrent jaundice in pregnancy which resolved after delivery. Pruritus was not mentioned in this report, but subsequent case reports published in the 1950s reported severe pruritus with or without jaundice associated with the condition, in addition to complete resolution after delivery and high recurrence rates in subsequent pregnancies (2).It is the most common pregnancy-specific liver disorder. The etiology of intrahepatic cholestasis in pregnancy (ICP) is poorly understood. It is likely to be a multifactorial disease, with genetic, environmental,

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- With peak bile acids 40 to 99 micromol/L and no other risk factors, the risk of stillbirth is similar to the background risk up to 38 to 39 weeks' gestation. Consider planned delivery at 38 to 39 weeks' gestation.
- For peak bile acids ≥100 micromol/L, the risk of stillbirth is higher than the background risk. Consider planned delivery at 35 to 36 weeks' gestation.
- The American College of Obstetricians and Gynecologist (ACOG)/Society for Maternal-Fetal Medicine (SMFM) recommends (40):
- For patients with total bile acid levels <100 micromol/L, delivery is recommended at 36/7 to 39/7 weeks' gestation or at diagnosis if diagnosed at >39/7 weeks.
- For patients with total bile acid levels ≥100 micromol/L, delivery is recommended at 36/7 weeks or at diagnosis if diagnosed later.

There are no special considerations for labour in patients with ICP. Continuous fetal monitoring during labour is indicated because of the increased incidence of fetal death and non-fatal asphyxia. Induction of labour does not necessarily increase the risk of caesarean section compared with expectant management. The risk of postpartum hemorrhage is not increased when ICP is treated with UDCA. Therefore, do not routinely assess coagulation parameters or prescribe vitamin K before delivery. In rare refractory cases, prothrombin time can be checked and vitamin K administered if it is prolonged (41, 42).

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