A190

POST-PARTUM PRIMARY BILIARY CHOLANGITIS (PBC) AFTER RESOLUTION OF INTRAHEPATIC CHOLESTASIS OF PREGNANCY (ICP) IN FIRST NATION'S PATIENTS OF BC: A CASE SERIES

D. Chahal[1], E.M. Yoshida[2], L. Arbour[3], J. Wallach[1]

1. Gastroenterology, University of British Columbia, Vancouver, BC, Canada; 2. Division of Gastroenterology, University of British Columbia, Vancouver, BC, Canada; 3. Medical Genetics, University of British Columbia, Victoria, BC, Canada

Background: PBC is a progressive cholestatic disease characterized by destruction of intrahepatic bile ducts, peri-portal inflammation and fibrosis. PBC is the leading indication for liver transplantation in First Nations of British Columbia. Diagnosis of PBC during pregnancy is difficult due to clinical overlap with ICP and pregnancy induced immune tolerance (4-6). We present 3 cases of clinically diagnosed ICP in First Nations women who were later diagnosed with PBC post-partum.
Aims: To investigate the potential relationship between ICP and PBC in the First Nations community of BC.
Methods: Retrospective review of relevant cases.
Results: Case 1: A 27-year-old woman with history of ICP at 31 weeks during her 3rd pregnancy and family history of PBC presented with ursodiol and cholestyramine responsive pruritis and jaundice 20 weeks into her 4th pregnancy. Bilirubin was 103 µmol/L, ALP 371 IU/L, ANA and AMA negative. Symptoms and biochemistry remained in remission after delivery at 33 weeks. Discontinuation of medications led to recurrent pruritis 4 months later. Bilirubin was 6 µmol/L, ALP 272 IU/L, GGT 153 IU/L and ALT 204 IU/L. Liver biopsy was consistent with PBC, F1. Pruritis has now been refractory to ursodiol, cholestyramine and rifampin.
Case 2: A 30-year-old woman with history of ICP at 20 weeks during 2 prior pregnancies presented with ursodiol responsive pruritis 20 weeks into her 3rd pregnancy. Symptoms and biochemistry remained in remission after delivery at 35 weeks. Post-partum discontinuation of ursodiol led to recurrent pruritis 2 months later. ALP was 876 and AMA >1:640. Re-initiation of ursodiol improved symptoms and biochemical abnormalities.
Case 3: A 30-year-old woman with family history of PBC (mother) presented with ursodiol responsive pruritis and jaundice 20 weeks into her 4th pregnancy. Symptoms and biochemistry remained in remission after delivery at 37 weeks. Post-partum discontinuation of ursodiol led to recurrence jaundice 4 months later. Bilirubin was 68, ALP 1279, total cholesterol 7.72, IgM 18.98, ANA >1:640 and AMA 1:320. Jaundice and biochemical abnormalities persisted despite re-initiation of ursodiol.

Obeticholic acid has been initiated.
Conclusions: First Nations communities of BC are disproportionately affected by PBC, due to both genetic and epigenetic phenomena. We present 3 patients who were diagnosed with ICP that resolved post-partum but with the subsequent development of PBC. The intrapartum cholestasis did not have clinical features of PBC during pregnancy. Although not previously reported, ICP may predispose to PBC in this specific community. It remains to be seen if there is a genetic association. Clinicians must remain suspicious of PBC during pregnancy in this population and ongoing monitoring in the post-partum period is paramount.

Funding Agencies: None