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Case Report

# Case Report : Intrahepatic Cholestasis of Pregnancy

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Abstract :
Intrahepatic cholestasis of pregnancy (ICP) is a cholestatic disorder characterized by pruritus, elevated serum aminotransferases and bile acid levels with gradual onset in the second or third trimester of pregnancy, and spontaneous relief of signs and symptoms within two to three weeks after delivery.

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## Introduction

In 1883, a case was reported for the first time, with unexplained pruritis associated with visible jaundice in a pregnant woman, during the last trimester of pregnancy. The symptoms persisted until delivery and resolved spontaneously afterwards. The disease remained unnamed until the mid-1950, when several Scandinavian clinicians described the clinical features in detail. In 1998, Shornick included ICP into the classification of the specific dermatoses of pregnancy. The incidence of ICP is higher in South America and Scandinavia, but the highest rate was detected in Chile (16%) [3]. In Middle Europe, the prevalence is approximately 0.2 - 2.4%. There are no available data concerning this impairment in Romania. The endemical occurrence and the positive family history in upto 50% of cases indicate a genetic background of the disease [1].

## Case History

A 22 year old Primigravida with 31weeks of gestation was admitted to antenatal ward with the complaints of yellowish discolouration of eyes, nailbuds and face, severe itching all over the body, passage of dark colored urine, change in the stool color (pale) and lack of sleep since one week . Client had a medical history of jaundice at the age of 4 yrs. Familial history of jaundice is absent, but history of Hypertension and Diabetes Mellitus is present. The clinical examination, revealed that client is weakly built, pale in appearance, jaundice and severe itching was evident which was not associated with secondary skin lesions. Client was lethargic. While her vital parameters remained normal.

Ultrasonography gave an impression of SLIUF of about 28-29wks of growth and estimated fetal weight of 1273+/-85gm and no markable anomalies. Urine report was positive for the presence of bile salts and pigments. Serum markers remained elevated with markable elevation of S. Bilirubin level 11.48mg/dL, SGOT – 46 IU/L , Alkaline phosphatase-287 IU/L, S. Albumin 2.98 g/dL, PT--- 14.0, aPTT--- 46.6, Hb---9.0 g/dL and peripheral smear report gave the impression of Normocytic normochromic anemia

with mild thrombocytosis. Hepatitis A and B report showed negative result (both rapid and ELISA) on admission. Client was treated with Ursodeoxycholic acid 300mg TID. 2 pints of FFP and 1pint of packed cell was transfused under strict supervision.When she attained 35wks of gestation, preterm labour pain started and mother was taken up for Emergency cesarean section, indication: fetal bradycardia and meconium stained liquor. APGAR score of the baby was 3,6, and 8 at 1st, 5th and 10th min. Baby was placed under oxygen hood and was shifted to NICU for Observation and further treatment. Baby weight: 1.035kg. No anomalies were detected at birth. Post operatively also mother had pruritis, bleeding was within the normal limit, while other parameters remained normal. Client had fatigue, appearance was lethargic and psychologically she was disturbed. Serum values started to reduce i. e S. Bilirubin dropped to 10.27mg/dL, Alkaline phosphatase—241 IU/L, PT – 11.6 and APTT--- 28.0 and Hb--- 10 g/dl. Post operatively, also ursodeoxycholic acid was continued along with the prophylactic antibiotic.

Discussion

The specific dermatoses of pregnancy are a heterogeneous group of severely pruritic inflammatory skin diseases specifically related to pregnancy and/or the immediate puerperium period. The most recent classification of the specific dermatoses of pregnancy, proposed by Ambros-Rudolph et al in 2006, includes the following diseases: pemphigoidgestationis (PG), polymorphic eruption of pregnancy (PEP), intrahepatic cholestasis of pregnancy (ICP), and atopic eruption of pregnancy (AEP). While some of them are pruritic but harmless, others may be associated with an increased risk for mother and child[1]. Different synonymous used are: Intrahepatic cholestasis of pregnancy /Obstetric cholestasis/ Recurrent jaundice of pregnancy / Pruritus gravidarum/ Icterus gravidarum / Idiopathic jaundice of pregnancy[2]. The etiology of ICP is *not completely understood* and is still under discussion. *Genetic, hormonal and* also *environmental factors* may contribute to the pathogenesis of ICP[2]. Mutations in the hepatocellular phospholipid transporter, ABCB4 (MDR3), that mediates secretion of the major human phospholipid, phosphatidylcholine (lecithine) into bile, have been estimated to account for up to 15% of all ICP cases[3,4,5]. Some of the clinical signs that a pregnant mother shows during the second or third trimester of pregnancy includes pruritis (range from mild to severe), insomnia; psychological sufferings with or without suicidal ideations, elevated liver enzymes with evidence of Jaundice, prolonged prothrombin time due to vitamin K deficiency, steatorrohoea due to malabsorption, gall stones, abdominal pain, malaise and other constitutional symptoms[2]. Pregnancy hormones affect gallbladder function, resulting in slowing or stopping the flow of bile. The gallbladder holds bile that is produced in the liver, which is necessary for the breakdown of fats in digestion. When the bile flow is stopped or slowed down, this causes a buildup of bile acids in the liver which can spill into the bloodstream[2].For a diagnosis of IHCP to be established, there must be no history of exposure to hepatitis viruses or hepatotoxic drugs or past history of liver or gall bladder diseases. Laboratory markers such as Liver function test values remain elevated specially serum bile acids, ALT, AST and serum bilirubin. Prolonged prothrombin time can be witnessed in mothers who are having vitamin k deficiency[6].

The newer diagnostic modalities include: Liver histology, Liver immunostaining, Electron microscopy,biliary lipid analysis, Molecular analysis, PMRS spectroscopy[7].

Therefore, the aim of treatment is toreduce bile acids in order to prolong the pregnancy and reduce both fetal risks and maternal symptoms. Obstetric surveillance is very important and the management consists of: - Weekly fetal cardiotocographic (CTG) registration, from 34 weeks of gestation; -Weekly assessment of liver function Tests - Control of prothrombin time

Medically ICP can be treated with the help of antihistamines, anion exchange resins (Colestyramine), phenobarbital, S-adenosylmethionine (SAM), and ursodeoxycholic acid (UDCA). In ICP, the use of S-adenosylmethionine is still a matter of debate. Some studies revealed a significant decrease of pruritus, bilirubin, and ALT after daily intravenous application of

SAM, but others showed no effect. The use of phenobarbital relieved pruritus in only 50% of patients and had no effect on liver parameters. UDCA has been successfully used in Chinese medicine for more than 5.000 years for the treatment of various liver diseases. It is a naturally occurring hydrophilic bile acid, which improves the clinical and biochemical tests in a variety of cholestatic liver diseases. UDCA is considered to be the first line of treatment option for patients with primary biliary cirrhosis (PBC), where it improves the survival rate without liver transplantation. The recommended dose for UDCA is 15mg/kg/day[1].

It has been reported that the incidence of respiratory distress syndrome in neonates born to mothers with ICP is twice that of a normal population based on analysis of bronchoalveolar lavage fluid of neonates born to mothers with ICP. Specifically, it has been hypothesized that bile acids can produce surfactant depletion in the alveoli[8].

ICP increases the risk of preterm delivery (up to 19–60%), meconium staining of amniotic fluid (up to 27%), fetal bradycardia (up to 14%), fetal distress (up to 22–41%), and fetal loss (sudden) (up to 0.4–4.1%), particularly when associated with fasting serum bile acid levels > 40 µmol/L. Maternal prognosis is good and symptoms resolve rapidly after delivery, accompanied by normalization of serum liver tests[2].

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