

SCRATCH THE TRUTH BEHIND THE ITCH

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Introduction:

Acute fatty liver of pregnancy (AFLP) was first described in 1940. It is a rare condition which affects 1:20 000 births a year¹ and can cause serious hepatic dysfunction. Mortality rates can range from 5%, with intensive treatment and soar to 75% if misdiagnosed¹. The pathophysiology of AFLP is poorly understood but it is usually observed in later pregnancy, 30-38 weeks². We present a case of AFLP occurring in a woman whose initial symptoms could have been attributed to intrahepatic cholestasis of pregnancy (ICP).

Case report:

A 34-year-old with 2 previous children born by caesarean section booked for maternity care. She had a history of asthma and ICP in both previous pregnancies. She was seen in the consultant clinic in the antenatal period and booked for a caesarean section at 39 weeks. She presented to the maternity day unit at 28 weeks complaining of itching. Biochemical investigations were normal and she was diagnosed as pruritis gestationis. At 36+6 weeks, she presented with worsening itch, along with polydipsia, vomiting, abdominal pain and reduced fetal movements. On assessment, she appeared dehydrated and was tachycardic at 109 beats/minute, but with normal blood pressure 103/62mmHg. Cardiotocography was normal. Serum biochemistry revealed raised bile acids 143umol/L, bilirubin 72umol/l, ALT 436iu/L and creatinine 103umol/L, with reduced glucose 3.3mmol/L. Lactate was 2.6mmol/L. Haematological tests showed INR 1.9 and WCC 17.8x10⁹/L. The differential diagnosis was ICP, AFLP or complications of atypical pre-eclampsia (PET).

Screening for viral or autoimmune hepatitis, liver genetic anomalies and hepatic vascular events was subsequently negative. She was managed by a multi-disciplinary team consisting of obstetrician, midwives, anaesthetist, obstetric physician, intensivist and hepatologist, in discussion with the tertiary liver centre.

She was stabilised with intravenous vitamin K and fresh frozen plasma (FFP). A category 3 caesarean section was performed under general anaesthetic with intraoperative cell salvage and blood loss of 500mls. There was meconium-staining in the liquor. Following birth, she was treated in the intensive care unit for 3 days, receiving further FFP and vitamin K, antibiotic prophylaxis, N-acetyl cysteine and careful support of fluid balance. She was discharged home on postnatal day 8. The neonate required intubation at birth, but was extubated after 1 hour and was well thereafter.

Discussion:

AFLP can be a diagnostic challenge. With many non-specific symptoms such as abdominal pain, vomiting, headaches and tiredness, it can be mistaken for other obstetric complications such as ICP or PET. The risk factors for AFLP are listed in table 1. AFLP leads to the damage of liver parenchyma due to fatty infiltration, which may be demonstrated on ultrasound (Image 1), but not reliably.

Diagnosis of AFLP can be assisted by the Swansea criteria (table 2), with a score of 6 or more for a likely diagnosis. Our patient scored 8/14. Treatment is to first correct any maternal metabolic derangements: hypoglycaemia, coagulopathy and renal dysfunction. Prompt delivery of the baby should be planned to arrest further hepatic damage. Timing of delivery should be individualised, taking prematurity into account.

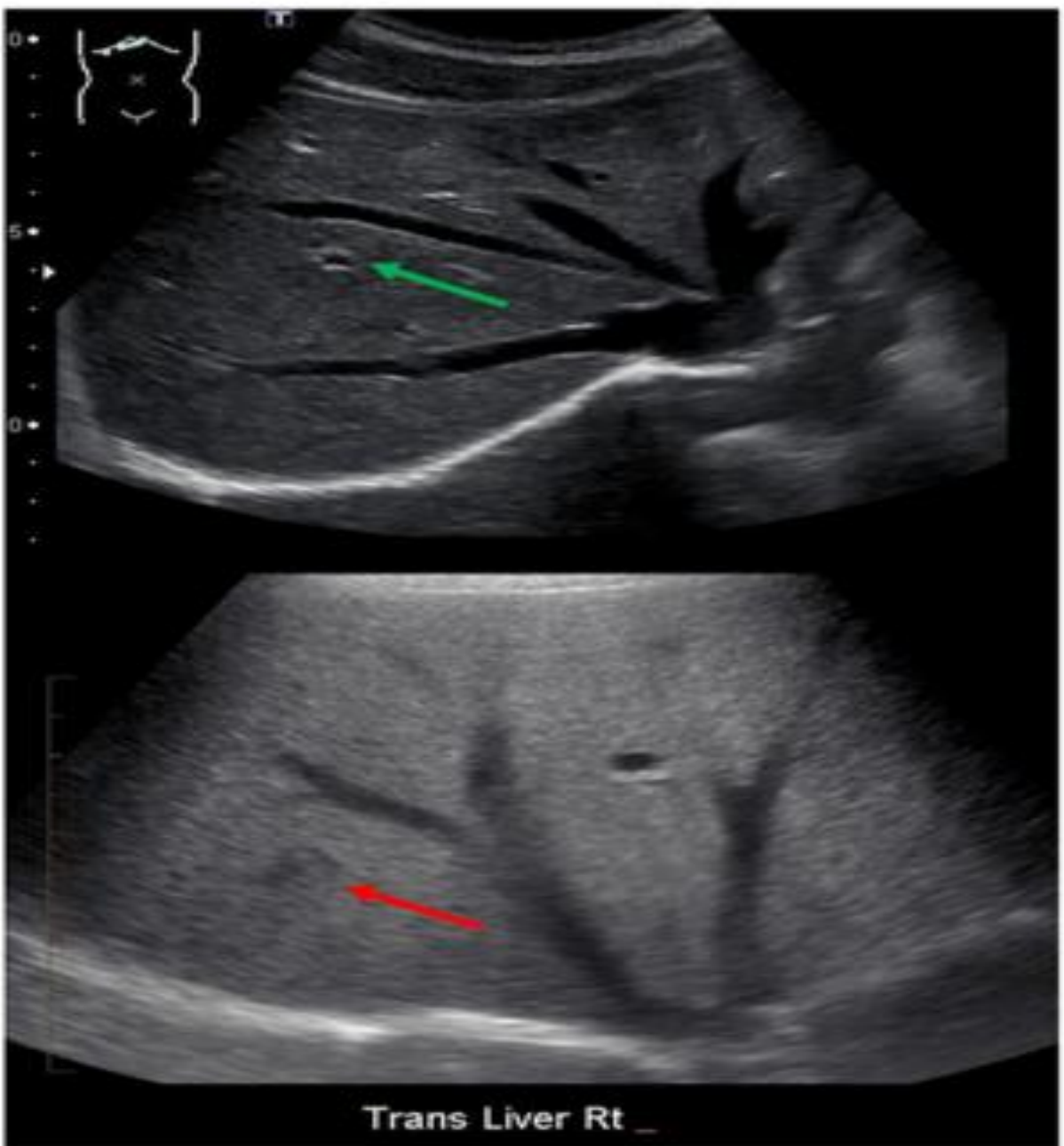
Conclusion:

AFLP is a rare obstetric emergency and can easily be mistaken for other more common diagnoses. Early recognition, initiation of supportive care, and prompt delivery of baby is essential to reduce maternal and fetal mortality³.

Table 1

- Fetal LCHAD deficiency (20% cases), other enzyme deficiencies
- Male fetal sex
- Previous AFLP
- Multiple pregnancy
- Nulliparity
- Low BMI
- Pre-eclampsia / HELLP

Normal



AFLP

Image 1: brightly echogenic liver parenchyma in AFLP (lower image), with loss of normal perivascular margins (red arrow). Image courtesy of Frank Gaillard, Radiopaedia.org, [rID: 7310](https://doi.org/10.5334/rtd-7310)⁴

Table 2- Swansea criteria

History and examination	Biochemistry / haematology
Vomiting	Bilirubin > 14 µmol/l
Abdominal pain	Transaminase (ALT/AST) >42 iu/l
Polydipsia and polyuria	Hypoglycaemia < 4 mmol/l
Encephalopathy	Creatinine > 150 µmol/l
	Urea > 3.4 mmol/l
Other	Ammonia > 47 µmol/l
Ascites or bright liver on US	Coagulopathy APTT > 34 or PT > 14 sec
Microvascular steatosis on biopsy	Leucocytosis > 11x10 ⁹ /l

References:

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