

Acute Fatty Liver of Pregnancy Presenting with Multiorgan
Dysfunction: A Case Report

The **best people**,
giving the **safest care**,
providing **outstanding experiences**

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25yrs P0 34/40 - Bg PCOS (normal BMI), in remission from ED, social drinker outside of pregnancy, previously raised umbilical PI - resolved, normal growth

UTI symptoms and brown PV discharge - Urine dip Protein ++, ketones ++, MSSU sterile pyuria - cephalixin - sent home after bloods taken

Asked to return 2 days later **as renal bloods abnormal** and on review symptoms included: nausea, anorexia, feverish, blurry vision, abdominal and lower back pain and urinary symptoms - struggling to pass with haematuria

Hb 115, plts 232, Wcc 15.9

CRP 16, urate 647, amylase 14

Urea 10.4 (9.1), Creat 203 (145), K+ 5.4 (5), normal Na

Yellow sclera, oedema to face and pitting to knees bilaterally, jittery

Tender right renal angle and RUQ

VBG: pH 7.27, lactate 2.5, glucose 2.7, HCO3 - 16, bilirubin 97

Abnormal antenatal CTG

Cat 1 GA EMCS (no clotting results) - **uncomplicated**, EBL 300mls

Immediate PN period

500mls 10% glucose in NaCL during c-section

BP 146/98 - nifedipine MR 10mg bd, HR 128 bpm

6hrs PN - pH 7.28, lactate 2.0, glucose 7.5

Hb 105, **Wcc 32.8**, plts 224, CRP 20, Na 128, K 5.1, **Urea 9.4 (10), Creat 197 (203), eGFR 30, Bilirubin 94, ALT 142, GGT 66**, PT 15.8, APTT 40, lactate 3.9

On IV tazocin and 2L O2, had 3L plasmalyte and 40 IU synto

Other Investiagtions

-Non-invasive liver screen - (Hepatitis A,B,C,D,E, EBV, CMV, Parvovirus, HIV, Ig liver antibodies) - negative

-Liver USS - NAD

-Bedside ECHO - small pericardial effusion

Day 2 PN

Ongoing tachycardia, as well as nausea and vomiting
+ve fluid balance 2.9L, urine output 30mls/hr

Bloods: Hb 99, Wcc 35, CRP 24, Na 129, K 4.5, Urea 13.9 (9) → 15.3, Creat 232 (197) → 262 → 270, eGFR 21, ALT 113 then 88, bilirubin 85 then 77, GGT 66, alk P 384, alb 28, PT 17.5, APTT 40.5, INR 1.1, elevated LDH, fibrinogen 1.1

Discussed with renal team and transferred to ITU day 3 PN

ITU Work-up

Wcc >40 - on IV tazocin ? renal infective cause

US liver - patent vessels, CT abdomen - ascites

Worsening AKI (Urea 19.6 (15.3), Creat 290 (270)) - urine output declined over the day, renal screen ordered

Abnormal clotting, and deranged LFTs but improving - vit k

For ascitic tap and consideration of referral to Liver Centre

Day 4 PN

Intermittent visual hallucinations, agitated

Hyperreflexia, worsening upper abdominal pain

Borderline glucose - just maintaining at 4 mmol/L

BP >140mmHg - labetalol 200mg BD added

Magnesium and 10% dextrose and furosemide 80mg with fluid restriction to 1500mls

Hb 87, Wcc 43, plts 170, Na 123, K 5.3, **Urea 33 (19), creat 351 (290), ALT 55, AlkP 236, GGT 72**, APTT 35, LDH 821,blood and urine cultures -ve

Continuous Veno-Venous Haemodialysis (CVVHD)

-stopped after 24 hours, urine output improved >150mls/hr

-Liver team consulted and advised to add in Vancomycin / teicoplanin and fluconazole

-2 x random cortisol <100 - started hydrocortisone 50mg QDS

-Hb 69 (normocytic, low haematocrit) - transfused 1 unit

Day 5-7 PN

BP improved with anti-HTNs gradually reduced to amlodipine 5mg

Fluid restriction relaxed and urine output improved - had polyuric phase (3L per day)

Clotting corrected after vit K

Renal function and LFTs improved: urea 12, creat 115, ALT 34

Bilateral pleural effusions

Large volume ascites abdo and pelvis on CT and liver hypoattenuation - diffuse hepatic steatosis

6cm C-section scar haematoma subcutaneous

Endocrine referral and short synacthen test

Latest Recovery Updates

Wound haematoma - conservative management with oral abx and FU with wound clinic

Having renal surveillance, BP managed with Amlodipine and Bisoprolol

Under further investigation by Endocrine team for possible

Sheehan's/hypopituitary - remains on hydrocortisone, normal Short

Synacthen test, awaiting MRI pituitary

Subjectively - Mum and baby are doing well

Learning Points

•**Atypical presentations are common:** Acute fatty liver of pregnancy (AFLP) may initially mimic sepsis or urinary tract infection → maintain diagnostic vigilance.

•**High index of suspicion is critical:** Non-specific symptoms and overlap with pre-eclampsia/HELLP can delay diagnosis → early consideration improves outcomes.

•**Rapid deterioration can occur:** AFLP can quickly progress to **multiorgan dysfunction** (hepatic failure, renal failure, coagulopathy, encephalopathy).

•**Early diagnosis = better outcomes:** Prompt recognition and timely intervention are key determinants of maternal and fetal survival.

•**Delivery is definitive management:** Stabilisation followed by **expedited delivery** remains the cornerstone of treatment.

•**Multidisciplinary care is essential:** Optimal outcomes require coordination between obstetrics, anaesthesia, hepatology, and critical care teams.

•**Supportive therapies are often needed:** Renal replacement therapy and intensive care support may be required in severe cases.

•**Evidence gaps remain:** Further research is needed to refine **diagnostic criteria and management pathways** in AFLP.

1. Vigil-De Gracia P, Montufar-Rueda C, Ruiz J. Management of acute fatty liver of pregnancy: clinical experience & literaturereview. International Journal of Gynecology and Obstetrics. 2011;113:161-4.

2. European Association for the Study of the Liver. EASL Clinical Practice Guidelines on liver diseases in pregnancy. Journal ofHepatology. 2023;79:1269-1306.