International Journal of Applied Research 2025; 11(7): 202-204

ISSN Print: 2394-7500
ISSN Online: 2394-5869
Impact Factor (RJIF): 8.69
IJAR 2025; 11(7): 202-204
www.allresearchjournal.com
Received: 10-05-2025
Accepted: 12-06-2025

Dr. Akshita Bhandari
Postgraduate, Department of Obstetrics and Gyanecology, Sri Ramachandra Institute of higher education and research, Porur, Chennai, Tamil Nadu, India

Dr. Bhuvana S
Department of Obstetrics and Gynaecology, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India

Dr. N Palaniappan
Department of Obstetrics and Gynaecology, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India

Corresponding Author:
Dr. Akshita Bhandari
Postgraduate, Department of Obstetrics and Gyanecology, Sri Ramachandra Institute of Higher Education and Research, Porur, Chennai, Tamil Nadu, India

Labetalol induced liver injury

Akshita Bhandari, Bhuvana S and N Palaniappan

Abstract
Drug induced liver injury is a well known cause of transaminitis, in this case, labetalolol, as an etiological factor of liver injury complexly intertwined with a clinical picture of biliary pancreatitis. This clinical case report holds significance in patients with gestational or chronic hypertension where a marked rise in liver enzymes obviates further workup in the direction of partial HELLP syndrome or differentials such as acute fatty liver of pregnancy, nonalcoholic steatohepatitis, cholelithiasis, infectious diseases or autoimmune diseases, therefore further necessitating delivery. Interestingly, in this case report, multiple exhaustive investigations after followup with a multidisciplinary team of experts were all rendered negative only to point to labetalol being the singular cause of hepatotoxicity and biliary pancreatitis, thereof potentially averting unnecessary iatrogenic preterm delivery measures. On delving deeper into the specifics of this case report after meticulous workup for other causes of liver injury and unsettling liver and pancreatic enzymes, a simple resort to stop labetalol and switch to alternate antihypertensive proved to be useful in improving the clinical picture liver injury.

Keywords: Drug-induced liver injury (DILI), labetalol hepatotoxicity, transaminitis, biliary pancreatitis, gestational hypertension, HELLP syndrome

Introduction
Labetalol is one of the first line and an extensively used antihypertensive agent in gestational hypertension. It is a selective alpha adrenergic antagonist and a nonselective beta adrenergic antagonist, it has an excellent safety profile, however as in this case, rarely it can be a cause of acute transaminitis, hepatotoxicity and hepatocellular injury. This case brings to light how a rare but eye opening side effect of labetalol presenting as transaminitis in a patient with gestational hypertension can mislead the diagnosis towards acute fatty liver of pregnancy or partial HELLP syndrome.

Case Report
A case of a 28 year old woman, primigravida at 29 weeks and 4 days, gestational diabetes mellitus on oral hypoglycemic agents, diagnosed as gestational hypertension, on Tab labetalol 100mg thrice a day since 26 weeks of gestation presented to OPD with complaints of severe epigastric pain and multiple episodes of vomiting and occasional headache for 1 day.

History
1st Trimester
Spontaneous conception, confirmed by UPT at 45 days of amenorrhea, tablet folic acid taken regularly, dating scan done, dates corresponded, NT scan and FTS were normal.

2nd Trimester
Quickening felt at 5th month of amenorrhea, Tablet iron and calcium taken regularly. Anomaly scan done, anomalies ruled out. Inj Td 2 doses taken. Diagnosed as Gestational diabetes mellitus, started on OHA.

3rd Trimester
Continued to perceive fetal movements regularly, continued iron and calcium regularly. Growth scan done, growth was satisfactory. Diagnosed as gestational hypertension and started on Tab Labetalol 100mg thrice a day at 26 weeks of gestation.

Examination

Vitals

No pallor, icterus, clubbing, cyanosis, lymphadenopathy, pedal edema

PR 88 bpm

RR 16/min

BP 140/100 mmHg

Systemic examination - normal

P/A -

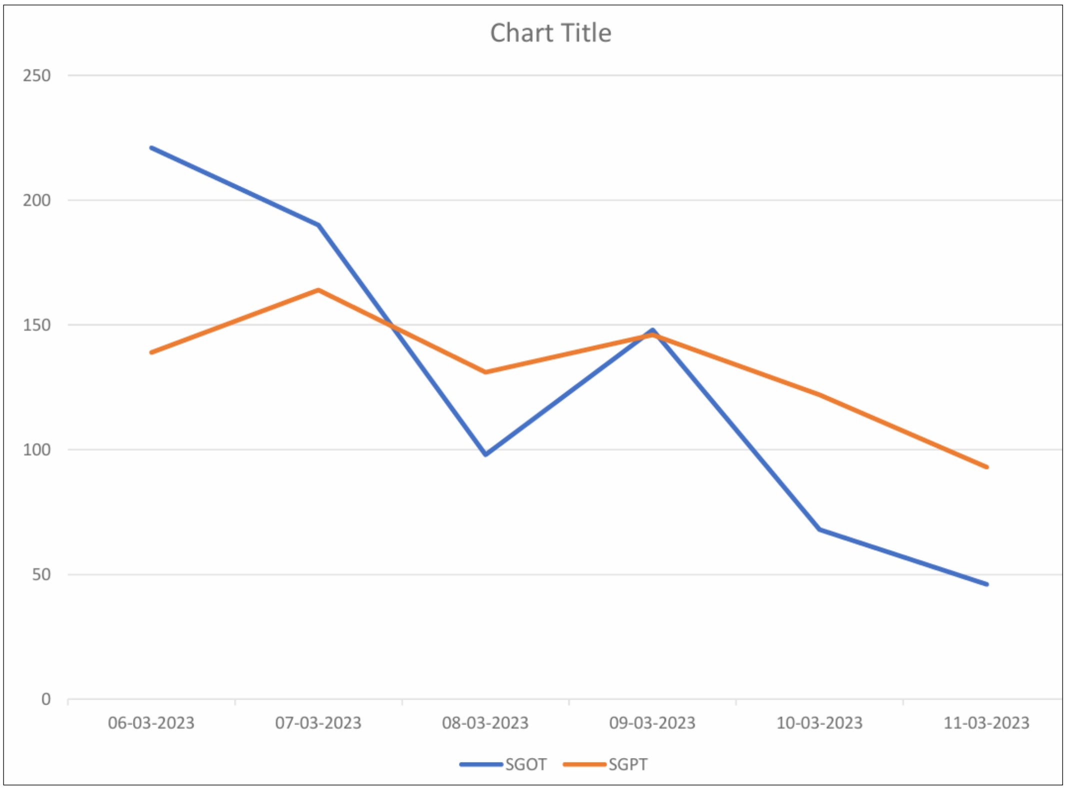
Uterus at term

Relaxed

Cephalic

FH present

Clinically liquor normal



DATE	SGOT	SGPT	Hb	Platelet	GGT	S. Amylase	S. Lipase
06-03-2023	221	139	10.6	2.57	-	-	-
07-03-2023	190	164	10.7	2.95-2.96	432	119	162
08-03-2023	98	131	-	2.44	0	2649	9240
09-03-2023	148	146	-	-	-	-	-
10-03-2023	68	122	-	-	-	-	-
11-03-2023	46	93	-	-	-	139	195

Fig 1: Serial monitoring of Investigation

Discussion

In view of the above mentioned findings, patient was shifted to labor room, urine albumin was trace, urine ketones were negative, blood pressure was 140/100 mmHg, steroids were covered and magnesium coverage of loading followed by maintenance dose was started with a working diagnosis of partial HELLP in mind.

Liver function test showed elevated values of AST and ALT of 221 and 139 respectively, platelet counts were 2.57 lakh/cumm and normal coagulation profile and peripheral smear on 6th March.

Ophthalmology opinion obtained showed normal fundus findings with no evidence of hypertensive retinopathy.

Continuous BP monitoring was done and magnesium sulphate charting was done, with SBP at 140-150 mmHg and DBP maintaining at 90-100 mmHg.

Hepatology opinion obtained in view of elevated liver enzymes, advised to stop Labetalol and start on an alternative hypertensive (Tab Nifedipine 20 mg twice a day), to send serum amylase, lipase and gamma glutamyl transferase, HIV, HBsAg and HCV.

Serum amylase was 119 and serum lipase was 162, gamma glutamyl transferase was 432, SGOT and PT were 190 and 164 respectively.

Viral markers were nonreactive.

Medical and surgical gastroenterology opinion was obtained in view of unsettling symptoms and elevated transaminases, advised ultrasound abdomen and MRCP.

Ultrasound abdomen showed mild hepatomegaly and grade 2 fatty infiltration of liver.

MRCP done showed choledocholithiasis with mild to moderate upstream dilatation of biliary tree, raising suspicion of choledocholithiasis, biliary or acute pancreatitis. Repeat SGOT and SGPT was 98 and 131 respectively, Serum amylase and Lipase showed a sharp increase and critical value of 2649 and 9240 respectively.

MGE and SGE reviews obtained, advised ERCP and the same was done, showed findings of cholelithiasis, choledocholithiasis, sphincterotomy done along with CBD and Pancreatic duct stenting.

Repeat SGOT, PT was 148, 146 respectively.

On day 3 of stopping labetalol and post ERCP, SGOT, PT returned to baseline values of 46, 93 and serum amylase, lipase of 139 and 195 respectively.

Repeat hepatology review

In view of normal INR, platelet count, RFT, serum electrolytes and elevated transaminases and GGT that it was unlikely pregnancy induced (HELLP, acute fatty liver of pregnancy), cause being drug induced liver injury.

Labetalol induced liver injury presents with transaminitis i.e. elevated aspartate and alanine aminotransferase, in this case also presenting as choledocholithiasis as an MRCP finding. An intriguing revelation in this case was of biliary pancreatitis with elevated Serum amylase and lipases further adding a layer of complexity to the case because biliary pancreatitis in itself can be a cause of transaminitis masking the actual cause and necessitating further evaluation, when in actuality, biliary pancreatitis is a less common but known complication associated with the use of labetalol.

Conclusion

A complex clinical picture of transaminitis in a patient of gestational hypertension could create a clinical picture and obviate the need to workup towards obvious diagnosis such as HELLP, AFL of pregnancy, when in reality a simple switch from labetalol to an alternate hypertensive can reverse the clinical picture to normalcy.

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