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Case Report

Constipation in obstetrics - an underrated sign

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ABSTRACT

An interesting case of normotensive hemolysis, elevated liver function tests, and low platelet counts (HELLP) syndrome with only presenting symptom of severe constipation near term; an occult sign of liver disease. She presented in active labour with mild jaundice and early signs of disseminated intravascular coagulation (DIC). This was an obstetric near miss, as it was a fine balancing act combined with aggressive monitoring and nursing management by highly skilled team of trained obstetric nurse midwives. This was a humbling lesson which reminded us not to ignore aggravated commonly occurring symptom in all patients of obstetrics. Availability of fresh whole blood makes it easier to manage such cases as fresh components are extremely difficult to procure and process in remote obstetric units.

Keywords: HELLP syndrome, Obstetric near miss, DIC, Jaundice in pregnancy, Obstetric HDU nursing

INTRODUCTION

Constipation in pregnancy is often ignored and fairly common physiological symptom in pregnancy managed by dietary advise, increase fluid and roughage intake, exercise with mild laxatives if required. It affects roughly 40% of women, and is often a result of hormonal and physical changes. Increased progesterone levels can slow down digestion due to decrease in gut motility and generally relaxing effect on all involuntary smooth muscles with tightening of sphincters.[1] The gravid uterus puts pressure on the intestines, making it harder for stool to pass. Additionally, iron supplements, frequently taken during pregnancy, can contribute to constipation. Dietary fads prevalent in various cultures along with unscientific, unnecessary advice of bed rest, lack of exercise, emesis with decreased fluid intake aggravates symptoms. Apart from the fact that constipation is an uncomfortable symptom, it also has the potential to cause permanent impairment. Patients with no history of bowel problems may develop constipation for the first time during pregnancy and, in addition, women who suffer with constipation prior to pregnancy will often find their symptoms are worse when pregnant. The causes of constipation in pregnancy are likely multifactorial.[2] Dietary factors and lifestyle issues play an important role but factors such as hormonal and mechanical changes are also important. There is evidence that straining to defecate can damage pudendal nerve and impair the supportive function of pelvic floor musculature.[3] Constipation is also an important factor in the development of uterovaginal prolapse. Constipation refers to difficulty in passing stool and infrequency of bowel motions, which is not secondary to an underlying cause. The colon's major functions are to conserve water, to facilitate bacterial digestion of dietary fibre and to retain and expel faeces. Colonic motility, absorption and the internal and external sphincters affect these functions leading to increase in gut transit time and further absorption of water thereby worsening constipation.

Objectives

The objectives were to spread awareness regarding one of the potentially life threatening maternal obstetric near-miss, management of HELLP and DIC in peripheral obstetric setting, importance of early recognition and a

"sixth-sense" for early diagnosis and treatment of labour room emergencies, emphasis on availability of fresh whole blood in absence of components, management of cases as per existing Guidelines in Standard Textbooks of Obstetrics, and reporting normotensive HELLP syndrome.

CASE REPORT

This case is being reported because our index patient was normotensive and landed in postpartum HELLP syndrome with early DIC. Her only symptom antenatally was severe aggravated constipation after appx. 36 weeks POG with no derangement in any biochemical or haemotlogical parameters. This symptom was a clue to underlying potentially life-threatening liver pathology which was investigated with routine tests which were normal and hence the common symptom managed conservatively. Her postpartum period was stormy which is presented as the events unfolded in labour room in sequence and further aggressive obstetric management in the ward with the good nursing care by a team of dedicated trained midwives.

A 31-year-old G2P1L1, one previous FTND, reported first to our centre at 31+2 POG with only diagnosed high risk profile of IGT of pregnancy on MNT. She was booked, normotensive, immunized with regular ANC visits at previous obstetric centre at Maharashtra. She was being reviewed by AYUSH homeopathic practitioner. All her biochemical and haematological parameters including obstetric USG performed at regular intervals were WNL till date. Her routine antenatal investigations including seven-point sugar profile were repeated which were normal. She was examined and advised to report after four weeks. Her next visit was uneventful and she offered no complaints. She reported at around 36 weeks with severe constipation and distress. Her GPE was unremarkable, vitals were pulse-82/mt, BP-128/82 mmHg, R/R-16/mt, temp-98.6°F.

Systemic examination including obstetric examination was normal and she was not in labour. She was given enema, routine biochemical and haematological investigations including LFT and obstetric USG were done which were WNL. She was relieved after enema and requested discharge and was sent home with advice to report after one week. She reported after 6 days in active labour and was delivered by midwife after around 2 hours. Nothing unusual was recorded, her vitals were normal and she was normotensive. Her haematological investigations were sent as per standard protocol which were WNL.

After around half an hour fresh active bleeding PV was noticed, bucket handle cervical tear was sutured at 6 O'clock position. Appx. blood loss was 700 ml. She was catheterized prior to suturing, 500 ml of high coloured urine was drained. She was simultaneously administered oxytocin, tranexamic acid, IV fluids, analgesics, PGF2α and Misoprostol sublingually and two units B positive whole blood demand was sent as per "WHO EMOTIVE PPH" Drill. Her vitals were stable for the entire duration of procedure. Breast feeding was initiated.

Table 1: Rapid clinical assessment.

S. No.	Assessment
1.	Two units Whole Blood Transfusion over 24 hrs.
2.	Inj MgSO4 by I.V Modified Sibai Regimen (MgSO4 Chart was maintained) for 24 hrs.
3.	Inj LMWH 40 mg SC OD x 10 days.
4.	Inj Vit K 10 mg I.V OD x 5 days.
5.	Inj Dexamethasone 8mg I.M 12hrly x 4 doses
6.	Inj Neurobion 1 amp I.V OD x 5 days.
7.	Inj 25% Dextrose I.V 12 hrly x 5 days.
8.	Ecosporin 75 mg OD (increased to 150 mg OD after 48 hrs) x 20 days.
9.	Tab Labetalol 200 mg TDS (stopped after 48 hrs).
10.	UDCA SR 300 mg TDS PO x 20 days.
11.	Oral Dextrose 25% TDS x 20 days.
12.	Syp Lactulose 15 ml HS x 20 days.
13.	Syp Sorbitol Tricholine Citrate 15 ml BD x 15 days.
14.	I.V Fluids 3RL, 3IGS for 48 hrs followed by 4 units daily for next 3 days. (I/O chart was maintained).
15.	I.V Oxytocin 5.I.U was administered in each alternate drip for 24 hrs.
16.	Inj TXA was stopped after 2 doses (being hepatotoxic).
17.	Catheter x 48 hrs.
18.	Vaginal haemostatic pack was removed after 4 hrs, no active bleeding noticed. Uterus was well contracted and retracted.
19.	No NSAIDS/ No Paracetamol/ No PGF2α/ No Lasix/ No Drugs without permission.
20.	Patient Liver Chart as advised by Dame Sheela Sherlock was advised to observe for micrographism and constructional apraxia. Nursing staff was advised to observe for reversal of sleep rhythm.
21.	High protein bland diet.
22.	Keep Calcium Gluconate or Calcium Chloride Injections ready to deal with MgSO4 toxicity.

On examination post suturing and packing; her BP was noticed to be high 172/94 mmHg and during clinical assessment of blood loss mild pallor and icterus were noticed. She gave retrospective history of high coloured urine of 2 days duration. A working diagnosis of "jaundice in pregnancy"; likely HELLP was made and all biochemical investigations were requested including viral markers. She did not have any other peripheral clinical signs of liver cell failure or IHCP.

Treatment for HELLP was initiated including $MgSO_4$ by I. V. modified Sibai regimen. Treatment initiated based on rapid clinical assessment is enumerated in Table 1.

She made rapid clinical recovery; her Vitals are recorded in tabular format in Table 2 (Original clinical dataset) highlighting most elevated/deranged parameters for the day. With aggressive management and dedicated nursing care she was discharged on D10. Although her serum bilirubin was not normal, a well deliberated clinical judgment to discharge the patient was made. She reported 6 days post discharge with normal reports. Her baby had no complaints and was active healthy and feeding well. We judiciously decided to manage her in our LR setting because her DIC score was consistently less than 3.

Table 2: Vitals in tabular format.

Vitals Date/day	Temp (°F)	Pulse (/min)	Resp. rate (/min)	Bp (mmHg) (highest recording)
Adm. in labour (31.05.2025/d1)	98.0	92	22	130/90
Intrapartum/d1	99.0	98	28	156/90
Imm postpartum/d1	97.6	102	24	172/94
Postpartum/d1	100	100	20	146/86
Postpartum (1800 hrs)/d1	101	102	22	142/88
01.06.2025/d2	99.2	98	20	134/76
02.06.2025/d3	98.6	96	16	124/80
03.06.2025/d4	98.4	92	18	118/76
04.06.2025/d5	98.8	88	20	114/80
05.06.2025/d6	98.6	74	16	116/74
06.06.2025/d7	98.6	76	16	112/70
07.06.2025/d8	98.6	80	18	118/74
08.06.2025/d9	98.6	76	16	108/70
09.06.2025/d10	98.6	72	16	110/68
16.06.2025/d17	98.6	78	16	110/70

Table 3: Investigations in tabular format.

Date → INV	31/05/2025 0830 hrs	31/05/2025 2230 hrs	02/06/2025	06/06/2025	16/06/2025	Remarks
Hb (g%)/ PCV%* urine Alb	11.9, neg	10.2/30, neg	11.2/31.9, neg	11.6, neg	11.8 g/32, neg	2 units whole blood on 31/05/2025
Platelet (cumm)	155×103	105×103	124×103	156×103	160×103	*
INR, C/T secs	1.2, 13/16.2	1.5, 13/18	1.26, 13/16.16	1.06, 13/13.6	1.1, 13/13.2	*
FDP (µg/ml)	>20	-	>20	>20	<5	*#
S. fibrinogen (mg/dl)	87	-	109	260	350	*#
D-Dimer (ng/ml)	-	-	3545.51	2226	302	$
Retics %	1.5	1.3	1.2	1.05	0.8	*
LDH (IU/l)	902	956	631	631	244	*
Na^+ (meq/l)	138	139	137	138	//N	
K^+ (meq/l)	3.4	3.2	3.6	3.8	//N	
Urea (mg/dl)	37	38	36	35	//N	
Creat (mg/dl)	2.0	1.9	1.6	1.2	//N	
Uric acid (mg/dl)	6.8	6.5	6.2	5.4	//N	
T. Prot. (gm/dl)	5.1	5.6	5.8	5.8	//N	
Alb (gm/dl)	3.5	2.3	3.3	3.5	//N	
Glob (gm/dl)	1.6	3.3	2.5	1.6	//N	
A:G ratio	2.1	0.69	1.3	2.1	//N	
T. Bil (mg/dl)	3.5	3.4	3.5	3.2	1.2 mg/dl	
SGOT (IU/l)	84	75	62	49	//N	
SGPT (IU/l)	67	53	47	42	//N	
GGT (IU/l)	156	135	127	87	//N	

Continued.

Date → INV	31/05/2025 0830 hrs	31/05/2025 2230 hrs	02/06/2025	06/06/2025	16/06/2025	Remarks
RBS (mg/dl)	84	103	96	98	//N	
Amylase (IU/l)	39	22	29	25	//N	
Alk PO[4] (IU/l)	260	242	236	166	//N	

*Benchmark criteria used for monitoring by our team, *# N/A at our centre, requires specialised lab, results were available after 48 hrs, $ not recommended by RCOG (fallacious), Highlighted values are gold standard for DIC Scoring.

Table 4: Tennesee diagnostic criteria for HELLP.[6]

S. no.	Variables
1	AST≥ 70 IU/l
2	LDH ≥600 IU/l
3	Platelets ≤100×10^9/l

Author does not recommend management of these cases with DIC score more than 3 in peripheral settings in the absence of Robust blood bank services with availability of components unless cornered. Her investigations reports investigations are shown in Table 3 (Original clinical dataset). Her urine albumin was consistently negative. Spot UACR was normal and was low risk for ecclampsia. DIC Score was maintained by ISTH criteria (Table 4).

DISCUSSION

HELLP syndrome is a severe form of preclampsia (sometimes called "atypical preclampsia") characterized by haemolysis (H), also expressed as microangiopathic hemolytic anemia, elevated liver enzymes (EL), and low platelets (LP). The condition usually occurs antepartum, between 27- and 37-weeks' gestation; 15% to 30% of cases present initially postpartum. Our index case was Postpartum. HELLP syndrome poses significant diagnostic and therapeutic challenges because only 80% to 85% of affected people present typically with hypertension and proteinuria. Our index case was normotensive. The disease is associated with progressive and sometimes rapid feto-maternal deterioration. Early detection and aggressive management with a combination of intravenous magnesium sulphate, intravenous dexamethasone, control of blood pressure, replacement of blood products, and timely delivery of the fetus and placenta seem to be the best and safest ways to arrest disease progression and reduce adverse outcomes. Maternal outcomes are improved considerably with this management; perinatal outcome depends predominantly on the gestational age when delivery occurs. A critical step is the early initiation of potent glucocorticoids as soon as the diagnosis of HELLP syndrome is made, so that severe maternal morbidity (stroke, liver hematoma/infarction/rupture, acute pancreatitis) and maternal mortality can be avoided. Although delivery is the only cure, serious manifestations continue into the immediate postpartum period.[4]

Some patients may present without these signs/symptoms when first evaluated for unexplained thrombocytopenia. The diagnosis of HELLP syndrome should be considered in any pregnant patient presenting in the second half of gestation or immediately postpartum with significant new-onset epigastric/RUQ pain until proven otherwise.

To meet diagnostic criteria, liver transaminases should be elevated >70 IU/l, or twice the upper limit of normal concentration not accounted for by alternative diagnoses, and the platelet count should be <100,000/mm^3 (Table 4 and 5) haemolysis may be indicated by elevated total bilirubin (>1.2 mg/dl), LDH and aspartate aminotransferase (AST) elevations, characteristic findings (schistocytes) on a peripheral blood smear, haematuria, worsening anaemia, and a low serum haptoglobin (ACOG).[5]

Table 5: Mississippi criteria for classification of HELLP.[6]

HELLP class	Criteria
I	Platelets ≤50×10^9/l
	AST or ALT ≥70 IU/l
	LDH ≥600 IU/l
II	Platelets ≤100×109/l, ≥50×10^9/l
	AST or ALT ≥70 IU/l
	LDH ≥600 IU/l
III	Platelets ≤150×10^9/l, ≥100×10^9/l
	AST or ALT ≥40 IU/l
	LDH ≥600 IU/l

Table 6: ISTH scoring for DIC.[7]

Parameter and value	Points allocation
Platelet count	
>100×10^{9}/l	0 points
<100×10^{9}/l	1 point
<50×10^{9}/l	2 points
Elevation of FDP (D-Dimer levels)	
No increase	0 points
Moderate increase	2 points
Marked increase	3 points
Prolongation of PT (Prothrombin time)	
<3 seconds	0 points
3-6 seconds	1 point
>6 seconds	2 points
Fibrinogen level	
<1g/dl	0 points
>1g/dl	1 point
≥ 5: compatible with overt DIC: Repeat values daily	
<5: suggestive of non overt DIC: Repeat values every 48 hrs	

Table 7: Preclampsia associated liver diseases.[8]

Differential Cohort value	Severe preclampsia and eclampsia	HELLP syndrome	Acute fatty liver of pregnancy
Time	After 22 wks POG	Late II trimester and early postpartum	III trimester
Prevalence	Increases in multiple gestations	0.10%	Increases in primis, male fetuses, multifetal gestations
Findings	High blood pressure; proteinuria; edema; seizure; renal failure; pulmonary edema	Abdominal pain, nausea/vomiting, overlap with findings in preclampsia	Abdominal pain, nausea/ vomiting, jaundice, hypoglycaemia and hepatic failure
Tests	Platelets > 70000; urine protein > 5 g/24 h; abnormal liver enzymes (10%)	Low platelets; hemolysis; elevated liver enzymes; prothrombin time may remain normal; normal fibrinogen	Platelets <100000; AST and ALT 300-1000 U/L; low antithrombin III; high prothrombin time; low fibrinogen; high bilirubin; DIC
Management	Blood pressure control; beta-blockers, methyldopa, magnesium sulfate, early delivery	Prompt delivery	Prompt delivery; liver transplant
Outcome	1% maternal death	5% maternal death 1% hepatic rupture, 1%-30% fetal death	≤10% maternal death (in HDU), upto 45% fetal death

Nearly 3% of pregnancies are complicated by some form of liver disease, and severe pregnancy-related liver diseases can have fatal consequences for both mother and child. Pregnant women undergo some physiological changes that can mimic liver disease; therefore, they must be considered in the diagnostic approach to women with the suspected liver disease.[6] Following diseases are always to be considered in differential and Jaundice in pregnancy of any underlying etiology is invariably fatal if the aggressive management is deferred. We followed the ISTH criteria for the DIC scoring in management of this patient (Table 6).[7]

Considering the various pathologies of jaundice in pregnancy, the presence of this sign is ominous and an indicator of high-risk pregnancy. Discussing jaundice or HELLP syndrome is beyond the scope of this case report. Author encourages clinicians to refer regularly to various RCOG and ACOG guidelines published every year along with GTG and good clinical practice. Latest article on HELLP Syndrome has been published in ACOG (Jul 2025) and emphasis is still on MgSO4, antihypertensives, steroids and termination. Spectrum of hypertensive diseases in pregnancy is beyond the scope of this article; however, three major differentials and their highlights is presented in Table 7.[8]

We have presented a challenging case wherein we adhered to age-old time-tested guidelines and were able to save the mother with aggressive management. Author cautions against managing patients with DIC score ≥5 in absence of blood bank services.

Table 8: Differential diagnosis

S. No.	Diagnosis
1.	Hyperemesis gravidarum
2.	AFLP
3.	Infectious hepatitis
4.	Ecclampsia
5.	IHCP (Intra hepatic cholestasis of pregnancy)
6.	SOJ including cholecystitis
7.	Hepato renal syndrome
8.	Budd-Chiari syndrome and DVT
9.	Peurperal sepsis
10.	Cirrhosis and portal hypertension
11.	Autoimmune diseases
12.	Acute pancreatitis.

Liver diseases and liver cell failure have a plethora of signs which are taught to every medical student during their clinical rotation in internal medicine. Rattling them out from tip to toe used to be the favourite pastime of attending consultant. Constipation is a fairly common physiological symptom in obstetrics due to progesterone load in the body due to pregnancy. But here we realized that intractable constipation requiring enema requires a second look and a keen eye to detect underlying liver dysfunction, especially new onset symptom in third trimester. Evaluation of liver enzymes is warranted in such cases as understood by this clinical case report.

CONCLUSION

Labour room emergencies are challenging for any obstetrician. Most are like a bolt of lightning and can be exhausting for all concerned. Dealing with maternal life-threatening emergencies requires swift teamwork, competent midwives, common sense, rapid clinical reassessment, energy of Thor with vigilance of Holmes, and a Herculean effort from ancillary staff with supportive family members. Blood bank is the life line of any labour unit and the limiting factor in all emergencies. Anticipate emergencies, evaluate limitations of your setting and strict due diligence in OPD with proper evaluation and assessment of risk factors. Yet often once in a while a potentially "low-risk" pregnancy waltzes in and teaches us a lesson and the experience can be humbling. For the uninitiated a few golden words: Last patient in the OPD is either an ectopic or preclampsia. Most haemorrhaging patients are Rh negative and only information needed to manage a maternal LR emergency is blood group. If the thought of hysterectomy crosses your mind; perform it early. No one can beat statistics-crowding of statistics can be demoralising for any obstetrician and is not a narration of our competence. Always look at the patient's face; Captain can sense an imminent "sense of doom" and knows when the ship is unsalvageable and yet cannot abandon. "Normal delivery" is a retrospective diagnosis. In the end "hands that rock the cradle rule the world" and the inevitable first rocking by midwife deals with a lifetime of happiness.

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