Original paper

HELLP Syndrome case study

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Abstract

Background: HELLP syndrome is a serious medical condition found in pregnant women. Usually in 3rd trimester [last 3 month], HELLP syndrome comprise Hemolytic ,i.e. breakdown of red blood cell , Elevated liver enzyme & Low Platelet count it's said to be an off – shot of pre-eclampsia or eclampsia i.e. hypertension during pregnancy . Usually it is found before the birth of baby, however sometime it can occur after the delivery

Objective: This was done to describe general experience with management of HELLP syndrome by analysis of patient outcome.

Patients And Methods: The patient M.K 30 year's old primigravida, developed generalize edema, high B.P., pallor, jaundice, haematuria &convulsion. She received anti-convulsion [MgSO4] & antihypertention (apresoline), also full investigation was done ,the result revealed P.C.V 28% ,T.S.B 8mg/dl, elevated liver enzyme ,low platelet [80,000]. The decision was termination of pregnancy after 6 hours stabilization, she also received antibiotic ,hydrocortisone. platelet, fresh frozen plasma &blood ,and because of unripe cervix, caesarean section was done under spinal anesthesia ,after operation she was kept in I.C.U. for few days.

Results & Discussion: The patient developed acute renal failure, but gradual improvement of her condition was achieved and patient become well after 3 weeks with complete recovery. Other medication used was MgSO4 for fit control prevention of recurrence.

safety, high dose steroid will accelerate recovery and that conservative treatment of renal failure is so effective.

Conclusion: The result support conclusion of previous author that rapid & early diagnosis and treatment of HELLP syndrome is very important to ensure favorable maternal & prenatal outcome, also in this study we showed that the patient should be stabilized before termination & regional anesthesia will increase

Introduction

HELLP syndrome is a serious medical condition found in pregnant women usually in the third trimester of pregnancy. HELLP is acronym for; H – hemolysis, EL -elevated liver enzymes, LP - low platelets. It is said to be an off-shoot of pre-eclampsia or eclampsia. Usually it occurs before the birth of the baby, however sometimes it can occur after the delivery. HELLP syndrome is also common in women who have had the same disease during their earlier pregnancy. (1)

HELLP syndrome was first described by Pritchard et al in 1954 however the first published article naming the syndrome as HELLP appeared in literature almost thirty years later.(1,2)

Its incidence is reported as 0.2-0.6% of all pregnancies, and 10-20% of women with comorbid preeclampsia. Caucasian women over the age of 25 are usually more affected. The outcome for mothers with HELLP syndrome is generally good. With treatment, maternal mortality is about 1 percent. However complications have been observed including placental abruption,

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acute renal failure, sub capsular liver hematoma, and retinal detachment. [1, 2]

The platelet count has been found to be moderately predictive of severity: under 50,000/mm3 is class1 (severe), between 50,000 and 100,000 is class 2(moderately severe) and more than 100,000 is class d 3 (mild) .This system is termed the Mississippi classification. [3-5]

Often, a patient who develops HELLP syndrome has already been followed up for pregnancy-induced hypertension or is suspected to develop pre eclampsia. Up to 8% of all cases present after delivery. There is gradual but marked onset of headache (30%), blurred vision, malaise (90%), nausea/ vomiting (30%), band pain around the upper abdomen (65%). If patient has a seizure or coma, the condition has progressed into full-blown eclampsia. Disseminated intravascular coagulation is also seen in 20% of all women with HELLP syndrome, and in 84% when HELLP is complicated by acute renal failure. [3]

In a patient with possible HELLP syndrome, a batch of blood tests is performed: a full blood count, liver enzymes, renal function and electrolytes and coagulation studies. Often, fibrin degradation products [FDPs], determined, which can be elevated. Lactate dehydrogenate is marked of hemolytic and is elevated [more than 600U/L]. Protinuria is present but can be mild. A positive D-dimer test in the presence of pre eclapmsia has recently been reported to be predictive of patients who will develop HELLP syndrome. D-dimer is more sensitive indicator of sub clinical coagulopathy and may be positive before coagulation studies are abnormal. [3,5]

The only effective treatment is prompt delivery of the baby. Several medications have been investigated for treatment of HELLP syndrome, but evidence is conflicting as to whether magnesium sulfate decreases the seizures and progress to eclampsia. The DIC is treated with fresh frozen plasma; the anemia may require blood transfusion.

In mild cases, corticosteroids and antihypertensive [labetalol, hydralazine, nifedipine] may be sufficient. Intravenous fluids are generally required. Hepatic hemorrhage can be treated with embolization as well if life-threatening bleeding ensues. [2, 3]

Case study

The patient M.K. is a 30 years old Muslim housewife, primigravida who was booked at 16 weeks gestation. Her antenatal was poor and irregular. She developed generalize edema, headache and malaise in the third trimester. Upon admission her blood pressure was 170/110, she was pale, deeply jaundice, edema and the uterine fundal height was less than dates.

She was commenced on Apresoline 15 mg. i.v. slowly and Atenolol 100 mg. orally and sent to urgent investigation with close observation.

The initial investigation showd : PCV :28%, B+ blood group, WBC count was 20000/mm, platelets 80ooo/l, total serum bilirubin was 8 mg/dl with raised liver enzymes.

Ultrasound examination reveal signal viable fetus cephalic presentation, F.L. equal to 36 weeks and liquor volume was below the 5th percentile.

The patient developed convulsion one hour after admission , emergency treatment had been done airway was inserted ,secretion aspirated ,Oxygen was given and MgSO4 was given as an intravenous bolus over minutes ,followed by MgSO4 infusion with glucose 1 g/h. Urine catheter revealed haemauria .

Blood pressure 180/120 mm and ophthalmic examination was normal. The diagnosis was eclampsia and HELLP syndrome.

To obtain the best result, the physician and obstetrician saw the patient together (combined care) and the decision was to terminate the pregnancy after stabilization of the mother.

Magnesium sulphate 5g slowly intravenous ,oxygen .Platelet and fresh frozen plasma was given and prepare fresh blood ,ceftriaxone 1gm intravenous, dexamethasone 12mg was given and close monitoring was done after 1 hour she retain conscious ,but drowsy B.P 160/100,urine output 200 cc frank haematuria and exaggerated reflexes.

After 4 hours, the patient became fully conscious, B.P 140/85, urine still haematuria, reflex was normal, but pelvic examination revealed the cervix not ripe and tightly closes

Delivery for a healthy baby was done by lower segment caesarean section under spinal anesthesia. During operation, the hemostasis was acceptable .The patient required intensive care unit for close observation, but the patient's condition deteriorated rapidly over the next 24 hours of postnatal period.

She developed anuria with raised blood urea above 20mmol /dl, after day 3, urine output start to increase, with one week generally stable expect blood pressure still rose, but it controlled under addition treatment combination B-blocker and diuretic.

Results

28 November 2010

10.00 A.M: Blood pressure 170/100. She was sent for investigations, received apresoline ampoule 15 I.V. and tenormine 100 mg orally

11. 00 A.M: Blood pressure 180/110 ,convolution ,result PCV =-28%,TSB.= 8 mg /dl ,B+, Platelet= 80,000,blood urea is 7mmol /l ,serum alkaline phosphates= 255 u/l,SGPT= 48 U/L and magnesium sulphate 5 g I.v. over 5 minutes followed by Mgso4 infusion 1 g/h 12.00 A.M: B.P. 160/100 ,coma ,W.B.C=. 17 000 ,haemturia 200 CC urine output/day receive apresoline 15 mg , dexamethasone 12 mg,1 Unite platelet

2.00 P.M: B.P. 150 /100, Drowsy, urine output 300 CC/day haematuria, receive 2nd unit of platelet

4.00 P.M: B.P 140/80, fully conciseness, urine output 330 CC, prepare for C.S., 1st unit of blood given [fresh]

6.00 P.M: B.P. 140/90, fully conciseness, urine output 400 CC haematuria, C.S done under spinal anesthesia

8 .00 P.M: BP.150 /100, conciseness ,urine output 440 CC/day haematuria ,W.B.C.= count 20 000, platelet count= 100000 ,admitted to R.C.U ,for close observation ,receive ceftriaxone 1 gm [every 12 hourly],I.V.Fluid Glucose water 5% slowly ,

10.00 P.M: B.P. 130/80, conciseness, urine output 460 CC, receive 2nd unite of blood and dexamethasone

29/10/2010

8.00A.M: B.P 130/80,Drawsy ,urine output 50 CC hematuria,W.B.C count 22000 ,platelet 102 000,blood urea= 9 mmol/dl .S.G.P.T =60 U/L , T.S.B= 7 mg /dl ,A.L.P. [Alkaline phosphates] was 610 IU/L, .P.C.V. was 7 gm /dl .she receive ceftriaxone 1 gm twice daily,dexamethasone 8 mg twice daily ,platelet unite 3 times daily ,fresh blood 3rd unite,tenormine 100 mg orally ,and establish oral liquid d

8.00P.M: B.P.130/70 , drowsy , W.B.C count= 18 000/mm ,platelet count= 105 000/mm, urine output= 25 CC haematuria/da, blood urea= 13 mmol /dl ,T.S.B=6 gm/dl

30/10/2010

8.00A.M: BP 125/80, Drawsy, urine output 10 cc haemturia

W.B.C =16 000. Platelet =110 000 ,blood urea 18 mmol /dl, TSB =5 gm /dl, ALP =400 IU/L, SGPT =55 U/L ,receive Glucose 5% 500 cc slowly infusion ,ceftiaxone 1 gm twice daily ,dexamethasone 8 mg twice daily , dressing of wound in which there some oozing twice daily

8.00 PM: BP was 130/80 urine output =50 cc, blood urea 20mmol /dl

31/10/2010

8.00 A.M: B.P 140/85, fully conciseness, urine output 300 cc. slight haematuria, WBC =12000, PCV 28 %, TSB 4 mg/dl, ALP =330 IU/L, SGPT = 38U/L

8.00 PM: BP 140 /90 fully conciseness, urine become clear =500cc

1/11/2010

8.00 AM: BP 150/95 ,Conciseness , ,WBC =10 000, urine output 600 cc clear ,platelet =125 000 ,blood urea =20 mmol dl, TSB =3mg /dl ,ALP =152 IU/L,SGPT=27U/L , PCV =29%,Patient receive tenormin 100 mg orally ,ceftiaxone 1 gm i.v. twice daily , dexamethason 4mg twice daily, one unite of blood [fresh] given ,dres

8.00 PM: BP150/100, urine output 1200cc /day clear, add glucose water 5% 500 cc infusion

2/11/2010

8.00 AM: BP 160 /100,Urine output 800cc/day clear ,WBC =8 000 ,Platelet =125 000 ,blood urea =18 mmol/dl ,TSB =2 mg /dl ,ALP =120 IU/L , SGPT =27U/L ,receive i.v fluid G.W Twice ,ceftiaxone 1gm stopped dexamethasone ,dressing of wound, calcium ampoule infusion ,tenormine 100 mg orally

8.00 PM: BP 160/100, urine 2000 cc/day

3/11/2010

8.00 AM ;BP 170/110 ,Urine output 1000 cc /day ,WBC =8000 ,Platelet =126 000,blood urea =14 mmo/dl ,TSB =1.5 mg /dl ,ALP=300 IU ,SGPT = , receive tenormine 100 ,add Hygrton [diuretic] ,one Alfa tab. ceftiaxone 1gm ,G. W 5% Twice daily dressing of wound

8.00 PM: BP 160 /100

4/11/2010

8.00 AM: BP 150 /90

Discussion

HELLP syndrome is a life threatening obstetric complication usually associated with pre eclampsia. Its incidence is 0.5% to 0.9% of all pregnancies. It is related with severe pre eclampsia (10% to 20%). Both conditions occur after twenty weeks of gestation and may occur after 5 to 12 weeks of childbirth.

The term HELLP syndrome was first used by Louis Weinstein in 1982 [6]. Although the same clinical features and investigations were noticed in 1954 by Pritchard [2]. The onset was gradual. Clinical features include severe headache (30%), malaise (90%); nausea, vomiting (30%); band pain around upper abdomen (65%), and edema. Disseminated intravascular coagulation is also seen in about 20%of all women with HELLP syndrome [7].

The main features of the disease are de novo hypertension after the 20th gestational week and proteinuria, and it is frequently accompanied by edema and other subjective symptoms. The origin of the disease is the placenta, but its sequel affect multiple organ systems [8] that produce a number of outcomes.

Sibai has proposed strict criteria for true or complete HELLP syndrome platelet <100000, AST>70IU/L, LDH>600IU/L [9]. Our patient fell into sever type of hellp syndrome. The only effective treatment of HELLP syndrome is immediate delivery of baby either by caesarean section or normal vaginal route [9]. Vigil De Gracia et al recommended that neuraxial anaesthesia may be safely administered in the patient with HELLP syndrome without DIC or prolonged Prothrombin time [10]. We decided to perform caesarean section under spinal anaesthesia even though d-dimer and prothrombin time were marginally raised because of our past experience in similar cases. Sibai et al [9] also recommended Spinal/epidural anaesthesia in HELLP syndrome if there is hemodynamic stability as epidural anesthesia is risky in patients with coagulopathy. This case was operated under spinal anesthesia because patient was hemo- dynamically stable. Administration of corticosteroid can minimize the degree of intravascular endothelial injury and improve blood flow while decreasing hepatocyte and platelet consumption in HELLP syndrome [11].

O Brien et al assessed the beneficial effects of steroids on the frequency of complications after regional anesthesia [12].

Conclusion

The result supports the conclusion of previous authors that rapid and early diagnosis of HELLP syndrome and the treatment of the symptoms are very important to ensure a favorable maternal and perinatal outcome. Also stabilization of patient with eclampsia before termination by operation under regional anesthesia will increase safety. High dose steroid will accelerate recovery and that conservative management of renal failure of HELLP syndrome is so effective.

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