An Assessment of Twelve Cases of HELLP Syndrome Treated at the King Fahad Central Hospital, Gizan, Saudi Arabia

Jacob A Unuigbe[1] *and Poonam Misra*[1]

ABSTRACT

A total of 11,051 deliveries were conducted during a period spanning four years, from July 1993 to June 1997. Twelve patients presented with the HELLP syndrome, a high incidence of 1.0 per 1000 deliveries. A significant proportion (75.0 per cent) of these women was multiparous with a mean age of 27.6 years, and majority (83.3 per cent) had preterm deliveries. Prominent presenting clinical features included preeclampsia (proteinuria and hypertension), eclampsia, jaundice, epigastric pain, anorexia and malaise. Relevant laboratory profiles on all patients met the criteria for confirmation of the diagnosis of HELLP syndrome. Important complications were disseminated intravascular coagulation, acute renal failure, transient cortical loss of vision, pulmonary oedema and transient nephrogenic diabetes insipidus. All patients required supportive care in the hospital's intensive care unit at the peak of their illnesses, and essential treatment consisted of control of fits and hypertension, transfusion of blood, fresh frozen plasma and albumin. Although, fortunately, there was no maternal death in spite of the severe and often stormy maternal morbidity, a very high perinatal mortality rate of 308 per 1000 was recorded. (*Afr J Reprod Health* 1999; 3(2):68-78)

RÉSUMÉ

Une évaluation de douze cas du syndrome du HELLP traités à l'hôpital central King Fahad à Gizan en Arabie Saoudite. Au total il y a eu 11, 051 accouchements pendant une période couvrant quatre ans, à compter du juillet 1993 au juin 1997.Douze patientes souffraient du syndrome du HELLP avec un taux élevé de 10 pour 1000 accouchements. Une bonne proportion de ces femmes (75%) étaient des multipares gées en moyenne de 27.6 ans alors que la majorité (83, 3%) avaient eu des accouchements avant terme. Parmi les importantes manifestations cliniques constatées étaient la prééclampsie (la proteinurie et l' hypertension), l'éclampsie, la jaunisse, les douleurs épigastriques, l'anoxerie et le malaise). Des profils laboratoires pertinants ont permis de confirmer le diagnostic du syndrome du HELLP chez toutes les patientes. De graves complications observées étaient: la coagulation introvasculaire disseminée, l'insuffisance rénale aiguë, la perte corticale de vision, l'oedème pulmunaire, la diabète insipide néphrogénique. Toutes les patientes ont bénéficié des soutiens dans l'unité de soins intensifs de l'hôpital au sommet de leur maladie. Le traitement essentiel était la maîtrise des crises et de l'hypertension, de transfusion sanguine, du plasme frais et de l'albumine. II n'y avait pas de décès maternel; mais malgré une morbidité maternelle grave, on a enrigistré un taux de mortalité périnatale de 308 pour 1000. (*Rev Afr Santé Reprod* 1999:3(2):68-78)

KEY WORDS: *HELLP syndrome, King Fahad Central Hospital, pre-eclampsia-eclampsia*

[1]*Department of Obstetrics and Gynaecology, King Fahad Central Hospital, Gizan, Kingdom of Saudi Arabia*

Correspondence: *Dr Jacob Unuigbe, Department of Obstetrics and Gynaecology, King Fahad Central Hospital, P. O. Box 204, Gizan, Saudi Arabia*

Introduction

HELLP syndrome is an acronym for haemolysis, elevated liver enzymes and low platelets. Even though these pathological changes associated with pregnancies complicated by hypertension have been noted in literature over the past hundred years,[1] the combination of abnormalities was formally documented in 1954 by Pritchard *et al.*[2] In 1982, Weinstein popularised the syndrome in his published article on the subject, initiating the use of the acronym HELLP for the first time.[3]

A summary of the pathology of HELLP syndrome is presented in Figure 1. The hallmark of the pathology of the syndrome, similar to that of pre-eclampsia-eclampsia, (leading to the often assumed conclusion that the former is an extension or a variant of the latter),[2,4] is microvascular endothelial damage, intravascular platelet activation and release of thromboxane A2, leading to extensive vasospasm, of which the most harmful pathological consequences affect the arterioles and capillaries. Prostacycline deficiency and increased platelet aggregation, secondary to thromboxane excess, causes destruction of platelets, leading to severe thrombocytopenia.[5] The net result of these pathological changes is the classic multiorgan involvement. However, the liver is often the primary target organ of major pathological sequelae in the HELLP syndrome. There are hepatic periportal or focal parenchymal necrosis, large hyaline deposits of microthrombi and fibrinogen deposits in otherwise normal sinusoids. These changes lead to hepatocellular necrosis, which in severe cases can result in bleeding into intrahepatic space or, in very rare cases, liver rupture or subcapsular haematoma.

The importance of HELLP syndrome in relation to pre-eclampsia-eclampsia is best visualised by considering maternal mortality. Globally, hypertensive disorders, notably pre-eclampsia-eclampsia, constitute one of the five most important causes of maternal deaths (currently more than 580,000 annually), the others being obstructed labour, genital sepsis, illicit abortion and haemorrhage. The 1994–1996 triennial confidential enquiries into maternal deaths in the United Kingdom lists hypertensive diseases as the second leading direct cause of maternal deaths.[6] In Saudi Arabia, of the 105 direct obstetric maternal deaths analysed in the first national triennial study from 1989 to 1992,[7] hypertensive diseases came third (13 deaths), behind haemorrhage and thromboembolism. Most analyses of these unfortunate deaths, while recognising the important aetiological role of hypertension, pre-eclampsia and eclampsia, do not often emphasise their close association with what many consider the most lethal consequence (or variant form) of these disorders, the HELLP syndrome. This issue appears important to the authors who in the past two years have treated a number of gravely ill women with severe pre-eclampsia-eclampsia complicated by the HELLP syndrome. A careful review was therefore conducted to determine the incidence and the maternal and perinatal outcome of patients treated for HELLP syndrome in King Fahad Central Hospital, Gizan, Saudi Arabia.

Patients and Methods

The Gizan Region is located in the southwestern part of Saudi Arabia, with the Republic of Yemen and the Red Sea on its southern and western boarders respectively. It has partly sub-urban but mostly rural population of about 650,000 inhabitants who are predominantly Saudis, with a very small percentage of expatriates of various nationalities. The King Fahad Central Hospital (KFCH), Gizan, is a tertiary level referral centre for the 12 peripheral secondary level hospitals and 131 primary health centres in the region. Only about 10 per cent of the obstetric population of women receive elective antenatal care in KFCH while the majority of women are referrals from the peripheral hospitals and health centres. Thus, the patients treated in KFCH represent majority of the high-risk obstetric population of the Gizan region.

This study covers a review of perinatal care and deliveries conducted at the KFCH from July 1, 1993, to June 30, 1997. From the records of deliveries, women who had hypertension, including pre-eclampsia-eclampsia, were identified. Various laboratory parameters were considered. Those who had inclusion criteria for establishing the diagnosis of HELLP syndrome were critically analysed. The last 18 months of the study was a prospective study during which the two authors followed up patients during their treatment in the hospital, utilising management protocol designed during the earlier

part of the study. The inclusion criteria for cases of HELLP syndrome were:

1. Evidence of intravascular haemolysis — abnormal peripheral smear, worsening thrombocytopenia and/or increase lactate dehydrogenase, and hyperbilirubinaemia (>25 mmol/L).
2. Evidence of hepatic dysfunction — increased levels of aspartate aminotransferase (>48 iu/L) and lactate dehydrogenase (>200 iu/L).
3. Thrombocytopenia, defined as platelet concentration less than 100×10^9/L.

In analysing the cases of HELLP syndrome, the following were considered: sources of referral, nationality, age, parity, gestation, mode of delivery, clinical presentation, laboratory profiles, maternal complications, management and perinatal outcome. The data were subjected to simple analysis.

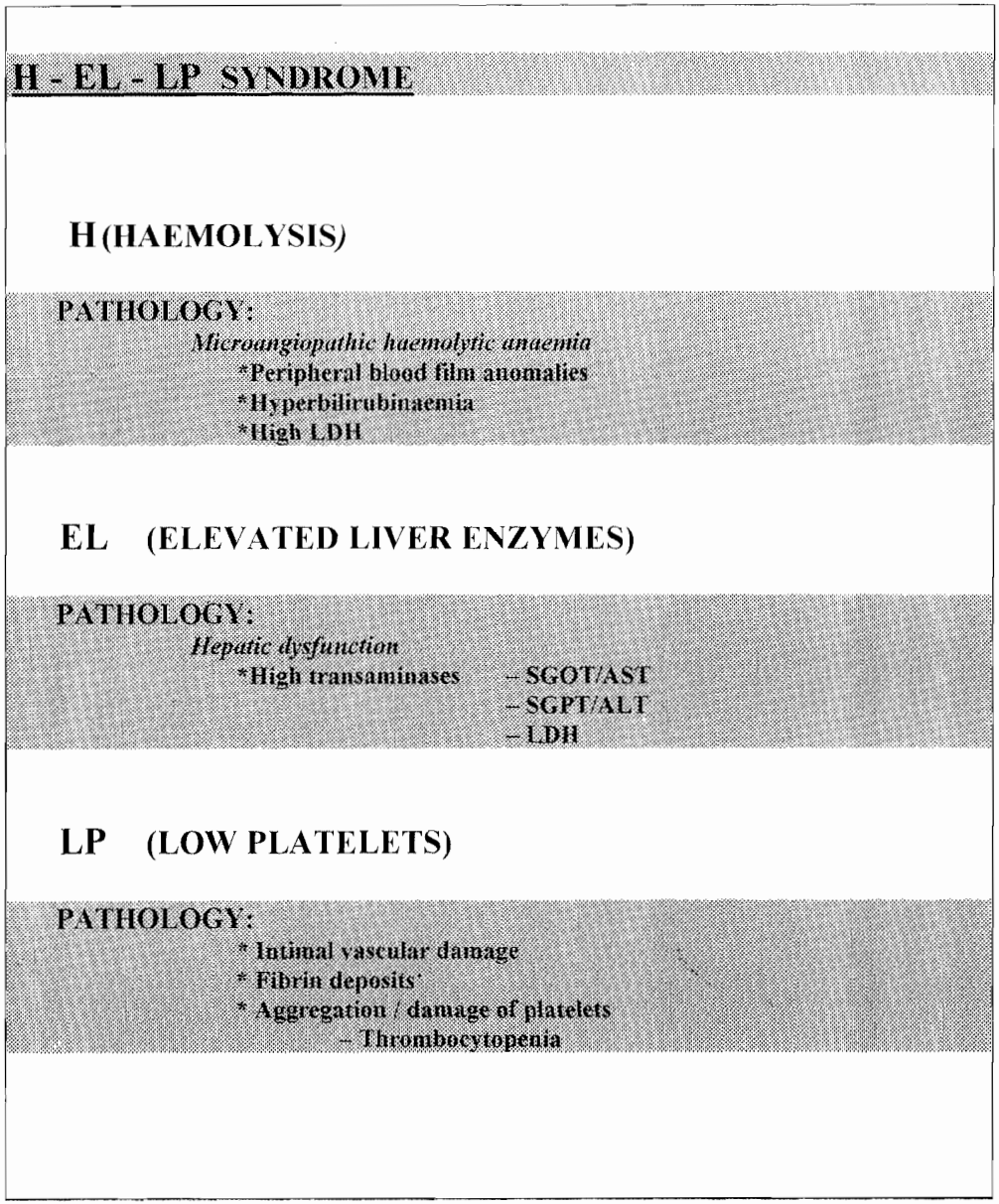

Figure 1 **Pathology of HELLP Syndrome**

Results

A total of 11,051 deliveries were recorded in KFCH during the four-year period, from July 1, 1993 to June 30, 1997. Of these, either pre-eclampsia or eclampsia complicated 483 (4.4 per cent) deliveries. A total of 82 women (0.7 per cent) had eclampsia, with 67 per cent of them developing in the early puerperium. Twelve patients (one per 1,000 of the delivery population and 2.3 per cent of those complicated by pre-eclampsia/eclampsia) met the criteria for HELLP syndrome. There was an even distribution of cases from amongst the referral general hospitals, with a slightly increased number of referrals from one general hospital that serves the mountainous area of the region with difficult access to medical facilities.

Table 1 shows some demographic characteristics of the 12 patients with HELLP syndrome. Nine women were Saudis. The ages of the 12 women ranged between 20 and 38 years (mean 27.6 years) and majority (75 per cent) were multiparous. Only two women (16.7 per cent) had term deliveries, while significantly 67 per cent had their babies before 32 weeks gestation. Five patients had assisted deliveries and the caesarean section rate was 33.3 per cent. The caesarean section rate for the entire obstetric population during the period was 14.6 per cent.

Table 2 shows the maternal clinical presentation. There was no difference in the severity of illness in primipara or multipara. There was varying degree of proteinuria in all the cases. Majority of the patients (83.3 per cent) were hypertensive. Two women were normotensive. Jaundice was an important clinical feature, so much so that one patient was referred through the liver unit of KFCH. Eclampsia complicated eight cases (67.7 per cent). Other important clinical features include epigastric pain, anorexia, malaise and pedal oedema.

Table 1 **Demographic Characteristics of Patients with HELLP Syndrome**

	Number of patients	Percentage
Saudis	9	75.0
Non Saudis	3	25.0
Age		
Range	20-38 years	
Mean	27.6 years	
Parity		
Primipara	3	25.0
Multipara 1-4	4	33.3
5 or more	5	41.7
Gestation (weeks)		
Less than 24	0	0
24-27	2	16.7
28-31	6	50.0
32-36	2	16.7
Term, 37 or more	2	16.7
Mode of delivery		
Normal vaginal delivery	7	58.3
Vaccuum delivery	1	8.3
LSCS	4	33.4

Table 2 HELLP Syndrome: Maternal Clinical Presentation

Patient order	Party (P)	Age	Epigastric pain	Anorexia	Jaundice	Pedal oedema	Eclamptic fits	Blood pressure	Proteinuria (mg/dl)
1	P11	38	+	+	+	+	–	140/100	100
2	P1	22	+	–	+	+	–	100/70	100
3	P4	30	–	–	–	+	+	200/120	500
4	P0	20	+	–	+	–	+	210/110	100
5	P0	20	+	+	+	+	+	160/112	100
6	P0	35	+	+	+	+	+	130/92	100
7	P2	28	–	–	+	–	+	180/120	500
8	P4	24	–	+	+	+	+	110/80	100
9	P5	25	+	–	+	+	+	139/114	500
10	P10	35	–	–	–	+	+	160/114	500
11	P5	27	+	–	–	+	–	150/100	100
12	P6	30	+	–	–	+	–	180/130	100

The detailed laboratory profile of the 12 patients is shown in Table 3. Majority (58.3 per cent) of the women was anaemic at the time of admission. After admission, there was generally a downward trend in haemoglobin levels until the second day postpartum, after which a plateau effect was observed. Lactate dehydrogenase (LDH) demonstrated the most dramatic rise in serum levels (up to 7139 iu/l). Aspartate aminotransferase (AST), alkaline phosphatase and bilirubin levels were raised in all the 12 patients. The haematological profiles (platelets, white cell count and peripheral blood smears) of the patients were assessed. A total of six women had their lowest platelet levels as $<50 \times 10^9/l$, and majority (nine) of all the patients reached platelet level of $100 \times 10^9/l$ within five days of delivery, the longest interval with one patient being eight days. Of the five peripheral blood smears performed, only two showed evidence of haemolysis (Howell-Jolly bodies, burr cells, polychromasia). Majority of patients had significantly elevated wide range of white cell count.

Abdominal (liver, spleen, kidneys and uterus) ultrasonic scans in all but one patient and abdominal computerised tomography scan (CT scan) in two patients showed no gross pathological changes. The most common serious complication was disseminated intravascular coagulation (DIC), which affected five patients. Other serious complications include two cases of acute renal failure and one case each of transient cortical loss of vision, pulmonary oedema and transient nephrogenic diabetic insipidus.

Table 3 Laboratory Profiles amongst Patients with HELLP Syndrome

Patient order	Day	Haemoglobin (10-14 g/dl)	Bilirubin (10-17 mmol/l)	Aspartate amino transferase (8-37 iu/l)	Lactate dehydrogenase (100-190 iu/l)	Alkaline phosphatase (50-135 iu/l)
	Pre-delivery	8.0	405	135	717	394
1	1	6.1	95	55	417	266
	2-4	6.6	34	44	349	207
	5-7	7.1	25	30	200	152
	Pre-delivery	–	–	–	–	–
2	1	5.5	180	120	355	261
	2-4	8.1	153	76	447	179
	5-7	9.5	101	67	506	274
	Pre-delivery	8.9	83	1015	503	143
3	1	6.0	40	318	232	101
	2-4	6.2	13	72	211	110
	5-7	6.2	10	52	NA	90
	Pre-delivery	5.0	76	2554	3024	170
4	1	7.9	183	4358	4123	120
	2-4	8.0	90	82	830	124
	5-7	8.0	82	70	456	90
	Pre delivery	–	–	–	–	–
5	1	6.1	95	436	1493	178
	2-4	8.5	30	162	713	170
	5-7	9.5	14	44	2600	136
	Pre delivery	12.1	95	1504	2600	347
6	1	10.3	128	930	2727	271
	2-4	8.9	39	236	689	175
	5-7	9.7	30	171	512	106
	Pre-delivery	–	–	–	–	–
7	1	11.7	106	941	1684	336
	2-4	8.5	118	465	1340	313
	5-7	8.2	284	155	148	206
	Pre-delivery	8.7	78	1381	483	124
8	1	8.0	82	1281	480	126
	2-4	8.9	46	750	322	110
	5-7	8.8	22	216	168	92

Table 3 (Continued)

Patient order	Day	Haemoglobin (10-14 g/dl)	Bilirubin (10-17 mmol/l)	Aspartate amino transferase (8-37 iu/l)	Lactate dehydrogenase (100-190 iu/l)	Alkaline phosphatase (50-135 iu/l)
	Pre-delivery	–	–	–	–	–
9	1	10.3	119	146	7139	289
	2-4	8.1	133	580	1212	514
	5-7	9.2	85	726	922	441
	Pre-delivery	–	–	–	–	–
10	1	12.2	32	201	1352	–
	2-4	10.4	24	133	499	–
	5-7	9.3	17	50	243	–
	Pre-delivery	9	40	120	502	261
11	1	7.9	30	76	–	199
	2-4	8.0	20	72	340	110
	5-7	8.1	18	65	104	96
	Pre-delivery	10.8	71	120	752	141
12	1	9.6	122	75	700	141
	2-4	9.7	34	72	552	130
	5-7	9.5	32	70	103	92

Table 4 **Management of Patients with HELLP Syndrome**

Management procedure*	Number of patients	Percentage
Control of fits, treatment of hypertension, and active supportive intensive care	9	75.0
Blood transfusion	11	91.6
Fresh frozen plasma transfusion	9	75.0
Albumin transfusion	1	9.1

*All patients required supportive care and monitoring in the hospital's intensive care unit at the peak of their illnesses, the duration of intesive care unit confinement ranging from12 to 144 hours (mean 34.5 hours).

Table 4 provides a summary of the management of the patients. All patients required supportive care and monitoring in the hospital's central intensive care unit at the peak of their illnesses, the duration of confinement ranging from 12 to 144 hours (mean 34.5 hours). Eclamptic fits were con-

trolled by bolus intravenous injection of diazepam (10mg) followed by intravenous infusion of 5 per cent dextrose in water containing 25mg of diazepam, delivered at a rate of 1.5mg per minute until the patient was drowsy, up to a total dose of 10-30mg diazepam. Infusion was then continued at a rate of 2.5-10mg diazepam per hour to maintain drowsiness until 24 hours after delivery or the last fit. Seven (87.5 per cent) of the patients with fits required ventilation for variable periods and they all had central venous pressures monitored. Acute hypertensive crisis was controlled with sublingual nifedipine or parenteral hydrallazine. All the patients had regular clinical assessment and, at least once daily, laboratory investigations of the liver, renal and coagulation functions during this critical period. Blood and blood products were transfused to correct anaemia and DIC. One patient was transfused with albumin to correct severe hypoalbuminaemia.

A summary of the obstetric management and perinatal outcome is presented in Table 5. Five patients were referred after delivery, from the peripheral general hospitals. Two babies were stillborn; one of which was the second of a set of twins. Five of the six babies delivered in KFCH were transferred to the hospital's neonatology unit and ventilated for periods ranging from 24-48 hours. With a total of four perinatal deaths, the perinatal mortality rate (PNMR) of 308 per 1000 was far in excess of the hospital's PNMR of 37.9 per 1000 total births during the study period. Prematurity was the most common contributive factor to the perinatal deaths.

Table 5 **HELLP Syndrome: Pregnancy Duration, Method of Delivery and Perinatal Outcome**

Patient order	Gestation (weeks)	Method of delivery	Birth weight (g)	Apgar scores (1, 5, 10 mins)	Complications and *perinatal outcome
1	28	LSCS (KFCH)	1210	4, 7, 7 alive and well	RDS, ventilated for 48 hours,
2	32	SVD (Sabya GH)	2000	–	Alive and well
3	30	LSCS (KFCH)	1920	4, 7, 8	Prematurity, mild birth asphyxia, ventilated for 24 hours, alive and well
4	37	Vaccuum delivery (KFCH)	3050	3, 5, 6 and well	Birth asphyxia, ventilated for 24 hours, alive
5	29	SVD (Abu Arish GH)	1160	–	Died on 5th day
6	26	SVD (KFCH)	1110	–	Stillborn (IUFD)
7	27	SVD (Gizan GH)	1120	–	Alive and well
8	.29	LSCS (KFCH)	(1) 1400	1, 2, 3	Ventilated, died within 24 hours
		Twins			
			(2) 1110	–	Stillborn, (IUFD)
9	36	SVD (Sabya GH)	2120	–	Alive and well
10	37	SVD (Gizan GH)	2500	–	Alive and well
11	30	SVD (KFCH)	1900	6, 8, 9	Ventillated for 48 hours. Alive and well
12	30	LSCS (KFCH)	1350	7, 9, 10	RDS on second day. Ventillated for 72 hours. Alive and well

** Perinatal deaths – 4/13; Perinatal mortality rate – 308 per 1000*

Discussion

The incidence of HELLP syndrome reported here is high in comparison with most studies,[8,9,10] including a study from Jeddah, Saudi Arabia,[11] with a reported incidence of one per 2285 deliveries. This high incidence reflects on KFCH as the only tertiary referral centre for all high-risk medical conditions in the Gizan Region. The incidence should therefore be considered more or less a regional compilation of cases of HELLP syndrome. The variable incidence and resulting controversies encountered in literature is explained by the variable clinical expressions of the syndrome and the differences in diagnostic criteria applied in different studies.[8] As with this study, most investigators[12,13] adopt Weinstein's criterion[3] of a platelet count of $<100 \times 10^9/l$ as indicating thrombocytopenia. Patients with platelets up to $150 \times 10^9/l$ are included in some other studies, based on Martin's (Types 1, 2 and 3) classification,[14] and these report higher incidence of HELLP syndrome. A significant proportion of patients develops the HELLP syndrome in the puerperium[10,15] with onset from a few hours to seven days postpartum, and majority occur within 40 hours.

The women with HELLP syndrome in this study were significantly older than women with pre-eclampsia-eclampsia without features of HELLP syndrome, and majority was multipara. Similar findings have been highlighted by Weinstein,[16] Barton and Sibai[17] and Martin *et al.*[9] The shorter gestation at pregnancy termination among the patients supports the observation by previous researchers,[9,10] that severe pre-eclampsia and eclampsia are likely to be associated with HELLP syndrome at earlier gestations, with implied potentials for increased perinatal loss.

The common clinical features that normally characterise pre-eclampsia-eclampsia (proteinuria, oedema and hypertension) were expectedly present in majority, if not all, of the patients. It is pertinent to note that a significant number of the patients had additional symptoms of malaise, anorexia and epigastric pain. Sibai[10] and Tomsen[18] have stressed the tendency of some patients to present with only vague symptoms, especially anorexia and malaise, during late pregnancy, thus causing delay with making the correct diagnosis. General physicians and surgeons thus need to be aware of the possibility of HELLP syndrome in all pregnant women under their care. This is particularly important, as some women may be normotensive at presentation, as was the case with 16.7 per cent of the patients studied. Hypertension may or may not develop at a later stage in such patients.[9,15,19] The development of fits and jaundice (evidence of pathological hepatic changes) represents the severest stage of the illness that must be taken very seriously to prevent maternal mortality. Hence, all patients in this series were invariably given ultimate intensive care.

The Gizan population is generally anaemic, with haemoglobin concentrations in the range of 8.5 to 10 gm/dl among majority of the obstetric patients at booking. Fortunately, further, anaemia and haemolysis in HELLP patients appeared to be neutralised by haemopoeisis, and hence the observed plateau effect on the serial haemoglobin levels within two days postpartum. Similar findings are reported by Martin *et al.*[15] Extremely high liver enzymes, notably LDH and AST, have been reported to positively correlate with extreme maternal morbidity and mortality.[20] However, such elevation does not necessarily always reflect liver damage. Haemolysed red blood cells constitute a rich source of AST, LDH and bilirubin.[13,15] So also do the release and elevation of creatinine phosphokinase (CPK), secondary to muscle injury resulting from impaired microcirculation in HELLP syndrome, cause elevated levels of ALT and AST.[21,22] There is not much significance with the elevated levels of alkaline phosphatase, as indeed it is the case with all pregnancies.

During pregnancy, white cell count is physiologically elevated. With the stress of HELLP syndrome, complicated by infections in many patients, it is not surprising that majority of women had overwhelming leucocytosis in this study. Spontaneous postpartum resolution of thrombocytopenia appears to be the rule, and this is exhibited among these patients in accord with similar findings by previous authors.[23-25] The longer time for platelet recovery in patients with a nadir below $50 \times 10^9/l$ seems to be directly proportional to the greater drop in platelet count in this group of patients.

Abdominal ultrasound and, when available, CT scan are very important in excluding such gross pathological lesions as subcapsular liver haematoma and intraperitoneal haemorrhage or ascites. The use of CT scan or magnetic resonance imag-

ing (MRI) is particularly useful in the differential diagnosis of HELLP syndrome and acute fatty liver of pregnancy (AFLP).[26] Other thrombotic microangiopathic haemolytic disorders such as thrombotic thrombocytopenic purpura (TTP) and adult haemolytic uraemic syndrome (HUS), which are often considered differential diagnosis of HELLP syndrome, predominantly affect the central nervous and renal systems respectively. HUS is commonly a childhood disorder.

DIC is a recognised common complication of HELLP syndrome[17] that featured prominently in this study. Transient nephrogenic diabetes insipidus, which complicated one of the cases, is a very rare complication.[27] This pathology is characterised by a resistance to antidiuretic hormone mediated by excessive vasopressinase secondary to impaired hepatic metabolism of the enzyme. In spite of the stormy maternal morbidity among the patients, there was no maternal mortality. In a series reported by Martin *et al*,[9] maternal mortality was 3.6 per cent. The quoted range of maternal mortality is from 1.1 to 24.2 per cent. [28]

There was an aggressive approach to the management of patients in this study because of the severity of the HELLP syndrome treated. Intensive supportive care and immediate termination of pregnancy by caesarean section, if necessary, are measures advocated by majority of clinicians.[29-31] When the patient's clinical condition is considered less severe, a few patients with extreme prematurity can be treated with corticosteroids for 24 hours before pregnancy is terminated. Temporising management, some of which consist of treatment with volume expanders including plasmapheresis, pharmacological vasodilators and varying doses of corticosteroids, constitutes the subject of discussion by many authors.[32-35] These measures did not feature much in this study because of the severity of the patients' illnesses.

The combination of severity of illness and the high proportion of preterm deliveries explain a high PNMR of 308 per 1000 in this review. In a recent review by Geary,[28] the quoted perinatal mortality rates were similarly very high with a range of 77 to 370 per 1000. In a careful critical review of women with HELLP syndrome and neonatal outcome, Harms *et al*[36] reported an overall PNMR of 56 per 1000 and significantly relatively high incidence of leukopenia, neutropenia and thrombocytopenia among the very low birthweight neonates. Because such severe complications as severe pre-eclampsia-eclampsia tend to complicate HELLP syndrome at earlier gestational age,[9] the grave implications of the above findings[36] on perinatal outcome are evident. In conclusion, a high incidence of severe HELLP syndrome in the Gizan Region of Saudi Arabia is reported. Active management with supportive intensive care formed the bedrock of management of majority of the patients in order to prevent more serious maternal morbidity and mortality. Extremely high perinatal mortality was sequel to unavoidable recourse to preterm termination of pregnancy because of severity of the mother's illness. Variable clinical presentation (anorexia, malaise and epigastric pain) seen in a significant number of the patients can result in delay of accurate diagnosis or inappropriate medical and surgical treatment. Obstetricians should therefore cultivate higher index of suspicion when patients present with these vague symptoms during the second half of pregnancy.

REFERENCES

1. Goodlin RC. Beware of the great imitator — severe pre-eclampsia. *Contemp Obstet Gynecol* 1982; 20: 215–228.
2. Pritchard JA, Weisman R, Ratnoff OD, *et al.* Intravascular haemolysis, thrombocytopenia and other haematologic abnormalities associated with severe toxaemia of pregnancy. *N Eng J Med* 1954; 250: 89–98.
3. Weinstein L. Syndrome of haemolysis, elevated liver enzymes and low platelet count: a severe consequence of hypertension in pregnancy. *Am J Obstet Gynecol* 1982; 142: 159–167.
4. Chesley LC. Diseminated intravascular coagulation. In Chesley LC, (Ed). *Hypertensive disorders in pregnancy.* New York: Appleton-Century-Crofts. 1978: 88–118.
5. Van Asche FA and Spitz B. Thromboxane synthetase inhibition in pregnancy induced hypertension. *Am J Obstet Gynecol* 1988; 159: 10–15.
6. Department of Health: Why mothers die. Report on confidential enquiries into maternal deaths in the United Kingdom, 1994–1996. London, HM stationery office, 1998.
7. Maternal mortality in Saudi Arabia, 1410–1412 Hegira (August 1989–June 1992). King Saud University Press, 1993.
8. Magann EF and Martin JN. Complicated postpartum pre-eclampsia-eclampsia. *Obstet Gynecol Clin North Am* 1995; 22: 337–356.

9. Martin JN Jr, Perry KG Jr, Miles JF, *et al.* The inter-relationship of eclampsia, HELLP syndrome and prematurity: co-factors for significant maternal and perinatal risk. *Br J Obstet Gynaecol* 1993; 100: 1095–1100.
10. Sibai BM. The HELLP syndrome (haemolysis, elevated liver enzymes and low platelets): much ado about nothing? *Am J Obstet Gynecol* 1990; 162: 311–316.
11. Barlas S, Al Mobarak MF, Shaheen TA, *et al.* HELLP syndrome: clinical profile of seven patients. *Ann Saudi Med* 1996; 16: 159–161.
12. Thiagarajah S, Bougeois FJ, Harbert GM, *et al.* Thrombocytopenia in pre-eclampsia: associated abnormalities and management principles. *Am J Obstet Gynecol* 1984; 150: 1–7.
13. Sibai BM, Taslim MM and El-Nazer A. Maternal-perinatal outcome associated with the syndrome of haemolysis, elevated liver enzymes and low platelets in severe pre-eclampsia-eclampsia. *Am J Obstet Gynecol* 1986; 155: 501–509.
14. Martin JN Jr, Blake PG, Lowry SL, *et al.* Pregnancy complicated by pre-eclampsia-eclampsia with the syndrome of haemolysis, elevated liver enzymes and low platelet count: how rapid is postpartum recovery? *Obstet Gynecol* 1990; 76: 737–741.
15. Martin JN Jr, Blake PG, Perry KG, *et al.* The natural history of HELLP syndrome: patterns of disease progression and regression. *Am J Obstet Gynecol* 1991; 164: 1500–1513.
16. Weinstein L. Pre-eclampsia-eclampsia with haemolysis, elevated liver enzymes and thrombocytopenia. *Obstet Gynecol* 1985; 66: 657–660.
17. Barton JR and Sibai. Care of the pregnancy complicated by HELLP syndrome. *Obstet Gynecol Clin North Am* 1991; 18: 165–179.
18. Tomsen TR. HELLP syndrome (haemolysis, elevated liver enzymes and low platelets) presenting as generalized malaise. *Am J Obstet Gynecol* 1995; 172: 1876–1880.
19. Magann EF, Chauhan SP, Naef RW, *et al.* Standard parameters of pre-eclampsia: can the clinician depend on them to reliably identify the patient with the HELLP syndrome? *Aust NZ J Obstet Gynaecol* 1993; 33: 122–126.
20. Catanzarite VA, Steiberg SM, Mosley CA, *et al.* Severe pre-eclampsia with fulminant and extreme elevation of aspartate aminotransferase and lactate dehydrogenase levels: high risk for maternal death. *Am J Perinatol* 1995; 12(5): 310–313.
21. Dreyfus JC and Schapiro G. Serum enzymes in pathophysiology of muscle. *Ann NY Acad Sci* 1959; 75: 235–241.
22. McMahon LP, O'Coigligh S and Redman CWG. Hepatic enzymes and the HELLP syndrome: a long-standing error? *Br J Obstet Gynaecol* 1993; 100: 693–695.
23. Katz VL, Thorp JM, Rosas L, *et al.* The natural history of thrombocytopenia associated with pre-eclampsia. *Am J Obstet Gynecol* 1990; 163: 1142–1143.
24. Neiger R, Contag SA and Coustan DR. The resolution of pre-eclampsia-related thrombocytopenia. *Obstet Gynecol* 1991; 77: 692–695.
25. Chandran R, Serra-Serra V and Redman CWG. Spontaneous resolution of pre-eclampsia-related thrombocytopenia. *Br J Obstet Gynaecol* 1992; 99(11): 887–890.
26. Farine D, Newhouse J, Owen J, *et al.* Magnetic resonance imaging and computed tomography scan for the diagnosis of acute fatty liver of pregnancy. *Am J Perinatol* 1990; 7: 316–319.
27. Mabie MC and Sibai BM. Transient diabetes insipidus in a patient with pre-eclampsia and HELLP syndrome. In Cefalo RC, (Ed). *Clinical decisions in Obstetrics and Gynecology.* Rockville, Maryland, Aspen, 1990, pp 136–138.
28. Geary M. The HELLP syndrome. *Br J Obstet Gynaecol* 1997; 104: 887–891.
29. Pillay M and Moodley J. The HELLP syndrome (haemolysis, elevated liver enzymes, low platelet count) in severe hypertension crisis of pregnancy — does it exist? *S Afr Med J* 1985; 67: 246–248.
30. Rath W, Loos W, Kulu W, *et al.* The importance of early laboratory screening methods for maternal and fetal outcome in cases of HELLP syndrome. *Eur J Obstet Gynecol Reprod Biol* 1989; 36: 43–51.
31. Van Dam PA, Reiner M, Baeckelandt M, *et al.* Disseminated intravascular coagulation and the syndrome of haemolysis, elevated liver enzymes and low platelets in severe pre-eclampsia. *Obstet Gynecol* 1989; 73: 97–102.
32. Heyborne KD, Burke MS and Porreco RP. Prolongation of premature gestation in women with haemolysis, elevated liver enzymes and low platelets: a report of five cases. *J Reprod Med* 1990; 35: 53–57.
33. Magann EF, Bass D, Chauhan SP, *et al.* Antepartum corticosteroids: disease stabilization in patients with the syndrome of haemolysis, elevated liver enzymes and low platelets (HELLP). *Am J Obstet Gynecol* 1994; 171: 1148–1153.
34. Katz VL, Watson WJ, Thorp JM, *et al.* Treatment of persistent postpartum HELLP syndrome with plasmapheresis. *Am J Perinatol* 1992; 9: 120–122.
35. Visser W and Wallenburg HC. Temporizing management of severe pre-eclampsia with and without the HELLP syndrome. *Br J Obstet Gynaecol* 1995; 102: 111–117.
36. Harmis K, Rath W, Herting E, *et al.* Maternal haemolysis, elevated liver enzymes, low platelet count and neonatal outcome. *Am J Perinatol* 1995; 12(1): 1–6.