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INTRAPARTUM ECLAMPSIA: A CASE SERIES PRESENTED AT TERTIARY CARE HOSPITAL.

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ABSTRACT: Preeclampsia is the complex disorder characterized by hypertension and proteinuria. Preeclampsia if complicated can progress to eclampsia, endangering life of both mother and fetus. Eclampsia occurring during delivery is referred as intrapartum eclampsia. Few studies have been conducted on intrapartum eclampsia, as it is less frequent in developed countries due to the availability of better health care facilities and awareness. Here we report a case series of patients presented with intrapartum eclampsia presented at a tertiary care hospital.

Key words: Case Series, Hypertension, Intrapartum Eclampsia, Preeclampsia, Proteinuria

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INTRODUCTION

Preeclampsia is characterized by a rise in blood pressure (systolic ≥ 140 mm Hg and diastolic ≥ 90 mm Hg) and development of proteinuria after 20 weeks of gestation. It may progress to eclampsia when patients develop fits along with hypertension and proteinuria.[1] Eclampsia occurring before delivery is referred as antepartum eclampsia whereas eclampsia occurring earlier in puerperium or after delivery is denoted as postpartum eclampsia. Although less frequent if the patient presents with eclamptic fits during delivery, it is referred as intrapartum eclampsia.[2,3] Four cases of intrapartum eclampsia presented at the department of Gynaecology and Obstetrics unit 1, Liaquat University of medical and health sciences Jamshoro during 1st June 2014 to 31st May 2015.

CASE 1

A 40 year old P_{14}^{+0} admitted after being referred from taluka hospital due to the intrapartum fits. She was admitted at a local hospital with 38 weeks of gestation and uterine contractions. The patient did not have antenatal visits during entire pregnancy. She had normal vaginal deliveries previously without obstetrical complications. Her past medical and obstetric histories were insignificant. Alive baby girl delivered weighing 2800 g with APGAR scores of 5 and 7 at 1 and 5 min, respectively at taluka hospital. Following her delivery she again developed generalized seizure and was referred to tertiary hospital for further management. On arrival her blood pressure recorded as 200/110 mmHg along with proteinuria (3+) on dipstick. An intravenous bolus of labetalol 20 mg was given. Magnesium sulphate (MgSO4) administered at a loading dose of 4g in 15-20 minutes and a maintenance dose of 1g per hour for 24 hours as a continuous intravenous infusion. Later patient did not develop seizures and her blood pressure decreased on subsequent maintenance dose of labetalol. She was monitored and discharged on the fifth

postpartum day.

CASE 2

A 25 years patient $G_2P_1^{+0}$ admitted with regular uterine contractions at 36 weeks of gestation. She had four antenatal visits that were normal and blood pressure recorded as 120/80 to 100/80 mmHg. Her medical and past obstetric history was insignificant. On admission, blood pressure recorded as 150/90 mmHg with traces of proteinuria (dipstick). A patient's ultrasound revealed dead baby with hydrocephalus without cardiac and cord pulsations. Abdominal and vaginal examination findings were normal with 6 cm os dilatation. The patient was monitored for progress of labour in pre labour room where she suddenly developed generalized convulsion lasting 7- 10 seconds. Supportive care was provided (positioning, airway support and oxygenation) and MgSO4 administered per protocol. No subsequent fits were observed. Due to the prolonged labour and intrapartum fits patient was shifted to operation theatre where an emergency caesarean section was performed and a dead baby delivered. The patient did not experience a second attack of seizures. She was continuously monitored for blood pressure and discharged on the fourth postpartum day.

CASE 3

A 32 year old patient $G_3P_2^{+0}$ admitted with complaints of swelling on face and feet, dizziness and no urine output. Ultrasonography report revealed dead baby at 32 weeks of gestation. She visited once to the local doctor during her entire pregnancy. Previously, she had a history of IUD in her one pregnancy. Her medical history was not significant. Urology opinion was taken where the consultant advised immediate emergency caesarean section, followed by renal dialysis. On admission her blood pressure was 140/100 mmHg. As soon patient was shifted to the operation theatre, she developed one tonic clonic seizure that lasted for 20 seconds. Her blood pressure suddenly increased to 200/170mm Hg. She was given supportive care, labetalol 20mg, and MgSO4 per protocol. Later, after patient stabilized caesarean section performed and dead baby delivered. On first postpartum day patient again developed generalized tonic clonic seizure that lasted for 25 seconds. Intravenous diazepam was administered and another dose of 2 g bolus MgSO4 was given. She was referred on sixth postpartum day to urology ward for opinion and subsequent management.

CASE 4

A 20 year old patient $G_1P_0^{+0}$ admitted with regular uterine contractions and with complaint of blurring of vision. She took one antenatal visit during 2nd trimester where she was prescribed iron isomaltoside infusion for the correction of anemia. At 35 weeks of gestation she referred to a local doctor with complaints of blurring of vision and headache from where she was referred to a tertiary care hospital for further management. At the time of admission her blood pressure was 140/100 mmHg with traces of proteinuria on dipstick. Ultrasonography, per abdominal and vaginal examination was normal. Cervical effacement was 50 % and os dilatation 6cm. In labour room patient suddenly experienced generalized tonic clonic fits thrice in one hour. Each fit lasted for 30 seconds. MgSO4 and labetalol per protocol were administered. Delivery was expedited with artificial rupture of membranes and intravenously 5 units of syntocinon were given. She delivered alive baby girl via normal vaginal delivery with APGAR scores 4 and 6 in 1 and 5 minutes respectively. After resuscitation baby was referred to the intensive care unit. The patient did not experience any fit thereafter. She was continuously monitored for blood pressure and discharged on the third postnatal day.

DISCUSSION

Eclampsia remains the most frequent cause of seizures during pregnancy; raising maternal mortality in developing countries, where one out of 300 patients may suffer from it. In Pakistan, it has been reported to contribute 10% of maternal deaths and 23.5% perinatal mortality.[2-4] Although, a number of studies have reported antepartum[5] and postpartum eclampsia[6] but limited literature is available on intrapartum eclampsia.

Variables	Case 1	Case2	Case 3	Case 4
Age	40	25	32	20
Gravidity and/or Parity	P_{14}^{+0}	G_2P^{1+0}	G_3P^{2+0}	$G_1P_0^{+0}$
Gestational week	38	36	32	35
Number of antenatal visits	0	4	1	2
Fetal outcome	Alive	IUD	IUD	Alive
Blood pressure	200/110	150/90 mmHg	200/170	140/100
Proteinuria (Dipstick)	3+	Traces	3+	Traces
Haemoglobin (g/dl)	8.1	9	**2.6**	12.2
Thrombocyte count (*platelets*/ mcl)	487,000	213,000	**63,000**	160,000
RBS (mg/dl)	101	97	**140**	90
Urea (mg/dl)	11	26	**88**	17
Creatinine (mg/dl)	0.6	1.2	**3.9**	**2.3**
LDH (u/l)	**501**	**1683**	18	**850**
Uric acid (mg/dl)	**6.9**	**13**	**13.2**	5.2
Total bilirubin (mg/dl)	0.5	0.5	0.5	0.6
SGPT (u/l)	16	31	22	27
Alkaline phosphatase (iu/l)	**509**	**520**	**450**	**931**
PT (seconds)	13	14	14	13
APTT (seconds)	30	32	32	32

Table-I. Clinical characteristics and laboratory investigations in presented case series of intrapartum eclampsia

One of the studies conducted in India reported the most frequent presentation of the eclampsia as antepartum eclampsia (70.9%) followed by intrapartum eclampsia (18.18%) and postpartum eclampsia (10.91%).[7]

In the present scenario, 3 patients developed late onset preeclampsia whereas one patient presented with early-onset preeclampsia, later followed by eclamptic fits. Early onset is defined as presentation of preeclampsia at less than 34 weeks of gestational age, whereas late onset refers to presentation at greater than 34 weeks.[8] In the present case series, only one patient made four antenatal visits, which is the least criteria of recommended antenatal visits by the World Health Organization.[9] Moreover, in two cases, fetal outcome was unfavourable and babies died in utero (IUD), which is clearly understood as studies have reported increased risks for the IUD in hypertensive disorders of pregnancy.[10] Higher incidence of IUD with intrapartum eclampsia has also been supported by a study conducted in Nigerian eclamptic women. Similar results have been achieved in Sinai and Anderson in America where perinatal mortality increased to 85% in patients with antepartum/ intrapartum eclampsia.[11] Another study reported 52.17% maternal deaths only due to intrapartum eclampsia.[12] In the current case series, 2 patients developed severe hypertension with 3+ proteinuria, whereas 2 patients presented with mild hypertension and traces of protein on dipstick test. Presentation of 50% of patients with mild hypertension and proteinuria suggest occurrence of intrapartum eclamptic fits independent of severity of hypertension and proteinuria. Although, in previous studies intrapartum eclampsia has shown to be associated with severe preeclampsia and poor feto-maternal outcome[13], nonetheless severity of both hypertension and proteinuria may not be good predictors for later on development of eclampsia. Moreover, nulliparity is considered as risk factor for preeclampsia[14], however only 1 patient with intrapartum eclampsia was nulliparous in our study.

Biochemical parameters revealed increased levels of LDH and uric acid in three patients and increased alkaline phosphatase levels in all patients. Platelet count was decreased in one patient only. Different presentation of patients suggests further studies to reach conclusion for risk factors and predictive markers for intrapartum eclampsia. Furthermore, improving antenatal care and earlier prediction of intrapartum eclampsia

may improve outcome in both mother and fetus.

CONCLUSION

Results from the studies suggest that feto-maternal outcome may be influenced by the presentation of eclampsia, where intrapartum eclampsia brings more challenges for clinicians regarding prompt diagnosis and management. Further, antenatal care and visits should be given importance, as earlier diagnosis of preeclampsia may prevent women from deadly consequences of eclampsia.

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