

# ECLAMPSIA ASSOCIATED WITH HAEMORRHAGIC DIATHESIS

## A CASE REPORT

SAROSH MEHER THANEVALA\*, M.R.C.O.G.

and

SAROJ CHUGH\*\*, M.B., B.S., D.G.O.

Eclampsia, the once dreaded complication of pregnancy, is fast disappearing with improved obstetric service. Yet from time to time we do encounter cases particularly from rural areas. Eclampsia associated with abruptio placentae and afibrinogenaemia is rarer still but when eclampsia is complicated by massive haemorrhages in practically all the organs of the body, in the absence of accidental haemorrhage, it warrants report and that is the only justification for presenting this case, the more so as the cause of the haemorrhage could not be elicited.

### Case Report

Patient R, age 26 years, admitted as an emergency on 1-4-60 at 5-30 p.m.

**History.** (Obtained from relatives) She had 8 months' amenorrhoea and from night before admission she had been having a number of fits of unconsciousness with twitchings of whole body. She was unconscious following the fits from the morning.

**Condition during Present Pregnancy.** She had headache off and on for last few days and oedema of feet also for last fortnight. Obstetric history: 7th gravida. 6 full-term normal deliveries, all alive and normal. Last delivery 2 years ago.

**Past history:** There was no history suggestive of preeclamptic toxæmia during previous pregnancies. No history suggestive of hypertension or renal disease in the past.

**Family history:** No history suggestive of hypertension in the mother. Father dead, cause not known.

**On admission:** Patient was semi-conscious, violent at times. Breathing was laboured; face was puffy. Patient was cyanosed; front incisor tooth was missing and there was profuse bleeding from the tooth socket, as well as copious haemetemesis and epistaxis. Due to excessive haemorrhage she was getting choaked.

Blood pressure, 205/145 mm. of Hg. Pulse, 108 per minute. Temperature, 99°F. Respiration, 32 per minute. Oedema of feet.

Heart, Tachycardia present. Lungs, Moist sounds heard all over. Per abdomen, Height of fundus 32 weeks, vertex presenting. Not engaged, foetal heart sounds absent.

She had a typical eclamptic fit soon after admission lasting for about one minute.

### Investigations

(1) Urine: On catheterisation about 1/2 oz. of smoky urine withdrawn. Sugar — + Albumin — ++ Microscopically—No casts seen; red blood cells ++.

(2) Blood urea—34 mgm. per 100 cc.  
(3) Clotting time, 4 minutes 52 seconds  
(4) Bleeding time, 2 minutes 33 seconds  
(5) Prothrombin time, 16 seconds (6) Clot retraction time, 5 minutes (7) Platelet count, 1,60,000/c.mm. (8) Blood, Haemoglobin—10 gms. per cent White cell count—

\* Obstetrician/Gynaecologist.

\*\* Assistant Gynaecologist, Irwin Hospital, New Delhi.

10,600 Polymorphs—72 per cent Lymphocytes—27 per cent Monocytes—1 per cent.

#### Treatment

(i) Injection morphia 1/4 grain, Injection atropine 1/100 grain (ii) Oxygen inhalations (iii) Injection serpacil 0.5 mgm. intramuscular and 0.25 mgm. orally thrice daily. (iv) Adrenaline pack to the bleeding socket and nostril. (v) Injection Vit. K 10 mgm. intramuscular. (vi) Intravenous calcium gluconate 10% 10 c.c. with Vit. C 500 mgm. (vii) Antibiotics. (viii) Suction of the aspirated blood from the air passages. (ix) Ryles tube put in and chlortride 2 tablets put in through it. Feeding through the Ryle's tube done. (x) A toxæmia chart was maintained.

A vaginal examination was carried out at 7.00 p.m. Cervix was taken up; one finger dilated. Membranes were intact. Vertex was presenting. Pelvis was adequate. Low rupture of membranes was done and blood-stained liquor came out. At the same time slow syntocinon drip 2½ units in 500 ml. of 5% glucose started.

Patient delivered a fresh dead-born male child at 10-30 p.m. Placenta and membranes expelled at 10-40 p.m. There were no retroplacental clots. She had no postpartum haemorrhage. As the patient had fair amount of bleeding from tooth socket etc., one unit of fresh blood transfusion was given. She had no more fits after the one soon after admission.

On 2nd day of admission—patient developed signs of meningeal irritation. She was unconscious and had neck rigidity. Pupils were constricted and reacting sluggishly to light. Planters were extensor. There was no obvious paralysis.

Bleeding had stopped from the nose and tooth socket. Blood pressure was 150/100 mm. of Hg. Urine was still smoky. Total output of urine in 12 hours was one ounce and it was loaded with albumin. A lumbar puncture was done. Fluid was blood-stained but not under pressure.

On 3rd day she was conscious. There was no neck rigidity. Pupils were still constricted but reacting to light. Planters were equivocal. There was no bleeding from any body surfaces.

Blood pressure was 160/110 mm. of Hg.

Urine was clearer than before and total output in 24 hours was 7 ounces. Her general condition gradually improved. Signs of meningeal irritation disappeared. Oedema diminished. Urinary output increased and urine became clear. Albumin in urine disappeared but her blood pressure remained high as shown in the toxæmia chart. In spite of the fact that blood pressure was pretty high at times, she did not have any more bleeding episodes. She absconded on 14th April 1959.

#### Discussion

This is a case of eclampsia complicated by haemorrhagic diathesis. Patient had high blood pressure, albumin-uria, oliguria, oedema of legs and convulsions. But what makes it interesting is that she also had haemorrhagic diathesis which was manifested by haemorrhage from the kidneys leading to haematuria, from the socket of a broken tooth, from the stomach revealed as haematemesis and also in the brain showing signs of meningism and blood-stained cerebrospinal fluid. Yet there was no intrauterine bleeding although there was intra-uterine death of the foetus. Investigations showed no decrease in serum fibrinogen content. Bleeding time, clotting time and platelet count were normal.

On going through literature we find no reference to hypertension per se giving rise to haemorrhagic diathesis. Hypertension associated with pre-eclampsia may cause haemorrhages due to generalized vascular spasms and consequent vascular damage. It is usually confined to capillaries as in the kidneys, liver, placenta etc. Whether it can cause massive haemorrhage as in this case is doubtful.

Date	Blood pressure		Oedema	Fits	Urine			M
	Morning	Evening			Quantity	Colour	Albumin	
1-4-1959	205	178	++	1	1 oz.	Highly smoky		No casts R.B.C.
	145	140						
2-4-1959	158	155	++	Nil	7 ozs.	Smoky		
	100	108						
3-4-1959	160	180	++	Nil	29 ozs.	Less smoky		
	110	135						
4-4-1959	210	225	++	Nil	42 ozs.	Clear		
	135	140						
5-4-1959	185	180	++	Nil	42 ozs.	Clear		No R.B.C.
	135	135						
6-4-1959	205	155	+	Nil	94 ozs.	Clear		
	135	110 (A.f.er)						
7-4-1959	130	180	Slight	Nil	92 ozs.	Clear		
	100	120						
8-4-1959	180	180	Slight	—	69 ozs.	Clear	Traces	
	120	130						
9-4-1959	140	165	Slight	—	72 ozs.	Clear	Traces	
	120	120						
10-4-1959	150		Nil	—	52 ozs.	Clear	Nil	
	100							
11-4-1959	150							
	102							
12-4-1959	156							
	98							

Another possibility and a more possible explanation is that there was defect in the clotting mechanism. In 1901, De Lee described death in a patient from post-partum haemorrhage as being due to haemophilia like phenomenon which followed concealed accidental haemorrhage. Diekmann in 1936 was the first to suggest that such bleeding might be due to decrease in fibrinogen and in 1949 Moloney successfully treated

such a case with fibrinogen and blood transfusion.

Normal clotting of blood as put forward by Morawitz in 1905 is

Prothrombin + Calcium + Thromoplastin = Thrombin. Thrombin + Fibrinogen = Fibrin.

But clotting mechanism is not so simple and several factors are involved in coagulation. Blood may not