Amniotic fluid embolism

(It can present without respiratory manifestations)
Case report

N. GAD

Mrs. A.F. was 20 years old, gravida 2 para 1. No past medical or surgical history of significance. Two years ago she had a normal vaginal delivery at 42 weeks of a male baby weighing 3,550 grams. She spent six hours in the first stage and fifty minutes in the second stage.

After a normal ante-natal course, she was admitted for induction of labour because of postmaturity at 42 weeks. On her admission she was normotensive with no proteinuria. The cervix was 2 cm dilated, effaced, posterior and thick, the presenting part (cephalic) was 2 to 3 cm above the ischial spines. Her haemoglobin and platelets were 12 gm/dl and 161×10^9/l respectively.

At 6 a.m. one mg of Prostaglandin gel was inserted into the posterior fornix. Six hours later the cervix was assessed and found to be 3 cm dilated, effaced, central and thick. The head was still 2-3 cm above the ischial spines. Artificial rupture of the membranes (ARM) was performed and clear liquor was obtained. Twenty minutes later she was contracting successively and strongly and required analgesia. She was given Entonox but 30 minutes later Pethidine and sparine injection was still given for pain relief. One hour and twenty minutes from the ARM the baby's head was delivered. On delivery of the anterior shoulder, the patient became unconscious, the attending midwife summoned for help while she continued with the delivery of the rest of the baby (baby birth weight was 4,595 grams with an Apgar score of 9 at one minute).

On attendance, two minutes from the episode, the patient was unconscious, with the blood pressure being immeasurable, and the pulse very weak at a rate of 176 beats per minute. The chest appeared clear. An oxygen 100% was given throught a mask. An intravenous line ((IV) was immediately inserted and blood was taken for full blood count, crossmatching and clotting screen. Intravenous infusion with Hartman's solution was started immediately. A second IV was inserted but it was noted that

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Table 1. — *The results of the clotting screen and haemoglobin.*

Day	Sample No.	Hb	Plat	PT	APTT	Fib
One	1st	11.7	132	12	31	3.5
	2nd *	12.6	42	> 180	> 120	< 1.0
	3rd	11	89 **	26	132	0.6
	4th	10.9	52	16	57	1.9
Two	1st	—	—	14	45	2.4
	2nd	10.6	59	13.6	44.6	2.9
	3rd	11.5	58	12	37	3.0
Three	1st	11.7	70	12.3	32.1	2.8
	2nd	13.0	82	11.3	33.2	2.9
Four		13.8	109	12.1	31.9	2.36
Five		13.4	151	11.5	32	4

(*) The 2nd sample of day one was taken only five minutes from the 1st sample, which was taken at the initial episode of unconsciousness.

(**) Note the relative increase of platelet count due to platelet transfusion.

Legend: Hb = Haemoglobin; Plat = platelets (N. = 150-400); PT = Prothrombin time (N. = 10.5 - 14 sec.); APTT = Activated partial thromboplastin time (N. = 24 - 37 sec.); Fib = Fibrinogen (N. = 2 - 5).

there was excessive bleeding during the procedure, so a further blood sample was sent for clotting screen.

The placenta was then delivered complete, uterus was well-contracted but there was very heavy post-partum haemorrhage despite the well-contracted uterus. The bleeding source appeared to be through the cervix and also from a small posterior wall vaginal laceration. There were no formed blood clots.

A clinical diagnosis of amniotic fluid embolism (AFE), and resulted in hypotension and disseminated intravascular coagulation (DIC), was made. Two units of uncrossmatched blood of the same blood group was started immediately using warming equipment and transfusion pump. Twelve units of crossmatched blood, fresh frozen plasma (FFP) and platelets were asked for. Intravenous infusion of Syntocinon 40 IU/500 ml normal saline was started at a rate of 40 ml/hour. The vaginal laceration was sutured, but, despite a good repair, the wound was still bleeding heavily. A tight vaginal pack to compress the vaginal wound was inserted.

The results of the clotting screen confirmed the clinical diagnosis of DIC (Table 1).

Chest X-ray and arterial blood gases were normal.

The patient's general condition was improving all the time. One and a half hours after the initial episode her blood pressure was 100/50 with a pulse rate of 160 beats per minute. There was gradual decrease of the vaginal bleeding. Within the first 24 hours she was transfused 14 units of blood, 7 units of fresh frozen plasma and 5 units of platelets.

The patient has made a complete recovery and she was discharged home on the sixth day.

DISCUSSION

Disseminated intravascular coagulation is never primary but it is due to a sti-

mulation of the coagulation activity. The triggers of DIC in pregnancy include leakage of amniotic fluid, placental tissue fragments, incompatible red blood cells or bacterial products into the maternal circulation. Obstetric conditions that can cause any of the above are amniotic fluid embolism, abruptio placentae, septic abortion and intra-uterine infection, retained dead fetus, hydatidiform mole, placenta accreta, pre-eclampsia and eclampsia and any prolonged shock ([9]).

Amniotic fluid embolism is the most dangerous and untreatable condition in obstetrics, the repeated incidence is from 1 in 3,360 to 1 in 80,000 pregnancies ([7]). In the United Kingdom from 1985 to 1987, it caused nine maternal deaths which represent 6.5% of all direct maternal deaths ([9]). The condition carries a mortality rate of 85%. The fetal mortality ranges from 40 to 50% ([3, 6, 10]).

The condition is usually presenting as sudden onset of dyspnoea, cough which is productive of pink frothy sputum, and cyanosis, hypertension, hypoxia and DIC ([8]) is reported in most of the cases. Left ventricular failure may develop. Convulsions may develop in 10 to 20% of women. Of those who survive the acute hypoxic episode, most of them develop DIC ([1, 2, 7, 8, 11]). In the presented case, there were no respiratory symptoms, chest X-ray was clear and arterial blood gases were normal.

Confirmation of the diagnosis of AFE is usually made by post-mortem examination by the presence of amniotic fluid and fetal tissue within the maternal lung on histological examination. In survived patients the diagnosis can be made by cytological examination of blood obtained from the central circulation via a central venous pressure on a Swan-Ganz catheter ([5]).

The treatment of AFE is mainly supportive. Oxygen administration sometimes through endotracheal intubation with mechanical ventilation is the first objective. The second step is the correction of the shock. Prompt intravenous fluid administration should be under central haemodynamic monitoring to avoid pulmonary oedema. Dopamine will be needed if the patient remains hypotensive. If heart failure develops, the patient should by rapidly digitalised ([13]).

Disseminated intravascular coagulation should be anticipated and promptly treated. Although whole fresh blood is the treatment of choice in coagulation failure, it is no longer generally available in the UK because of the time needed to complete hepatitis, HIV (AIDS) antibody and blood grouping tests before it is released from the Transfusion Centre ([9]). Transfusion of packed red cells and fresh frozen plasma and may be platelets now replace the whole fresh blood in treatment of DIC. There is insufficient data to warrant the use of heparin or anti-fibrolytic agents in this condition. It is important to reduce the placental site bleeding by the use of oxytocics. The use of prostaglandin F2-alpha has been tried with success ([1, 2, 8, 11]).

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