Clinical Notes

Afibrinogenaemia Associated with Accidental Haemorrhage

P. G. S. KENNEDY, M.B., B.CH., D.OBST., R.C.O.G.

Plaistow

In this condition a deficiency of fibrinogen in the blood results in failure of the clotting mechanism. It has been found to occur in three conditions during pregnancy.

- 1. Abreptio placentae.
- 2. Amniotic fluid embolism.
- 3. Delay in expulsion of foetus from uterus, when it has died as a result of rhesus incompatibility.

It has been shown that the foetus, decidua, and liquor amnii all contain large amounts of thromboplastin. It is suggested that in the conditions listed above this thromboplastin occasionally gains access to the maternal circulation. There the thromboplastin reacts with the fibrinogen of the maternal blood, which is precipitated as fibrin intravascularly. As a result the maternal blood becomes depleted of fibrinogen and will not clot. This failure to clot results in haemorrhage from the placental site (or from any other wound) despite good uterine contraction and retraction.

The diagnosis, which can easily be made at the bed-side without any other apparatus than a hypodermic syringe, is based on the following findings:—

- 1. History of accidental haemorrhage, amniotic fluid embolism syndrome, or retention of dead foetus in utero in a Rh. negative mother.
- 2. Continued uterine haemorrhage despite good uterine contraction and without any other obvious cause.
- 3. Failure of the blood collected in a receiver or even in bed to clot.
- 4. Clot observation test. (Weiner et al. 1950, 1953.) This test is simple. 5cc. of blood are collected by venipuncture and allowed to stand in a test-tube. Normal blood will set almost solid in less than half-an-hour. In afribrinogenaemia there will be no clot or, at the most, a very small piece (and even such small clots may dissolve again if incubated at 37 degrees for a short time).

Treatment consists of replacement by intravenous injection of sufficient amounts of human fibrinogen.

A number of cases have been reported in the American medical press but comparatively few in the British press so far. It therefore seemed of sufficient interest to report two successive admissions to Plaistow Maternity Hospital for accidental haemorrhage, both of which proved to have afibrinogenaemia.

Case 1. Age 34. Two previous pregnancies normal. Attended ante-natal clinic regularly. No abnormality observed, maximum B.P. 130/80mm, blood group A. Rh. positive.

Admitted to hospital at 36th week of pregnancy with a history of half-pint

vaginal haemorrhage shortly before admission.

Examination. Fundus uteri at 36 weeks, slightly tender. Foetus L.O.A. position. Head had entered brim. No foetal heart sounds heard. P.V. vertex presenting. Cervix thin and 1-in. dilated. No placenta felt. Colour good. P.80, B.P. 118/82mm. Blood taken for cross matching was noted not to have clotted one hour later.

Diagnosis. Accidental haemorrhage and afibrinogenaemia.

Progress. Patient was in labour and having regular contractions and a small amount of blood was passed with each pain. She was treated with intravenous Dextran solution (400cc.) and 1.8gm. human fibringen. Subsequently she was given a further 3.6gms. fibrinogen and 400cc. blood. She delivered herself of a still-born foetus weighing 5lb. 12ozs. The placenta followed immediately and showed evidence of partial separation.

Age 28. Six normal previous pregnancies. Patient was admitted as an emergency, with a history of a recent brisk haemorrhage. She had no pain. No ante-natal examination had been made. Calculation from last

menstrual period suggested that she should be 20 weeks pregnant.

Examination. Fundus uteri at 24 weeks, rather hard and a little tender. Foetal parts not distinguished and foetal heart sounds not heard. P.V. No dilation of cervix. Skin a little pale. P.98, B.P. 110/72mm., no oedema. Blood taken for cross matching showed a clot only the size of a pea after threequarters of an hour. Blood group O. Rh. positive. Catheter specimen of urine set solid on boiling.

Diagnosis. Accidental haemorrhage and afibrinogenaemia.

Progress. Patient continued to have a small but continuous trickle of blood P.V. and, despite rupture of membranes and small doses of Pitocin, labour did not commence within 24 hours. During the course of 36 hours, she received seven pints of blood and two pints of six per cent. dextrose intravenously. It was not until after the third ampoule that her blood showed evidence of clotting satisfactorily. When other means failed to start uterine contractions an intravenous drip of five units of Pitocin in 400cc. of six per cent. dextrose solution was put up, and after the rate of flow had been increased from eight to sixteen drops a minute vigorous uterine contractions started. She delivered herself of a 20-week foetus and placenta with a considerable amount of old clot adherent to it. Recovery was rapid and uncomplicated.

Conclusion.

Afibrinogenaemia is a somewhat rare condition. The second case mentioned above shows how serious the results may be. The first case on the other hand suggests that early diagnosis and adequate treatment will prevent serious haemorrhage. diagnosis can readily be made by clinical findings, and by the use of the simple clot observation test done at the bed-side. The condition should be kept in mind whenever one has to deal with ante-partum haemorrhage.

REFERENCES

Weiner, A. E., Reid, D. E., and Robey, C. C. (1953) Amer. J. Obstet. Gynec., Weiner, A. E., and Diamond, L. K. (1950) Amer. J. Obstet. Gynec., 60, 1015.