ABRUPTIO PLACENTAE*

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SUMMARY

Abruptio placentae occurs on an average in 11 patients per month at Baragwanath Hospital. In 7 years (1962 - 1968) 1 228 patients presented with this complication and were treated in the obstetric department. The present report deals with the results of treatment of 138 patients with abruptio placentae in the year 1968. Maternal mortality was nil, 15 babies survived the condition (10·5%); premature infants under 1 800 g comprised 94 of the 142 babies delivered.

The hospital postpartum haemorrhage rate was increased from 1·5 to 34·1 as a result of the blood coagulation defect which developed in 43 patients in this series. In 4 patients failure of the uterus to contract aggravated the blood loss. Correction of the clotting defect before delivery was successful in 30 of 35 severely affected patients. In 5 patients the therapy had to be continued in the post-delivery period because of failure of the blood to clot in the presence of a well-contracted uterus.

Therapy consisted of the use of bank blood, double- or triple-strength plasma, and a minimum of 2 g fibrinogen. The institution of therapy was governed by the clinical observation that the clotting time was prolonged. Artificial rupture of membranes and the administration of Syntocinon 5 U in an intravenous drip formed a basic part of the therapy. Prolongation of the interval between artificial rupture of the membranes and delivery was associated with an increase in the postpartum haemorrhage rate.

A review of the results of treatment of 138 patients at this hospital confirms the view that correction of the blood coagulation defect must be instituted promptly and before delivery of the placenta in spite of views that this could in fact be harmful, or even dangerous.

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Abruptio placentae is a common complication of pregnancy found in the patients treated in the obstetric section of Baragwanath Hospital. In a recent article Basu[1] analysed the treatment of 322 instances of this condition collected over 16 years. In the 7-year period 1962 - 1968, 1 228 patients were treated for accidental haemorrhage out of a total of 73 995 patients delivered at our hospital, an incidence of 1·6%. Because of the divergent opinions in articles on the subject of treatment of abruptio placentae which have appeared in the literature recently, and because the statements made by the authors on the subject were at such variance with what was found to occur in the Bantu patients, it was decided to review the cases which occurred in the unit in one year. The year 1968 was chosen simply because it was the most recent year with completed statistics.

MATERIAL

In the year under review, 18 611 patients were admitted to the obstetric section, of whom 12 330 were delivered in the section. Abruptio placentae occurred in 138 of these patients and in a further group of 139 patients the

diagnosis of 'antepartum haemorrhage of unknown origin' was made. Placenta praevia, proved by radioactive iodinated serum albumin, visual evidence or palpation of the placenta, was present in 91 patients.

Age and Parity

Accidental haemorrhage occurred most frequently in parous women 1 - 4 and in the age-group 21 - 35 years (Table I).

TABLE I. ACCIDENTAL HAEMORRHAGE RELATED TO AGE AND PARITY

Age (years)	*No. of cases*	*Parity*	*No. of cases*
15 - 20	13	Primiparae	15
21 - 25	35	Para 1 and 2	53
26 - 30	43	Para 3 and 4	38
31 - 35	28	Para 5 and 6	19
36 - 40	14	Para 7 and 8	8
40 - 47	5	Over para 8	5
Total	138		138

In this series 90 patients had not received any antenatal care, 42 patients had attended the antenatal clinics conducted by the City Council of Johannesburg in the Bantu townships and only 6 had attended the clinic operated by the hospital. The most severe cases of separation of the placenta occurred, probably fortuitously, in the patients who had not received antenatal care.

The Infant

In the group, 142 babies were delivered (4 sets of twins) and the sex distribution was 80 male and 62 female infants. There were 94 infants (66·2%) with birthweight less than 1 800 g (4 lb 8 oz). Of the 25 infants born alive, 10 died in the immediate neonatal period and 15 (10·5%) survived and were discharged from hospital in a satisfactory condition. The babies that survived all weighed in excess of 2 721 g (6 lb) and were delivered of mothers who were less severely affected. There were 117 stillbirths (84·9%) in the series, of which 87 were fresh and 30 were macerated stillbirths.

The Clinical Manifestations

Frank vaginal haemorrhage without pain was the presenting symptom in 72 patients (52·1%). Haemorrhage commencing with or soon after the onset of labour pains occurred in 60 patients (43·4%) and in 6 patients vaginal haemorrhage became apparent some time after the clinical manifestations of abruptio placentae had become established, and the presenting symptom was pain in the abdomen.

In 35 instances, the patient arrived at the admission ward of the obstetric unit in a severe state of shock and required a prolonged period of intensive resuscitation. The external blood loss was not marked, but all complained of severe abdominal pain. The uterus was tense and tender on palpation and the lie and presentation of the foetus could not be elicited. The foetal heart sounds were heard soon after admission in 32 patients but disappeared later in 7 patients.

The uterus was noted to be tense and tender in 81 patients and the presenting part and foetal parts could not be made out on palpation. In only 2 of these patients did the measurement of the abdominal girth increase in the time preceding delivery.

Spontaneous vaginal delivery occurred in 126 of the 138 patients in the series. Delivery was effected in 8 patients by means of lower uterine segment caesarean section, because of a history of a previous section in 3 patients and a wrong diagnosis of the type of haemorrhage in 5 patients.

The foetus was delivered with the assistance of the vacuum extractor in 2 patients and in the remaining 2 patients, internal version and breech extraction was performed because of transverse presentation of the foetus. In the 12 patients delivered by intervention the foetal loss was extremely high and only 1 infant was salvaged. Mild pre-eclamptic toxaemia was present in 8 patients with abruptio placentae (5·7%). Essential hypertension was found in 8 patients after delivery had been completed.

TREATMENT

The treatment at this hospital of patients who developed abruptio placentae is standard and was formulated in 1952. The patient is admitted to bed and is given 15 mg of morphine sulphate intramuscularly. An intravenous infusion is set up, using 1 litre of dextrose/water and all other necessary resuscitative measures are instituted. A sample of blood is taken and sent to the hospital blood bank and the necessary amount of compatible blood is obtained. The blood-clotting time is measured on another sample of blood and the tube is strapped to the drip stand in order to observe the stability of the clot.

When the patient is considered to have recovered from her initial shock, a vaginal examination is performed and the forewaters are ruptured. Syntocinon 5 units is added to the intravenous infusion and the rate of flow is adjusted to 12 drops per minute. The response of the uterus to the Syntocinon is carefully observed and the rate and intensity of the contractions are charted. The blood-clotting time is repeated at frequent intervals and at the first indication that the clotting time is becoming prolonged the regimen formulated many years ago is instituted. Dried human plasma is reconstituted into a triple-strength solution and replaces the dextrose/water infusion. In 15 minutes the clotting time is repeated and, if found to be above normal, 2 g fibrinogen is administered immediately. Blood transfusion is controlled by the needs of the patient as assessed clinically and on haemoglobin levels. Fibrinogen in 2 g units is administered until the clotting time has returned to normal.

The progress of labour in these patients is usually rapid and the placenta is delivered soon after the birth of the foetus. The contraction and retraction of the now empty uterus is noted carefully and if contraction does not occur the uterus is massaged to induce it to contract and the rate of the Syntocinon drip is increased. If the blood lost *per vaginam* fails to clot or if the clotting time is prolonged then further plasma and/or fibrinogen is administered.

The patient is closely observed for 2 hours postpartum with particular reference to the state of the uterus. Only when this time has elapsed is she transferred to the lying-in ward. A full blood count is ordered for the third postpartum day and treatment is carried out if required to correct anaemia.

RESULTS

The treatment outlined above has proved to be completely adequate for 134 of the 138 patients in the series. Four patients developed atony of the uterus at the end of the third stage of labour which necessitated continuation of therapy. Within 30 minutes all 4 were reported to have a uterus which was well contracted.

Lysis of the formed clot occurred in 3 cases only in the series. These patients were given Trasylol 250 000 U with success. The postpartum haemorrhage rate was raised in the patients with abruptio placentae. The hospital postpartum haemorrhage rate for the year was 1·5% but in the abruptio placentae group it rose to 34·1%. This increase in blood loss was due to failure of the blood to clot in 43 patients, in the presence of a well-contracted uterus. In 4 patients atony of the uterus added to the coagulation defect.

The amount of blood lost postpartum was 600 - 1 000 ml in 22 patients, 1 001 - 1 200 in 13 patients, 1 201 - 1 600 in 6 patients, and 2 000 - 3 000 in 6 patients.

The time interval between artificial rupture of membranes and delivery of the foetus did not exceed 11 hours in this series. The result bear out the well-recognized fact that increase in the interval between artificial rupture of the membranes and the time of delivery is associated with increased postpartum haemorrhage. When the interval was up to 3 hours the postpartum haemorrhage rate was 39·1% but increased sharply to 71·4% when the interval was between 4 and 11 hours.

Artificial rupture of membranes was performed on 60 patients who were classified as severe; spontaneous rupture of membranes occurred in 9 patients and in 69 patients labour commenced spontaneously. The postpartum haemorrhage rate in these groups was 28%, 2% and 17% respectively. There does not appear to be any correlation between the amount of blood clot present behind the placenta and the incidence of postpartum haemorrhage. Traumatic accidental haemorrhage as a result of falls or blows on the abdomen accounted for abruptio placentae in 3 patients only.

DISCUSSION

The incidence of abruptio placentae at Baragwanath hospital is very much higher than that found at other hospitals in this country and certainly much higher than that of overseas hospitals. The reason for this is not completely understood.

The significance of the utilization of available blood-clotting components in abruptio placentae, with consequent failure of the blood to clot intra- and postpartum was realized for the first time at this hospital in 1952. Up to that time it was believed that some substance which caused or resulted from placental separation interfered with coagulation, which produced the haemorrhage. It was labelled a haemorrhagic diathesis. When the part played by fibrinogen loss was recognized, the failure of the blood to clot was brought under control and the deaths from this condition were soon reduced to a minimum. In the present series in spite of severe affection in 69, and very severe affection in 35, no maternal deaths occurred.

The treatment of abruptio placentae did not change significantly at this hospital until late in 1969 when as a result of articles in the literature doubt was cast on the advisability of using fibrinogen and similar substances before the completion of the third stage of labour. Basu[1] stated: 'specific therapeutic measures with fibrinogen or other agents to correct any coagulation defect present before delivery are not indicated. They can only make matters worse, not better.' In the late 1969/early 1970 period 4 of our patients died as a direct result of blood-clotting defects which were not treated until the delivery of the foetus and placenta—by which time it was too late.

A further remark made by Basu concerning the postpartum haemorrhage rate and causation is not confirmed in our series. He states that increased postpartum haemorrhage is due to atony of the uterine muscle which is caused by the formation of fibrin degradation products in the uterus and that 'except in those cases in which abruptio placentae is virtually complete, the maximum rise in serum levels of fibrin degradation products occurs after delivery. This may have a bearing on the development of uterine atony only after delivery.' This again has not been the experience at this hospital. In 9 cases the placenta was completely separated and delivered with the foetus. Only 3 patients developed a postpartum haemorrhage *but in the presence of a well-contracted uterus.* In fact there were only 4 patients in the 138 in the series who developed a postpartum haemorrhage as a result of atony of the uterus. The postpartum haemorrhage rate was raised significantly in the series from 1·5% to 34·1% and the blood loss was due to failure of the blood to clot.

Thirty-five patients were classed as 'very severe' accidental haemorrhages. All were treated as described above and in 30 the coagulation failure was controlled before delivery and 5 required continuation of therapy for up to an hour postpartum. It must be stated that facilities for the estimation of the so-called fibrin degradation products do not exist at this hospital. The ability of the clinician to assess the clotting time of a sample of venous blood, or to observe that the vaginal blood is failing to clot, were the criteria used to assess failure of blood coagulation and to institute therapy to combat this.

'The condition is so short lived that it is unwise to be dogmatic about the management of abruptio placentae.'[2] This has been our experience and until such time as it has been proved conclusively that the use of substances to correct coagulation defects 'can only make matters worse not better', we consider it unwise to state that they be not used.

It is not our intention to deny the existence of fibrin degradation products, or to belittle the part played by laboratory investigations in the management of the type of case, but it is felt that the clinical approach and experience must take precedence over the theory expounded by Basu[1] and others if tragedy is to be avoided. The management of a patient with abruptio placentae who develops a blood coagulation defect as a result of the condition is a clinical emergency which must be treated vigorously and promptly.

REFERENCES

1. Basu, H. K. (1969): J. Obstet. Gynaec. Brit. Cwlth. **76**, 481.
2. Bonnar, J., McNichol, G. and Douglas, A. R. (1969): *Ibid.*, **76**, 799.