

Silent Abruptio of the Posteriorly Inserted Placenta

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SUMMARY

Four illustrative cases of severe, but 'silent', abruptio placentae are presented. Apart from variable degrees of vaginal bleeding, low backache was the only other constant symptom. In each instance the abruptio was of a posteriorly imbedded placenta. The significance of posterior placental abruptio is emphasised and an approach to its clinical management suggested.

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Significant abruptio of the normally situated placenta is commonly associated with painful antepartum haemorrhage. Clinical examination usually reveals a uterus which is tense and tender to the touch. Depending upon the degree of abruptio, foetal heart tones are either absent, or are indicative of acute foetal distress.

Contrary to this usual presentation of abruptio placentae, a number of severe, but 'silent', abruptions—severe enough to cause foetal death—have been noted among patients attending the Addington Obstetric Service. The placental site in these cases was invariably on the posterior wall of the upper uterine segment. With antepartum haemorrhage, a dull but persistent low backache was the only other abnormal presenting feature. Four cases are reported to illustrate the clinical presentation and significance of abruptio of the posteriorly placed placenta.

CASE REPORTS

Case 1

A Coloured multiparous patient of 34 weeks' gestation was admitted with a history of unprovoked vaginal bleeding. The bleeding had been preceded by some lower abdominal pain. The patient estimated the blood loss to be 'about half a pint' (300 ml).

On clinical examination, the abdomen was soft and non-tender. The lie of the foetus was longitudinal with a cephalic presentation. A few isolated contractions were felt and the patient was thought to be in early labour. The foetal heart sounds were more clearly heard at 124 beats/min. No local abnormalities to account for the bleeding could be seen on the cervix.

A diagnosis of placenta praevia was made and, as the patient was in labour and still bleeding (although only slightly), an 'examination in theatre' was undertaken. This confirmed the

above features. In addition, the cervix was soft and 4 cm dilated. On digital examination, placental tissue was not found over the internal os, and the membranes were therefore artificially ruptured. Clear liquor was obtained.

The patient was returned to the labour ward and closely observed for signs of foetal/maternal distress, vaginal bleeding, and the progress of labour. Approximately 5 hours after the rupture of the membranes and despite good progress, the foetal heart tones suddenly disappeared. There had been no premonitory signs to suggest foetal distress. Examination of the patient at this stage showed a maternal pulse of 78 beats/min, and a blood pressure of 130/70 mmHg. Clinically she appeared pale but not shocked. On abdominal palpation, the uterus relaxed well between contractions and was non-tender. The head was engaged. On vaginal examination the cervix was almost fully dilated. The foetal skull bones were moulded and extremely soft. There was no bleeding. An elective forceps delivery was performed and a fresh stillborn female foetus weighing 1.45 kg was delivered. On digital exploration the placenta was found situated in the fundus, posteriorly. There was a large retroplacental clot.

Case 2

A White grand multipara had had 8 term pregnancies. Two of these resulted in stillbirths associated with antepartum haemorrhage, and so she was booked for an elective induction of labour at the 38th week of her present gestation. The patient refused to be induced and preferred to wait for the spontaneous onset of labour. When she was approximately 39 weeks pregnant, she presented with a history of painless vaginal bleeding and progressive abdominal distension. On examination, the patient was found to be cold and clammy, with a poor peripheral circulation, a thready pulse of 120 beats/min, and a systolic blood pressure of 75 mmHg. The abdomen was distended and non-tender. The foetus was lying longitudinally with the vertex presenting. An ill-defined foetal heart of 96 beats/min was heard. A vaginal examination was not done at this stage. After rapid resuscitation and correction of acidosis (under CVP control), the patient was examined vaginally in the theatre. The cervix was found tightly closed and uneffaced. At this stage she began to bleed profusely and Caesarean section was promptly performed. The clotting mechanism had been checked prior to surgery and was normal.

A stillborn female infant weighing 3 kg was delivered. The placenta was partially attached to the posterior aspect of the upper segment. Approximately 2.5 litres of blood was present in the uterus.

Case 3

A Coloured multipara, known to have aortic incompetence, had been admitted previously for treatment. Her cardiac state was well controlled by digoxin. When she was 34 weeks pregnant she began to bleed vaginally. The bleeding was associated with a continuous backache, despite the fact that she was not in labour.

When examined on admission, she had a pulse of 80/min, and a blood pressure of 120/80 mmHg. Although not clinically shocked, she was extremely pale. The haemoglobin was 10 g

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100 ml. The height of the fundus was compatible with the calculated period of gestation. The uterus was soft and there was no tenderness. She was not in labour. The foetus presented longitudinally with the vertex easily palpable three-fifths above the pelvic brim. The foetal heart was irregular and varied between 80-100 beats/min. On vaginal examination the cervix was found to be 2 cm dilated and only partially effaced. The blood in the vagina was dark, but there were no clots.

A diagnosis of posterior placental abruption was made, and the patient prepared for emergency Caesarean section.

At operation a live male infant weighing 2,7 kg was delivered. The Apgar score at birth was 1/10, but this rapidly improved on resuscitation to 8/10. The placenta was imbedded posteriorly in the upper segment. There was 500 g of retroplacental blood clot.

Case 4

A White gravida 3, para 2, was mildly hypertensive in the early part of her pregnancy, the blood pressure varying between 130/80 and 150/80 mmHg. When she was 34 weeks pregnant, she was admitted with a history of having bled approximately 4 cupfuls of blood. This was associated with some low backache. There were no contractions and foetal movement could be felt.

On general examination she did not appear shocked, but she had a marked tachycardia (132/min). Her blood pressure was 150/90 mmHg but there was no oedema or albuminuria. The height of the fundus was compatible with the patient's period of amenorrhoea. The uterus was tense but not tender. The lie of the foetus was longitudinal with a cephalic presentation. Four-fifths of the head was palpable above the pelvic brim. The foetal heart was regular at 176 beats/min.

A diagnosis of posterior placental abruption with foetal distress was made and Caesarean section performed. A female infant weighing 2 kg was delivered. The Apgar score at birth was 4/10, but improved to 10/10 on resuscitation. A posteriorly inserted placenta with 600 ml of retroplacental blood clot, was found.

DISCUSSION

Abruptio placentae is initiated by haemorrhage into the decidua basalis. The source of the haemorrhage may be either from the placenta or from some pathological change in the smaller uterine vessels.¹ The decidua then splits, so that a thin layer remains in contact with the maternal surface of the placenta, while a thicker layer adjoins the myometrium. The decidual haematoma thus formed leads to separation, compression, and ultimate destruction of the portion of the placenta adjacent to it.¹ In its early stage there may be no clinical symptoms. Groenewald *et al.*² refer to this stage as asymptomatic 'premature separation of the placenta' and differentiate it from the clinical condition of symptomatic 'acute abruption'. Pathological changes in the placenta are frequently found in the former condition, whereas with acute abruption the changes found on the maternal surface of the placenta are often consistent with those that result from normal separation.² Although these two conditions may occur separately, they are also found in association and it has been postulated that premature asymptomatic separation may occur in several bouts, one of which results in clinical symptomatic abruption.²

The aetiology of placental abruption is not germane to this discussion. A review of this subject was recently published by Pritchard *et al.*³

The clinical presentation of a typical case of severe abruptio placentae, is vaginal bleeding of a greater or lesser degree; a tonically contracted wood-hard uterus; uterine tenderness which may be generalised or localised; and the absence of foetal heart sounds.^{1,4} Although the amount of pain is variable with lesser degrees of abruptio placentae, uterine tenderness is almost always present. The cause of the pain has not been determined, but it is probably due to the disruption of the myometrial fibres and intravasation of blood at the site of the abruption. When it is severe, effusions of blood may occur beneath the uterine serosa, and elsewhere, such as the tubal serosa and the connective tissue of the broad ligaments.

With this accepted presentation of severe abruptio placentae, it was rather surprising to find patients who had obviously suffered severe disruption of their placental attachments, but in whom the symptoms, other than external vaginal haemorrhage, were minimal. Thus, of the 4 patients presented, none had uterine tenderness, while only 3 complained of pain. The pain was dull, persistent, and localised to the lower part of the back. A common feature in every instance was the posterior site of the placenta.

Scrutiny of recent English literature revealed only 1 report of silent abruptio placentae. Wisat⁵ described 2 patients who had had severe, yet silent, premature separation of the placenta associated with marked hypofibrinogenaemia. Although both patients were delivered by Caesarean section, the placental site was only commented on in one instance—it was located on the posterior wall of the uterus, and was completely separated with about 500 ml of adherent retroplacental blood clot.

Conservative management of premature separation of the abnormally situated placenta (placenta praevia) is associated with a marked improvement in perinatal mortality⁶ due, largely, to a reduction of the incidence of prematurity. Because the mechanism of placentation in placenta praevia is essentially normal, bleeding from the abnormal situation is only likely to occur when the lower segment begins to form, approximately 2-3 weeks from term. With separation of the normally situated placenta (abruptio placentae), however, it is doubtful whether a conservative approach will be as effective, since the inherent fault in this instance is a defect in either the implantation and 'anchoring' of the placenta, or in the maternal 'bed', the decidua. A further discouraging feature, demonstrated by 3 of our patients, is that both abruption and asymptomatic premature separation of the placenta predispose to premature delivery. The mechanism responsible for the initiation of labour is not known, but may be due to the irritating effect of the resulting decidual necrosis and haemorrhage.²

Clinical management is difficult. There are no tests or diagnostic methods to detect early separation of the placenta, and the bleeding may be entirely concealed. Pain and tenderness are fairly early signs of abruption, but may be completely absent, especially if there is posterior placentation. Because of these disadvantages, patients who present with antepartum haemorrhage associated with a fundal insertion of the placenta, and in whom other causes for antepartum haemorrhage have been excluded,

should be treated along lines similar to those advocated by Macafee⁶ for placenta praevia. This opinion is based on the premise that vaginal bleeding results from some form of trauma, and if a cause cannot be found, it is safest to assume that the bleeding originated from detachment of the placenta. Conservative treatment includes hospitalisation, strict bed rest (the patient, preferably, lying on her side), investigation for predisposing factors (hypertension, folate deficiency, supine hypertension), and close observation to detect renewed haemorrhage and/or pain. Precise localisation of the placenta is essential, as abruption of the posteriorly inserted placenta is particularly 'silent'. Although the timing of delivery will obviously vary according to the clinical situation, the pregnancy should be terminated when the patient is 37 weeks pregnant, is in active labour, or has excessive bleeding and/or pain. The method of delivery is governed by a consideration of the following points: clinical acute abruption may be preceded by previous episodes of asymptomatic premature separation of the placenta;² there are no tests currently available which can readily determine the degree of placental separation or the integrity of the remainder of the placenta; the early warning sign of uterine tenderness and pain is absent when the placenta is situated posteriorly; the process of abruption can occur very rapidly without necessarily producing signs of foetal distress (case 1). Under these circumstances early delivery is in the best interests of the foetus. Elective Caesarean section, by preventing the possible disrupting effect of labour on the placental site, will help to ensure a live-born infant. It is therefore to be preferred, especially in instances of posteriorly inserted placenta. Hibbard and Jeffcoate⁷ do not agree and maintain that the more liberal use of Caesarean section in their series would have made little difference to the perinatal mortality. Acceptance of their viewpoint would, however, depend on the clinical condition of both mother and foetus on admission. The 2 live births obtained in our series (cases 3 and 4), were due to the recognition of a major degree of abruption and the early recourse to Caesarean section. Conversely, the baby born to case 1 would have been saved had a Caesarean section been performed earlier.

Abruptio placentae is said to recur in subsequent pregnancies,³ but the scales are, nevertheless, weighted against this possibility. Normal spontaneous vaginal deliveries can, therefore, be anticipated unless an additional recurring factor, e.g. pelvic contracture, is present. Provided the patient's clotting mechanism has been checked and/or corrected prior to surgery, Caesarean section per-

formed for foetal distress associated with abruptio placentae has been found to be safe.

CONCLUSION

Attention has been drawn to the occurrence of 'silent' abruption of the normally imbedded placenta. Its significance is that, besides experiencing vaginal bleeding of variable amount and a dull persistent backache (easily confused with the low backache frequently encountered in pregnancy), these patients are frequently asymptomatic. The uterus is invariably soft and non-tender despite the presence of significant abruption of the placenta. In every instance, the placenta is attached to the posterior wall of the upper uterine segment.

Despite the apparently benign presentation of this condition, the associated intra-uterine haemorrhage may be excessive, and the foetal mortality high. Early recourse to Caesarean section is advised to ensure a live birth. The antenatal management of patients known to have minor posterior placental abruption is more difficult, due to the inadequacy of techniques for assessing the degree of placental damage, function, and disruption.

Because of the tendency of patients with asymptomatic premature separation of the placenta to progress to clinical abruption rapidly and silently, it is suggested that the pregnancy be terminated by Caesarean section at 37 weeks' gestation, or earlier if labour sets in, or if the haemorrhage and/or pain become excessive.

The above approach should be judged in the light of the following: the so-called 'unclassified' group of antepartum haemorrhages is one of the largest, and accounts for almost half of the foetal wastage from antepartum haemorrhage.¹ As stated by Donald,⁴ 'The risks to the foetus in placenta praevia are obvious, but it has only now become recognised that the baby is at even greater risk when the source of antepartum bleeding is not determinable.' Premature separation or abruption of the posteriorly inserted placenta, or both, fall into this category.

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