Frequent fetal heart-rate monitoring for early detection of abruptio placentae in severe proteinuric hypertension

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Summary

Abruptio placentae occurred in 16 of 132 patients with severe pre-eclampsia who were admitted to an obstetric high-risk ward before 34 weeks' gestation. These 16 patients were compared with those who did not develop abruptio placentae. Systolic and diastolic blood pressure levels, proteinuria and birth weights did not differ significantly between the two groups. Apgar scores were significantly lower in the abruptio placentae group. There were 6 intra-uterine and 2 neonatal deaths in the abruptio placentae group (50% perinatal mortality (PNM)) and 3 intra-uterine and 16 neonatal deaths in the other group (18% PNM). Four patients with abruptio placentae presented with abnormal fetal heart-rate patterns and 8 with abdominal pain. No warning signs were present in 3 patients and the fetal heart-rate pattern before delivery was not available in 1 patient. Abnormal fetal heart-rate patterns were present in 5 of the 8 patients who presented with pain. Abruptio placentae occurring in patients with severe proteinuric hypertension carries a high PNM. Frequent monitoring of the fetal heart rate sometimes helps to diagnose fetal distress before the clinical signs of abruption become apparent.

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Abruptio placentae is the most important cause of fetal intra-uterine death in patients with severe proteinuric hypertension. In a recent study of 129 patients with severe proteinuric hypertension, Odendaal *et al.*[1] found that abruptio placentae caused 36% of intra-uterine deaths. Brink and Odendaal[2] also calculated that placental abruption occurred in 13% of patients with severe proteinuric hypertension. Sibai *et al.*[3] reported that conservative management of severe proteinuric hypertension in the midtrimester was complicated by abruptio placentae in 22% of patients. The same researchers found that abruptio placentae occurred in 20% of patients with the haemolysis, elevated liver enzymes and low platelets syndrome.[4]

A study was undertaken to determine whether the patient with severe proteinuric hypertension at risk for abruptio placentae could be identified and whether frequent monitoring of the fetal heart rate (FHR) could detect fetal distress before the well-known clinical signs of abruptio placentae become apparent.

Patients and methods

Clinical records of 132 patients with severe proteinuric hypertension, 16 of whom developed abruptio placentae, were studied. All patients with severe proteinuric hypertension were admitted to a high-risk obstetric ward where all patient information was accurately recorded. Treatment of these patients has been previously described in detail.[1] Essentially it consists of prevention of convulsions with magnesium sulphate and the control of hypertension with hydralazine. Patients whose fetuses were beyond a gestational age of 34 weeks were delivered soon after stabilisation but those less than 34 weeks were treated expectantly and the condition of both mother and fetus was carefully monitored. Non-stress tests were done three or four times daily or more frequently when suspicious patterns were seen. Tests were interpreted as reactive when two or more accelerations (at least 15/min for 15 s or more) were present. Absence of sufficient accelerations indicated a non-reactive test. Stress tests were negative when sufficient uterine contractions (3 in 10 minutes, each with a duration of at least 45 seconds) did not cause late decelerations of the FHR. Repeated late decelerations in the absence of supine hypotension or over-stimulation indicated a positive test. Tachycardia was defined as a heart rate of 160/min or more for at least 5 minutes.

Pregnancies were terminated for specific maternal or fetal indications or when a gestational age of 34 weeks was reached. After delivery the placenta was examined for signs of abruption. A retroplacental clot exceeding 15% of the uterine surface was accepted for the diagnosis of abruptio placentae. Patients with abruptio placentae were then compared with those who did not abrupt in respect of age, gravidity, gestational age, hypertension and proteinuria and infants' birth weight, Apgar score, FHR patterns and perinatal outcome.

Results

Since 10 patients had either spontaneous or induced abortions, the actual study comprised 122 patients of whom 16 had abruptio placentae (Table I). Maternal age, gravidity, gestational age, severity of hypertension, amount of proteinuria and infants' birth weight did not differ significantly. The 5-minute Apgar score was significantly lower in the newborns of the abruption group (Table II). There were 6 intra-uterine deaths in the abruption group and 3 in the other group. There were 18 neonatal deaths of which 2 occurred after abruptio placentae. Abruptio placentae was therefore accompanied by a perinatal mortality (PNM) rate of 50% in contrast with the 18% PNM rate of patients without abruption (Table III).

Eight patients who had abruptio placentae presented with abdominal pain and 4 with abnormal FHR patterns. There were no warning signs in 3 patients and 1 patient had no abdominal pain but the FHR recording was lost; in 2 of these patients abruptio placentae was discovered after delivery and in the remaining 2 when the fetal heart could not be heard.

The FHR was monitored immediately before delivery in 7 of the 8 patients who presented with abdominal pain. It was reactive in 2 patients and non-reactive in 1. Four patients demonstrated either late or variable decelerations. Basal tachycardia was noted in 5 patients with abruptio placentae and in 3

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TABLE I. ABRUPTIO PLACENTAE IN PATIENTS WITH PRE-ECLAMPSIA

Patient	Fetal monitoring			First warning sign	Delivery	Outcome	Birth weight (g)	Abruptio placentae (%)
	Day before	2nd last test	Last test					
1	Late decelerations	Non-reactive	Non-reactive (4 h before IUD)	Pain	Vaginal	IUD	1 200	50
2	Negative ST	Reactive	Basal tachycardia (8 h later), variable decelerations, basal bradycardia	Pain	C/S	Good	1 780	30
3	—	—	Basal tachycardia, late deceleration, typical contraction pattern	Pain	C/S	Good	1 770	15
4	Non-reactive	Negative ST	Negative ST	None	Vaginal	IUD	960	80
5	Reactive	Reactive	Reactive	Late decelerations during prostaglandin induction	C/S	Good	1 690	30
6	Non-reactive	Non-reactive	Basal tachycardia, late decelerations	Abnormal FHR	C/S	Neonatal death	820	40
7	Non-reactive	Non-reactive	Late decelerations	Pain	C/S	Good	1 500	10
8	Negative ST	—	—	Vague pain	Vaginal	IUD	1 760	60
9	Reactive	Reactive	Reactive	None	Vaginal	Good	1 500	10
10	Non-reactive	Late decelerations	Basal tachycardia, late decelerations	Abnormal FHR	C/S	Good	1 255	?
11	—	Basal tachycardia, late decelerations	Basal tachycardia, late decelerations	Abnormal FHR	C/S	Good	1 555	35 (couvellaire uterus)
12	Reactive	Non-reactive	Non-reactive	Pain	Vaginal	IUD	900	80
13	Reactive	Negative ST	Reactive	Pain	C/S	IUD	1 540	60
14	Reactive	Reactive	Non-reactive	None	Vaginal	IUD	760	25
15	—	—	—	None	C/S	Good	1 030	10
16	—	—	Reactive	Pain	C/S	Neonatal death	935	10

ST = stress test; C/S = caesarean section; FHR = fetal heart rate; IUD = intra-uterine death.

TABLE II. COMPARISON (MEAN ± SD) BETWEEN PATIENTS WITH AND WITHOUT ABRUPTIO PLACENTAE

	Abruptio placentae ($N = 16$)	No abruptio placentae ($N = 106$)	Significance*
Age (yrs)	25,9 ± 5,6	26,2 ± 6,7	NS
Gravidity	2,4 ± 1,6	2,5 ± 1,7	NS
Duration of pregnancy (wks)	29,5 ± 2,3	29,7 ± 3,1	NS
SBP (mmHg)	157 ± 15	159 ± 24	NS
DBP (mmHg)	101 ± 9	103 ± 14	NS
Proteinuria (+)	2-3 ± 1	2-3 ± 1	NS
Birth weight (g)	1 310 ± 368	1 322 ± 499	NS
5-min Apgar score	4,6 ± 4,1	8 ± 2,4	< 0,01

*Paired Student's *t*-test.
SBP = systolic blood pressure; DBP = diastolic blood pressure; NS = not significant.

TABLE III. PERINATAL DEATHS (PND)

	Abruptio placentae	No abruptio placentae	Total
PND	8 (50%)	19 (18%)	27
No PND	8 (50%)	87 (82%)	95
Total	16	106	122

Chi-square test = 6,5424; $P < 0,01$.

TABLE IV. FETAL HEART RATE PATTERNS

	Abruptio placentae	No abruptio placentae	Total
Reactive	4 (29%)	37 (43%)	41
Non-reactive	10 (71%)	50 (57%)	60
Total	14	87	101

Chi-square test = 0,4878; $P > 0,1$.

Late deceleration	6 (43%)	14 (16%)	20
No late deceleration	8 (57%)	73 (84%)	81
Total	14	87	101

Chi-square test = 3,9107; $P < 0,05$.

in the other group. Reactive FHR patterns were found in 29% and 43% of the abruptio placentae group and the other group respectively. In 1 patient with a reactive pattern the abruptio placentae which developed shortly afterwards was severe enough to have caused intra-uterine death and in another patient with a reactive pattern, the abruption was followed by neonatal death. Late decelerations were found in 43% and 16% of these two groups respectively (Table IV).

Discussion

Clinical parameters such as the severity of the hypertension or degree of proteinuria were of no help in determining the risk of abruptio placentae. All patients with severe proteinuric hypertension should therefore be regarded as at risk for abruptio placentae. Although it has been shown that smoking increases the risk for abruptio placentae,[5] it is uncertain whether it further increases the risk in patients with proteinuric hypertension.

The high PNM rate of abruptio placentae was again demonstrated in this study. The 50% PNM rate is higher than the 39% overall rate of this unit[2] and the 30 - 50% reported elsewhere.[6-8] This is probably owing to the underlying placental insufficiency in patients with severe proteinuric hypertension.

There were more late decelerations of the FHR in patients with abruptio placentae. This was to be expected although late decelerations were present in some patients before a clinical diagnosis of abruptio placentae could be made. On the other hand, most of the patients with a clinical diagnosis of abruptio placentae demonstrated abnormal FHR patterns before delivery. It therefore seems that abnormal FHR patterns could be an early indication of fetal distress caused by abruption. Possible mechanisms for the fetal distress are a smaller gas exchange area in the placenta, a higher uterine tone reducing intervillous space perfusion and a pre-existing placental insufficiency.

Although most of the FHR patterns were abnormal before delivery, 1 patient experienced intra-uterine death caused by abruptio placentae in spite of the reactive pattern recorded earlier in the day. In another patient with a reactive pattern, the abruption was only diagnosed at delivery.

The PNM rate of 50% questions the conservative management of patients with severe proteinuric hypertension but if one considers the fact that there was only 1 intra-uterine death in the 8 patients where late decelerations were seen and 1 neonatal death where the newborn weighed 820 g, the PNM rate of this subgroup was not high. Frequent monitoring of the FHR in patients with severe proteinuric hypertension could prevent some of the intra-uterine deaths caused by abruptio placentae.

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