COAGULATION FAILURE FOLLOWING INDUCTION OF MIDTRIMESTER ABORTIONS WITH PROSTAGLANDINS

(A Case Report)

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It is well known that midtrimester abortions with intra-amniotic saline is associated with significant coagulopathy due to intravascular Coagulation (Stander *et al*, 1971) Induction with hyperosmolar solutions of Urea and manitol have also given rise to similar complications (Rajan, 1976; Deshmukh *et al*, 1980). Prostaglandins are claimed to be safe abortifacients and rarely lead to coagulation defects.

In our experience of 90 midtrimester abortions with Prostoglandin during last one year, we encounted one case of coagulation failure following intraamniotic prostaglandin and because of its rarity it is reported.

CASE REPORT

Mrs. S. 35 yrs., gravida 4, Para 3 was admitted for medical termination of pregnancy on 26-10-79 with history of 4 months amenorrhoea. On examination, her general condition was good. Abdominal and Vaginal examination confirmed that she was 18 weeks pregnant. The cervix was soft and closed, uterus was enlarged to 18 weeks and adenexa were free.

All investigations were within normal limits.

Four hours after admission 10 ml of solution containing 2.5 mg of 15 S 15 M PGF_2 (Prostin-M Upjohn) was injected into the amniotic cavity after aspirating about 5 ml of clear liquor. Nine hours later, uterus started acting and within 16 hours she expelled the foetus. As the placenta was retained, it was removed 25 mins. later. Half an hour after the abortion was completed she had a severe bout of haemorrhage. Immediately general condition deteriorated. Blood Pressure and pulse were not recordable. Exploration of the uterus showed no placental tissue or cervical injury.

Blood taken immediately, did not clot. Tests for fibrinolysis were negative. She was given a total of 5 units of blood, triple strength plasma—1 bottle, and 5 gms of fibrinogen intravenously. The coagulation defect could not be corrected. She failed to respond to the therapy and expired 6 hours following the abortion.

Discussion

Blood coagulation studies during midtrimester abortion with prostaglandins do not indicate any significant changes in coagulation mechanism. In a study of 30 such patients. Philips *et al* (1974) found that intra-amniotic prostaglandin resulted in increased levels of fibrinogen, platelets factors VIII and V profibrinolysis and fibrinolytic inhibitors during 24 hours after medication. These values returned to normal after abortion was completed.

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Moreover Kleiner *et al* (1974) observed that coagulation failure during Prostaglandin abortions may show a minor degree of defibrination "Which is substantially less than that seen with saline abortions". Earlier Badraoui *et al* (1973) had concluded after serial blood coagulation studies during second trimester abortions with extra-amniotic and intra-amniotic prostoglandins, the transient changes in the coagulation system are much less than those accompanying saline abortions or abdominal deliveries in late pregnancies. Unlike with saline abortions where the induction abortion interval is prolonged to 32-34 hours, with Prostaglandin analogues, the abortions occur in a much shorter interval of 14-18 hours. Thus the risk of defibrination occurring with Prostaglandin is much less, yet occasionally it does occur.

This has been reported following intra-amniotic injection of 15 (s) 15 M Prostaglandin $F_2\alpha$, by Dutta *et al* (979). The induction abortion interval was 23 hours and the patient had profuse haemorrhage soon after placental expulsion. She survived, after 6 units of blood transfusion. The fibrinogen level was 88 mgs%. Severe and fatal coagulation failure following extra-amniotic 15 (s) 15 M PGE_2 α has been reported by Mukherjee *et al* in an ICMR report (1979). In the latter case, the induction-abortion interval was 39 hours. She died 8 hours later inspite of blood transfusion and supportive therapy. In our case the induction abortion interval was shorter i.e. 16 hours and severe degree of coagulation defect occurred.

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