Induction of labor with vaginal prostaglandin-E2 in women with one previous cesarean section

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Induction of labor is considered necessary in certain clinical situations. These include postdate pregnancy, term premature rupture of membranes, preeclampsia, intrauterine growth retardation, and significant medical diseases such as diabetes mellitus at term. Intravaginal or intracervical dinoprostone (prostaglandin-E2 [PGE2]) is the most used pharmacologic method to ripen the cervix and induce labor. Cervical ripening and labor induction in women with one previous low transverse cesarean section (CS) is controversial. The objective of this study was to assess the safety and effectiveness of induction of labor with vaginal PGE2 in candidate women for vaginal birth after cesarean (VBAC).

From January 1995 to December 2000, there were 510 induction of labor with vaginal PGE2 (dinoprostone; ProstinTM E2, Upjohn, London, United Kingdom) at King Fahad Armed Forces Hospital, Jeddah, Kingdom of Saudi Arabia. The outcome of induction of labor with vaginal PGE2 (3 mg tablet every 6 hours for a maximum of 3 doses) in 41 women with one previous low transverse CS was compared to the outcome of spontaneous labor of 82 women with one previous CS matched for age, parity, and gestational age at delivery. There was no statistical significant differences in the duration of labor, fetal birth weight, estimated blood loss at delivery, and Apgar scores in the 2 groups. In the induction group, 27 (65.9%) women delivered vaginally and 14 (34.1%) women by emergency cesarean section (ECS). In the control group, 58 (70.7%) women delivered vaginally and 24 (29.3%) women by ECS. However, in the induction group the mean duration of the hospital stay of the mother was longer (p = 0.019) and there was one (2.4%) asymptomatic uterine dehiscence discovered at CS compared to one (1.2%) in the control group. Both cases of asymptomatic uterine dehiscence of the CS scar were easily repaired and no hysterectomy was performed. In a review of 10 studied published in 2000,[1] the incidence of uterine scar disruption was not differ from women who received PGE2 and women who entered labor spontaneously (1.6% versus 1.23%, odds ratio of 1.46, 95% confidence interval 0.96-2.22). However, more recent studies showed an increase in the uterine rupture and decreased vaginal delivery rates with induction with PGE2.[2,3] The American College of Obstetrics and Gynecologist[4] based on limited or inconsistent scientific evidence recommended that the use of prostaglandin gel for VBAC "requires close patient monitoring". It has been shown repeatedly from many studies of VBAC that one of the important prognostic factors for success is history of previous vaginal delivery. This was not carried out in other studies. Our findings suggest that induction of labor with PGE2 in women candidates for VBAC is effective and may be safe. However, the couple should be counseled on the potential increased risk of uterine scar dehiscence or rupture.

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The association of acute myocardial infarction and pregnancy loss in young female with primary antiphospholipid syndrome

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Acute arterial and venous thrombosis is a common manifestation of antiphospholipid syndrome.[1] Acute myocardial infarction may be a clinical manifestation in young adults.[2] Pregnancy in women who are positive for antiphospholipid antibodies may have recurrent pregnancy loss.[3] We describe a young female with no history of venous thrombosis, presented with an incomplete abortion with complicated acute myocardial infarction, thrombolytic therapy was given with no complications. Her blood test was positive for antiphospholipid antibody and the case proved to be primary antiphospholipid syndrome.

A 21-year-old female patient of 10 weeks gestation presented with 2 days history of mild vaginal bleeding and fever. She was admitted to the hospital and managed with intravenous (I.V.) fluid and I.V. metronidazole and amoxicillin clavulanate. She was planned for dilatation and curettage in <24 hours in the hospital, but she developed acute retrosternal chest pain followed by dyspnea, oxygen desaturation of 85%. She developed acute pulmonary edema clinically and radiologically. The 12 leads electrocardiogram showed acute ischemic changes with ST segment elevation of 2 mm. At leads I, III and a augmented unipolar left leg lead with reciprocal ST segment depression at precordial and lateral leads. Clinical examination revealed blood pressure of 95/60 mm Hg, heart rate of 110 beat per minute, respiratory rate of 24 beat per minute and temperature of 37.70C. Chest with fine bibasal rales and S3 gallop with soft systolic murmur of 2/6 in the apical area. Laboratory blood result showed white blood cell of 8000/uL, hemoglobin of 10 gm/dl, and platelet of 345,000/uL. Peaked creatinine kinase isoenzyme of 132 U/L, total creatinine kinase of 1990 U/L and aspartate aminotransferase of 148 U/L, alanine aminotransferase of 70 U/L, urea of 8 mmol/L, creatinine of 90 umol/L, bilirubin 12 umol/L, alkaline phosphatase of 124 U/L, albumin of 40 g/L, globulin 30 g/L, cholesterol 4.2 mmol/L, triglyceride 2.1 mmol/L, international normalized ratio (INR) of 1.5, activated partial thromboplastine time 52 per second, complement C3 of 0.02 mg/dl (N = 0.2-0.5), C4 of 0.5 mg/dl (N = 0.5-1.2). Antiphospholipid antibody and anticardiolipin antibody were both positive. Antinuclear factor (ANF) was positive. Anti-double strand DNA antibody and smooth muscle antibodies were negative. Furosemide and dopamine I.V. infusion at 3 ug/kg per minute were given. Streptokinase I.V. infusion of 1.5 million units administered over one hour. Post thrombolytic electrocardiogram showed reduction of ST amplitude in inferior leads and normalization of precordial leads in favor of successful thrombolytic therapy. Echo showed inferior wall hypokinesia, mild mitral regurgitation, no echo dense masses seen. Left ventricle wall and cavity dimensions were normal, overall left ventricle ejection fraction of 45%. Coronary angiography showed no atheromatous narrowing in the left anterior descending or the left circumflex artery or the right coronary artery. She had unremarkable hospital recovery and underwent dilation and curettage later with no complications. She was discharged and maintained on long term Aspirin 100 mg daily and oral anticoagulant to keep INR of 2-3. The most common manifestation of antiphospholipid syndrome is deep venous thrombosis of the leg; half of these patients develop pulmonary emboli. Arterial thromboses are less common than venous thromboses and manifest with ischemia or infarction. The brain is the most common site of arterial thrombosis with stroke and transient ischemic attacks, account for 50% of arterial occlusion, coronary artery occlusion account for 23%; other sites are the subclavian, renal, retinal and pedal arteries.[4] In this case, a young female presented for the first time with incomplete abortion complicated with acute inferior myocardial infarction. She was diagnosed as primary antiphospholipid antibody syndrome, as the blood assays were positive for antiphospholipid antibody and anticardiolipin antibody. Primary antiphospholipid syndrome is one of several prothrombotic states that occur in young patients, in which recurrent thrombosis can occur with both venous and arterial beds. The administration of thrombolytic therapy although is debatable in proven cases of primary antiphospholipid syndrome due to the high risk of bleeding, was given successfully in this case, with no bleeding complication that, in keeping with others who had reported the safe administration of thrombolytic therapy in a similar case.[5] Primary coronary angioplasty is regarded as a superior alternative to thrombolytic therapy in such case, but it was not contemplated due to the lack of accessibility. The absence of atheromatous narrowing in the precordial artery on angiography indicated the likelihood of arterial thrombus as a cause of acute myocardial infarction that probably dissolved by thrombolysis. So in the clinical setting where a young patient presented with pregnancy loss and acute myocardial infarction, screening for antiphospholipid antibody as an attributable factor with other assays that are sensitive for lupus anticoagulant antibodies is mandatory, as the long term line of management is different.

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