SHOULD CERVIX BE DILATED AT NON-LABOR CAESAREAN SECTION? MAJOR POSTPARTUM HEMORRHAGE AFTER NON-LABOR CESAREAN FOLLOWED BY DISSEMINATED INTRAVASCULAR COAGULATION DUE TO MARKEDLY STENOTIC CERVICAL OS

Shashikant L Sholapurkar1*, Peter Forster2

1Shashikant L Sholapurkar, Department of Obstetrics and Gynaecology, Royal United Hospital Bath NHS foundation Trust, Bath, BA1 3NG, UK.
2Department of Anesthesiology, Royal United Hospital Bath NHS foundation Trust, Bath, BA1 3NG, UK.

*Corresponding Author: Shashikant L Sholapurkar, Department of Obstetrics and Gynaecology, Royal United Hospital Bath NHS foundation Trust, Bath, BA1 3NG, UK. Tel: 44 (0) 1225 825634, Fax: 44 (0) 1225 825464, Email: s.sholapurkar@nhs.net

ABSTRACT

There is insufficient evidence for mechanical dilatation of cervix at non-labor cesarean section for reducing postpartum haemorrhage or infection; hence it has been recently proposed that this practice be discontinued. We present a very rare but highly significant case of major postpartum haemorrhage following non-labor cesarean when a completely stenotic cervix was found from previous diathermy loop excision for cervical intraepithelial neoplasia. Four hours after caesarean the patient collapsed with sudden major vaginal bleeding with disseminated intravascular coagulation (DIC) requiring expeditious resuscitation and return to theatre. Blood loss came under control with replacement of blood and clotting factors. Although this complication would remain very rare, in future, there would

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be increasing incidence of caesareans after cervical excisional treatment for precancer and cancer. We recommend checking the cervical patency with a finger or a 10 mm dilator during non-labor caesarean. This is not just to avoid occasional cases of mild atonic haemorrhage but also to prevent very rare but catastrophic complication of DIC due to absorption of thromboplastins from trapped blood inside the uterine cavity due to intrauterine pressure created by a well retracted uterus.

**Keywords:** Cervical dilatation; elective caesarean; non-laborcesarean; pre-laborcesarean; postpartum haemorrhage; disseminated intravascular coagulation.

**INTRODUCTION**

Caesarean section is by far the commonest major surgical procedure across the world. It used to be a standard practice for obstetricians to dilate (check) the cervical os using a finger or an instrument during non-laborcesarean section. A recent Cochrane systematic review reported that there is insufficient evidence of mechanical dilatation of cervix at non-labor (pre-labor or elective) caesarean section for reducing postoperative morbidity (postpartum haemorrhage or infection) but more randomised trials (RCTs) are required. [1] However, despite insufficient evidence, over the last few years it has become common practice not to examine patency of the cervix during non-labor caesareans with the British national guideline [2] making no recommendation or any mention. The recent edition of a leading Textbook “Williams Obstetrics” [3] specifically mentions that it is unnecessary to check (or dilate) cervical patency reversing its previous recommendation. [4] It is tempting to simplify surgical practice with wholehearted adoption of available evidence (even if inconclusive or lacking) to avoid a supposedly extra operative step. On the other hand, checking cervical patency is a simple and very quick procedure which has been shown not to have any adverse effects in the trials and systematic review [1]. It is not uncommon for the conclusions based on evidence-based medicine (EBM) to radically change over time. Moreover, very rare but highly significant complications may not be picked up by RCTs of a few hundred cases. Contentious evidence from empiricism if inconsistent with common experience in surgical practice should be treated cautiously.

We present a very rare but important anonymised case of major postpartum haemorrhage (PPH) after elective caesarean due to disseminated intravascular coagulation (DIC) most likely caused by markedly stenotic cervical os. The implications for clinical practice are discussed in details.

**CASE REPORT**

A fit slim woman in her mid-twenties underwent elective (pre-labor) caesarean section at 39 weeks of gestation for breech presentation. The caesarean section under a spinal anaesthetic took 40 minutes. A girl weighing 3.5kg was delivered in good condition by breech extraction. Placenta was removed complete with membranes by cordtraction. Uterine incision was closed in two layers. Estimated blood loss was 600ml and the patient remained stable throughout the operation. The upper and lower uterine cavity was explored digitally and was confirmed to be empty. As a long-standing personal practice, the Obstetrician checked the cervical patency. However, there was barely a dimple in place of internal cervical os and an index finger could not be passed even a slightest distance into
the os. At this stage, on direct enquiry about any cervical surgery, the patient (who was awake) confirmed that she had had a diathermy loop excision of cervical intraepithelial neoplasia (CIN) a few years earlier. The obstetrician passed a long artery forceps from above through the internal os well past the external os, opened it partially and withdrew it. This was thought to be sufficient to allow drainage of blood. The patient was informed of this.

1500 hours: Start of elective caesarean section during daytime hours.

1550 hours: Patient transferred out of the theatre to recovery area.

1700 hours: The patient was transferred to postnatal ward. No vaginal bleeding or any abnormality of vital signs was noted at this time. Uterus was recorded to be “well retracted / contracted”.

1730 hours: Patient complained to the midwife of feeling quite unwell (intermittent feeling of “passing out”). Pulse was noted to be high (113bpm) but blood pressure was normal at 114/78 mmHg. She was on maintenance infusion of Harman’s solution 1L/ 6 hours. Midwife recorded normal lochia and well-retracted uterus.

1820 hours: Patient had continued to have intermittent feeling of “passing out”. Pulse was 125bpm and BP 113/76. No increased vaginal blood loss had been noticed /recorded so far. An obstetric registrar happened to be on the ward seeing some other patient. The midwife requested the obstetric registrar to review this lady in view of her symptoms. The Registrar witnessed a sudden blood loss of 300ml (weighed and confirmed) but noted the uterus to be well retracted. Patient was looking pale and feeling sick. There was no evidence of any intraperitoneal haemorrhage at all. A full blood count was sent and the result lateron revealed Hb of 90g/L.

1850 hours: BP stable but high pulse of 114 bpm. Normal lochia and well contracted uterus recorded.

1915 hours: Vital signs stable and minimal lochia.

1936 hours: Almost four hours after the caesarean, a small bleed followed by major vaginal bleeding noted by midwife who pulled an emergency bell. The overnight obstetric team urgently attended and resuscitation was commenced. The uterus was described to be boggy and distended for the first time. Two doses of syntometrine were administered. Two litres of Hartman’s solution were infused by now. Abdominal uterine massage was commenced.

1942 hours: A senior anaesthetist attended. The total blood loss was now estimated to be about 2 L with BP 60/30 mmHg. Decision was taken to return the patient to operation theatre immediately for exploration.

1950 hours: Patient in theatre. Major Obstetric haemorrhage protocol [5] initiated. The anaesthetist described patient’s skin as “mottled” and condition as “pre-arrest”. A clotting screen was sent at 1948 hours. The result was available later which showed very low fibrinogen level of 0.4g/L (normal 1.8 - 3.8g/L), activated partial thromboplastin time (APPT) of 65 sec (normal 23 - 32), prothrombin time of 19.2 sec (normal 9.5 - 11.7) and international normalised ratio (INR) of 1.92 (normal 0.8- 1.2).

1957 hours: General anaesthesia was induced. One gram of Tranexamic acid was given intravenously. High dose oxytocin infusion (40 units/4 hrs) was started and a Foley’s catheter was inserted.

2007 hours: On vaginal exploration, the cervix was found to be long and stiff. Two fingers could be forced through. Blood and blood clots were extracted. The weighed blood loss in the theatre was 300ml. 800 mcg of misoprostol was administered per rectally.
2020 hours: A Bakri intrauterine haemostatic balloon was inserted and distended with 200ml saline. Arterial blood gas analysis at this stage showed marked metabolic acidosis despite expeditious resuscitation (pH 7.14, base deficit of 14.1 and lactate of 8 g/L)

2023 hours: Senior obstetric consultant on call arrived in theatre. Second senior anaesthetist from Intensive Care Unit (ITU) also attended. Patient was receiving 4th unit of blood and 3 units of fresh frozen plasma (FFP) by now. Systolic BP remained at 80mmHg and slowly picked up to 120mmHg after blood transfusion.

2057 hours: A midwife had continued vigorous trans-abdominal uterine massage all through for almost an hour. Abdomen was noticed to be getting distended at this stage with evidence of intraperitoneal haemorrhage. Decision was taken to perform a laparotomy through the previous Pfannensteil incision. Cryoprecipitate was administered intravenously. At laparotomy, there was no bleeding from the uterine caesarean scar itself but diffuse bleeding was noted from abdominal wall muscles and peritoneal areas. Four units of platelets were transfused. With replacement of clotting factors with fresh frozen plasma and a few haemostatic sutures, fair haemostasis was achieved and abdomen was closed with two drains in the peritoneal cavity. The patient was transferred to ITU where supportive treatment was continued.

2150 hours: A full blood count was sent. The result later revealed Hb72g/L, white cell count of 18.7 and low platelet count of 70x10^9/L (normal 150 – 400). Clotting screen at this stage had improved with fibrinogen 1.2g/L(normal 1.8-3.8), APTT of 38 sec (normal 23-32) and INR of 1.32. Patient remained apyrexial and C reactive protein (CRP) was 4 with no suggestion of any infection. The total estimated blood loss was about 5 L.

24 hours later: Patient remained stable on ITU and was extubated. There was continued drop of Hb to 77g/L despite blood transfusions and platelet count was still low at100 ×10^9/L. Fibrinogen, APTT and INR had normalised (2.2g/L, 27 sec and 1.11 respectively). There was no continued drainage of blood in the drains and a CT scan after 48 hours confirmed absence of any intraperitoneal blood or haematoma. Patient and the family were debriefed.

Day 5: The patient was discharged after 5 days in good physical condition with a plan to keep under follow up for further debriefing. At discharge blood tests showed Hb 100g/L and platelet count of 277 ×10^9/L.

**DISCUSSION**

This case appears to be very rare and perplexing complication after an apparently straightforward non-labor (elective) cesarean. The overnight obstetric team dealing with the sudden major PPH four hours after the cesarean were not aware of the excessively stenotic cervix encountered during the caesarean and in any case, it would not have been considered during the life-saving emergency management of the catastrophic bleeding. The emergency teams responded in very expeditious manner with excellent management of what was thought to be an atonic uterine bleeding which is the commonest cause of PPH sometimes with retained adherent placental tissue. Indeed, the initial presumption remained that this lady had massive atonic uterine bleeding which caused “consumptive coagulopathy” which then lead to disseminated intravascular coagulation (DIC) causing further vaginal and intraperitoneal bleed. During the case review, this belief was questioned. It was rightly argued that even major bleeding inside the uterine cavity or externally would not “consume” the platelets and clotting factors from the intravascular compartment. There is an exceedingly rare entity called “Dilutional Coagulopathy” which requires around 80% of original blood
volume is replaced by large infusions of replacement fluids. [6] Although, the intravascular clotting factors can be “diluted” by improper resuscitation with fluids, this scenario will not co-exist with DIC or lead to it at all. Hence, the confusing term “consumptive coagulopathy” is best avoided. The Harrison’s text book of internal medicine does not use this term at all. [7] All other authorities state that consumptive coagulopathy is better known as disseminated intravascular coagulation (DIC). Thus, they are synonyms (one and the same) because only DIC will “consume” intravascular clotting factors and not extra vascular haemorrhage. [8]

This case had evidence of DIC right at the outset of massive uterine haemorrhage. Hence, it seems most reasonable that clotting failure from DIC was the cause of this haemorrhage. It seems unlikely that after minimal bleeding and contracted uterus repeatedly documented for four hours after the caesarean, there would be sudden catastrophic “atonic” PPH. Also, atonic PPH cannot explain the presence of coagulopathy (DIC) at the time of haemorrhage. Many hours of prolonged poorly treated severe hemorrhagic shock with widespread microvascular damage would be necessary for DIC to result from atonic PPH. This was clearly not the case in this patient at the outset of haemorrhage or even later. The best (and probably only) explanation which would explain the chain of events, record of vital signs and blood test results was thought to be as follows (Figure 1). More or less normal amount of post-delivery bleeding from placental site got trapped in the uterine cavity due to stenosed cervix. As the intrauterine pressure increased to force this blood out through the cervix, the thromboplastins released from the clotted blood were gradually forced into the systemic circulation through the venous sinuses in the placental bed. This started the cascade or chain reaction of DIC via activation of extrinsic coagulation pathway (somewhat akin to abruption). The systemic absorption of thromboplastins is the well accepted mechanism of DIC in Obstetrics e.g. DIC associated with abruptio placentae or amniotic fluid embolism. [3, 4, 9] This required a very well contracted uterus (as repeated postoperative observations showed in this case) rather than an atonic one. The increased intrauterine pressure ultimately forced open the cervix with loss of about 300 ml of blood about three and a half hours after the caesarean. But the chain reaction of DIC was on-going by this stage which would explain the peripheral shutdown, increasing metabolic acidosis, tachycardia and sympathetic overdrive (patient having a “sinking” feeling) despite stable blood pressure and absence of much bleeding. The DIC presented with coagulation failure causing sudden PPH (distending the uterus as the cervix was only slightly open). Further dilatation of cervix under general anaesthesia to drain the accumulated blood and clots stopped further progression of DIC. It would not have been possible to predict, anticipate or prevent such an exceedingly rare and almost unknown complication. Probably it was better to think of common causes in such a dire emergency. The emergency teams were right to treat the case expeditiously with blood transfusion, replacement of clotting factors to correct coagulation and operative intervention to achieve haemostasis, whatever cause was considered.

The Debate about cervical dilation:

We are unaware of any similar case report where cervical stenosis led to DIC and massive PPH after a non-labor cesarean. Similar rare cases may go unreported or even undiagnosed especially if cervical patency is not examined during caesarean which is more commonly the case in last few years. Confirming the cervical patency used to be a standard procedure for the older generation of obstetricians with a concern that retained blood may lead
to uterine atony but probably very few thought of the possibility DIC from blood clot products (thromboplastins) being forced back into maternal circulation due to intrauterine pressure.

**Figure 1: Chain of Events - DIC leading to uterine and intraperitoneal hemorrhage.**

<table>
<thead>
<tr>
<th>Very tight cervix (despite attempt to probe)</th>
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<tbody>
<tr>
<td>Entrapment of normal bleeding in the uterus with clot formation</td>
</tr>
<tr>
<td>Thromboplastins from blood clot pushed in to maternal circulation through placental bed from high intrauterine pressure (not atonic uterus)</td>
</tr>
<tr>
<td>DIC triggered (similar to abruption), patient feeling unwell (sympathetic overdrive) due to progressive peripheral shutdown and metabolic acidosis</td>
</tr>
<tr>
<td>Clotting factors depleted + Fibrinolytic system activated</td>
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<tr>
<td>Profuse bleeding from placental bed, uterine distension and vaginal bleeding (Evidence of DIC at the onset of bleeding)</td>
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<tr>
<td>Expeditious resuscitation blood transfusion</td>
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<tr>
<td>Examination under anesthesia, Extraction of clots through tight long cervix, Vigorous abdominal massage (in the presence of coagulation failure) leads to intraperitoneal hemorrhage from the operative site</td>
</tr>
<tr>
<td>Hemorrhage comes under control with correction of clotting factors and hemostatic sutures</td>
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The 18th edition of the most widely read textbook Williams Obstetrics in 1989 made the following statement, "If the cervical canal is not known to be patent, it should be probed with a long clamp to assure patency". The recent MOET (Managing Obstetric Emergencies and Trauma) Course manual [6] states, “Check the patency of the internal cervical os and that it is not covered with membranes.” The British national guideline [2] on
caesarean section makes no mention of cervical patency check at all. The 24th edition of William’s Obstetrics (2014) states, “The practice of dilating cervix does not improve infection rates from potential hematometra and is not recommended.” The Cochrane systematic review reported that there is insufficient evidence for mechanical dilatation of cervix at non-labor (pre-labor or elective) caesarean section for reducing postoperative morbidity (PPH or infection) but more randomised trials (RCTs) are required. [1] However, very rare but highly important cases (like the one described above) are unlikely to be captured in the studies including a few hundred cases in the systematic review. [1, 10] Insufficient evidence does not mean lack of evidence of harm due to stenosed cervix. Cervical patency check is a very quick and simple procedure. Moreover, long experience and published studies have not shown risk of infection or any other harm from cervical patency check, which should have become apparent even with a small number of studies available each including a few hundred cases. [1, 10] The obstetricians should pass a gloved finger down through the os and change the glove if desired (for theoretical but unlikely risk of infection). An alternative would be to pass a 10 or 12 mm metal cervical dilator which should then be available on the instrument trolley. However, when cervix is ostensibly tight to allow a finger or 10mm dilator to pass through, we would recommend a progressive dilatation of cervix from above starting with a small dilator and increasing to a 10 mm dilator. Simply passing an artery forceps may not be enough as a learning lesson from this case. There are increasing number of patients having caesareans after cervical surgery for cervical intraepithelial neoplasias (CIN) or even cancer, which can lead to scarring and stenosis. Some patients may even have an abdominal cervical suture following radical trachylectomy for early cervical cancer, although in these cases cervical incompetence may be more of a concern than stenosis. These patients may need lesser dilatation of about 5-6 mm if the cervix found to be obstinately closed. It is not envisaged that cervical dilatation from above could fail but in such an event combined vaginal- abdominal approach could be tried. Awareness of this very rare complication of DIC due to obstinate cervical stenosis is required. Routine checking of cervical patency following non-labor cesarean section and cervical dilatation when required seems a prudent practice.

Declaration of interest:

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