Rupture of uterine muscle hematoma following diagnostic amniocentesis at 18 weeks and uterine dehiscence at term

Zane Krastiņa1,3, Jānis Šavlovskis2,3, Anna Langrate3*, Toms Znotiņš3

INTRODUCTION

Globally, caesarean section operation incidence increases so as proportionally the number of uterine ruptures, reaching 20-80 / 10,000 women who have scar tissue in the uterus after a previous SC. In contrast, relatively less frequent cases with incidence of 1 / 5700-1 / 20,000 when a rupture occurs, are associated with healthy uterine wall muscle. In all these situations it is always difficult to determine whether to manage expectantly or surgically, including repair of the uterine wall in termination of the pregnancy.

Uterine rupture implies a defect in the uterine musculature which can be divided into two groups-complete and incomplete. With term-complete uterine rupture we understand separation of the uterine wall and extravasation of foetal parts, intra-amniotic contents into the peritoneal cavity. It is often associated with acute symptoms and/or blood loss. Factors that can predispose to uterine rupture are multiparity, advanced maternal age, a scarred uterus, a big foetus, mal presentation, and others. Uterine dehiscence / incomplete uterine rupture / silent rupture is classically defined as a disruption of the uterine musculature, without extravasation of the intra-amniotic contents and foetal parts into the peritoneal cavity, and with intact serosa. In most cases it is asymptomatic.

CASE REPORT

Clinical case report and analysis of literature. We searched online databases (EBSCO, PubMed, Scopus, UpToDate) under search names: “uterine rupture”, “uterine dehiscence”, “amniocentesis”, “angiography in...”
pregnancy” to find clinical cases, trials and reviews that are, in our opinion, most relevant to our case.

A 46-year old, gravida 5, para 1-1-0-0 presented in our hospital at 18+5 with symptoms of acute abdomen. At 16 weeks gestation, genetic amniocentesis was performed within a 22-gauge (single insertion) under direct real time ultrasonographic guidance. Clear yellow amniotic fluid was obtained. The ultrasonographic findings were normal. Cytogenetic analysis, later known, revealed a norm; 46, XX chromosome pattern. Now presented at hospital with acute abdominal pain, nausea and abnormal stool. On examination patient was normotensive (120/80 mmHg) with a heart rate of 82 bpm and respiratory rate 16 breaths per minute, oxygen saturation 99%. The heart sounds were regular, both lungs were clear on auscultation. Body temperature was normal (Table 1).

Table 1: Laboratory test.

<table>
<thead>
<tr>
<th>Blood count</th>
<th>On admission (16.05.15 at 12.20)</th>
<th>Pre-operative (16.05.15 at 19.12)</th>
<th>Post-operative (17.05.15 at 12.20)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erythrocytes (106/µL)</td>
<td>3.1</td>
<td>2.43</td>
<td>2.92</td>
<td>4.20-5.40</td>
</tr>
<tr>
<td>Hemoglobin (g/L)</td>
<td>85</td>
<td>67</td>
<td>81</td>
<td>120-160</td>
</tr>
<tr>
<td>MCV (fL)</td>
<td>86.2</td>
<td>82</td>
<td>84</td>
<td>80-100</td>
</tr>
<tr>
<td>MCH (pg)</td>
<td>27</td>
<td>28</td>
<td>28</td>
<td>27-33</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>27</td>
<td>20</td>
<td>24</td>
<td>37-47</td>
</tr>
<tr>
<td>Platelets (103 /µL)</td>
<td>248</td>
<td>200</td>
<td>191</td>
<td>150-400</td>
</tr>
<tr>
<td>Leukocytes (103 /µL)</td>
<td>9.7</td>
<td>7.9</td>
<td>7.2</td>
<td>4.0-10.0</td>
</tr>
<tr>
<td>Granulocytes (%)</td>
<td>83.0</td>
<td>77.9</td>
<td>77.9</td>
<td>30.0-70.0</td>
</tr>
<tr>
<td>Biochemistry</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C-reactive protein (mg/L)</td>
<td>7.6</td>
<td>5.2</td>
<td>-</td>
<td>0-5.0</td>
</tr>
<tr>
<td>Creatinin (µmol/l)</td>
<td>40</td>
<td>32</td>
<td>34</td>
<td>44-97</td>
</tr>
<tr>
<td>Coagulogramm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>APTT (s)</td>
<td>24.7</td>
<td></td>
<td></td>
<td>26.0-36.0</td>
</tr>
<tr>
<td>INR</td>
<td>0.9</td>
<td></td>
<td></td>
<td>0.8-1.2</td>
</tr>
<tr>
<td>PT (%)</td>
<td>135.2</td>
<td></td>
<td></td>
<td>70.0-120.0</td>
</tr>
<tr>
<td>PT (%)</td>
<td>135.2</td>
<td></td>
<td></td>
<td>70.0-120.0</td>
</tr>
<tr>
<td>Fibrinogen (g/l)</td>
<td>4.1</td>
<td></td>
<td></td>
<td>1.8-3.6</td>
</tr>
</tbody>
</table>

MCV, mean corpuscular volume; MCH, mean corpuscular haemoglobin, MCHC, mean corpuscular haemoglobin concentration; RDW, red blood distribution; APTT, Activated Partial Thromboplastin Time.

On first examination, the abdomen was soft and distended. Abdominal pain was located in left upper quadrant and epigastrium. Peritoneal symptoms were negative. Uterus cervix was closed, thick and posterior. Transabdominal ultrasound showed intrauterine pregnancy with positive foetal cardiac activity. In abdominal cavity, free fluid was visualized between liver and left kidney and in left ileocecal region.

Acute appendicitis was suspected. An emergency upper midline laparotomy under general anaesthesia was performed. Upon opening the abdominal cavity, a hematoma was revealed on the right side of uterus – at the site where amniocentesis needle was placed. Incision on hematoma was performed and approximately 1500 mL of dark coloured blood was released. The defect was sutured several times and haemostatic sponge was applied. Right uterine artery was ligated. With suspicion of difficult patient haemostasis vascular embolization was sought. During angiography, no active bleeding was visualized and embolization was not performed (Figure 1 and 2).

Figure 1: Digital subtraction angiography of the visceral branches of the right internal iliac artery, shows a ligated right uterine artery (black arrow) and several permeable branches of the right uterine artery (white arrows) without signs of extravasation.
Patient was discharged from hospital 10 days after surgery with ongoing pregnancy.

At gestational age of 37+6 woman was admitted for planned C-section. A healthy boy was born 3370g, 51 cm, Apgar 8/9. Upon entering the abdominal cavity suture dehiscence was observed in uterine wall (4x4cm) covered only with serosa and surrounded by fibrinous Tachosil remainings and placenta increta revealed during operation. Postoperative management was without complications.

Figure 2: Digital subtraction angiography of the left internal iliac artery. A large diameter left internal iliac artery (white arrows) is shown. Note the intensive contrasting of the uterus due to left-to-right shunting throw collateral vessels without signs of extravasation. The centre line is marked by a dashed line.

**DISCUSSION**

Genetic amniocentesis is a common invasive diagnostic procedure performed between 15 and 20 week of gestation. Early amniocentesis (performed before 14 week) is not recommended because of increased risk of membrane rupture, development abnormalities (clubfoot) and fetal loss. Amniotic culture failures are more common after early amniocentesis procedure.

Indications of amniocentesis include high risk at triple/double test, abnormal ultrasound findings, history of child with Down syndrome and baby anomalies, advanced maternal age, hydrops fetalis, severe IUGR, prenatal infection. Even though amniocentesis is reliable diagnosis method, it causes certain complications and these risks should be kept in mind prior to the procedure. Minor complications due to amniocentesis are amniotic fluid leakage and transient vaginal spotting that occurs 1 – 2% of all cases. Preterm premature rupture of membranes after amniocentesis has a better perinatal outcome compared with spontaneous rupture of membranes at similar gestational ages. In a study by Borgida et al., preterm premature rupture of membranes after midtrimester genetic amniocentesis occurred in 1% of cases. The perinatal survival rate was 91% and the mean time of delivery 34.2 weeks.

Cases of fetal needle injuries have been reported, but are rare when amniocentesis is performed under continus ultrasonographic guidance.

Amniocentesis carries a risk for mother-to-child transmission of blood borne disease such as hepatitis B virus, hepatitis C virus and Human immunodeficiency virus (HIV).

The data regarding amniocentesis in women with hepatitis C virus (HCV) are limited. In a French study, 22 pregnant women who were positive for HCV underwent midtrimester genetic amniocentesis. 16 women had detectable virus RNA in serum. Only one patient had detectable viral RNA in amniotic fluid after the procedure. Non of the resulting 10 newborns, who were tested were positive for HCV RNA, including the one with virus in amniotic fluid.

Current evidence suggest that the risk of vertical transmission of HIV in women treated with combination antiretroviral therapy (CART), and where viral load is undetectable, is not increased. Counseling should be done prior to the procedure. In a French Perinatal Cohort study the transmission rate due to amniocentesis in women who received CART therapy was 0.00%. The transmission rates were higher in women who received no therapy (25%), zidovudine monotherapy (6.1%) and a combination of two anti-retroviral agents (3.3%).

Current evidence suggest that amniocentesis increases the risk of mother-to-child transmission in hepatitis B positive women and the rate of transmission depends on viral load.

The faster trial by Eddleman et al showed a procedure-related foetal loss rate of 0.15%. A recent meta-analysis estimated the risk of miscarriage due to procedure 0.11% (1/900).

Retrospective cohort study reported a risk of miscarriage of 0.48% (1/208). The ACOG Bulletin nr. 162 states that the procedure related risk of miscarriage due to amniocentesis is approximately 0.1 – 0.3%. Eddleman et al. stated that the median time to pregnancy loss after amniocentesis, if considered at all, was 3 weeks.

Maternal complications related to the procedure, such as amnionitis, are rare, occurring in less than 1/1000 procedures. Investigating literature, we found only one published case report in which amniocentesis is proposed to be the leading cause of uterine rupture. Williams and Stallworthy reported a case of a deadly uterine hematoma at the puncture site. Cases of significant maternal blood loss secondary to lacerated uterine artery or vein have been reported. Angiography possesses two important risk factors for the developing foetus: exposure to ionizing radiation and iodinated contrast media (ICM) exposure.
The absorbed radiation dose to the foetus can be estimated prior to the procedure (prospectively) and appropriate safety precautions such as shielding can be done. Diagnostic imaging procedures typically expose the foetus to less than 50 mGy (5 rads) of ionizing radiation. There is no evidence of an increased risk of foetal anomalies, intellectual disability, growth restriction, or pregnancy loss from ionizing radiation at doses less than 50 mGy.\textsuperscript{19} According to American College of Radiology (ACR), the use of iodinated contrast media (ICM) during pregnancy is safe and women should not be routinely screened for pregnancy prior to the procedure.\textsuperscript{20} ACR states that the risk of thyroid dysfunction in newborns exposed to iodinated contrast media during foetal development is negligible.

American College of Radiology concludes that it is safe for women to breastfeed after receiving contrast medium. If the mother is concerned about the potential side effects of ICM in breastmilk (mainly allergic reactions) she may abstain from breastfeeding for 12-24 hours after receiving ICM injections.\textsuperscript{20}

**CONCLUSION**

Obstetric complications after invasive diagnostic amniocentesis has been reported in extremely rare cases ranging from 0.5-1.2%. Uterine rupture must be ruled out in all pregnant women complaining of acute abdominal pain especially if there is a history of curettage, increased maternal age and multiparity, and amniocentesis in current pregnancy. Excessive suturing on the pregnant uterus above the placental bed might be the cause for placenta increta in this case.

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**REFERENCES**

18. ACR Practice Guideline for imaging pregnant or potentially pregnant adolescents and women with...

