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Atopobium Vaginae Bacteremia with Fetal Loss after Chorionic Villus Sampling: A Case Report

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Abstract Chorionic villus sampling (CVS) is a procedure in which biopsies of placental tissue are obtained for prenatal genetic diagnosis. Risk of infection after CVS is low and only sporadic cases of postprocedural sepsis have been reported. Clostridium perfrigens and Escherichia coli are the most commonly described pathogens in cases of sepsis after prenatal invasive diagnostic procedures. However, this report describes a case of sepsis with fetal loss after CVS caused by Atopobium vaginae.

Keywords: *sepsis, chorionic villous sampling, Atopobium vaginae, fetal loss*

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1. Introduction

Chorionic villus sampling (CVS) was introduced in the early 1980's for the detection of chromosomal abnormalities. It is an invasive procedure to biopsy placental tissue for prenatal genetic testing without entering the amniotic sac. [1] CVS carries a very small but finite risk of miscarriage (0.5-1%) [2], although recent literature shows the procedure-related risks to be negligible. [3,4] The procedure is generally performed between a gestational age (GA) of 10 and 13 weeks through a transabdominal or transcervical approach, depending on the preference of the provider and placental location. [5,6] With the arrival of noninvasive prenatal testing (NIPT) as a screening method, the need for invasive diagnostic procedures decreased considerably. [7] Several indications still require invasive diagnostic testing: when a patient is assessed as high-risk based on the results of the NIPT or first-trimester combined test, ultrasound abnormalities such as hydrops fetalis and a predisposed risk of a genetic or chromosomal disorder. [6] The advantage of CVS is that it can be performed early in pregnancy allowing the prospecting parents to make a decision regarding the pregnancy at an earlier stage. In 1-2% of cases the result can be complicated by confined placental mosaicism, requiring a confirmatory amniocentesis after a GA of 15 weeks. [8]

The risk of infection after CVS is low, with incidences ranging from 0% to 0.6% as described in literature. [9-13] Chorioamnionitis following CVS can be caused by ascending infection, inadvertent puncture of the bowel, skin contaminants or pathogens present on the materials and can lead to spontaneous abortion. [2] Transient maternal bacteremia without the presence of clinical infection has been reported by Silverman et al. in two women undergoing transcervical CVS (1.8%). [14] However, only a few cases of postprocedural sepsis are known, which is a very rare but life-threatening complication. [15-19] Sepsis is a clinical syndrome defined as a dysregulated immune response to infection. [20] It is one of the leading causes of death globally which requires early identification to provide adequate treatment and to avoid progression to multiple organ failure or septic shock which can lead to death.

Escherichia coli is the most commonly described pathogen in cases of sepsis after invasive prenatal diagnostic procedures through the transabdominal route, most likely following inoculation of intestinal bacteria. [15,16,17,18,21,22,23]

We present a case of a 30-year old pregnant women who developed sepsis following transcervical CVS caused by Atopobium vaginae, from which invasive infections have only been sporadically described.

2. Case Presentation

A 30-year old gravida 2 para 1 underwent transcervical CVS in the 12th week of pregnancy due to increased nuchal translucency. Her obstetric history included gestational diabetes and a spontaneous vaginal birth, followed by manual removal of a retained placenta. The CVS was performed under ultrasound guidance. The procedure went without complications. Eight days after the procedure the patient developed fever up to 39.7°C and chills. She presented herself at the outpatient clinic at a GA of 13+4 weeks where a normal temperature of 37°C

was measured. The patient was tachycardic (115 bpm) and there was no abdominal pain or vaginal blood loss. Transabdominal ultrasound showed a vital pregnancy. Laboratory results were as follows: C-reactive protein (CRP) 107 mg/L, white blood count (WBC) 3.4 x10^9/L. The urine sample showed no signs of a urinary tract infection. A SARS-CoV-2 PCR and Influenza virus PCR were performed which came back negative. A vaginal culture swab was performed which showed only normal vaginal microbiota after two days. The patient was sent home with clear instructions. The following day the patient returned at the outpatient clinic with ongoing peaking fever up to 40°C. CRP had raised up to 263 mg/L. The patient was admitted due to suspected sepsis following CVS. Two sets of blood cultures (including aerobic and anaerobic incubation) were taken after which intravenous antibiotic treatment with meropenem 3dd1000mg was initiated. At a GA of 13+6 weeks fetal demise was established through transabdominal ultrasound. Induction of labor was initiated with vaginal misoprostol. Due to a decrease of oxygen saturation to 86% and tachypnoea up to 30/min 4.0 liters of oxygen per minute was administered through a nasal cannula with modest effect. Because of clinical deterioration additional diagnostics were performed. Chest X-ray and abdominal CT showed signs of fluid overload, although pneumonia could not be excluded. The ECG was normal. Since there was no sign of clinical improvement, a single dose of gentamicin 250mg was administered intravenously while antibiotic treatment with meropenem was continued. Laboratory results showed a hypokalemia which was supplemented with intravenous potassium 80mmol/24h. Due to increased dyspnea and the presence of crepitations upon lung auscultation the chest X-ray was repeated the following day which showed bibasilar infiltrates suspect for pneumonia. A pulmonary CT was performed to exclude the presence of a lung embolism. The SARS-CoV-2 PCR and Influenza virus PCR were repeated; both were negative. Since the patient expressed progressive abdominal pain with tenderness of the right upper quadrant upon palpation an abdominal ultrasound was performed by the radiologist which showed no abnormalities. Differential diagnosis of the sepsis remained pneumonia or intra-uterine infection. Vancomycin was started to supplement the antibiotic treatment with meropenem and gentamicin. Serum vancomycin concentrations were monitored and vancomycin dose was adjusted accordingly. Vaginal misoprostol was continued without clear effect. Serum potassium levels normalized after which intravenous potassium suppletion was stopped. Urine output remained adequate during admission. Table 1 shows the course of the laboratory results during admission. At the fourth day of admission both the anaerobic blood culture bottles became positive with streptococci after two days of incubation. Two days later, the strain was determined using MALDI-TOF MS (Bruker Daltoniks, Germany) as Atopobium vaginae, confirming a gynecological origin of the sepsis. Antimicrobial resistance was determined using Etest (bioMérieux, France) and EUCAST criteria for Gram-positive anaerobes. The strain was susceptible to penicillin (MIC 0,094 mg/L) and clindamycin (MIC 0,016 mg/L), but resistant to metronidazole (MIC 16 mg/L). The blood culture set taken 24 hours after initiation of meropenem, remained negative. Due to the ongoing fever and chills without the occurrence of an abortion following four days of vaginal misoprostol treatment an aspiration curettage was performed at GA of 14+3 after which the temperature normalized. The evacuated material, a fetal swab and amniotic fluid was sent for culture but resulted in no growth. Antibiotic treatment was switched to oral amoxicillin/clavulanic acid 4dd500/125mg for two weeks. Two days later, the patient was discharged. The patient returned for a checkup after 2 weeks. WBC and CRP normalized (resp. 4.4 x10^9/L and 2.1 mg/L). Transvaginal ultrasound showed a normal uterus with triple layer endometrium.

Table 1. Development of laboratory results during admission (* day of aspiration curettage)

Day of admission	-1	0	1	2	3	4	5*	6	7	
Laboratory test										Reference range
Hemoglobin, blood (mmol/L)	-	-	5.5	5.1	5.5	6.2	5.6	4.8	4.6	7.5-10.0 mmol/L
Hematocrit	-	-	0.25	0.23	0.25	0.30	0.27	0.23	0.22	0.35-0.45
C-reactive protein	107	263	211	211	304	196	153	113	46	<5 mg/L
White blood count	3.4	3.4	5.7	4.1	6.4	5.5	4.8	6.5	6.0	4.0-10.0 x10^9/L
Thrombocytes	-	-	85	77	-	157	151	244	291	150-400 x10^9/L
Activated partial thromboplastin time (aPTT)	-	-	41	41	41	37	39	-	-	25-34 sec
Partial thromboplastin time (PTT)	-	-	15.3	15.2	14	13.5	14	-	-	12.1-15.6 sec
Fibrinogen	-	-	4.3	4.0	5.9	6.6	5.7	-	-	2.0-4.0 g/L
Sodium	-	-	137	136	138	136	140	138	139	135-145 mmol/L
Potassium	-	-	3.0	2.9	3.6	3.7	4.0	3.9	3.9	3.5-5.0 mmol/L
Ureum	-	-	1.3	1.1	1.1	2.0	2.4	1.8	2.5	2.5-6.4 mmol/L
Creatinine	-	-	39	35	36	39	31	34	32	49-90 µmol/L
Glomerular filtration rate (GFR)	-	-	>90	>90	>90	>90	>90	>90	>90	>90 ml/m/1.73m2
Bilirubin	-	-	14	11	9	9	9	6	5	<17 µmol/L
Alkaline phosphatase (ALP)	-	-	94	80	88	105	80	80	73	33-98 U/L
Gamma-glutamyl transferase (GGT)	-	-	55	46	41	39	34	32	31	<38 U/L
Aspartate aminotransferase (AST)	-	-	81	71	49	27	21	18	18	<31 U/L
Alanine aminotransferase (ALT)	-	-	69	64	57	41	34	27	22	<34 U/L
Lactate Dehydrogenase (LDH)	-	-	230	200	240	260	200	190	170	<247 U/L

Table 2. Summary of cases of Atopobium vaginae infection reported in the literature

Reference	Age	Obstetric history	GA	Clinical presentation	Site of isolation	Identification method	Treatment	Outcome
Geissdörfer, 2003 [36]	39y	G0	n.a.	Tuboovarian abscess 2 months following transvaginal oocyte recovery	Abscess swab	16s rRNA sequencing	Surgery (hysterectomy, bilateral salpingectomy, left-sided ovariectomy, appendectomy), antibiotics (cefoxitin i.v. 3dd2g and metronidazole i.v. 2dd0.5g for 5 days)	Discharged 10 days postoperative
Yamagashi, 2011 [37]	33y	P1	n.a.	Endometritis	Intrauterine content	16s rRNA sequencing	Antibiotics (amoxicillin 3dd500mg p.o. for 4 days then meropenem i.v. 2dd0.5g for 2 days)	Discharged after clinical remission
Knoester, 2011 [38]	40y	G7P3	12	Sepsis following CVS	Blood culture and cervical swab	MALDI-TOF MS and 16s rRNA sequencing	Antibiotics (cefuroxime i.v. 3dd750mg then amoxicillin 4dd1g for 14 days) and aspiration curettage	Discharged 5 days after admission
Chan, 2012 [39]	33y	G4P2	39^{+2}	Spontaneous intrapartum sepsis and fetal distress leading to emergency caesarean delivery	Blood culture	MALDI-TOF MS and 16s rRNA sequencing	Antibiotics (amoxicillin/clavulanic acid i.v. 1.2g every 8 hours) then amoxicillin/clavulanic acid p.o. for 14 days. Emergency caesarean section was performed.	Discharged 4 days after delivery
Jacqmin, 2018 [40]	38y	G3P2	12	Sepsis following subchorionic hematoma infection	Blood culture	MALDI-TOF MS and 16s rRNA sequencing	Antibiotics (amoxicillin/clavulanic acid i.v. 1g then amoxicillin p.o. 3dd1g for 21 days)	Termination of pregnancy following PPROM at 20 weeks of gestation
Dauby, 2019 [41]	29y	G1P0	39^{+3}	Spontaneous intrapartum sepsis and fetal distress leading to vacuum assisted vaginal delivery	Blood culture	MALDI-TOF MS and 16s rRNA sequencing	Antibiotics (amoxicillin/clavulanic acid i.v.)	Discharged 3 days after delivery
Taillandier 2020 [42]	57y	G5P0	n.a.	Postoperative peritonitis and septic shock following total hysterectomy and adnexectomy	Peritoneal fluid	Not described	Antibiotics (piperacillin/tazobactam and gentamicin for 7 days)	Discharged after clinical remission
Present case	30y	G2P1	12	Sepsis following CVS	Blood culture	MALDI-TOF MS and 16s rRNA sequencing	Antibiotics (meropenem i.v. 3dd1000mg combined with vancomycin i.v. then amoxicillin/clavulanic acid p.o. for 14 days)	Discharged 8 days after admissions

3. Discussion

Atopobium vaginae is a Gram positive, anaerobic, elliptical or rod-shaped coccobacillum which produces large amounts of lactic acid. [24] In a Gram stain they can occur alone, in pairs, in clumps or in short chains. Its species was first described in 1999 by Rodriguez as nonpathogenic and considered part of the healthy vaginal flora. [24,25] However, since 2004 an association between A. vaginae and bacterial vaginosis (BV) has frequently been described. [26-31] Vaginal loads of A. vaginae and other typical BV pathogens are significantly higher in women with BV than in healthy controls. [32] High vaginal loads are also associated with premature birth. [33,34,35] Whether in our case a high vaginal load of A. vaginalis was present and responsible for bacteremia is unknown since this was not specifically determined. Due to the high recurrence rate of BV (30%) [29], De Backer et al. tested susceptibility to antibiotic treatment of A. vaginae. [31] A variable susceptibility with a minimum inhibition concentration (MIC) ranging from 2 to more than 256 µg/ml for metronidazole, which is the main BV treatment in many countries, was described. [31] Beta-lactam antibiotics have been successfully used as antibiotic treatment in all reported cases in literature. In our case, antimicrobial susceptibility also showed resistance to metronidazole and susceptibility for penicillin.

Invasive infections caused by A. vaginae are rare. To our knowledge, only 7 cases have been described in literature which all were infections involving the female genital tract (Table 2). [36-42] Of these, there was 1 case of sepsis after CVS, 2 cases of intrapartum sepsis, 1 case of sepsis after subchorionic hematoma infection, 1 case of infection after transvaginal oocyte retrieval and 2 other cases. In 5 of these 7 cases A. vaginae caused sepsis. Since its first identification two decades ago, the presence of A. vaginae in areas of the human body other than the female genital tract have not been described. Therefore, when the blood cultures in our case came back positive for A. vaginae, the differential diagnosis of a pulmonary cause of the bacteremia and sepsis was rejected. In retrospect, pulmonary symptoms developed after the patient was diagnosed with sepsis.

The increasing identification of A. vaginae as a cause of invasive infection is probably due to the arrival of newer diagnostic techniques such as 16S rRNA gene sequencing, which allowed accurate identification of A. vaginae in

previous years. Conventional methods such as phenotypic and biochemical tests probably often misidentified A. vaginae as other non-spore-forming Gram-positive bacilli. Currently, A. vaginae can easily be determined using MALDI-TOF MS since the library (8468 MSP) [P2] contains 4 A. vaginae strains. Due to this technique, A. vaginae might be recognized as a pathogen more often in the future.

We suggest two hypotheses for the mechanism of bacteremia and fetal loss in the present case.

In the first hypothesis A. vaginae was transported from the patients vagina or cervix into the maternal bloodstream during the invasive procedure of CVS, as described by Silverman et al., causing bacteremia leading to maternal sepsis. [14] Maternal sepsis can result in fetal loss. High maternal fever, poor oxygenation or a systemic reaction to the pathogen may cause the fetus to die without direct transmission of pathogens to the placenta or fetus. [43,44] Fetal loss can also be caused by subsequent fetal or placental infection. Pathogens can directly damage vital organs of the fetus or placental blood flow can be reduced following infection of the placenta. [43,44] The presence of clinical sepsis in our case a few days before fetal loss occurred substantiates this first hypothesis. Although the uterine microenvironment has been considered a sterile compartment in the past, it is now clear that some commensal bacteria are present in the uterus. [45] Therefore, the following hypothesis seems more likely where A. vaginae was transported from the patients vagina or cervix into the uterus during CVS and caused an intrauterine infection leading to maternal bacteremia with subsequent sepsis. The intrauterine infection may have caused fetal loss by direct fetal infection or isolated placental infection through the mechanisms described above. In our case clinical improvement occurred only after aspiration curettage was performed, which advocates for the second hypothesis. However, cultures of the evacuated material did not confirm the presence of A. vaginae infection. This may be attributed to antecedent antibiotic treatment.

4. Conclusion

To conclude, A. vaginae bacteremia leading to clinical sepsis is a rare but very severe complication following CVS. Only one other case of sepsis caused by A. vaginae after CVS has been described before, with this case being the second. [38] The incidental occurrence of bacteremia due to A. vaginalis in the literature refutes its identification as a nonpathogenic bacterium. It should be considered as a possible cause of infection in pregnant women presenting with sepsis following transcervical CVS.

Abbreviations

Bacterial vaginosis (BV)
C-reactive protein (CRP)
Chorionic villus sampling (CVS)
Gestational age (GA)
Minimum inhibition concentration (MIC)
Noninvasive prenatal testing (NIPT)
White blood count (WBC)

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