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Case Report

Case of a healthy infant born following antenatal enterovirus myocarditis and hydrops

Aurore Bonnin[a], Mikael Tassin[a], Christelle Vauloup-Fellous[b], Emmanuelle Letamendia[c], Bertrand Stos[d], Damien Bonnet[e], Vincent Gajdos[f], Mylène Mabille[g], Alexandra Benachi[a,*]

[a] Service de Gynécologie-Obstétrique et Médecine de la Reproduction, Hôpital Antoine Béclère, AP-HP, Université Paris Sud, Clamart, France
[b] Service de Virologie, Hôpital Paul Brousse, AP-HP, CNR Infections Rubéoleuses Materno-Fœtales, Université Paris-Sud, UMR-S 785, INSERM U785, Villejuif 94804, France
[c] Service de Néonatologie, Hôpital Antoine Béclère, AP-HP, Université Paris Sud, Clamart, France
[d] Service de Cardiologie, Hôpital Marie Lannelongue, Université Paris Sud, Le Plessis Robinson, France
[e] Service de Cardiologie, Hôpital Necker-Enfants Malades, AP-HP, Université René Descartes, Paris, France
[f] Service de Pédiatre, Hôpital Antoine Béclère, AP-HP, Université Paris Sud, Clamart, France
[g] Service de Radiologie, Hôpital Antoine Béclère, AP-HP, Université Paris Sud, Clamart, France

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ABSTRACT

Fetal hydrops and myocarditis were diagnosed in a woman at 32 weeks of gestation (WG). Transplacental enterovirus infection was suspected because all other causes of myocarditis and hydrops were excluded, it was during an endemic period, and there was a setting of maternal infection (fever a few days before). We opted for in utero treatment because of the risk of resuscitating a neonate with myocarditis and hydrops. We administered dexamethasone 12 mg twice for pulmonary maturation and presumed it would partially improve the myocarditis. Fetal arrhythmia was noted at 35 WG and we decided to deliver the infant as postnatal treatment of the heart disorder would be more effective. RT-PCR (ARGENE®) showed that the neonate's throat and anal tissues and cord blood sampled on the day of birth contained enterovirus ribonucleic acid and coxsackievirus B5, as did the mother's anal sample. Laboratory tests, heart MRI and probably brain MRI indicated neonatal enterovirus infection. Findings were normal at two-year follow-up.

1. Why this case is important

Enteroviruses such as coxsackievirus and echovirus are responsible for a wide range of symptoms in adults: asymptomatic, fever only, myocarditis, encephalopathy. Infection is more prevalent in the summer. Mother-to-child transmission occurs mainly at delivery. Enterovirus infection in neonates can be responsible for serious diseases such as myocarditis, hepatitis, and general sepsis and for death [1]. Transplacental transmission has been described [2–5] but its frequency is unknown. Late term stillbirths associated with maternal enterovirus infections have been reported [6]. Enteroviruses, especially some coxsackieviruses B, are known to have a predilection for myocardial tissues [7]. Hydrops fetalis and arrhythmia were detected antenatally by ultrasonography in a single case of a fetus infected with coxsackievirus B3 [8], but there is no literature report of the birth of a healthy infant following antenatal myocarditis caused by enterovirus infection. We report the case of a healthy boy born following transplacental enterovirus infection, with hydrops and myocarditis, complicated by fetal heart rate anomalies.

2. Case report

A 30-year-old woman, gravida 2 para 2, consulted at 34 weeks of amenorrhea (WA), in July, as an emergency because of 38.2 °C fever at home throughout the previous week. She had previously delivered vaginally a normal term infant following an uneventful pregnancy. Her medical history was unremarkable. No other member of the family was sick.

* Corresponding author at: Department of Obstetrics, Gynecology and Reproductive Medicine, Hospital Antoine Béclère, 92140 Clamart, France.
Tel.: +33 1 45374476; fax: +33 1 45374967.
E-mail address: alexandra.benachi@abc.aphp.fr (A. Benachi).

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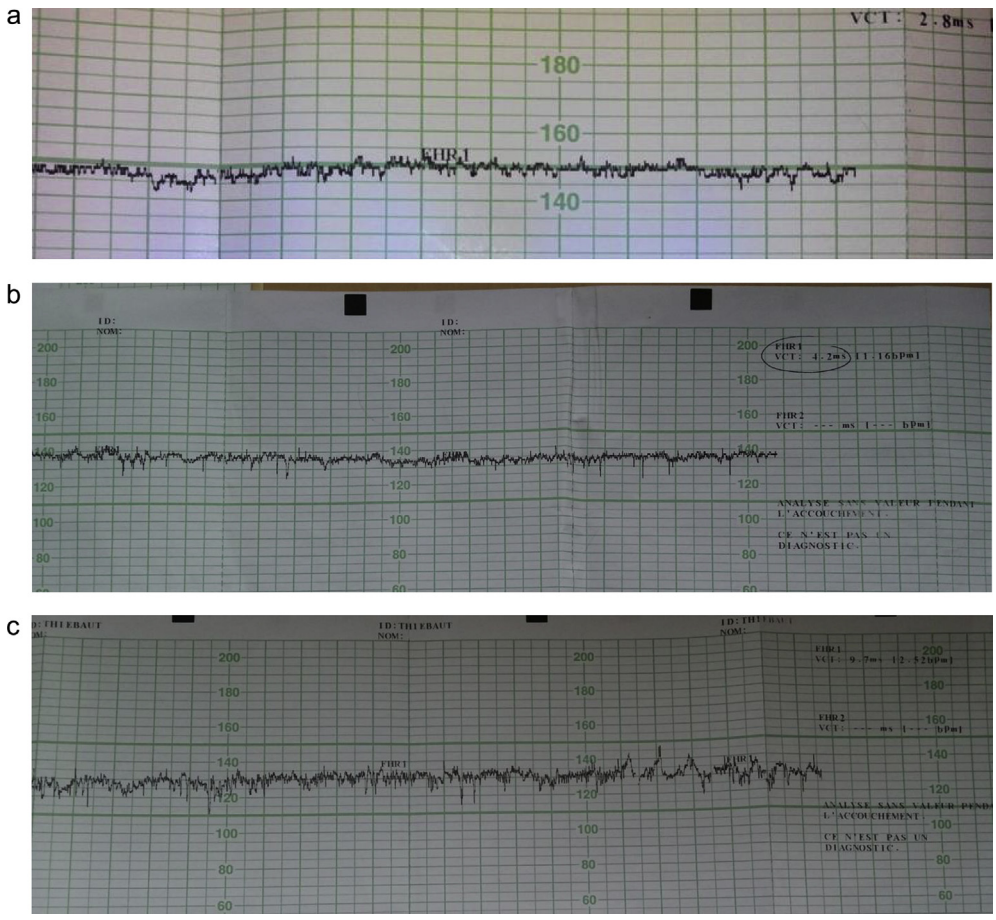

Fig. 1. Fetal hearth rate on STV score. (a) On the 07/12/2012 (9.40 pm) at 34 + 2 WA. STV score = 2.8 ms. (b) On the 07/13/2012 (7.45 pm). STV score: 4.2 ms. (c) On the 07/14/2012 (1.25 pm).

In hospital the patient had no fever and no signs of infection. Fetal movements and fetal heart rate (and oscillation) were normal. C-reactive protein (CRP) was negative and white blood cell count (WBC) normal. Serologies were negative for toxoplasmosis, cytomegalovirus, and parvovirus B19. The patient was discharged with the usual advice.

She returned two days later, at 34^{+2} WA, because of decreased fetal movements. Oscillation amplitude was reduced. The fetus displayed hydrops with ascites, pericardial and pleural effusion and myocarditis. The diagnosis was made on the basis of ventricular septal thickening, decreased contractility and a hyperechogenic appearance of the ventricular walls. There were no calcifications or hepatomegaly, and no brain anomalies.

Maternal–fetal enteroviral infection was suspected because of (i) fetal acute myocarditis, (ii) in the setting of maternal infection. It was decided to pursue the pregnancy in order to avoid birth in the acute period of infection.

Dexamethasone (12 mg × 2) was administered prophylactically to accelerate fetal lung maturity and we hypothesized that it would also improve, at least partially, the myocarditis. The parents were informed that the cause of the fetal pathology was most likely enteroviral myocarditis, which is associated with a risk of in utero death due to arrhythmia. We explained the lack of benefit of giving birth in the context of hydrops and cardiomyopathy, and the neonatal risks. We informed the parents that there is no antenatal treatment for enteroviral infection. Furthermore, we discussed the rare possibility of a metabolic or mitochondrial myocarditis. Metabolic diagnosis using amniotic fluid was not considered as there was little time till term and no antenatal treatment. After being informed, the parents chose to pursue the pregnancy.

During hospitalization the mother remained asymptomatic, with normal CRP and WBC. There were still no fetal movements and short-term fetal heart rate variation (STV) dropped to 2.8 ms (millisecond) but we decided to wait for the dexamethasone to take effect, bearing in mind that it reduces STV and fetal movements (Fig. 1a). After three days, cardiac ultrasound findings had improved and fetal movements were back to normal. STV increased to 5, then 8.5 (Fig. 1b and c). Fetal hydrops was unchanged, but it is well known that whatever the cause it takes time to resolve.

We looked for causes of intrauterine myocarditis and hydrops. Epstein–Barr virus (EBV) serology was IgG-positive but IgM-negative. The coxsackievirus test was negative as was the lupus antibody test. The Kleihauer test was negative.

At 35^{+1} WA, fetal heart rate was difficult to interpret, suggesting a heart rhythm disorder. Ultrasound showed a run of premature atrial beats with stable hydrops. After discussion with pediatric cardiologists and the antenatal team, we decided, given the high risk of severe arrhythmia, to deliver the fetus by cesarean section.

A boy weighing 2380 g was delivered. The Apgar score was 5/6/6/8 and umbilical pH 7.44. He showed incipient circulatory failure. He needed tracheal intubation at 9 min of life. Ventilatory support was stopped at day one.

Heart rate was 129 bpm with premature atrial beats on cardiotocography and bradycardia episodes (80 bpm). Cordarone 90 mg was administered at hour 8. Cardiotocographic findings were normal at day 2. Heart ultrasound at day 1 and day 7 was normal. Cordarone was stopped at day 7.

Laboratory tests suggested infection, with severe thrombopenia (transfusion needed), cytolysis and cholestasis.