CASE REPORT

Resolution of tachyarrhythmia-related fetal hydrops after corticosteroid administration for fetal lung maturation

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SUMMARY

A case of hydrops fetalis (HF) that resolved after corticosteroid therapy despite persisting fetal tachycardia of 190 bpm is reported. The fetus with confirmed normal karyotype had HF in the presence of atrial flutter and sustained ventricular rate of 200–210 bpm at gestational week (GW) 31^{–6/7}. Aetiologies of HF other than fetal tachyarrhythmia were unlikely in this infant. The patient's family declined pharmacological treatment with maternal digoxin and desired to continue pregnancy with only intensive monitoring of fetal condition. Maternal transplacental β -methasone was given to facilitate fetal lung maturation. The fetal tachyarrhythmia ameliorated to 190 bpm at GW 32^{–5/7} and HF gradually disappeared by birth. The otherwise healthy female neonate weighing 3050 g born at GW 37^{–0/7} was successfully treated with cardioversion performed 60 min after birth. This case suggested that corticosteroid therapy contributed to the resolution of HF associated with tachyarrhythmia.

BACKGROUND

Fetal tachycardia constitutes a rare but important cause of non-immune hydrops fetalis (HF), premature delivery and perinatal morbidity and mortality.¹ The American Heart Association states that, "For sustained tachycardia, noted for the majority of the time of evaluation (more than 50%), decisions about treatment depend on and maternal risk analysis with little data to support the specific treatment protocol that is likely to be most effective and to carry the lowest risk."¹ Maternal transplacental administration of antiarrhythmic medication is recommended for all but near-term fetuses with sustained tachycardia with or without HF.^{1 2}

Excessive fluid accumulation within the fetal extravascular compartments and body cavities occurs in HF, approximately 20% of which are caused by cardiovascular abnormalities.^{2 3} Except in cases associated with microorganisms, such as parvovirus B19, it is generally considered that non-immune HF does not resolve spontaneously. We recently encountered a case of tachyarrhythmia and HF in which the HF resolved after transplacental corticosteroid therapy administered for fetal lung maturation. The patient's family gave informed consent for this presentation.

CASE PRESENTATION

A 27-year-old nulliparous Japanese woman in gestational week (GW) 31^{–6/7} was referred to us for

fetal tachycardia of >200 bpm, which was found incidentally at a regular antenatal check-up on the same day as the referral. This female fetus with proven normal karyotype was confirmed to have HF (figure 1) and atrial flutter with 2:1 atrioventricular block on the day of admission (figure 2A, B). The fetal cardiovascular profile score⁴ was estimated as 6 on admission. The HF in this case was unlikely to be due to Rh alloimmunisation, anaemia, structural anomalies or congenital infections, as fetal middle cerebral artery peak systolic velocity was 37.4 cm/s (0.89 multiple of the median (MoM)), serological test results for parvovirus B19, syphilis, *Toxoplasma*, cytomegalovirus, herpes simplex and herpes zoster were negative, and there were no autoimmune antibodies against Sjögren's syndrome-related antigen A (SS-A) or SS-B. The parents declined treatment involving maternal administration of digoxin, perhaps due to excessive concern regarding possible adverse effects of antiarrhythmic agents, and desired to continue pregnancy with only intensive monitoring of fetal condition. Maternal transplacental administration of 12 mg of β -methasone was performed twice at GW 31^{–6/7} and 32^{–0/7} for fetal lung maturation. Daily fetal heart rate (FHR) monitoring (1–2 h/day) was continued, and ultrasound study was performed every day until GW 34^{–6/7}, and three times a week thereafter. The ventricular rate ameliorated to 190–200 bpm at GW 32^{–5/7} (figure 2C) with persisting 2:1 atrioventricular block, but no ventricular rates slower than 190 bpm were seen at any time before birth. Unexpectedly, the HF gradually disappeared (no pleural effusion at GW 34^{–3/7}, no ascites at GW 35^{–6/7}, no pericardial effusion at GW 36^{–5/7} and no skin oedema on the day of birth). An otherwise healthy infant, apart from tachyarrhythmia of 192–202 bpm, was born weighing 3050 g with Apgar scores of 8 (1 min) and 9 (5 min), and umbilical cord blood haemoglobin concentration of 14.7 g/dL, by planned caesarean section at GW 37^{–0/7}. This neonate underwent cardioversion once 60 min after birth, with successful conversion to sinus rhythm with a heart rate of 150–152 bpm (figure 2). She exhibited no recurrence of tachyarrhythmia as determined by ECG monitoring during the period of hospitalisation, and left hospital 8 days after birth. Twenty-four hour Holter monitoring performed at the age of 1 month revealed no arrhythmia. The infant was doing well when last seen at the age of 3 months, showing no symptoms suggestive of recurrence of tachyarrhythmia.

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Figure 1 Ultrasound study on gestational week 31^{-6/7}. (A) Pericardial effusion. (B) Pleural effusion. (C) Subphrenic ascites. (D) Breast oedema.

Figure 2 Fetal heart rate patterns, fetal M-mode finding and postnatal ECG. (A and C) Fetal heart rate tracing on gestational week (GW) 31^{-6/7} and GW 32^{-5/7}, respectively. (B) M-mode imaging of the fetal heart on GW 31^{-6/7}. (D and E) ECG of the neonate before and after cardioversion, respectively. The atrial/ventricular rates were measured from the time interval between two neighbouring atrial (arrows)/ventricular (arrowheads) contractions (B). The fetal atrial rate was 420 bpm, whereas ventricular rate was 210 bpm; a diagnosis of fetal atrial flutter with 2:1 atrioventricular block was made. Postnatal ECG (D) showed atrial flutter with an atrial rate of 384 bpm and 2:1 or 3:1 atrioventricular block. After cardioversion (E) successful conversion to sinus rhythm occurred.

DISCUSSION

Maternal transplacental β -methasone was given to facilitate fetal lung maturation, but no specific treatment for tachyarrhythmia was given in this case. However, this case exhibited resolution of HF. To the best of our knowledge, this is the first report of tachyarrhythmia-related HF that resolved spontaneously despite persistent and sustained tachyarrhythmia with a ventricular rate of ≥ 190 bpm.

The present case had HF exhibiting four types of abnormal fluid collection, namely, pericardial effusion, pleural effusion, ascites and skin oedema (figure 1). Common aetiologies leading to non-immune HF include cardiovascular (such as cardiac structural abnormalities, tachyarrhythmia, supraventricular tachycardia, atrial flutter and atrial fibrillation, accounting for 20% of cases), chromosomal and haematological abnormalities.^{2–3} This case did not have structural, chromosomal or haematological abnormalities. Although not all of the possible aetiological infections were excluded, the postnatal condition did not suggest congenital infection. Among the most common tachyarrhythmias leading to HF are supraventricular tachycardia and atrial flutter,⁵ and atrial flutter was seen in this case. Not all fetuses with supraventricular tachycardia develop HF.^{6–7} In a study dealing with 30 infants with fetal supraventricular tachycardia of >200 bpm and normal cardiac anatomy, including 12 with HF and 23 with 'sustained tachycardia' defined as prolonged uninterrupted tachycardia lasting more than 12 h at initial assessment,⁶ the mean GW at presentation was significantly lower (33 ± 1 vs 38 ± 1 , respectively, $p < 0.001$) and 'sustained tachycardia' was more frequent (100% (12/12) vs 61% (11/18), $p = 0.024$) for HF cases than for non-HF cases.⁶ Therefore, as our case presenting at GW 31 with 'sustained tachycardia' was indeed at higher risk of developing HF, the tachyarrhythmia may have been causally associated with HF in this case.

It is unclear at present whether β -methasone administered for fetal lung maturation favourably affected the clinical course of the present case with respect to the decrease in ventricular rate from 200–210 to 190–200 bpm occurring at GW 32^{–5/7} and the resolution of HF. Spontaneous decrease in FHR can occur in cases of supraventricular tachycardia/flutter.⁷ However, antenatal corticosteroids have a direct, but transient effect on FHR.⁸ Maternal cortisol infusion induces increase in fetal heart size and wall thickness in sheep.⁹ These effects of corticosteroid on haemodynamics may cause a decrease in FHR. Moreover, corticosteroid therapy with β -methasone was reported to be effective for treatment of HF associated with congenital pulmonary airway malformation (CPAM)^{10–11} and is recommended for the treatment of HF associated with CPAM.² These observations together with the clinical course of our case raised the possibility that corticosteroid therapy contributed to the resolution of HF in the present case with tachyarrhythmia.

In conclusion, a case of tachyarrhythmia-induced HF resolving after corticosteroid therapy administered for fetal lung

maturation was reported. This case suggested that corticosteroid therapy may be effective for resolution of HF associated with fetal tachyarrhythmia. Although further clinical studies are required, corticosteroid may alleviate fetal and maternal allostatic overload, and diminish the stress reaction resulting from fetal heart failure.

Learning points

- It is not yet known whether transplacental corticosteroid therapy is effective for hydrops fetalis associated with tachyarrhythmia.
- We report a case of tachyarrhythmia-related hydrops fetalis that resolved after corticosteroid therapy administered for fetal lung maturation.
- It is possible that corticosteroid therapy contributed to the resolution of hydrops fetalis in this case.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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