Fetal Atrial Flutter Associated With Atrial Septal Aneurysm

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Research Article

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Abstract

Objective: To provide evidence that fetal atrial flutter (AF) caused by atrial septal aneurysm (ASA) can be completely cured by delivery.

Cases: Three fetuses with ASA complicated by AF in late gestation, including hydrops fetalis in one case, were collected and completely followed up. AF in all cases completely disappeared shortly after birth. New insights gained from this study are as follows: 1) PACs or bigeminy associated with ASA can progressively change to AF. 2) AF associated with ASA can cause hydrops fetalis and intrauterine treatment is needed but delivery is definitive treatment. 3) AF associated with ASA completely resolves after birth.

Conclusion: This report may have clinical impact because of providing evidence that 1) In case of AF associated with ASA, the prognosis is much better than other causes and delivery should be strongly considered. 2) Fetuses diagnosed with AF should always be looked for the presence of ASA. 3) PAC/bigeminy related to ASA, different from isolated PAC, needs close follow-up for development of SVT and AF. 4) The fetuses remote from term can benefit from intrauterine treatment to avoid hydrops fetalis, and to prolong gestation for maturity and early delivery is recommended once lung maturity is confirmed.

Introduction

Atrial septal aneurysm (ASA) in newborns, defined as an excursion ratio (maximal excursion of the septum primum to left atrial diameter ratio) of 25% or greater, is common in newborns, possibly as many as 7.6% ⁽¹⁾, and all cases disappear at the end of the first year of life, mostly in one month, without any complications related to the lesion during the follow-up period ⁽¹⁾. Thus, ASA can be considered that it is benign and transient observation. Nevertheless, ASA associated with dysrhythmias in newborns or adults has been recognized ^(2,3). ASA in the fetuses should be defined differently, since an excursion ratio of 25% can be a physiologic movement caused by blood flow crossing the foramen ovale. In fetuses, the cut-off point for diagnosis of excessive excursion is defined as greater than 1.3 MoM or the excursion ratio of greater than 55% ⁽⁴⁾. ASA associated with premature atrial contraction (PAC) has been reported several times ^(5,6). However, atrial flutter (AF) secondary to ASA in fetuses is rarely described prenatally. To the best of our knowledge, fetal ASA associated with AF has never been reported. The objective of this study is to describe the cases of fetal ASA complicated by AF in late gestation, including hydrops fetalis in one case, with complete disappearance shortly after birth, to underline clinical impact of AF secondary to ASA and to emphasize on the close follow-up the fetuses with PAC associated with ASA.

Case Presentation

The report was ethically approved by the institutional review board (Faculty of Medicine, Chiang Mai University) and written informed consent has been obtained from the patient(s) to publish this paper.

Case 1

A 33-year-old pregnant woman, G2 P1001, attended the scheduled antenatal care at 35 weeks of gestation at a community hospital. On physical examination, fetal tachycardia with heart rate greater than 200 bpm was detected. The pregnancy was referred to our center for further management. Her history of prenatal course was uneventful. Fetal anomaly screening at 20 weeks of gestation showed normal and chromosome study result was low-risk for aneuploidy. At our center, at 35 weeks, detailed ultrasound showed a normally formed female fetus with normal growth and fetal biometry, occasional PAC, alternate with occasional atrial flutter (AF) (Fig. 1A, 1B) and with normal rhythm. However, the fetal heart had atrial septal aneurysm (ASA), with abnormally wide excursion of the foramen ovale flap (approximately 80% of left atrial diameter: Fig. 1C) with turbulent reversed flow in the septum back to the right atrium (Fig. 1D), instead of crossing the foramen. The patient was admitted for close follow-up with ultrasound and expectantly treated. The dysrhythmias were more progressive, turning to be persistent AF. During surveillance, at 36⁺⁵ weeks ultrasound showed fetal atrial rate was 505 bpm and ventricular rate was 245 bpm. AF with 2:1 AV block was diagnosed. AF was persistent after observation over a period of four hours. The cardiac size was within normal limit. No hydropic sign was detected. The last fetal heart rate 2 weeks before this admission was normal (150 bpm), recorded by Doppler device.

Because the AF was persistent, without alternate normal rhythm, early treatment was needed. Since the gestational age was nearly term, rather than in utero treatment, cesarean delivery was performed for early treatment after birth. The female newborn had birthweight of 2,750 g, no gross structural anomaly, and Apgar score 10 at 5 min. Just immediately after birth, the heart rate was firstly detected to be approximately 240 bpm. Ten minutes later, the heart rate was suddenly reduced to 124 bpm, no longer tachycardia. Neonatal echocardiography, the heart was otherwise normal except the small size of atrial septal aneurysm with minimal redundant the atrial septum, bulging predominantly into the right atrium, no significant turbulent flow in the septum as previously shown in utero. The AF completely disappeared without recurrence.

Case 2

A 20-year-old pregnant woman, G0 P0, attended routine antenatal care at 33⁺⁴ weeks of gestation at a private hospital. On physical examination, fetal tachycardia with heart rate of approximately 230 bpm was detected. The pregnancy was referred to our center because of fetal tachycardia. The pregnancy was low-risk and prenatal course was unremarkable. Fetal anomaly screening and fetal biometry at 21 weeks of gestation revealed normal. Fetal Down syndrome screening was low-risk. At our center, fetal heart rate was sometime irregular and sometime very fast (approximately 240 bpm). The detailed ultrasound examination at 33⁺⁴ weeks revealed a normally formed male fetus with proper growth. Nevertheless, the fetal tachycardia was the fetal heart had tortious ductus arteriosus and ASA (abnormally wide excursion of the foramen ovale flap: approximately 80% of left atrial diameter) with turbulent reversed flow in the septum, circulating back to the right atrium. The fetal atrial rate was varied, occasional bigeminy of PAC with conduction with the rate of about 150 bpm (Fig. 2a), occasional normal rate of 140–150 bpm, but

mostly tachycardia with atrial rate of 480 bpm and ventricular rate was 240 bpm (Fig. 2b). AF with 2:1 AV block was diagnosed and the AF took most of the time. Because AF was not persistent and no any hydropic signs, other than mild cardiomegaly (cardio-thoracic diameter ratio of 65%), expectant management with close follow-up was given. Fetal well-being was assessed by biophysical profile. At 36 weeks of gestation AF was persistent. We decided to have cesarean delivery for postnatal treatment, giving birth to a 2,540-g male newborn, Apgar scores 10 at 5 min. Just immediately after birth, the heart rate was regular, 120 bpm and no longer tachycardia. Neonatal echocardiography revealed normal cardiac structures except the atrial septal aneurysm with the redundant atrial septum, no flow crossing the foramen ovale. The AF and hydropic signs completely disappeared without any treatment.

Case 3

A 25-year-old pregnant woman, G2 P1001, attended our antenatal care at 37⁺⁴ weeks of gestation with incidental findings atrial septal aneurysm with fetal tachycardia, the heart rate of approximately 220 bpm. She attended her first antenatal visit at 12⁺⁴ weeks of gestation. The antenatal course was low-risk and uneventful. Fetal anomaly screening at 20⁺⁵ weeks of gestation was unremarkable. The second trimester serum screening was low-risk. At 33⁺⁴ weeks of gestation the fetus developed occasional PACs with conduction and also ASA was firstly noted, with the maximal excursion of about 80% of the left atrial width. Fetal heart rate was relatively normal, though sometime irregular. Expectant management and close follow-up as out-patient care was provided. At 34⁺⁴ weeks, the fetus developed persistent SVT, atrial rate to ventricular rate ratio of 1:1 (220 bpm) (Fig. 3a). Also, hydropic signs including pleural effusion and ascites were observed. Because SVT was persistent during preterm period, intrauterine treatment with flecainide via maternal circulation was instituted. Drug administration and dosage flecainide of dosage described elsewhere ⁽⁷⁾. Approximately 24 hours after treatment, cardioversion to normal sinus rhythm was successful. However, four days after the initiation of intrauterine treatment, AF developed in spite of intrauterine treatment (Fig. 3b). Then the combination of digoxin (0.5 mg/d) and flecainide were administered instead. Nevertheless, cardioversion was not successful. We decided to have delivery for early postnatal treatment. Cesarean delivery was performed at 35⁺⁶ weeks of gestation, giving birth to a normally-formed male newborn, birthweight of 2,550 g., and Apgar score of 9 at 5 min. The neonatal heart (ventricular) rate at the delivery room was 240 bpm. Shortly after that, at the NICU, the heart rate was suddenly lowered to 130 bpm, no need of any anti-arrhythmic medication. Neonatal echocardiography showed a markedly decrease in the maximal excursion of the foramen ovale flap, no significant turbulent flow in the septum as previously shown in utero. There was a trivial low velocity left to right shunt across the patent foramen ovale. The AF and hydropic signs completely disappeared without recurrence.

The summary of characteristics of the three cases with AF associated with ASA is presented in Table 1.

Table 1
Characteristics of the three cases with atrial flutter associated with septal aneurysms

Case no.	Maternal age	GA of diagnosis	Intrauterine treatment	GA of delivery	Hydrops fetalis	Preceding arrhythmia	Birth weight
1	33	35	No	36 ⁺⁵	No	Bigeminy	2750
2	20	33 ⁺⁴	No	36	No	PACs, Bigeminy	2540
3	25	34 ⁺⁴	Yes	35 ⁺⁶	Yes	PACs, SVT	2550

Discussion

New insights gained from this study are as follows: 1) Though PACs or bigeminy are usually benign, those associated with ASA have a tendency to subsequently develop SVT or AF. Hypothetically, the dysrhythmias may be activated by mechanical stimulation of turbulent flow produced by ASA. 2) AF associated with ASA has a tendency to develop in late pregnancy. 3) AF associated with ASA can cause hydrops fetalis and intrauterine treatment is needed, especially in case of remote from term, to prolong gestation for maturity. However, delivery is definitive treatment. 4) AF associated with ASA has a good prognosis, usually resolves shortly after birth. This is probably associated with changes of circulation after birth, no more blood flow across the foramen ovale, no turbulent flow in the septum with reversal to hit the right atrial wall.

AF is a serious disorder for the fetus in utero and newborns, probably leading to heart failure, hydrops fetalis or fetal death. However, the prognosis of the disorder may depend on the etiology. We have encountered three cases of AF with good prognosis, which completely resolved by delivery and all of them had septal aneurysms. Thus, it is reasonable to assume that AF is likely caused by septal aneurysm, since after delivery resulting in dramatic change in fetal to neonatal hemodynamics with functional close of foramen ovale or no more blood flow crossing the foramen. In other words, delivery gets rid of turbulent reversed flow, which could interfere with atrial ectopic pacemakers, leading to disappearance of AF.

Of interest, though the number of cases is very small, the three cases strongly suggested that septal aneurysm is likely associated with SA node dysfunction or activation of an ectopic foci in the right atrial wall. We hypothesize that the turbulent flow in the aneurysms, circulating back to the right atrium instead of crossing the foramen ovale to the left atrium, may activate the ectopic pacemaker foci to initiate dysrhythmias. Fetal ASA may be associated with autonomic dysfunction, as reported in adult patients ⁽⁸⁾. However, the natural course of AF secondary to excessive septal excursion in the fetus can be reversed by delivery, different from the persistent form seen in adult. Because delivery gets rid of the abnormal flow which is definitive treatment, the prognosis of AF associated with ASA is very good.

Hemodynamics of the fetal life is much different from that of a newborn. In fetal life, most blood from the ductus venosus enters the inferior vena cava and freely across the foramen secundum and foramen primum of the interatrial septum to the left atrium. Nevertheless, after birth this channel is functionally closed because neonatal breathing causes increased lung perfusion and pulmonary venous return resulting in an increase in pressure in the left atrium, producing back pressure on the foramen ovale flap to fuse with the septum secundum. We hypothesize that this functional change, which certainly causes no longer blood flow crossing foramen ovale and no turbulent flow in the ASA, might resume the function of SA node pacemaker or get rid of the activation of atrial ectopic foci. Actually, in our cases ASA were still present just after birth, but the sizes were much dramatically reduced or became just only a redundant atrial septum, back-and-forth movement between both atria.

Clinical impact

We provide evidence that fetuses diagnosed with AF should always be carefully looked for the presence of ASA. Different from isolated PAC or bigeminy, PAC or bigeminy associated with ASA needs close follow-up for development of SVT and AF. In case of atrial flutter associated with ASA, the prognosis is much better than other causes and delivery should be strongly considered. The fetuses remote from term can benefit from intrauterine treatment to avoid hydrops fetalis, and to prolong gestation for maturity and early delivery is recommended once lung maturity is confirmed.

In conclusion

AF can be caused by ASA and has a tendency to develop in late pregnancy. It may be preceded by PAC or bigeminy or supraventricular tachycardia. Hypothetically, turbulent flow in the aneurysms returning to the right atrium may mechanically activate ectopic pacemaker foci or SA node dysfunction. Different from most cases of AF, spontaneous disappearance can be expected by delivery. However, untreated AF associated with ASA can cause hydrops fetalis and intrauterine treatment should be instituted.

Declarations

Ethical approval and informed consent statement: The report was ethically approved by the institutional review board (Faculty of Medicine, Chiang Mai University) and written informed consent was obtained from the patient(s) to publish this paper.

Data Availability Statement: The data of this report are available from the corresponding authors upon request.

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Conflict of Interest: none declared

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Figures

Figure 1

A) M-mode shows ventricular rate (v) 245 bpm and atrial rate (a) 490 bpm; B) Doppler waveforms of renal vein (a-rate: 245 bpm) and renal artery (v-rate: 505 bpm); C: Atrial septal aneurysm (*) : wide excursion of the foramen ovale flap; D: Color flow mapping in the septal aneurysm shows turbulent reversed flow returning back to the right atrium (FFo: flap of foramen ovale; LA: left atrium; RA: right atrium)

Figure 2

A) Umbilical artery waveforms show premature atrial contraction / bigeminy (a): B) M-mode shows atrial flutter; atrial rate (a) : ventricular rate (v) 2:1

Figure 3

A) M-mode shows supraventricular tachycardia; atrial rate (a) : ventricular rate (v) (A: aortic; LA: left atrium; RA: right atrium); B) Cardiac Doppler waveforms at left inflow and outflow shows atrial flutter; atrial rate (a) : ventricular rate (v) 2:1 (440:226)