Original articles

Prevalence and prognosis of atrial septal aneurysm in high risk fetuses without structural heart defects

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Background. **The aim of this study was to evaluate the prevalence and prognostic implications of the association between atrial septal aneurysm (ASA) and fetal arrhythmias in a population of high risk fetuses.**

Methods. **One thousand three hundred and two fetal echocardiograms performed during high risk pregnancies from the 17th to the 41st week of gestation were retrospectively evaluated for the presence of ASA and/or arrhythmias. An ASA was defined as redundant tissue extending at least halfway across the left atrium. Patients with an ASA were distinguished in two subgroups according to whether there was (subgroup 1) or was not (subgroup 2) cyclical contact of the atrial septum with the left atrial wall or with the mitral valve. Arrhythmias were documented during mono/two-dimensional echocardiography and Doppler evaluation.**

Results. **Out of 1223 patients considered for the study, 93 (7.6%) fetuses had an ASA; among these 93 fetuses, 33 (36%) had premature atrial beats ($p < 0.001$). Ten of these patients were included in subgroup 1, and 7 of them (70%) exhibited premature atrial beats ($p = 0.016$ vs subgroup 2). No arrhythmias other than premature atrial beats were observed in these patients. None of them received any therapy during observation. All of them developed a regular sinus rhythm within 3 months of life.**

Conclusions. **On the basis of these data, we can speculate that, if accurately searched for, ASA is often present (7.6%) and is likely to represent a mechanical stimulus for the generation of premature atrial beats. Indeed, our data show an important correlation between the degree of bulging and the presence of arrhythmias, supporting the hypothesis of a mechanical stimulus. However, the observed arrhythmias did not appear to be prone to degeneration. In conclusion, ASA observed during fetal life is often associated with premature atrial beats, which are apparently in direct relation with the degree of bulging of the atrial septum. On the other hand, an ASA almost invariably disappears at birth and is not associated with major arrhythmias.**

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Introduction

Fetal atrial septal aneurysm (ASA) and arrhythmias should be detected during the first level echographic evaluation in pregnant women[1-5]. Although fetal atrial arrhythmias without congenital heart disease occur in 1 to 2% of all pregnancies, their etiology has not yet been well established[1]. Previous reports had shown a significant association between ASA and fetal atrial arrhythmias[1-4]. Nevertheless, the significance of a potential mechanical stimulus by a redundant septum primum flap is still to be determined[1,2].

The aim of this study was to evaluate the relationship between an ASA and arrhythmias and to determine its significance in a group of fetuses referred for evaluation to our fetal-pediatric echocardiography laboratory.

Methods

Study patients. Between January 1996 and May 2001, 1302 fetal echocardiograms were performed in our Institute in high risk pregnancies. We retrospectively searched for the presence of ASA and/or arrhythmias. The gestational age of the fetuses ranged from 17 to 41 weeks. Fifty-five fetuses had congenital heart disease and were excluded from the study. Twenty-one others were excluded because, owing to the position of the fetus or to thickness of maternal abdominal wall, the atrial anatomy could not be adequately evaluated. Nevertheless none of them had arrhythmias. Three fetuses were lost at follow-up, after the 25th week of pregnancy and were therefore excluded from the study. None of them showed an ASA or arrhythmias at the time of evaluation. All patients with an ASA

and/or arrhythmias underwent ECG and echocardiographic evaluation at 1 and 3 months of age.

According to whether patients were referred for a high risk pregnancy (Table I) or for arrhythmias, the study population was divided in group 1 and 2, respectively.

Table I. High risk pregnancies for congenital heart disease.

Family history
Extracardiac anomaly
Maternal diabetes
Maternal teratogen
Suspicious level 1 scan
Aneuploidy
Hydrops

Echocardiography. M-mode, two-dimensional, pulsed and color Doppler echocardiography studies were performed with a Hewlett-Packard (Andover, MA, USA) Sonos 2500 or 5500 with 3.5, 5 MHz or S8 transducers and recorded on VHS.

Definitions. The criteria adopted for the definition of a high risk pregnancy for congenital heart disease are listed in table I[6,7].

An ASA was defined as redundant tissue extending at least halfway across the left atrium[2] (Figs. 1 and 2). Patients with evidence of a septum primum flap, which appears smaller and less mobile[2], or with bulging of the entire atrial septum[8] were considered as having a normal heart morphology.

Patients with an ASA were distinguished in two subgroups according to whether there was (subgroup 1) or was not (subgroup 2) cyclical contact with the left atrial wall or with the mitral valve.

Arrhythmias were documented during mono, two-dimensional echocardiography and Doppler evaluation lasting an average of 47 ± 14 min (range 30 to 60 min)[3]. Atrial ectopic activity was considered significant when it ranged from atrial bigeminy to two ectopic beats during observation[2]. Supraventricular tachycardia was defined by a 1:1 atrioventricular conduction with a fetal heart rate > 180 b/min. Supraventricular tachycardia was considered sustained when observed for the whole echocardiographic examination time and intermittent when lasting < 50% of the examination time[9]. Second or third degree atrioventricular block was diagnosed when the atrial contraction was not followed by a ventricular contraction or when the atrial contraction was irregularly related to the ventricular contraction.

Statistical analysis. The statistical correlation between an ASA and atrial arrhythmias was analyzed by means of the χ^2 method. A p value of < 0.05 was considered statistically significant.

Results

Detailed results are shown in figure 3. Out of 1223 patients considered for the study, 93 (7.6%) fetuses had an ASA; among the latter, 33 (36%) showed premature atrial beats ($p < 0.001$). Ten of these patients were included in subgroup 1, and 7 of them (70%) exhibited premature atrial beats ($p = 0.016$ vs subgroup 2). No arrhythmias other than premature atrial beats were observed in these patients.

Seventy-nine fetuses (6.4% of the total study population) were referred for arrhythmias (group 2) but only in 36 of them (45.5%) could the presence of arrhythmias be confirmed. Nineteen fetuses (52.8%) had premature atrial beats but no ASA (Fig. 3); 14 out of 36

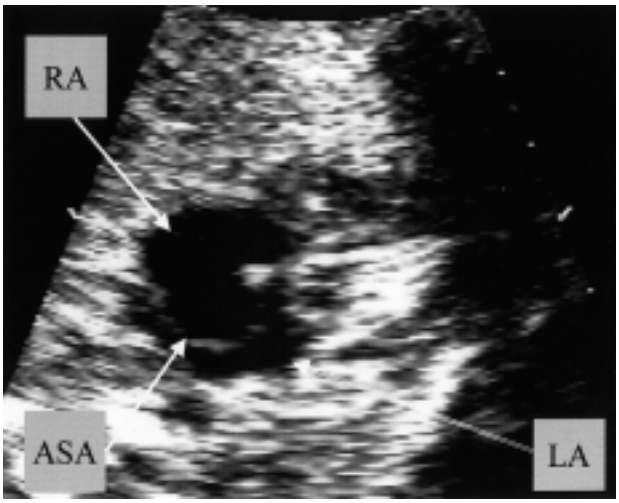

Figure 1. *Two-dimensional echocardiography in the short-axis view of the atria of a fetus with an atrial septal aneurysm (ASA) bulging* (white arrows) *into the left atrium (LA). RA = right atrium.*

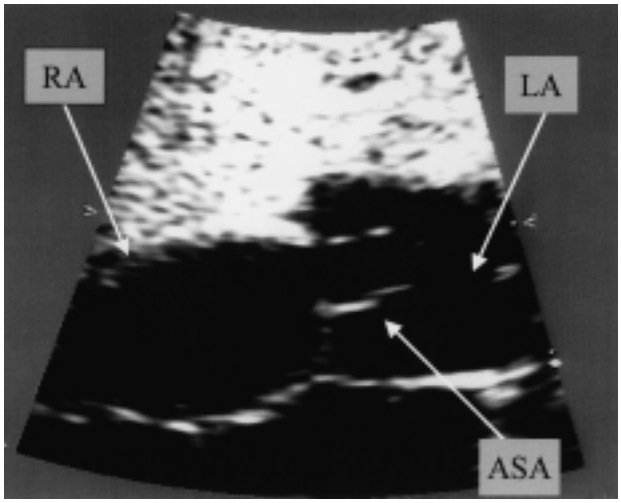

Figure 2. *Two-dimensional echocardiography in the 4-chamber view of the atria of a fetus with an atrial septal aneurysm bulging* (white arrows) *into the left atrium. Abbreviations as in figure 1.*

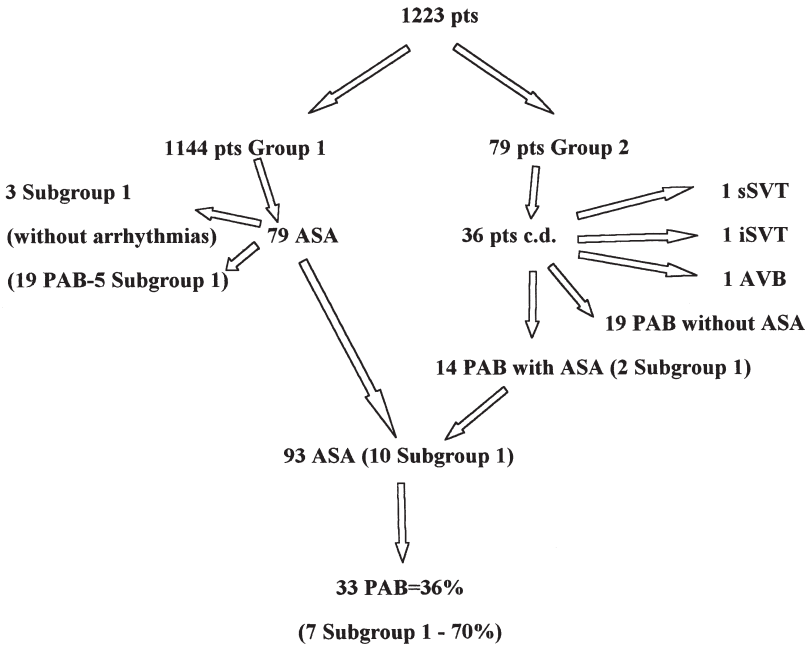

Figure 3. *Schematic representation of the study results. ASA = atrial septal aneurysm; AVB = atrioventricular block; c.d. = confirmed diagnosis; iSVT = intermittent supraventricular tachycardia; PAB = premature atrial beats; sSVT = sustained supraventricular tachycardia.*

(38.9%) had an ASA associated with premature atrial beats (2 of them included in subgroup 1). The remaining 3 fetuses, all without an ASA, presented with sustained supraventricular tachycardia, intermittent supraventricular tachycardia, and complete atrioventricular block, respectively.

All group 1 fetuses developed sinus rhythm within term. Of the 33 patients with premature atrial beats, referred for arrhythmias (group 2), 29 (11 with an ASA) showed sinus rhythm within term; 3 patients with an ASA (one of them in subgroup 1) and 1 without an ASA, presented with premature atrial beats until the first week of life. However, 24-hour Holter monitoring did not confirm the presence of arrhythmias at 3-month follow-up. None of them received any therapy during observation. In all subjects, the ASA disappeared within the first month after birth.

Discussion

An association between fetal atrial arrhythmias and an ASA has been previously reported[1,2,4]. In our series, 35.5% of fetuses with an ASA were affected by arrhythmias confirming a significant association between the two conditions ($p < 0.001$). Nevertheless, in these fetuses we observed only supraventricular ectopic beats. None of them received any therapy during observation. In most of them the premature atrial beats disappeared during pregnancy; in all, within 3 months of birth.

The disappearance of the ASA at birth, most likely depending on the increase in the left atrial pressure to values higher than those in the right atrium with a consequent compression of the aneurysm into the atrial septum, probably determines the development of a normal cardiac rhythm. The observations that most fetal atrial arrhythmias resolve within a few months of birth, either when the obligatory right-to-left shunt through the foramen ovale disappears or after surgical excision of the ASA[10,11], further support the latter hypothesis. Indeed, we can speculate that an ASA may constitute a mechanical stimulus for the generation of premature atrial beats which, however, do not appear to be prone to degeneration.

Previous reports have surmised an abnormally weakened septal tissue in the presence of altered atrial hemodynamics that could synergistically contribute to the formation of an ASA and arrhythmias[8]. Alternatively, arrhythmias could be related to the contact between redundant atrial tissue and the left free atrial wall[2,4]. Indeed, our data show an important correlation between the degree of bulging and the presence of arrhythmias. In fact, premature atrial beats were present in 70% of patients with a higher degree ASA, as highlighted by the cyclical contact of the atrial septum with the left atrial wall or with the mitral valve, supporting the hypothesis of a mechanical stimulus for the generation of arrhythmias.

In our series of patients, major arrhythmias were observed only in 3 cases all without an ASA: one with sustained supraventricular tachycardia, one with intermittent supraventricular tachycardia, and the last with complete atrioventricular block. Therefore, in our series, the presence of an ASA was not able to predict the only cases in which major arrhythmias developed.

In conclusion, an ASA observed during fetal life is often associated with premature atrial beats the severity of which appears to be directly related to the degree of bulging of the atrial septum. On the other hand, an ASA almost invariably disappears at birth and is not associated with major arrhythmias.

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