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Pregnancy Issues in Women with Atrial Septal Defect

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1. Introduction

Success of surgical repair of congenital heart defects in the past five decades has enabled survival into adulthood. Women born with congenital heart diseases reach child-bearing age with or without surgical repair of those lesions. Specifically, atrial septal defect (ASD) is a non-lethal, acyanotic lesion in which survival into adulthood with or without surgery is common place. Types of ASD and hemodynamics have been discussed elsewhere in this book. Briefly, ASD causes left to right shunting leading to right atrial and right ventricular enlargement and increased pulmonary blood flow. Heart failure is uncommon before 4th decade. Mild pulmonary arterial hypertension may occur with advancing age. However, Eisenmenger's syndrome is rare.

Presence of ASD may be diagnosed for the first time during adulthood – probably during pregnancy when an asymptomatic murmur is evaluated using echocardiography. Pregnancy causes cardiovascular changes due to fetal demand for oxygen and nutrition and due to effect of maternal hormones on blood volume and hematocrit. In this chapter, effects of these cardiovascular changes on ASD hemodynamics and effect of ASD on the pregnancy will be discussed. Management strategies will be reviewed.

2. Physiologic changes during pregnancy

2.1 Cardiovascular changes

Physiologic demands of pregnancy lead to significant changes in cardiovascular system during pregnancy, labor and postpartum (Strong et al. 1992). Cardiac output increases constantly in the first 30 weeks of pregnancy reaching ~140% of the pre-gestational level. After 30 weeks, the increase is minimal. Initially, stroke volume increases more than the heart rate. In later part of pregnancy, heart rate increases. There is a 10-fold increase in blood flow to placenta and uterus during pregnancy. Since placenta offers very low vascular resistance, maternal systemic vascular resistance decreases. A reduction in left ventricular afterload occurs (Metcalfe & Ueland 1974). A corresponding increase in cardiac output occurs during the same period, keeping blood pressure stable in spite of the reduction in afterload.

Overall, circulatory changes can be summarized as follows: Increase in stroke volume by 18-25% with heart rate increase by 20%. Net effect of this is a 30-50% increase in cardiac output.

There is increased extraction of oxygen by the placenta leading to increase in arterio-venous difference of oxygen content. Both systemic and pulmonary vascular resistance decrease. However, the reduction in systemic vascular resistance is higher leading to decrease in left to right shunting across ASD. Systemic blood pressure shows a reduction in both systolic and diastolic pressure with a higher reduction in diastolic pressure, thus leading to a wide pulse pressure. (Perloff, JK et al.1992).

2.2 Respiratory changes

Concurrent to cardiovascular changes, changes occur in the respiratory system as well. There is a 45% increase in minute ventilation and 20% increase in oxygen consumption. There is a mild increase in respiratory rate. Functional residual capacity increases by approximately 40% (Perloff, JK et al. 1992).

2.3 Hematologic changes

Due to hormonal changes, blood volume increases to about 150% of the pre-gestational value (Figure 1). This is accomplished by increase in plasma volume out of proportion to the increase in red cell mass. Therefore, "physiologic" anemia occurs which in turn imposes a hyperdynamic circulatory state (Figure 1).

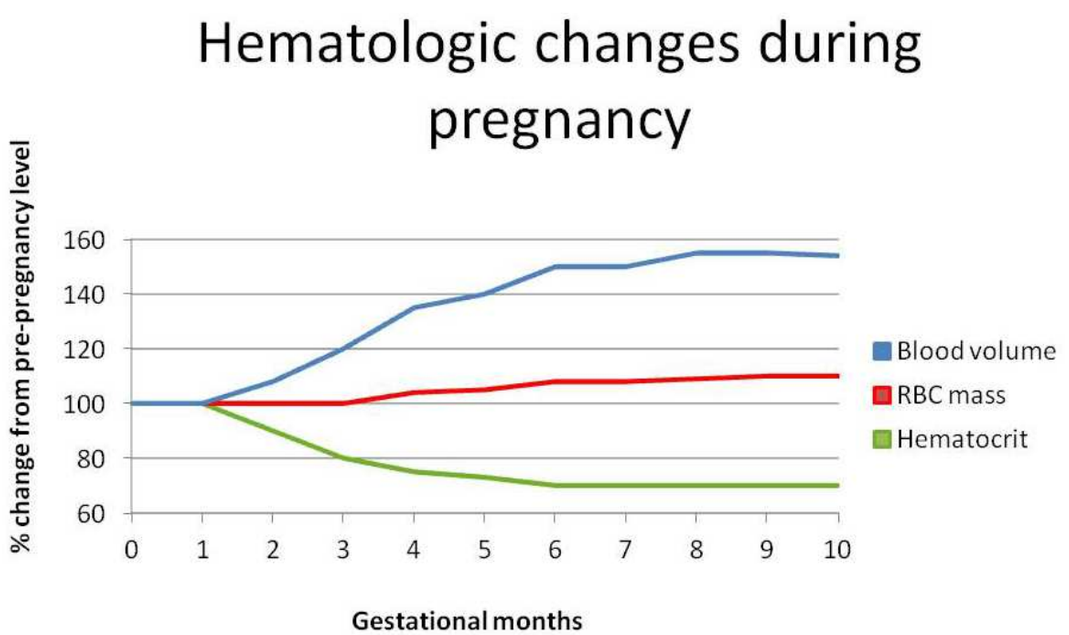

Fig. 1. Hematologic changes during pregnancy. Changes in blood volume, red cell mass (RBC mass) and hematocrit occur during pregnancy. RBC mass increases only approximately 110% while blood volume increases by approximately 150% resulting in "physiologic" anemia of pregnancy. (Figure adapted from Strong et al. 1992).

Greatest demand on the cardiovascular system occurs after 30 weeks of gestation. This is usually tolerated well in woman with normal heart or mild heart disease before conception (New York Heart Association Functional Class I and II) because the demand increases gradually. Increasing fetal demand of oxygen is met with increasing placental blood flow and slight increase in maternal hyperventilation in late pregnancy (Pitkin et al. 1990).

2.4 Changes in last trimester and labor

During the last trimester, enlarging uterus compresses the inferior vena cava (IVC) especially in supine posture resulting in two effects. One, decrease in venous return leading to decrease in cardiac output in supine posture. Second, predisposition to deep vein thrombosis in lower part of the body by creating sluggish circulation. (Metcalfe 1978).

During labor, significant changes occur in a short period of time. In first stage of labor, uterine contractions lead to compression of IVC and decrease in venous capacitances and increase in systemic vascular resistance. This leads to reduction in cardiac output during uterine contractions. However, when the uterus relaxes the cardiac output increases back to pre-contraction levels. In second stage of labor, cardiac output increases significantly due to vigorous expulsive effort. (Ueland & Metcalfe 1975).

After delivery, removal of placenta leads to increase in systemic vascular resistance. Blood loss during labor is a stress on the heart. Equally important is the fact that several hormonal stimuli that occur during pregnancy are receding during postpartum, leading to return of blood volume, hematocrit and cardiac output to pre-conception levels.

3. Effect of pregnancy on ASD

Hemodynamic changes outlined above will affect the hemodynamics of ASD. In a study of 54 pregnant women with ASD, an increase in right atrial and right ventricular size was found, more than the control group of pregnant women who did not have any heart defect. Indirect parameters of right ventricular strain were worse and estimated proportion of pulmonary blood flow to systemic blood flow was lower. There was a higher incidence of supraventricular tachycardia during pregnancy in women with ASD. (Peisiewicz et al. 2004).

Higher incidence of thromboembolism during pregnancy due to venous stasis in the lower part of the body and hypercoagulable state due to high progesterone level was detected. Increased respiratory effort during pregnancy from hemodynamic changes and anemia, and straining during parturition increase the chances of paradoxical embolism. These risks have been reported in several cases in the literature and raise the issue of whether closure of ASD during pregnancy is indicated.

4. Effect of ASD on pregnancy

Asymptomatic ASD without functional compromise (NYHA Class I and II) usually have no effect on pregnancy. However, if there is pre-existing heart failure, there is decrease in

uterine blood flow leading to compromised fetal growth (reflected in incidence of small for gestation age babies) and viability. In a review that consolidated outcome of 123 pregnancies in women with ASD, 1 arrhytmic event, no heart failure events, 2 cardiovascular events and 1 endocarditis were reported. In this study, incidence of these reported events were less than what is expected for healthy women. Similarly, incidence of adverse neonatal events was also low including small for premature birth, small for gestational age and neonatal mortality (Drenthem et al. 2007).

5. Risk of inheritance of ASD in the offspring

If there is a recognizable genetic syndrome in the mother associated with ASD, the inheritance pattern of such syndrome will dictate the risk of congenital heart disease in the fetus. In the absence of such genetic syndrome in the mother, incidence of congenital heart disease is 2-4% if mother has congenital heart disease which is higher than 0.6 – 0.8% incidence in general population. In a Danish study of 1.7 million persons, a recurrence risk of 7.1 in first-degree relatives of individuals with congenital heart disease. (Oyen et al. 2009). Another European study from Netherland, reported an occurrence of ASD in 2.4% of the offspring in a cohort of 291 pregnancies in women with ASD. (Drenthen et al. 2007)

6. Management of pregnancy with ASD

Management pregnancy in women with ASD depends on whether the ASD was diagnosed prior to pregnancy, size of ASD and pre-existing cardiac issues related to ASD such as chamber enlargement, heart failure, arrhythmia and pulmonary artery hypertension. In general, uncomplicated ASD need not be treated during pregnancy.

6.1 Diagnosis known before pregnancy

If the diagnosis of ASD were known prior to pregnancy and ASD is large associated with hemodynamically-significant shunt, associated with moderate or severe chamber enlargement, a potential for supraventricular tachycardia and thromboembolic events during pregnancy, labor or postpartum. Therefore, the patient should have ASD closed prior to planned-pregnancy. Current practice is to electively close asymptomatic, but significant size ASDs prior to child-bearing years. Transcatheter or surgical technique should be applied based on the location and size of ASD and presence of adequate rim of tissue around the ASD suitable for transcatheter device placement. If unsuitable for transcatheter closure, surgical repair is sought.

6.2 ASD diagnosed during pregnancy

6.2.1 Medical management

ASD does not always require surgical or transcatheter closure. If the pregnant woman is asymptomatic (NYHA Functional Class I and II) without heart failure, atrial arrhythmia or pulmonary hypertension or history of stroke, an expectant management of ASD during pregnancy is acceptable. However, if any of the above stated risk factors exist, ASD may

need to be closed during pregnancy. Indications for intervention (surgical or transcatheter options) include severe hemodynamic compromise, NYHA class > II, recurrent stroke prior to or during pregnancy, etc. This usually constitutes a small number of patients. Medical treatment of arrhythmia may be necessary during pregnancy.

6.2.2 Transcatheter closure of ASD during pregnancy

Indication for treatment of ASD during pregnancy includes high risk of recurrent stroke, high risk for taking anticoagulation throughout pregnancy such as intracranial hemorrhage, prior intolerance to anticoagulation with complicating bleeding, thrombocytopenia, hypertension, preeclampsia or other system impairment such as renal or liver dysfunction.

Several precautions are taken if such procedure is required. Radiation of the fetus and its teratogenic effect are an important consideration during transcatheter therapy for ASD. First trimester irradiation will be associated with higher incidence of fetal malformation. Therefore, catheterization is performed in second trimester (13-28 weeks). Use of long venous sheath avoids direct radiation exposure and reduces radiation dose to pelvic area. Use of intracardiac echocardiography for balloon sizing and guidance of device deployment greatly reduces overall radiation exposure to the mother as well as the fetus. (Orchard et al. 2011) (Schrale et al. 2007). General anesthesia may be avoided by use of local anesthesia with conscious sedation for the catheterization procedure.

6.2.3 Surgical closure of ASD during pregnancy

Indication for surgical closure of ASD is rare. However, if the abovementioned indications for ASD closure exist and the ASD is unsuitable for transcatheter closure, surgical closure of ASD is indicated. Following precautions are suggested based on several observations. Ideal period for open heart surgery during pregnancy is second trimester (13 – 28 weeks) in order to avoid any fetal malformations (first trimester) and to avoid preterm labor, unfavorable maternal hemodynamics and increased maternal mortality (third trimester). During surgery, fetal bradycardia at the start of cardiopulmonary bypass may be avoided by infusion of high-concentration glucose to provide energy for fetus and intraoperative monitoring of fetal well being with cardiotachometer and fetal echocardiogram. During cardiopulmonary bypass, high-flow and high mean arterial pressure (60 mmHg), hyperoxygenation and maintenance of high hematocrit (> 25%) are advised. (Arnoni et al. 2003).

7. Outcome

A study compared pregnancy outcome in women who have had surgical repair of ASD before conception with women who have not had repair. 60 women (115 pregnancies) had surgery for ASD while 20 women (48 pregnancies) had unrepaired ASD. Incidence of still births, recurrence of congenital heart defect in the offspring or long term cardiac complications were similar in both groups. However, incidence of miscarriage, preterm delivery and cardiac symptoms during pregnancy were higher in women who had unrepaired ASD. (Actis Dato et al. 1998).

Drenthen et al. (2007) reported 0.8% incidence of arrhythmias, no cases of heart failure during pregnancy, 5% occurrence of thromboembolic events in women with ASD. Figure 2 provides the perspective of risk to the offspring in women with ASD compared with various congenital heart diseases. While outcome of pregnancy relatively better with ASD compared to other congenital heart diseases, a certain incidence of complications have been reported including preterm delivery (6%), small for gestational age at birth (2%), fetal mortality (2.4%), perinatal mortality (1.7%) and recurrence of heart disease (2%). (Drenthen et al. 2007)

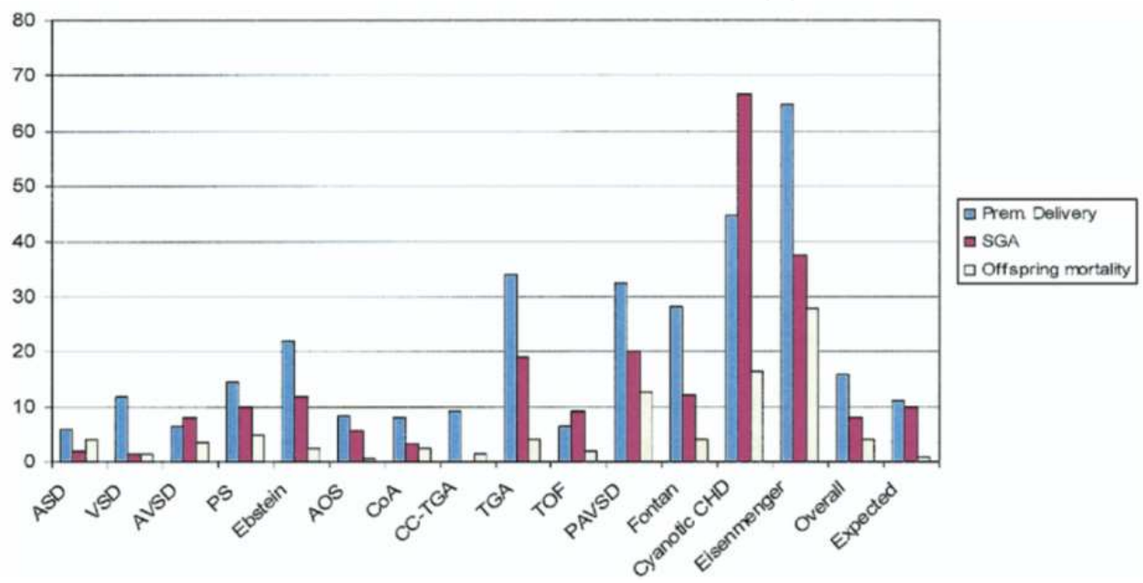

Fig. 2. Risk to the offspring from pregnancy in women with ASD compared to women with other congenital heart diseases. (Reproduced with permission. Drenthen et al 2007).

8. Contraception

Pregnancy is not contraindicated in women with ASD. However, there may be social and other clinical circumstances that may require delaying ASD closure and therefore, the pregnancy. In such circumstances, conception may be used. Since ASD is an acyanotic heart disease, there are more options compared to women with cyanotic heart disease. Barrier methods and intrauterine devices may be used. When these devices are inserted or removed, endocarditis prophylaxis is not indicated in patients with ASD. Though not crucial, progesterone-only pills have lesser incidence of thromboembolism compared to combination pills of estrogen and progesterone. However, progesterone-only pills may cause irregular bleeding especially in the first month of use. Efficacy of progesterone-only pills is slightly lower than combined contraceptive pills. Similarly, the morning-after pills are also safe and effective for women with heart disease. There are injectable forms of long-acting contraceptives available. From cardiac point of view, permanent methods of

sterilization such as tubal ligation or vasectomy for the partner are unwarranted in cases with ASD.

9. Conclusion

Pregnancy in women without heart disease has its own risks. Women with ASD who are asymptomatic, NYHA Class I or II are likely to have uneventful pregnancy. The ASD may be closed either surgically or by transcatheter technique if found suitable. For high risk patients, treatment of ASD during pregnancy may be undertaken. Second trimester is considered suitable for either transcatheter or surgical repair of ASD. Radiation dose is minimized by using intracardiac echocardiogram and modifications of the catheter procedure. Similarly, precautions are taken during open heart surgery should surgical repair were necessary. However, such need for closure of ASD during pregnancy should be rare and if possible avoided.

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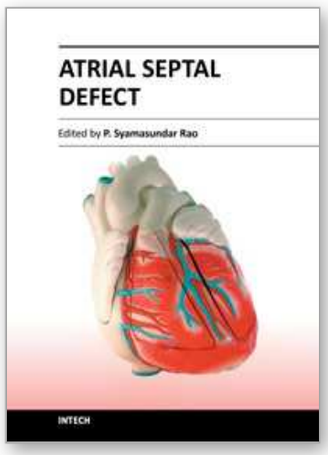

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Atrial Septal Defects (ASDs) are relatively common both in children and adults. Recent reports of increase in the prevalence of ASD may be related use of color Doppler echocardiography. The etiology of the ASD is largely unknown. While the majority of the book addresses closure of ASDs, one chapter in particular focuses on creating atrial defects in the fetus with hypoplastic left heart syndrome. This book, I hope, will give the needed knowledge to the physician caring for infants, children, adults and elderly with ASD which may help them provide best possible care for their patients.

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