IS LOW BIRTH WEIGHT ASSOCIATED WITH CONGENITAL HEART DISEASE?

Nutan Nalini Bage¹, Atul Mukul Bage²

¹Associate Professor, Department of Anatomy, PIMS, Pondicherry. ²Professor, Department of ENT, SMVMCH, Pondicherry.

ABSTRACT

BACKGROUND

Daily we see lots of still birth cases and the agony associated with it after carrying the fetus for so many months with the negative outcome it is quite disheartening. Malnutrition is quite rampant in the country as far as the females are concerned and in the name of the religion and rituals, it complicates the issue further. If the mother is malnourished, the chance of low birth weight baby is high.

OBJECTIVES

To correlate the prevalence of cardiac anomalies in low birth weight fetuses.

MATERIAL AND METHOD

The study was carried out in 40 stillbirth fetuses with detailed account of nutritional status of the mother, consanguinity, history of previous pregnancies, miscarriages, socio-economic status and exposure to drugs/radiation. The number of cases with fetus having cardiac anomalies was quite significant.

RESULTS

Out of total 40 stillbirth fetuses, 29 (72.5%) stillbirths were from less than 2.5kg and 11 (27.5%) were from more than 2.5kg. Cardiac anomalies were present in 16 and 02 cases respectively. Fused heart-01, mesocardia-01, Dextrocardia with CORV-02, Interrupted aortic arch-02, Abnormal origin of right subclavian artery-01, Tetralogy of Fallot-01, VSD-07, ASD-01, transposition of great vessels-01, persistent truncus arteriosus with tricuspid atresia, elongated hypertrophied abnormally positioned Rt. Atrium, rudimentary Rt. Ventricle-01, mal-attachment of ductus arteriosus-03, double superior vena cava-01, dilated caval system-01.

CONCLUSION

Considering the high incidence of cardiac anomalies in fetuses of low birth weight, we must try to create awareness and support the expectant mothers to avoid the low birth weight babies and thus the congenital anomalies.

KEYWORDS

Still birth, Low Birth Weight (LBW), Congenital Heart Disease (CHD).

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INTRODUCTION

In this study we wanted to know–is there any association of the low birth weight fetus and cardiac defects? The causes are many for cardiac anomalies including poor socio-economic status, exposure to drugs/radiation, consanguineous marriages. Poor nutrition means there is every possibility that the baby would be of low birth weight. Malnutrition is quite rampant in the country as far as the females are concerned, in the name of the religion and rituals it is further compounded. With low birth weight baby the chances of cardiac and extracardiac anomalies are very high. Low body weight in cardiology is deemed as 2.5kg or below.^(1,2,3) though it can be 2.0kg and below.^(4,5)

Establishing the prevalence of Low Birth Weight (LBW) is particularly important, since perinatal morbidity and mortality are more frequent in LBW than in normal infants and has become the second cause of death in this period, after

Financial or Other, Competing Interest: None. Submission 18-01-2016, Peer Review 13-02-2016, Acceptance 19-02-2016, Published 01-03-2016. Corresponding Author: Dr. Atul Mukul Bage, Plot No. 39,40. Messiah Street, Raja Annamalainagar, Gorimedu, Pondicherry-605006. E-mail: atulbage@yahoo.co.in DOI: 10.14260/jemds/2016/206 premature birth.⁽⁶⁾ Furthermore, socio-economic factors like habitat, education, birth order, age and religion also affect birth weight.⁽⁷⁾ Moreover, women born with LBW are more likely to give birth to infants with LBW, contributing to the transgenerational cycle of malnutrition and poverty.⁽⁸⁾ Among other causes of stillbirth, the small for gestational age fetus had an incidence of stillbirth of 46.8 per 1000, whereas the appropriate-for-gestational-age fetus had a rate of 4.0 per 1000.⁽⁹⁾

MATERIAL AND METHODS

A total number of 40 stillbirth fetuses of varying gestational age of both sexes of known and unknown causes were procured from the Department of Obstetrics and Gynaecology, JIPMER. The study was conducted in the Department of Anatomy, JIPMER, Pondicherry. Detailed history of mother was taken, especially in terms of nutritional status, consanguinity, history of previous pregnancies, miscarriages, socio-economic status and exposure to drugs/radiation, possible cause of fetal death and mode of termination.

Consent for autopsy and embalming was obtained from the parents, the fetuses were subjected to detailed external physical examination and the anomalies were noted. A midline incision was given below the chin to pubic symphysis to open the neck, thorax and peritoneal cavity. The detailed anomalies both cardiac and extracardiac was recorded and analysed. In this article, we are emphasizing only the cardiac defects which were associated with low birth weight.

RESULTS

Weight	CVS Malformations				Total	
	Present		Absent		IUtai	
<2.5kg	16	55.17%	13	44.83%	29	
>2.5kg	02	18.18%	09	81.82%	11	
Table 1						

Chi sq. - 4.409. Degrees 0 - 1. P- Value - 0.0357

Table 1 shows the CVS malformation in <2.5kg of birth weight fetus present in 16 (55.17%) and absent in 13 (44.83%), total no.=29. In >2.5kg the defects were found in only 02 (18.18%) and absent in 09 (81.82%) cases, total no.=11. P-value -0.0357.

CARDIAC AND GREAT VESSELS ANOMALIES	NO.				
Fused heart	01				
Mesocardia	01				
Dextrocardia with CORV	02				
Interrupted aortic arch	02				
Abnormal origin of right subclavian artery	01				
Tetralogy of Fallot	01				
V.S.D	07				
A.S.D	01				
Transposition of great vessels	01				
Persistent truncus arteriosus with tricuspid atresia, elongated hypertrophied abnormally positioned Rt. Atrium, rudimentary Rt. Ventricle	01				
Mal-attachment of ductus arteriosus	03				
Double superior vena cava	01				
Dilated caval system	01				
Table 2					

Table 2 showing the different types of cardiac malformation, which was found during dissection. Fused heart-01, mesocardia-01, dextrocardia with CORV-02, interrupted aortic arch-02, abnormal origin of right subclavian artery-01, Tetralogy of Fallot-01, V.S.D-07, ASD-01, transposition of great vessel-01, persistent truncus arteriosus with tricuspid atresia, elongated hypertrophied abnormally positioned Rt. Atrium, rudimentary Rt. Ventricle-01, malattachment of ductus arteriosus-03, double superior vena cava-01, dilated caval system-01.

DISCUSSION

Stillbirth pregnancies are associated with increased prevalence of Small for Gestational Age (SGA) fetuses compared with live births. In fact, the risk of stillbirth increases with declining percentile of birth weight.⁽¹⁰⁾

However, it is fetal growth impairment and not small fetal size that is associated with stillbirth. Unlike SGA fetuses, the fetuses with impaired growth are those that are smaller than expected after physiologic factors determining fetal size were considered. A study of over 300,000 pregnancies clearly demonstrated that stillbirth is more frequent only among these foetuses.⁽¹¹⁾

The cross-sectional studies of association between fetal size and growth and stillbirth have a significant limitation. The

time of death is not precisely known and gestational age at delivery might be considerably longer than at the time of demise, falsely increasing incidence of SGA and growth restricted fetuses. Despite this uncertainty, the relation between fetal growth impairment and stillbirth seems real. Several recent studies demonstrated strong, "Dose dependent" and biologically plausible associations between concentration of early or mid-pregnancy placental hormones and stillbirth.^(12,13,14)

In contrast, for live births the frequency of congenital heart defects in various epidemiological studies has ranged between 4 and 8 per 1000.(15-18) Necropsies on neonates (Still births and live born infants dying shortly after birth) have shown incidence rates for congenital heart defects varying between 6% and 13%.(19-24)

Max Godfrey, et al.⁽²⁵⁾ in their observation shows a sevenfold higher incidence of CHD in the VLBW neonates and an eleven-fold higher incidence in the extremely low birth weight population as compared to the reported incidence of 0.5–0.8% in the general population.⁽²⁶⁻²⁸⁾ A population-based study has shown low birth weight, small for gestational age and preterm birth to be associated with many forms of CHD.⁽²⁹⁾ Interestingly, transposition of the great arteries and aortic stenosis were not associated with these variables in that study.

A more recent study of non-syndromatic CHD showed a two-fold increase in likelihood of being small for gestational age, a three-fold increased risk of prematurity and a reduced mean birth weight in the CHD population.⁽³⁰⁾ The latter study found the associations to be valid for all types of CHD tested. Their results did not show a statistically significant relationship between birth weight and incidence of CHD within the VLBW population, although this may have been due to the small numbers involved. It could represent a true increase in CHD in the VLBW population. The reasons for the higher incidence of CHD observed in premature infants remain unclear. They speculated that it may be due to the fact that small septal defects close spontaneously in utero and thus fewer may be apparent in mature infants.

This would not, however, explain the reported increased incidence of non-septal defects in our LBW population. It is also possible that CHD may be independently associated with premature birth and/or low birth weight. It is generally accepted that the causes of CHD are multifactorial involving a wide range of genetic (Including non-syndromatic) and environmental risk factors.^(31,32) Thus, prematurity and/or intrauterine growth retardation or small for gestational age weight may be causally related to the same factor(s) that precipitated the CHD.

C Tennstedt, R Chaoui, et al.⁽³³⁾ found in their study of aborted fetuses, ventricular septal defect the most common defect (28%). In no case did it occur as an isolated lesion. In 56%, it was associated with other cardiovascular anomalies (Double outlet right ventricle, coarctation of the aorta, Tetralogy of Fallot, tricuspid atresia, atrial septal defect, transposition of the great arteries and truncus arteriosus communis. Ventricular septal defect has been reported to be the most frequent cardiac defect in various other necropsy studies of live births and stillbirths.^(19,20) and in those studies the proportion was higher than in ours (Between 32% and 42%). We totally agree that VSD is the most common finding always clubbed with other anomalies.

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Infants with congenital malformations have on average lower birth weight than do infants without malformations with a few exceptions.^(34,35) The relation between growth restriction and malformations may be explained by either the growth restriction as primary and predisposing the fetus to malformations or as secondary to the presence of malformations or by coexisting with a malformation due to common etiologic factor(s).^(34,35)

CONCLUSION

It is amply clear from our reports and the review of literature and discussion that definitely there is a correlation between CHD and low birth weight fetus. The era is different and with the advent of latest equipment we can prevent the poor outcome. What we personally believe is awareness is the key and with it at least we can minimize the tragedies.

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