Risk Factors of Small for Gestational Age

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Abstract

Objectives: A case-control study conducted in Al-Yarmouk & Al-Kadhmya Teaching Hospitals for the period from the 1st of March 2000 to the 28th of February 2001, to identify the risk factors for small for gestational age neonate in our hospitals.

Method: The study was done in the delivery rooms & the intensive care units, detailed history; including mother health before & during pregnancy.100 small for gestational age neonate &100 normal were evaluated within the 1st hour of delivery. Risk factors include antenatal care, parity, birth order, exposure to radiation, drugs or smoking, placental infarction, previous history of abortion, stillbirth, or premature delivery, age of the mother & weight, history of toxemia, hypertension, & renal diseases.

Result: Among the 100 small for gestational age neonates, we found 52% male &48% female; there is significant relation between small for gestational age & asymmetrical growth, with twin pregnancy, 62% in primigravida, was more in those with inadequate antenatal care 52%, with decrease placental size 40%. 33%had exposure to drugs & smoking. Small for gestational age was more with chronic illnesses, more with cesarean section, mothers who had low weight before pregnancy were at high risk to have small for gestational age neonate.

Conclusion: SFD is associated with significant risk factors, which should be considered by both Obstetrician & Pediatrician to decrease the incidence of the problem.

Keywords: Risk factors, small for gestational age.

Introduction:

World Health Organization (WHO) in 1950 defined a baby as SGA when it was below the 10th weight centile for gestation [1,2]. IUGR is often classified as reduced growth that is symmetrical (head circumference, length, weight are affected) or asymmetrical (with relative head growth sparing) [3].

The causes of SGA include maternal, fetal, and placental causes. The maternal causes include; hypertension & renal diseases "both have significant association with SFD by reducing utero-placental perfusion. [4], increased renal insufficiency may be accompanied by retarded growth [5], also the circulating IGF-1&1, 25-OH 2D level in both maternal & umbilical cord compartment are low in pre-eclampsia [6], malnutrition &chronic illnesses "most often the fetus grows normally despite significant decreased maternal calories intake. The famine persists for 28weeks & there were an average birth weight. Decrease of 250gm per infant. [7], for women having chronic illness like diabetes mellitus, among with pregestational diabetes mellitus the frequency of pre-eclampsia rise with increasing severity of diabetes [8]", drugs "chronic use of drugs like opium derivatives, barbiturates, &amphetamines in large doses is harmful to the fetus, [7], smoking during pregnancy is associated with average decrease in fetal weight of 200 gm [3], the negative effect on fetal growth from maternal smoking was found to affect the male fetus proportionally more than the female [9]". The fetal causes include; multiple gestations "the incidence of SFD is higher (14.3%) among the twins [10],twin pregnancies complicated by pre-eclampsia show higher rate of SFD neonates among second twin than those with normal pressure [11], chronic fetal infection "cytomegalovirus, congenital rubella, syphilis, toxoplasmosis, Herpes simplex are associated with fetal growth retardation [7]".

The placental causes; birth weight increases with increasing placental weight in both animals & humans IUGR without other anomalies are usually associated with a small placenta. A small placenta is not always associated with an IUGR newborn, but the large infant from otherwise normal pregnancy does not have a small placenta. In placentas of IUGR infants, the mean surface area &the capillary surface area are reduced, implying a diminished diffusing capacity, placental infarction & chorionic villitis are commonly present in placentas of IUGR infants [12].

Others causes include; inadequate prenatal care, women who not have insurance coverage for prenatal care at increase risk of low birth

weight. [13], the incidence of SGA in teenagers' nilliparous is higher with fewer cesarean & instrumental deliveries [14], Small women have typically smaller babies & mothers" birth weight is strongly associated with the weight of her baby [15] & SGA girls had a smaller uterus & reduced ovarian volume [16, 17], approximately 40% of total birth weight variation is due to genetic contributions from mother & fetus, approximately half in each, the other 60% from the fetal environment. Although both parents' genes affect childhood growth & final adult size, the maternal genes have the main influence on birth weight [12].

Patient & Method

The study was done in the delivery rooms & neonatal intensive care units, detailed history, including mothers" health before & during pregnancy. 100 SGA neonates& 100 normal wt neonates, as a control group, were evaluated within the 1st hour after delivery. All neonates were weighed naked using a standard beam scale, to nearest 10gm.

A full general examination was done for each baby, to detect any abnormality. Gestational age was assessed by the last menstrual period, ultrasound & expanded new Ballard score. Mothers' prepartum weight was measured before labour using a standard health-0-meter, 160 kg capacity.

Statistical analysis was done with (EPI-INFO version-6); data presented through simple frequency distribution tables for each variable (Odd Ratio for case control study). The term risk, which was, used in this study means the likelihood that an individual will contract a disease. [8,19]. Risk factors in this study include, antenatal care, parity, birth order, exposure to radiation, drugs, smoking, placental infarction, previous history of abortion, stillbirth & preterm deliveries, age and weight of mother, history of toxemia, hypertension or renal disease.

Results:

Total number was 200 neonates, 100 were SGA, & the others100 were the control group. In this study 48 were female, & 52 were male, as shown in the table; 30 neonates had asymmetrical growth & 70 neonates had symmetrical growth, the risk of twin pregnancy & associated twin-twin transfusion is shown also. Regarding birth order higher percentage of SGA neonates were born to primigravida, 62%, only 38% to multigravida. In52%of SGA neonates their mothers had inadequate ANC (delayed infrequent irregular visits). The relation between placenta & SGA is also shown in the table as well as for the relation of toxemia & hypertension with SGA. SGA had significant relation with previous history of stillbirth, but not with abortion. There was significant relation with drug exposure shown in this study. Mothers with chronic illness, like Rheumatoid arthritis, Thyrotoxicosis had significant risk. The risk of SGA was more with history of infertility 5 times more than the control group. Neonates delivered by cesarean section had 1.35 times risked being SGA. Pregestational weight of mothers was a significant factor for SGA delivery, mothers with low weight had increased incidence of LBW neonates

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Table: The different factors associated with SGA.

	SGA		Control		χ2;P	OR (95% C.I.)
	No.	%	No.	%		
Sex Females	48	48	47	47	0.02; 0.887	1.04 (0.58-1.88)
Males	52	52	53	53		
Growth Asymmetrical	30	30	4	4	23.95; 0.0001*	10.29 (3.37-41.57)
Symmetrical	70	70	96	96		
Multiple pregnancy Twin	12	12	4	4	4.35; 0.037*	3.27 (0.94-14.36)
Single	88	88	96	96		
Twin-Twin transfusion	3	3	-	-	-	-
Normal pregnancy	97	97	100	100		
Placental surface Large	1	1	-	-	26.43; 0.0001*	6.85 (2.97-17.12)
Reduced	40	40	9	9		
Normal	59	59	91	91		
Placental infarction	7	7	-	-	-	-
Normal	93	93	100	100		
Placental infection	2	2	1	1	0.34; 0.561	2.02 (0.10-120.4)
No	98	98	99	99		
Toxemia	30	30	11	11	11.0; 0.0009*	3.47 (1.55-8.19)
No	70	70	89	89		
Hypertension	8	8	7	7	0.07; 0.788	1.16 (0.35-3.91)
No	92	92	93	93		
History of previous abortion	21	21	20	20	0.03; 0.861	1.06 (0.5-2.24)
No	79	79	80	80		
History of previous stillbirth	11	11	1	1	8.87; 0.003*	12.24 (1.7-531.9)
No	89	89	99	99		
Exposure to drug	33	33	5	5	25.47; 0.0001*	9.36 (3.36-31.98)
None	67	67	95	95		
Chronic illness	10	10	1	1	7.79; 0.005*	11.0 (1.5-482.1)
No	90	90	99	99		

Discussion:

In this study we found that the incidence of SGA was nearly the same in both sex, the male: female ratio was 1.08:1 that was not consistent with Thomas study which showed female neonates were smaller than male [20] & also with Resnic [12].

70% of neonate had symmetric IUGR & 30% had asymmetric IUGR, which means, intrinsic fetal insults occurring early in pregnancy, such as infection, exposure to certain drugs, or other chemical agents, etc .are likely to affect fetal growth at time of development when cell division is the predominant mechanism of growth, consequently musculoskeletal

dimension &organ size may be adversely affected & symmetric growth restriction observed, at the other end of the spectrum an extrinsic insult occurring later in pregnancy usually fetal nutrition is more likely to result in a symmetrical growth retardation characterized by inadequate.[12]

Twin pregnancies especially those who had twin-to-twin transfusion, had increased risk for SGA neonate& this is agreed with a study done by Okogbo [10] & also with Yu [21]. 62% were primigravida& 38%were multiparous mothers, this is consistent with study done by Lao [14]. The study showed, good ANC was an important factor in lowering the percentage of SGA this is consistent with study done by Wisp [13]

There was increased risk of SFD with reduced placental surface, placental infarction; this is consistent with study done by Fox [22]. Mothers with toxemia, & those with hypertension had more risk to have SGA; this is consistent with study done by Janunsas [23]. Mothers who had previous history of stillbirth had significant risk of SGA; this is consistent with study done by Wisp [13]. 33%of mothers had history of drug ingestion, mothers exposed to smoking showed two times risk getting SGA; this is consistent with Bjelovar [24]. Chronic illnesses were significant risk& this is agreed with study done by Skomsvoll [25], & Moser [26].

Higher percentage of neonates resulted from CS deliveries, this means that their mothers had complicated pregnancies; this factor had 1.35-time risk, this is agreed with study done by Janusas [23].
Mother wt before conception was a significant factor, low wt increase the risk of SFD& this is consistent with a study done by Jacquet & colleagues in Paris [27] and also with Al-Bayatti & Al-Ani in Iraq [28], Isaksen [29] and with Clausson [30].

Conclusion:

SFD is associated with significant risk factors, which should be considered by both Obstetrician &Pediatrician to decrease the incidence of this problem.

Recommendation:

1. Good ANC, especially to those mothers at risk.
2. Education of mothers to avoid certain risk factors.

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