

Caring for Fetal Growth Retarded Infants. Are we Getting it Right?

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Fetal growth restriction affects 10 to 15% of live births in Western rich countries, and up to 30% of all births in low and middle-income countries [1-3]. Being born small for gestational age (SGA) is associated with high morbidity and mortality both in the perinatal period and beyond, making it an important public health matter of concern. It is well established that SGA children are at higher risks of poor neurodevelopmental outcomes, and also of developing metabolic syndrome, a constellation of several conditions such as insulin resistance, dyslipidemia, high blood pressure, and cardiovascular complications in adulthood [4]. Additionally, 5 to 7% of these children fail to show catch-up growth by the age of 3 - 4 years and require endocrine care [5].

The exact prevalence of SGA remains unknown as a consequence of the discrepancies in its definition (height or weight separately, or height, weight, and head circumference together) and reference growth curves used. Published epidemiological studies also add to the existing confusion as some analyze term-born and preterms infants in the same cohorts [6]. Causes of SGA are numerous and maybe maternal (pregnancy-induced hypertension, tobacco consumption, uterine malformations, alcohol and drugs consumption, etc.), fetal (chromosomal abnormalities, fetal toxoplasmosis, other [syphilis, varicella-zoster, parvovirus B19], rubella, cytomegalovirus and herpes [TORCH] infections, etc.), environmental (high altitude, poor socio-economic conditions) and idiopathic. Although these various causes lead to fetal growth restriction, it is reasonable to consider that their physiopathological mechanisms are likely different. Maternal smoking during pregnancy affects fetal growth through several mechanisms such as chronic hypoxia, IGF-I and leptin synthesis [7,8], nicotine acts as a central nervous system toxicant. Maternal smoking also modifies fetal DNA methylation and more often causes symmetrical SGA which has poorer neurological outcomes. Maternal pregnancy-induced hypertension (PIH) affects primarily the fetoplacental unit through changes in its vasculature leading to chronic hypoxia and induces an asymmetrical type of SGA, often sparing fetal brain development as shown by a normal head circumference at birth. Maternal alcohol and drug consumption are essentially toxicants and impair neuronal development, they lead to reduced head circumference. Infants with chromosomal abnormalities have complex structural genetic derangements, etc.

In low and middle-income nations, fetal growth restriction is consequent to several mechanisms, of which malnutrition is an important component. This deprives the fetus of a supply of essential nutrients, etc.

It, therefore, appears difficult to interpret SGA outcomes without considering their primary causes individually. Although some attempts have been made [6,9], we still need to design future studies that evaluate outcomes in SGA children per etiological groups and separating term-born from preterm infants. Taking into account each child's ethnic origin is another point to consider, knowing that Sub-Saharan mothers often present with PIH, whereas South-East Asians give birth to constitutionally small babies [10]. Different causes are likely to produce different consequences and therefore, different outcomes.

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