Mini Review

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Finding Facility-Based Cost Effective Options for Improving the Survival of Preterm Neonates in Low Income Countries

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Abstract

In this retrospective study we did a relative analysis of the outgrowth of 28 1 to 320 weeks gravidity babies between the State of Qatar and some high income countries with an ideal of furnishing an substantiation base for perfecting the survival of preterm babes in low income countries. Data covering a five time period (2002-2006) was caught on on a pre-designed Performa. A relative analysis with the most recent data from VON, NICHD, UK, France and Europe was accepted. Qatar's 281 to 320 weeks Prematurity Rate (9.23 per 1000 births) was lower than the UK's($p < 0.0001$). Of the 597 babies born at 28 1 to 320 weeks of gravidity didn't bear any respiratory support, while31.1 needed only CPAP remedy.80.12 of the MV and 96.28 of CPAP remedy was needed.

Keywords: Epidemiology • Gravid age • Mortality • Morbidity • Qatar • Developing countries

Introduction

Veritably Preterm Babies (≤ 32 weeks of gravidity) constitute 1-2 of all live births in high income countries, but account for at least one third of perinatal mortality, the maturity of neonatal mortality, as well as both short- term (pre discharge, in sanitarium) and long- term (at two times corrected age) morbidity. Although neonatal mortality and morbidity are known to worsen with dwindling gravid age and weight at birth, the dramatic enhancement in the complete survival of preterm babies has been one of the most remarkable features of neonatology in high income countries over the last three decades. similarly, the question of how 'small is small' has, over the last two decades, gradationally dropped in terms of gravidity period from 32 weeks to 28 weeks and also 24 weeks; and in terms of birth weight from,500 g to 1000 g, to 800 g and also 500 g (10-15). Still, the long term issues, the cost of care of babies born ≤ 28 weeks (particularly ≤ 26 weeks) and or birth weight ≤ 1000 g (and particularly ≤ 750 g), and the futility of intervention at the edge of viability remains a hot debate, indeed among the utmost resource rich countries and the care of extremely unseasonable babies (≤ 28 weeks and ≤ 1,000 g at birth) isn't an option for resource confined developing countries. Styles this retrospective logical study was approved by the Institutional Research Board of Hammad Medical Corporation protocol No. 7004/07, and accepted in the Neonatal ferocious Care Unit Women's Sanitarium in Doha the only tertiary care motherliness and neonatal unit in the State of Qatar. The Hospital was accredited in 2006 and re-accredited in 2009 by the Joint Commission International (JCI) USA for its norms and Quality of Care [1-4].

About 99 of deliveries in Qatar take place in this sanitarium. All inborn and out born babies delivered ≤ 32 week gravidity in the State are admitted to the Women's Sanitarium. thus the data with respects to ≤ 32 week gravidity babies from our study not only directly represents Qatar's public data, for all practical purposes, it's original to a population-grounded study. We employed three major orders of outgrowth measures 281 to 320 weeks gravidity birth rates; in sanitariumpre-

discharge mortality (aggregate and gravid age specific); and in sanitarium pre-discharge morbidity (CLD, NEC, Characteristic PDA, IVH Grade III and IV, Cystic PVL, ROP ≥ Stage 3). The most common and unifying condition associated with hepatic carcinogenesis is cirrhosis, which develops after long latencies (20-40 years) of chronic liver disease. HCC risk remains low during chronic liver disease but dramatically increases at the cirrhotic stage. Hepatic carcinogenesis remains partly obscure. Initially, a variety of genetic and epigenetic alterations have been detected in human and experimental HCCs. Later on, DNA microarray analysis has led to an extensive integrative approach, leading to identification of clusters of HCCs that allow comparison between phenotypes in experimental and human HCCs, and may predict outcome of patients. However, none of the identified genes is universally expressed by tumor cells that are heterogeneous in their morphology, clinical behaviour, and molecular profiles in the tumor bulk. These observations lead to the suspicion that the current studies might have focused only on the heterogeneous "end products" i.e. "adult" tumor cells within the tumor bulk but not the "root" cell. More interestingly, molecular signatures from cirrhotic no neoplastic tissues can predict occurrence/recurrence of HCC, letting hypothesize that although "root" cells exist within the heterogeneous "end product" tumor bulk, they also might be present in cirrhotic tissues prone to develop HCC [5, 6].

Results

Aggregate of 689 live births were recorded during the study period with 597 of these babies being babies between 28 1 to 320 weeks gravidity, therefore giving a punctuality rate of 9.23 per 1000 total births for the target gravid age order during the study period. Our 281 to 320 weeks gravidity punctuality rate was lower than the rates for the same gravidity group in the UK ($p < 0.0001$). The patient characteristics of our sample in comparison to VON, which is an transnational database of further than 800 Neonatal ICU's; further than 95 of which are located in North America, Western Europe and Australia. 86.44 of our babies had birth weights between, 001 and, 000 g and only 3.9 were SGA as compared to 12 in VON database ($p < 0.0001$). These latter are not terminally differentiated and can respond to injury by highly regulated proliferation. Although the cell type giving rise to HCC has been shown as dependent on many factors in experimental hepatocarcinogenesis, few is known in humans. One can speculate that "root" cells might come from the neoplastic transformation of different normal liver cell types: periodical stem cells, bipolar ductal committed progenitor cells, or differentiated hepatocytes. Thus, it would not be surprising to find HCC as arising differentially from one (or several) of them, depending on extrinsic factors such as viral infection, or deregulation of intrinsic key pathways. The maturation arrest of cells at various stages of differentiation in a hierarchical cell lineage may best explain the various types of human liver cancer. From analysis of established HCCs, we might speculate that HCCs contain Cancer Stem Cells (CSC) i.e. cells with stem-cell-like properties of immortality, resistance to therapy, and transplant ability [7].

As hepatocarcinogenesis is likely a dynamic process leading from a normal cell towards an initiated root cell and thereafter a hugely heterogeneous tumor bulk, abnormalities useful for initiation of root cells are not mandatory found in all cancerous cells forming the tumor bulk. In addition, abnormalities found in tumor bulks might evolve with time and/or under pressure of anti-cancer therapies, thus strengthening the need of cautious when interpreting a molecular profile. However, some fundamental events have been described as key steps in cellular transformation and very likely necessary and sufficient to allow each cell to get and keep the cancerous phenotype [8].

Discussion

Among the low income countries, Sri Lanka stands out for having achieved a significant decline in its neonatal mortality rates by sustained investment in primary health care installations and without establishing numerous high tech precious neonatal ferocious care units. Still, any farther reduction in neonatal mortality in low income countries, particularly among the preterm babies, will need some installation- grounded interventions. Some low cost interventions like regular use of prenatal steroids for preterm labor and the adding use of noninvasive respiratory support in the neonatal units has contributed significantly to the survival of extremely preterm babies in high income countries. The operation of these low cost interventions may increase the complete survival of preterm babies at the limit of viability in the low income countries (281 to 320 weeks gravidity) at an affordable cost. In order to explore this proposition, we carried out

this logical and relative study of the outgrowth of 281 to 32 0 weeks gravidity babies in the State of Qatar. The selection of Qatar is a model of study was grounded on two explanations first, the Perinatal and Neonatal survival rates in Qatar have significantly bettered over the last thirty times to the extent that its current rates are similar [9].

Senescence mechanisms in hepatocytes and in liver tissue are not well known. However, a limited number of in vitro studies with hepatocytes, as well as numerous descriptive in vivo studies in liver tissue provide sufficient evidence that hepatocytes can undergo senescence type changes. Limited proliferative capacity of somatic cells is controlled by replicative senescence. By contrast to primary hepatocytes which do not proliferate in culture, fetal hepatocytes display better proliferation capacity and can enter replicative senescence. This is accompanied by progressive shortening of telomeres in a context of telomerase-free activity. In contrast to in vitro studies, in vivo senescence of human hepatocytes is better known. Replicative senescence displays a gradual increase from 10% in normal liver, to more than 80% in cirrhosis, being detected in 60% HCCs [10].

Conclusion

The State of Qatar has achieved an excellent rate of complete survival of 28 1 to 320 weeks gravidity babies which is similar to that of numerous high income countries. Qatar's data demonstrates that low cost ways (prenatal steroids for preterm labor and post natal use of CPAP remedy), can potentially save the maturity of 28 1 to 320 weeks gravidity babies by low income countries with minimum in sanitarium pre discharge morbidity. The assessment of long term morbidities (at two times neurodevelopmental follow up) will be the true determinant of the ultimate outgrowth. Further, up to date studies in this area will be helpful for prioritizing health care investments in resource constrained countries. Telomere shortening during aging is slow and stabilizes at mid age in healthy liver, so that the loss of telomere DNA does not reach a level to induce telomere dysfunction and DNA Damage Response (DDR). On the other hand, telomere loss is accelerated in chronic liver disease to reach lowest levels in the cirrhotic liver. Therefore, one plausible mechanism involved in cirrhosis is probably telomere-dependent senescence, the so called replicative senescence [11-15].

Acknowledgement

None

Conflict of Interest

None

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