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Review Article

Birth Weight and Susceptibility to Chronic Kidney Disease

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ABSTRACT. The worldwide prevalence of noncommunicable diseases (NCDs) is projected to increase substantially over the next few decades. Chronic kidney disease (CKD) is a key determinant of poor health outcomes for major NCD. Genetic predisposition and environmental exposures are contributory factors, but increasingly, it is being recognized that fetal development is also an important modulator of the NCD risk. Low birth weight (LBW) and CKD affect more disadvantaged populations and ethnic minorities and, therefore, causes a disproportionate burden on the poor. Human nephron number is highly variable and may range from under half a million to almost over two million. Significant variability is already present at birth, highlighting the importance of early nephrogenesis. Nearly 60% of nephrons are developed in the third-trimester of pregnancy. Nephron numbers increase in proportion to birth weight and gestational age. This wide-variability probably contributes to individual susceptibility to develop CKD where individuals with nephron numbers on the lower side of the spectrum are those at higher risk of developing kidney dysfunction at higher rate and progress more toward end-stage CKD. This article aims at discussing LBW and the susceptibility to CKD. Furthermore, in postnatal environment, the weight gain or change at adult life increases the metabolic demand and determines the phenotypic expression of disease along with the spectrum of nephron number. Hence, a cycle of hyperfiltration mechanism of these nephrons leads to proteinuria, glomerulosclerosis, and progressive development of larger glomeruli, a greater risk of proteinuria and progressive CKD. Therefore, LBW offspring are at risk of developing CKD (defined as albuminuria, a reduced glomerular filtration rate, or renal failure) in later life. Furthermore, the impact of prenatal programming is expected to be compounded with age, and the association of LBW with the risk of CKD seen in younger adults may become greater with age. It would be prudent, to adopt policies of intensified life-long surveillance of LBW people, anticipating this risk.

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Introduction

The noncommunicable diseases (NCDs) are spreading across the globe in recent years. Various metabolic syndromes, with obesity as its integral component, are fuelling various

risk factors and end-organ failure. Furthermore, genetic predisposition and environmental exposure are contributory factors, but increasingly, it is being recognized that fetal development is also an important modulator of the risk of NCD.

Birth weight varies across the globe. In Oman, low birth weight (LBW) was 10.2% out of a total live births of over 66,000 in the year 2013. The worldwide prevalence of LBW is 15.5% which amounts to about 20 million LBW infants born each year 96.5% of them in the developing countries. LBW has been increasing globally and regionally with various advancements in medical care, including obstetric and neonatal care and technological developments, with reduced fetal growth and pregnancy-related complications are being seen among live births. For example, in Oman, the prevalence of LBW was 4.2% in 1980, which doubled (8.1%) in 2000 and has shown a slow but steady increase reaching 10.2% in 2013.[1] This also causes an increase in the rate of LBW infants, and subsequently an increased rate of long-term medical sequelae. It is now well proven that LBW, reflecting a poor intrauterine environment, is associated with diminished nephron endowment and kidney functional reserve, and a predisposition to progressive kidney failure in a variety of kidney diseases.

Birth Weight, Kidney Size, and Kidney Volume

Evidence is mounting that nephron deficits result from environmental changes *in utero*. Konje et al reported that kidneys are disproportionately affected as compared with other organs in "small for gestational age" fetuses.[2] Demographic studies have shown that people with a very high incidence of hypertension have a relatively small kidney size to their body mass, suggesting a diminished number of nephrons.[3-5]

Several studies have compared kidneys of fetuses with intrauterine growth retardation (IUGR) to those of normally grown fetuses by ultrasound examination. They found that kidneys in fetuses with IUGR were significantly smaller than kidneys of fetuses that were normally grown.[4,5] Furthermore, Spencer et al concluded that after correction for the current body size, LBW children have lower kidney volumes than children of higher birth weights.[6] The susceptibility to kidney disease associated with LBW in many populations, especially the most deprived population, might be mediated through reduced nephron endowment. Hence, smaller kidneys predispose to higher blood pressure and albuminuria. An autopsy study has shown persuasively that nephron number is correlated with kidney mass and with total glomerular mass, representing filtration surface area.[7]

Nephron number

The number of nephrons is characteristic of a species. It ranges from a few thousand in the mouse to several millions in the elephant.[8,9] It has been generally agreed for many decades that a human kidney contains about one million nephrons.[10-14] In some recent studies, the exact number varied between 400,000 and 1,000,000 nephrons per kidney,[15] while another study reported an even greater range of between 210,000 and 1,800,000.[7] Nephron number in human kidneys follows a normal distribution curve with a mean of 600,000 and a standard deviation of about 200,000.[15]

An autopsy study of 71 infants that died acutely *in utero* or within 24 h after birth found that the number of glomerular generations formed within the fetal kidneys was directly proportional to gestational age, body weight and kidney weight, with variability between individuals in the ultimate number of glomerular generations and in the timing of the cessation of nephrogenesis.[16] Higher nephron number provide a good vascular capacitance reserve to maintain an appropriate kidney function even after postnatal environmental insult, such as infections and certain drugs that may render a good number of nephrons functionless. Autopsy studies demonstrate that indigenous Japanese[17] and African-Americans[18] have smaller kidneys than Caucasians. These changes maybe related to prenatal or genetic

factors.

A study of the kidneys of stillborn human infants with IUGR, compared to appropriate for gestational-age (AGA) infants, using the dissector method (a stereologic procedure unbiased by feature size, shape, or tissue-processing methods, for the estimation of total glomerular number), showed that the IUGR group had 35% fewer nephrons than AGA infants.[10] Cross-sections of kidneys from full-term infants who died from nonkidney causes found that infants with a birth weight below the 10th percentile had lower kidney weights and fewer glomeruli than did infants with an appropriate birth weight for gestational age[19] (Figure 1).

A strong correlation between glomerular number (direct) and size (inverse) with LBW had been demonstrated.[19] While the number of glomeruli is directly proportional to the birth weight with high birth weight being associated with higher number of glomeruli, the size of the glomeruli was inversely related to the birth weight with LBW associated with larger glomeruli. A number of autopsy studies have shown reduced nephron numbers in infants, children and adults of lower birth weight compared to higher birth weight[7,14,15,17-24] (Figure 2). Endowment with decreased nephron numbers might be a risk factor for the rate of progression of kidney disease. Glomerular size varies in each individual and between individuals, and significantly enlarged glomeruli are susceptible to premature sclerosis.[7] Kidneys of preterm infants have been found to have a greater percentage of morphologically abnormal glomeruli and a significantly larger cross-sectional area of the renal corpuscle, suggestive of renal hyperfiltration.[16,25] These observations suggest that the preterm kidney may have fewer functional nephrons, thereby increasing vulnerability to impaired renal function in both the early postnatal period and later in life.[16,25]

Birth Weight and Nephropathy

A consistent relationship between short stature and microalbuminuria and nephropathy has been described in both nondiabetic and diabetic subjects.[26] In patients with type 1 diabetes, those with no proteinuria had a birth weight greater than those with microalbuminuria or frank proteinuria.[27] Seventy-five percent of women, with Type 1 diabetes, below the 10th percentile of birth weight (≤2700 g) had persistent albuminuria compared with 35% of those above the 90th percentile. Patients with Type 1 diabetes with severe diabetic glomerulopathy had significantly fewer glomeruli compared with type 1 diabetes patients with mild or no glomerulopathy.[28,29]

A continuous u-shaped association between birth weight and elevated urinary albumin

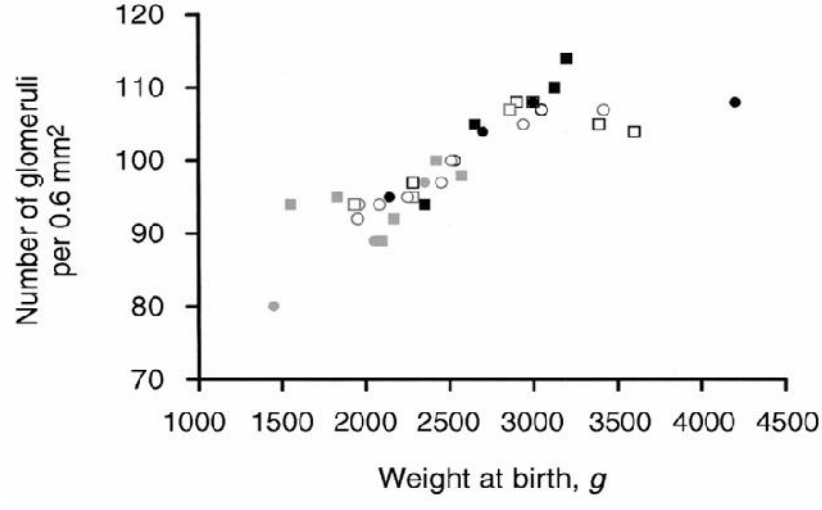

Figure 1. Relationship between the weight at birth and the number of glomeruli.

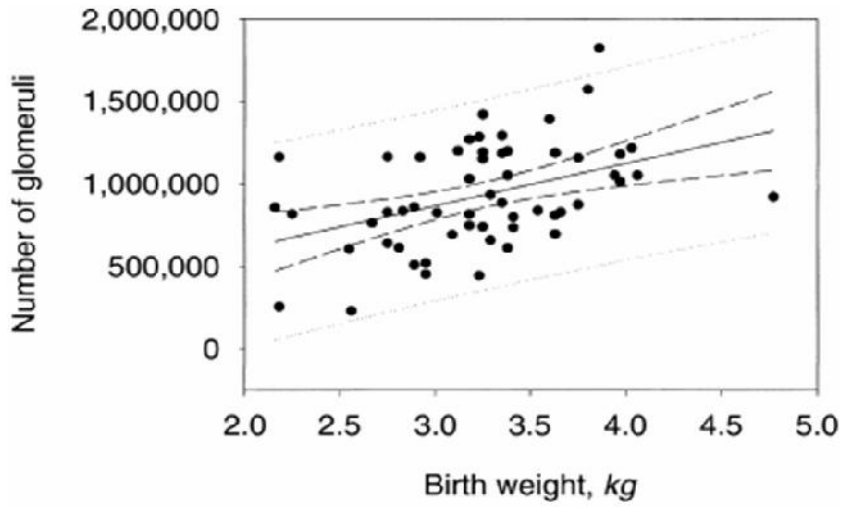

Figure 2. Relationship between birth weight and total glomerular number among all cases that includes infants, children, and adults.

excretion was reported in Pima Indians, even after adjustment for age, sex, duration of diabetes, glycated hemoglobin (HbA1c), and blood pressure. The rate of elevated albuminuria in subjects of LBW was 2.3 times that in those subjects of normal birth weight.[30]

In a retrospective study, Bendtsen and Nyengaard found that patients with severe glomerulosclerosis had 30% fewer nephrons than diabetic patients who had either no, or mild glomerular lesions.[31] This might imply that nephron number increases susceptibility to nephropathy. In addition, it has been found that albumin creatinine ratio (ACR) inversely correlated with birth weight, with a relative risk for overt proteinuria (ACR ≥34 g/mol) of 2.8 for individuals with LBW (Figure 3).

A multivariate analysis showed that LBW, body mass index (BMI), blood pressure, and diabetes amplify the risk of proteinuria with increasing age.[32] The risk of proteinuria, glomerulopathy, and progressive kidney failure appears to be the highest in patients with a reduction in overall kidney mass of more than 75%, and these changes are more likely to appear over a period of ≥10 years.[33] Moreover, many experimental studies have shown that a reduction of kidney mass causes

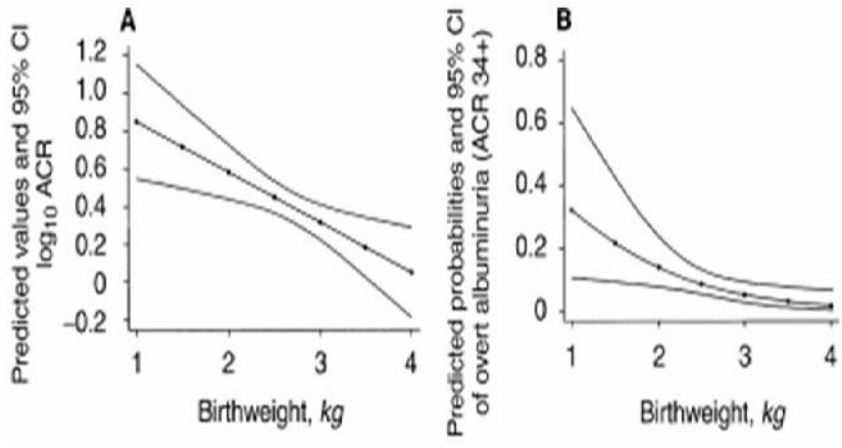

Figure 3. Albumin-creatinine ratio and birth weight.

adaptive increases in the size and function of the remaining nephrons, and that focal glomerulosclerosis eventually develops in association with proteinuria[34] (Figure 4).

Birth Weight and Kidney Failure

The "oligonephron theory" proposes that compensatory mechanisms for lower numbers of nephrons lead to deterioration of the function of the remaining nephrons. The morphological response to a nephron deficit is hypertrophy and hyperfiltration of the remaining nephrons. This manifests as proteinuria/albuminuria, and results in premature sclerosis of hypertrophied glomeruli. Factors that further enlarge nephrons such as obesity, cause nephron loss, and hence compound this phenomenon.

Environmental factors in genetically susceptible individuals increase the risk of reduction of number of nephrons, development of hypertension and end-stage-CKD (ESKD). Glomerular volume was found to be high in ethnic groups with a predilection for kidney failure.[7,18,24] In other words, people born with reduced number of nephrons compensate with increasing the size (volume) of their glomeruli and this renders them susceptible to kidney failure.

Birth Weight and Nephropathy in Various Ethnic Groups

LBW was significantly associated with a greater risk of ESKD in both African-Americans and Caucasians.[35] African-Americans and Caucasians, with ESKD attributed to hypertension are more likely to have been born with LBW compared to age, race, and gender-matched patients without ESKD.[36] The increased risk of ESKD in relation to LBW combined with a two-fold increase in the rate of LBW will contribute to a greater "population attributable risk" for African-Americans than Caucasians.[36] Among Pima Indians, a U-shaped association was found between birth weight and albumin excretion in diabetics, i.e., both LBW and HBW (largely due to gestational diabetes) correlated with increased albumin excretion. A nationwide screening program of 2083 in multiracial Southeast Asian pediatric population found that LBW and early malnutrition was associated with a

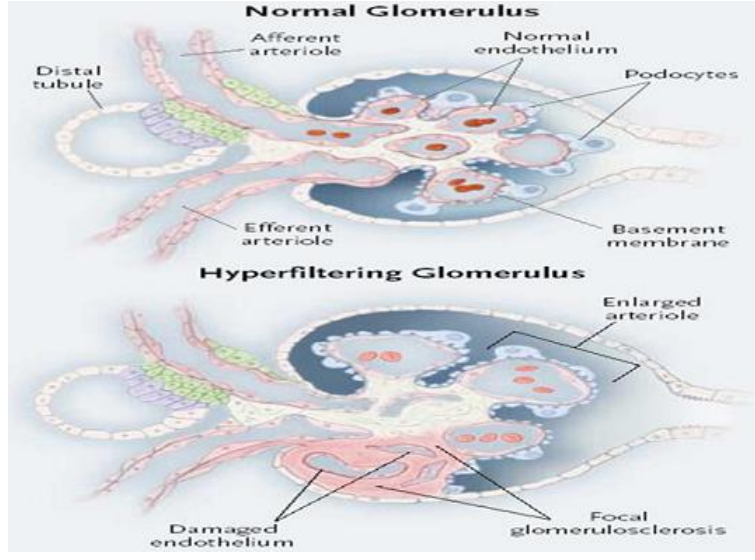

Figure 4. Hyperfiltration of glomeruli.
Hyperfiltration.
The presence of relatively few glomeruli leads to increased filtration by each glomeruli. Over time, this hyperfiltration may cause glomerular injury.

high prevalence of proteinuria, elevated blood pressure, and chronic kidney disease (CKD) in later life.[37] A correlation between LBW and CKD has also been described in poor African-Americans and Caucasians living in the southeastern United States. A continuous u-shaped association between birth weights and elevated urinary albumin excretion was reported in Pima Indians, even after adjustment for age, sex, duration of diabetes, HbA1c, and blood pressure. The rate of elevated albuminuria in subjects with LBW was 2.3 times that in those with normal birth weight.[30] Studies in Australian Aboriginal population showed lower nephron number and larger glomerular volume, as well as, more broadly, a direct correlation of birth weight with nephron number and, inversely, glomerular volume and subsequently development of CKD.[38-40] We reported previously that patients with CKD regardless of their age had, lower birth weight than the general population. People with CKD had a 3.9-fold increased risk of LBW.[41]

Hughson et al found that birth weight is a strong determinant of glomerular number and of glomerular size in the postnatal kidney from 37 African Americans and 19 Caucasians.[42] They found that glomerular volume is strongly and inversely correlated with glomerular number (Figure 5). This powerful inverse relationship between glomerular size and number implied an adaptive process, whereby glomerular size increased to compensate for innately low nephron number that may increase the susceptibility to glomerular scarring, and subsequent development of kidney failure (Figure 4).

Birth Weight and Chronic Kidney Disease Progression

Systematic review and meta-analysis of observational studies by White et al showed that LBW is associated with subsequent risk of CKD, although there is scope for additional well-designed population-based studies with accurate assessment of birth weight and kidney function and consideration of important confounders, including maternal and socioeconomic factors.[43]

In a study of risk factors for the progression of kidney failure in patients with idiopathic membranous nephropathy, weights at birth correlated with the gradients of reciprocal serum creatinine (1/SCr) regression lines.[44] In addition, 23 young women with scarred kidneys but stable kidney function had significantly heavier birth weights than 17 matched patients with progressive chronic kidney failure.[45] Furthermore, IUGR was associated with the higher prevalence of biopsy-proven glomerulosclerosis and with later risk of the development of hypertension in 50 children with IgA glomerulonephritis followed for 3–

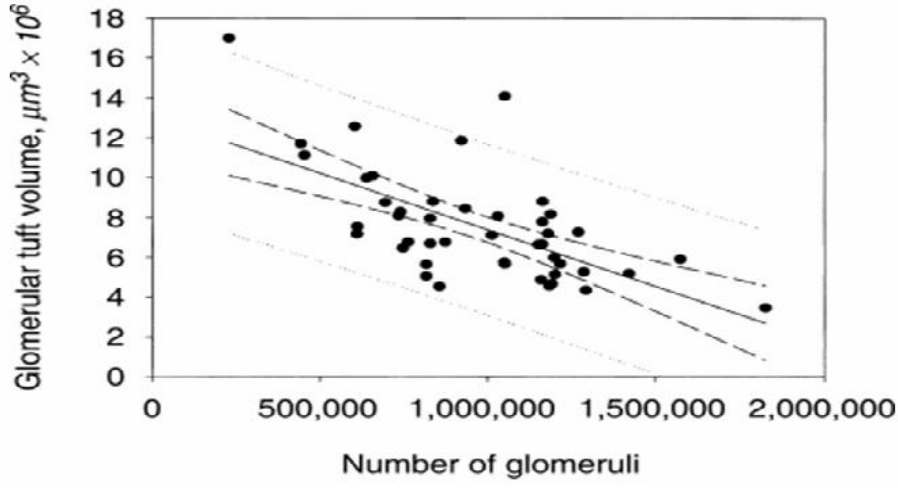

Figure 5. Relationship between total glomerular number and mean glomerular volume in adults.

20 years.[46]

Abitbol et al reported the long-term follow-up of pre-term infants with extremely LBW, who had consultations for kidney problems in the neonatal period.[47,48] Nine of 20 patients showed compromised kidney function.[48] Prominent risk factors for progression were urinary protein/creatinine >0.6 at one-year of age, serum creatinine >53 μmol/L (>0.6 mg/dL) at one-year of age, and a tendency to obesity with BMI >85th percentile.[47]

When kidney mass is reduced, sodium excretion through the kidney becomes less. Subsequently, blood pressure rises in the systemic arterial circulation and glomerular capillaries, thus increasing single-nephron glomerular filtration rate and promoting salt and water excretion.[49] This ongoing persistent elevation in glomerular capillary hydraulic pressure leads to the development of focal and segmental glomerular sclerosis, with further loss of nephrons. This results in a continuous vicious cycle of hypertension and progressive nephron loss leading to CKD.[50,51]

Relevance and Future Landscape

Prenatal programming has more potential significance in developing countries, where birth weights, compatible with smaller adult stature, are lower than in western populations, where IUGR is often superimposed, and where improving infant mortality is allowing lower birth weight infants to increasingly survive to adult life. This is further enhanced by the wide spread of NCDs, especially obesity.

In Oman, we have found that almost 1% of the population aged ≥40 years have severe renal failure, 9% have moderate renal failure and 29% have mild renal failure.[52-54] LBW, prematurity and CKD affects more disadvantaged populations and ethnic minorities and therefore, causes a disproportionate burden on the poor. Kidney disease is, therefore, a global public health priority. Given the very high individual and societal costs of treatment, prevention is the most effective strategy to sustainably address the growing global burden of kidney disease.[55,56] Hence, there is a need for various levels of intervention from a population health perspective point of view. Primary level of intervention includes good maternal care even before conception, with good maternal and fetal baby prenatal care. Secondary level of care is at the time of peridelivery with multidisciplinary care of LBW and premature babies to avoid further compromise of various organ functions such as kidney. Third level of care is at the postnatal stage with life screening programs to enable early detection of various risk factors. Fourth level of care is to manage as early as possible any risk factors or early end-organ deterioration.

Conclusion and Significance

LBW predisposes to increased risk for non-communicable diseases in adult life.[57-62] Studies highlighting regional distribution or geographical dispersal of LBW and prematurity are of paramount importance to identify risk factors and focus implementation strategies. The progressive improvement of health-care for mothers and babies would lead to survival of very LBW and prematurity into adult life. It would be prudent, to adopt policies of intensified life surveillance of lower birth weight people. Early detection of organ dysfunction is the best strategy to delay the progression of CKD. The earliest risk factor to be noticeable in postnatal life is birth weight and gestational age, which can be easily sought out by any health worker.

Conflict of interest: None declared.

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