

Obesity and Transplantation

Lavjay Butani *

Chief Pediatric Nephrology, University of California, Davis, USA

*Corresponding author: Lavjay Butani, Professor of Pediatrics, Chief Pediatric Nephrology, University of California, Davis, 2516 Stockton Blvd, Room 348, Sacramento, CA 95817, USA, Tel: 916-734-8118; Fax: 916-734-0629; E-mail: lbutani@ucdavis.edu

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Editorial

The obesity epidemic continues unabated, especially in children and adolescents, in spite of extensive multidisciplinary approaches to curb this societal scourge [1]. An elevated body mass index (BMI), especially leading to an overweight or obese body habitus, has been clearly linked to the development and progression of chronic kidney disease (CKD) in adults [2]. This is due to the higher prevalence of diabetes and hypertension, but also likely due to the inflammation that can be induced by adipose tissues [3]. Further compounding this problem is the higher morbidity and mortality that is seen in once these patients develop severe enough CKD to need a renal transplant; overweight and obese transplant recipients are at higher risk of postoperative wound infections, and also delayed graft function, leading to lower long-term graft survival [4]. Moreover, the risk of post-transplant diabetes is also increased in obese transplant recipients, further adding fuel to the fire. All of these risks are further worsened in the most severely obese [5]. In the post-transplant setting, the reason for the higher incidence of delayed graft function and worse graft survival are not entirely clear. Whether these are related to technical issues pertaining to the surgical procedure, to the effect of hypertension and post-transplant diabetes, or directly from the

inflammatory milieu induced by obesity, remains to be elucidated. One can hypothesize that all of these factors play a role. In addition to ongoing attempts to tackle obesity in general and preventing the development of an obese body habitus, trying to tease out the precise pathomechanism might help identify how best to target approaches to improve graft and patient survival.

References

1. Ogden CL (2016) Trends in obesity prevalence among children and adolescents in the United States. *JAMA* 315: 2292-2299.
2. William G, Herrington WG (2017) Body-mass index and risk of advanced chronic kidney disease: Prospective analyses from a primary care cohort of 1.4 million adults in England.
3. Butani L (2016) Preliminary report of inflammatory markers, oxidative stress, and insulin resistance in adolescents of different ethnicities. *Metab Syndr and Relat Disord* 14: 3.
4. Von-During ME (2017) Visceral fat is strongly associated with post-transplant diabetes mellitus and glucose metabolism 1 year after kidney transplantation. *Clin Transplant* 31: e12869.
5. Kanthawar P (2016) Kidney transplant outcomes in the super obese: A national study from the unos dataset. *Wor J Surg* 40: 2808-2815.