



Simultaneous Liver Kidney Transplantation

Kirk B. Russ¹ and Ashwani K. Singal^{2*}

¹Internal Medicine Residency Program, University of Alabama at Birmingham, USA

²Division of Gastroenterology & Hepatology, University of Alabama at Birmingham, USA

*Corresponding author: Ashwani K. Singal MD, MS, FACP, Division of Gastroenterology & Hepatology, University of Alabama at Birmingham, AL, 1808 7th Ave S, BDB 351, USA, Tel: 205-934-5623, E-mail: aksingal@uab.edu

Since the introduction of model for end-stage liver disease (MELD) score in 2002 for listing patients for liver transplantation, frequency and proportion of simultaneous liver kidney (SLK) transplantation has increased by over 300% [1]. This is mainly due to incorporation of renal function and serum creatinine as one of the major factors in determining the MELD score [2]. Renal insufficiency occurs in about 20-30% of patients with cirrhosis [3]. For good post-transplant outcomes, it then becomes crucial which patients would recover renal function after receiving liver transplant alone (LTA) and may not need SLK transplantation [4]. Current recommendations for allocating SLK transplantation among patients with cirrhosis who have renal insufficiency are: a) chronic kidney disease with estimated glomerular filtration rate (eGFR) ≤ 30 mL/min, proteinuria > 3 g/d, or $> 30\%$ glomerulosclerosis or interstitial fibrosis and b) acute kidney injury with requirement of dialysis and/or sustained eGFR < 25 mL/min for > 6 weeks [5]. However, these criteria on SLK allocation are based on consensus of experts and are without strong evidence behind them [5]. This is due to lack of objective criteria or biomarkers to distinguish renal insufficiency from hepatorenal syndrome (HRS) from intra-renal or mixed pathology. Moreover, about 25% of HRS type 1 patients may not recover renal function and remain dialysis dependent after receiving LTA [6]. Recently, data are emerging on the accuracy of urinary and/or serum biomarkers such as neutrophil gelatinase-associated lipocalin (NGAL), interleukin-18 (IL-18), kidney injury molecule-1, liver-type fatty acid binding protein (L-FABP) to distinguish acute tubular necrosis from HRS [7]. More prospective data are needed with well-designed studies to define whether these biomarkers would be useful in accurate prediction of renal function recovery among cirrhosis patients after receiving LTA. There seems to be light at the end of tunnel with hope of ability to appropriately allocate SLK transplantation to patients with cirrhosis and renal insufficiency, with optimal utilization of donor kidneys, which are already in scarcity.

Reference

1. Singal AK, Salameh H, Kuo YF, Wiesner RH (2014) Evolving frequency and outcomes of simultaneous liver kidney transplants based on liver disease etiology. *Transplantation* 98: 216-221.
2. Kamath PS, Wiesner RH, Malinchoc M, Kremers W, Therneau TM, et al. (2001) A model to predict survival in patients with end-stage liver disease. *Hepatology* 33: 464-470.
3. Ginès P, Schrier RW (2009) Renal failure in cirrhosis. *N Engl J Med* 361: 1279-1290.
4. Gonwa TA, McBride MA, Anderson K, Mai ML, Wadei H, et al. (2006) Continued influence of preoperative renal function on outcome of orthotopic liver transplant (OLT) in the US: where will MELD lead us? *Am J Transplant* 6: 2651-2659.
5. Eason JD, Gonwa TA, Davis CL, Sung RS, Gerber D, et al. (2008) Proceedings of Consensus Conference on Simultaneous Liver Kidney Transplantation (SLK). *Am J Transplant* 8: 2243-2251.
6. Marik PE, Wood K, Starzl TE (2006) The course of type 1 hepato-renal syndrome post liver transplantation. *Nephrol Dial Transplant* 21: 478-482.
7. Belcher JM, Garcia-Tsao G, Sanyal AJ, Bhogal H, Lim JK, et al. (2013) Association of AKI with mortality and complications in hospitalized patients with cirrhosis. *Hepatology* 57: 753-762.