

ics in treating hepatic encephalopathy and that the use of oral antibiotics carries certain risks. However, a Cochrane review found that antibiotics were superior to nonabsorbable disaccharides in improving hepatic encephalopathy.<sup>1</sup> Furthermore, in a recent multicenter, randomized, placebo-controlled trial (whose findings are currently available only in abstract form),<sup>2</sup> among patients who had cirrhosis and hepatic encephalopathy that was not controlled with lactulose alone, the oral administration of rifaximin at a dose of 550 mg twice a day was more effective than placebo during a 6-month period. Thus, there is some evidence that antibiotics are beneficial in the treatment of hepatic encephalopathy.

Michael R. Lucey, M.D.

University of Wisconsin School of Medicine and Public Health  
Madison, WI  
mrl@medicine.wisc.edu

Philippe Mathurin, M.D., Ph.D.

Hôpital Claude Huriez  
Lille, France

Timothy R. Morgan, M.D.

VA Long Beach Healthcare System  
Long Beach, CA

1. Als-Nielsen B, Gluud LL, Gluud C. Nonabsorbable disaccharides for hepatic encephalopathy. *Cochrane Database Syst Rev* 2004;2:CD003044.

2. Bass N, Mullen K, Sigal S, et al. Rifaximin is effective in maintaining remission in hepatic encephalopathy: results of a large, randomized, placebo-controlled study. *J Hepatol* 2009;50: Suppl 1:S39. abstract.

## Indigenous Australians and Living Kidney Donation

**TO THE EDITOR:** Indigenous Australians comprise 2.5% of the total population of Australia, but like other minority groups worldwide, rates of chronic kidney disease are higher among this indigenous group than among the general population. Transplantation remains the preferred form of renal-replacement therapy, and the use of living donors maximizes transplantation rates with a generally minimal long-term effect on donors.<sup>1</sup> However, the consequences of the use of this strategy in indigenous Australians are unclear, given the likelihood of a family or community history of chronic kidney disease in potential donors.

We performed a retrospective analysis of all living kidney donors from the Northern Territory of Australia since 1985. A total of 50 people (22 of whom were self-reported to be indigenous) were accepted as kidney donors and were evaluated according to standard pretransplantation protocols at the same transplantation center, irrespective of their race or ethnic group. Donors were assessed by a transplantation nephrologist, and each donor was found to have normal kidney function (a calculated glomerular filtration rate [GFR] of >90 ml per minute per 1.73 m<sup>2</sup> of body-surface area), a normal intravenous pyelogram and renal angiogram, normal blood pressure (<140/80 mm Hg), a normal oral glucose-tolerance test, a normal electrocardiogram, and no proteinuria.

Sixteen of 22 indigenous donors (73%) had

medical follow-up over a median period of 16.1 years. At the last follow-up of these carefully selected donors, two (12%) had died, three (19%) required renal-replacement therapy, and the majority had hypertension, diabetes, established proteinuria, and an estimated GFR of less than 60 ml per minute (Table 1). Of 16 nonindigenous donors, only 6 (38%) had an estimated GFR of less than 60 ml per minute; none had other clinically significant coexisting conditions.

Clearly, “standardized assessment” has failed these indigenous donors. Hoy et al.<sup>2</sup> best described the natural history of kidney disease in indigenous Australian communities. The confirmed rates of proteinuria and end-stage kidney disease in our study were significantly worse among indigenous donors than among the general population, and the estimated GFR at the last follow-up provides support for this concern (although estimated GFR validation studies involving indigenous Australians have not yet been completed).

Our present study also raises the questions of whether donors with any family history of kidney disease should be accepted and of whether more extensive testing before donation would avert these adverse outcomes. A kidney biopsy performed before donation may be of benefit, since low nephron endowment and glomerulomegaly have been described,<sup>5</sup> but this procedure increases donor risk. The heterogeneity of indigenous

**Table 1. Characteristics of Living Kidney Donors and the General Population among Indigenous and Nonindigenous Australians from the Northern Territory.\***

Variable	Indigenous Population		Nonindigenous Population		P Value
	Kidney Donors	General Population†	Kidney Donors	General Population‡	
<b>Before donation</b>					
Total donors — no.	22		28		
Donors identified by name — no./total no. (%)	19/22 (86)		24/28 (86)		
Female sex — no./total no. (%)	9/19 (47)		12/24 (50)		
Age at donation — yr					
Median (range)	35.1 (19.1–60.2)		44.9 (19.6–67.6)		
<b>Pretransplantation workup</b>					
Medical records available — no.§	15		21		
BMI					
Median (range)	24.3 (21.3–30.3)	23.2 (13.6–43.5)	23.0 (20.9–26.5)		
Men with BMI >25 — % of population				45	
Women with BMI >25 — % of population				26	
Blood pressure >140/80 mm Hg — no. of patients/total no.	0/15		0/21		
Normal OGTT — no. of patients/total no.	15/15		21/21		
Normal electrocardiogram — no. of patients/total no.	15/15		21/21		
24-hr urine volume					
Median (range) — liter	1.65 (1.53–2.1)		1.75 (1.24–2.67)		
No. of patients	12		3		
24-hr urine protein <0.22 g/24 hr					
Median (range)	0.05 (0.03–0.15)		0.05 (0.04–0.08)		
Urinary albumin-to-creatinine ratio <3.5 — no. of patients/total no.	—		18/18		
Normal urine microscopy — no. of patients/total no.	15/15		18/18		
GFR — ml/min					
Median (range)	98 (90–120)		101 (95–124)		
No. of patients¶	15		21		
<b>Last follow-up</b>					
Total donors with medical follow-up — no./total no. (%)	16/22 (73)		16/28 (57)		
Donor alive but no medical follow-up — no./total no. (%)	2/22 (9)		5/28 (18)		
Donor status unknown — no./total no. (%)	1/22 (5)		3/28 (11)		
Duration of medical follow-up after donation — yr					
Median (range)	16.1 (1.27–20.2)		6.37 (2.54–21.2)		
Age at last medical follow-up — yr					
Median (range)	41.2 (30.9–79.0)		52.6 (27.0–66.1)		
Estimated life expectancy — yr					
Men		59		77	
Women		65		82	

Deaths due to cardiac causes and sepsis						
% of population			5.9		0.22	
No./total no. (% [95% CI])	2/16 (12 [1.6–38])			0.34		0.99
Time to death after donation — yr						
Range	14.0–18.5					
Age at death — yr						
Range	37–79					
Serum creatinine — $\mu\text{mol/liter}$						
Median (range)	142 (95–271)				101 (85–229)	
Estimated GFR — no./total no. (%)**						
≥60 ml/min/1.73 m <sup>2</sup>	3/16 (19)				10/16 (62)	
<60 ml/min/1.73 m <sup>2</sup>	10/16 (62)				6/16 (38)	
<10 ml/min/1.73 m <sup>2</sup> (end-stage kidney disease)	3/16 (19)		12/825 (1.5)	<0.001	0	115 pmp (0.012)
Time to renal-replacement therapy — yr						
Median (range)	17.5 (9.3–21.5)					
Blood pressure >140/80 mm Hg						
% of population			34–54			30
No./total no. (% [95% CI])	8/16 (50 [25–75])			0.76	1/16 (6 [0.16–30.0])	0.05
Median (range)					6	
Diabetes mellitus						
% of population			17–30			8
No./total no. (% [95% CI])	9/16 (56 [30–80])			0.04		0.24
Proteinuria (urinary albumin-to-creatinine ratio >3.5 mg/mmol)						
% of population			15–28			8
No./total no. (% [95% CI])	13/16 (81 [54–96])			<0.001	1/16 (6 [0.16–30.0])	0.81

\* Pretransplantation workup protocols were identical, regardless of race or ethnic group. Donor outcomes were compared with expected population rates from available epidemiologic studies. Indigenous donors showed a trend toward poorer outcomes as compared with the general population, although because of the small sample size (and unconfirmed or missing data), confidence intervals are large. Body-mass index (BMI) is the weight in kilograms divided by the square of the height in meters. CI denotes confidence interval, GFR glomerular filtration rate, OGTT oral glucose-tolerance test, and pmp per million population.

† Data are from Hoy et al.<sup>2</sup> and Hoy et al.<sup>3</sup>

‡ Data are from Begg et al.<sup>4</sup>

§ Documentation was available at the time of writing; medical records of four indigenous and three nonindigenous donors were destroyed.

|| The GFR was calculated with the use of the Cockcroft–Gault formula.

¶ The measurements of serum creatinine exclude three donors with end-stage kidney disease.

\*\* The estimated GFR was calculated with the use of the Modification of Diet in Renal Disease (MDRD) four-variable equation. Three indigenous donors underwent hemodialysis, and one patient subsequently received a deceased-donor kidney transplant.

peoples within and outside Australia limits the ability to generalize these observations; donor outcomes in other minority groups have not, to our knowledge, been reported. Until comprehensive, prospective outcome data on kidney donors from different minority groups are available, the extrapolation of modest long-term risk to all donors may do a disservice to the integrity of the living-donor kidney program.

Natasha M. Rogers, M.D.

Queen Elizabeth Hospital  
Adelaide, SA, Australia

Paul D. Lawton, M.D.

Royal Darwin Hospital  
Darwin, NT, Australia

Matthew D. Jose, M.D., Ph.D.

Menzies Research Institute  
Hobart, TAS, Australia

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1. Ibrahim HN, Foley R, Tan L, et al. Long-term consequences of kidney donation. *N Engl J Med* 2009;360:459-69.
2. Hoy WE, Wang Z, VanBuynder P, Baker PR, McDonald SM, Mathews JD. The natural history of renal disease in Australian Aborigines. 2. Albuminuria predicts natural death and renal failure. *Kidney Int* 2001;60:249-56.
3. Hoy WE, Kondalsamy-Chennakesavan S, Wang Z, et al. Quantifying the excess risk for proteinuria, hypertension and diabetes in Australian Aborigines: comparison of profiles in three remote communities in the Northern Territory with those in the AusDiab study. *Aust N Z J Public Health* 2007;31:177-83.
4. Begg S, Vos T, Barker B, Stevenson C, Stanley L, Lopez A. The burden of disease and injury in Australia 2003. Canberra: Australian Institute of Health and Welfare, 2003. (Accessed September 17, 2009, at <http://www.aihw.gov.au/publications/index.cfm/title/10317>).
5. Hoy WE, Hughson MD, Singh GR, Douglas-Denton R, Bertram JF. Reduced nephron number and glomerulomegaly in Australian Aborigines: a group at high risk for renal disease and hypertension. *Kidney Int* 2006;70:104-10.

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#### CORRECTIONS

Telaprevir with Peginterferon and Ribavirin for Chronic HCV Genotype 1 Infection (April 30, 2009;360:1827-38). In the disclosure statement (page 1837), the disclosure information for Dr. Jacobson should have read, "Dr. Jacobson, consulting fees and grant support from Vertex, Schering-Plough, and Roche, and lecture fees from Schering-Plough." The article has been corrected at NEJM.org.

The Hypertension Paradox — More Uncontrolled Disease despite Improved Therapy (August 27, 2009;361:878-87). In Table 1 (page 879), "β-Adrenergic-receptor agonists" should have read, "β-Adrenergic-receptor antagonists." In the text, in the second sentence of the second paragraph under the heading "Increase in the Prevalence of Hypertension" (page 883), "blood pressures of 120–130/80–89 mm Hg" should have read, "blood pressures of 120–139/80–89 mm Hg." The article has been corrected at NEJM.org.

Case 14-2009: A 36-Year-Old Man with Chest Pain, Dysphagia, and Pleural and Mediastinal Calcifications (April 30, 2009;360:1886-95). The disclosure statement (page 1894) should have read, "Dr. Mark reports serving as an expert witness at trial for people who had diffuse malignant mesothelioma and who believed that they had been harmed by asbestos." The article has been corrected at NEJM.org.

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