

# FREQUENCY OF T-CELL MEDIATED REJECTION IN NEW ONSET DIABETES AFTER KIDNEY TRANSPLANTATION

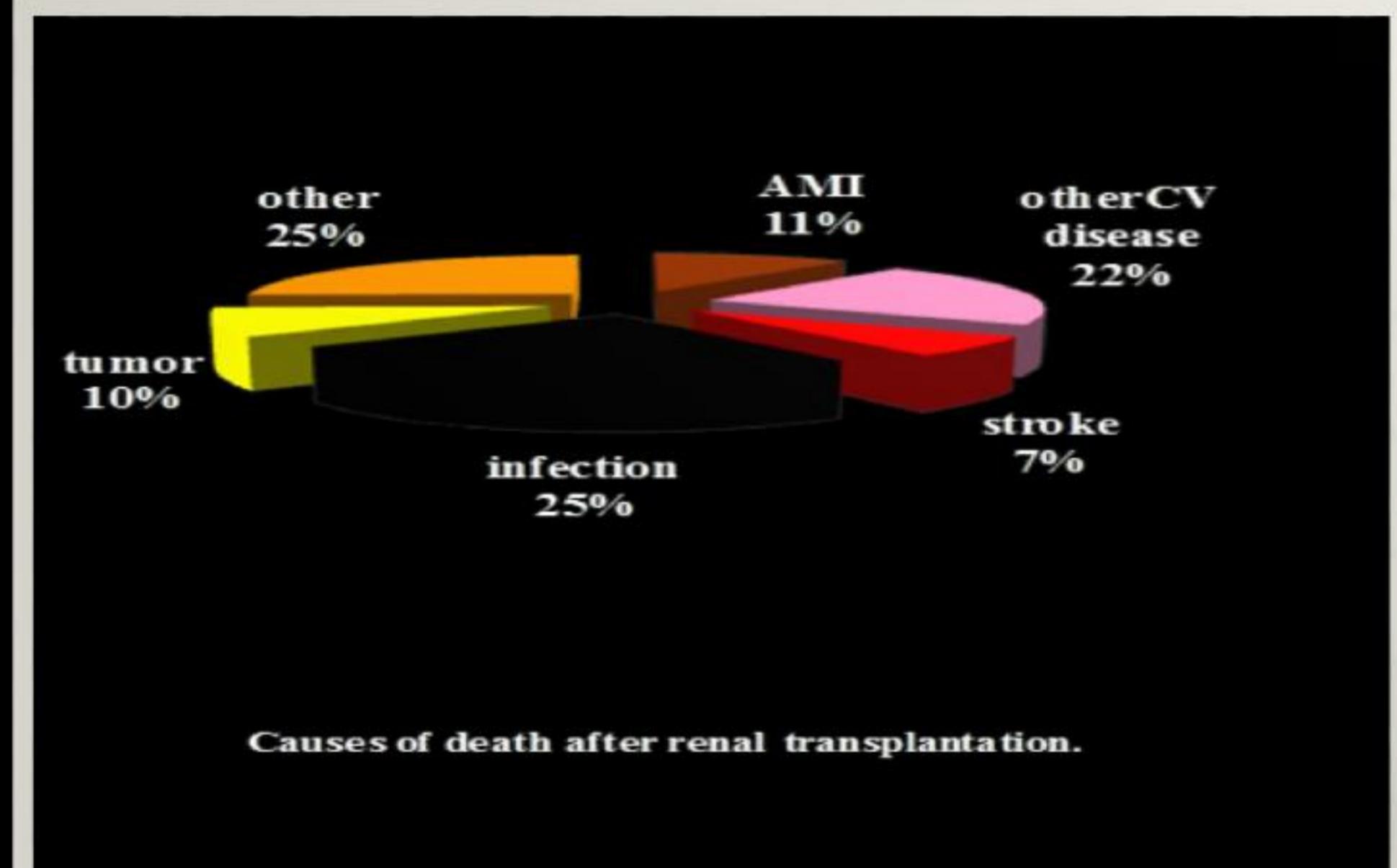
Borda B.<sup>1</sup>, Szederkényi E.<sup>1</sup>, Hódi Z.<sup>1</sup>, Iványi B.<sup>2</sup>,  
Kemény É.<sup>2</sup>, Keresztes Cs.<sup>3</sup>, Lázár Gy.<sup>1</sup>



University of Szeged, Faculty of Medicine<sup>1</sup> Department of Surgery, <sup>2</sup>Institute of Pathology, <sup>3</sup>Department for Medical Translation and Communication Szeged, Hungary

## INTRODUCTION

New-onset diabetes after transplantation (NODAT) is one of the most common complications following kidney transplantation. The diagnosis of NODAT is often late or missed; therefore, it impairs the implanted renal allograft. Not only does untreated NODAT negatively influence the allograft, but it increases the risk of cardiovascular diseases and death. NODAT is the same risk factor of cardiovascular diseases as diabetes diagnosed before the transplantation.



## PATIENTS AND METHODS

University of Szeged, Department of Surgery, Hungary (between 2006 and 2013)

### Excluded from the study:

- diabetes mellitus before the transplantation
- younger than 18 years
- living donor
- died during the study
- "0" biopsy was not normal

### Randomised

- CsA n = 95
- Tac n = 102

### morphology:

*2007 modification of the Banff classification*

- Normal
- Antibody mediated changes
- Borderline changes
- T-cell mediated rejection
- Interstitial fibrosis/tubular atrophy
- Other

### function:

- se. creatinine ( $\mu\text{mol/L}$ )
- eGFR- CKD-EPI formula ( $\text{mL/min}/1.73\text{m}^2$ )

### 1 year after kidney transplantation

OGTT (75g glucose, 120. min  $\geq 11.1$  mmol/L)

- ✓ N – normal
- ✓ IFG/IGT – increased fasting glucose/impaired glucose tolerance
- ✓ NODAT – new - onset after kidney transplantation

## RESULTS

	CsA (n = 95)	Tac (n = 102)	n = (197)
NODAT	11 (12%)	24 (24%)	35 (17%)
IFG/IGT	17 (18%)	19 (18%)	36 (18%)
N	67 (70%)	59 (58%)	126 (65%)

\* NODAT, CsA vs Tac p = 0.021

	NODAT	IFG/IGT	N	P value
eGFR ( $\text{mL/min}/1.73\text{m}^2$ )	37.7±13.1	39.51±16.24	40.73±13.06	0.583
se. creatinine ( $\mu\text{mol/L}$ )	188.5±58.7	186.50±62.70	184.69±122.60	0.236

## CONCLUSIONS

**Diabetes not diagnosed and treated in time not only damages the graft but increases the cardiovascular risk as well. In case of kidney recipients, long term survival of the graft may be increased, and the cardiovascular risk may be decreased with diagnosing and treating NODAT in time.**

## REFERENCES

1. Vincenti F, Friman S, Scheuermann E, et al: Results of an International, Randomized Trial Comparing Glucose Metabolism Disorders and Outcome with Cyclosporine Versus Tacrolimus. Am J Transplant 7: 1506, 2007
2. Borda B, Szederkényi E, Lengyel C et al: Functional and histopathological changes in renal transplant patients with new-onset diabetes and dyslipidemia. Transplant Proc 43: 1254, 2011
3. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 35(Suppl. 1): S64, 2012
4. Racusen L, Colvin RB, Solez K et al: The Banff 97 working classification of renal allograft pathology. Kidney Int 55: 713, 1999
5. Borda B, Lengyel Cs, Szederkényi E et al. Post-transplant diabetes mellitus – Risk factors and effects on the function and morphology of the allograft. Acta Phys Hung 99: 206, 2012
6. Helanterä I, Ortiz F, Räisänen-Sokolowski A et al: Impact of glucose metabolism abnormalities on histopathological changes in kidney transplant protocol biopsies. Transpl Int 4: 374, 2010

Dr Bernadett Borda PhD.  
University of Szeged, Faculty of Medicine,  
Department of Surgery  
Email: borda.bernadett@med.u-szeged.hu

