SR TR

SCIENTIFIC REGISTRY OF TRANSPLANT RECIPIENTS

Introduction

- Cardiovascular disease is a leading cause of nonimmunologic morbidity and mortality after kidney transplant (KTx). To date, much attention has focused on ischemic heart disease and heart failure in this population.
- In recent years, **pulmonary** hypertension (P-HTN) has gained recognition as a clinically important cardiovascular condition among patients suffering from chronic kidney disease, including **KTx** candidates and recipients.
- The incidence and mortality implications of P-HTN after KTx have not been described in large, national samples.

Methods

- We examined a linkage of Scientific Registry of Transplant **Recipients (SRTR)** data with Medicare claims to investigate P-HTN diagnoses among Medicareinsured KTx recipients in 2000-2016 (**N=59,610**).
- We identified diagnoses of "primary" and "secondary" P-HTN based on International Classification of Diseases, Clinical Modification (ICD-CM) diagnostic codes (ICD-CM-9 through October 2015, then **ICD-CM-10**) on billing claims.
- **Cox regression** was used to identify independent correlates of P-HTN (adjusted hazard ratio, 95%) LCLaHR 95%UCL), and to examine P-HTN diagnoses as time-dependent mortality predictors, stratified by baseline clinical factors.

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Incidence, Clinical Correlates, and Outcomes of Pulmonary Hypertension After Kidney Transplantation: Analysis of Linked U.S. **Registry and Medicare Billing Claims**

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Results

- At **3 years** post-KTx, P-HTN was diagnosed in **7.0%** of patients without P-HTN in the year before KTx, but in **45.8%** of those with **P**-HTN in the year before KTx.
- The incidence of new onset P-HTN was higher with baseline factors including older age (aHR for age <u>>60 vs <18-30, _{2.46}3.12_{3.95}), obesity</u> (aHR, _{1.04}1.15 _{1.28}), **limited** functional status (aHR, 1.091.251.43), coronary artery disease (aHR, 1.18 1.34_{1.52}), chronic obstructive lung disease (aHR, 1.571.972.48), longer pre-KTx dialysis duration (aHR for >5 yrs vs preemptive, $_{1,30}$ 1.47 $_{1,66}$), dialysis modality (aHR for hemo- vs peritoneal, $_{1.28}$ 1.50 $_{1.77}$), and lower organ quality (aHR Kidney Donor Profile Index >85 vs 20-25, 1 17 1.32 1.48**)**-



Figure. Variation in associations of P-HTN after kidney transplant with subsequent morality risk, according to baseline clinical factors.

Results

P-HTN diagnosis was associated with **3.0**fold increased risk of subsequent **mortality**, with relative risk being highest in young recipients, those of nonwhite/non-black race, those with kidney failure due to glomerulonephritis or polycystic kidney disease, those without pretransplant P-HTN, and those who underwent transplant in more recent years (Figure).

Limitations

- Retrospective design limits ability to objectively confirm clinically coded P-HTN diagnoses.
- The ICD- diagnostic scheme does not discriminate cause of P-HTN.
- Clinical covariates are recorded with limited granularity in the registry.

Conclusions

- Clinical diagnosis of P-HTN after KTx is correlated with increased risk of subsequent mortality.
- More work is needed to **refine diagnostic** and management strategies to improve outcomes in KTx recipients who develop this challenging complication.