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Hello, my name is Tony Okoro and I'd like to speak to you today on behalf of Bayer Pharmaceuticals about Kerendia® (finerenone), which is a non-steroidal mineralocorticoid receptor antagonist, indicated to reduce the risk of sustained eGFR decline, end stage kidney disease, cardiovascular death, non-fatal myocardial infarction, and hospitalization for heart failure in adult patients with chronic kidney disease (CKD) associated with type 2 diabetes (T2D).[1]

Kerendia® blocks MR mediated sodium reabsorption and MR overactivation in both epithelial (e.g., kidney) and nonepithelial (e.g. heart, and blood vessels) tissues. MR overactivation is thought to contribute to fibrosis and inflammation.[1]

To begin, I'd like to highlight the large unmet need of these patients with CKD associated with T2D. CKD increases commercial and Medicare patients' costs exponentially with each advancing stage of the disease.[2] In addition, kidney damage, marked by increased albuminuria, is a cost driver in CKD associated with T2D. In a retrospective claims analysis of 23,235 adults with T2D conducted between 2004 and 2014, patients with T2D and high or very high albuminuria had significantly higher risks of progressing to a more severe disease stage, receiving dialysis, nephropathy-related inpatient admissions, emergency department and outpatient services.[3] Also, in an analysis of 2006 adults with T2D from the NHANES data set from 2007-2012, patients with CKD associated with T2D reported nearly 2x greater rates of myocardial infarction and approximately 3.5x greater rates of congestive heart failure than patients with T2D alone.[4]

Diabetes is a major cause of CKD in the US. In fact, approximately 40% of patients with T2D develop CKD.[5] However, CKD goes largely undiagnosed, in earlier stages.[6,7,8] Based on developments in the CKD landscape and implementation of new quality measures,[9] goals for the optimal patient journey should include a shorter undiagnosed period, earlier diagnosis and treatment directed at the drivers of disease progression.[10-12]

Since launch, some new clinical data has become available that I'd like to share with the committee. FIGARO-DKD is the second phase III trial and studied time to CV death, non-fatal MI, non-fatal stroke, or HHF. The time to the CV composite outcome was reduced with Kerendia® (12.4%) vs placebo (14.2%) (HR 0.87; 95% CI 0.76-0.98). The secondary kidney composite endpoint as measured by time to first onset of kidney failure, sustained ≥57% decrease in eGFR from baseline over ≥4 weeks, or renal death, was lower with Kerendia® than placebo, however the difference was not statistically significant.[13] In addition, FIDELITY was a prespecified, meta-analysis of the phase III FIGARO-DKD and FIDELIO-DKD studies comprising over 13,000 patients with CKD associated with T2D. At baseline, patients had a range of CKD (stages 1-4), with a mean eGFR of 58 mL/min/1.73 m^2, and 99% of patients were treated with a maximum labeled tolerated dose of an ACEi or ARB. The prespecified meta-analysis analysis showed positive results in two composite endpoints. First, Kerendia® demonstrated a 14% reduction in the risk of CV composite endpoint as measured by time to first onset of CV death, non-fatal MI, non-fatal stroke, or hospitalization for heart failure compared with placebo (HR 0.86; 95% CI 0.78–0.95). The reduction in the CV composite outcome was primarily driven by hospitalization for heart failure (3.9% Kerendia® vs 5% placebo) (HR 0.78, 95% CI 0.66-0.92). Second, a 23% reduction in the risk of kidney composite endpoint as measured by time to first onset of kidney failure, sustained ≥57% decrease in eGFR from baseline over ≥4 weeks, or renal death, compared with placebo (HR 0.77; 95% CI 0.67-0.88). Lastly, the analysis showed a 32% greater reduction in UACR between baseline and month 4 vs. placebo (ratio of LS mean 0.68; 95% CI 0.65-0.70). Regarding safety, hyperkalemia occurred in 14% of patients who received Kerendia® vs 6.9% with placebo.[7, 13]

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