



Oral Communications 8 Kidney Transplant, Anemia, and MBD

December 6, 2025 (Saturday) 11:00~12:30

Venue : Room 8 (602)

Chair(s)

Sunny Sze Ho Wong, Ya-Chung Tian

11:00-11:09

Distinct Subtypes of Kidney Transplant-associated Urothelial Carcinomas Harboring BK Polyomavirus Integration and Aristolochic Acid Mutational Signatures
APCN20251137

Ya-Chung Tian

Kidney Research Center and Department of Nephrology, Linkou Chang Gung Memorial Hospital

11:09-11:18

Therapeutic Targeting of the IL-6/IL-17 Amplifier Loop in Fibroblasts: Translational Insights from Chronic Antibody-Mediated Rejection in Kidney Transplant Recipients
APCN20251085

Mantabya Kumar Singh

Department of Nephrology, Sanjay Gandhi Post Graduate Institute of Medical Sciences (SGPGIMS)

11:18-11:27

Endometrial Regeneration Cell-Derived Exosomes Carrying siCD3 Inhibit Kidney Allograft Rejection through Suppression of α -2,6 Sialylation
APCN20250750

Yini Xu

General Hospital of Tianjin Medical University

11:27-11:36

Impact of Metabolic Variability of Tacrolimus on Post-Transplant Clinical Outcomes in Kidney Transplant Recipients
APCN20250135

Woo Yeong Park

Division of Nephrology, Department of Internal Medicine, Keimyung University School of Medicine, Keimyung University Dongsan Hospital

11:36-11:45

The Role of Mesenchymal Stem Cells in Treating Diabetic Kidney Disease: Immunomodulatory Effects and Kidney Regeneration
APCN20250457

Po-Jen Hsiao

Division of Nephrology, Department of Internal Medicine, Taoyuan Armed Forces General Hospital





11:45-11:54	Efficacy and Safety of Rabbit versus Equine Anti-Thymocyte Globulin as Induction Therapy in Kidney Transplantation with Modern Immunosuppression: A Single-Center Retrospective Cohort Study APCN20251052	Ramalakshmi Thullimalli Department of Nephrology, Virinchi People's Hospital
11:54-12:03	Mortality, Malignancy, And CMV Infection Risks With mTOR Inhibitors Versus Mycophenolate in Kidney Transplantation: A Matched Real-World Analysis APCN20250039	Naveen Department of General Medicine, Gulmi Durbar Basic Hospital
12:03-12:12	Multi-Omics Machine Learning Model Predicts Long-Term Graft Outcome Based on Urinary Renal Progenitor Epigenomic Reprogramming After Kidney Transplantation APCN20250944	Prihantini AI-BioMedicine Research Group, IMCDS-BioMed Research Foundation
12:12-12:21	Prevalence And Correlates of Adynamic Bone Disease in Patients With End-Stage Kidney Failure in Singapore APCN20250797	Shuit Siew Kit Department of Renal Medicine, Tan Tock Seng Hospital
12:21-12:30	Interactive Effects of Hemoglobin and Iron Deficiency on kidney prognosis and Major Adverse Cardiovascular Events: A Retrospective Cohort Study APCN20251046	ZHU XiHuan Qi-huang Chinese Medicine School, Beijing University of Chinese Medicine



Oral Communications : Kidney Transplant, Anemia, and MBD

Abstract Submission No. : APCN20251137

Distinct subtypes of kidney transplant-associated urothelial carcinomas harboring BK polyomavirus integration and aristolochic acid mutational signatures

YA-CHUNG TIAN^{1,2}; Jiun-Wen Wang^{1,2}; Yi-Jung Li^{1,2}; Tzu-Hao Huang³; Hsin-Hsu Wu^{1,2}; Hsu-Han Wang⁴; Yang-Jen Chiang⁴; See-Tong Pang⁴; Abner Herbert Lim⁵; Cedric Chuan Young Ng⁵; Wei Liu⁵; Jing Yi Lee⁵; Jing Han Hong⁶; Bin Tean Teh^{7,8}; Jason Yongsheng Chan^{5,7}

¹ Kidney Research Center and Department of Nephrology, Linkou Chang Gung Memorial Hospital, Taiwan

² Department of Medicine, Chang Gung University, Taiwan

³ Department of Urology, Taipei Veterans General Hospital, Taiwan

⁴ Department of Urology, Linkou Chang Gung Memorial Hospital, Taiwan

⁵ Cancer Discovery Hub, National Cancer Centre Singapore, Singapore and

⁶ Cancer and Stem Cell Biology Programme, Duke-NUS Medical School, Singapore

⁷ Oncology Academic Clinical Program, Duke-NUS Medical School, Singapore

⁸ Laboratory of Cancer Epigenome, National Cancer Centre Singapore, Singapore

Abstract

Urothelial carcinoma (UC) is the most prevalent cancer of kidney transplant recipients in some regions. The integration of BK polyomavirus (BKPyV) genome into the human genome is observed in some cases of UC. The prevalence of aristolochic acid (AA) mutational signatures in urothelial carcinoma is high in the general population of Taiwan. To better understand the mechanisms of UC carcinogenesis in kidney transplant recipients, 19 UC specimens were analyzed by whole genome sequencing (WGS). BKPyV genome integration into human genome was observed in 6 of 19 (32%) UC specimens. The total number of integrations sites in these 6 UC specimens was 21, with chromosome 8 and 12 exhibiting a higher frequency of integration sites. Of these 21 sites, 76% were located in introns, 19% in intergenic regions, and only one breakpoint was identified in an exon. PCR analysis and Sanger sequencing confirmed BKPyV genome integration. In addition, the WGS result showed a high prevalence of the AA mutational signature in these UC specimens (84%). High percentage of UC had mutated genes involving chromatin modification [KMT2C (68%), KMT2D (53%), CREBBP (42%), and ARID1A (42%)], the cell-cycle pathway [TP53 (58%), NF1 (47%), and ATM (32%)], and oncogenic signaling pathways [ERBB2 (21%), PIK3CA (16%), FGFR3 (11%), and HRAS (11%)]. Interestingly, compared with UC with AA mutational signatures, UC with BKPyV genome integration had far less mutated genes, suggesting different mechanisms of carcinogenesis in these two types of UC. In conclusion, the present study provides evidence of different mechanisms of UC development in Taiwanese kidney transplant recipients.

Keywords : genomics, mutational signatures, aristolochic acid, BK polyomavirus

Oral Communications : Kidney Transplant, Anemia, and MBD

Abstract Submission No. : APCN20251085

Therapeutic Targeting of the IL-6/IL-17 Amplifier Loop in Fibroblasts: Translational Insights from Chronic Antibody-Mediated Rejection in Kidney Transplant Recipients

Mantabya Kumar Singh¹; Mohit Rai²; Vikas Agarwal²; Narayan Prasad¹

¹ Department of Nephrology, Sanjay Gandhi Post Graduate Institute of Medical Sciences (SGPGIMS), Lucknow, India

² Department of Clinical Immunology, Sanjay Gandhi Post Graduate Institute of Medical Sciences (SGPGIMS), Lucknow, India

Abstract

Background and Aims:

Organ transplants are the preferred treatment for end-stage organ failure. Potent and specific immunosuppressive agents have significantly decreased acute allograft rejection following renal transplantation. Chronic antibody-mediated kidney rejection (CABMR) is significant cause of chronic allograft injury. Chronic inflammation is a major cause of late graft loss that is mediated by soluble mediators released by immune and non-immune cells. Fibroblasts, have been implicated in mediating this injury through activation of the IL-6 amplifier loop (IL-6 + IL-17), driven by NFκB and STAT3 signalling pathways. This study investigates the activation of the IL-6 amplifier loop in fibroblasts isolated from renal biopsies of CABMR patients and evaluates the effects of IL-6 and IL-17 inhibition.

Method:

Fibroblasts were cultured from six CABMR patient biopsy samples and treated with anti-IL-6 (100 ng/mL) and anti-IL-17 (0.75 μg/mL), both before and after stimulation with IL-6/sIL-6R (20 ng/μL), IL-17 (50 ng/μL), or combination. IL-6, CCL2, and CCL20 levels were measured in culture supernatants by ELISA. mRNA expression of IL-6, CCL2, CCL20, and SOCS3 was assessed using RT-PCR, protein expression was evaluated by western blot for phosphorylated STAT3 and NFκB p65.

Results:

IL-6 and IL-17 synergistically induced more IL-6, CCL-20 & MCP-1 production from fibroblasts in culture supernatant. Gene expression analysis of IL-6, MCP1, and CCL20 was significantly higher with synergistic activation of IL-6 and IL-17 as compared to either IL-6 or IL-17 alone, while SOCS3 gene expression was downregulated. Our results also suggested that IL-6 Amplifier loop activation induces the NFκB and STAT3 signalling pathway activation in the non-immune cells like fibroblast derived from CABMR patients.

There was a significant reduction in IL-6, CCL20 and MCP-1 concentration in culture supernatant and their m-RNA expression with IL-6 and IL-17 inhibitor together was significantly reduced while SOCS3 gene expression was upregulated. The phosphorylated NFκB and STAT3 proteins expression was also downregulated in the presence of IL-6 and IL-17 inhibitor together.

Conclusion:

In humans after kidney transplantation, IL-6 amplifier activation plays an active role in chronic rejection responses. Inhibition of IL-6 with Anti-IL-6 (Tocilizumab) and inhibition of IL-17 with Anti-IL-17 together reduces markers of tissue injury (IL-6, MCP1, CCL20) and rejection of allografts. so, IL-6 amplifier may be a therapeutic target for Chronic transplant rejection.

Keywords : Chronic antibody-mediated rejection, IL-6 Amplifier loop, CCL2, CCL20, SOCS3, Tocilizumab, Anti-IL-17

Oral Communications : Kidney Transplant, Anemia, and MBD

Abstract Submission No. : APCN20250750

Endometrial Regeneration Cell-derived Exosomes Carrying siCD3 Inhibit Kidney Allograft Rejection through Suppression of α -2,6 Sialylation

Yini Xu^{1,2}; Hao Wang¹; Xiulan Zhao²; Baocun Sun^{1,2}

¹ General Hospital of Tianjin Medical University

² Tianjin Medical University

Abstract

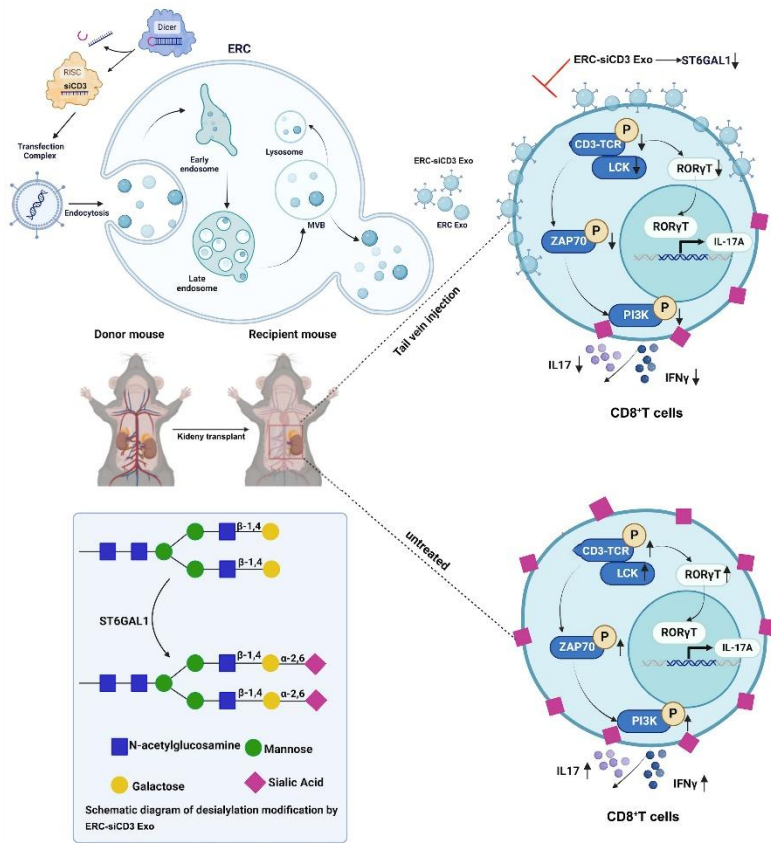
Objective: Kidney transplant rejection is a major component in the poor prognosis of organ transplantation. Due to the multiple complicated mechanisms involved, a novel therapy remains under exploration. Endometrial regenerative cells (ERCs) have been ubiquitously applied to various refractory immune-related diseases, but the role of ERC-derived exosomes (ERC-Exos) in alleviating transplant rejection has not been thoroughly studied. Cluster of differentiation 3 (CD3) plays an important role in regulating immune responses. In this study, we have demonstrated for the first time that ERC-Exo carried with siCD3 attenuated allograft rejection by inhibiting T-cell proliferation and differentiation.

Methods: C57BL/6 mouse recipients receiving bm12 mouse kidney allografts were randomly divided into four groups. Graft pathological changes were evaluated by H&E staining. Splenic immune cell populations were analyzed using flow cytometry. Serum cytokine profiles of recipients were measured by ELISA. The proliferation capacity of CD8+T cell populations was also assessed in vitro. α -2,6-sialylation levels in CD8+T cells were measured by SNA blot.

Results: In vivo, mice treated with ERC-siCD3 Exos achieved significantly prolonged allograft survival. Serum cytokine profiles of recipients were significantly changed in ERC-siCD3 Exo-treated recipients. In vitro, we found that ERC-siCD3 Exos considerably down-regulated the α -2,6-sialyltransferase (ST6GAL1) expression in CD8+T cells, and significantly reduced α -2,6-sialylation levels. Through desialylation modification, ERC-siCD3 Exo therapy significantly decreased CD8+T cell proliferation and inhibited CD8+T cell differentiation into Th1 and Th17 cells while promoting Treg differentiation.

Conclusions: ERC Exo carrying siCD3 reduces the sialic acid connected to α -2,6 at the end of the N-glycan chain on the CD8+T cell surface, increases the number of therapeutic exosomes endocytosed into CD8+T cells and inhibits activation of T-cell receptor signaling pathways, which prolongs allograft survival. This study confirms the feasibility of ERC-derived exosomes as natural carriers combined with gene therapy, which could be used as a potential therapeutic strategy to alleviate allograft rejection.

Keywords : Cardiac allograft rejection, Endometrial regeneration cell-derived exosome, Desialylation modification, T cell receptor signaling pathway



Oral Communications : Kidney Transplant, Anemia, and MBD

Abstract Submission No. : APCN20250135

Impact of Metabolic Variability of Tacrolimus on Post-Transplant Clinical Outcomes in Kidney Transplant Recipients

Woo Yeong Park¹; Yaerim Kim¹; Jin Hyuk Paek¹; Kyubok Jin¹; Seungyeup Han¹

¹ Division of Nephrology, Department of Internal Medicine, Keimyung University School of Medicine, Keimyung University Dongsan Hospital, Daegu, Republic of Korea

Abstract

Background: Tacrolimus (Tac) is a main immunosuppressant in kidney transplantation (KT), but, its narrow therapeutic window and potential for nephrotoxicity and neurotoxicity necessitate careful monitoring. In particular, Tac metabolism can affect clinical outcomes after KT, but it is difficult to measure the factors like cytochrome P450 or P-glycoprotein contributing to the Tac metabolism in clinical practice. Recently, it has been reported that Tac metabolism rate defined as the blood concentration normalized by its daily dose (the C/D ratio) can be used, but the efficacy is still controversial.

Methods: We enrolled 60 patients performed KT between 2013 and 2016. We aimed to investigate the efficacy of C/D ratio as a prognostic biomarker in kidney transplant recipients (KTRs). We divided them into fast metabolizer group (<1.05 ng/mL x 1/mg) and slow metabolizer group (>1.05 ng/mL x 1/mg) based on the Tac C/D ratio. We analyzed the incidence of delayed graft function (DGF), de novo donor specific antibody (DSA), acute rejection, cytomegalovirus and BK virus infection, graft function and graft survival according to the C/D ratio.

Results: The mean age of KTRs was 45 ± 9.2 years and the proportion of male was 43.3%. In particular, the proportion of elderly KTRs was higher in the slow metabolizer group than the fast metabolizer group. The incidence of DGF was significantly higher in the slow metabolizer group than the fast metabolizer group. The incidence of de novo DSA was significantly higher in the fast metabolizer group than the slow metabolizer group. However, there were no significant differences in the graft function (eGFR) at 3 months, 6 months and 12 months after KT, the incidence of acute rejection and cytomegalovirus and BK virus infection between the two groups. In the Kaplan-Meier analysis, there was no significant difference in the death-censored graft survival rate between the two groups.

Conclusion: In our study, the Tac metabolism rate was associated with the incidence of DGF and de novo DSA although there was no significant difference between the Tac metabolism rate and the incidence of acute rejection and infection, graft function within 1 year after KT. The Tac C/D ratio might be helpful as a prognostic biomarker to predict post-transplant clinical outcomes in KTRs.

Keywords : Kidney transplantation; Immunosuppression; Biomarkers; Outcome

Oral Communications : Kidney Transplant, Anemia, and MBD

Abstract Submission No. : APCN20250457

The Role of Mesenchymal Stem Cells in Treating Diabetic Kidney Disease: Immunomodulatory Effects and Kidney Regeneration

PO-JEN HSIAO^{1,2,3}; Wen-Yi Kao⁴; Li-Chin Sung^{5,6,7}; Chia-Yi Lin^{8,9}; Liam Li-An Tsou^{10,11}; Yung-Hsi Kao³; Chu-Lin Chou^{7,12,13,14}; Kung-Ta Lee⁴

¹ Division of Nephrology, Department of Internal Medicine, Taoyuan Armed Forces General Hospital, Taoyuan, Taiwan.

² Division of Nephrology, Department of Internal Medicine, Tri-Service General Hospital, National Defence Medical Centre, Taipei, Taiwan.

³ Department of Life Sciences, National Central University, Taoyuan, Taiwan.

⁴ Department of Biochemical Science and Technology, National Taiwan University, Taipei, Taiwan.

⁵ Division of Cardiology, Department of Internal Medicine, Shuang Ho Hospital, Taipei Medical University, New Taipei City, Taiwan.

⁶ Division of Cardiology, Department of Internal Medicine, School of Medicine, College of Medicine, Taipei Medical University, Taipei, Taiwan.

⁷ Taipei Medical University-Research Centre of Urology and Kidney, Taipei Medical University, Taipei, Taiwan.

⁸ Division of Cardiology, Department of Internal Medicine, Tri-Service General Hospital, Taipei, Taiwan.

⁹ Graduate Institute of Clinical Medicine, College of Medicine, National Taiwan University, Taipei, Taiwan.

¹⁰ Nephrology Division, Department of Medicine, Mount Sinai Hospital, New York, NY, USA.

¹¹ Biochemistry, Department of Chemistry, Hofstra University, Hempstead, New York, USA.

¹² Division of Nephrology, Department of Internal Medicine, School of Medicine, College of Medicine, Taipei Medical University, Taipei, Taiwan.

¹³ Division of Nephrology, Department of Internal Medicine, Shuang Ho Hospital, Taipei Medical University, New Taipei City, Taiwan.

¹⁴ Division of Nephrology, Department of Internal Medicine, Hsin Kuo Min Hospital, Taipei Medical University, Taoyuan City, Taiwan.

Abstract

Introduction: Diabetic kidney disease (DKD), also known as diabetic nephropathy (DN), is characterized by progressive glomerulosclerosis and chronic inflammation. The potential of mesenchymal stem cells (MSCs) in treating DKD could be explored.

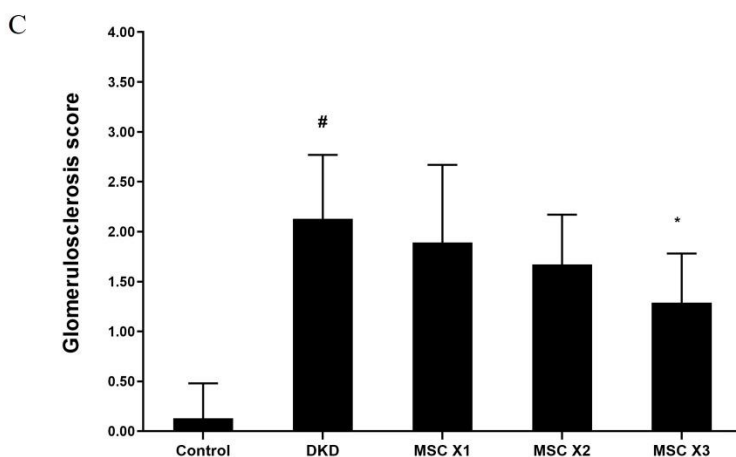
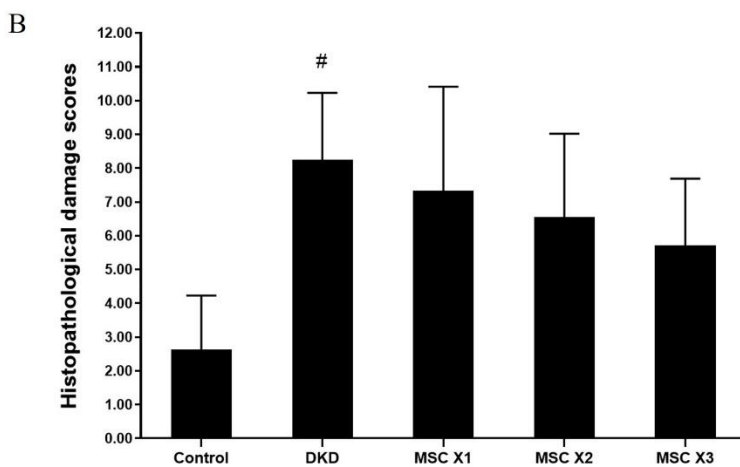
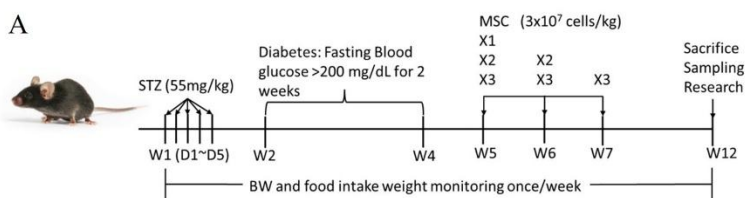
Methods: In this study, a streptozotocin (STZ)-induced type 1 diabetes mellitus (T1DM) DKD mouse model was utilized to investigate the renoprotective potential of human umbilical cord-derived mesenchymal stem cells (hUC-MSCs) through immunohistochemical, histopathological, and biochemical analyses. Two separate experiments were conducted to assess the therapeutic efficacy of hUC-MSCs in a DN mouse model. The first experiment determined the optimal dose by assigning the body weight and food intake alterations, serum cytokines and kidney function changes post hUC-MSCs treatment. STZ-induced DKD mice were divided to four groups: DKD control and other three hUC-MSCs treatment groups (low-dose: 3×10^6 , intermediate (middle)-dose: 1×10^7 , and high-dose: 3×10^7 cells/kg), with intravenous administration at weeks 8, 10, and 12 over 14 weeks. The second experiment evaluated treatment frequency, with mice assigned to hUC-MSCs x1, x2, and x3 groups (3×10^7 cells/kg) administered at weeks 5, 6, and 7 across 12 weeks, assessing the

kidney histology and morphometry changes.

Results: In the first experiment, the body weight and food intake showed no significant alterations among the DN and other 3 hUC-MSCs treatment groups. Compared to the DKD control group, only high-dose hUC-MSCs (3×10^7 cells/kg) treatment group significantly reduced the levels of inflammatory cytokines (IL-1 β , and TNF- α) ($p < 0.05$). Additionally, the urine albumin-to-creatinine ratio (UACR) levels in the high-dose hUC-MSCs (3×10^7 cells/kg) treatment group showed a decreasing trend compared to those in the DN control group ($p = 0.06$). In the second experiment, the hUC-MSCs x3 treatment group (3×10^7 cells/kg) significantly alleviated kidney histopathology compared to the DKD group ($p < 0.05$).

Conclusion: hUC-MSCs treatment may present a potential avenue for reversing glomerulosclerosis and mitigating inflammation in DKD mice. The long-term therapeutic benefits of MSCs-based treatments in patients with DKD and other kidney diseases could be further investigated.

Keywords : mesenchymal stem cells, diabetic kidney disease, immunomodulatory effects, kidney regeneration



Oral Communications : Kidney Transplant, Anemia, and MBD

Abstract Submission No. : APCN20251052

Efficacy and Safety of Rabbit versus Equine Anti-thymocyte Globulin as Induction Therapy in Kidney Transplantation with Modern Immunosuppression: A Single-Center Retrospective Cohort Study

Naveen Kumar Mattewada¹; Ramalakshmi Thullimalli²; Lekha Laxmi Pradhan²; Ravi Kumar Naredla¹; K S Nayak¹

¹ Department of Nephrology, Gleneagles Hospital, Lakdikapul, Hyderabad, India - 500004

² Department of Nephrology, Virinchi People's Hospital, Banjara Hills, Hyderabad, India - 500034

Abstract

Introduction: Polyclonal anti-thymocyte globulins, including rabbit (rATG) and equine (eATG) formulations, are potent induction agents used to prevent acute rejection in kidney transplantation. While historical trials compared these agents with older maintenance regimens, their relative efficacy and safety profiles in the context of contemporary triple immunosuppression (tacrolimus, mycophenolate mofetil, and steroids) are less defined. This study aimed to compare the clinical outcomes of rATG versus eATG induction in a modern cohort of adult kidney transplant recipients.

Methods: We conducted a single-center, retrospective analysis of adult patients who received a living donor kidney transplant between 2017 and 2023. All recipients had a negative pre-transplant complement-dependent cytotoxicity crossmatch and donor-specific antibody screen. Patients received induction therapy with either rATG (Thymoglobulin, 1.5 mg/kg) or eATG (Atgam, 10 mg/kg). The primary endpoints, assessed at 12 months post-transplant, were the incidence of biopsy-proven acute rejection (BPAR), graft loss, and patient mortality. Secondary endpoints included infection rates, duration of post-induction leukopenia, and renal function.

Results: A total of 58 patients were included: 17 received rATG and 37 received eATG. Baseline characteristics were comparable between groups. At 12 months, the incidence of BPAR was low and not statistically different between the groups (rATG: 5.9% (1/17) vs. eATG: 5.4% (2/37)). There were no episodes of graft loss in either group. One patient death occurred in the rATG group due to sepsis. The rATG group experienced a numerically higher incidence of infectious complications (8 events in 17 patients, including 2 cases of CMV viremia) compared to the eATG group (3 events in 37 patients, with no CMV). Furthermore, rATG was associated with a significantly more prolonged duration of leukopenia (median 24 days, range 2-78) compared to eATG (median 8 days, range 1-43).

Conclusion: In this real-world cohort of kidney transplant recipients on modern maintenance immunosuppression, rATG and eATG demonstrated comparable efficacy in preventing acute rejection. However, rATG was associated with a higher burden of infectious events and more protracted leukopenia. These findings suggest that eATG may offer a preferable safety profile in this setting. This provides a strong rationale for prospective studies and formal health-economic analyses to determine if eATG represents a more cost-effective induction strategy, particularly in regions with a high prevalence of infectious diseases

Keywords : Kidney Transplantation; Equine ATG; Thymogam

Oral Communications : Kidney Transplant, Anemia, and MBD

Abstract Submission No. : APCN20250039

Mortality, Malignancy, And CMV Infection Risks With mTOR Inhibitors Versus Mycophenolate In Kidney Transplantation: A Matched Real-World Analysis

Naveen¹; Balkrishna Subedi²

¹ Department of General Medicine, Gulmi Durbar Basic Hospital, Gulmi Durbar 03, Gulmi, Nepal

² Department of Internal Medicine, Einstein Medical Center Montgomery. 559 West Germantown Pike, East Norriton, PA 19403

Abstract

Introduction: Maintenance immunosuppression following kidney transplantation typically involves a calcineurin inhibitor (CNI) like tacrolimus plus mycophenolate (MMF/MPA). Mammalian target of rapamycin inhibitors (mTORi; sirolimus/everolimus) offer an alternative, potentially reducing viral infections and malignancy but with different side effect profiles. Large-scale real-world comparisons of long-term outcomes between tacrolimus+mTORi (Tac+mTORi) and tacrolimus+MMF/MPA (Tac+MMF) are limited. We aimed to compare the effectiveness and safety of these regimens using global real-world data.

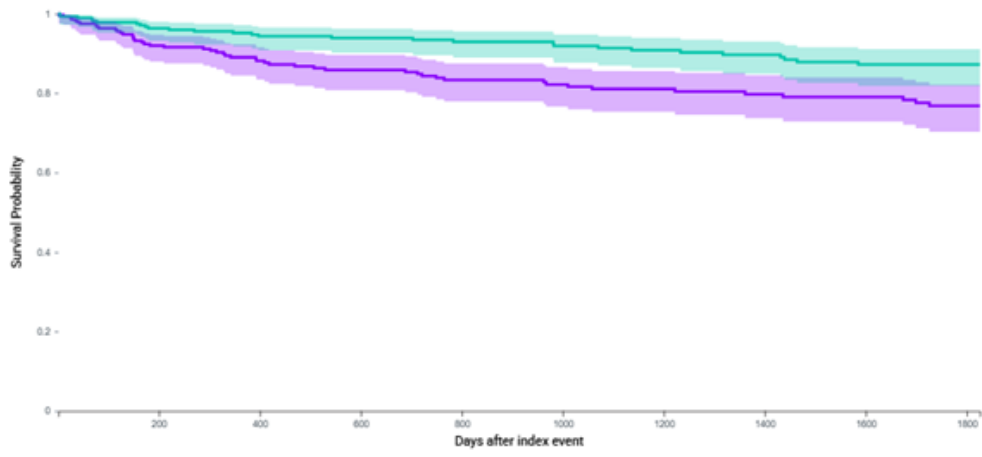
Methods: We conducted a retrospective cohort study using the TriNetX global federated health research network (144 HCOs). We identified adult kidney transplant recipients (ICD-10 Z94.0) initiating either Tac+mTORi or Tac+MMF at least 3 months post-transplant, excluding multi-organ transplants and those with dialysis dependence prior to the index date (first day of concurrent regimen). Patients initiating Tac+mTORi (N=302 before matching) were matched 1:1 using propensity scores to patients initiating Tac+MMF (N=8,552 before matching) based on baseline demographics, diagnoses (including diabetes, hypertension, cardiovascular disease, malignancy history), and medications (including steroids, antihypertensives, statins, insulin). The final analysis included 284 matched pairs. Outcomes were assessed over 5 years using Cox proportional hazards models. Key outcomes included composite graft failure (return to dialysis, re-transplant, graft failure codes), CMV infection (ICD-10 B25), non-melanoma skin cancer (NMSC; ICD-10 C44), and all-cause mortality.

Results: The 284 matched pairs were well-balanced on baseline characteristics. Over 5 years, there was no significant difference in composite graft failure between the Tac+mTORi and Tac+MMF groups (HR 1.14; 95% CI 0.68-1.92; p=0.623). Patients receiving Tac+mTORi had a significantly lower risk of CMV infection (HR 0.11; 95% CI 0.03-0.47; p<0.001) but a significantly higher risk of NMSC (HR 3.05; 95% CI 1.39-6.71; p=0.003) and all-cause mortality (HR 2.03; 95% CI 1.28-3.23; p=0.002). Trends towards lower leukopenia/neutropenia (HR 0.42, p=0.063) were observed with mTORi.

Conclusion: In this large, propensity-score matched real-world analysis of kidney transplant recipients, initiating a Tac+mTORi regimen \geq 3 months post-transplant was associated with significantly lower CMV infection risk compared to Tac+MMF over 5 years. However, this potential benefit was offset by significantly increased risks of non-melanoma skin cancer and all-cause mortality, with no difference in graft failure. These findings highlight the complex risk-benefit profile of mTORi-based regimens and underscore the need for individualized treatment strategies.

Keywords : Kidney Transplantation; mTOR Inhibitors; Mycophenolate; Propensity Score Matching; Real-World Evidence

Kaplan-Meier Survival Curve



Oral Communications : Kidney Transplant, Anemia, and MBD

Abstract Submission No. : APCN20250944

Multi-Omics Machine Learning Model Predicts Long-Term Graft Outcome Based on Urinary Renal Progenitor Epigenomic Reprogramming After Kidney Transplantation

Prihantini¹; Sahnaz Vivinda Putri²; Rini Winarti³; Elfiany Syafruddin⁴; Rifaldy Fajar¹

¹ AI-BioMedicine Research Group, IMCDS-BioMed Research Foundation, Jakarta, Indonesia

² Health Management Laboratory, International University Semen Indonesia, Gresik, Indonesia

³ Department of Biology, Yogyakarta State University, Sleman, Indonesia

⁴ Computational Science Research Laboratory, BLK Muhammadiyah University, Bulukumba, Indonesia

Abstract

Introduction: Long-term graft deterioration remains a major cause of morbidity after kidney transplantation. Despite routine monitoring, current methods fail to predict chronic injury progression with sufficient lead time. Urinary renal progenitor cells may reflect early graft pathophysiology through transcriptional activity and epigenomic remodeling. This study aimed to develop a machine learning model integrating transcriptomic and methylomic profiles of urine-derived progenitor cells to predict long-term graft outcomes, including fibrosis progression and renal function decline.

Methods: Transcriptomic and methylomic data were retrieved from the Gene Expression Omnibus (GEO), specifically GSE235813 (RNA-seq from 37 urine-derived renal progenitor cell samples post-transplant) and GSE213458 (methylation data from 41 matched samples using Illumina EPIC arrays). Samples were stratified by donor type (living vs deceased) and by 24-month clinical outcome (stable vs deteriorating graft). A multi-modal machine learning pipeline was constructed using a late-fusion framework. RNA-seq data were preprocessed using variance stabilizing transformation and modeled via a stacked autoencoder followed by Random Forest classification. DNA methylation data were normalized using noob method and processed through SHAP-informed XGBoost after feature selection based on mutual information and variance thresholds. Predictions from both branches were fused using logistic regression to generate a composite Long-Term Graft Risk Score (LTGRS). Model performance was evaluated using stratified 5-fold cross-validation and an independent 20% test set.

Results: The fused model achieved an AUROC of 0.83 (95% CI: 0.78–0.88) and AUPRC of 0.79, surpassing transcriptomic-only (AUROC 0.72) and methylomic-only (AUROC 0.76) baselines. In the high-risk LTGRS group, 85.7% of patients progressed to CKD stage ≥ 3 within 24 months, versus 22.2% in the low-risk group ($p < 0.0001$). Fibrosis score (Banff ci ≥ 2) was significantly higher in the high-risk group (mean score 2.1 vs 0.9, $p = 0.002$), and LTGRS was negatively correlated with eGFR slope ($r = -0.48$, $p = 0.004$). Top predictive features included downregulation of KLF9 and LINC01133 and hypermethylation at cg13725483 (NR3C1). These molecular signals reflect altered stress response and glucocorticoid sensitivity, particularly in deceased-donor grafts. Deceased-donor grafts showed stronger stratification accuracy (AUROC 0.86) than living-donor grafts (AUROC 0.78), and among deceased-donor recipients, increased methylation at immune regulation loci correlated with microvascular inflammation severity and interstitial tissue remodeling on protocol biopsy.

Conclusion: This study presents an integrative multi-omics ML model using urine-derived renal progenitor data to predict long-term graft outcomes. The approach offers an accurate, non-invasive, and interpretable tool for early risk stratification and long-term monitoring in kidney transplant recipients.

Keywords : Kidney Transplantation, Machine Learning, Urine Progenitor Cells, Multi-Omics Integration, Graft Outcome Prediction

Oral Communications : Kidney Transplant, Anemia, and MBD

Abstract Submission No. : APCN20250797

Prevalence And Correlates Of Adynamic Bone Disease In Patients With End-Stage Kidney Failure In Singapore

Shuit Siew Kit¹; Teh Yuan Kai¹; Chen Fangxia¹; Regina Lim Shaoying¹; Manohar Bairy¹

¹ Department of Renal Medicine , Tan Tock Seng Hospital, Singapore

Abstract

Introduction

The spectrum of chronic kidney disease-mineral and bone disorder (CKD-MBD) is changing and adynamic bone disease (ABD) is now believed to constitute the majority of CKD-MBD in the developed world.¹⁻⁴ However, its prevalence and risk factors are poorly described in the literature. Its diagnosis requires bone biopsy but biochemical criteria including parathyroid hormone (PTH) levels show a good correlation.⁵ This study aims to understand the prevalence of ABD in our end-stage kidney failure (ESKF) hemodialysis (HD) population, identify the risk factors for its development, and in doing so enable early intervention to modify the risk factors specific to our population.

Methods

This is a retrospective cross-sectional study. 201 prevalent adult patients on maintenance HD and no previous parathyroidectomy were recruited. Relevant clinical and biochemical parameters, management and outcomes were collected. ABD was diagnosed if any intact PTH (iPTH) level during the study period was <15pmol/L.

Results

Of the 201 patients in the study (median age 64.5 years), 35 (17.4%) patients had ABD. In the multivariable model, the adjusted OR of ABD was higher with higher mean adjusted serum calcium level while concurrent use of non-calcium-based binders was associated with lower odds of ABD. Activated vitamin D non-use was also associated with higher odds of ABD likely reflecting past occurrence of ABD. 17% of patients had had fractures without significant association with ABD. The mean PTH level was in the target range (15-60pmol/L) in 41% of the cohort. Cardiovascular complications were not significantly associated with ABD.

Conclusions

Approximately one in every six haemodialysis patients in our care has ABD as diagnosed by the iPTH level. Targeting a lower serum calcium level and using non-calcium-based binders may reduce the occurrence of ABD and will need to be tested in prospective studies.

Keywords : adynamic bone disease, parathyroid hormone, renal hyperparathyroidism

Variables	Overall (N=201)	Adynamic Bone Disease (N = 35)	No Adynamic Bone Disease (N = 166)	Multivariable model	
				Adjusted OR (95% CI)	p- value*
Age in years, mean (SD)	64.5 (12.5)	68.7 (12.5)	63.6 (12.4)	1.02 (0.98-1.07)	0.26
Gender, N (%)					
Male	123 (61.2)	21 (60.0)	102 (61.5)	1.39 (0.56-3.45)	0.48
Female	78 (38.8)	14 (40.0)	64 (38.6)	Reference	
ESKF cause, N (%)					
Diabetes mellitus (DM)	124 (61.7)	25 (71.4)	99 (59.6)	-	
Hypertension (HTN)	34 (16.9)	5 (14.3)	29 (17.5)	-	
Chronic glomerulonephritis (CGN)	35 (17.4)	3 (8.6)	32 (19.3)	-	
Others	8 (4.0)	2 (5.7)	6 (3.6)	-	
Vintage in months, median (IQR)	54 (30, 80)	41 (18, 78)	56 (31, 90)	1 (0.99-1.01)	0.36
DM, N (%)					
Yes	144 (71.6)	29 (82.9)	115 (69.3)	2.42 (0.82-7.1)	0.12
No	57 (28.4)	6 (17.1)	51 (30.7)	Reference	
Dialysate Calcium (mmol/L)					
Normal (1.5 mmol/L)	111 (55.2)	22 (62.9)	89 (53.6)	Reference	
Low (1.25 mmol/L)	90 (44.8)	13 (37.1)	77 (46.4)	0.58 (0.23-1.42)	0.23
Serum Calcium, mean (SD), mmol/L	2.27 (0.14)	2.30 (0.13)	2.27 (0.14)	163.42 (3.4- 7847.73)	0.01
Serum Phosphate, mean (SD), mmol/L	1.56 (0.42)	1.47 (0.37)	1.57 (0.43)	0.94 (0.27-3.26)	0.92
Serum Alkaline Phosphatase, mean (SD), U/L	143.73 (159.33)	100.9 (113.91)	152.76 (166.22)	1 (0.99-1)	0.33
Serum Albumin, mean (SD), g/L	39.25 (4.60)	38.91 (5.08)	39.32 (4.50)	-	
Calcium-based phosphate binder, N (%)					
Yes	160 (79.6)	30 (85.7)	130 (78.3)	-	
No	41 (20.4)	5 (14.3)	36 (21.7)	-	
Non-calcium-based phosphate binder, N (%)					
Yes	50 (24.9)	1 (2.9)	49 (29.5)	Reference	
No	151 (75.1)	34 (97.1)	117 (70.5)	20.39 (2.06- 202.33)	0.01
Calcitriol use, N (%)					
Yes	119 (59.2)	10 (28.6)	109 (65.7)	Reference	
No	82 (40.8)	25 (71.4)	57 (34.3)	5.99 (2.28- 15.75)	<0.001
Calcimimetic use, N (%)					
Yes	22 (11)	0 (0)	22 (13.3)	-	
No	179 (89.1)	35 (100)	144 (86.8)	-	

CI – Confidence Interval, OR – Odds Ratio * Bolded values indicate statistical significance of p < 0.05

Oral Communications : Kidney Transplant, Anemia, and MBD

Abstract Submission No. : APCN20251046

Interactive Effects of Hemoglobin and Iron Deficiency on kidney prognosis and Major Adverse Cardiovascular Events: A Retrospective Cohort Study

ZHU XiHuan¹; Xiaomeng Lin²; Xudong Cai²; Guanghui Zhong²; Ji Wang^{3,4}; Qi Wang^{3,4}; Huai-Yu Wang^{3,4}

¹ Qi-huang Chinese Medicine School, Beijing University of Chinese Medicine, Beijing, China

² Ningbo Municipal Hospital of Traditional Chinese Medicine, Affiliated Hospital of Zhejiang Chinese Medical University, Ningbo, China

³ National Institute of Traditional Chinese Medicine Constitution and Preventive Medicine, Beijing University of Chinese Medicine, Beijing, China

⁴ School of Traditional Chinese Medicine, Beijing University of Chinese Medicine, Beijing, China

Abstract

Introduction: Anemia and iron deficiency are prevalent and interrelated comorbidities in patients with chronic kidney disease (CKD) and are linked to worse clinical outcomes. However, their combined effects on kidney and cardiovascular prognosis remained under-investigated. The draft of 2025 KDIGO guidelines for anemia in CKD emphasized the need to investigate the interactions between anemia and iron deficiency and their impact on kidney and cardiovascular outcomes.

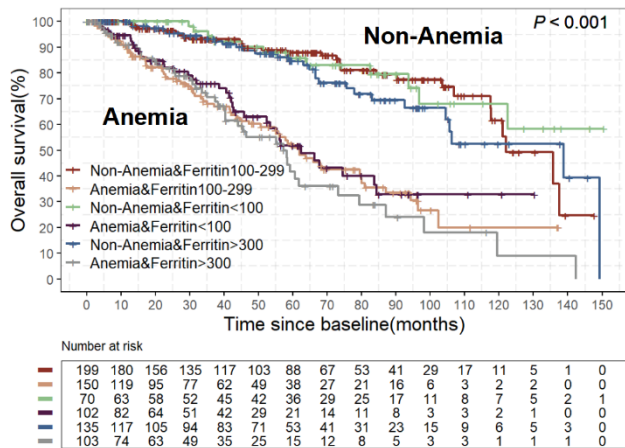
Methods: This retrospective cohort study included non-dialysis CKD patients from Ningbo Municipal Hospital of Traditional Chinese Medicine. Anemia was defined by hemoglobin (Hb) levels, and iron status was assessed using ferritin. Adverse kidney outcomes were defined as a $\geq 30\%$ decline in eGFR, doubling of serum creatinine, or progression to end-stage kidney disease (ESKD, eGFR < 15 mL/min/1.73 m²). Major adverse cardiovascular events (MACE) were evaluated as cardiovascular outcomes. Cox and Kaplan-Meier analyses were used to evaluate the associations of anemia/iron deficiency with outcomes. Restricted cubic spline (RCS) were applied to explore nonlinear relationships between hemoglobin/ferritin and clinical outcomes.

Results: A total of 829 non-dialysis CKD patients were included and followed by 34.63 [13.17, 65.97] months. Kaplan-Meier analyses showed that patients with or without anemia exhibited distinguished risk of kidney prognosis (log-rank $P < 0.001$), while no significant difference in the risk of MACE was observed among subgroups (log-rank $P = 0.246$). (Figure A-B) Cox regression models revealed that ferritin > 300 ng/mL was associated with increased risks of eGFR 30% decline and ESKD. Anemia was linked to higher risks of kidney prognosis, regardless of ferritin levels. (Figure C) RCS analyses demonstrated a linear relationship between lower Hb and higher risk of kidney prognosis across all ferritin subgroups. (Figure D-G) For MACE, the association with Hb was nonlinear: elevated risk was observed at Hb < 102 g/L and 115–142 g/L, while reduced risk was observed at Hb > 158 g/L. This pattern persisted in patients with ferritin < 100 ng/mL. In contrast, no significant association between Hb and MACE was found in ferritin 100–300 ng/mL subgroup, and Hb < 105 g/L was linked to elevated risk in ferritin > 300 ng/mL subgroup. (Figure H-K) Ferritin > 418 ng/mL was linked to elevated risk for kidney prognosis in non-anemia patients but not in those with anemia. (Figure L-N) No significant association was found between ferritin and MACE.

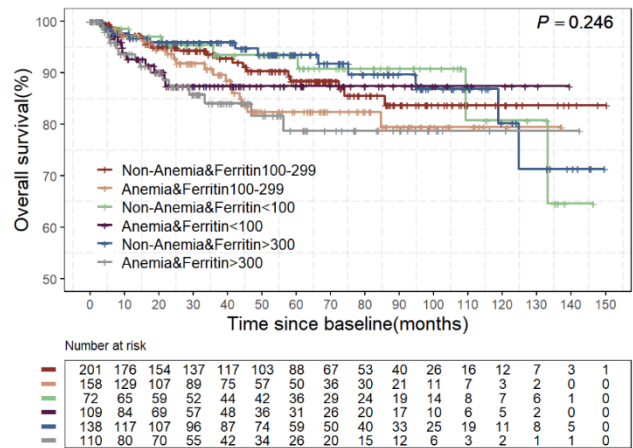
Conclusion: Hb show a linear association with kidney prognosis across ferritin subgroups, while the relationship between Hb and MACE is nonlinear and modified by iron status. Higher Ferritin levels are linked to worse kidney outcomes only in non-anemia patients.

Keywords : Anemia; Iron deficiency; Kidney prognosis; MACE

A. K-M curves for kidney prognosis(30% eGFR decline)



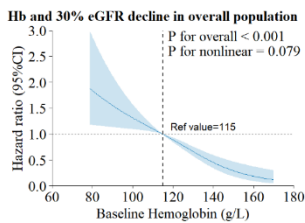
B. K-M curves for MACE



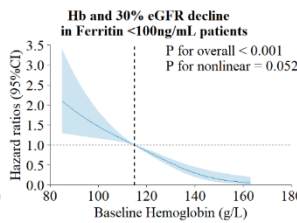
C. Association between anemia and iron deficiency and the risk of kidney prognosis and MACE

Variables	30% eGFR decline		Scr doubling		ESKD		MACE		
	HR (95%CI)	P value	HR (95%CI)	P value	HR (95%CI)	P value	HR (95%CI)	P value	
Seperate effect									
Anemia#									
No	Ref.		Ref.		Ref.		Ref.		
Yes	2.69(1.93,3.76)	<0.001	4.28(2.60,7.06)	<0.001	10.75(4.53,25.5)	<0.001	0.88(0.54,1.43)	0.609	
Iron de ficiency#									
Ferritin 100-300 ng/mL	Ref.		Ref.		Ref.		Ref.		
Ferritin <100 ng/mL	1.10(0.77,1.57)	0.607	0.73(0.46,1.18)	0.199	1.81(0.99,3.3)	0.053	1.04(0.59,1.84)	0.884	
Anemia&Ferritin >300 ng/mL	1.64(1.11,2.42)	0.013	1.32(0.80,2.18)	0.270	1.80(1.02,3.16)	0.042	1.24(0.67,2.28)	0.493	
Joint effect									
Anemia&Iron de ficiency#									
Non-Anemia&Ferritin 100-300 ng/mL	Ref.		Ref.		Ref.		Ref.		
Anemia&Ferritin 100-300 ng/mL	2.69(1.70,4.25)	<0.001	4.26(2.04,8.90)	<0.001	13.66(3.11,59.95)	0.001	0.78(0.40,1.53)	0.475	
Non-Anemia&Ferritin <100 ng/mL	0.80(0.41,1.57)	0.513	1.07(0.37,3.12)	0.899	2.36(0.33,17.16)	0.395	0.86(0.36,2.05)	0.726	
Anemia&Ferritin <100 ng/mL	2.55(1.53,4.26)	<0.001	6.24(2.89,13.46)	<0.001	27.06(5.94,123.35)	<0.001	0.83(0.39,1.80)	0.644	
Non-Anemia&Ferritin >300 ng/mL	1.60(0.95,2.68)	0.077	2.04(0.93,4.48)	0.075	2.50(0.41,15.19)	0.320	1.08(0.52,2.23)	0.831	
Anemia&Ferritin >300 ng/mL	3.71(2.26,6.09)	<0.001	7.23(3.39,15.4)	<0.001	20.91(4.74,92.32)	<0.001	1.03(0.51,2.08)	0.929	

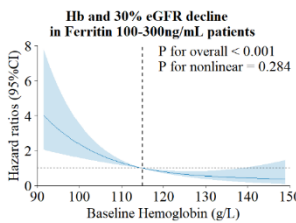
D.



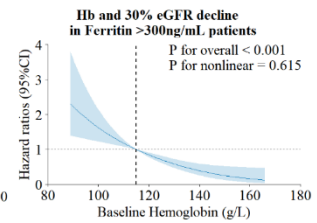
E.



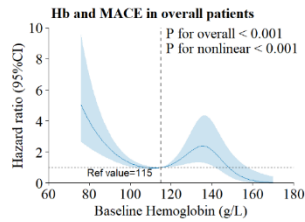
F.



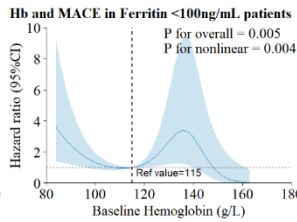
G.



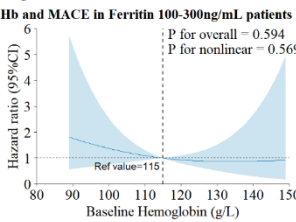
H.



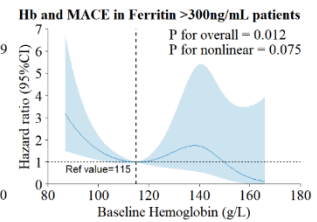
I.



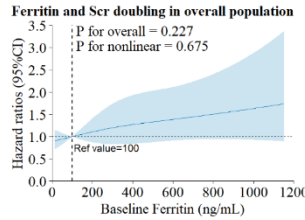
J.



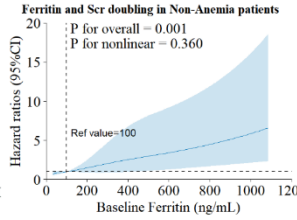
K.



L.



M.



N.

