ERA 2026

The atmosphere throughout the Congress embodied the theme, ‘Open Your Mind’, encouraging attendees to embrace innovation

Congress Review

Review of the European Renal Association (ERA) Congress 2026

Location:	Glasgow, UK
Date:	3rd–6th June 2026
Citation:	EMJ Nephrol. 2026;14[1]:10-23. https://doi.org/10.33590/emjnephrol/N217T1QF

THE QUAINT city of Glasgow provided the backdrop for Europe's premier nephrology meeting, the 63rd annual European Renal Association (ERA) Congress. Held from 3rd–6th June 2026 at the iconic Scottish Event Campus, the Congress brought together a record number of delegates from around the world. The venue, which has previously hosted major entertainment events, international conventions, and exhibitions, reflected the scale and ambition of this year's meeting. The atmosphere throughout the Congress embodied the theme, 'Open Your Mind', encouraging attendees to embrace innovation, collaboration, and fresh perspectives in nephrology.

Giuseppe Palladino, Research, Development and Innovation Director at ERA, opened the ceremony by welcoming delegates and introducing Kate Stevens, Local Congress President, and Rosa Torra, ERA President.

In her opening remarks, Stevens celebrated Glasgow's unique character and heritage, referencing the well-known phrase, 'People Make Glasgow', which reflects the warmth, friendliness, and hospitality of the city's communities. She also highlighted Glasgow's rich scientific legacy, noting the contributions of Thomas Graham, the Scottish chemist whose pioneering work on diffusion laid the foundations for modern dialysis and earned him recognition as the 'father of dialysis'. Stevens further acknowledged Joseph Lister, whose development of antiseptic surgical techniques transformed healthcare practice worldwide and has saved countless lives. She also recognised the historic achievements of James McCune Smith, who studied at the University of Glasgow and became the first African American to obtain a medical degree.

Stevens went on to discuss ERA's commitment to community engagement and sustainability through its congress legacy project. Initiatives included support for the Glasgow Children's Hospital Charity and the Kidney and Bee Adventure, a programme designed to encourage local children to engage with nature, while learning about kidney health and environmental stewardship.

Delegates were then welcomed by Bailie Marie Garrity, a long-serving member of Glasgow City Council, who further emphasised the city's enduring contribution to kidney care and medical innovation. Drawing parallels between the values of Glasgow and the ERA community, Garrity highlighted their shared commitment to fairness, inclusion, and opportunity for all. She noted that the Congress theme, 'Open Your Mind', reflects

the importance of curiosity, innovation, and collaboration in addressing current and future healthcare challenges.

ERA President Rosa Torra then delivered the Presidential Address, expressing her gratitude to the nephrology community for its continued engagement with and support of the association. She noted that the high level of participation at ERA reflects a collective commitment to shaping the future of nephrology. Torra also encouraged delegates to embrace the spirit of 'challenging their thinking' by critically evaluating emerging evidence and new developments in the field. She remarked that, rather than a lack of information, the modern challenge lies in effectively interpreting and applying the vast amounts of data now available to clinicians and researchers. Torra concluded by thanking everyone involved in organising the congress, including staff, committee members, and partner organisations.

Before the scientific programme commenced, Torra invited Serhan Tuğlular, Chair of the ERA 2026 Scientific Committee, to the stage to receive a diploma in recognition of her contributions to the congress.

The ceremony then celebrated the recipients of the ERA 2026 awards. Manuel Praga Terente, Complutense University, Madrid, Spain, received the ERA Award for Outstanding Clinical Contributions to Nephrology. Robert Unwin, University College London, UK, received the ERA Award for Outstanding Basic Science Contributions to Nephrology. Mehmet Kanbay, Koç University Hopsital, Istanbul, Türkiye, was honoured with the ERA Award for Research Excellence in Nephrology, while Danilo Fliser, Saarland University, Saarbrücken, Germany, received the ERA Award for Outstanding Contribution to the Society.

The ERA Award for Excellence in Sustainable Nephrology was presented to Groupe Néphrologie Verte (Société Francophone de Néphrologie, Dialyse et Transplantation), recognising its leadership in promoting environmentally sustainable kidney care.

Among the early-career award recipients, Verónica Miguel Herranz, RWTH Aachen University Hospital, Germany, received the Rosanna Gusmano Award for Young Investigators in Basic Science, while Michael Balzer, University of Kiel, Germany, was presented with the Stanley Shaldon Award for Young Investigators in Translational Science. Rik Olde Engberink, Amsterdam University Medical Center; and Michele Eisenga, University Medical Center Groningen, the Netherlands, received the Eberhard Ritz Award for Young Investigators in Clinical Science. Katharina Artinger, Medical University of Graz, Austria, was honoured with the Young Nephrologist ERA Council Award.

The opening ceremony also recognised Rosanna Coppo, University of Turin, Italy, who received Honorary Membership of the ERA in recognition of her longstanding contributions to nephrology and the association.

This year's Opening Ceremony concluded with a vibrant celebration of Scottish culture, beginning with an exuberant pipe band performance that showcased the musical heritage of the host nation and provided a memorable close to the evening's proceedings.

Pre-dialysis Exercise May Protect the Heart Across Haemodialysis Sessions

A RANDOMISED crossover trial suggests that a single bout of exercise performed before haemodialysis can reduce dialysis-induced cardiac injury during not only the same treatment session, but also the subsequent session, according to findings presented at ERA 2026.[1] These results may support the concept of exercise-induced cardiac preconditioning.

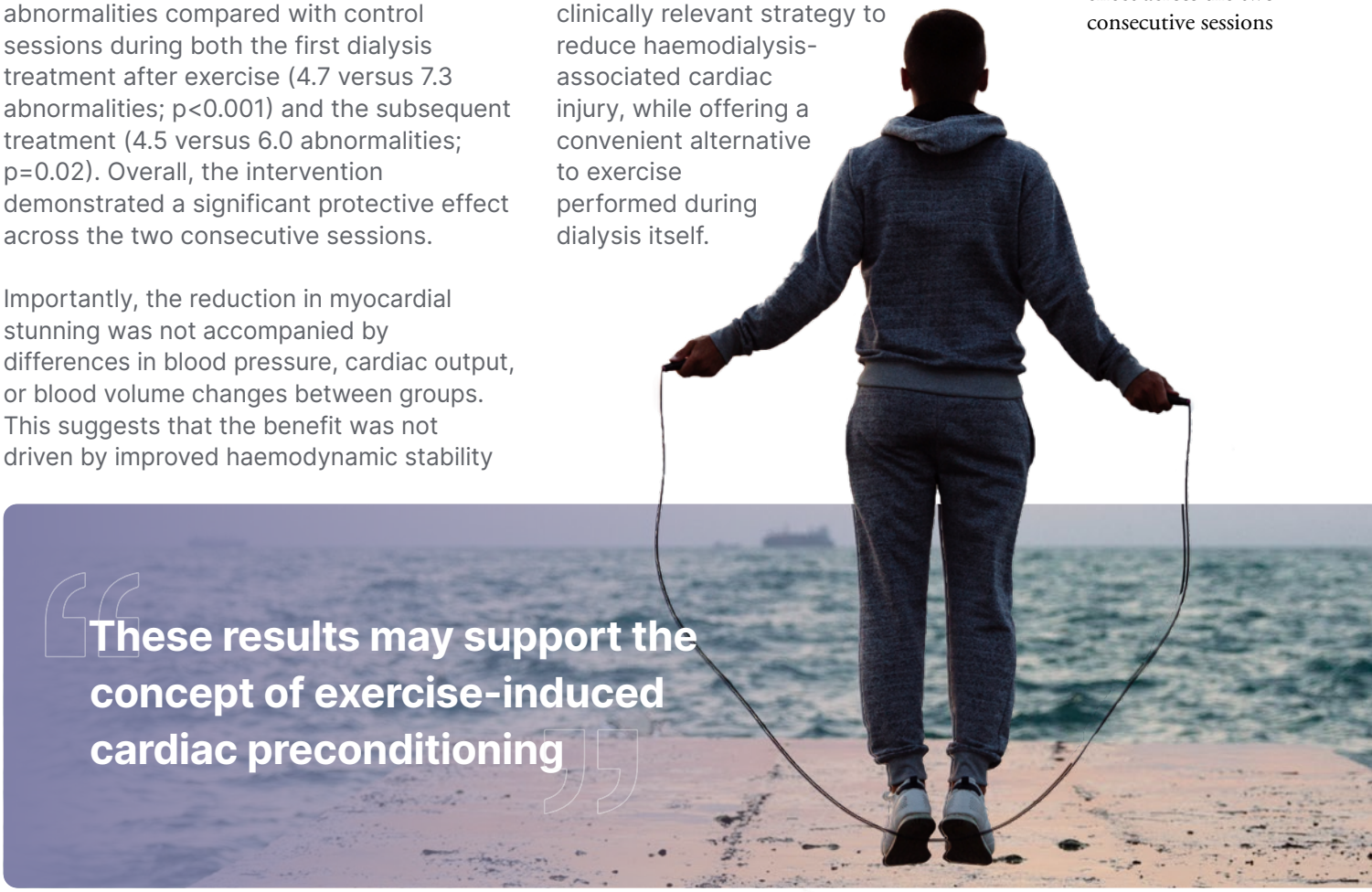
Researchers enrolled 26 patients receiving maintenance haemodialysis and compared two conditions: a 30-minute exercise session performed 1 hour before dialysis followed by a second dialysis session without exercise, versus two standard dialysis sessions without exercise. Cardiac function was assessed using speckle-tracking echocardiography, with dialysis-induced regional wall motion abnormalities, a marker of myocardial stunning and transient ischaemic injury, measured during treatment.

Pre-dialysis exercise significantly reduced the number of regional wall motion abnormalities compared with control sessions during both the first dialysis treatment after exercise (4.7 versus 7.3 abnormalities; $p<0.001$) and the subsequent treatment (4.5 versus 6.0 abnormalities; $p=0.02$). Overall, the intervention demonstrated a significant protective effect across the two consecutive sessions.

Importantly, the reduction in myocardial stunning was not accompanied by differences in blood pressure, cardiac output, or blood volume changes between groups. This suggests that the benefit was not driven by improved haemodynamic stability or vascular refilling, mechanisms traditionally thought to explain the cardioprotective effects of intradialytic exercise.

Instead, the findings support the hypothesis that exercise triggers a biphasic preconditioning response, with an immediate protective effect and a delayed 'second window' of protection that persists to the next haemodialysis session. The authors suggest that neural or humoral signalling pathways may be involved, although the precise mechanisms remain unclear.

The investigators conclude that pre-dialysis exercise could represent a practical and clinically relevant strategy to reduce haemodialysis-associated cardiac injury, while offering a convenient alternative to exercise performed during dialysis itself.



> Overall, the intervention demonstrated a significant protective effect across the two consecutive sessions

> **These results may support the concept of exercise-induced cardiac preconditioning**

TTV Trajectories May Help Identify Rejection and Infection Risk After Kidney Transplantation

MONITORING changes in torque teno virus (TTV) levels may help predict rejection and opportunistic infections during the first months after kidney transplantation, according to research presented at ERA 2026.[2]

Achieving the right balance of immunosuppression following transplantation remains challenging, with excessive treatment increasing infection risk and insufficient treatment raising the likelihood of rejection. TTV has emerged as a potential biomarker of overall immunosuppressive burden, but most studies have focused on population-wide thresholds rather than individual patient patterns.

Researchers from Queen Mary University of London and Royal London Hospital, UK, prospectively followed 268 kidney transplant recipients, measuring TTV levels from transplantation to 24 weeks post-transplant.

TTV levels increased during the first 12 weeks before plateauing, but substantial variation was observed between patients. Importantly, interval-to-interval increases in TTV were associated with a significantly lower risk of biopsy-proven rejection. In contrast, absolute TTV levels were not associated with rejection risk.

Higher TTV levels were linked to a greater likelihood of opportunistic infections. Each one-log increase in TTV was associated with a 57% increase in the odds of opportunistic infection, including BK virus and cytomegalovirus infections. No significant association was observed with bacterial infections.

The findings suggest that individual TTV trajectories may provide more clinically useful information than fixed universal thresholds. Rather than relying solely on absolute TTV values, monitoring how levels change over time could help clinicians assess immunosuppressive burden and identify patients at risk of either rejection or opportunistic infection.

The authors concluded that TTV shows promise as a patient-specific biomarker in the early post-transplant period and highlighted the need for randomised multicentre trials evaluating TTV-guided immunosuppression strategies.

57%

Each one-log increase in TTV was associated with a 57% increase in the odds of opportunistic infection

> **The findings suggest that individual TTV trajectories may provide more clinically useful information than fixed universal thresholds**

Chronicity Score Improves Kidney Allograft Risk Stratification

MAYO Clinic Chronicity Score was independently associated with long-term kidney transplant outcomes and may enhance risk stratification beyond established pathological classification systems, according to new research analysing kidney allograft biopsies, which was presented at ERA 2026.[3]

Current transplant pathology classifications place greater emphasis on active inflammation than chronic injury, despite chronic histological damage being irreversible and strongly linked to graft outcomes. Researchers investigated whether the Mayo Clinic Chronicity Score could provide a standardised approach to assessing chronic injury in kidney allograft biopsies.

The study evaluated 359 kidney allograft biopsies, with chronic lesions scored across four tissue compartments: glomerular sclerosis, interstitial fibrosis, tubular atrophy, and arteriosclerosis. The primary endpoint was progression to end-stage kidney disease within 5 years. Survival analyses and adjusted Cox regression models accounted for age, sex, estimated glomerular filtration rate, and proteinuria at the time of biopsy.

Results showed that higher Mayo Clinic Chronicity Score grades were independently associated with progression to end-stage kidney disease beyond 5 years. This association remained significant regardless of diagnostic category in both crude ($p<0.0001$) and adjusted analyses ($p=0.02$).

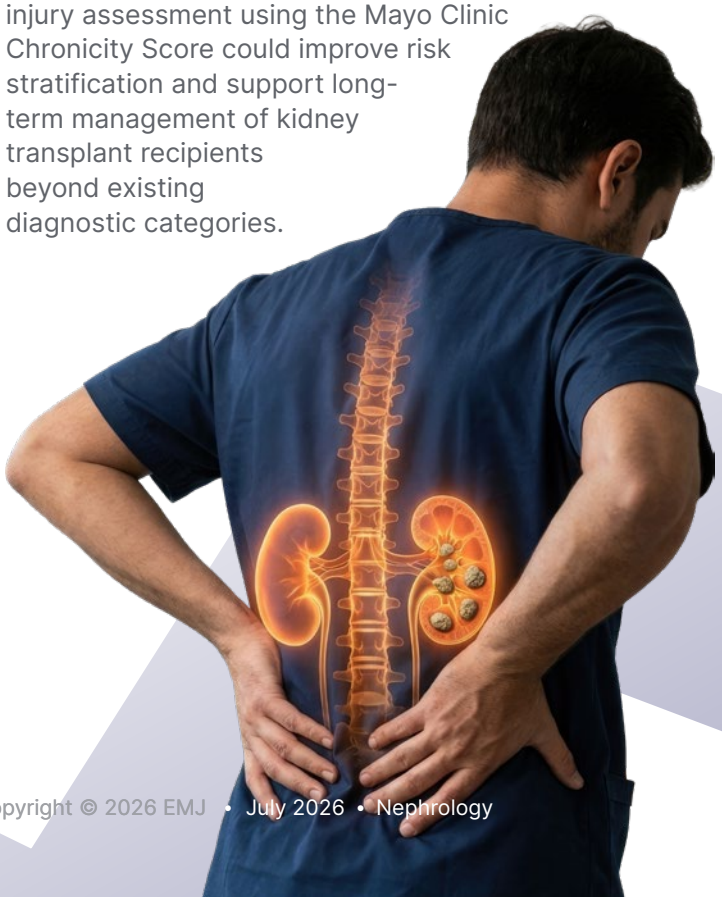
Among the individual histological components, interstitial fibrosis and tubular atrophy emerged as the strongest predictor of graft survival, demonstrating significant associations in both crude ($p<0.0001$) and adjusted analyses ($p<0.003$). Glomerular sclerosis was also associated with outcomes in unadjusted analyses ($p=0.0013$), although this relationship was attenuated after adjustment ($p=0.2$). Arteriosclerosis showed a non-significant trend towards poorer outcomes ($p=0.079$).

The composite Mayo Clinic Chronicity Score demonstrated the highest predictive accuracy at earlier time points, achieving area under the curve values of 0.836 at 12 months and 0.816 at 36 months.

At 60 months, interstitial fibrosis and tubular atrophy alone showed the strongest predictive performance, with an area under the curve of 0.746. Combining interstitial fibrosis and tubular atrophy with either glomerular sclerosis or arteriosclerosis further improved long-term predictive ability, with area under the curve values ranging from 0.745–0.770. In contrast, glomerular sclerosis and arteriosclerosis alone became less informative over time.

The findings represent the first systematic application of the Mayo Clinic Chronicity Score in kidney allograft biopsies and suggest the tool is practical, reproducible, and clinically relevant. The authors conclude that incorporating chronic injury assessment using the Mayo Clinic Chronicity Score could improve risk stratification and support long-term management of kidney transplant recipients beyond existing diagnostic categories.

At 60 months, interstitial fibrosis and tubular atrophy alone showed the strongest predictive performance, with an area under the curve of 0.746



Study Reveals that EKFC and CKD-EPI Yield Similar KFRE Performance

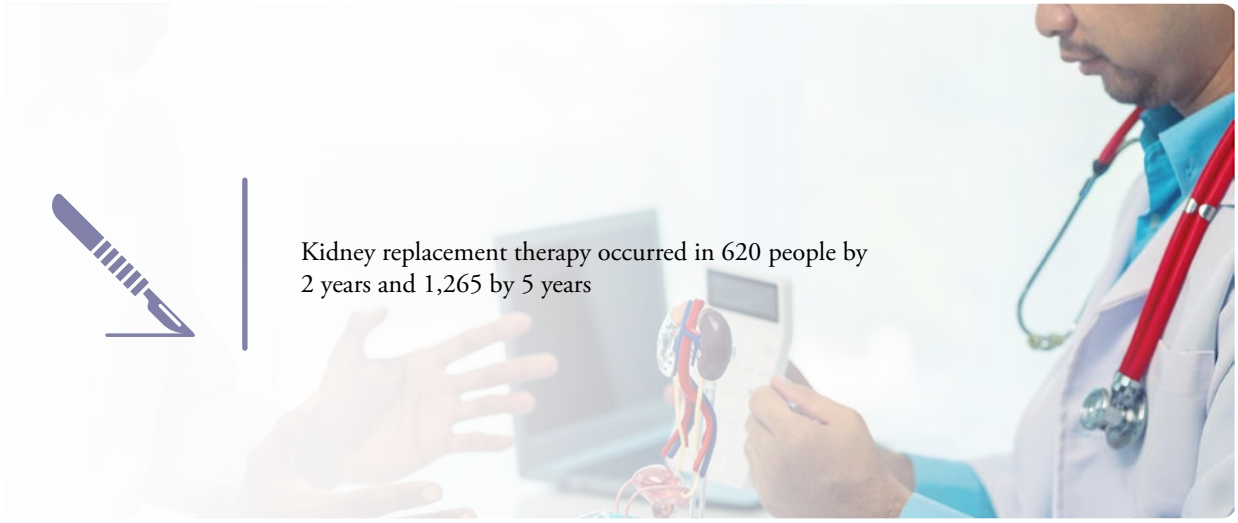
SWAPPING the newer European Kidney Function Consortium (EKFC) equations for established Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) ones makes little difference to how well the Kidney Failure Risk Equation (KFRE) predicts kidney failure, a large Swedish study reported, though EKFC sharpens accuracy when cystatin C is used.[4]

The KFRE guides European clinicians on when to refer patients to nephrology and prepare for kidney replacement therapy. It was built on CKD-EPI-based estimated glomerular filtration rate (eGFR). As centres increasingly adopt the EKFC equations, which match or outperform CKD-EPI, it is unclear whether substituting them disturbs the tool's reliability.

The team used the Stockholm CREAtinine Measurements (SCREAM) project, a population-based cohort exceeding three million residents. They identified adults who, between 2011–2021, had creatinine and cystatin C measured the same day plus an adjacent albuminuria test. Two- and five-year kidney failure risks were computed with the four-variable KFRE, covering eGFR, albuminuria, age, and sex. eGFR came from creatinine, cystatin C, or their combination, via CKD-EPI or EKFC. The final sample comprised 27,125 individuals (median age: 75 years; 45% women).

Kidney replacement therapy occurred in 620 people by 2 years and 1,265 by 5 years, while 4,214 and 8,287 respectively died without reaching kidney replacement therapy. KFRE discrimination was excellent across every equation and filtration marker, with areas under the curve spanning 0.95–0.97. For creatinine-based eGFR, EKFC and CKD-EPI delivered nearly identical calibration at both horizons. When cystatin C or the creatinine-cystatin C combination was used, EKFC produced better KFRE calibration than CKD-EPI. Net benefit at the KDIGO-recommended thresholds for guiding nephrology care was similar at 2 and 5 years, irrespective of the equation applied.

The results demonstrate that, in a northern European system, estimating creatinine-based eGFR with EKFC or CKD-EPI does not impact the KFRE's predictive performance or clinical utility, reassuring clinics adopting the newer equations.



Cystatin C eGFR Improves Chronic Kidney Disease Risk Stratification

CYSTATIN C estimated glomerular filtration rate (eGFR) may improve chronic kidney disease risk stratification by identifying patients at greater risk of mortality and hospitalisation who are not detected using creatinine-based eGFR, according to new findings from a large population-based analysis.[5]

Researchers analysed data from 372,454 participants in the UK Biobank to investigate whether cystatin C-based eGFR could identify individuals with chronic kidney disease at increased risk of adverse outcomes compared with conventional creatinine-based measures.

Participants were categorised according to chronic kidney disease status using both methods. During a median follow up of 12.53 years, there were 23,470 deaths and 113,378 first hospitalisation episodes.

The analysis showed substantial differences in the number of participants identified as having chronic kidney disease. Creatinine-based eGFR exclusively identified 2,584 individuals, whereas cystatin C eGFR alone identified 11,691 participants.

Among both the full cohort and participants eligible for chronic kidney disease screening, those identified exclusively through cystatin C eGFR consistently experienced higher rates of all-cause mortality and first hospitalisation.

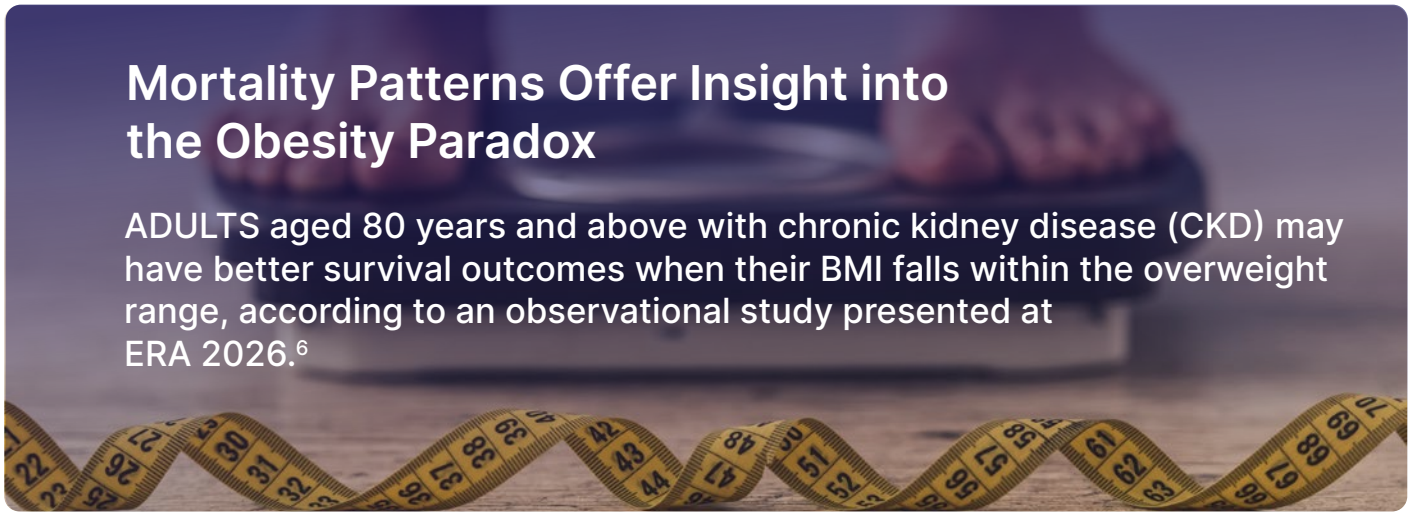
In contrast, researchers found no consistent associations between adverse outcomes and participants classified as having no chronic kidney disease or those identified solely through creatinine-based eGFR.

Importantly, the relationship between cystatin C eGFR defined chronic kidney disease and adverse outcomes remained evident among participants without albuminuria and across body mass index extremes. These findings suggest that cystatin C based assessment may provide prognostic information beyond traditional markers currently used in clinical practice.

The investigators also evaluated cohorts reflecting current eligibility criteria for disease modifying chronic kidney disease therapies. Within these groups, participants identified as having chronic kidney disease only through cystatin C eGFR experienced at least double the rates of mortality and hospitalisation compared with those identified exclusively using creatinine-based eGFR.

The authors concluded that reliance on creatinine-based eGFR alone may systematically exclude higher risk populations from future chronic kidney disease trials. The findings support the potential role of cystatin C eGFR in improving risk stratification and informing participant selection in future studies.

These results highlight the possibility that incorporating cystatin C eGFR into chronic kidney disease assessment frameworks could enable more accurate identification of patients at greatest risk of adverse clinical outcomes.



Mortality Patterns Offer Insight into the Obesity Paradox

ADULTS aged 80 years and above with chronic kidney disease (CKD) may have better survival outcomes when their BMI falls within the overweight range, according to an observational study presented at ERA 2026.[6]

The study explored the so-called 'obesity paradox' in the oldest-old population with CKD, a population often characterised by multimorbidity and age-related changes in body composition that have been largely overlooked in previous research.

Researchers analysed data from 1,267 adults aged 80 years or older with CKD, representing an estimated 4.2 million people in the USA. During a median follow-up period of 4.8 years, 795 deaths were recorded, 320 of these being cardiovascular.

Using restricted cubic spline analyses and adjusted Cox proportional hazards models, the investigators assessed relationships between BMI and all-cause, cardiovascular, and non-cardiovascular mortality. The analyses accounted for a range of factors including demographic characteristics, smoking status, comorbidities, kidney function measures, and laboratory values.

Results showed a U-shaped association between BMI and cardiovascular mortality, while non-cardiovascular mortality decreased as BMI increased. The analyses indicated that the BMI associated with the most favourable cardiovascular survival fell within the overweight range.

Among participants whose BMI exceeded the point associated with the lowest cardiovascular risk, each one-unit increase in BMI was linked to a 7% higher risk of cardiovascular mortality. However, each additional BMI unit was associated with a 4% lower risk of non-cardiovascular mortality. Compared with overweight individuals, those with a BMI of 35 kg/m^2 or higher had a significantly greater risk of cardiovascular death (HR: 1.81; 95% CI: 1.15–2.86).

The authors suggest that the obesity paradox in the oldest-old CKD population may be largely explained by lower rates of non-cardiovascular mortality among individuals with higher BMI, as higher BMI appeared protective for non-cardiovascular mortality.

As an observational study, a cause-and-effect relationship cannot be established. However, the results indicate that an overweight BMI may represent the most favourable range for survival in very elderly people with CKD and could help inform future guidance for this growing patient population.

> **The BMI associated with the most favourable cardiovascular survival fell within the overweight range**

Air Pollution Linked to CKD Progression in Portugal

A LARGE Portuguese cohort study, presented at ERA 2026, suggests that exposure to air pollution, particularly fine particulate matter ($PM_{2.5}$) and ozone (O_3), may contribute to chronic kidney disease (CKD) development and progression, even in regions with generally good air quality.[7]

The IMPA-R-PT study analysed data from 23,620 adults followed for a median of 6.7 years. Participants lived within 2 km of air quality monitoring stations, allowing researchers to assess long-term exposure to pollutants including $PM_{2.5}$, PM_{10}, nitrogen dioxide (NO_2), sulphur dioxide (SO_2), and O_3.

CKD prevalence in the cohort was 33.8%, with an incidence rate of 25.2 cases per 1,000 person-years. Patients with CKD had significantly higher exposure to $PM_{2.5}$, PM_{10}, and O_3 than those without kidney disease. After adjustment for demographic and clinical factors, $PM_{2.5}$, O_3, and the PM_{10}/ $PM_{2.5}$ ratio remained independently associated with CKD prevalence.

Higher pollutant exposure was also linked to faster kidney function decline. Patients classified as rapid progressors experienced greater exposure to $PM_{2.5}$, SO_2, and O_3, while those who progressed to advanced CKD (Stage 5) had significantly higher levels of $PM_{2.5}$ and O_3 exposure than the rest of the cohort.

Notably, $PM_{2.5}$ emerged as an independent predictor of incident CKD in adjusted analyses, with each increase in average $PM_{2.5}$ concentration associated with a 15% higher risk of developing CKD. Elevated levels of PM_{10}, SO_2, NO_2, and O_3 were also associated with increased CKD incidence, while a higher $PM_{10}/PM_{2.5}$ ratio appeared protective.

The authors note that air quality was rated good or very good on more than 90% of study days, suggesting that adverse renal effects may occur even at pollutant concentrations that meet current regulatory standards. The findings add to growing evidence that chronic exposure to air pollution may contribute to kidney disease through inflammatory and oxidative stress pathways and highlight potential implications for environmental health policy.

Myeloid HIF-2α Deficiency Linked to Renal Fibrosis and Immune Dysregulation

DISRUPTION of myeloid hypoxia-inducible factor-2 α (HIF-2α) may drive renal fibrosis through altered communication between immune cells, according to research presented at ERA 2026.[8]

Renal fibrosis is a key contributor to chronic kidney disease progression, but the mechanisms linking hypoxia, immune dysfunction, and tissue scarring remain incompletely understood. While HIF-2α is known to play important roles in kidney biology, anaemia, and cancer, its influence on immune cell interactions in kidney disease has been less clear.

To investigate this, researchers generated a mouse model lacking HIF-2α specifically in myeloid cells and compared these animals with healthy control mice. The team examined kidney function, immune cell behaviour, and markers of fibrosis to determine how loss of myeloid HIF-2α affected renal health.

The investigators observed spontaneous and early development of renal fibrosis in mice lacking myeloid HIF-2α. By 3 months of age, affected animals showed evidence of kidney dysfunction, including increased serum creatinine levels and alterations in urinary biomarkers. Histological analysis revealed prominent glomerular sclerosis, while molecular studies demonstrated increased expression of genes associated with fibrosis, inflammation, and kidney injury.

Further analysis identified significant changes in immune cell behaviour. Macrophages isolated from younger mice displayed a pro-inflammatory profile, while those from older animals shifted towards a more anti-inflammatory phenotype. Despite this apparent change, the cells remained metabolically hyperactive, exhibiting increased glycolysis and oxidative phosphorylation.

Proteomic analysis highlighted neutrophil degranulation as one of the most strongly enriched pathways in the kidneys of HIF-2α-deficient mice. Consistent with this finding, researchers identified hyperactive neutrophils with immunosuppressive and anti-apoptotic characteristics in the bone marrow.

Importantly, signalling molecules released by both HIF-2α-deficient macrophages and neutrophils promoted a profibrotic response in cultured mesangial cells, increasing expression of genes associated with tissue scarring and cellular transformation.

The findings suggest that abnormal communication between macrophages and neutrophils may be a key driver of fibrosis development. According to the authors, loss of myeloid HIF-2α promotes excessive neutrophil recruitment and activation within the kidney, accelerating tissue injury and disease progression.

These results identify a previously unrecognised role for myeloid HIF-2α in regulating immune res

ponses within the kidney and provide new insight into how hypoxia-related pathways contribute to renal fibrosis. The researchers suggest that targeting this immune crosstalk could represent a novel therapeutic strategy for slowing the progression of chronic kidney disease.

> **“The investigators observed spontaneous and early development of renal fibrosis in mice lacking myeloid HIF-2α”**

Extracellular Vesicles Show Potential to Block Immune-Driven Acute Kidney Injury in Sepsis

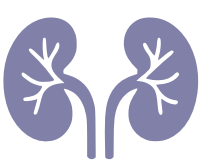
NEW FINDINGS presented at ERA 2026 redefine the biology of sepsis-associated acute kidney injury (SA-AKI), identifying monocytes intracellular complement (complosome) activation as a key driver of organ damage and a promising target for intervention.[9]

Building on their earlier discovery that reduced podocyte-derived cluster of differentiation 35-positive extracellular vesicles (CD35-EVs) are associated with poor outcomes in SA-AKI, the researchers investigated whether these vesicles play a functional role in disease progression in cecal ligation and puncture mice. The study reveals that CD35-EVs act as natural regulators of monocyte complement activity, blocking a newly identified intracellular pathway that drives organ injury.

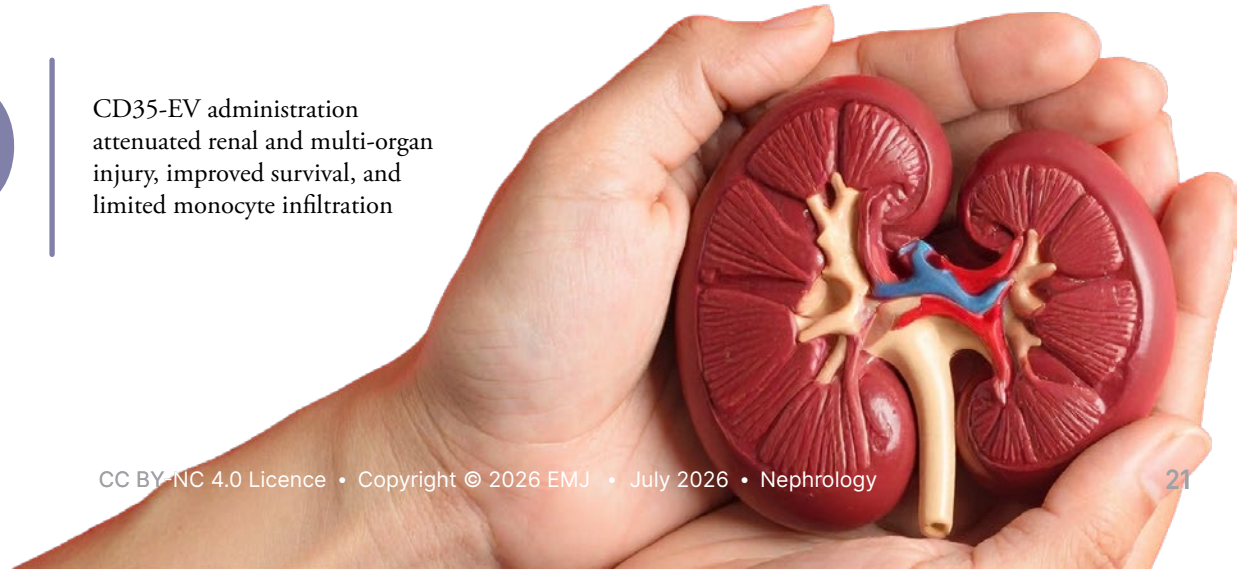
Monocyte-tracking and molecular assays were used to trace the original of renal component depositions, analyse organ infiltration, complosome activation, and CD35-EV-complosome interaction. Injury pathways driven by infiltrating monocytes were defined via multi-omics as well validation of the functional necessity of complosome through genetic and transplantation models. Monocytes from sepsis patients were also collected to assess the correlation between complosome activation and the severity of organ injury.

CD35-EV administration attenuated renal and multi-organ injury, improved survival, and limited monocyte infiltration. Injury was driven by infiltrating monocytes exhibiting intracellular complosome activation linked to mitochondrial C5a receptor 1 signalling and glycolytic reprogramming that promoted migration. CD35-EVs were internalised by monocytes, where they neutralised intracellular C3b and suppressed alternative pathway activity, reversing metabolic and chemotactic programming. Monocyte-specific C3 deletion reproduced these effects and eliminated additional CD35-EV benefit, confirming pathway specificity. In patients, complosome activation correlated with disease severity.

This study identifies intracellular complosome activation in monocytes as a key driver of SA-AKI and multi-organ injury. It further demonstrates the therapeutic potential of extracellular vesicles as nanocarriers for targeted delivery of CD35, disrupting complosome-dependent monocyte chemotaxis and tissue infiltration. Collectively, these findings establish a mechanistically defined EV-based intervention strategy and provide a foundation for targeting intracellular complement in inflammatory disease. This reshapes current understanding of complement biology, expanding it beyond extracellular pathways and positioning the complosome as a tractable target for precision immunomodulation in sepsis and related organ dysfunction.



CD35-EV administration attenuated renal and multi-organ injury, improved survival, and limited monocyte infiltration



PPIs Linked to Fewer Upper GI Bleeds in Patients on Haemodialysis

NEW DATA presented at ERA 2026 has demonstrated that proton pump inhibitors (PPI) cut the risk of upper gastrointestinal bleeding by nearly 40% in patients on haemodialysis on oral anticoagulants.[10]

Patients on haemodialysis carry a high risk of upper gastrointestinal bleeding (UGIB), and that danger climbs once an oral anticoagulant is added. PPIs are widely prescribed, but their ability to avert bleeding in these patients has never been firmly established.

The investigators emulated a target trial using the French Renal Epidemiology and Information Network (REIN) registry, linked to the national health database. Eligible adults on haemodialysis had started an oral anticoagulant between January 2012–December 2023, with no PPI dispensed in the prior 3 months. They were followed for 12 months or until a first UGIB event, death, kidney transplantation, dialysis cessation, anticoagulant discontinuation, or loss to follow-up. The primary outcome, a first UGIB event, was identified through 10th revision of the International Classification of Diseases (ICD-10) hospital discharge codes. Risk in PPI users versus non-users was estimated with inverse probability of treatment weighting and a cause-specific Cox model, plus an as-treated secondary analysis. Of 8,205 eligible patients, 7,503 were included, 2,323 starting a PPI the same day as their anticoagulant.

Over a median follow-up of 1 year, 145 patients experienced a first UGIB event, with melena most common at 38% of cases. After weighting, the 12-month cumulative incidence of UGIB was slightly lower among PPI users than non-users. The risk reduction was statistically significant, with a weighted hazard ratio of 0.62 (95% CI: 0.41–0.95). The as-treated secondary analysis of PPI initiation versus no initiation gave a closely matching weighted hazard ratio of 0.63 (95% CI: 0.39–0.99). Subgroup and sensitivity analyses, though underpowered, aligned with the main result.

The authors concluded that PPIs are linked to a lower risk of upper gastrointestinal bleeding after patients on haemodialysis begin oral anticoagulation, supplying rare evidence for a protective approach in this high-risk group. They cautioned that PPIs carry their own potential for serious harm, urging clinicians to review such prescriptions regularly rather than indefinitely.

References

1. Josse M et al. Biphasic cardioprotection by pre-dialysis exercise in hemodialysis patients: a randomized controlled trial. Abstract 2891. ERA Congress, 3-6 June, 2026.
2. Chakraborty R et al. Individualised torque teno virus trajectories predict rejection and opportunistic infection early after kidney transplantation. Abstract 114. ERA Congress, 3-6 June, 2026.
3. Berti GM et al. Five-year prognostic performance of the Mayo Clinic Chronicity Score in kidney transplant biopsies. Abstract 1381. ERA Congress, 3-6 June, 2026.
4. Créon A et al. Predictive performance and clinical utility of the kidney failure risk equation using EKFC versus CKD-EPI estimated GFR. Abstract 1377. ERA Congress, 3-6 June, 2026.
5. Tabinor M et al. Risk stratification in chronic kidney disease: outcomes using cystatin-C and creatinine based eGFR. Abstract 912. ERA Congress, 3-6 June, 2026.
6. Yang C et al. The obesity paradox is primarily driven by decreased non-cardiovascular mortality risk among the oldest old with chronic kidney disease. Abstract 1362. ERA Congress, June 3-6, 2026.
7. Laranjinha I et al. Air pollution and chronic kidney disease progression in Portugal - IMPA-R-PT study (impact of air pollution on renal health in Portugal). Abstract 1201. ERA Congress, 3-6 June, 2026.
8. Barrenechea-Barrenechea JA et al. Myeloid HIF-2α: a key player in immune crosstalk and renal fibrosis. Abstract 442. ERA Congress, 3-6 June, 2026.
9. Li N et al. CD35-EVs ameliorate sepsis-associated AKI and multi-organ injury by regulating monocyte complosome activation. Abstract 1784. ERA Congress, 3–6 June, 2026.
10. Laville S et al. Proton pump inhibitors reduce upper gastrointestinal bleeding in hemodialysis patients initiating oral anticoagulation: a nationwide registry study. Abstract 637. ERA Congress, 3-6 June, 2026.