Received: 29 July 2024 | Revised: 13 October 2024 | Accepted: 14 October 2024

DOI: 10.1111/iwj.70122

ORIGINAL ARTICLE

IWJ WILEY

The dialysis foot- the impact of presenting estimated glomerular filtration rate on clinical outcomes in patients hospitalized with diabetic foot infections

Arthur Tarricone[1] | **Tyler L. Coye**[2] | **Allen Gee**[3] | **Bijan Najafi**[2] | **Michael C. Siah**[4] | **Lawrence A. Lavery**[1,5]

[1]Department of Orthopedic Surgery, The University of Texas Health Science Center, San Antonio, Texas, USA

[2]Michael E DeBakey Department of Surgery, Baylor College of Medicine, Houston, Texas, USA

[3]Nova Southeastern University Dr. Kiran C. Patel College of Osteopathic Medicine, Davie, Florida, USA

[4]Department of Surgery, University of Texas Southwestern Medical Center, Dallas, Texas, USA

[5]Department of Plastic Surgery, University of Texas Southwestern, Dallas, Texas, USA

Correspondence
Arthur Tarricone, Department of Orthopedic Surgery, The University of Texas Health Science Center, San Antonio, TX, USA.
Email: tarria01@outlook.com

Abstract

To evaluate the association between presenting estimated glomerular filtration rate (eGFR) and clinical outcomes in patients hospitalized with diabetic foot infections. This retrospective cohort study included 344 patients with moderate to severe diabetic foot infections. Patients were categorized into three groups based on presenting estimated eGFR: eGFR ≥60 (eGFR >60 mL/min), eGFR 30–60 (eGFR 30–60 mL/min) and eGFR <30 (eGFR <30 mL/min). Outcomes assessed included wound healing, time to heal, re-infection, amputation, mortality and re-hospitalization for infection. Compared with patients with eGFR <30, patients with eGFR ≥60 had significantly lower rates of retinopathy, peripheral arterial disease and use of beta blockers or calcium channel blockers. Glycated haemoglobin levels were inversely related to eGFR, decreasing as eGFR severity increased. Haemoglobin levels were significantly lower, and inflammatory markers (ESR and CRP) were significantly higher in patients with eGFR <30. There were no significant differences among eGFR groups in rates of wound healing, time to heal, re-infection or amputation. However, mortality increased with decreasing eGFR (1.9% in eGFR ≥60 vs. 3.2% in eGFR 30–60 vs. 8.1% in eGFR <30; $p = 0.04$). Similarly, re-hospitalization for infection at a different site also increased with decreasing eGFR (20.5% in eGFR ≥60 vs. 28.1% in eGFR 30–60 vs. 48.4% in eGFR <30; $p < 0.01$). In diabetic foot infections, presenting eGFR severity did not affect rates of wound healing, time to heal, re-infection or amputation. However, decreasing eGFR was associated with increased mortality and re-hospitalization for infection at a different site. In this study, presenting eGFR was not a predictive value for wound healing or time until healing, however was

associated with rehospitalization and overall mortality this diabetic foot population.

KEYWORDS
chronic kidney disease, diabetic foot infection, eGFR, foot, osteomyelitis

Key Messages

- Decreased eGFR is associated with higher mortality and increased risk of re-hospitalization for infections, indicating that reduced kidney function significantly impacts clinical outcomes beyond wound healing alone.
- Presenting eGFR levels did not significantly influence wound healing rates, healing time, re-infection, or amputation rates in patients hospitalized with diabetic foot infections, suggesting that while eGFR affects broader outcomes like survival and re-hospitalization, it may not directly impact the wound healing process itself.

1 | INTRODUCTION

Diabetes mellitus (DM) affects over 10% of the United States population. The prevalence increases in the elderly population to approximately 30% in people over the age of 65.[1] It has been estimated that 19%–24% of patients with diabetes will develop a foot ulcer in their lifetime. Developing a foot ulcer is a pivotal event associated with increased risks of infection, amputation and death.[2–4] Diabetes related foot complications are the leading cause of non-traumatic lower extremity amputations and are a significant source of disability and mortality with 5-year mortality rates reported as high as 70%.[5]

The causal pathway to amputation is multivariable and has been associated with social determinants of health including sex, race and socioeconomic status as well as physiologic factors such as chronic kidney disease (CKD), peripheral artery disease (PAD) and diabetes mellitus (DM).[6,7] CKD is a significant risk factor for infection, amputation and re-amputation.

This is due to a complex interplay of factors in CKD patients including increased prevalence and severity of peripheral arterial disease (PAD) and peripheral neuropathy. This makes foot complications more likely to develop and less likely to be detected early, increasing the risk of amputation and mortality. The aim of this study is to evaluate presenting estimated Glomerular Filtration Rate (eGFR) and diabetic foot outcomes predictor.

2 | METHODS

This is a retrospective cohort study comparing the effect of presenting eGFR on clinical outcomes in patients admitted to hospital with moderate or severe diabetic foot infection. This study was approved by the institutional review board. Clinical, demographic and intraoperative parameters we collected on all subjects. The patients were categorized into three groups based on their presenting eGFR: patients with: eGFR >60 mL/min, eGFR 30–60 mL/min and eGFR <30: GFR <30 mL/min. The primary endpoint of the study was to determine the effect of presenting eGFR with wound healing and the time required for healing in patients with diabetic foot infections. Data was collected from patients' index hospitalization and at standard intervals over a 12-month period. For the index hospitalization, we assessed infection severity, surgeries, amputations, vascular interventions and length of stay. During the 1-year follow-up, we evaluated wound healing, re-infection, amputation, surgery, readmissions, length of hospitalizations and mortality. We assessed co-morbidities, baseline lab data history of ulceration and amputation. The diagnosis of diabetes was based on the American Diabetes Association criteria. Peripheral arterial disease (narrowing of the arterial walls) was defined as non-compressible vessels at the ankle, ankle-brachial index <0.9, claudication symptoms or history of vascular surgery in the extremity. Sensory neuropathy was defined as abnormal vibration sensation or abnormal sensation with 10-g Semmes-Weinstein monofilament exam. We used the International Working Group of the Diabetic Foot Infection Classification to define the presence and severity of infection.[8,9]

2.1 | Statistical analysis

Continuous variables were presented as mean ± standard deviation, and categorical variables were presented as

n (percentage). Shapiro–Wilk test was used to determine normality for wound subjects ($n \leq 50$). Skewed variables were presented as median [IQR] (mean). Pearson's chi square was used to compare dichotomous variables and t-test to compare continuous variables. Mann–Whitney U test was applied for non-normally distributed data and findings were reported as Median [IQR]. Statistical significance was indicated by p values ≤0.05. All laboratory and demographic information were determined from EPIC patient medical records and procedural characteristics were extracted from catheterization reports. All statistical analyses were completed using STATA(BE18) College Station, TX.

3 | RESULTS

Among 344 patients with diabetic foot infections, 219 patients had presenting eGFR ≥60; 63 patients had eGFR 30–60; and 62 had eGFR <30. Table 1 includes demographics and clinical outcomes. Significantly differences were found in sex across presenting eGFR categories (eGFR ≥60: $n = 174$ (males = 79.4%), eGFR 30–60: $n = 43$ (males = 68.2%), eGFR <30: $n = 38$ (males = 61.3%); $p = 0.008$). eGFR <30 patients had significantly higher rates of retinopathy (22.8% vs. 34.9% vs. 41.9%; $p = 0.006$), PAD (19.2% vs. 74.6% vs. 87.1%; $p = 0.002$) use of beta blockers (27.4% vs. 45.3% vs. 74.2%;

TABLE 1 Comparison of demographic data based on eGFR severity.

Variable	eGFR ≥ 60 (*N* = 219)	eGFR 30–60 (*N* = 63)	eGFR < 30 (*N* = 62)	*p*-value*
Age, years of age	54 [47; 61]	53 [43; 61]	56 [46; 65]	0.43
BMI, kg/m^2	30 [25; 36]	33 [28; 38]	32 [27; 38]	0.12
Male	174 (79.4%)	43 (68.2%)	38 (61.3%)	0.008
Medical history				
Retinopathy	50 (22.8%)	22 (34.9%)	26 (41.9%)	0.006
Sensory neuropathy	194 (88.6%)	58 (92.0%)	59 (95.2%)	0.30
PAD	42 (19.2%)	47 (74.6%)	54 (87.1%)	0.002
Previous amputation	77 (35.2%)	29 (46.0%)	22 (35.4%)	0.30
Monkenberg sclerosis	83 (37.9%)	41 (65.1%)	47 (75.8%)	<0.001
Previous ulceration	140 (63.9%)	47 (74.6%)	36 (58.1%)	0.14
Dialysis	1 (0.5%)	2 (3.1%)	33 (53.2%)	<0.001
Medications				
Insulin	172 (78.5%)	57 (89.1%)	46 (74.2%)	0.076
Steroid oral/inhaled	5 (2.3%)	5 (7.8%)	4 (6.5%)	0.234
Beta blocker	60 (27.4%)	29 (45.3%)	46 (74.2%)	<0.001
Calcium channel blocker	45 (20.5%)	26 (40.6%)	31 (50.0%)	<0.001
Infection severity				
Severe infection (SIRS)	86 (39.3%)	37 (58.7%)	19 (30.6%)	0.501
Osteomyelitis	109 (49.8%)	35 (54.7%)	30 (48.4%)	0.70
Admission laboratory values				
Glycated haemoglobin	9.4 [7.6; 11.4]	8.6 [6.9; 10.3]	7.15 [6.1; 8.6]	<0.001
A1C >12	126 (57.5%)	15 (23.4%)	7 (11.3%)	<0.001
Haemoglobin	12.0 [10.9; 13.4]	10.3 [9.1; 11.6]	9.8 [8.5; 11.0]	<0.001
CRP, mg/dL	4.0 [1.2; 9.7]	4.0 [1.0; 10.0]	7.0 [2.1; 14.0]	0.09
ESR, mm/h	57 [38.0; 90.0]	88 [58.0; 129.0]	100.0 [70.0; 130.0]	<0.001
WBC, 10^9/L	9.5 [7.3; 12.5]	9.9 [8.0; 13.2]	10.2 [7.9; 14.1]	0.17
WBC >15 000	26 (11.9%)	8 (12.7%)	13 (21.0%)	0.158
Albumin, g/dL	3.4 [3.1; 3.8]	3.15 [2.7; 3.8]	3.2 [2.9; 3.6]	0.02
Albumin <3.0	49 (22.3%)	28 (44.4%)	21 (33.9%)	0.005

Note: Continuous variables represented as median and quartiles 1 and 3. Categorical variables are represented as *n* and percentage.
*Wilcoxon rank sum test; Pearson's Chi-squared test.

$p < 0.001$) and calcium channel blockers (20.5% vs. 40.6% vs. 50.0%; $p < 0.001$). Glycated haemoglobin levels (9.4% vs. 8.6% vs. 7.2%; $p < 0.001$) and haemoglobin (12.0 mg/dL vs. 10.3 mg/dL vs. 9.8 mg/dL; $p < 0.001$) were significantly different in eGFR ≥60, eGFR 30–60 and eGFR <30, respectively. Inflammatory marker, erythrocyte sedimentation rate, was significantly higher (57 mm/h vs. 88 mm/h vs. 100 mm/h; $p < 0.001$), and C-reactive protein (CRP) was elevated in eGFR <30 compared with eGFR ≥60 and eGFR 30–60 (7.0 mg/L vs. 4.0 mg/L vs. 4.0 mg/L); however, this did not meet significance ($p = 0.07$). Mockenberg medical sclerosis (MMSC) was significantly different across groups (eGFR ≥60: $n = 83$ (37.9%) eGFR 30–60: $n = 41$ (65.1%), eGFR <30 $n = 47$ (75.8%); $p < 0.001$).

During the 1-year follow-up, there were no differences in the incidence wound healing (63.0% vs. 58.1% vs. 58.1%; $p = 0.70$), median time to wound healing (106 days vs. 155 days vs. 116 days; $p = 0.30$), re-infection (45.2% vs. 50.0% vs. 35.5%, $p = 0.33$) or amputation (36.6% vs. 48.4% vs. 37.1%, $p = 0.16$) between the three groups (Tables 2 and 3 and Figure 1).

However, patients with lower levels of presenting eGFR had higher mortality rates (1.9% vs. 3.2% vs. 8.1%; $p = 0.04$) and higher rates of hospital admission for infection at a different site (20.5% vs. 28.1% vs. 48.4%,

TABLE 2 Comparison of outcomes based on eGFR severity.

Variable	eGFR ≥60 ($N = 219$)	eGFR 30–60 ($N = 63$[a])	eGFR <30 ($N = 62$[a])	*p*-value*
Index hospitalization				
Vascular intervention	20 (9.2%)	7 (11.0%)	10 (16.0%)	0.30
Number of surgeries	2.0 [1; 3]	2.0 [1; 4]	2.0 [1; 3]	0.60
Any amputation	78 (35.6%)	29 (45.3%)	23 (37.1%)	0.33
Minor amputation	48 (21.9%)	14 (21.9%)	14 (22.6%)	0.98
Major amputation	30 (13.7%)	15 (23.4%)	9 (14.5%)	0.16
Length of hospitalization (days)	21.1 [18.3, 23.8]	27.6 [21.2, 34.0]	26.7 [20.5, 32.8]	0.06
12 month follow-up				
Wound healing	138 (63.0%)	36 (58.1%)	33 (58.1%)	0.70
Days to heal	106 [53; 241]	155 [85; 240]	116 [56; 225]	0.30
Re-infection	99 (45.2%)	32 (50.0%)	22 (35.5%)	0.06
Hospital admission (Same)	99 (45.2%)	33 (51.5%)	22 (35.5%)	0.183
Hospital admission (Different)	45 (20.5%)	18 (28.1%)	30 (48.4%)	<0.001
Number of surgeries	2.0 [1; 3]	2.0 [1; 4]	2.0 [1; 3]	0.25
Any amputations	78 (36.6%)	31 (48.4%)	23 (37.1%)	0.60
Minor amputation (Foot)	64 (29.2%)	25 (35.9%)	18 (28.1%)	0.58
Major amputation (BKA-AKA)	14 (6.4%)	6 (9.4.%)	5 (8.2%)	0.68
Mortality	4 (1.9%)	2 (3.2%)	5 (8.1%)	0.04

Abbreviation: eGFR, presenting estimated glomerular filtration rate.
*Wilcoxon rank sum test; Pearson's Chi-squared test.
[a]Continuous variables represented as median and quartiles 1 and 3. Categorical variables are represented as *n* and percentage.

TABLE 3 Intergroup analysis of significant 12-month outcomes: The analysis compared eGFR ≥60 to eGFR 30–60 and eGFR <30 and eGFR (30–60)to eGFR <30. The association of mortality between eGFR <30 compared with eGFR ≥60 was OR: 2.17 (1.11–4.27, $p = 0.02$). The hospital readmission for a different site association between eGFR <30 compared with eGFR ≥60 was OR: 1.92 (1.42–2.56, $p = 0.0$). The readmission association between eGFR 4/5 compared with eGFR 3 was OR:2.42 (1.15–5.08, $p = 0.2$).

Variables	eGFR > 60 vs. eGFR 30–60	eGFR > 60 vs. eGFR < 30	eGFR 30–60 vs. eGFR < 30
Mortality	OR = 1.73 [0.31–9.71] $p = 0.524$	OR = 2.17 [1.11–4.27] $p = 0.024$	OR = 2.72 [0.508–14.60] $p = 0.242$
Hospital admission different	OR = 1.52 [0.80–2.88] $p = 0.198$	OR = 1.92 [1.42–2.56] $p = 0.000$	OR = 2.42 [1.15–5.08] $p = 0.020$

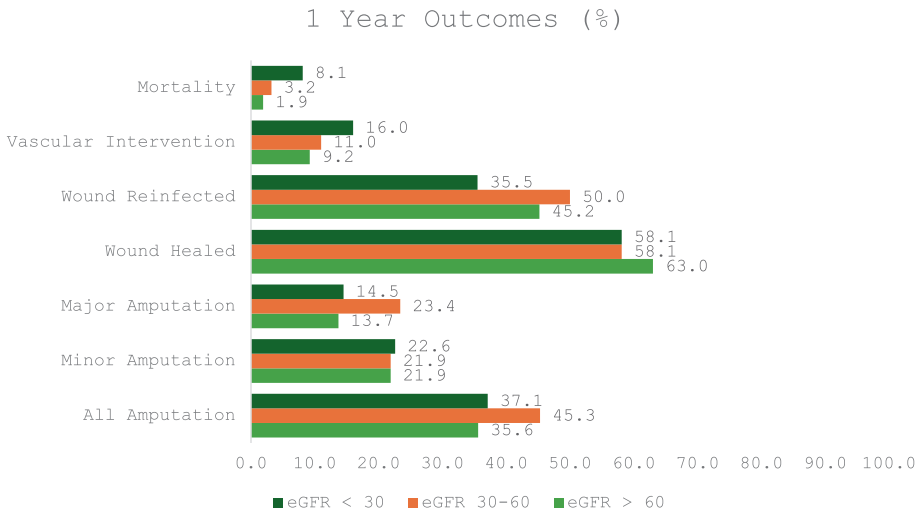

FIGURE 1 Bar chart of 1 year outcomes by eGFR classification. All numbers beside bars indicate percentages. Variables are listed on the Y axis. The X axis is percent. Green bars represent eGFR >60, red bars represent eGFR 30–60 and blue bars represent eGFR <30. *p* values for each variables are as follows: Mortality ($p = 0.04$), Vascular Intervention ($p = 0.30$), Wound Re-infection ($p = 0.06$), Wound Healed ($p = 0.70$), Minor amputation ($p = 0.98$), Major amputation ($p = 0.16$), and All amputation ($p = 0.33$).

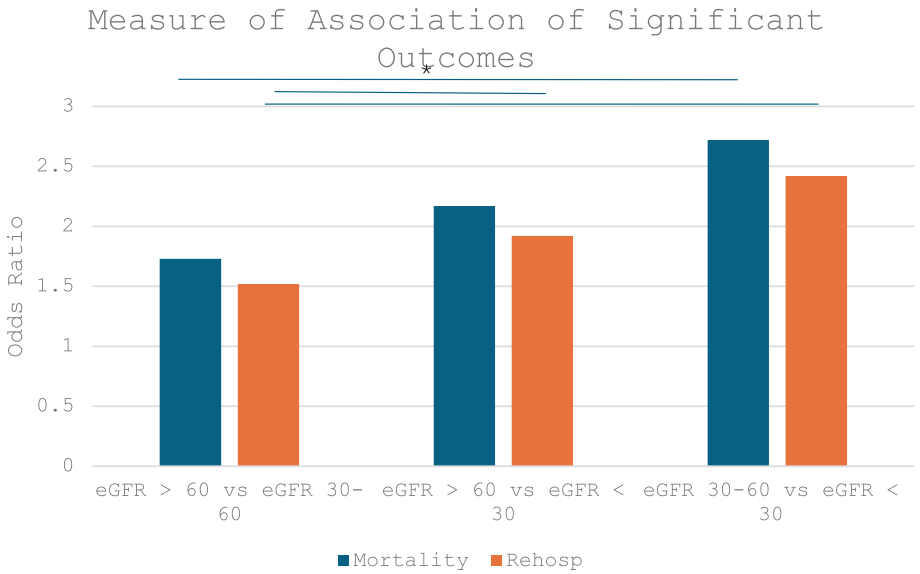

FIGURE 2 Bar graph presenting the measure of Association (odds ratios) for significant outcomes. Blue bars represent the outcome of mortality, orange bars represent the outcome of re-hospitalization for the different site. Comparisons were made between eGFR >60 versus eGFR 30–60, eGFR >60 versus eGFR <30 and eGFR 30–60 versus eGFR <30. The Y axis represents odds ratios.

$p < 0.01$). Re-hospitalization for the same site of due to infection was not significant between any eGFR group (45.2% vs. 51.5% vs. 35.5%, $p = 0.183$). Figure 2 shows the bar charts for 1-year clinical outcomes. Figure 3 depicts the Kaplan–Meier Survival analysis for wound healing. There was no difference in the time to heal (106 days vs. 155 days vs. 116d, $p = 0.30$) based on eGFR (Figure 3).

An intergroup analysis was performed on the significant 12-month outcomes of this study. The analysis compared eGFR ≥60 with eGFR <30 and eGFR 30–60 with eGFR <30. The association of mortality between eGFR <30 compared with eGFR ≥60 was OR: 2.17 (1.11–4.27, $p = 0.02$). The hospital readmission for a different site association between eGFR <30 compared with eGFR ≥60 was OR: 1.92 (1.42–2.56, $p = 0.0$). The readmission for a different site association between eGFR <30 compared with eGFR 30–60 was OR:2.42 (1.15–5.08, $p = 0.02$). All other comparisons between eGFR groups for these two variables were not significant.

4 | DISCUSSION

The results of this paper indicate that presenting eGFR may not be a significant predictor of wound healing, or the time required for healing in patients with moderate and severe diabetic foot infections. We did not find a difference in healing or median days to heal between the three groups. Even though there was a significantly higher prevalence of PAD in people with eGFR <30, there was no difference in vascular surgeries or the incidence or level of lower extremity amputation. These results contrast most other studies that report higher rates of ulcers, amputations and death as eGFR decreases. Most other studies addressing this topic have

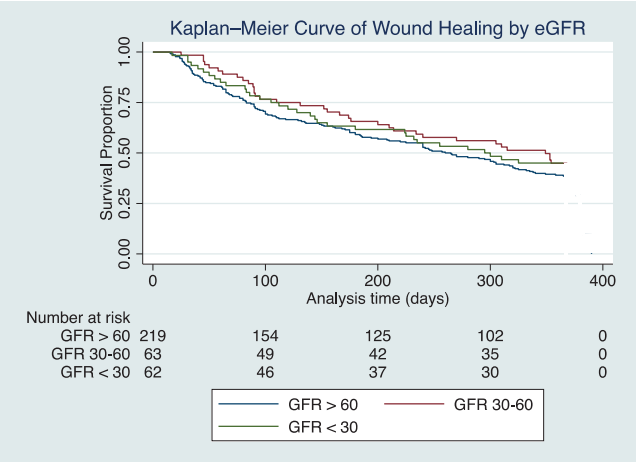

FIGURE 3 Kaplan–Meier Curve for Wound Healing by CKD Level. Kaplan–Meier Curve for wound healing by eGFR. The Y axis represents the proportion of wounds healed and the X axis represents the time (days). eGFR was categorized by GFR <30 (blue), GFR 30–60 (green) and GFR >60 (red). There was no significant difference in time until wound healing between each group ($p = 0.365$).

indicated that impaired renal function, as reflected by a decreased eGFR, is associated with adverse outcomes in patients with foot ulcers and infections. Epidemiology studies have consistently shown much higher amputation incidence rates, more proximal amputations and higher mortality incidence in people with decreased eGFR and in end-stage renal disease (ESRD).[10–13] It is unclear from our data if there was a significant difference in the severity of disease or if our ability to stratify disease severity was adequate.

There are several co-morbidities that were more common in people with worsening eGFR such as retinopathy, PAD and anaemia. Perhaps the most important factor associated with wound healing is the presence and severity of peripheral arterial disease. There was a significantly higher prevalence of PAD in subjects with eGFR<30; however, there was also a high rate of arterial calcification in all three study groups, so ABIs were not reliable for many of our subjects.[14] In addition, there was no difference in the prevalence of revascularization procedures. Unfortunately, we did not collect data for toe pressures or toe brachial indices or if patients were more likely to have direct or indirect revascularization for this retrospective study. This may have given us better insights into perfusion.

The correlation between increasing glycated haemoglobin and microvascular disease, macrovascular complications and mortality have been reported in several studies.[15–17] However, CKD obscures this relationship. Glycated haemoglobin is known to represent blood glucose levels across a period of up to 3 months and is determined by three factors: circulating blood glucose, the kinetics of haemoglobin and the length of time of interaction between the two.[18] In CKD patients, there is an array of pathological abnormalities that alter the components of glycated haemoglobin such as a shorter red blood cell (RBC) half-life and diminished erythropoietin production.[19,20] These changes create an anaemic state that results in less interaction time, and thereby less glycosylation of haemoglobin by blood glucose. Studies have shown the resultant effect as a weaker correlation between glycated haemoglobin levels and true blood glucose in CKD patients.[18–20] Dialysis patients exhibit similar changes, with fluctuating RBC half-life and varying interaction times between haemoglobin and glucose.[21] These mechanisms have presented clinically, with many studies finding no association between glycated haemoglobin and wound healing, amputations, re-infection or mortality.[21–24]

The difference in our clinical outcomes compared with other studies may be explained by the interventions of a multidisciplinary diabetic limb salvage service. There are many examples of improved outcomes such as reduced lower amputations, hospitalizations and emergency room visits in high-risk populations when a collaborative team provides care.[25–30] In our hospital, the diabetic limb salvage service is consulted to see patients in the emergency room before patients are admitted, so additional consultations and interventions can be initiated early in the admission process. Chung and colleagues described the reduction in amputations and mortality by our multidisciplinary service in a cohort of CLI patients compared with people treated with standard care.[31]

Our infectious diseases service developed an OPAT (out patient antimicrobial therapy) programme to provide self-administered intravenous antibiotics at home for people with osteomyelitis.[32] An extension of the hospital service is the transition back to outpatient specialty clinics. Podiatry and physical therapy participate in a dedicated wound clinic. This type of format provides continuity of care and communication among team members to improve the transition from hospital to clinic. Multidisciplinary amputation prevention programmes report 39%–56% reduction in amputation.[33,34] Specialty foot care has also been demonstrated to be effective in people with diabetes and end stage renal disease.[35,36] The results of this study are an extension of the results reported by Chung and Bhavan with a focus on CKD subpopulations.

There are advantages and limitations in this study. We included eGFR <30 in the highest risk group rather than just people with ESRD (stage 5). The number of subjects in presenting eGFR 30–60 and <30 were

disproportionately small compared with the subjects with a presenting eGFR >60. This may introduce risk for a potential Type 2 error. Our hospital serves a disproportionately poor, minority population that is uninsured or underinsured. This can limit access to medical services after hospital discharge such as home intravenous antibiotic services, physical therapy and negative pressure wound therapy. We only included study subjects that had at least 1 year of follow-up after their index hospitalization. We probably lost patients that had good clinical outcomes because they did not require long-term, continued medical care.

5 | CONCLUSION

In diabetic foot infections, presenting eGFR severity did not affect rates of wound healing, time to heal, re-infection or amputation. However, eGFR was associated with increased mortality and re-hospitalization for infection at a different site.

While eGFR severity may not directly influence wound healing or days to healing in patients with moderate and severe diabetic foot infections, it does have implications for peripheral arterial disease and readmission rates. These findings may underscore the significance of comprehensive management strategies that address both renal and foot complications in diabetic patients with compromised eGFR. Further research is warranted to evaluate the effectiveness of diabetic limb salvage programmes in high-risk patients with eGFR and ESRD.

CONFLICT OF INTEREST STATEMENT

The authors report no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data available on request due to privacy/ethical restrictions.

ORCID

Arthur Tarricone https://orcid.org/0000-0001-5311-6283

REFERENCES

1. Barnes JA, Eid MA, Creager MA, Goodney PP. Epidemiology and risk of amputation in patients with diabetes mellitus and peripheral artery disease. *Arterioscler Thromb Vasc Biol*. 2020;40(8):1808-1817.
2. Armstrong DG, Boulton AJM, Bus SA. Diabetic foot ulcers and their recurrence. *N Engl J Med*. 2017;376(24):2367-2375.
3. Hoffstad O, Mitra N, Walsh J, Margolis DJ. Diabetes, lower-extremity amputation, and death. *Diabetes Care*. 2015;38(10):1852-1857.
4. Meloni M, Izzo V, Giurato L, Lazaro-Martinez JL, Prevalence UL. Clinical aspects and outcomes in a large cohort of persons with diabetic foot disease: comparison between neuropathic and ischemic ulcers. *J Clin Med*. 2020;9(6).
5. McLatchie GR, McDonald M, Lawrence GF, Rogmans D, Lisai P, Hibberd M. Soft tissue trauma: a randomised controlled trial of the topical application of felbinac, a new NSAID. *Br J Clin Pract*. 1989;43(8):277-280.
6. Tarricone A, Gee A, De La Mata K, et al. Health disparities in nontraumatic lower extremity amputations. A systematic review and meta-analysis. *Ann Vasc Surg*. 2023;88:410-417.
7. Lavery LA, Suludere MA, Ryan E, et al. The infected diabetic foot: analysis of diabetic and non-diabetic foot infections. *Wound Repair Regen*. 2024;32:360-365.
8. Lipsky BA, Senneville E, Abbas ZG, et al. Guidelines on the diagnosis and treatment of foot infection in persons with diabetes (IWGDF 2019 update). *Diabetes Metab Res Rev*. 2020;36-(Suppl 1):e3280.
9. Senneville E, Albalawi Z, van Asten SA, et al. IWGDF/IDSA guidelines on the diagnosis and treatment of diabetes-related foot infections (IWGDF/IDSA 2023). *Clin Infect Dis*. 2023;40(3):1–25.
10. Lavery LA, Hunt NA, Ndip A, Lavery DC, Van Houtum W, Boulton AJ. Impact of chronic kidney disease on survival after amputation in individuals with diabetes. *Diabetes Care*. 2010;33(11):2365-2369.
11. Ndip A, Lavery LA, Boulton AJ. Diabetic foot disease in people with advanced nephropathy and those on renal dialysis. *Curr Diab Rep*. 2010;10(4):283-290.
12. Ndip A, Rutter MK, Vileikyte L, et al. Dialysis treatment is an independent risk factor for foot ulceration in patients with diabetes and stage 4 or 5 chronic kidney disease. *Diabetes Care*. 2010;33(8):1811-1816.
13. Lavery LA, Lavery DC, Hunt NA, La Fontaine J, Ndip A, Boulton AJ. Amputations and foot-related hospitalisations disproportionately affect dialysis patients. *Int Wound J*. 2015;12(5):523-526.
14. Suludere MA, Danesh SK, Killeen AL, et al. Monckeberg's medial calcific sclerosis makes traditional arterial Doppler's unreliable in high-risk patients with diabetes. *Int J Low Extrem Wounds*. 2023;31(4):542–546.
15. Mohr DC, Zhang L, Prentice JC, et al. Association of hemoglobin A1c time in range with risk for diabetes complications. *BMJ Open Diabetes Res Care*. 2022;10(4):e002738.
16. Paprott R, Schaffrath Rosario A, Busch MA, et al. Association between hemoglobin A1c and all-cause mortality: results of the mortality follow-up of the German National Health Interview and examination survey 1998. *Diabetes Care*. 2015;38(2):249-256.
17. Riddle MC, Ambrosius WT, Brillon DJ, et al. Epidemiologic relationships between A1C and all-cause mortality during a median 3.4-year follow-up of glycemic treatment in the ACCORD trial. *Diabetes Care*. 2010;33(5):983-990.
18. Coelho S. What is the role of HbA1c in diabetic hemodialysis patients? *Semin Dial*. 2016;29(1):19-23.
19. Hoshino J, Mehrotra R, Rhee CM, et al. Using hemoglobin A1c to derive mean blood glucose in peritoneal dialysis patients. *Am J Nephrol*. 2013;37(5):413-420.
20. Uzu T, Hatta T, Deji N, et al. Target for glycemic control in type 2 diabetic patients on hemodialysis: effects of anemia and

erythropoietin injection on hemoglobin a(1c). *Ther Apher Dial*. 2009;13(2):89-94.
21. Okada T, Nakao T, Matsumoto H, et al. Association between markers of glycemic control, cardiovascular complications and survival in type 2 diabetic patients with end-stage renal disease. *Intern Med*. 2007;46(12):807-814.
22. Shima K, Komatsu M, Kawahara K, Minaguchi J, Kawashima S. Stringent glycaemic control prolongs survival in diabetic patients with end-stage renal disease on haemodialysis. *Nephrology (Carlton)*. 2010;15(6):632-638.
23. Williams ME, Lacson E Jr, Teng M, Ofsthun N, Lazarus JM. Hemodialyzed type I and type II diabetic patients in the US: characteristics, glycemic control, and survival. *Kidney Int*. 2006; 70(8):1503-1509.
24. Salinero-Fort MA, San Andres-Rebollo FJ, Cardenas-Valladolid J, et al. Glycemic variability and all-cause mortality in a large prospective southern European cohort of patients with differences in glycemic status. *PLoS One*. 2022;17(7): e0271632.
25. Anichini R, Zecchini F, Cerretini I, et al. Improvement of diabetic foot care after the implementation of the international consensus on the diabetic foot (ICDF): results of a 5-year prospective study. *Diabetes Res Clin Pract*. 2007;75(2):153-158.
26. van Netten JJ, Price PE, Lavery LA, et al. Prevention of foot ulcers in the at-risk patient with diabetes: a systematic review. *Diabetes Metab Res Rev*. 2020;32(Suppl 1):84-98.
27. Lavery LA, Lavery DC, Hunt NA, Fontaine J, Lavery RD. Does the start of dialysis initiate a period of increased risk of ulceration or amputation? *J Am Podiatr Med Assoc*. 2018; 108(1):1-5.
28. Lavery LA, Wunderlich RP, Tredwell JL. Disease management for the diabetic foot: effectiveness of a diabetic foot prevention program to reduce amputations and hospitalizations. *Diabetes Res Clin Pract*. 2005;70(1):31-37.
29. Broersma A. Preventing amputations in patients with diabetes and chronic kidney disease. *Nephrol Nurs J*. 2004;31(1):53-62; quiz 3–4.
30. van Netten JJ, Raspovic A, Lavery LA, et al. Prevention of foot ulcers in the at-risk patient with diabetes: a systematic review. *Diabetes Metab Res Rev*. 2020;36(Suppl 1):e3270.
31. Bhavan KP, Brown LS, Haley RW. Self-administered outpatient antimicrobial infusion by uninsured patients discharged from a safety-net hospital: a propensity-score-balanced retrospective cohort study. *PLoS Med*. 2015;12(12):e1001922.
32. Chung J, Modrall JG, Ahn C, Lavery LA, Valentine RJ. Multidisciplinary care improves amputation-free survival in patients with chronic critical limb ischemia. *J Vasc Surg*. 2015;61(1):162-169.
33. Albright RH, Manohar NB, Murillo JF, et al. Effectiveness of multidisciplinary care teams in reducing major amputation rate in adults with diabetes: a systematic review & meta-analysis. *Diabetes Res Clin Pract*. 2020;161:107996.
34. Monteiro-Soares M, Vale-Lima J, Martiniano J, Pinheiro-Torres S, Dias V, Boyko EJ. A systematic review with meta-analysis of the impact of access and quality of diabetic foot care delivery in preventing lower extremity amputation. *J Diabetes Complications*. 2021;35(4):107837.
35. King LB. Impact of a preventive program on amputation rates in the diabetic population. *J Wound Ostomy Continence Nurs*. 2008;35(5):479-482; quiz 83–4.
36. Ahmad Z. Putting your best foot forward: focusing on preventive foot care in hemodialysis patients. *Nephrol News Issues*. 2009;23(6):25.

How to cite this article: Tarricone A, Coye TL, Gee A, Najafi B, Siah MC, Lavery LA. The dialysis foot- the impact of presenting estimated glomerular filtration rate on clinical outcomes in patients hospitalized with diabetic foot infections. *Int Wound J*. 2025;22(5):e70122. doi:10.1111/iwj.70122