Characteristics, trends and outcomes in acute coronary syndromes in the renal replacement therapy population: a 10-year retrospective analysis of the Midlands Region, New Zealand

Lance Ng, Tze Vun Liew, Andrew Henderson

ABSTRACT

Acute coronary syndrome (ACS) is one of the leading causes of mortality in the renal replacement therapy (RRT) population. We aimed to understand the characteristics, trends and outcomes of ACS in our local RRT population as a means to improve care and outcomes for this high-risk population. Using the ANZACS-QI database, we conducted a retrospective analysis of all ACS occurring in RRT patients between 1 January 2010–31 December 2019 managed at Waikato Hospital (n=135 at index ACS). In our cohort made up predominantly of Māori (55%) and European (34%) patients, 58% had diabetic nephropathy as their primary disease. Twenty-seven percent presented atypically and 65% had a delay of >72 hours from diagnosis to angiogram. There was a 49% mortality rate at one year post-index ACS. Factors associated with mortality at one year included: atypical presentation (chi-square statistic (X^2) 7.250; p=0.0071), troponin delta >20% (X^2 5.682; p=0.0171), peak troponin (point biserial correlation; r=0.2086; p=0.0473) and no revascularisation (X^2 5.2419; p=0.0221). The findings in our cohort reiterate that patients on RRT are a vulnerable population who have poor outcomes associated with ACS, driven by multifactorial delays in diagnosis and treatment.

Acute coronary syndrome (ACS) is one of the leading causes of mortality in the renal replacement therapy (RRT) population.[1–4] Using the ANZACS-QI database, we conducted a retrospective analysis of all acute coronary syndromes occurring in those documented to be receiving long-term dialysis at the time of their recorded acute coronary event over the last 10 years. We sought to understand the characteristics and trends in our local dialysis population as a means to improve care and outcomes for this high-risk population.

Methods

We used the ANZACS-QI database to identify a cohort of patients who were receiving long-term dialysis at the time of their index acute coronary syndrome. These patients were domiciled in the Midlands Region (Waikato, Taranaki, Bay of Plenty, Lakes and Tairawhiti District Health Boards) and a 10 year interval (between 1 January 2010–31 December 2019) was subsequently assessed. The All New Zealand Acute Coronary Syndrome Quality Improvement

(ANZACS-QI) registry is a rigorously maintained and audited web-based electronic database which captures a wide array of data on each ACS event across New Zealand's public hospitals. The specific data flows, outputs and cohorts have been previously described.[5]

Of 8,633 patients admitted to Midlands Region hospitals with ACS in the prespecified 10-year period, 135 were noted to be on dialysis at the time of index ACS. Of this 135, 18 were not on dialysis long-term, five were non-ACS diagnoses and 10 were repeat entries into the database. Therefore, a total 102 cases were included in the final analysis.

Our analysis included ST-elevation myocardial infarctions (STEMIs), non ST-elevation myocardial infarctions (NSTEMIs) and unstable angina under the umbrella term of acute coronary syndrome (ACS) as defined by the 4th Universal Definition of Myocardial Infarction.[6–7] Typical chest pain consistent with acute coronary syndrome in our cohort was defined as having at least two features of: (i) central chest pain; (ii) radiation to arm or neck; (iii) diaphoresis; (iv) associated with exertion or if it was documented to be "cardiac-sounding".

Comparative data from our cohort in comparison to a New Zealand-wide sample was derived from the Australia and New Zealand Dialysis and Transplant Registry (ANZDATA).[8–9] Renal transplantation candidacy was defined by having a National Renal Advisory Board (NRAB) score of greater than 80% at the time of the index ACS.[10]

Statistical analyses were undertaken for chi-squared test for association and the biserial correlation coefficient using Prism 8.

Results

Demographic data (Table 1)

Over half our sample consisted of patients of Māori ethnicity, with the second largest group consisting of those who identified as European. Our Māori cohort is relatively over-represented in the context of acute coronary syndromes in comparison to the Australia and New Zealand Dialysis and Transplant Registry (ANZDATA) database, whereby Māori patients make up 32% of the New Zealand dialysis population (as of 2019).[5]

The age distribution of the cohort followed a relatively bell-shaped curve, with a median age of 62 at time of first

Table 1: Demographic data of cohort.

Category	Number of cases (n) (%)	ANZDATA values (for NZ)
Ethnicity		
Māori	56 (54.9%)	32%
European	35 (34.3%)	28%
Pacific Islander	5 (4.9%)	28%
Asian	6 (5.9%)	9%
Age		
<40	9 (8.8%)	13%
40–49	8 (7.8%)	14%
50–59	21 (20.6%)	22%
60–69	45 (44.1%)	26%
70–79	18 (17.6%)	19%
>80	1 (1.0%)	6%
Gender		
Male	70 (68.6%)	60%
Female	32 (31.4%)	40%

acute coronary syndrome for those on renal replacement therapy. The distribution of the cohort is similar to that demonstrated in the ANZDATA database.[5]

Our cohort had a male predominance, which is reflective of the gender split overall in the New Zealand dialysis population.[5]

Renal-specific characteristics and outcomes (Table 2)

Across New Zealand in 2018, 54% of patients at time of RRT entry are classed as having type 2 diabetes.[5] This is reflected fairly similarly in our cohort. Concordantly, well over half of our cohort was noted as having diabetic nephropathy as their primary renal disease. This is in keeping with the ANZDATA registry, which identifies diabetic nephropathy as the primary aetiology in roughly 47% of cases in 2018.[5]

A greater proportion of our cohort received haemodialysis compared to peritoneal dialysis. Our Midlands cohort has a slightly higher proportion of peritoneal dialysis and in-centre (versus home) haemodialysis compared to the proportion seen across New Zealand.[6] Over 90% of our cohort had a dialysis vintage between 0–7 years with a mean vintage of 3.22 years across the cohort.

With regards to transplant status, just over one in six of the cohort were candidates for renal transplant at the time of their acute coronary syndrome.

From a biochemistry perspective, over two-thirds of the cohort who suffered an ACS while on long-term RRT had serum phosphates over the target range of 0.80–1.59mmol/L. Notably, less than half the cohort analysed had serum haemoglobins in the target range of 100–119g/L. Of those not within the target range, there tended to be a trend towards having a lower-than-target haemoglobin in our cohort (48% had Hb <100g/L at time of index ACS). Despite this, our cohort demonstrated a wide distribution of erythropoiesis stimulating agent (ESA) doses with the higher-end dosing (eg, 10,000 units three times per week) being relatively uncommon.

In terms of fluid status, approximately one in six were fluid-overloaded on clinical assessment at the time of their ACS event.

Cardiac-specific characteristics and outcomes (Table 3)

Over a quarter of presentations were deemed to be atypical in our cohort. Of the atypical presentations, dyspnoea and dizziness/syncope made up a large proportion.

Of the types of ACS, three-quarters of the cohort were defined as having NSTEMIs at the time of presentation.

Delay from diagnosis of ACS to time of angiography was recorded and demonstrated a wide range of wait times for coronary angiography; indeed, almost two-thirds of our cohort did not receive angiography within 72 hours of diagnosis of their ACS.

With regard to serum troponin (high-sensitivity troponin T; hsTnT) levels, there was a relatively broad distribution of peak troponins across the cohort. Peak troponin in our cohort was defined as the highest measured troponin within the admission for the ACS and does not have any temporal relation to the onset of symptoms. Of note is that 40% of the cohort did not have delta (change in) troponins of greater than 20%—a general threshold used in the Midlands region for identifying a dynamic change in troponin and ultimately in diagnosing type I myocardial infarctions.[6]

One-fifth of the cohort did not receive an assessment of left ventricular ejection fraction (LVEF) during their index ACS admission. Notably, with respect to the distribution of LVEF across the cohort, 60% had sub-normal LV function at the time of index ACS.

In our cohort, almost half had three-vessel disease (3VD) at the time of their index ACS. This being said, the majority of the cohort had either a conservative approach (medical therapy) (19.8%) or had percutaneous coronary intervention (PCI) (66.7%) and only a small proportion of our cohort received coronary artery bypass grafting (CABG) (13.6%)—irrespective of coronary anatomy.

One-third of the cohort suffered at least one further ACS. Of those who had at least one further ACS, 31/34 (91.2%) were initially managed with either medical therapy or PCI, compared to the 3/34 (8.8%) that were managed initially with CABG.

Table 2: Renal-specific characteristics and outcomes of cohort.

Category	Number of cases (n) (%)	ANZDATA values (for NZ)
Prevalence of diabetes		
None	35 (34.3%)	42%
Type 1	2 (2.0%)	4%
Type 2	65 (63.7%)	54%
Primary renal disease		
Diabetic	59 (57.8%)	47%
Hypertensive	6 (5.9%)	7%
Unknown/idiopathic	13 (12.7%)	4%
Reflux	3 (2.9%)	2%
Glomerulonephritis	11 (10.8%)	18%
Other	10 (9.8%)	16%
Modality of RRT		
Peritoneal	41 (40.2%)	30%
Home HD	13 (12.7%)	45%
In-centre HD	48 (47.1%)	18%
Vintage of dialysis		
<3 years	53 (52.5%)	-
>3 years	48 (47.5%)	-
Transplant candidacy at time of ACS		
Yes	17 (16.7%)	-
No	85 (83.3%)	-
Serum phosphate		
<1.59mmol/L	32 (31.4%)	-
>1.60mmol/L	69 (68.6%)	-
Serum Hb		
<99g/L	49 (48.0%)	-
100–119g/L	40 (39.2%)	-
≥120g/L	16 (15.7%)	-
ESA dose		
No ESA	28 (28.0%)	-
<10,000u/wk	41 (41.0%)	-
>10,000u/wk	31 (31.0%)	-
Fluid status		
Euvolaemic	16 (15.7%)	-
Overloaded	86 (84.3%)	-

RRT – renal replacement therapy; HD – haemodialysis; ACS – acute coronary syndrome; Hb – haemoglobin; ESA – erythropoiesis stimulating agent; u/wk – units per week.

Table 3: Cardiac-specific characteristics and outcomes.

Category	Number of cases (n) (%)
Nature of presentation (n=102)	
VF/VT arrest	7 (6.9%)
Typical	66 (64.7%)
Atypical	
- Dyspnoea	11 (10.8%)
- Dizziness/syncope	4 (3.9%)
- Musculoskeletal	2 (2.0%)
- Pleuritic	3 (2.9%)
- GI upset	3 (2.9%)
- Other	4 (3.9%)
Unclear	2 (2.0%)
Type of ACS (n = 102)	
Unstable angina	7 (6.9%)
NSTEMI	76 (74.5%)
STEMI	19 (18.6%)
Time from diagnosis of ACS to angiogram (n=89)	
0–24 hours	12 (13.5%)
24–48 hours	13 (14.6%)
48–72 hours	6 (6.7%)
>72 hours	58 (65.2%)
Peak troponin (hsTnT) (n=101)	
<1,000	54 (53.5%)
>1,000	47 (46.5%)
% Change in troponin (n=90)	
<20%	36 (40.0%)
>20%	54 (59.9%)
LVEF (%) (n=82)	
<40%	31 (37.8%)
>40%	51 (62.2%)
Coronary anatomy (n=90)	
NOCAD	9 (10.0%)
1VD	20 (22.2%)
2VD	19 (21.1%)
3VD	42 (46.7%)

Table 3: Cardiac-specific characteristics and outcomes (continued).

Revascularisation strategy (n=81)			
	Medical therapy	**PCI**	**CABG**
1VD	3 (3.7%)	16 (19.8%)	1 (1.2%)
2VD	1 (1.2%)	16 (19.8%)	2 (2.5%)
3VD	12 (14.8%)	22 (27.2%)	8 (9.9%)
Total	16 (19.8%)	54 (66.7%)	11 (13.6%)
Recurrent ACS (n=102)			
Yes		34 (33.3%)	
No		68 (66.7%)	
Angiogram-related complications (n=90)			
None		81 (90.0%)	
Yes			
- Vascular		5 (5.6%)	
- Other bleeding		2 (2.2%)	
- ADHF		2 (2.2%)	
Mortality at 1 month (n=102)			
Alive		85 (83.3%)	
Deceased		17 (16.7%)	
Mortality at 1 year (n=94)**			
Alive		48 (51.1%)	
Deceased		46 (48.9%)	

** 8 cases in most recent calendar year excluded.
VF – ventricular fibrillation; VT – ventricular tachycardia; GI – gastrointestinal; ACS – acute coronary syndrome; NSTEMI – non-ST-elevation myocardial infarction; STEMI – ST-elevation myocardial infarction; hsTnT – high sensitivity troponin T; LVEF – left ventricular ejection fraction; NOCAD – non-obstructive coronary artery disease; 1VD – one vessel disease; 2VD – two vessel disease; 3VD – three vessel disease; PCI – percutaneous coronary intervention; CABG – coronary artery bypass graft; ADHF – acute decompensated heart failure.

There was an overall complication rate associated with coronary angiography of 9% with a broad range of issues including vascular complications (perforation, dissection, pseudoaneurysm), bleeding and development of acute decompensated heart failure (ADHF).

In our cohort, there was a 17% mortality rate at one month post-index ACS and this increased to 49% at one year. Specific data on in-hospital mortality was not captured in our analysis.

Table 4 outlines the variables assessed in our cohort and their association with death at one year post-ACS. Atypical nature of presentation, peak troponin, % change in troponin (>20%) and no revascularisation being undertaken (by either PCI or CABG) were the parameters with statistically significant associations with death at one year.

Discussion

Our review into 10 years of data on recorded acute coronary syndromes in those on long-term dialysis has uncovered a number of valuable insights into the trends and outcomes of the dialysis population who experience a coronary event.

Table 4: Independent factors associated with death at one year post-index acute coronary syndrome event.

Independent factor	Chi square statistic (for association)	p-value
Renal-specific factors		
Māori vs non-Māori ethnicity	2.133	0.1442
PD vs HD	0.147	0.7016
Diabetic nephropathy vs other	2.859	0.0909
Vintage <3y vs Vintage >3y	2.074	0.1498
Transplant vs non-transplant candidate	0.089	0.7651
Target vs hyperphosphataemic	0.907	0.3408
Hb <100 vs 100–120 vs >120	0.961	0.6184
No ESA vs. ESA	0.560	0.4425
Cardiology-specific factors		
Atypical vs typical presentation	7.250	0.0071
STEMI vs NSTEMI/UA	0.1301	0.7183
Time to angio <72h vs >72h	0.2833	0.5945
Troponin delta >20% vs <20%	5.6842	0.0171
LVEF <40% vs >40%	0.2766	0.5989
3VD vs 1/2VD	0.1735	0.6770
No revascularisation vs any revascularisation (PCI or CABG)	5.2419	0.0221
PCI vs CABG	0.9376	.3329
	Point biserial correlation	**p-value**
Peak troponin	r=0.2086	0.0473

PD – peritoneal dialysis; HD – haemodialysis; Hb – haemoglobin; ESA – erythropoiesis stimulating agent; STEMI – ST-elevation myocardial infarction; NSTEMI – non-ST-elevation myocardial infarction; UA – unstable angina; LVEF – left ventricular ejection fraction; 3VD – three vessel disease; 2VD – two vessel disease; 1VD – one vessel disease; PCI – percutaneous coronary intervention; CABG – coronary artery bypass graft.

One of the most notable issues from a demographic perspective is the marked over-representation of Māori in our cohort. According to the ANZDATA registry, which collates data on the dialysis population across Australia and New Zealand, the dialysis population in New Zealand is made up approximately 33% Māori patients.[9] In contrast, our cohort was made up of 55% Māori patients—indeed, it would seem that at the convergence of two health-defining end points: end-stage renal failure and acute coronary syndromes—that Māori are heavily over-represented. This is likely a reflection of not only the demographic make-up of the Midlands region (which has a relatively higher representation from Māori and a relatively lower representation from Pacific Island and Asian/South Asian groups compared to areas such as Waitemata, Auckland and Counties Manukau),[11] but also the ongoing inequities and inequalities that exist within our health system. A recent kaupapa Māori analysis highlights that even when socioeconomic, demographic and geographical factors are equivalent, matched non-Māori patients still derive better outcomes from our health services across New Zealand.[12] It is clear that more needs to be done in Aotearoa/New Zealand to not only prevent disease but promote health in our Māori population.

From a renal perspective, the prevalence of diabetes and subsequent diabetic nephropathy as well as the balance of RRT modalities was relatively well-aligned with current ANZDATA findings.[9] Of note in our cohort was the strong tendency for sub-optimal phosphate control (66% were above target range phosphate)—a finding consistent across Australasia as noted in the ANZDATA Registry.[13] This trend is consistent with the contemporary understanding of hyperphosphataemia as an independent cardiovascular risk factor in those with kidney disease—as well as a tendency for patients to be anaemic with sub-normal haemoglobin levels.[14–15]

Although the literature regarding risk of cardiovascular events and having supratherapeutic levels of erythropoiesis stimulating agents driving serum haemoglobins over 120g/L is well-understood,[16] the data from our cohort demonstrates a trend in acute coronary syndromes and serum haemoglobins less than 100g/L. Mechanistically, one could hypothesise that relative anaemia and a subsequent supply-demand mismatch may serve to compromise myocardium which is already at-risk with diffuse coronary artery disease and ultimately predispose to cardiovascular events.

From a cardiological perspective, it is clear that diagnosis of acute coronary syndromes in patients with end-stage renal disease is challenging. Our data demonstrates that from history alone, one in four will present atypically and then biochemically, 40% will have a change in serum troponin (delta troponin) of less than 20%. Furthermore, although the majority of peak troponins exceeded the 99% cut-off (for "troponin positivity"), it is known that troponinaemia is a ubiquitous finding amongst the dialysis population.[17] Additionally, the utility of "serial troponins" in the RRT population is unknown given the conflicting data around the effect of dialysis on serum troponin concentrations.[18–19] As such, as we continue to use conventional means to diagnose acute coronary syndromes (as per the 4th Universal Definition of MI),[6] it is conceivable that this then leads to potential misdiagnosis or delays in diagnosis and ultimately delays in timely therapy.

Indeed, these delays—driven by the demonstrated diagnostic challenges posed by the dialysis population—are evident in our cohort whereby almost two-thirds (65%) of our cohort did not receive a coronary angiogram within 72 hours of diagnosis of ACS and the median time from diagnosis (in some instances the diagnosis was made many days after the day of admission) to invasive angiogram was four days. Over the same 10-year period, for all-comers at Waikato Hospital (n=8,865), the median time from admission to invasive angiogram was two days. As per the European Society of Cardiology (ESC) (and other international) guidelines, all acute coronary syndromes should receive a coronary angiogram within at least 72 hours of diagnosis (and for higher risk cases such as STEMIs and high-risk NSTEMIs, they should receive invasive angiography urgently).[7] The proportion of those not receiving angiography within 72 hours in our cohort—in contrast to the general cohort of ACS patients—demonstrates that guideline-appropriate management can be difficult to achieve in this unique population. Delays in diagnosis, communication between different in-hospital specialty services, concurrent medical issues such as anaemia, fluid overload and biochemical disturbances delaying angiography, the coordination of the timing of invasive angiography with dialysis sessions as well as a potential sentiment of "renalism" all play a part in shaping the way in which the dialysis population have their acute coronary events managed.[18]

Furthermore, despite almost half of the cohort having triple vessel disease, there was a relative paucity revascularised with CABG in comparison to multivessel PCI approaches. Given the prevalence of diabetes in the cohort, one would expect a higher rate of CABG in this population, particularly for those who are renal transplant candidates. However, because many decisions around revascularisation strategy are based around perioperative risk—the widespread use of tools such as the STS Score and EUROSCORE in cardiosurgical meetings held in the Midlands Region means that any renal dysfunction and in particular dialysis, is scored extremely unfavourably.[21–23] As such, there is a tendency to push dialysis patients towards a PCI approach, which in and of itself has inherent risks and shortcomings.

The challenges of a PCI strategy in the dialysis population is well documented

in the literature, whereby the nature of coronary plaque in those with ESRF is typically multi-vessel with heavy calcification—this in turn poses a higher risk for under-expansion of stents, subsequent stent malapposition and ultimately a higher risk of stent thrombosis.[24–27] Furthermore, contemporary second-generation drug-eluting stents available on the market do not include patients with end-stage renal disease in their randomised studies—the most robust evidence is extrapolated from observational studies and thereafter collated into meta-analyses.[28] Thus, although cardiosurgical meetings might emphasise perioperative risk as a detractor for surgery, the limitations and shortcomings of PCI in the dialysis population are likely under-appreciated.

In addition, there is data from large observational studies which suggest that for those on dialysis, although short-term mortality remains higher for CABG, their long-term outcomes (including mortality, rate of reinfarction and rate of reintervention) are superior to PCI for multivessel disease.[29] Contrarily, local data from Pilmore et al (NZMJ 2017) studied a cohort of 288 patients in New Zealand with ESRF (from the ANZDATA database) who received diagnostic angiograms of which one-third received revascularisation (with either PCI or CABG) and two-thirds were medically managed. In contrast to other larger overseas cohorts, there was no statistically significant difference in median survival between CABG (2.9 years), PCI (3.3 years) and medically-managed (2.9 years) groups. It must be noted however, that a significant number of this cohort studied received angiograms as part of renal transplantation workup, rather than for acute coronary syndromes—as such, these findings cannot be extrapolated to fit our cohort with acute coronary pathology.[30] In short—there is a paucity of quality, randomised data to ascertain the optimal management strategy for acute coronary syndromes in patients on renal replacement therapy.

Indeed, the above is reflected in the rates of reinfarction in our own cohort, whereby a third had further ACS events. This is far higher than in the non-dialysis population, where reinfarction rates tend to sit between 6–7 percent (at three years, as demonstrated in the HORIZONS-AMI trial).[31]

Our angiography complication rate of 9% is roughly in keeping with other observational data sets.[32] This slightly higher complication rate—in comparison to the non-dialysis population—is expected given the inherent vasculopathic and bleeding tendencies of those with ESRF.[33]

The 49% mortality at one year is also in keeping with other observational data sets,[34–35] and serves as a strong reminder that cardiovascular events are a key issue with all patients on renal replacement therapy and a significant prognostic factor. The poor prognosis associated with acute coronary syndromes in the dialysis population in our cohort reinforces the findings of the New Zealand-wide data linkage study, which assessed the impact of chronic kidney disease on mortality and cardiovascular outcomes after acute coronary syndromes (ANZACS-QI 44).[36] The New-Zealand wide data linkage study which evaluated over 20,000 patients (294 of whom had chronic kidney disease (CKD) stage 5), found that compared to those with normal renal function, patients with CKD stage 5 had up to 16 times greater rate of all-cause mortality (after adjustment for non-cardiac covariates) and additionally, that 70% of those with CKD stage 5 would go on to have a further adverse cardiovascular event within two years, compared to 12% of those with normal renal function.[36]

Our independent factor analysis has highlighted an association with atypical presentations and mortality at one year. This is likely a multifactorial finding, driven by the fact that both diabetic and elderly patients will tend to present atypically as well as the potential for atypical presentations to be diagnosed later and managed less aggressively. In addition, the statistically significant association between those who are conservatively managed (not revascularised) and those who are revascularised is potentially also borne out of the tendency for those who are more comorbid with a limited life expectancy to be offered conservative care rather than invasive management strategies.

The other significant association highlighted by our analyses was related to serum troponin values—both absolute peak value (during index admission) as well as the delta (a greater than 20% change in) troponin. Given that troponinaemia has been

repeatedly observed in multiple data sets to be a poor prognostic marker among CKD patients,[37] it is biologically plausible that a more significant troponinaemia would indicate more severe myocardial injury and therefore point towards a poorer outcome.

Ultimately, our retrospective analysis has highlighted a number of challenges in the care and management of acute coronary syndromes in the dialysis population. Additionally, the potential predictors for mortality at one year highlighted in our analysis, although suggestive of potential association, require validation with randomised trial data. Overcoming the challenges posed by systemic and ingrained ethnic disparities, as well as the inherent diagnostic and therapeutic challenges associated with the renal replacement therapy population remain the keys to seeing our dialysis population in the Midlands region receive equitable, best-practice care when they experience acute coronary events.

Competing interests:
Nil.

Author information:
Lance Ng, Department of Cardiology, Waikato District Health Board, Hamilton;
Tze Vun Liew, Department of Cardiology, Waikato District Health Board, Hamilton;
Andrew Henderson, Renal Department, Waikato District Health Board, Hamilton.

Corresponding author:
Lance Ng, C/- Cardiology Registrar Office, Level 2 Waiora Building, Waikato Hospital, Pembroke Street, Hamilton West, Hamilton 3204.
lance.ng@waikatodhb.health.nz

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REFERENCES:

1. Foley RN, Parfrey PS, Sarnak MJ. Epidemiology of cardiovascular disease in chronic renal disease. J Am Soc Nephrol. 1998; 9 Suppl:S16–23.
2. Collins AJ, Kasiske B, Herzog C, et al. Excerpts from the United States Renal Data System 2004 annual data report: atlas of end-stage renal disease in the United States. Am K Kidney Dis. 2005; 45 Suppl 1:A5–7,S1–280.
3. Cheung AK, Sarnak MJ, Yan G, et al. HEMO Study Group. Cardiac diseases in maintenance haemodialysis patients: results of the HEMO study. Kidney Int. 2004; 65:2380–9.
4. ANZDATA Registry. 42nd Report, Chapter 3: Mortality in End Stage Kidney Disease. Australia and New Zealand Transplant Registry, Adelaide, Australia. 2019. Available at: http://www.anzdata.org.au
5. Kerr A, Williams MJ, White H, et al. The all New Zealand acute coronary syndrome quality improvement programme: implementation, methodology and cohorts (ANZACS-QI 9). NZMJ. 2016; 129:23–36.
6. Thygesen K, Alpert JS, Jaffe AS, et al. ESC Scientific Document Group, Fourth universal definition of myocardial infarction (2018). European Heart Journal. 2019 Jan; 40(3):237–269.
7. Roffi M, Patrono C, Collet J, et al. ESC Scientific Document Group, 2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC). European Heart Journal. 2016 Jan; 37(3):267–315.
8. ANZDATA Registry. 42nd Report, Chapter

1: Incidence of Renal Replacement Therapy for End Stage Kidney Disease. Australia and New Zealand Transplant Registry, Adelaide, Australia. 2019. Available at: http://www.anzdata.org.au

9. ANZDATA Registry. 42nd Report, Chapter 2: Prevalence of Renal Replacement Therapy for End Stage Kidney Disease. Australia and New Zealand Transplant Registry, Adelaide, Australia. 2019. Available at: http://www.anzdata.org.au

10. Ministry of Health (2012). Policy for use of New Zealand Survival After Kidney Transplant Predictor system in patients assessed for deceased donor transplantation in New Zealand (Version 1.0). Wellington, New Zealand: Cross N, Dittmer I, Matheson P. Retrieved from http://www.health.govt.nz/about-ministry/leadership-ministry/expert-groups/national-renal-transplant-service/nrts-papers-and-reports

11. Te Pou o te Whakaaro Nui. (2018). DHB population profiles, 2018-2028: Statistics New Zealand projections 2017 update. Auckland: Te Pou o te Whakaaro Nui.

12. Huria T, Palmer S, Beckert L, et al. Inequity in dialysis related practices and outcomes in Aotearoa/New Zealand: a Kaupapa Māori analysis. Int J Equity Health. 2018; 17:27.

13. ANZDATA Registry. 41st Report, Chapter 4: Haemodialysis. Australia and New Zealand Dialysis and Transplant Registry, Adelaide, Australia. 2018. Available at: http://www.anzdata.org.au

14. McGovern AP, de Lusignan S, van vlymen J, et al. Serum phosphate as a risk factor for cardiovascular events in people with and without chronic kidney disease: a large community based cohort study. PLoS ONE 8(9): e74996. http://doi.org/10.1371/journal.pone.0074996

15. Palmer SC, Hayen A, Macaskill P, et al. Serum levels of phosphorus, parathyroid hormone, and calcium and risks of death and cardiovascular disease in individuals with chronic kidney disease: a systematic review and meta-analysis. JAMA. 2011; 305:1119–1127.

16. Palmer SC, Navaneethan SD, Craig JC, et al. Meta-analysis: erythropoiesis-stimulating agents in patients with chronic kidney disease. Ann Intern Med. 2010; 153(1):23. Epub 2010 Jun 24.

17. Dubin RF, Li Y, He J, et al. Predictors of high sensitivity cardiac troponin T in chronic kidney disease patients: a cross-sectional study in the chronic renal insufficiency cohort (CRIC). BMC Nephrol. 2013; 14:229. Epub 2013 Oct 22.5

18. Lippi G, Tessitore N, Montagnana M, et al. Influence of sampling time and ultrafiltration coefficient of the dialysis membrane on cardiac troponin I and T. Arch Pathol Lab Med. 2008 Jan; 132(1):72–6.

19. Wayand D, Baum H, Schatzle G, et al. Cardiac troponin T and I in end-stage renal failure. Clin Chem. 2000 Sep; 46(9):1345–50.

20. Chertow GM, Normand SLT, McNeil BJ. "Renalism": inappropriately low rates of coronary angiography in elderly individuals with renal insufficiency. J Am Soc Nephrol. 2004; 15:2462–2468.

21. Shahian DM, O'Brien SM, Filardo G, et al. The Society of Thoracic Surgeons 2008 cardiac surgery risk models: part 1--coronary artery bypass grafting surgery. Ann Thorac Surg. 2009; 88:S2.

22. Cooper WA, O'Brien SM, Thourani VH, et al. Impact of renal dysfunction on outcomes of coronary artery bypass surgery: results from the Society of Thoracic Surgeons National Adult Cardiac Database. Circulation. 2006; 113:1063.

23. Roques F, Nashef SA, Michel P, et al. Risk factors and outcome in European cardiac surgery: analysis of the EuroSCORE multinational database of 19030 patients. Eur J Cardiothorac Surg. 1999; 15:816.

24. Stenvinkel P, Pecoits-Filho R, Lindholm B. Coronary artery disease in end-stage renal disease: no longer a simple plumbing problem. J Am Soc Nephrol. 2003; 14:1927–39.

25. Nakamura S, Ishibashi-Ueda H, Nizuma S, et al. Coronary calcification in patients with chronic kidney disease and coronary artery disease. Clin J Am Soc Nephrol. 2009; 4:1892–900.

26. Mintz GS and Weissman NJ. Intravascular ultrasound in the drug-eluting stent era. J Am Coll Cardiol. 2006; 48:421–9.

27. Kawamura Y, Nagaoka M, Ito D, et al. A case of percutaneous coronary intervention procedure successfully bailed out from multiple complications in hemodialysis patient. Cardiovasc Interv Ther. 2013; 28:76–80.

28. Abdel-Latif A, Mukherjee D, Mesgarzadeh P, et al. Drug eluting stents in patients with end-stage renal disease: meta-analysis and

systematic review of the literature. Catheter Cardiovasc Interv. 2010; 76:942–8.

29. Aoki J, Ikari Y. Cardiovascular disease in patients with end-stage renal disease on haemodialysis. Ann Vasc Dis. 2017; 10(4):327–337.

30. Pilmore H, Webster M, Sidhu K, et al. Management of coronary artery disease in patients on dialysis. NZMJ. 2017; 130(1467):11–22.

31. Stone GW, Witzenbichler B, Guagliumi G, et al. Heparin plus a glycoprotein IIb/IIIa inhibitor versus bivalirudin monotherapy and paclitaxel-eluting stents versus bare-metal stents in acute myocardial infarction (HORIZONS-AMI): final 3-year results from a multicentre, randomised controlled trial. Lancet. 2011; 377:2193.

32. Baim, DS, Grossman, W. Complications of cardiac catheterization. In: Cardiac Catheterization, Angiography and Intervention, Baim, DS, Grossman, W (Eds), Williams & Wilkins, Baltimore 1996. p.17

33. Le Feuvre C, Dambrin G, Helft G, et al. Clinical outcome following coronary angioplasty in dialysis patients: a case-control study in the era of coronary stenting. Heart. 2001; 85(5):556–560. doi:10.1136/heart.85.5.556

34. Chertow GM, Normand SL, Silva LR, McNeil BJ. Survival after acute myocardial infarction in patients with end-stage renal disease: results from the cooperative cardiovascular project. Am J Kidney Dis. 2000; 35:1044–1051.

35. Herzog CA, Ma JZ, Collins AJ. Poor long-term survival after acute myocardial infarction among patients on long-term dialysis. N Engl J Med. 1998; 339(12):799–805. doi:10.1056/NEJM199809173391203

36. Pilmore H, Xiong F, Choi Y et al. Impact of chronic kidney disease on mortality and cardiovascular outcomes after acute coronary syndrome: a nationwide data linkage study (ANZACS-QI 44). Nephrology. 2020; 25(7):535–543.

37. Stacy SR, Suarez-Cuervo C, Berger Z et al. Role of troponin in patients with suspected acute coronary syndrome: a systematic review. Ann Intern Med. 2014; 161(7):502.