

ORIGINAL ARTICLE

Improved metabolic parameters of people with diabetes attending an Aboriginal health service in regional Victoria

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Key words

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Abstract

Background: Aboriginal and Torres Strait Islander people have higher rates of diabetes and its complications than non-Aboriginal people. Rumbalara Aboriginal Co-operative is the major primary healthcare provider for Aboriginal people in the Greater Shepparton region.

Aims: To evaluate the baseline metabolic parameters and presence of diabetes complications in people with type 2 diabetes attending Rumbalara Aboriginal Co-operative in 2017 and compare it with other Aboriginal and Torres Strait Islander studies and Australian specialist diabetes services.

Methods: Clinical and biochemical characteristics, including diabetes type, age, weight, body mass index (BMI), blood pressure, micro- and macrovascular complications, glycosylated haemoglobin (HbA1c), haemoglobin, renal function, lipid profile, urine albumin:creatinine ratio, diabetes medications, renin angiotensin system inhibition therapies, HMG-CoA reductase inhibitors and antiplatelet agents, were determined.

Results: One hundred and twenty-six individuals had diabetes, 121 had type 2 diabetes. One hundred and thirteen identified as Aboriginal and/or Torres Strait Islander. Median age was 57.5 (48–68) years, median HbA1c was 7.8% (6.8–9.6) and median BMI was 33.4 kg/m² (29–42.3). Compared with other Australian Aboriginal and Torres Strait Islander populations, this population was older and had more obesity, but with better glycaemia management. Compared with specialist diabetes services, this population was of similar age, with greater BMI but comparable HbA1c.

Conclusions: Aboriginal people living with type 2 diabetes attending this regional Aboriginal health service have comparable glycaemic management to specialist diabetes services in Australia, managed largely by primary care physicians with limited access to specialist care for the past 5 years.

Introduction

Diabetes mellitus is a chronic health condition that leads to complications including heart disease, stroke, and end-stage renal failure. Diabetes and its related complications

cause significant mortality and morbidity, costing the Australian community an estimated A\$14.6 billion annually.¹

Diabetes is common in the general Australian population, but its prevalence is 3–4 times greater in the Aboriginal and Torres Strait Islander population,² and even more so in rural and remote communities.^{3,4} Life expectancy of Aboriginal and Torres Strait Islander people is approximately 10 years less than their non-Aboriginal counterparts,² and diabetes is one of the major chronic health conditions contributing to this health gap.^{5,6} Aboriginal and Torres Strait Islander people not only have

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higher rates of diabetes,⁷ but a disproportionately higher incidence of diabetes-related complications and mortality compared with non-Aboriginal people.^{8,9} Furthermore, the onset of type 2 diabetes occurs at younger age in Aboriginal and Torres Strait Islander people,^{2,10,11} adversely affecting Aboriginal and Torres Strait Islander people in their most productive years of life.¹²

The Rumbalara Aboriginal Co-operative¹³ Medical Clinic (RAC) is an Aboriginal Community-Controlled Health Organisation (ACCHO) in Mooroopna, approximately 180 km north of Melbourne, Victoria. Mooroopna is part of the Greater Shepparton region and has a population of approximately 8000 people.¹⁴ RAC is the major primary healthcare provider for Aboriginal and Torres Strait Islander people in the Greater Shepparton region. In the 2016 census, there were 2696 Aboriginal and Torres Strait Islander people residing in the Greater Shepparton area, making up 4.56% of the total population.¹⁵ The Greater Shepparton region has the second-largest Aboriginal and Torres Strait Islander population in Victoria, outside of Melbourne.¹⁵

The Rumbalara Diabetes Service commenced in 2017 as a collaborative partnership between RAC, The University of Melbourne, and Austin Health Department of Endocrinology. There was extensive consultation with the Rumbalara executive and community leaders to identify community needs, and both the Rumbalara executive and community members wanted the addition of specialist diabetes care to their health service. This service provides a monthly 'in-house' multidisciplinary diabetes service with a team comprising of four visiting endocrinologists, an endocrinology registrar, diabetes nurse educator, diabetes chronic disease nurse and a dietitian.

The Rumbalara Diabetes Service commenced in 2017 and is the second specialist diabetes service to be run within RAC. The first service was an integrated specialist diabetes service, the Rumbalara Aboriginal Health Service Diabetes Programme,¹⁶ which ran from 1999 until August 2001. This was a pilot programme that involved a weekly service with a sole diabetes specialist integrated into an Aboriginal primary care setting. Forty-seven people were seen over a 2-year study period, with significant improvement in their glycaemic management.¹⁶ There was no 'in-house' specialist diabetes service in RAC between 2001 and 2017, and diabetes care was largely provided through primary care.

This present study aimed to evaluate the baseline metabolic parameters of people living with type 2 diabetes and diabetes-related complications, attending the Rumbalara Aboriginal Co-operative in 2017. These data were compared with data from other Aboriginal and Torres Strait Islander diabetes cohorts and Australian specialist diabetes services from the Australian National Diabetes Audit Australian Quality Clinical Audit (ANDA-AQCA). Analysis

focused on data prior to the establishment of the Rumbalara Diabetes Service, so the impact of the Rumbalara Diabetes Service could be measured at a later date. Data from this study represent data almost 20 years from the original RAC integrated specialist diabetes service.

Methods

Retrospective audit of patients with a diagnosis of diabetes mellitus recorded on the RAC Medical Centre electronic medical records (EMR). The date range for data extraction was from 1 January 2017 to 31 December 2017. The following clinical characteristics were obtained: diabetes type, age, weight, body mass index (BMI), blood pressure (BP) and presence of micro- and macrovascular complications. The use of diabetes and other cardiovascular medications was recorded. Biochemical characteristics collected included haemoglobin, renal function and electrolytes, urine albumin:creatinine ratio, glycosylated haemoglobin (HbA1c) and lipid profile. Where an individual had one or more recordings of the above parameters, the first value recorded in 2017 was used. Individuals were deemed to no longer have diabetes if their most recent HbA1c was <6.5% and they were not taking glucose-lowering medications. This study focussed on individuals with type 2 diabetes mellitus (T2DM), as there were small numbers of people with type 1 diabetes mellitus (T1DM).

Comparison data were sourced from the ANDA-AQCA 2017¹⁷ for Australian specialist diabetes care. Comparison data from Aboriginal and Torres Strait Islander studies were sourced from the original publications and supplementary material of the Rumbalara Diabetes Clinic (1999),¹⁶ Fremantle Diabetes Study (FDS),¹⁸ Darwin Region Urban Indigenous Diabetes Study (DRUID),¹⁹ and two studies^{20,21} of remote Aboriginal and Torres Strait Islander populations.

This study was approved by the Human Research Ethics Committee, Goulburn Valley Health, Shepparton, Victoria and supported by the Rumbalara Aboriginal Co-operative. Statistical analyses were performed using Stata for Mac (version 15.1; StataCorp, TX, USA). Data were described as median and interquartile range, mean $\pm$ standard deviation and *n* (%). Categorical data were compared using the Pearson Chi-squared test, while continuous data were compared using the Wilcoxon rank-sum test. A significance level of <0.05 was used.

Results

Baseline characteristics

There were 126 individuals with diabetes mellitus attending RAC in 2017. Approximately 89.7% identified as

Aboriginal or Torres Strait Islander. The majority of people had T2DM. One hundred and twenty-one people had T2DM, four people had T1DM and one person had gestational diabetes mellitus. Females comprised 57.9% of the population. BP was at target in the majority of individuals, with a median BP of 127/79 mmHg (117/70–136/87 mmHg) for the whole cohort and 128/79 mmHg (119/70–137/87 mmHg) for people with T2DM. The median BMI was 33.4 kg/m² (29–42.3 kg/m²), but when BMI was further categorised into normal, overweight and obese categories, only individuals with T2DM were overweight or had obesity. In people with T2DM, 14.9% were overweight and 60.3% had obesity (Table 1).

Table 1 Baseline characteristics of individuals with diabetes

Characteristic	(n = 126)
Age (years)	57.5 (48–68)
Aboriginal or Torres Strait Islander, % (n)	89.7 (113/126)
Sex, % female (n)	57.9 (73/126)
Weight (kg)	92.40 (74.95–113.3)
BMI (kg/m ²)	33.4 (29–42.3)
Normal BMI, % (n)	8.7 (11/126)
Overweight, % (n)	14.3 (18/126)
Obese, % (n)	57.9 (73/126)
SBP (mmHg)	127 (117–136)
DBP (mmHg)	79 (70–87)
1 or more microvascular complications, % (n)	35.7 (45/126)
1 microvascular complication, % (n)	27.0 (34/126)
2 microvascular complications, % (n)	7.9 (10/126)
3 microvascular complications, % (n)	0.8 (1/126)
1 or more macrovascular complications, % (n)	43.7 (55/126)
1 macrovascular complication, % (n)	32.5 (41/126)
2 macrovascular complications, % (n)	10.3 (13/126)
3 macrovascular complications, % (n)	0.8 (1/126)
HbA1c (%) (mmol/mol)	7.8 (6.8–9.6)
	62 (51–81)
Urinary ACR (mg/mmol)	2.55 (0.85–7.60)
Microalbuminuria, % (n)	14.3 (18/126)
Macroalbuminuria, % (n)	4.8 (6/126)
GFR† CKD EPI (mL/min)	88.63 (67.36–102.09)
Total cholesterol (mmol/L)	3.90 (3.50–4.70)
HDL-C (mmol/L)	1.03 (0.86–1.22)
LDL-C (mmol/L)	2.00 (1.40–2.50)
Triglycerides (mmol/L)	2.15 (1.65–3.05)

†CKD-EPI GFR.

Data are median (interquartile range (IQR)), or % (n) where specified. Normal BMI < 25 kg/m²; overweight BMI = 25–30 kg/m²; obese BMI > 30 kg/m²; microalbuminuria = alb:cr 3.4–34 mg/mmol; macroalbuminuria = alb:cr > 34 mg/mmol; microvascular complications = retinopathy, nephropathy and peripheral neuropathy; macrovascular complications = stroke, coronary artery disease and peripheral vascular disease.

ACR, albumin:creatinine ratio; BMI, body mass index; DBP, diastolic blood pressure; GFR, glomerular filtration rate; HbA1c, glycosylated haemoglobin; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; SBP, systolic blood pressure.

Diabetes management and complications

The median HbA1c was 7.8% in those with T2DM. Of people with T2DM, 14.3% had microalbuminuria and 4.8% had macroalbuminuria. 35.7% of individuals with T2DM had one or more microvascular complications, and 43.7% had one or more macrovascular complications. Microvascular complications included retinopathy (*N* = 13), nephropathy (*N* = 26) and peripheral neuropathy (*N* = 15). Macrovascular complications included stroke (*N* = 14), coronary artery disease (*N* = 46) and peripheral vascular disease (*N* = 10). Only individuals with T2DM had macrovascular complications.

Medication use

People with T2DM were on varying combinations of oral hypoglycaemic agents²² and/or injectable glucose-lowering medications (insulin and/or glucagon-like peptide 1, GLP-1 agonists). Metformin was the most commonly prescribed oral hypoglycaemic agent, used by nearly 80% of people. Insulin was used by just over 26% of individuals, and their mean daily insulin requirement was 62.7 ± 42.8 units. Four of the 32 people with T2DM using insulin (12.5% of T2DM on insulin and 3.3% of all people with T2DM) were on insulin monotherapy for diabetes. Their mean insulin requirement was 45.5 units/day (Tables A1–A3).

There was 60.3% of people with T2DM who were taking renin–angiotensin–aldosterone system (RAAS)-inhibitor therapies; 68.3% were on a 3-hydroxy-3-methyl-glutamyl-coenzyme A (HMG-CoA) reductase inhibitor, 73.8% (93/126) were on at least one lipid-lowering medication (HMG-CoA reductase inhibitor, ezetimibe or fenofibrate) and 15.9% (20/126) were on both a HMG-CoA reductive inhibitor plus ezetimibe and/or fenofibrate. Only individuals with T2DM were taking RAAS inhibitor and/or lipid-lowering therapies.

Medication use and complications

When medication use was assessed according to the presence of micro- and macrovascular complications, there were differences in some medications used by those with macrovascular complications, but no difference for microvascular complications. The use of HMG-CoA reductase inhibitors (*P* < 0.005) and antiplatelet agents (*P* < 0.005) was greater in people with macrovascular complications compared to those without macrovascular complications. Fifty-two of the 55 individuals with macrovascular complications used HMG-CoA Reductase inhibitors compared with 36 of

Table 2 Medication use by presence of macrovascular complications

	No macrovascular complications (n = 67)	1 or more macrovascular complications (n = 55)	P-value
SGLT2i, % (n)	7.5 (5/67)	10.9 (6/55)	0.508
ACEi/ARB, % (n)	53.7 (36/67)	70.9 (39/55)	0.052
HMG-CoA reductase inhibitor, % (n)	50.7 (34/67)	94.5 (52/55)	0.000
Any antiplatelet, % (n)	41.8 (28/67)	78.2 (43/55)	0.000
Fenofibrate or ezetimibe, % (n)	20.9 (14/67)	23.6 (13/55)	0.717

Data are presented as % (n). P-values have been calculated using the Pearson Chi-squared test.

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin 2 receptor blocker; HMG-CoA, 3-hydroxy-3-methyl-glutamyl-coenzyme A; SGLT2i, sodium-glucose co-transporter 2 inhibitor.

the 67 individuals without macrovascular complications. Forty-three of the 55 individuals with macrovascular complications used antiplatelet agents compared with 28 of the 67 people with macrovascular complications. The use of RAAS-inhibitor therapy was also greater in those with macrovascular complications; however, the *P*-value did not reach statistical significance (*P* = 0.052), most likely due to the small sample size. For macrovascular complications (Table 2), there was no difference in the rates of use of sodium-glucose co-transporter 2 inhibitor (SGLT2i) or fenofibrate/ezetimibe use. For microvascular complications, there was no difference in rates of SGLT2i, RAAS or fenofibrate/ezetimibe use.

Albuminuria

There was 19% of people with diabetes who had albuminuria; 14.3% had microalbuminuria (urinary albumin-to-creatinine ratio (ACR): 3.4–34 mg/mmol) and 4.8% had macroalbuminuria (urinary ACR > 34 mg/mmol). Those with albuminuria were older and had higher BP than those with normoalbuminuria. Mean BP was 133/79 mmHg and 126/77 mmHg respectively. In people with albuminuria, the mean urinary ACR was 23.37 mg/mmol, and 70.8% of these individuals were treated with RAAS-inhibitor therapy. Glomerular filtration rate (GFR), as calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation, was lower in those with albuminuria, with mean GFR being 77.87 mL/min compared with 84.65 mL/min in those with normoalbuminuria. HbA1c in those with normoalbuminuria was significantly lower than in those with albuminuria (Table 3).

There was no significant difference in the rates of RAAS-inhibitor therapy use between people with normoalbuminuria and those with albuminuria. Furthermore, there was no difference when those with albuminuria were further categorised into micro- and macroalbuminuria. For micro- and macroalbuminuria,

there was no difference in the rates of SGLT2i and HMG-CoA reductase inhibitor use.

Comparison with other studies

The 2017 ANDA-AQCA¹⁷ provided an overview of the clinical status of people with diabetes who attended surveyed services for diabetes care in 2017, and represented the general Australian population with diabetes with a small proportion of Aboriginal and Torres Strait Islander people (4.2%). Compared with the 2017 ANDA-AQCA group, the population at RAC in 2017 had comparable diabetes management, but had a greater BMI (mean BMI 35.6 vs 31.4 kg/m²). Metformin use was similar to the ANDA-AQCA population. There was twice as much insulin use in the ANDA-AQCA population compared to the RAC population. Despite this, the RAC population were able to achieve similar mean HbA1c levels.

Compared with other studies of Aboriginal and Torres Strait Islander populations with diabetes (Arnhem Land,²¹ FDS¹⁸ and DRUID¹⁹), the RAC population were older and had a higher BMI. The average person with T2DM lived with obesity (World Health Organization (WHO) class 2), with mean BMI of 36.0 kg/m² (overall mean 35.6 kg/m²). In contrast, the average person from other Aboriginal and Torres Strait Islander populations was overweight/obese (WHO class 1), with mean BMI between 26.7 and 32.4 kg/m². The RAC population had a greater proportion of people treated with metformin, but lower rates of sulphonylurea and thiazolidinedione use, compared to other Aboriginal populations. Compared to the RAC diabetes programme from 1999 to 2001,¹⁶ the RAC population in 2017 were slightly older, heavier (mean weight 95.4 ± 28.4 vs 82.5 ± 20.3 kg), and had better glycaemic management (mean HbA1c 8.3 ± 2.4% vs 10.4 ± 2.2%). With respect to data from the DRUID study, 135 individuals were assessed for diabetes complications, but were categorised into known diabetes (n = 99) and new diabetes (n = 36), pooled data had not been presented. The data of the 99 individuals from the DRUID study reflect those with known diabetes.

Table 3 Normoalbuminuria versus albuminuria

	Normoalbuminuria (n = 102)	Albuminuria (n = 24)	P-value
Age (years)	55.4 ± 14.6 55.5 (45.0–66.0)	62.3 ± 11.4 63.5 (52.5–70.0)	0.0521
Sex, % female (n)	58.8 (60/102)	54.2 (13/24)	
Weight (kg)	96.36 ± 28.20 93.30 (77.05–113.30)	92.19 ± 29.44 82.20 (65.2–112)	0.3995
BMI (kg/m ²)	36.04 ± 9.40 34.0 (29.9–42.4)	33.86 ± 8.64 32.80 (26.4–39.4)	0.2960
SBP (mmHg)	126.2 ± 15.2 125.0 (114.0–136.0)	133.0 ± 13.75 130.5 (125.5–138)	0.0773
DBP (mmHg)	77.3 ± 12.3 78.0 (70.0–86.0)	79.4 ± 10.3 81 (72.5–87)	0.3856
ACEi/ARB, % (n)	57.8 (59/102)	70.8 (17/24)	0.293
HbA1c (%)	8.1 ± 1.9 7.5 (6.7–8.8)	9.9 ± 2.4 9.3 (8.5–11.6)	0.0006
Urinary ACR (mg/mmol)	1.39 ± 0.90 1.00 (0.70–2.05)	23.37 ± 21.48 17.95 (6.80–32.60)	0.0000
GFR† CKD EPI (mL/min)	84.65 ± 26.14 89.49 (73.15–103.68)	77.87 ± 25.44 51.01 (87.26–97.10)	0.2323
Total cholesterol (mmol/L)	4.26 ± 1.09 3.90 (3.60–4.70)	3.91 ± 0.88 3.70 (3.15–4.55)	0.1967
HDL-C (mmol/L)	1.06 ± 0.34 1.02 (0.84–1.18)	1.25 ± 0.64 1.11 (0.94–1.43)	0.1374
LDL-C (mmol/L)	2.11 ± 0.93 2.00 (1.55–2.60)	1.84 ± 0.73 1.80 (1.20–2.30)	0.2987
Triglycerides (mmol/L)	2.81 ± 2.67 2.10 (1.65–2.90)	2.85 ± 1.83 2.2 (1.65–3.55)	0.7410

†CKD-EPI GFR.

Data are presented as % (n), mean ± standard deviation, or median and interquartile range. P-values have been calculated using the Wilcoxon rank-sum test; normoalbuminuria = urinary ACR < 3.4 mg/mmol; albuminuria = urinary ACR ≥ 3.4 mg/mmol.

ACR, albumin:creatinine ratio; BMI, body mass index; DBP, diastolic blood pressure; GFR, glomerular filtration rate; HbA1c, glycosylated haemoglobin; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; SBP, systolic blood pressure.

Discussion

People with diabetes at Rumbalara Aboriginal Co-operative represent a cohort of mostly Aboriginal and Torres Strait Islander people with T2DM in regional Victoria. In 2017, this population had diabetes management and cardiometabolic parameters similar to Australian diabetes services,¹⁷ achieving more of the targets compared with other Aboriginal and Torres Strait Islander populations (Table 4).^{16,18,20,21}

In particular, the mean HbA1c was 8.3% (median HbA1c was 7.8%), which is particularly encouraging because it is comparable to that of Australian diabetes services, where there is a much smaller proportion of Aboriginal and Torres Strait Islander people. Aboriginal and Torres Strait Islander people with diabetes attending Rumbalara Aboriginal Co-operative had lower HbA1c levels than Aboriginal and Torres Strait Islander populations in other Australian studies, in contrast to findings in other studies that demonstrated 1% higher HbA1c levels in Aboriginal and Torres Strait Islander versus non-Aboriginal populations.²³ Like

other Aboriginal and non-Aboriginal populations, BP was well managed. Lipid profile was comparable with Australian diabetes services. This population had lower total cholesterol, triglyceride and low-density lipoprotein cholesterol (LDL-C) levels, but higher high-density lipoprotein cholesterol (HDL-C) levels than the Australian diabetes population which is very encouraging. Rates of renin-angiotensin inhibition therapies and lipid-lower medication use were similar to that of Australian diabetes services.¹⁷ The difference in statin use between people with and without macrovascular complications at RAC was both statistically and clinically significant. This likely reflects adherence to the current guidelines that recommend the use of a statin for secondary prevention. The difference in the proportion of people who were on RAAS in those with and without macrovascular complications did not reach statistical significance, likely due to the relatively small sample size of the cohort. The use of lipid-lowering therapies could be increased in order to achieve the LDL target <1.8 mmol/L for primary prevention in high-risk populations.²⁴

Table 4 Characteristics of people at Rumbalara Aboriginal Co-operative versus other Australian Aboriginal populations

	Rumbalara Aboriginal Co-operative Diabetes Audit 2017				Australian National Diabetes Audit Australian Quality Clinical Audit (ANDA- AQCA) 2017 ¹⁷				
	All (n = 126)	T2DM (n = 121)	Rumbalara Diabetes Clinic 1999 ¹⁶ (n = 30)	Remote Australian (Arnhemland) ²¹ (n = 37)	Urban Australian (DRUID) ¹⁹ (n = 99)	Fremantle Diabetes Study Aboriginal ¹⁸ (n = 18)	Remote Australian ²⁰ (Central Australian) (n = 124)	ANDA 2017 All Diabetes (n = 5719)	ANDA 2017 T2DM (66.9%)
Data year	2017	2017	2001	2001	2003–2005	1993–1996	2014	2017	2017
Age (years)	56.7 ± 14.3	57.7 ± 13.7	54 ± 13 (n = 40) ^{†‡}	51 ± 8	53 ± 10	51.2 ± 11.8	48.6 (40.9–57.2) [†]	55.4 ± 17.8	63.1 ± 12.9
Mean (±SD)	57.5 (48.0–68.0)	58.0 (49.0–68.0)							
Median (IQR)	89.7 (113/126)	89.3 (108/121)						4.2	
Aboriginal or Torres Strait Islander, % (n)									
Sex, % female (n)	57.9 (73/126)	57.9 (70/121)	65(26/40) ^{†‡}	54 (20/37)	76 (75/99)	72.2	76.6 (95/124)	48.8	
Weight (kg)	95.4 ± 28.4	96.4 ± 28.2	82.5 ± 20.3	76.3 ± 14.0	86.4 ± 17.2				
BMI (kg/m ²)	35.6 ± 9.2	36.0 ± 9.0	33.6 ± 8.7 (n = 40) ^{†‡}	26.7 ± 3.8	32.4 ± 6.1	30.8 ± 5.8		31.4 ± 7.6	33.1 ± 7.5
SBP, mean (±SD) (mmHg)	127.7 ± 15.1	128.6 ± 14.5	138 ± 20	131 ± 26	128 ± 18	138 ± 24	120 (110–137) [†]	130 ± 17	
Median (IQR)									
DBP, mean (±SD)	77.7 ± 11.9	78.3 ± 11.6	78 ± 11	76 ± 11	78 ± 10	79 ± 11	80 (70–85) [†]	75 ± 11	
Median (IQR)									
HbA1c, † mean (±SD) (%)	8.3 ± 2.4	8.2 ± 2.2	10.4 ± 2.2	8.7 (7.8–9.6)	8.1 (7.7–8.4)	9.8 (7.8–11.0)	10.2 (7.7–11.9)	8.1 ± 1.8	8.1 ± 1.7
Median (IQR)	7.8 (6.8–9.6)	7.8 (6.8–9.5)							
Urinary ACR (mg/mmol) [†]	2.55 (0.85–7.60)	2.60 (0.80–7.60)		10.3 (5.7–18.5)	2.4 (1.6–3.8)	10.1 (1.1–93.6)	11.7 (2.3–34.8)		
GFR [‡] (mL/min)	83.1 ± 26.0	82.3 ± 26.2			83.6 [§] (77.6–90.1) [†]				
CKD-EPI									
MDRD 175	81.8 ± 30.9	82.4 ± 31.1							
Total cholesterol, † mean (±SD)	4.17 ± 1.05	4.15 ± 1.04	6.0 ± 1.8	4.8 (4.5–5.0)	5.0 (4.8–5.3)	5.7 ± 0.9	4.3 (3.8–5.0)	4.4 ± 1.3	4.3 ± 1.2
Median (IQR)	3.90 (3.50–4.70)	3.90 (3.50–4.70)							
HDL-C, † Mean (±SD) (mmol/L)	1.11 ± 0.45	1.08 ± 0.41		0.82 (0.76–0.89)	0.96 (0.91–1.02)	1.01 ± 0.26	0.9 (0.80–1.00)	1.3 ± 0.5	1.2 ± 0.4
Median (IQR)	1.03 (0.86–1.22)	1.02 (0.86–1.22)							
LDL-C, † mean (±SD) (mmol/L)	2.04 ± 0.88	2.03 ± 0.88				3.6 ± 0.9	2.40 (1.80–3.20)	2.3 ± 1.0	2.2 ± 1.0
Median (IQR)	2.00 (1.40–2.50)	2.00 (1.40–2.50)							
Triglycerides, † mean (±SD) (mmol/L)	2.82 ± 2.46	2.86 ± 2.48	4.0 ± 2.2	2.4 (2.0–2.8)	2.4 (2.1–2.8)	2.4 (1.5–3.9)	2.50 (1.75–3.70)	2.1 ± 2.2	2.2 ± 2.2

Table 4 Continued

	Rumbalara Aboriginal Co-operative Diabetes Audit 2017		Australian National Diabetes Audit Australian Quality Clinical Audit (ANDA- AQCA) 2017 ¹⁷			
	All (n = 126)	T2DM (n = 121)	Rumbalara Diabetes Clinic 1999 ¹⁶ (n = 30)	Remote Australian (Arnhemland) ²¹ (n = 37)	Urban Australian (DRUID) ¹⁹ (n = 99)	Fremantle Diabetes Study Aboriginal ¹⁸ (n = 18)
Median (IQR)	2.15 (1.65–3.05)	2.15 (1.70–3.10)				
Metformin, % (n)	76.2 (96/126)	79.3 (96/121)	56.7 (17/30)	65 (24/37)	57 (52/99)	71.8
Sulphonylurea, % (n)	15.1 (19/126)	15.7 (19/121)	33.3 (10/3)	30 (11/37)	35 (32/99)	24.3
Thiazolidinedione, % (n)	0.8 (1/126)	0.8 (1/121)		0 (0/37)	7 (6/99)	0.6
DPP4 inhibitor, % (n)	20.6 (26/126)	21.5 (26/121)				17.5
SGLT2 inhibitor, % (n)	8.7 (11/126)	9.1 (11/121)				18.5
GLP1 agonist, % (n)	7.1 (9/126)	7.4 (9/121)				10.3
Insulin, % (n)	27.8 (35/126)	26.4 (32/121)	43.3 (13/30)	0 (0/37)	19 (18/99)	58.2
ACEi/ARB, % (n)	60.3 (76/126)	62.8 (76/121)	73.3 (22/30)	62 (23/37)	63 (59/99)	
HMG-CoA reductase inhibitor, % (n)	68.3 (86/126)	71.1 (86/121)	33.3 (10/30)	24 (9/37)	37 (35/99)	

[†]Data are median (interquartile range, IQR).

^{*}CKD-EPI GFR.

[§]MMDR GFR.

^{||}ACEi only reported in the Fremantle Diabetes Study.

[‡]Lipid-lowering therapy reported in the Fremantle Diabetes Study.

^{††}Data reported in text of the study where the total study population was 40 people; other data taken from a table containing data of patients not seen for hypoglycaemia (n = 30).

Data are mean ± standard deviation or % (n) unless stated otherwise.

ACEi, angiotensin-converting enzyme inhibitor; ACR, albumin:creatinine ratio; ARB, angiotensin 2 receptor blocker; BMI, body mass index; DBP, diastolic blood pressure; DPP4, Dipeptidyl peptidase 4; GFR, glomerular filtration rate; GLP1, glucagon-like peptide-1; HbA1c, glycosylated haemoglobin; HDL-C, high-density lipoprotein-cholesterol; HMG-CoA, 3-hydroxy-3-methyl-glutaryl-coenzyme A; LDL-C, low-density lipoprotein-cholesterol; SBP, systolic blood pressure; SGLT2, sodium-glucose co-transporter 2; T1DM, type 1 diabetes mellitus; T2DM, type 2 diabetes mellitus.

The RAC population had higher rates of insulin use, which might explain why they had better glucose management but higher BMI, as insulin therapy leads to weight gain. The greater BMI in this population represents an opportunity to use newer diabetes agents that are weight neutral or lead to weight loss (DPP4i, SGLT2i and GLP-1) instead of insulin. The lower rates of sulphonylurea and thiazolidinedione use in this population were likely due to the availability of newer glucose-lowering medications including SGLT2i, dipeptidyl peptidase 4 inhibitors (DPP4i) and glucagon-like peptide-1 (GLP-1) agonists. These agents were not available when most of the other Aboriginal and Torres Strait Islander studies were conducted. At the time of this study, the evidence for cardio-renal protective medications was not fully realised as some of the larger randomised clinical trials had not been completed. After this audit was completed in 2018, the American Diabetes Association guidelines were changed to reflect recommendations to use SGLT2 inhibitors and GLP-1 analogues with cardio-renal protection in those who are at high risk or with a prior history.²⁵ Over time, it is hoped that using these pharmacotherapies will lead to reductions in diabetes complications, such as end-stage renal failure and heart failure, and diabetes-related hospital admissions in Aboriginal and Torres Strait Islander populations.

While diabetes mellitus causes significant morbidity and mortality in Aboriginal and Torres Strait Islander people, there are few studies that detail their cardiometabolic outcomes. Of the available published studies, these have either studied Aboriginal people in remote or urban areas. This study provides information of the cardiometabolic outcomes of Aboriginal people with diabetes mellitus who live in regional Australia, of which there is limited data. This study showed that Aboriginal people living in the Shepparton region had comparable diabetes management to non-Aboriginal populations. Compared to the earlier RAC diabetes program study,¹⁶ this current study represents all people with diabetes at RAC, whilst the earlier study does not. The sample size in the earlier study¹⁶ was smaller than the present study. These individuals had been referred for specialist diabetes management were likely to be those with higher HbA1c levels and rates of complications, most in need of specialist care.

We recognise the limitations of the present study. First, the accuracy of clinical data was dependent on entry from healthcare professionals. However, data collection was reliable because the Aboriginal and Torres Strait Islander population were more likely to attend RAC as it is an Aboriginal Health Service (AHS) compared with non-Aboriginal health services. Furthermore, people were more likely to attend this AHS as they received timely reminders for health checks. In

particular, there is an Aboriginal and Torres Strait Islander-specific Medicare Benefits Schedule (MBS) item (MBS Item 715), which allows a health assessment for Aboriginal and Torres Strait Islander people as frequently as every 9 months.²⁶ Consequently, there is likely to be ascertainment bias as individuals attending RAC were more motivated than individuals who declined attending RAC. Therefore, the population in this audit may not be representative of and generalisable to other Aboriginal and Torres Strait Islander populations. Second, this study is likely to have underestimated the number of people with diabetes, as there may be people with diabetes who were not listed in their medical history, who would not have been included in this audit. These findings are similar to other studies where the surveyed population is a snapshot of the entire population. Third, while Rumbalara Aboriginal Co-operative is the major primary healthcare provider for Aboriginal and Torres Strait Islander people in the greater Shepparton region, not all Aboriginal and Torres Strait Islander people attend RAC. Some individuals see multiple healthcare providers across the region, therefore RAC might not have all of their records. Of available data, not all patients have diabetes duration recorded on the EMR. As such, links between diabetes complications and diabetes duration were unable to be established. All of these factors contribute to missing data.

The COVID-19 pandemic has significantly delayed the publication of this data. This publication was intended to show the impact of a specialist diabetes service on the cardiometabolic outcomes of people living with diabetes attending Rumbalara Aboriginal Co-operative, with this data being the baseline characteristics prior to the commencement of the service and subsequent data 5 years after the start of the service. There were very limited specialist diabetes services undertaken at RAC throughout 2020 and 2021 as a result of the public health measures in place. Data collection that would have been undertaken 5 years after the commencement of the clinic could have been collected from 2022. However, due to the significant reduction in services during the pandemic, data collection at this time may not demonstrate the full impact of the specialist diabetes service.

These data are an example of primary care in Aboriginal Health Services effectively providing diabetes care to Aboriginal populations in regional Victoria. These results are encouraging as they have been achieved despite limited access to specialist diabetes services for several years. These positive results may have been achieved at RAC because RAC is a good ACCHO, with good relationships between staff members, community and clients. In addition, this study was performed sometime after most of

the comparative studies, when newer diabetes medications including SGLT2 inhibitors and GLP-1 analogues had become available for use. Therefore some of the improvements in diabetes management may reflect the efficacy of these newer agents, but it is difficult to differentiate the degree to which each factor contributes. It is hoped that other ACCHO will be able to achieve similar results. It is anticipated that increased access to specialist diabetes care could potentially add to further improvements in these outcomes which will hopefully reduce the disease burden that diabetes mellitus has on this community. It is hoped this will help to close the gap in health outcomes between the Aboriginal and Torres Strait Islander population and the Australian population in the Greater Shepparton region.

Conclusion

The data presented in this study represents the baseline characteristics of a largely Aboriginal and Torres Strait Islander population with T2DM residing in regional Victoria. Aboriginal and Torres Strait Islander people with

diabetes attending Rumbalara Aboriginal Co-operative had lower HbA1c levels than Aboriginal and Torres Strait Islander populations in other Australian studies, and levels of glycaemia were comparable with non-Aboriginal populations. People with diabetes at Rumbalara Aboriginal Co-operative have diabetes management that is comparable with non-Aboriginal populations in metropolitan areas, despite being without an 'in-house' diabetes specialist service for several years. This is a credit to the Aboriginal and Torres Strait Islander population in the Greater Shepparton region, their primary health care providers and Aboriginal Health Workers at Rumbalara Aboriginal Co-operative.

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Appendix A

Table A1 Medication use

	All (<i>n</i> = 126)
Metformin, % (<i>n</i>)	76.2 (96/126)
Sulphonylurea, % (<i>n</i>)	15.1 (19/126)
Thiazolidinedione, % (<i>n</i>)	0.8 (1/126)
DPP4 inhibitor, % (<i>n</i>)	20.6 (26/126)
SGLT2 inhibitor, % (<i>n</i>)	8.7 (11/126)
GLP1 agonist, % (<i>n</i>)	7.1 (9/126)
Insulin, % (<i>n</i>)	27.8 (35/126)
Insulin (mean ± standard deviation) (units)	64.1 ± 43.3
ACEi/ARB, % (<i>n</i>)	60.3 (76/126)
HMG-CoA reductase inhibitor, % (<i>n</i>)	68.3 (86/126)
Other lipid-lowering therapy,† % (<i>n</i>)	21.4 (27/126)

†Fenofibrate or ezetimibe.

Data are presented as % (*n*), or mean ± standard deviation.

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin 2 receptor blocker; DPP4, dipeptidyl peptidase 4; GLP1, glucagon-like peptide-1; HMG-CoA, 3-hydroxy-3-methyl-glutamyl-coenzyme A; SGLT2, sodium-glucose co-transporter 2.

Table A2 Medication use by presence of microvascular complications

	No microvascular complications (<i>n</i> = 77)	1 or more microvascular complication (<i>n</i> = 44)	<i>P</i> -value
SGLT2i, % (<i>n</i>)	10.4 (8/77)	4.5 (2/44)	0.261
ACEi/ARB, % (<i>n</i>)	57.1 (44/77)	68.2 (30/44)	0.231
HMG-CoA reductase inhibitor, % (<i>n</i>)	64.9 (50/77)	79.5 (35/44)	0.091
Any antiplatelet, % (<i>n</i>)	53.2 (41/77)	65.9 (29/44)	0.175
Fenofibrate or ezetimibe, % (<i>n</i>)	19.5 (15/77)	27.3 (12/44)	0.322

Data are presented as % (*n*). *P*-values have been calculated using the Pearson Chi-squared test.

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin 2 receptor blocker; HMG-CoA, 3-hydroxy-3-methyl-glutamyl-coenzyme A; SGLT2i, sodium-glucose co-transporter 2 inhibitor.

Table A3 Medication use patients with albuminuria

	Microalbuminuria (<i>n</i> = 18)	Macroalbuminuria (<i>n</i> = 6)	<i>P</i> -value
SGLT2i, % (<i>n</i>)	16.7 (3/18)	16.7 (1/6)	1.0
ACEi/ARB, % (<i>n</i>)	66.7 (12/18)	83.3 (5/6)	0.437
HMG-CoA reductase inhibitor, % (<i>n</i>)	88.9 (16/18)	100 (6/6)	0.394
Any antiplatelet, % (<i>n</i>)	83.3 (15/18)	66.7 (4/6)	0.384
Fenofibrate or ezetimibe, % (<i>n</i>)	27.8 (5/18)	50 (3/6)	0.317

Data are presented as % (*n*). *P* values have been calculated using the Pearson Chi-squared test.

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin 2 receptor blocker; HMG-CoA, 3-hydroxy-3-methyl-glutamyl-coenzyme A; SGLT2i, sodium-glucose co-transporter 2 inhibitor.