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ORIGINAL ARTICLE

Incidence and predictors of transurethral resection of prostate in men with and without type 2 diabetes: the Fremantle Diabetes Study Phase I

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Key words
type 2 diabetes, transurethral resection of the prostate, incidence, risk factors.

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Abstract

Background: The relationship between type 2 diabetes and the incidence of transurethral resection of the prostate (TURP) remains uncertain.
Aims: To utilise data from the Fremantle Diabetes Study Phase I (FDS1) to examine the association between type 2 diabetes and incident TURP and investigate risk factors in men with type 2 diabetes.
Methods: First TURP hospitalisations were ascertained for males from the Fremantle Diabetes Study Phase I ($n = 581$) and age- and postcode-matched men without diabetes ($n = 2361$) between entry (1993–1996) and end (2017). Incidence rate ratios (IRRs) were calculated. Cox proportional hazards and competing risk models generated cause-specific (cs) and subdistribution (sd) hazard ratios (HRs) for incident TURP.
Results: There were 86 and 338 TURP hospitalisations in participants with and without type 2 diabetes, respectively, during 42 236 person-years of follow-up. The IRR (95% confidence interval) for diabetes versus no diabetes was 1.23 (0.96, 1.56). A 10-year age increase more than doubled the risk of incident TURP (csHR 2.51 (2.02, 3.12), sdHR 2.59 (2.11, 3.18)), but type 2 diabetes was not a significant predictor in multivariable models. In participants with type 2 diabetes, a 10-year age increase was predictive (csHR 2.94 (1.93, 4.47), sdHR 1.92 (1.51, 2.44)); Anglo-Celt versus other ethnic groups was significant in the Cox (csHR 1.87 (1.17, 3.00)) but not competing risk (sdHR 1.60 (0.99, 2.57)) models.
Conclusions: Type 2 diabetes does not increase TURP risk in community-based Australians. There are no diabetes-specific variables associated with incident TURP.

Introduction

At least one third of Australian men aged >50 years experiences the typical lower urinary tract symptoms (LUTSs) of benign prostatic hyperplasia (BPH) and the proportion increases with age.[1] When LUTSs become severe despite conventional non-surgical management, transurethral resection of the prostate (TURP) is usually recommended. The rates of TURP for BPH in Australia increase from around 2/1000 person-years in younger men and those with relatively mild LUTSs to >10/1000 person-years in those aged >75 years or with severe LUTSs.[2]

The interrelationships between type 2 diabetes, LUTSs, BPH and TURP are unclear. A recent meta-analysis found that, consistent with preclinical data,[3] diabetes of unspecified type was associated with significantly larger prostatic volumes and greater LUTS severity scores versus no diabetes in men with BPH.[4] However, there was substantial heterogeneity between studies, and the majority of included data were from China[4] against a background of significant racial variability in the incidence of BPH.[5] A further potential complicating factor

Abbreviations: ABSI, a body shape index; BMI, body mass index; BPH, benign prostatic hyperplasia; CCI, Charlson Co-morbidity Index; csHR, cause-specific hazard ratio; FDS1, Fremantle Diabetes Study Phase I; HDMC, Hospital Morbidity Data Collection; ICD, International Classification of Disease; IR, incident rate; IRR, incident rate ratio; LUTS, lower urinary tract symptoms; sdHR, subdistribution hazard ratio; TURP, transurethral resection of the prostate; WA, Western Australia; WADLS, Western Australian Data Linakge System

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that can increase the severity of LUTS independently of BPH in diabetes is neurovascular effects on bladder function.[6] In contrast to diabetes-specific aggravating factors, preclinical studies provide evidence that the blood glucose-lowering therapy metformin may lower the risk of BPH.[7] In addition, South Korean population-based administrative data show that its use may reduce the need for TURP.[8] However, short-term[9] and long-term[10] epidemiological studies have shown no relationship between type of blood glucose-lowering therapy and the incidence of BPH. Australian population-based data do not suggest that doctor-diagnosed diabetes increases the risk of TURP.[2] Since metformin is recommended as first-line therapy for type 2 diabetes,[11] it is possible that a neutral effect of diabetes on the need for TURP in Australian men reflects a balance between factors that increase and reduce the severity of BPH.

In light of this background, there is a need for a long-duration, prospective study of TURP in community-based, well-characterised Australian men with or without type 2 diabetes. The aim of this study was, therefore, to utilise longitudinal data from the Fremantle Diabetes Study Phase I (FDS1) (i) to examine the aetiological association between type 2 diabetes and incident TURP using well-characterised community-based cohorts and (ii) to investigate risk factors predicting incident TURP in men with type 2 diabetes.

Participants and methods

Study site, participants and approvals

The FDS1 is a cohort study of people living with diabetes in a postcode-defined urban community of 120 097 people. Descriptions of recruitment, sample characteristics including classification of diabetes type and details of non-recruited patients have been published.[12] Of 2258 people with diabetes identified from a population of 120 000 between 1993 and 1996, 1426 (63%) were recruited of whom 1296 (91%) had clinically defined type 2 diabetes. The FDS1 protocol was approved by the human rights committee at Fremantle Hospital and all subjects gave informed consent before participation. Four age-, sex- and postcode-matched residents with no prior documentation of diabetes were randomly selected from the catchment area for each FDS1 participant at the time of their enrolment using the Western Australian (WA) electoral roll.[13] For the purposes of the present study, we included 561 adult men with type 2 diabetes (mean ± SD age 62.7 ± 10.6 years) and 2296 matched men without diabetes (aged 63.0 ± 10.8 years) after excluding those with a prior hospitalisation for TURP.

Clinical and laboratory methods

Each FDS1 participant underwent a baseline assessment including a comprehensive questionnaire, a physical examination, additional investigations relevant to chronic complications and fasting biochemical tests performed in a single nationally accredited laboratory.[12] Details of medical conditions and their management, demographic, socioeconomic and lifestyle data were recorded. Racial/ethnic background was categorised according to self-selection, country/countries of birth and parents'/grandparents' birth, and language(s) spoken at home as either Anglo-Celt, Southern European, Other European, Asian, Aboriginal or mixed/other. Body mass index (BMI) was calculated, together with a body shape index (ABSI), which represents a more reliable estimate of visceral adiposity in relation to mortality.[14] Baseline micro- and macrovascular complications of diabetes were identified using standard criteria.[15]

Prior and incident TURP were ascertained through the WA Data Linkage System (WADLS),[16] which facilitates linkage with all hospitalisations (public and private) in WA. Data linkage was approved by the WA department of health human research ethics committee. TURP was identified using the code of surgical operations (1968) procedure code 06.33 (from January 1970 to December 1978), international classification of procedures in medicine (1978) procedure code 56.01 (from January 1979 to December 1987), International Classification of Disease (ICD)-9-CM (Australian v1) procedure code 60.2 (from January 1988 to mid-1996) and ICD-9-CM (Australian v2) procedure code 60.21 (from mid-1996 to mid-1999), or the later Australian Classification of Health Interventions code 37203–00 (from mid-1999 to mid-2017) and 37 224–03 (from mid-2017 onwards). Participants were followed from entry (or the equivalent date for each matched individual without diabetes) to first record of incident TURP, death or end-December 2017, whichever came first, and prior TURP was ascertained through linkage with the WA Hospital Morbidity Data Collection (HMDC) for operations from 1970 to study entry. Other pre-existing co-morbidity during the 5 years prior to study entry for both the type 2 diabetes and matched non-diabetes cohorts, excluding diabetes and its complications, was used to derive the Charlson Co-morbidity Index (CCI).[17]

Statistical methods

Data are presented as percentages (%), mean ± SD, geometric mean (SD range) or median (interquartile range). Two independent samples were compared using Fisher's exact test for proportions, Student's *t*-test for normally distributed variables and Mann–Whitney U test for variables

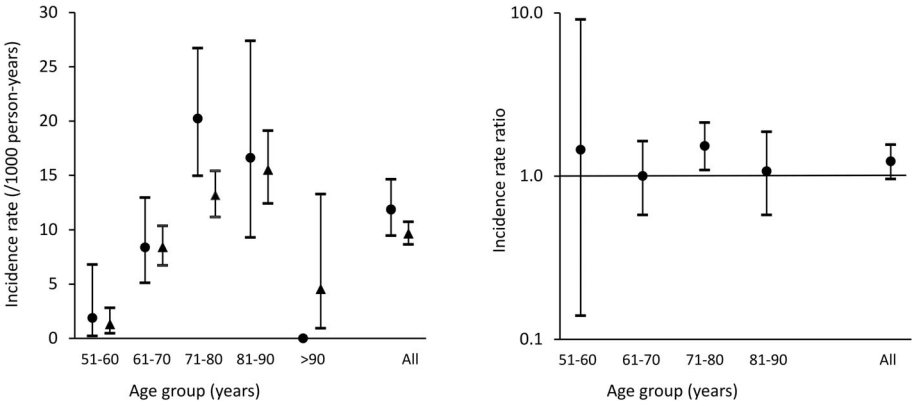

Figure 1 Incidence rates (left panel; type 2 diabetes ●, no diabetes ▲) and incidence rate ratios (●) and 95% confidence intervals (right panel). 95% confidence intervals are shown as vertical bars.

that were not normally or log-normally distributed. Ten-year age-specific TURP incident rates (IRs) in the type 2 diabetes cohort were compared with those derived for the non-diabetes cohort, and incident rate ratios (IRRs) were calculated. Cox proportional hazards modelling was used to generate cause-specific hazard ratios (csHRs) for incident TURP. Since the matched cohort without diabetes was deidentified, only variables which could be derived from linkage with the HMDC could be adjusted for, namely age and CCI. For the type 2 diabetes cohort, baseline risk factors for incident TURP were explored utilising the wide range of variables measured in the FDS1 clinical assessment and identified through data linkage. Variables with bivariable $P \leq 0.20$ were considered for entry into the Cox models (backward conditional variable selection with $P < 0.050$ for entry and ≥0.050 for removal). The competing risk of death was accounted for using Fine-Gray subdistribution (sd) hazards modelling.[18] The proportional hazards assumption was assessed, and if it was violated, significant time-varying covariates were included in the models. A two-tailed significance level of $P < 0.05$ was used throughout.

Results

Incidence of TURP in participants with or without type 2 diabetes

At baseline, 68 of 629 men with type 2 diabetes in the FDS2 cohort (10.8%) and 218 of 2514 of the matched men without diabetes (8.7%) had already had a TURP ($P = 0.103$). There were 86 and 338 TURP hospitalisations in participants with and without type 2 diabetes, respectively, during 42 236 person-years of follow-up. The IRs and IRRs by older age groups and for the total cohorts are shown in Figure 1. Over the total 42 236 person-years of follow-up, the overall IRR was 1.23 with 95% confidence interval that included unity (0.96 to 1.56). There were similar findings for deciles of age except for the 71–80 year age-group in which men with type 2 diabetes were 53% more likely to undergo TURP during follow-up than men without diabetes. For men older than 90 years, there were no first hospitalisations for TURP in the type 2 diabetes participants and a low IR in those without diabetes (Fig. 1). There were no documented TURP hospitalisations in either group in men younger than 50 years.

The Cox and competing risk models for incident TURP in the pooled type 2 diabetes and non-diabetes cohorts are summarised in Table 1. Increasing age was a significant predictor in both but with an effect that attenuated with longer duration of follow-up. Type 2 diabetes was not associated with incident TURP, while a CCI ≥3 halved the risk of incident TURP in competing risk analysis.

Predictors of TURP in men with type 2 diabetes

The characteristics of FDS1 male participants with type 2 diabetes categorised by incident TURP status are summarised in Table 2. Those who required a TURP during follow-up were significantly older at diabetes

Table 1 Cox and competing risk models of time to first hospitalisation for TURP ($n = 424$) in 2855 pooled FDS1 men with type 2 diabetes and their age- and postcode-matched counterparts without diabetes excluding those with a prior hospitalisation for TURP

	csHR	sdHR
Age (increase of 10 years)	2.51 (2.02, 3.12)	2.59 (2.11, 3.18)
CCI 0	1.00 (reference)	1.00 (reference)
1–2	1.13 (0.85, 1.50)	0.85 (0.64, 1.13)
≥3	1.06 (0.59, 1.90)	0.51 (0.28, 0.92)
Type 2 diabetes	1.25 (0.98, 1.59)	1.11 (0.87, 1.42)
Time-varying		
Age (increase of 10 years)	0.86 (0.77, 0.95)	0.72 (0.66, 0.79)

Cause-specific hazard ratios (csHRs) and subdistribution hazard ratios (sdHRs) are shown together with 95% confidence intervals.

Table 2 Baseline characteristics by incident TURP in men with type 2 diabetes in FDS1 and no prior hospitalisation for TURP

	No TURP	Incident TURP	*P*-value
Number (%)	475 (84.7)	86 (15.3)	
Age (years)	62.3 ± 11.0	65.5 ± 7.7	0.001
Ethnic background (%): Anglo-Celt	58.7	73.3	
Southern European	17.7	11.6	
Other European	9.7	7.0	0.113
Asian	4.2	0	
Aboriginal	1.3	1.2	
Mixed/other	8.4	7.0	
Not fluent in English (%)	14.9	7.0	0.059
Educated beyond primary level (%)	77.6	81.0	0.568
Age at diabetes diagnosis (years)	56.0 ± 11.4	59.8 ± 8.5	<0.001
Diabetes duration (years)	4.0 (1.0–10.0)	4.0 (1.0–8.0)	0.521
Fasting serum glucose (mmol/L)	8.4 (6.9–11.1)	8.9 (7.1–10.6)	0.643
HbA_{1c} (%)	7.6 (6.4–8.8)	7.5 (6.4–8.8)	0.806
HbA_{1c} (mmol/mol)	60 (46–73)	58 (46–73)	0.806
Diabetes treatment (%) Diet	29.1	41.9	
Oral hypoglycaemic agents (OHAs)	58.2	47.7	0.069
Insulin ± OHAs	12.7	10.5	
Metformin therapy (%)	32.4	24.4	0.164
Smoking status (%) Never	25.6	24.4	
Ex	53.2	61.6	0.238
Current	21.2	14.0	
Alcohol consumption (standard drinks†/day)	0.3 (0–1.5)	0.3 (0–1.5)	0.432
BMI (kg/m^2)	29.1 ± 4.7	28.3 ± 4.1	0.150
ABSI ($m^{11/6}\ kg^{-2/3}$)	0.084 ± 0.004	0.084 ± 0.004	0.702
Central obesity (by waist circumference‡ (%))	54.8	50.6	0.480
Systolic blood pressure (mmHg)	149 ± 23	152 ± 24	0.183
Diastolic blood pressure (mmHg)	82 ± 11	82 ± 12	0.635
Antihypertensive medication (%)	43.8	46.5	0.639
Angiotensin converting enzyme inhibitor (%)	18.9	11.6	0.125
Prazosin use (%)	3.4	9.3	0.020
Total serum cholesterol (mmol/L)	5.3 ± 1.1	5.1 ± 0.8	0.184
Serum HDL-cholesterol (mmol/L)	0.96 ± 0.28	0.98 ± 0.27	0.448
Serum triglycerides (mmol/L)	1.9 (1.1–3.5)	1.8 (1.0–3.2)	0.272
Lipid-modifying medication (%)	10.4	10.6	>0.999
Fibrate therapy (%)	3.2	0	0.144
Aspirin therapy (%)	23.7	32.6	0.104
Cerebrovascular disease (%)	10.1	9.3	>0.999
Coronary heart disease (%)	33.3	32.6	>0.999
Peripheral arterial disease (%)	28.8	22.1	0.239
Distal symmetrical polyneuropathy (%)	35.1	28.9	0.315
Any retinopathy (%)	19.2	17.9	0.880
eGFR category§ (%) ≥90 mL/min/1.73m^2	24.2	22.4	
60–89 mL/min/1.73m^2	54.2	60.0	
45–59 mL/min/1.73m^2	15.9	12.9	0.705
30–44 mL/min/1.73m^2	3.8	4.7	
<30 mL/min/1.73m^2	1.9	0	
Urinary albumin: creatinine ratio (mg/mmol)	3.0 (0.6–14.5)	2.3 (0.6–9.3)	0.120
CCI (%) 0	69.5	72.1	
1 or 2	24.8	20.9	0.721
≥3	5.7	7.0	

†One standard drink = 10 U ethanol.
‡≥102 cm in males and ≥88 cm in females.
§Estimated glomerular filtration rate category based on the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.[19]

diagnosis and study entry and were more likely to be treated with prazosin compared to those who did not undergo TURP, but there were no other statistically significant differences. The Cox and competing risk models for incident TURP in the males with type 2 diabetes are summarised in Table 3. Increasing age, Anglo-Celt ethnicity and prazosin use were significant predictors in both models, with the effect of age attenuating with longer duration of follow-up in the competing risk model.

Discussion

The present data do not support an independent role for type 2 diabetes, or for diabetes-specific risk factors

Table 3 Cox and competing risk models of time to first hospitalisation for TURP ($n = 88$) in 562 FDS1 men with type 2 diabetes (HR (95% CI)), excluding those with a prior hospitalisation for TURP

	csHR	sdHR
Age (increase of 10 years)	1.98 (1.53, 2.55)	2.87 (1.90, 4.34)
Anglo-Celt ethnicity	1.94 (1.20, 3.13)	1.66 (1.02, 2.69)
Prazosin use	2.42 (1.17, 5.02)	2.39 (1.16, 4.90)
Time-varying		
Age (increase of 10 years)		0.65 (0.54, 0.78)

Cause-specific hazard ratios (csHRs) and subdistribution hazard ratios (sdHRs) are shown together with 95% confidence intervals.

including metformin therapy, in modifying the risk of TURP in community-based Australian men. Closely paralleling previously published Australian general population data,[2] increasing age was the most powerful predictor of incident TURP in both pooled multivariable analyses with IRs increasing from <2/1000 person-years in men aged 51–60 years to >13/1000 person-years in participants aged 71–90 years. The fact that the influence of age declined with time even in the competing risk analysis suggests that this was not a survivor effect. In an Australian general population study, there was a flattening of the rise in incidence rates at age ≥75 years,[2] consistent with the present findings. In the FDS1 men with type 2 diabetes, Anglo-Celt ethnicity increased the risk of TURP in the Cox model but this was reduced to borderline significance in the competing risk analysis.

The attenuation of the effect of age on incident TURP with duration of follow-up likely reflects concerns associated with invasive surgery in older patients who may have limited life expectancy, as well as a relatively high risk of perioperative complications and a poor functional outcome.[20] This latter consideration includes the possibility that age-related detrusor underactivity, which can increase the risk of surgical failure,[21,22] may be worsened by the neurovascular effects of diabetes. Consistent with this observation is that fact that the peak TURP IR decile for men with type 2 diabetes was 10 years younger than in men without diabetes and there were no hospitalisations for TURP in men with diabetes aged >90 years. Nevertheless, careful patient selection, including a preoperative geriatric assessment as has been recommended in older men in the general population,[23] may lead to satisfactory outcomes including reduction in polypharmacy and avoidance of the adverse effects of medications prescribed for LUTSs in men with type 2 diabetes. The independent positive association between baseline prazosin use and incident TURP likely reflects confounding by indication. At the time of FDS1 recruitment, alpha-1 adrenergic receptor antagonists were the only approved and subsidised medication for LUTSs in Australia, the 5-alpha reductase inhibitors and especially the phosphodiesterase-5 inhibitors only becoming available towards the end of the FDS1 follow-up period.[24]

The reasons why there was some evidence, at least from the Cox model, that Anglo-Celt FDS1 participants with type 2 diabetes appeared more likely to proceed to TURP than other ethnic groups are uncertain. In a large US study ($n = 31\,699$ followed to first TURP with higher rates of surgery in White vs non-White men),[25] the authors concluded that racial and ethnic disparities were the primary reason for the differences given that confounders such as geographical region of residence, CCI and baseline comorbidities were controlled for. Other US studies showed inconsistent results,[26] but ethnic differences have been attributed to inequitable healthcare access and other psycho-socioeconomic factors. The Australian study of incident TURP[2] did not include ethnicity in multivariable analyses but concluded that around 30% higher TURP rates observed in men with higher income and private health insurance, after accounting for need, also suggested inequity in the use of the procedure. We had a range of sociodemographic variables including lack of fluency in English, which was associated with a close to 50% lower rate of incident TURP in bivariable analysis, but this was not independently significant. There may be other unmeasured contributory variables that could vary by ethnicity such as thresholds for reporting LUTS and their severity.[27]

We did not find that metformin therapy influenced the risk of incident TURP. This is consistent with a population-based Danish study showing that the 10-year TURP incidence in nearly 4000 men initiating metformin as first-line therapy for type 2 diabetes was similar to that in approaching 6000 men started on a sulfonylurea after adjustment for a range of confounding variables.[10] The South Korean study showing a protective effect of metformin against TURP used a much more limited number of variables in adjusted statistical models.[8] Our data did not reveal any other diabetes-specific variables such as glycaemic control and chronic complications that might influence incident TURP.

The main limitation of the present study is the use of administrative data for ascertainment of TURP with the possibility of coding errors. This issue is common to a range of other epidemiological studies,[2,8,10,25] but since TURP is a relatively simple procedure to classify this seems unlikely. We did not ascertain LUTS severity using a validated symptom score or have access to measures of prostate morphology or urodynamics, which may have strengthened the regression analyses. The strengths of our study are the large cohorts and long duration of follow-up, which enabled us to assess the risk of TURP stratified by diabetes status. In addition, TURP events were extracted from independent data sources through the WADLS,[16] which are regularly validated and would limit ascertainment bias, consistent with the close overall agreement in age-specific TURP incidence rates between the present study and a recent Australian population-based report.[2]

In conclusion, we did not find that type 2 diabetes significantly increases the risk of TURP in a community setting after adjustment for important confounding variables. The suggestion of an increased risk of incident TURP in Anglo-Celt participants with type 2 diabetes

may be worthy of further study directed at establishing whether patient- or system-specific discrepancies are at play.

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References

1 Homma Y, Kawabe K, Tsukamoto T, Yamanaka H, Okada K, Okajima E *et al*. Epidemiologic survey of lower urinary tract symptoms in Asia and Australia using the international prostate symptom score. *Int J Urol* 1997; **4**: 40–6.
2 Joshy G, Soga K, Korda RJ, Patel MI, Banks E. Factors related to receipt of non-cancer-related transurethral prostatectomy: findings from a large prospective study of 106 769 middle-aged and older Australian men. *BMJ Open* 2017; **7**: e013737.
3 Vignozzi L, Gacci M, Maggi M. Lower urinary tract symptoms, benign prostatic hyperplasia and metabolic syndrome. *Nat Rev Urol* 2016; **13**: 108–19.
4 Xin C, Fan H, Xie J, Hu J, Sun X, Liu Q. Impact of diabetes mellitus on lower urinary tract symptoms in benign prostatic hyperplasia patients: a meta-analysis. *Front Endocrinol (Lausanne)* 2021; **12**: 741748.
5 Hoke GP, McWilliams GW. Epidemiology of benign prostatic hyperplasia and comorbidities in racial and ethnic minority populations. *Am J Med* 2008; **121**: S3–S10.
6 Van Den Eeden SK, Ferrara A, Shan J, Jacobsen SJ, Quinn VP, Haque R *et al*. Impact of type 2 diabetes on lower urinary tract symptoms in men: a cohort study. *BMC Urol* 2013; **13**: 12.
7 Yang T, Yuan J, Peng Y, Pang J, Qiu Z, Chen S *et al*. Metformin: a promising clinical therapeutical approach for BPH treatment via inhibiting dysregulated steroid hormones-induced prostatic epithelial cells proliferation. *J Pharm Anal* 2024; **14**: 52–68.
8 Hong Y, Lee S, Won S. The preventive effect of metformin on progression of benign prostate hyperplasia: a nationwide population-based cohort study in Korea. *PLoS One* 2019; **14**: e0219394.
9 Murff HJ, Roumie CL, Greevy RA, Grijalva CG, Hung AH, Liu X *et al*. Thiazolidinedione and metformin use and the risk of benign prostate hyperplasia in veterans with diabetes mellitus. *J Mens Health* 2014; **11**: 157–62.
10 Norgaard M, Darvalics B, Thomsen RW. Metformin use and long-term risk of benign prostatic hyperplasia: a population-based cohort study. *BMJ Open* 2020; **10**: e041875.
11 Australian Diabetes Society. Australian Blood Glucose Treatment Algorithm for Type 2 Diabetes. 2025 [cited 2025 Jan]. https://treatment.diabetessociety.com.au/plan/.
12 Davis TM, Bruce DG, Davis WA. Cohort profile: the Fremantle Diabetes Study. *Int J Epidemiol* 2013; **42**: 412–21.
13 Magliano DJ, Davis WA, Shaw JE, Bruce DG, Davis TM. Incidence and predictors of all-cause and site-specific cancer in type 2 diabetes: the Fremantle Diabetes Study. *Eur J Endocrinol* 2012; **167**: 589–99.
14 Krakauer NY, Krakauer JC. Anthropometrics, metabolic syndrome, and mortality hazard. *J Obes* 2018; **2018**: 9241904.
15 Norman PE, Davis WA, Bruce DG, Davis TM. Peripheral arterial disease and risk of cardiac death in type 2 diabetes: the Fremantle Diabetes Study. *Diabetes Care* 2006; **29**: 575–580.
16 Holman C, Bass A, Rouse I, Hobbs M. Population-based linkage of health records in Western Australia: development of a health services research linked database. *Aust N Z J Public Health* 1999; **23**: 453–459.
17 Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. *J Chronic Dis* 1987; **40**: 373–83.
18 Fine JP, Gray RJ. A proportional hazards model for the subdistribution of a competing risk. *J Am Stat Assoc* 1999; **94**: 496–509.
19 Levey AS, Stevens LA, Schmid CH, Zhang Y(L), Castro AF III, Feldman HI *et al*. A new equation to estimate glomerular filtration rate. *Ann Intern Med* 2009; **150**: 604–12.
20 Eredics K, Meyer C, Gschliesser T, Lodeta B, Heissler O, Kunit T *et al*. Can a simple geriatric assessment predict the outcome of TURP? *Urol Int* 2020; **104**: 367–72.
21 Evrard PL, Mongiat-Artus P, Desgrandchamps F. Morbi-mortality of transurethral resection of the prostate in patients aged 75 and over. *Prog Urol* 2017; **27**: 312–8.
22 Antoniou V, Edris F, Akpobire W, Voss J, Somani B. Surgical outcomes for elderly patients undergoing transurethral resection of the prostate for chronic urinary retention and proposal of a management algorithm. *J Endourol* 2023; **37**: 581–6.
23 Lotterstatter M, Seklehner S, Wimpissinger F, Gombos J, Bektik J, Stolzlechner P *et al*. Transurethral resection of the prostate in 85+ patients: a retrospective, multicentre study. *World J Urol* 2022; **40**: 3015–3020.
24 Jiwrajka M, Yaxley W, Perera M, Roberts M, Dunglison N, Yaxley J *et al*. Review and update of benign prostatic hyperplasia in general practice. *Aust J Gen Pract* 2018; **47**: 471–5.
25 Narang GL, Rojanasarot S, Cutone B, Humphreys MR. Is race associated with

the surgical treatment for benign prostatic hyperplasia? An analysis of 30,000 medicare lives. *J Racial Ethn Health Disparities* 2024; **11**: 528–534.

26 Ayangbesan A, Kavoussi N. Racial disparities in diagnosis and management of benign prostatic hyperplasia: a review. *Curr Urol Rep* 2022; **23**: 297–302.

27 Fowke JH, Murff HJ, Signorello LB, Lund L, Blot WJ. Race and socioeconomic status are independently associated with benign prostatic hyperplasia. *J Urol* 2008; **180**: 2091–6.