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Original Article

Acute aortic syndrome: nationwide study of epidemiology, management, and outcomes

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Abstract

Background: Epidemiological studies on acute aortic syndrome (AAS) have relied largely on unverified administrative coding, leading to wide-ranging estimates of incidence. This study aimed to evaluate the incidence, management, and outcomes of AAS in Aotearoa New Zealand.

Methods: This was a national population-based retrospective study of patients presenting with an index admission of AAS from 2010 to 2020. Cases from the Ministry of Health National Minimum Dataset, National Mortality Collection, and the Australasian Vascular Audit were cross-verified with hospital notes. Poisson regression adjusted for sex and age was used to investigate trends over time.

Results: During the study interval, 1295 patients presented to hospital with confirmed AAS, including 790 with type A (61.0 per cent) and 505 with type B (39.0 per cent) AAS. A total of 290 patients died out of hospital between 2010 and 2018. The overall incidence of aortic dissection including out-of-hospital cases was 3.13 (95 per cent c.i. 2.96 to 3.30) per 100 000 person-years, and this increased by an average of 3 (95 per cent c.i. 1 to 6) per cent per year after adjustment for age and sex adjustment on Poisson regression, driven by increasing type A cases. Age-standardized rates of disease were higher in men, and in Māori and Pacific populations. The management strategies used, and 30-day mortality rates among patients with type A (31.9 per cent) and B (9.7 per cent) disease have remained constant over time.

Conclusion: Mortality after AAS remains high despite advances over the past decade. The disease incidence and burden are likely to continue to increase with an ageing population. There is impetus now for further work on disease prevention and the reduction of ethnic disparities.

Introduction

Acute aortic syndrome (AAS), including aortic dissections, intramural haematomas (IMHs) and penetrating aortic ulcers (PAUs), are a life-threatening group of conditions associated with significant morbidity and mortality. Key risk factors include uncontrolled hypertension[1,2] and atherosclerotic disease[3,4]. Despite improving treatments and established guidelines[5], mortality from this condition remains significant[1].

Sudden onset of disease, diagnostic difficulties, and high early mortality rates make quantification of the true disease incidence difficult. Previous nationwide and statewide studies[6,7] have relied on administrative coding to estimate incidence, systematically classifying type A or B dissections based on algorithms incorporating procedure codes. Studies using patient-level data to estimate incidence are rare[8,9]. Furthermore, many studies lack data on out-of-hospital death. Variations in epidemiological estimates are likely the result of a combination of true variation and a consequence of differences in research methodology, and range widely from 2.12 to 9 or more per 100 000 person-years[7–11].

In Aotearoa New Zealand (AoNZ), a centralized public healthcare system is responsible for all hospital admissions, allowing comprehensive coverage of all patients with AAS reaching hospital, regardless of surgical intervention. In more recent years, the development of the Australasian Vascular Audit (AVA) has also allowed prospective capture of all vascular surgery procedures in Australia and AoNZ, supplementing existing data sets[12]. The National Mortality Collection (MORT) with autopsy diagnoses is available in AoNZ for unexplained or sudden out-of-hospital deaths. These data provide the opportunity to verify the incidence of this disease rigorously.

The aim of this study was to estimate and determine trends in AAS incidence, management, and mortality in AoNZ over the past decade from 2010 to 2020. The secondary aim was to determine the discrepancy between administrative database and manual patient-level estimates of AAS incidence.

Methods

A population-based cohort study was conducted in accordance with the STROBE guidelines for observational studies[13]. Ethical approval was obtained before study commencement (HDEC 20/NTB/217).

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Data sources

Data for this study were obtained from three sources. Data from the National Minimum Dataset (NMDS) were sourced from the Ministry of Health. These data cover all public hospital admissions. Hospital admissions are recorded in the NMDS with corresponding ICD-10 Australian Modification (ICD-10-AM) diagnosis and procedure codes[14].

Publicly funded secondary and tertiary healthcare services in AoNZ were delivered through 20 district health boards (DHBs) based on geographical areas. Each DHB has at least one secondary-care or higher hospital delivering services to its geographical catchment area. Specialist cardiothoracic and vascular surgery services undertaking emergency aortic dissection surgery are restricted to five cardiothoracic and five vascular surgery centres providing tertiary care to a national population of 5.084 million (2020)[15].

The AVA data set, a prospectively maintained data set of all vascular surgery procedures in Australia and AoNZ[12], was cross-checked to supplement the NMDS with patients undergoing vascular or endovascular procedures for AAS.

The MORT captures the underlying cause of all deaths in New Zealand, both in and out of hospital[16]. Sudden or unexpected deaths are referred to the coroner in AoNZ, and a post-mortem examination is routinely considered if the cause of death is unclear. This data set has an administrative delay of 3 years, and data were available until 31 December 2018 at the time of analysis.

Patient inclusion

Patients with an ICD-10 code of thoracic or thoracoabdominal aortic dissection (I17.01 or I17.03 respectively) presenting to hospital from 1 January 2010 to 31 December 2020 were identified from the NMDS. Additional out-of-hospital deaths or deaths before admission were captured using the MORT data set. Data extracted included ICD-10-AM codes for discharge diagnoses and procedure codes using Australian Classification of Health Intervention system. Patients were linked using National Health Index numbers between the NMDS, MORT, and AVA data sets.

Because of the high readmission rates in AAS[17], a 10-year lookback interval was used to determine whether patients were presenting for an index event of AAS rather than a follow-up or unrelated presentation. The latter patients were excluded from the study. For patients with multiple hospital events and transfers, the date of first presentation to hospital was taken as the start of the index admission.

Exclusion criteria were: patients with an incidental chronic dissection, non-index presentations, iatrogenic or traumatic dissections, non-AoNZ residents, and those discharged from the emergency department alive with no admission or procedure. Patients with suspected dissections without radiological, surgical, or post-mortem confirmation of diagnosis were excluded.

Study variables and outcomes

Demographic characteristics of patients with AAS were examined including age, sex, ethnicity, and socioeconomic deprivation. Six ethnic categories were used for the purposes of this study: NZ European encompassing the Caucasian majority of the AoNZ population, NZ Māori encompassing the indigenous population, Pacific Island Peoples, Asian, Middle Eastern, North African/Latin American (MENA/LA), and other.

The New Zealand Index of Deprivation (NZDep2018) score which is calculated on nine domains (access to transport, access to communication, living space, income, recipient of benefit, single-parent family, home ownership, qualifications, and employment), is automatically assigned based on domiciliary address at each census. Patient NZDep2018 score was grouped into quintiles from least to most deprived.

Date of death was collected from the NMDS and MORT, and data were censored on 15 September 2021. Population data were sourced from publicly available data on Stats NZ based on national census data[15].

Patient management strategies were broadly categorized into open surgical management, endovascular management with or without debranching surgery, and medical management encompassing those not fit for surgery, those who died before intervention, and those managed without surgery. An endovascular procedure for type B AAS that occurred in the subacute phase within 6 weeks of the index admission was classified as endovascular management.

Pathological classification

The ICD-10 administrative coding system does not differentiate between Stanford type A or type B aortic dissections. Therefore, patients were classified into type A or type B AAS by manual review of radiology images and clinical notes. True aortic dissections were classified using the Stanford classification. IMHs and PAUs were classified according to their location in a similar fashion[18]. For patients in whom more than one type of AAS was present, the most severe pathology took priority in the classification (dissection followed by IMH followed by PAU). A vascular surgeon reviewed the images to determine the final classification.

Patients were classified as having uncomplicated or complicated type B aortic dissection based on the presence of intractable pain or intractable hypertension for more than 24 h, rapid expansion on CT, rupture, haemodynamic shock, or presence of malperfusion syndrome[19].

Outcomes

The primary outcome of the study was AAS incidence per 100 000 person-years.

Secondary outcomes included 30-day and 1-year mortality calculated from the time of the start of the index admission for AAS.

Sensitivity analysis

A sensitivity analysis was conducted to determine the discrepancy in incidence calculated based on patient-level data *versus* that based on systematic classification of administrative data using methods similar to those of McClure *et al.*[6]. The classification algorithm for AAS based on administrative procedural coding is presented in *Tables S1 and S2*. The degree of agreement between the two methods in classifying subtypes of AAS was reported as Cohen's κ coefficient. The absolute difference in AAS incidence between the two methods was calculated.

Statistical analysis

Data are presented as number with percentage for categorical data and mean(s.d.) or median (i.q.r.) for continuous variables, and compared between groups using independent-samples χ^2 test, one-way ANOVA, and Kruskal–Wallis test respectively. Age-, sex-, and ethnicity-adjusted rates of AAS were calculated

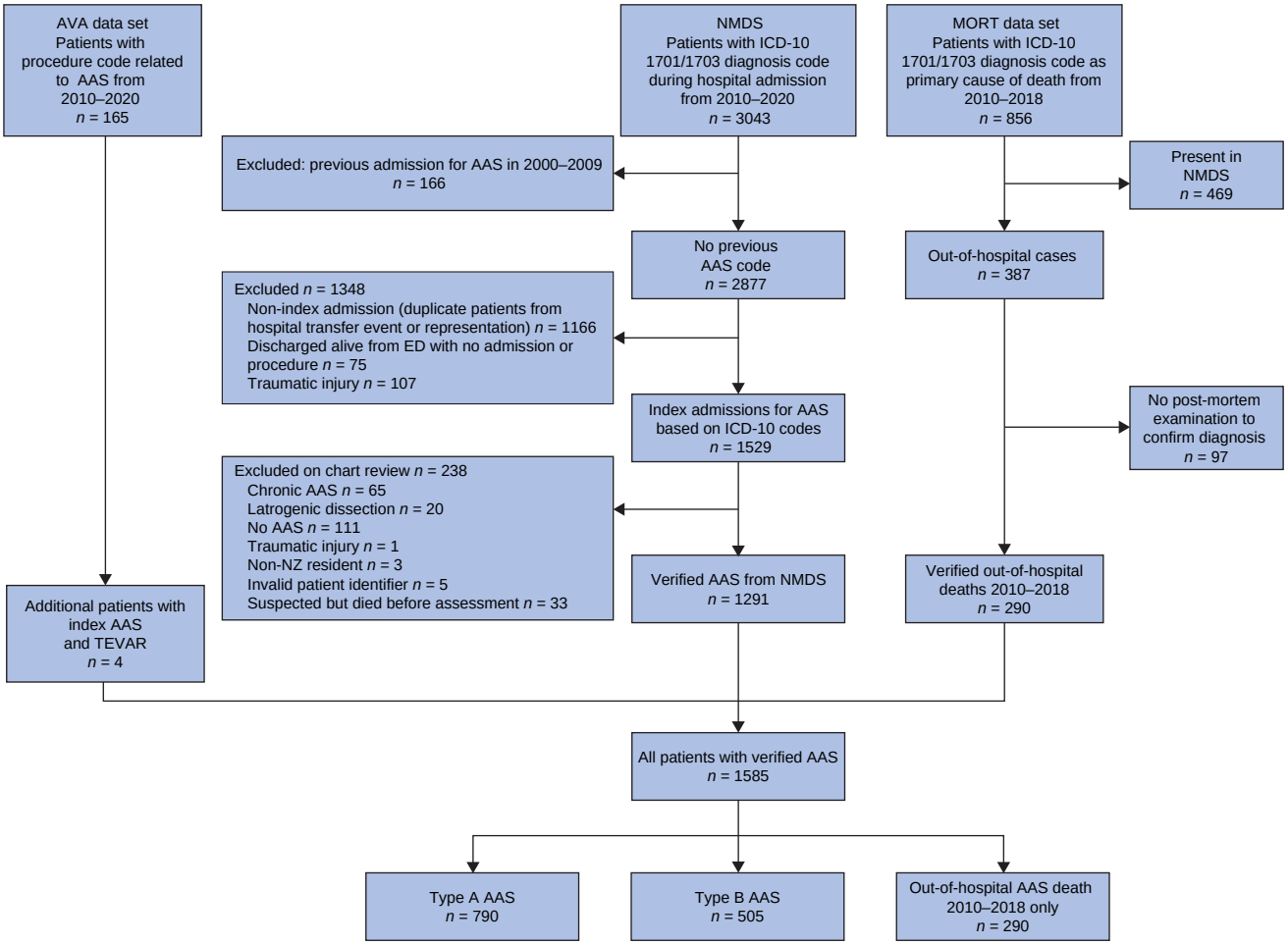

Fig. 1 Study flow chart

AVA, Australasian Vascular Audit; AAS, acute aortic syndrome; TEVAR, thoracic endovascular aortic repair; NMDS, National Minimum dataset; ED, emergency department; NZ, New Zealand; MORT, National Mortality Collection.

using available population data from the AoNZ census[15]. Age standardization was completed using standard populations modelled after the AoNZ Māori population and AoNZ non-Māori population in addition to the WHO standard population[20], in accordance with the methods of Robson *et al.*[21]. As out-of-hospital death data were available until 2018, a separate analysis including only cases from 2010 to 2018 was conducted to estimate AAS incidence in New Zealand including both in-hospital and out-of-hospital cases. The denominator populations for each year and age group were estimated by linear interpolation between the 2013 and 2018 census populations[15]. The number of events per year grouped by sex and prespecified age groups per year were modelled using a Poisson regression model with the logarithm of population size as the offset variable to determine the relative change in incidence per calendar year, accounting for changing population structures[22]. A statistical significance threshold of $P < 0.050$ was used. All analyses were performed in R version 4.0.3 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Patients and demographics

Some 3043 patients with a relevant ICD-10-AM diagnostic code were identified from the NMDS (*Fig. 1*). After exclusions and patient verification, there were 1585 patients with AAS. An additional 4 patients not previously included were identified from the AVA dataset. A total of 1295 index AAS cases presented to hospital, including 790 type A (61.0 per cent) and 505 type B (39.0 per cent) dissection. Some 290 patients with AAS died outside hospital and were identified by post-mortem examination from 2010 to 2018 (*Table 1*).

The average mean age of patients was 65.5 (range 19–94) years, and there was a male preponderance (57.7 per cent) (*Table 1*). Patients affected by AAS presenting to hospital were over-represented in the higher deprivation quintiles (13.7 per cent of patients in lowest quintile *versus* 26.5 per cent in highest quintile; $P < 0.001$). The most common co-morbidities in patients presenting to hospital included hypertension (66.3 per cent), smoking (ex-smoker or current smoker 42.1 per cent), ischaemic heart disease (13.4 per cent), and a history of stroke or transient ischaemic attack (10.6 per cent). A total of 114 patients (8.8 per cent) had a history of abdominal aortic aneurysm at time of presentation to hospital with AAS.

Overall, 326 of 1295 in-hospital cases were classified as IMH (20.6 per cent) including 141 of 790 type A IMH (17.8 per cent) and 185 of 505 type B IMH (36.6 per cent) (*Table 1*). Isolated PAUs were found in 15 of 1295 patients (1.1 per cent).

Incidence of acute aortic syndrome

The incidence of in-hospital AAS across 2010–2020 was 2.53 (95 per cent c.i. 2.39 to 2.67) per 100 000 person-years. Incidences for

Table 1 Baseline demographics

	Prehospital death (*n* = 290)	Type A (*n* = 790)	Type B (*n* = 505)	Total (*n* = 1585)	*P**
Age (years), mean(s.d.)†	65.7(12.8)	64.2(14.4)	67.3(14.4)	65.5(14.2)	0.001†
Sex (F : M)*	138 : 152	333 : 457	199 : 306	670 : 915	0.080
Ethnicity*					< 0.001
European	188 (64.8)	464 (58.7)	308 (61.0)	960 (60.6)	
Māori	0 (0.0)	155 (19.6)	91 (18.0)	246 (15.5)	
Pacific Island Peoples	23 (7.9)	94 (11.9)	60 (11.9)	177 (11.2)	
Asian	18 (6.2)	61 (7.7)	35 (6.9)	114 (7.2)	
MENA/LA	1 (0.3)	6 (0.8)	3 (0.6)	10 (0.6)	
Other	0 (0)	10 (1.3)	8 (1.6)	18 (1.1)	
Missing	60 (20.7)	0 (0)	0 (0)	60 (3.8)	
NZDep18 centiles*					0.593
1–2		115 (14.9)	59 (11.9)	174 (13.7)	
3–4		140 (18.2)	98 (19.7)	238 (18.8)	
5–6		139 (18.1)	96 (19.3)	235 (18.5)	
7–8		174 (22.6)	110 (22.1)	284 (22.4)	
9–10		202 (26.2)	134 (27.0)	336 (26.5)	
Missing	290 (100)	–	–	–	
AAS classification*					< 0.001
Dissection	–	635 (80.4)	302 (59.8)	937 (59.1)	
IMH	–	141 (17.8)	185 (36.6)	326 (20.6)	
PAU	–	4 (0.5)	11 (2.2)	15 (0.9)	
Missing	–	10 (1.3)	7 (1.4)	17 (1.3)	
Hypertension*					0.141
Yes		507 (64.2)	351 (69.5)	858 (66.3)	
No		231 (29.2)	125 (24.8)	356 (27.5)	
Missing		52 (6.6)	29 (5.7)	81 (6.3)	
Diabetes mellitus*					0.011
Yes		51 (6.5)	56 (11.1)	107 (8.3)	
No		700 (88.6)	428 (84.8)	1128 (87.1)	
Missing		39 (4.9)	21 (4.2)	60 (4.6)	
Ischaemic heart disease*					0.004
Yes		86 (10.9)	87 (17.2)	173 (13.4)	
No		667 (84.4)	398 (78.8)	1065 (82.2)	
Missing		37 (4.7)	20 (4.0)	57 (4.4)	
Congestive heart failure*					0.762
Yes		56 (7.1)	35 (6.9)	91 (7.0)	
No		696 (88.1)	450 (89.1)	1146 (88.5)	
Missing		38 (4.8)	20 (4.0)	58 (4.5)	
Stroke/TIA*					0.440
Yes		88 (11.1)	49 (9.7)	137 (10.6)	
No		662 (83.8)	436 (86.3)	1098 (84.8)	
Missing		40 (5.1)	20 (4.0)	60 (4.6)	
Connective tissue disease*					0.370
Yes		19 (2.4)	18 (3.6)	37 (2.9)	
No		731 (92.5)	466 (92.3)	1197 (92.4)	
Missing		40 (5.1)	21 (4.2)	61 (4.7)	
Abdominal aortic aneurysm*					< 0.001
Yes		41 (5.2)	73 (14.5)	114 (8.8)	
No		718 (90.9)	415 (82.2)	1133 (87.5)	
Missing		31 (3.9)	17 (3.4)	48 (3.7)	
Tobacco smoker*					< 0.001
Current smoker		148 (18.7)	92 (18.2)	240 (18.5)	
Ex-smoker		156 (19.7)	150 (29.7)	306 (23.6)	
Non-smoker		403 (51.0)	222 (44.0)	625 (48.3)	
Missing		83 (10.5)	41 (8.1)	124 (9.6)	

Values are *n* (%) unless indicated otherwise. Out-of-hospital death data are complete only to 2018 rather than the full 2020 study interval. MENA/LA, Middle Eastern, North African/ Latin American; NZDep, New Zealand Index of Deprivation; AAS, acute aortic syndrome; IMH, intramural haematoma; PAU, penetrating aortic ulcer; TIA, transient ischaemic attack. *χ^2 test, † one-way ANOVA.

type A and B AAS were 1.54 (1.43 to 1.65) and 0.99 (0.90 to 1.08) per 100 000 person-years respectively (*Table S3*).

Including autopsy-confirmed out-of-hospital AAS cases from 2010 to 2018, the overall incidence of AAS was 3.13 (2.96 to 3.30) per 100 000 person-years (*Table S4*). The incidence of type A, type B, and out-of-hospital cases was 1.49 (1.37 to 1.61), 0.94 (0.84 to 1.03), and 0.70 (0.62 to 0.79) per 100 000 person-years respectively. In the interval 2010– 2018, out-of-hospital deaths comprised 22.5 per cent of the total 1288 AAS cases.

The peak incidence of cases was reached in the eighth decade of life (*Tables S3 and S4*). Type A dissections were more common than type B in almost all subgroups examined, except for patients of the ninth decade of life (*Table S3 and S4*). Considering only patients aged over 70 years, including out-of-hospital deaths, the incidence was 13.30 (12.20 to 14.40) per 100 000 person-years.

When stratified by NZDep index for in-hospital cases, the incidence of disease in the most deprived cohort was 3.28 (2.93

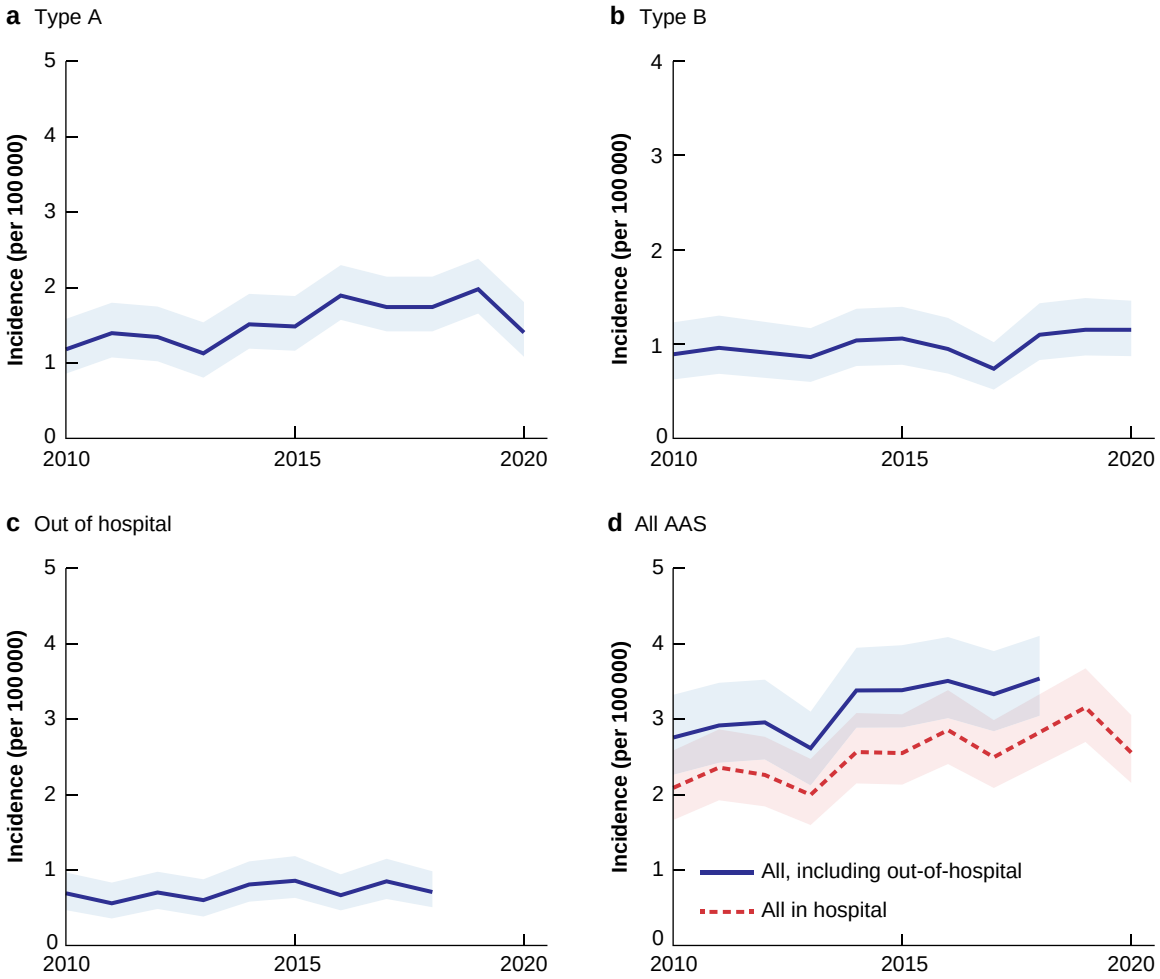

Fig. 2 Yearly incidence of aortic dissection over study interval

Incidence of **a** type A and **b** type B acute aortic syndrome (AAS), and **c** out-of-hospital mortality owing to AAS over time; **d** incidence of all in-hospital cases of AAS until 2020 year end, and of all cases of AAS including out-of-hospital deaths until 2018 year end. Data for out-of-hospital deaths from AAS are complete to only 2018. Rates presented are age- and sex-standardized to the Aotearoa New Zealand population based on 2013 and 2018 census data, with linear interpolation. Shaded areas represents 95 per cent confidence intervals.

to 3.63) per 100 000 person-years, significantly higher than that in the least deprived quintile (1.7 (1.45 to 1.95) per 100 000 person-years).

Age- and sex-standardized incidence over time

There was an overall mild increasing trend in the age-standardized incidence of aortic dissection in AoNZ (*Fig. 2d*) (2010: 1.98 (95 per cent c.i. 1.58 to 2.44) per 100 000 person-years; 2020: 2.65 (2.22 to 3.14) per 100 000 years), with an average 3 (95 per cent c.i. 1 to 5) per cent increase per year ($P < 0.001$). This trend remained when out-of-hospital deaths from 2010 to 2018 were included (*Fig. 2c*) (2010: 1.91 (1.66 to 2.18) per 100 000 person-years; 2018: 2.34 (2.10 to 2.61) per 100 000 person-years), with an average 3 (1 to 6) per cent increase in incidence per year ($P = 0.020$).

The increasing overall incidence was attributable to an increase in the incidence of type A AAS presenting to hospital (2010: 1.18 (0.88 to 1.57) per 100 000 person-years; 2020: 1.4 (1.10 to 1.75) per 100 000 years) with an average 4 (1 to 6) per cent increase per year ($P < 0.001$) (*Fig. 2a*). There was no significant change in the incidence of type B dissections over the study interval (2010: 0.91 (0.64 to 1.26) per 100 000 years; 2020: 1.12 (0.85 to 1.44) per 100 000 person-years) (*Fig. 2b*). There was also no change in the incidence of out-of-hospital deaths from 2010 to 2018 (2010: 0.64 (0.41 to 0.88) per 100 000 person-years; 2018: 0.71 (0.48 to 0.95) per 100 000 person-years; risk ratio 1.03 (95 per cent c.i. 0.98 to 1.07) per year, $P = 0.257$) (*Fig. 2c*).

The age-adjusted incidence of aortic dissection was higher in men than women (2.68 (2.54 to 2.82) *versus* 1.63 (1.53 to 1.74) per 100 000 person-years standardized to the WHO standard population). The age-standardized incidence of aortic dissection was significantly higher for Māori (3.31 (2.84 to 3.83) per 100 000 person-years) and Pacific (6.68 (5.65 to 7.85) per 100 000 person-years) populations than for patients of European descent when adjusted for the WHO standard population, as well as when adjusted for the Māori and non-Māori overall population (*Table 2*).

Management of aortic dissection

Most patients with type A AAS underwent open surgical repair (596 of 790,75.4 per cent). Some 194 of 790 patients with type A AAS (24.6 per cent) were managed without surgical intervention, including 74 (9.4 per cent) who died within 24 h of presentation before intervention. Most type B dissections were managed medically (387 of 505, 76.6 per cent) (*Table S5*); nine of these patients (1.8 per cent) died within the first 24 h of presentation

Table 2 Age-standardized incidence according to ethnicity and sex

	No. of cases	Population	Standardized rate (per 100 000 person-years)		
			Using Māori standard	Using non-Māori standard	Using WHO standard
Age-and sex-standardized incidence by ethnicity including all cases from 2010 to 2018					
Asian	89	5 216 270	1.52 (1.22, 1.87)	3.47 (2.72, 4.3)	2.00 (1.60, 2.47)
European and other	861	26 694 460	1.36 (1.26, 1.48)	2.82 (2.64, 3.02)	1.72 (1.60, 1.85)
Māori	186	6 543 490	2.84 (2.45, 3.28)	4.87 (4.13, 5.71)	3.31 (2.84, 3.83)
Pacific	152	2 681 125	5.61 (4.75, 6.58)	10.11 (8.43, 12.02)	6.68 (5.65, 7.85)
Age-standardized incidence by sex including all cases from 2010 to 2018					
F	1102	41 824 200	1.26 (1.18, 1.35)	2.75 (2.59, 2.91)	1.63 (1.53, 1.74)
M	1474	40 446 490	2.25 (2.13, 2.38)	4.05 (3.85, 4.27)	2.68 (2.54, 2.82)
Age- and sex-standardized incidence by ethnicity including in-hospital cases only from 2010 to 2020					
Asian	96	6 848 890	1.24 (1.00, 1.52)	2.77 (2.17, 3.48)	1.59 (1.27, 1.97)
European and other	799	32 764 600	1.00 (0.92, 1.09)	2.1 (1.95, 2.25)	1.25 (1.15, 1.34)
Māori	246	8 227 760	2.99 (2.62, 3.39)	5.25 (4.53, 6.05)	3.46 (3.03, 3.94)
Pacific	154	3 361 005	4.56 (3.86, 5.35)	8.15 (6.79, 9.70)	5.34 (4.52, 6.27)
Age-standardized incidence by sex including in-hospital cases only from 2010 to 2020					
F	1064	51 965 350	0.99 (0.92, 1.06)	2.13 (2.00, 2.26)	1.25 (1.17, 1.33)
M	1526	50 439 160	1.87 (1.77, 1.98)	3.38 (3.21, 3.55)	2.20 (2.09, 2.31)

Values in parentheses are 95% confidence intervals. Values are standardized with respect to the Aotearoa New Zealand Māori and non-Māori populations, and the WHO standard population according to the methods of Robson *et al.*[20,21]. Data are stratified by all acute aortic syndrome cases and in-hospital cases only.

without intervention. Endovascular management by thoracic endovascular aortic repair was undertaken in 112 of 505 patients (22.2 per cent) with type B AAS in the acute or subacute phase (within 6 weeks), whereas a small minority underwent open surgical repair (6 of 505, 1.2 per cent). Trends in management strategies, including rates of endovascular repair, did not change over the study interval (*Fig. S1*).

A total of 155 of 505 type B dissections (30.7 per cent) were classified as complicated (29 for expansion, 18 for rupture, 43 for malperfusion, 45 for intractable hypertension, 17 for intractable pain, and 3 for haemodynamic shock). Of 155 patients with complicated type B dissections, 82 (53.0 per cent) were managed with endovascular techniques in the subacute phase (within 6 weeks). Some 63 patients (40.6 per cent) underwent medical management at initial presentation, of whom 30 (48 per cent) eventually underwent delayed endovascular interventions. Overall, 6 of the 155 patients (3.9 per cent) with complicated type B dissections died before intervention, whereas 4 (3.9 per cent) underwent open surgical procedures.

Mortality of aortic dissection

Overall, the 30-day all-cause mortality rate among patients with type A and B AAS admitted to hospital in AoNZ was 31.9 and 9.7 per cent respectively, increasing to 35.4 and 16.8 per cent respectively at 1 year after presentation (*Table S5*). In those with type A AAS, 30-day and 1-year mortality rates were higher in those who did not have surgery, as expected (30 days: 70.6 *versus* 19.3 per cent, $P < 0.001$; 1 year: 76.3 *versus* 22.1 per cent, $P < 0.001$). In those with type B AAS, unadjusted 30-day and 1-year mortality rates were comparable between patients who were managed medically and those who had endovascular treatment (30 days: 8.5 *versus* 12.5 per cent, $P = 0.278$; 1 year: 16.3 *versus* 17.0 per cent; $P = 0.978$).

In analyses stratified by sex, there was a trend towards higher mortality rates among women after type A AAS at 30 days (118 of 333 women (35.4 per cent) *versus* 134 of 457 men (29.3 per cent); $P = 0.081$) and 1 year (131 of 333 (39.3 per cent) *versus* 149 of 457 (32.6 per cent) respectively; $P = 0.060$). After type B AAS, there were no significant sex differences in mortality at 30 days (21 of 199 women (10.6 per cent) *versus* 28 of 306 men (9.2 per cent); $P = 0.714$) or 1 year (35 of 199 (17.6 per cent) *versus* 49 of 306 (16.0 per cent) respectively; $P = 0.732$).

Short-term 30-day mortality rates among patients with type A and B AAS, or all AAS together did not change significantly over the study interval (OR 1.01 (95 per cent c.i. 0.97 to 1.05) per year; $P = 0.570$) (*Fig. 3*).

Accuracy of administrative coding

Of the 1529 patients who were classified as having an acute aortic dissection based on administrative coding alone, 3 patients (0.2 per cent) were excluded as non-AoNZ residents and 5 (0.3 per cent) owing to invalid identifiers. Overall, 65 patients (4.3 per cent) had chronic dissections, 20 had iatrogenic dissections (1.2 per cent), and 1 had a traumatic aortic injury (0.1 per cent); 33 patients (2.2 per cent) had a suspected dissection without radiological diagnosis, and 111 (7.3 per cent) were misclassified as having an aortic dissection (*Fig. 1*). Therefore, a total of 230 patients (15.0 per cent) were misclassified as having acute non-iatrogenic, non-traumatic aortic dissection.

In analyses with aortic dissection stratified into type A AAS managed procedurally, type B AAS managed procedurally, and aortic AAS managed medically, there was agreement in 80 per cent of cases ($\kappa = 0.702$) (*Table S6*). The estimated national in-hospital incidence of aortic dissection based on administrative data was 2.99 (95 per cent c.i. 2.84 to 3.14) per 100 000 person-years, compared with 2.53 (2.39 to 2.67) per 100 000 person-years based on verified diagnoses (*Table 3* and *Table S6*).

Discussion

AAS remains a rare condition with a high mortality rate in AoNZ. Through rigorous patient-level verification of diagnoses, an increasing in-hospital incidence in AoNZ was identified, driven by higher numbers of type A dissections. Despite advances in

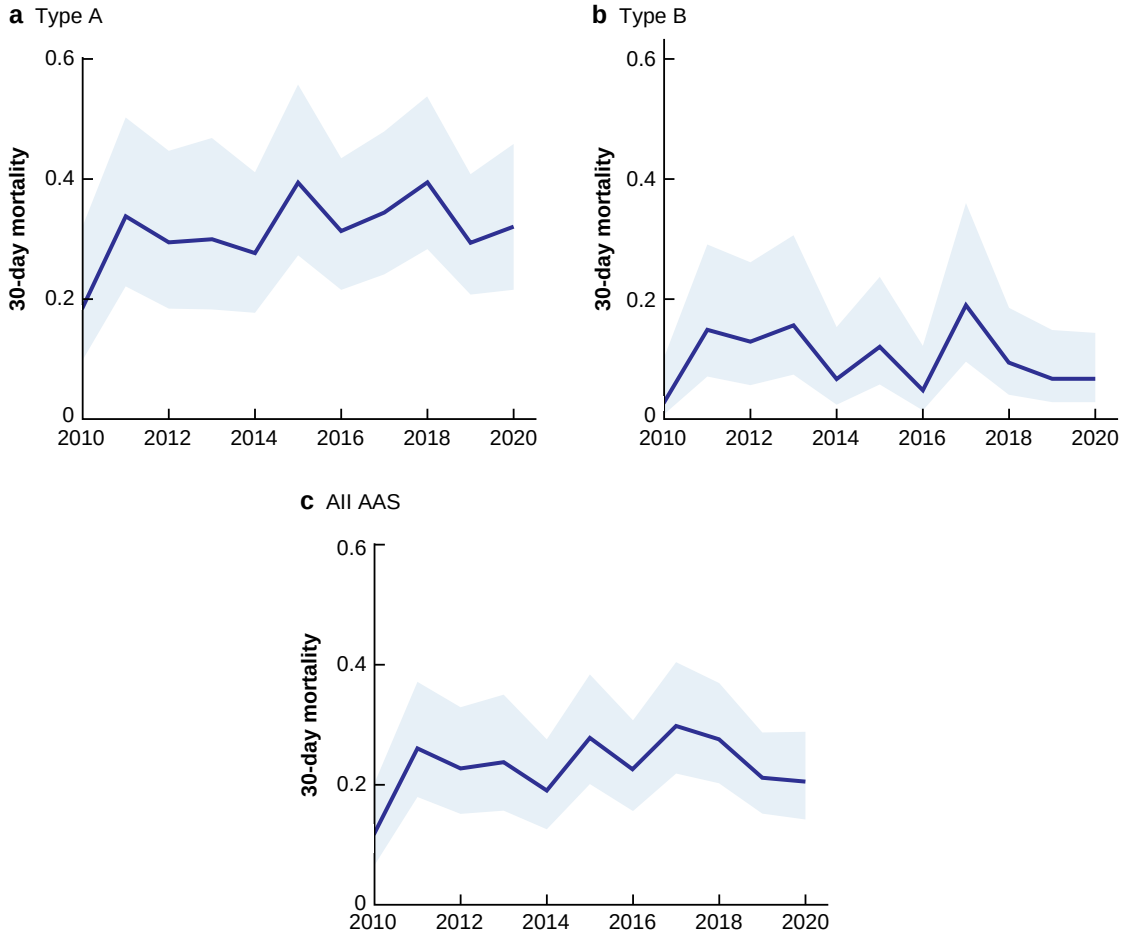

Fig. 3 Yearly 30-day mortality rate of in-hospital cases of acute aortic syndrome over study interval
Shaded areas represents 95 per cent confidence intervals. Stratified by **a** type A, **b** type B, and **c** all cases.

Table 3 Discrepancy between administrative coding and verified incidence of in-hospital cases

	Administrative coding-determined incidence per 100 000 person-years	Verified incidence per 100 000 person-years
Overall	2.99 (2.84, 3.14)	2.53 (2.39, 2.67)
Type A—procedural	1.21 (1.11, 1.3)	1.16 (1.07, 1.26)
Type B—procedural	0.29 (0.25, 0.34)	0.24 (0.19, 0.28)
Medical	1.49 (1.39, 1.6)	1.13 (1.04, 1.22)

Values in parentheses are 95% confidence intervals.

surgical and perioperative care, the mortality rate for this condition has remained significant without improvement over the past decade.

After patient-level verification and inclusion of out-of-hospital deaths, this population-based study estimated the incidence of AAS to be 3.13 (95 per cent c.i. 2.96 to 3.30) per 100 000 person-years. Although this is lower than the overall pooled global estimate of AAS incidence in a recent systematic review[11] of population-based studies, the present result is consistent with estimates from Iceland (1992–2013), where the incidence was 2.53 per 100 000 person-years after manual verification of cases[8]. Administrative coding is likely to misclassify a significant portion of AAS cases[23], and is limited in its ability to differentiate true acute from chronic aortic dissections, incidental findings, or other aortic pathology such as abdominal aortic aneurysms. Previous administrative studies[6] without verification have shown that type B is more common than type A AAS, which contrasts with the expected distribution of type A and B dissections[1]. In total, 58 per cent of patients who were initially coded as having AAS within the national database were excluded, including 15.0 per cent of patients who were classified incorrectly despite application of previously published classification algorithms. This further emphasizes the need for verification to ensure the accuracy of population-level estimates of disease.

Hospital-based studies underestimate the true community-based incidence of disease by up to 33 per cent owing to in-community deaths[9,24]. A Swedish study[25] reported a rate of 7.2 per 100 000 person-years after inclusion of out-of-hospital deaths, which comprised 29 per cent of all cases, and the Oxford Vascular Study[9] reported the community incidence of AAS to be 5.6 per 100 000 person-years. The present findings are in line with these previous studies, showing that, from 2010 to 2018, 22.5 per cent of patients with confirmed AAS died out of hospital, highlighting the importance of including out-of-hospital deaths in epidemiological estimates.

This study showed a mild increase in incidence driven by a slowly increasing rate of type A dissections. This finding has been replicated in other studies[6,10,26], but not universally[7,8]. It remains unclear whether this increase in observed disease is due to the increasing availability of acute CT, better diagnostic awareness, improved out-of-hospital patient stabilization, or a true increasing disease burden[6]. The prevalence of hypertension in New Zealand has increased since the early 2000s[27], but may have plateaued in recent years[28]. Whether increases in incidence can be explained by improved case ascertainment or an increasing prevalence of risk factors remains to be determined. Regardless, given that the peak incidence of AAS in this study was in the eighth decade of life, an increasing ageing population in New Zealand will lead to an increased absolute number of events[29].

A significant finding of the study was that the mortality risk for this disease has remained constant since 2010. Improvements in short-term mortality have been demonstrated inconsistently[6,8], although some studies[1,7,30], including a single-centre study from AoNZ, have shown improvements in mortality for surgically repaired type A AAS. The reason why improving mortality was not observed in the present study is likely multifactorial. Socioeconomic disparities, geographical isolation, and differential access to acute imaging all may contribute to the unchanging 30-day mortality rate after hospital admission[31]. Given the high risk of death in the early phase of disease, prevention of primary disease by management of hypertension and smoking cessation remains key to reducing the impact and burden of AAS nationally[9].

The continued advancements in endovascular technology have resulted in increasing use of endovascular intervention for both complicated and now uncomplicated type B AAS with high risk of late complications[1,32]. Future nationwide studies investigating patient selection, timing of intervention, and indication for the procedure may yield useful insights into variation in practice and quantify long-term outcomes adjusted for co-morbidities.

Particular attention needs to be drawn to the disparities highlighted in this study. AoNZ, Māori, and Pacific populations had a higher age-standardized incidence of AAS. Poorer health outcomes and higher cardiovascular disease burden in Māori and Pacific communities are documented widely across a number of different disease processes[33–36], driven by entrenched systemic inequalities and institutionalized racism rooted in colonialism, and may arise further from implicit biases among clinicians[37]. AoNZ, like its European counterparts, has a free national tertiary healthcare system but a privatized primary care system. Socioeconomic barriers to preventative care likely contributed to these observed disparities and are a focus of ongoing policy reform. Although the present study was not designed to explore the effect of these disparities on disease survival, these findings highlight yet again the need for targeted action to address health inequalities[33].

The single-provider healthcare system in AoNZ allowed complete national analysis with cross-matching of supplementary registries and individual-level case verification. However, this study has its limitations. Although extensive action was taken to verify all cases included in the present study and maximize accuracy, this was at the cost of excluding cases without radiological, surgical, or autopsy diagnostic confirmation. Furthermore, this study was still reliant on clinical coding to assign an AAS diagnosis and could not to account for diagnoses not coded as AAS.

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Disclosure

The authors declare no conflict of interest.

Supplementary material

Supplementary material is available at *BJS* online.

Data availability

Data used for analysis by authors will be shared upon reasonable request.

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