BMI is a key risk factor for early periprosthetic joint infection following total hip and knee arthroplasty

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ABSTRACT

AIM: To identify patient and surgical risk factors that are associated with periprosthetic joint infection (PJI), especially whether obesity is a risk factor following total hip arthroplasty (THA) and total knee arthroplasty (TKA).

METHODS: New Zealand Surgical Site Infection Improvement Programme data was analysed using deep infection within 90 days of the index procedure as the outcome. This was tested against surgical and patient factors for statistical associations in a multivariate model.

RESULTS: A total of 10,690 primary THAs and 9,481 primary TKAs were recorded by the NZSSIIP between 2013 and 2015. Multivariate analysis showed statistically significant associations with deep infections for BMI (BMI >40kg/m^2 OR 5.62, 95% CI 2.25–14.0), male gender (OR 1.7, 95% CI 1.05–2.74) and age greater than 75 for THAs (age <55 years OR 0.35, 95% CI 0.14–0.87). For TKAs, multivariate analysis showed statistically significant associations with deep infection for BMI (BMI >40kg/m^2 OR 1.94, 95% CI: 0.63–5.70) and male gender (OR 2.96, 95% CI 1.51–5.80).

CONCLUSIONS: These findings show that obesity is one of the most important modifiable patient factors in predicting PJI following THA and TKA.

Periprosthetic joint infection (PJI) is a devastating complication of total joint arthroplasty. It results in significant disability for the patient and burdens the healthcare system with significant costs.[1–3] In New Zealand, each PJI adds an excess mean treatment cost of $40,121 and an additional 42 days in hospital, with an overall burden of $8 million per annum to the New Zealand healthcare system.[4] In the last four decades, large strides have been made in improving surgical technique, theatre environment and prophylactic antibiotic use.[5,6] Despite these efforts, the rate of PJI has not reduced and may have increased over the same time period.[7,8] This finding may be due to changes in modifiable patient risk factors.[9] One factor in particular is obesity—in New Zealand, 32% of adults are now obese.[10] This figure is higher in patients undergoing THA and TKA and is expected to rise further in the coming years. Obesity has been linked in a number of studies to an increased risk of PJI.[11,12] The majority of these studies rely on national joint registry data, which underestimates the true incidence of PJIs. A study of the New Zealand Joint registry found a sensitivity of only 63% in capturing PJI.[13]

The New Zealand Surgical Site Infection Improvement Programme (NZSSIIP) is a Health Quality and Safety Commission initiative to capture all inpatient infections within 90 days of the primary operation. Its reporting is independent of the New Zealand joint registry, and has high accuracy in capturing infections following THA and TKAs. Using NZSSIIP data, this study aims to identify patient and surgical risk factors that are associated with PJI; especially whether obesity is a risk factor following THA and TKA.

Methods

Patients

The NZSSIIP began in March 2013, and monitors surgical site infections following orthopaedic procedures across all 20 district health boards in New Zealand.[14,15] It collects accurate data regarding infection within a 90-day follow-up period in THA and TKA patients.

All primary TKAs and THAs recorded by the NZSSIIP between July 2013 and September 2015 were analysed. Demographic details including age, gender, BMI, type of procedure, total risk score and ASA score were recorded. Four different BMI classifications were used in an attempt to determine which best stratified the differences in TKA outcomes: WHO classification, Dowsey, BMI greater or less than 30 and BMI greater or less than 35. Total risk score combines the patient's ASA score with a surgical wound score and duration of the operation and is an indicator of prognosis.[15] The following surgical details were also included in the study: surgical duration, prophylactic antibiotic type, prophylaxis timing, antibiotic dose and skin preparation type (Tables 1A and 2A).

Outcome

The NZSSIIP uses criteria from the Centre for Disease Control and Prevention (CDC) to group surgical site infections (SSIs) into superficial, deep and organ space.[16] The outcome measured in this study was a deep or organ space infection (defined according to the CDC) that developed within 90 days of the index procedure. Deep and organ space SSIs were grouped together, as both constitute PJI infection in the context of hip and knee arthroplasty. Infection was recorded by Infection Prevention and Control practitioners specifically trained by the NZSSIIP in identifying perioperative infection using a predefined checklist using CDC criteria.[15] Cases in which the diagnosis was not clear were reviewed by clinical microbiologists or infectious disease specialists. A 90-day cut off is used by the SSII programme, focusing on infections related to the initial surgical episode rather than late infection due to haematogenous spread.

Statistical analysis

The incidence of PJIs was calculated as a percentage for each of the patient and surgical factors. Association of these factors to deep/organ space infection was calculated as an odds ratio with 95% confidence intervals. A univariate analysis of p values, to determine whether associations were statistically significant, was conducted using a Pearson Chi Squared test. Variables with p values less than 0.05 were then initially forced into a multivariate model. Variables that were not found to be significant ($p>0.05$) in the multivariate model were removed and then a stepwise regression analysis of those remaining factors was performed.

Results

Data from 22,178 TKA and THA patients across all district health boards was recorded within the near two-year time frame. Of these, 20,171 of those were primary procedures and were included in this study, consisting of 10,690 primary THAs and 9,481 primary TKAs.

THAs

The overall PJI rate for THAs was 1.09%. The average age of patients was 70 years old. The average BMI was 30.7kg/m^2. Thirty-seven percent of THA patients were classified as obese (BMI>30kg/m^2) Compared to the general New Zealand population, which has an obesity rate of 32%, obese patients are over represented in people requiring THA.[10] On univariate analysis, BMI (all classifications), ASA and total risk score were associated with a statistically significant increase in risk of PJI following THA (Table 1A).

Using the WHO classification, which is the most widely used and accepted BMI classification, patients that were class III obese (BMI >40 kg/m^2) had an odds ratio of 3.73 (95% CI: 1.57–8.82, p=0.0035) when compared to 'normal weight' patients (BMI <25kg/m^2). Patients with a BMI greater than 35kg/m^2 were almost 2.5 times (OR: 2.33, 95% CI 1.41–3.81) more likely to develop PJI than patients with a BMI less than 35kg/m^2. Patients with a BMI greater than 30kg/m^2 were almost two times (OR: 1.83, 95% CI 1.15–2.91) more likely to develop PJI than patients with a BMI less than 30kg/m^2.

Table 1A: Univariate analysis of variables in relation to infection following THA.

Factor	Number of THAs	Deep/organ space infection (<90 days)		Percentage (%)	Odds ratio (with 95% CI)	P-value
		No	Yes			
Body mass index (WHO)						
Normal (<25)*	2,016	2,005	11	0.55	1.00	
Overweight (25-30)	3,398	3,378	20	0.59	1.08 (0.52–2.26)	0.0035
Obese I (30-35)	2,373	2,355	18	0.76	1.39 (0.66–2.96)	
Obese II (35-40)	1,152	1,138	14	1.22	2.24 (1.01–4.96)	
Obese III (>40)	499	489	10	2.00	3.73 (1.57–8.82)	
NR	1,252	1,243	9	0.72	1.32 (0.55–3.19)	
Body mass index (Dowsey)						
Normal* (<25)	2,016	2,005	11	0.55	1.00	
Overweight (25–30)	3,398	3,378	20	0.59	1.08 (0.52–2.26)	0.0036
Obese (30–40)	3,525	3,493	32	0.91	1.67 (0.84–3.32)	
Obese Morbid (>40)	499	489	10	2.00	3.73 (1.57–8.82)	
NR	1,252	1,243	9	0.72	1.32 (0.55–3.19)	
Body mass index (< or >35)						
<35*	7,787	7,738	49	0.63	1.00	
>35	1,651	1,627	24	1.45	2.33 (1.42–3.81)	0.0005
NR	1,252	1,243	9	0.72	1.14 (0.56–2.33)	
Body mass index (< or >30)						
<30*	5,414	5,383	31	0.57	1.00	
>30	4,024	3,982	42	1.04	1.83 (1.15–2.92)	0.0098
NR	1,252	1,243	9	0.72	1.26 (0.60–2.65)	
ASA						
1–2*	7,235	7,199	36	0.50	1.00	
3	3,067	3,036	31	1.01	2.04 (1.26–3.31)	0.0017
4–5	146	143	3	2.05	4.20 (1.28–13.9)	
NR	242	240	2	0.77	1.56 (0.37–6.50)	
Total risk score						
0*	6,559	6,521	38	0.58	1.00	
1	3,470	3,431	39	1.12	1.95 (1.25–3.06)	0.012
2	402	399	3	0.75	1.29 (0.40–4.20)	
NR	259	257	2			
Age						
<55yr	1,273	1,266	7	0.55	0.76 (0.33–1.76)	
55–64yr	3,352	3,319	33	0.98	1.37 (0.82–2.30)	0.40
65–74yr	2,224	2,208	16	0.72	1.00 (0.54–1.87)	
>75yr*	3,613	3,587	26	0.72	1.00	
NR	28	28	0	-	-	
Gender						
Female*	6,026	5,986	40	0.66	1.00	0.16
Male	4,659	4,617	42	0.90	1.36 (0.88–2.10)	
NR	5	5	0	-	-	
Surgical duration						
<40mn	121	121	0	0	0	
40–59mn*	1,405	1,394	11	0.78	1.00	
60–89mn	4,703	4,672	31	0.66	0.84 (0.42–1.7)	0.45
90–119mn	3,230	3,203	27	0.84	1.1 (0.53–2.2)	
120–180mn	1,102	1,091	11	1.0	1.3 (0.55–3.0)	
>180mn	98	96	2	2.1	2.7 (0.58–12)	
NR	31	31	0	0	0	

Table 1A: Univariate analysis of variables in relation to infection following THA (continued).

Prophylactic antibiotic type						
Cephazolin*	9,825	9,747	78	0.79	1.00	
Cefuroxime	635	632	3	0.47	0.59 (0.19–1.9)	NA
Flucoxacillin	2	2	0	0	0	
Gentamicin	63	62	1	1.6	2.0 (0.28–15)	
Vancomycon	34	34	0	0	0	
Other	79	79	0	0	0	
NR	52	52	0	0	0	
Prophylactic antibiotic dose						
<2g*	1,768	1,761	7	0.40	1.00	
2g ≤ x <3g	8,537	8,466	71	0.83	2.1 (1.0–4.6)	0.13
≥3g	278	275	3	1.1	2.7 (0.71–11)	
NR	107	106	1			
Prophylactic antibiotic timing						
0–60 minutes before incision*	10,118	10,041	77	0.76	1.00	
>1h before incision	106	105	1	0.94	1.24 (0.17–9.0)	0.087
After incision	117	114	3	2.5	3.3 (1.0–11)	
NR	349	348	1	0.29	0.37 (0.052–2.7)	
Skin preparation						
Chlorhexidine + alcohol*	8,372	8,305	67	0.58	1.00	
Povidone iodine + alcohol	2,079	2,067	12	0.80	0.72 (0.39–1.3)	0.47
Aqueous chlorhexidine	48	47	1	2.1	2.6 (0.36–19)	
Aqueous povidone iodine	76	75	1	1.3	1.7 (0.23–12)	
NR	115	114	1	0.87	1.1 (0.15–7.9)	

*Reference category for odds ratio determination.

Table 1B: Multivariate analysis of factors showing significant independent association to deep/organ space infection following THA.

Factor	Odds ratio (with 95% CI)	P-value
Body mass index (WHO) [stepwise]		
Normal (<25)*	1.00	
Overweight (25–30)	1.12 (0.52–2.43)	<0.0001
Obese I (30–35)	1.65 (0.75–3.62)	
Obese II (35–40)	3.03 (1.31–7.00)	
Obese III (>40)	5.62 (2.26–14.0)	
Age (stepwise)		
<55yr	0.35 (0.14–0.87)	
55–64yr	0.53 (0.28–1.01)	0.042
65–74yr	0.53 (0.30–0.95)	
>75yr*	1.00	
Gender (stepwise)		
Female*	1.00	0.030
Male	1.70 (1.05–2.74)	
ASA (forced model)		
1–2*	1.00	
3	1.33 (0.80–2.22)	0.424
4–5	1.99 (0.46–8.66)	

*Reference category for odds ratio determination.

Table 2A: Univariate analysis of variables in relation to infection following TKA.

Factor	Number of TKAs	Deep/organ space infection (<90 days)		Percentage (%)	Odds ratio (with 95% CI)	P-value
		No	Yes			
Body mass index (WHO)						
Normal (<25)*	983	978	5	0.51	1.00	
Overweight (25–30)	2,712	2,707	5	0.18	0.36 (0.10–1.26)	0.028
Obese I (30–35)	2,757	2,745	12	0.44	0.86 (0.30–2.45)	
Obese II (35–40)	1,570	1,558	12	0.76	1.51 (0.53–4.31)	
Obese III (>40)	923	915	8	0.87	1.72 (0.56–5.27)	
NR	536	534	2	0.37	0.74 (0.14–3.81)	
Body mass index (Dowsey)						
Normal* (<25)	983	978	5	0.51	1.00	
Overweight (25–30)	2,712	2,707	5	0.18	0.36 (0.10–1.26)	0.036
Obese (30–40)	4,327	4,303	24	0.55	1.10 (0.42–2.88)	
Obese morbid (>40)	923	915	8	0.87	1.72 (0.56–5.27)	
NR	536	534	2	0.37	0.74 (0.14–3.81)	
Body mass index (< or > 35)						
<35*	6,452	6,430	22	0.34	1.00	
>35	2,493	2,473	20	0.80	2.36 (1.28–4.34)	0.0042
NR	536	534	2	0.37	1.09 (0.26–4.67)	
Body mass index (< or > 30)						
<30*	3,695	3,685	10	0.27	1.00	
>30	5,250	5,218	32	0.61	2.26 (1.11–4.60)	0.021
NR	536	534	2	0.37	1.38 (0.30–6.32)	
ASA						
1–2*	6,592	6,569	23	0.35	1.00	
3	2,667	2,650	17	0.64	1.83 (0.98–3.44)	0.088
4–5	70	69	1	1.42	4.14 (0.55–31.1)	
NR	152	149	3	1.97		
Total risk score						
0*	5,959	5,942	17	0.29	1.00	
1	3,011	2,991	20	0.66	2.34 (1.22–4.47)	0.0041
2	337	333	4	1.19	4.20 (1.40–12.55)	
NR	174	171	3	1.72	6.13 (1.78–21.12)	
Age						
<55yr	609	606	3	0.49	0.99 (0.28–3.45)	
55–64yr	2,365	2,347	18	0.76	1.53 (0.76–3.09)	0.038
65–74yr	3,670	3,661	9	0.25	0.49 (0.21–1.14)	
>75yr*	2,812	2,798	14	0.50	1.00	
NR	25	25	0			
Gender						
Female*	5,098	5,085	13	0.26	1.00	
Male	4,379	4,348	31	0.71	2.79 (1.46–5.34)	0.0012
NR	4	4	0	-	-	
Duration						
<40mn	75	75	0	0	0	
40–59mn*	934	929	5	0.54	1.0	
60–89mn	4,295	4,282	13	0.30	0.56 (0.20–1.6)	0.12
90–119mn	3,077	3,061	16	0.52	0.97 (0.35–2.7)	
120–180mn	999	990	9	0.91	1.7 (0.60–5.1)	
>180mn	71	70	1	1.4	2.7 (0.31–23)	
NR	30	0	0	0	0	

Prophylactic antibiotic type						
Cephazolin	8,759	8,717	42	0.48	1.0	
Cefuroxime	504	504	0	0		NA
Flucoxacillin	1	1	0	0		
Gentamicin	39	38	1	2.6	5.5 (0.73–41)	
Vancomycon	29	29	0	0		
Other	94	94	0	0		
NR	55	55	0	0		
Prophylactic antibiotic dose						
<2g*	1,312	1,307	5	0.38	1.0	
2g ≤ x <3g	7,771	7,735	36	0.46	1.2 (0.48–3.1)	0.38
≥3g	306	303	3	0.98	2.6 (0.61–11)	
NR	92	92	0	0	0	
Prophylactic antibiotic timing						
0–60 minutes before incision*	8,959	8,918	41	0.46	1.0	
>1h before incision	131	129	2	1.5	3.4 (0.81–14)	0.17
After incision	82	82	0	0	0	
NR	309	308	1	0.32	0.71 (0.097–5.2)	
Skin preparation						
Chlorhexidine + alcohol*	7,465	7,427	38	0.51	1.0	
Povidone iodine + alcohol	1,832	1,826	6	0.33	0.64 (0.27–1.5)	0.68
Aqueous chlorhexidine	31	31	0	0	0	
Aqueous povidone iodine	72	72	0	0	0	
NR	81	81	0	0	0	

*Reference category for odds ratio determination.

Patients with an ASA score of 3 had an odds ratio of 2.04 (95% CI: 1.27–3.31); scores of 4–5 had an odds ratio of 4.20 (95% CI: 1.28–13.8); p=0.006, when compared to patients with ASA score 1–2. Male gender was associated with an increase in risk of PJI (OR: 1.36, 95% CI 0.88–2.10) while age less than 55 had a slightly lower risk (OR: 0.76, 95% CI 0.33–1.76).

The variables BMI, ASA score, age and gender were analysed using a multivariate model. BMI, age and gender all showed significance. In a stepwise multivariate analysis with ASA removed, BMI continued to show a strong association with PJI. 'Obese III' patients had an odds ratio of 5.62 (95% CI: 2.25–14.0) compared to 'normal weight' patients with a p-value less than 0.0001. After adjusting for BMI and age, men were 1.7 (1.05–2.74) times more likely to develop PJI than women. Patients younger than 75 were found to have approximately half the risk of PJI for patients older than 75 (Table 1B).

TKAs

The overall PJI rate was 0.46%. The average age of patients was 70 and the average BMI was 30.7kg/m^2. Fifty-five percent of TKA patients were classified as obese. On univariate analysis BMI, total risk score, age and gender were associated with a statistically significant increase in risk of PJI following TKA (Table 2A).

Using the WHO classification, patients that were class III obese (BMI >40kg/m^2) had an odds ratio of 1.72 (95% CI: 0.56–5.27, p=0.028) when compared to 'normal weight' patients (BMI <25 kg/m^2). In absolute terms, 8 out of 923 (0.87%) class III obesity patients developed PJI compared to 5 out of 983 (0.51%) 'normal weight' patients. Patients with a BMI greater than 35kg/m^2 were over twice as likely (OR: 2.36 with 95% CI 1.28–4.34) to develop PJI than patients with a BMI less than 35kg/m^2. The results were similar using the greater or less than 30kg/m^2 stratification (OR: 2.26, 95% CI 1.11–4.60).

Table 2B: Multivariate analysis of factors showing significant independent association to deep/organ space infection following TKA.

Factor	Odds ratio (with 95% CI)	P-value
Body mass index (WHO)		
Normal (<25)*	1.00	
Overweight (25–30)	0.31 (0.09–1.09)	0.015
Obese I (30–35)	0.73 (0.25–2.12)	
Obese II (35–40)	1.44 (0.50–4.16)	
Obese III (>40)	1.94 (0.63–5.97)	
Gender		
Female*	1.00	0.002
Male	2.96(1.51–5.80)	
ASA (forced model)		
1–2*	1.00	
3	1.54 (0.78–3.02)	0.268
4–5	3.64 (0.47–28.4)	
Age (forced model)		
<55yr	0.54 (0.12–2.54)	
55–64yr	1.13 (0.50–2.56)	0.138
65–74yr	0.45 (0.19–1.10)	
>75yr*	1.00	

*Reference category for odds ratio determination.

Males had an almost three-fold (OR = 2.79, 95% CI: 1.46–5.34, p-value = 0.0012) greater risk of PJI than females. Age was also shown to be a significant factor. Fifty-five to 64 year-olds had an OR of 1.53 (95% CI: 0.76–3.09) when compared with patients older than 75. Patients with ASA scores 4–5 were greater than four times more likely to develop PJI than patients with scores 1–2.

Surgical duration, antibiotic dose, type, timing and skin preparation did not provide statistically significant results in the univariate analysis and were, therefore, not included in the multivariate analysis.

BMI, ASA score, age and gender were analysed using a multivariate model (Table 2B). Using this model, BMI and gender remained statistically significant but ASA and age did not. In a stepwise multivariate analysis with ASA and age removed, BMI showed a strong association with PJI.

'Obese III' patients had an odds ratio of 1.94 (95% CI: 0.63–5.70, p=0.015) compared to 'normal weight' patients. Patients who were classed as overweight and obese I showed a reduced risk of developing PJI with odds ratios of 0.31 and 0.73 respectively. After adjusting for BMI, men were 2.96 (1.51–5.80, p=0.002) times more likely to develop PJI than women.

Discussion

Infection is a major cause of failure in TKA and THA, accounting for up to 24% of early (within 24 months) failures.[17] There is strong international evidence that accurate data to inform and monitor efforts to reduce PJI is beneficial. A Norwegian group reported a 57% reduction in SSIs over an 11-year period after the implementation of a surveillance system.[18] The French ISO-RAISIN system (Infection du Site Opératoire—Réseau Alerte Investigation Surveillance des Infections)

reported a 36% reduction in hip SSI over seven years.[19] An Australian study of the Victorian Healthcare Associated Infection Surveillance System (VICNISS) reported that for every year of participation in the surveillance system there is a 9% decrease in SSI risk.[20] Hence the NZSSIIP will be important going forward.

Reducing PJI following arthroplasty first requires an accurate understanding of the factors important in its incidence. This study, using data from the NZSSIIP, found patient factors including an elevated BMI and gender to be strongly associated with early infection following both TKA and THA. The use of data from a targeted national infection surveillance program, rather than a national joint registry, is relevant as PJI rates are underreported by registries both in New Zealand and overseas.[13,21,22] Zhu et al found the New Zealand Joint Registry (NZJR) has a sensitivity of only 63% for reporting PJI when compared to an audit of hospital records.[13] The NZSSIIP supports more vigilant surveillance and therefore reduces the likelihood of both missed reports and missed diagnoses. In this study, the overall 90-day infection rate for TKAs was 0.46%, compared to NZJR data revision rates for PJI of TKAs of 0.16% at six months and 0.28% (at 12 months).[23] This highlights the benefit of a targeted surveillance programme, as true PJI rates can be recorded and the effectiveness of any measures to reduce PJI rates can be monitored.

We found an increased risk of PJI in males for both THAs (OR=1.70) and TKAs (OR=2.96). This association has been previously reported in a number of large registry studies.[23–26] Namba et al found that males had a 1.89 higher risk of PJI than females following TKA.[24] Tayton et al similarly reported a 1.78 times higher risk in males.[23] However, the reason for this association is unclear. There may be a number of potential confounding variables, such as smoking and diabetes, that may contribute. However, male gender may also be an independent risk for the development of PJI and the strength of the association seen in this study would support this hypothesis.

There is debate whether age is an independent risk factor for PJI.[12,23–26] Namba et al studied a population with an average age of 67.4 years, which did not show an association with age for PJI. This contrasts with a study based on data from the Korean Nosocomial Infection Surveillance System (KONIS), which showed an odds ratio of 1.75 and 1.64 for age 60–69 for THAs and TKAS respectively.[27] In our study, the risk of PJI in patients with age greater than 70 was almost two times higher in THAs, but no association with age was found in TKAs. The average age of patients undergoing arthroplasty procedures is around 70 years old. This is an important factor to consider as, if indeed the rate of PJI is higher in older patients, this will represent a larger proportion of the arthroplasty population.

We found BMI was strongly associated with PJI, consistent with findings from previous studies. Wagner et al used joint registry data from the Mayo Clinic to evaluate BMI as a risk factor for PJI. In THAs (study of 21,361 patients) there was a 9% increase in risk per unit of BMI above a threshold of 25kg/m^2.[28] In TKAs (study of 22,289), there was a 7% increase in risk per unit of BMI above a threshold of 35kg/m^2.[29] There are a number of potential reasons for this association. Obesity is associated with poor penetration of prophylactic antibiotics and an impaired immune response to infection, which may increase the likelihood of bacterial contamination during surgery progressing to clinical infection.[30]

While we found a graduated effect-response relationship between PJI risk and increasing BMI across all the classifications used, we were unable to define a clear BMI threshold at which the risk of PJI increased to unacceptable levels. There is some debate whether patients above a certain BMI threshold should be refused arthroplasty.[31] Patients with a high BMI are known to benefit from THA and TKA. Lash et al compared the change in Oxford functional scores for BMIs less than 30kg/m^2, between 31 and 35kg/m^2 and greater than 35kg/m^2. All three categories showed a similar absolute change in functional scores for both THAs and TKAs.[32] Furthermore, in this study while the odds ratio for PJI in THA patients with BMI >40kg/m^2 was 5.6, the absolute risk difference was only 1.45% comparing those two BMI groups. Similarly, the odds ratio for TKA patients was 1.9, but again the absolute risk difference was 0.36%. Therefore, for every 100 THA patients with

BMI greater than 40 kg/m^2, 1.5 more people will develop PJI compared to the equivalent number of normal BMI patients, and just 0.4 more for TKA based on this data set. Should an infection threshold be clearly defined, a weight reduction intervention, even bariatric surgery, before TKA or THA may have a role although data is mixed.[33] Werner et al reported that bariatric surgery prior to TKA resulted in reduced post-operative complications.[34] However, Inacio et al reported no significant association with prior bariatric surgery and improved surgical outcomes.[35]

The overall PJI rate in THAs was 1.06% compared to 0.46% in TKAs, a significant difference. This could be associated with the prevalence of abdominal obesity, which affects the hips more than the knees. While abdominal obesity is important in abdominal surgery, there are no studies that have specifically investigated the impact of abdominal obesity on orthopaedic surgery.[36]

There are a number of limitations to this study. Firstly, The NZSSIIP does not record data on some factors that may have a correlation to increasing rates of PJI, in particular patient smoking and diabetes. The use of a multivariate analysis aims to limit confounding, but without data on these factors this was not possible. However, our findings are consistent with previous registry-based studies where data on these factors was available, and BMI appears to be an independent risk factor in PJI causation.[9,11,12,23–30,33,34] Also, information is only collected up to 90 days, which will mean that a small number of infections due to intra-operative contamination will not be collected in the data set.

This study has shown that male gender and BMI are key risk factors in the development of early periprosthetic joint infection following THA and TKA. PJI has significant impacts on patient mortality and morbidity and also resource expenditure by the public healthcare system. The NZSSIIP will be important in the future by ensuring that quality and safety markers are met and allowing the accurate reporting of the SSI rate following interventions aimed at its reduction.

Competing interests:
Dr Morris is the Clinical Lead for the NZ Surgical Site Infection Improvement Programme.

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