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ORIGINAL ARTICLE

Investigation of preoperative asymptomatic bacteriuria as a risk factor for postvertebroplasty infection

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Abstract

Background: Postvertebroplasty infection (PVI) is a catastrophic complication after vertebroplasty (VP). Although the urinary tract has been considered as a source of infectious pathogens, whether asymptomatic bacteriuria (ASB) is a risk factors for PVI remains unknown.

Methods: This retrospective study included 716 patients (207 males; 509 females) treated with VP for osteoporotic vertebral fractures in a single medical center between May 2015 and December 2019. Clinical symptoms, urinalysis results, and culture data were collected preoperatively to identify patients with ASB. The primary outcome was PVI at the index level during follow-up. Demographic data and laboratory test results were compared between the PVI and non-PVI groups.

Results: The mean age of the cohort was 78.6±9.6 (range, 63–106). The prevalence of ASB was 14.1%, with female predominance (63.4%). The overall PVI rate was 1.26% (9/716). The PVI group had more patients with ASB (4/9, 44.4%) than did the non-PVI group (97/707, 13.7%) (p = 0.027). The rate of ASB treatment was similar between the PVI and non-PVI groups (25% vs. 23.7%, respectively). No case of PVI was caused by the urine culture pathogen. Multivariate analysis identified the following risk factors for PVI: ASB (odds ratio [OR], 5.61; 95% CI, 1.14–27.66; p = 0.034), smoking (OR, 16.26; 95% CI, 2.58–102.65; p = 0.003), and malignancy (OR 7.27; 95% CI, 1.31–40.31; p = 0.023).

Conclusion: ASB was not uncommon among patients admitted for VP and should be considered a marker of relatively poor host immunity. Preoperative ASB, a history of malignancy, and smoking were identified as significant risk factors for PVI.

Keywords: Asymptomatic bacteriuria; Postvertebroplasty infection; Risk factor; Vertebral fracture; Urinary tract infection

1. INTRODUCTION

Vertebroplasty (VP) has gained in popularity as an effective treatment for painful osteoporotic vertebral fracture since its introduction by Galibert in 1987.[1] Although the procedure is generally safe and effective, catastrophic complications such as post-VP infection (PVI) occur at a rate of 0.32% to 0.46%.[2–4] Surgical management of PVI often requires an anterior approach to remove the cement, anterior fusion, and posterior instrumentation. This aggressive procedure is not well tolerated in aged and frail patients. The mortality rate of PVI is 10% to 33%.[2,3] With less than 60 cases reported to date, the potential risk factors for PVI remain elusive.[2]

In primary vertebral osteomyelitis, hematogenous seeding from distant sites via the arterial route is the predominant pathway for pathogen infection of the vertebral column. Sites of infection that result in hematogenous spread include the respiratory tract, gastrointestinal tract, skin and soft tissue, and, most often, urinary tract infection (UTI).[5] Thus, VP is contraindicated in patients with active infection.[6] Asymptomatic bacteriuria (ASB), a condition that does not require treatment in most cases,[7] is another potential concern when considering VP. While ASB is found to be a risk factor for prosthetic joint infection,[8–10] the relationship between ASB and PVI is unknown.

This study aims to investigate the incidence of ASB among patients who underwent VP to treat painful osteoporotic vertebral fractures, identify the risk factors for PVI, and determine whether ASB is a risk factor for PVI.

2. METHODS

2.1. Study design and population

This study was approved by the institutional review board of our hospital (2020-02-013AC). A prospectively compiled database of patients who were admitted as VP candidates for the treatment of painful osteoporotic vertebral fractures between May 2015 and December 2019 was retrospectively analyzed to select the study cohort. Patients were excluded from the study

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CA9_V86N2_Text.indb 233 13-Jan-23 14:17:11

if they had a follow-up of less than 12 months, an indwelling urinary catheter, previous VP, received VP for malignant metastasized vertebral fractures or multiple myeloma, or if VP was not performed. Patients who had a history malignancy and suffered from fragility-related vertebral fracture were not excluded. The patients' demographic data, clinical characteristics, laboratory data, imaging findings, and biopsy results were collected from the database and medical records. The primary outcome was PVI at index level during follow-up.

2.2. Treatment protocol

All patients receiving conservative treatment for at least 6 weeks and still experiencing refractory back pain were considered as candidates for VP at our institute. The diagnosis of primary vertebral fracture was based on the following criteria: (1) magnetic resonance imaging (MRI) showing vertebral marrow edema in a T2-weighted image or STIR views; (2) air accumulation, vacuum phenomenon, or pseudarthrosis observed in the CT or MRI scan.

If CT or MRI scans were still ambiguous, a CT-guided biopsy was performed by radiologists to exclude the possibility of primary vertebral osteomyelitis.[11] Biopsy samples were sent for culture of aerobic and anaerobic bacteria, tuberculosis, fungi, and permanent pathology sections. The diagnosis of vertebral osteomyelitis was always taken into careful consideration in case of any clinically suspicious observations before the VP procedure.

Urinalysis and urine culture were performed for all patients 1 week before admission. The urine was cultured using conventional methods in the microbiology laboratory, and all isolated microorganisms were identified using standard biochemical protocols. Urinalysis samples with more than 8 squamous epithelial cells per low-power field were excluded.[12] A positive culture was defined as more than 1 bacterial species isolated in a quantitative count $\geq 10^5$ colony-forming units/mL in a voided urine specimen. On admission, the patients were evaluated by the treating physicians for the symptoms of UTI, including urinary frequency, dysuria, gross hematuria, and suprapubic discomfort. Patients with a positive culture and symptoms of UTI were treated with antibiotics, and the VP was canceled. ASB was defined as a positive culture without symptoms. Whether to treat a patient with ASB with preoperative antibiotics was determined by the treating physician. Patients who were treated for ASB were given a 7-day course of oral empirical antibiotics according to in vitro susceptibility, starting from the date of admission. Due to the retrospective nature of this study, the ASB treatment protocol was neither mandatory nor consistent among the study cohort.

VP was performed under local anesthesia by specialty-trained spine surgeons. Preoperative prophylactic parenteral antibiotics were not routinely administered. Through a 2–3 mm incision, a Jamshidi needle was inserted percutaneously into the pedicle under C-arm imaging guidance, through which polymethylmethacrylate cement was inserted.[13,14] The patient was discharged on the same day or the next day if no acute complications or discomfort occurred postoperatively. Postoperative bracing was prescribed for 3 months. All patients received outpatient clinic follow-up at 1 week, 4 weeks, 8 weeks, 12 weeks, 6 months, and 12 months after surgery. A plain radiograph of the spine was taken at each follow-up.

Of the 758 patients who were admitted for VP in our hospital during the study period, 42 did not meet the inclusion criteria (15 lost to follow-up, 12 for a history of previous VP, 7 for malignant metastasized vertebral fractures, 4 for multiple myeloma, and 4 who did not undergo VP due to active UTI), leaving 716 patients in the final cohort (Fig. 1). In the total cohort, 509 patients were female (71.1%), and the mean age was 78.6 ± 9.6 years (range, 63–106). No patient had an indwelling urinary catheter when admitted for VP (Table 1).

2.3. Diagnosis of post-VP infection

Post-VP infection was diagnosed based on either of the following assessments: (1) pathology: CT-guided vertebral biopsy

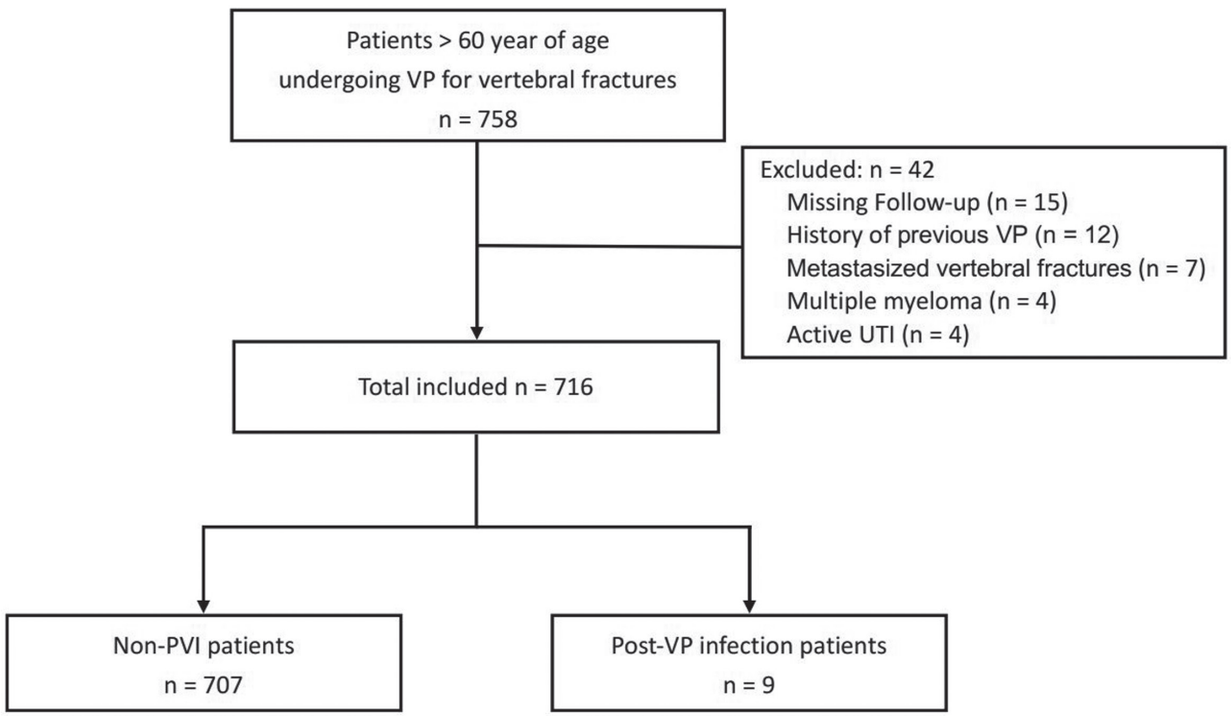

Fig. 1 Number of patients enrolled, excluded, and included in data analysis.

CA9_V86N2_Text.indb 234 13-Jan-23 14:17:12

at the index level revealing osteomyelitis with infiltration of neutrophils or lymphocytes, or granulomatous change in TB cases[15,16]; (2) microbiology: biopsy culture yielding growth of bacteria or tubercle bacillus; (3) a combination of clinical findings, laboratory data, and imaging: refractory back pain or fever after the procedure, elevated CRP (normal range, <0.5 mg/dL), and a change in MRI signal in the involved vertebral body at index level with postcontrast enhancement of bone marrow or abscess margins.[16,17] Patients were divided accordingly into two groups: PVI and non-PVI groups.

2.4. Statistical analysis

All data analysis and calculations were performed using IBM SPSS Statistics for Windows, version 25.0 (IBM, Armonk, NY). Data were compared between patients with and without PVI. Independent-sample t-tests were used to compare continuous variables, while chi-squared or Fisher's exact tests were used to compare categorical variables. To identify risk factors for infection after VP, the variables that differed significantly between groups were initially evaluated using univariate logistic regression analysis. Those significantly associated with PVI at $p \leq 0.10$ in univariate analysis were entered into the multivariate logistic regression model. The statistical power of each significant variable was analyzed. Potential modifier effects of the variables were also studied. The results are expressed as odds ratios (ORs) with 95% confidence intervals (CIs). A 2-tailed p value <0.05 was considered statistically significant.

3. RESULTS

Of the 716 patients in the final cohort, nine (1.26%) were diagnosed with PVI occurring at a mean time of 4.1 ± 2.3 weeks (range, 2–9 weeks) after the index VP. Seven patients were diagnosed based on pathological and microbiological findings, and 2 were diagnosed based on typical clinical presentation and image features (Table 1). Patients in the PVI group were older (p = 0.03), had a higher incidence of malignancy (p = 0.01), and had a history of smoking (p = 0.01). No significant difference was observed in any of the other demographic characteristics or clinical features. Additional information is shown in Table 2.

Comparison of the laboratory test results between patients in the PVI and non-PVI groups was shown in Table 3. No significant difference in the preoperative CRP levels or the proportion of patients with abnormal CRP level was observed between the two groups. Preoperative urinalysis showed that abnormal urinalysis was present in 55.6% (5/9) of the PVI patients and 30.5% (251/707) of the non-PVI patients (p = 0.21). While squamous epithelial cells were found in 596 of the 716 urine samples (83%), none of the samples presented more than 8 cells per low-power field. Culture of the urine specimens yielded positive growth and met the diagnosis of ASB in 101 patients (14.1%), with 4 patients in the PVI group (44.4%; 4/9) and 97 patients in the non-PVI group (13.7%; 97/707). The fraction of patients with ASB was significantly higher in the PVI group than in the non-PVI group (p = 0.03). The types of microorganisms isolated from urine in each group are compared in Table 4.

Detailed demographic data for patients in the PVI group are shown in Table 1. The microorganisms found in urine differed from those cultured from the infected vertebrae, indicating that no case of PVI was caused by a pathogen present in the preoperative urine. Of the 4 patients with preoperative ASB in the PVI group, only one was treated before VP. The fraction of patients receiving treatment for ASB did not differ significantly between the PVI and non-PVI groups (25% vs. 23.7%, respectively).

Univariate logistic regression analysis identified multiple risk factors for PVI, including smoking, hypertension, a history of

Table 1

Patient demographics of the PVI group

Patient number	Age/sex	Comorbidities	BMI (kg/m²)	Smoking	Level	Urine culture	Treatment for ASB	Causative organism of PVI	Pathology from biopsy	Time to PVI (weeks)
1	85/M	HTN	22.3	No	T12	No growth	–	No biopsy	No biopsy	6
2	75/F	DM, HTN	24.4	No	L2, L5	Escherichia coli	No	No growth	Osteomyelitis	4
3	84/M	DM, HTN, HCC, gout	27.4	Yes	T12	Proteus mirabilis	No	No biopsy	No biopsy	2
4	79/F	HTN, Parkinsonism	26.2	No	T8, T12	Escherichia coli	No	Staphylococcus aureus	Chronic necrosis	2
5	85/F	HTN, colon cancer	27.7	No	L1	No growth	–	No growth	Chronic abscess	3
6	87/M	HTN	32.1	Yes	L1	No growth	–	Proteus mirabilis	Osteomyelitis	5
7	88/M	HTN, RCC	33.3	Yes	L1	Enterobacter cloacae	Yes	Cutibacterium acnes	Osteomyelitis	2
8	78/F	HTN	19.9	No	T10	No growth	–	No growth	Osteomyelitis	4
9	82/F	HTN, tuberculosis	19.9	No	T12, L1	No growth	–	No growth	Osteomyelitis	9

BMI = body mass index; DM = diabetes mellitus; HCC = hepatic cell carcinoma; HTN = hypertension; PVI = post-VP infection; RCC = renal cell carcinoma.

CA9_V86N2_Text.indb 235 13-Jan-23 14:17:12

Table 2

Patient demographic data

	Overall (n = 716)	PVI group (n = 9)	Non-PVI group (n = 707)	*p*
Sex (n [%])				0.30
Male	207 (28.9%)	4 (44.4%)	203 (28.7%)	
Female	509 (72.1%)	5 (55.6%)	504 (71.3%)	
Age (years)	78.6 ± 9.6	82.6 ± 4.4	78.5 ± 9.7	0.03*
BMI (kg/m^2)	23.59 ± 4.11 (14–35.8)	25.91 ± 4.82 (19.9–33.3)	23.56 ± 4.1 (14–35.8)	0.09
Smoking (n [%])	41 (5.7%)	3 (33.3%)	38 (5.4%)	0.01*
Medical history (n [%])				
Hypertension	401 (56.0%)	8 (88.9%)	393 (55.6%)	0.046*
Diabetes mellitus	152 (21.2%)	2 (22.2%)	150 (21.2%)	1.00
Malignancy	37 (5.2%)	3 (33.3%)	34 (4.8%)	0.01*
ESRD	20 (2.8%)	0 (0%)	20 (2.8%)	1.00
Involved segment (n)	820	12	808	1.00
Thoracic spine (n [%])	338 (41.2%)	5 (41.6%)	333 (41.2%)	1.00
T6	4	0	4	
T7	11	0	11	
T8	20	1	19	
T9	19	0	19	
T10	26	1	25	
T11	58	0	58	
T12	200	3	197	
Lumbar spine (n [%])	482 (58.8%)	7 (58.3%)	475 (58.8%)	
L1	230	4	226	
L2	105	2	103	
L3	85	0	85	
L4	47	0	47	
L5	15	1	14	
Number of VP segment (n [%])				
Single segment	620 (86.6%)	6 (33.3%)	614 (86.8%)	0.11
Multiple segments	96 (13.4%)	3 (66.7%)	93 (13.2%)	0.14
Time from injury to VP (weeks)	9.8 ± 0.4 (1–78)	9.8 ± 8.1 (1–26)	9.9 ± 11.3 (1–78)	0.99
Length of hospitalization (days)	4.2 ± 0.3 (1–45)	8.2 ± 14.3 (2–45)	4.4 ± 7.4 (1–18)	0.45
Follow-up (months)	20.1 ± 9.4 (0–40)	16.9 ± 9.4 (0–34)	20.3 ± 9.3 (6–40)	0.28

Data are presented as the mean ± standard deviation (range) if not otherwise specified.

*Statistically significant.

BMI = body mass index; ESRD = end-stage renal disease; PVI = post-VP infection.

Table 3

Preoperative lab test results

Characteristic	Overall (n = 716)	PVI group (n = 9)	Non-PVI group (n = 707)	*p*
CRP on admission (mg/L)	0.89 ± 2.12	3.87 ± 9.47	0.88 ± 1.87	0.40
>0.5 mg/L (n [%])	229 (32.0%)	3 (33.3%)	226 (32.0%)	1.00
>1.0 mg/L (n [%])	129 (18.0%)	2 (22.2%)	127 (18.0%)	0.62
Abnormal Urinalysis (n [%])	256 (35.7%)	5 (55.6%)	251 (35.5%)	0.21
Nitrite positive	109 (15.2%)	3 (33.3%)	106 (15.0%)	0.14
Leukoesterase positive	191 (26.7%)	3 (33.3%)	188 (26.6%)	0.71
Pyuria	243 (33.9%)	4 (44.4%)	239 (33.8%)	0.50
Bacteriuria	232 (32.4%)	3 (33.3%)	229 (31.1%)	1.00
ASB (n [%])	101 (14.1%)	4 (44.4%)	97 (13.7%)	0.03*
Treated ASB	24 (23.8%)	1 (25.0%)	23 (23.7%)	1.00
Untreated ASB	77 (76.2%)	3 (75.0%)	74 (76.3%)	1.00

Data are presented as the mean ± standard deviation if not otherwise specified.

*Statistically significant.

ASB = asymptomatic bacteriuria; CRP = C-reactive protein; PVI = post-VP infection.

malignancy, and ASB. Multivariable logistic regression analysis revealed that independent risk factors for PVI include smoking (OR, 16.26; 95% CI, 2.58–102.65; $p < 0.01$), a history of malignancy (OR, 7.27; 95% CI, 1.31–40.31; $p = 0.02$), and ASB (OR, 5.61; 95% CI, 1.14–27.66; $p = 0.03$). A post hoc statistical power calculation using the group size (n= 716), the incidence of the PVI (1.26%), and an alpha value of 0.05, all ORs of the four significant factor in univariate logistic regression yields a power of 100%. However, accounting for risk variables using multivariable logistic regression, hypertension was not an independent predictor of the occurrence of PVI (Table 5).

CA9_V86N2_Text.indb 236 13-Jan-23 14:17:12

Table 4

Microorganisms isolated from preoperative urine cultures

Isolated species	PVI group (n = 4)	Non-PVI group (n = 97)	*p*
Gram positive	**1 (25.0%)**	**18 (18.6%)**	**0.57**
Staphylococcus aureus		4 (4.1%)	
Enterococcus spp.	1 (25.0%)	14 (14.4%)	
Gram negative	**3 (75.0%)**	**73 (75.3%)**	**1.00**
Klebsiella pneumoniae	1 (25.0%)	12 (12.4%)	
Escherichia coli	2 (50.0%)	42 (43.3%)	
Morganella morganii		3 (3.1%)	
Pseudomonas aeruginosa		4 (4.1%)	
Proteus mirabilis		6 (6.2%)	
Citrobacter koseri		3 (3.1%)	
Gardnerella vaginalis		3 (3.1%)	
Fungi		**6 (6.2%)**	**1.00**
Candida albicans		6 (6.2%)	

Data are presented as number (%).

Table 5

Univariate and multivariate logistic regression analysis of PVI data

	Univariate logistic regression		Multivariate logistic regression	
Variables	**OR (95% CI)**	***p***	**OR (95% CI)**	***p***
Age	1.05 (0.97 to 1.14)	0.21		
Smoking	8.79 (2.12 to 36.51)	<0.01*	16.26 (2.58–102.65)	<0.01*
Hypertension	6.39 (0.80 to 51.38)	0.08	8.28 (0.95–72.32)	0.06
History of malignancy	9.06 (2.18 to 37.79)	<0.01*	7.27 (1.31–40.31)	0.02*
Asymptomatic bacteriuria	5.03 (1.33 to 19.06)	0.02*	5.61 (1.14–27.66)	0.03*

*Statistically significant.

CI = confidence interval; OR = odds ratio.

4. DISCUSSION

The overall prevalence of ASB in our patient cohort was 14.1% in patients who underwent VP. We found that 44.4% of the patients with PVI had preoperative ASB. Further analysis revealed that the likelihood of developing PVI was 5.61 times higher in patients with ASB than in those without. No case of PVI was caused by pathogens present in the pre-VP urine culture. We observed that smoking habit and a history of malignancy were also risk factors for PVI.

We observed a PVI rate of 1.26% (9/716), which is higher than the rates of 0.32% to 0.46% reported in previous studies.[2–4] This difference in findings possibly resulted from our determination of PVI based on not only pathology and microbiological data but also a clinical diagnosis supported by MRI features. No standard diagnostic criteria are reported in the literature.[2,3,18] Previous studies have described cases with radiographic characteristics typical of PVI but failed to identify the causative organisms or acquire positive pathology results.[4,19] Among previous studies, the rate of identification of organisms causing PVI is 50% to 78%.[2–4] In our study, two of the nine patients were diagnosed based on typical radiologic findings and clinical presentation (cases 1 and 3 in Table 4).

No clear risk factors for PVI have been identified in previous studies, likely due in part to its very low incidence. Previously identified risk factors for complications and mortality following VP include American Society of Anesthesiologists physical status class 4, history of COPD, kidney disease, or disseminated cancer.[20,21] These results are consistent with our finding that the incidence of malignancies was higher among patients with PVI than without. This finding likely reflects the greater comorbidity burdens and weakened immunity resulting from cancer or related therapy. Smoking is a known risk factor for surgical site infection after spinal surgery,[22] a finding consistent with our observations. Studies have reported that several compounds present in cigarette smoking have immunosuppressive effects that include impairment of innate defenses against pathogens, modulation of antigen presentation, and promotion of inflammatory processes.[23,24]

The preoperative management of ASB in specific orthopedic procedures has received much attention.[8–10] The prevalence of ASB increases with age, occurring in about 20% of women and 10% of men over 80 years of age.[25] The rate in long-term care facilities is even higher, ranging from 25% to 50% in women and 15% to 35% in men.[26] In our study cohort, the prevalence of ASB was 14.1%, and patients with ASB were 5.61 times more likely to have PVI than were those without. Several studies report coincident UTI at the time of VP followed by a subsequent surgical infection.[27–29] Abdelrahman et al[3] reported that of the six cases of VP infection investigated in their study, five presented with UTI as a comorbidity. Since the method used to diagnose UTI was not clearly defined in these studies, we assume that these patients might have had ASB. The symptoms of UTI can be clinically subtle and difficult to differentiate by physicians. Nevertheless, our study shows that a positive preoperative urinary culture, even without symptoms, is a risk factor for PVI.

Possible explanations for the increased risk of PVI in patients with ASB include direct contamination, hematogenous spread, and ASB serving as a marker of poor immunity. Wound contamination and hematogenous spread were highly suspected in our series, since the majority of infections occurred within 6 weeks after surgery (8 of 9). However, in the two patients with positive urine and surgical site cultures, the bacterial species responsible for the VP and urine infections differed. Previous studies

CA9_V86N2_Text.indb 237 13-Jan-23 14:17:12

of coincident UTI at the time of VP followed by infection did not report the species identified in urine cultures.[3,27–29] Studies on ASB before arthroplasty have also noted differences in culture results between urine and periprosthetic infections.[8,9] Thus, ASB is likely a host marker that indicates a poorer host immune response to pathogens in vivo, leading to a higher bioburden at the time of surgery. A recent study found that patients with ASB may have specific gene deletions that disrupt innate immune activation, increasing the susceptibility to infection by decreasing the efficiency of bacterial clearance.[30]

According to the literature, ASB treatment has been proven beneficial only for urological procedures and during pregnancy.[7,31] Studies investigating the necessity of treating ASB before total joint arthroplasty have found no decrease in the risk of infection with such treatment.[9,32] In our study cohort, the fraction of patients receiving preoperative treatment for ASB was similar in the PVI and non-PVI groups (25% vs. 23.7%). However, the preoperative treatment protocol for ASB was neither mandatory nor randomized. Further study is required to determine whether preoperative antibiotic treatment is beneficial for preventing PVI.

To date, little evidence supports the use of prophylactic antibiotics before vertebral augmentation procedures. While such treatment was not used in this study, others have suggested the routine use of prophylactic antibiotics given the serious morbidities associated with PVI.[33] In a series of 1150 kyphoplasty patients treated with prophylactic antibiotics, no case of infection was found.[34] However, prophylactic antibiotics may have side effects such as rashes, hypotension, intramembranous colitis, and Stevens-Johnson syndrome.[35] The findings of this study suggest that the candidates for antibiotics prophylaxis can be reasonably narrowed down to those with the risk factors of smoking, history of malignancy, and ASB.

This study has several limitations. First, the study was conducted retrospectively, resulting in selection bias for the defined cohorts and incomplete biopsy results and insufficient data regarding possible confounding risk factors for ASB. Second, the diagnosis of ASB was based on a single urine sample, a practice that is not fully in accordance with the diagnosis criteria proposed by the Infectious Diseases Society of America.[7] These guidelines recommend two consecutive specimens containing the same bacterial strain to diagnose ASB in women. Third, the treatment of ASB before VP was not mandatory nor randomized. Physicians may have decided to treat patients with abnormal urinalysis based on individual factors. A prospective, randomized study in a larger cohort is needed to determine whether treating ASB before VP should be recommended. However, this is the first study in search for risk factors associated with PVI. The findings may provide valuable information in treating patients who are planning to undergo index VP.

In conclusion, ASB was not uncommon among patients admitted for VP and should be considered as a marker of relatively poor host immunity. Preoperative ASB, a history of malignancy, and smoking were identified as significant risk factors associated with PVI. Although it remains unclear whether treating ASB preoperatively is necessary to prevent PVI, these risk factors may help identify candidates for preoperative antibiotic prophylaxis.

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CA9_V86N2_Text.indb 238 13-Jan-23 14:17:12

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CA9_V86N2_Text.indb 239 13-Jan-23 14:17:12