Contents lists available at ScienceDirect



Journal of Orthopaedics, Trauma and Rehabilitation

Journal homepages: www.e-jotr.com & www.ejotr.org

Original Article

Spinal Implants Can Be Retained in Patients with Deep Spine Infection: A Cohort Study 深層脊椎感染的患者可以保留脊柱植入物 - 一項隊列研究





Hey Hwee W.D. ^{a, *}, Ng Li W.N. ^a, Kumar Nishant ^a, Lau Tze-Chun E. ^a, Thambiah Joseph ^a, Kumar Naresh ^a, Lau Leok-Lim ^a, Liu Ka-Po G. ^a, Vasudevan Anupama ^b, Fisher Dale ^b, Wong Hee-Kit ^a, Tambyah Paul A. ^b

^a University Orthopaedics, Hand and Reconstructive Microsurgery Cluster, National University Health System, Singapore ^b Department of Medicine, National University Health System, Singapore

ARTICLE INFO

Article history: Received 8 January 2017 Received in revised form 21 March 2017 Accepted 3 May 2017

Keywords: deep spine infection implant removal mortality relapse reoperation spinal implant

ABSTRACT

Background/Purpose: It is unclear whether implant removal is necessary when deep spine infection of spinal instrumentation occurs. This study compares mortality, relapse, and reoperation rates between such patients with and without removal of spine implants.

Methods: A total of 20 patients were retrospectively reviewed. Baseline characteristics of the implant removal and nonremoval groups were compared. Outcome measures between groups were compared using multivariable logistic regression and predictors of each outcome identified.

Results: There were no significant differences in mortality, relapse, or reoperation rates between groups. Multiple vertebral level involvement was common (85%), and the L4 (30%) and L5 (35%) levels were most commonly involved. The majority of patients had osteomyelitis/spondylodiscitis (50%) and *Staphylococcus aureus* infections (60%). Thoracic spine infection was associated with relapse (odds ratio = 1.26) and reoperation (odds ratio = 1.101).

Conclusion: Implant removal is not always necessary in cases of deep spine infection as retention of implants is not associated with higher mortality, relapse, or reoperation rates.

中文摘要

背景/目的:目前還不清楚是否需要在已有脊柱內固定植入物發生深層脊椎感染時將脊柱植入物移除。這項研 究比較了有移除或沒有移除脊柱植入物的患者之間的死亡率、復發率和再手術率。 方法:回顧性分析20例受試者。比較植入物去除和未去除組別的基線特徵。以多變量邏輯和預測因子將兩組 別每個確定的結果測量進行比較。 結果:兩組別之間的死亡率、復發率或再手術率無統計學意義的差異。多站點脊椎發生是常見的(85%)。

結果, 网起为之间的死亡率、该数率或舟于闸率照 統計季息義的差共。夕始為育准数主定常兒的(65%)。 L4(30%)和L5(35%) 最常見的。大多數患者有骨髓炎/脊椎椎間盤炎 (50%) 和金黃葡萄球菌感染(60%)。胸椎感 染與復發 (OR=1.26) 和再次手術 (OR=1.101)有關。

Introduction

Deep infection involving the instrumented spine is an unfortunate complication of spine surgery with an incidence ranging from 0.2% to 6.7%.^{1–4} Infection could result from haematogenous seeding, adjacent spread, or contamination during the time of spinal instrumentation.^{5–7} Management should aim at timely diagnosis⁸ and instituting early treatment. This often involves a prolonged course of appropriate antibiotics, surgical debridement, with or without the removal of existing implants.⁸

Microorganisms form a layer of biofilm on implants, leading to difficulty in eradication, and frequent relapses.⁹ The surgical dilemma is whether the spinal implants should be routinely removed to aid bacterial clearance, or should they be retained to provide spinal stability.

^{*} Corresponding author. *E-mail:* dennis_hey@nuhs.edu.sg.

http://dx.doi.org/10.1016/j.jotr.2017.05.003

^{2210-4917/}Copyright © 2017, Hong Kong Orthopaedic Association and the Hong Kong College of Orthopaedic Surgeons. Published by Elsevier (Singapore) Pte Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

When stabilization is critical, removal of implants may lead to neurological sequelae, problems with brace fitting or prolonged bed immobilisation that has to be balanced with the potential benefits of eradicating infection and reducing recurrence.¹⁰

In this study, we compared the outcomes in terms of mortality, relapse, and reoperation in patients with spinal instrumentation who either underwent implant retention or removal. We hypothesized that there would be no difference in any of the outcomes between these two groups.

Methods

This was part of a large retrospective study of all patients with pyogenic deep spine infection treated at an academic medical centre in Singapore from 1999 to 2012.¹¹ The hospital is a tertiary healthcare centre with seven spine specialists and eight active infectious disease specialists. Surgery for all deep spine infection was decided based on a clinical consensus made during grand rounds attended by the same panel of spine specialists in consultation with the infectious disease team.

This study defined deep spine infection as patients with clinically and radiologically apparent typical features, with or without the isolation of microorganisms because of the well-documented risk of false-negative sampling.^{12,13} Patients without radiological evidence of deep spine infection were excluded. In our inclusion criteria, patients had clinical features of back pain or constitutional symptoms (fever, loss of weight and appetite) and radiological evidence on magnetic resonance imaging (MRI) scans, including increased signal intensity on T2-weighted images in the vertebral body or disc space, or decreased signal intensity in the disc and adjacent endplates on T1-weighted images, with or without the presence of epidural and paraspinal abscesses. Patients with suspected tuberculous infections, which are endemic in the region, were excluded.

All study patients were identified from electronic databases maintained by both the orthopaedic and infectious disease departments. Further verification was performed by two independent auditors not directly involved in the study, to ensure that all patients met the inclusion criteria before enrolment.

Following Domain Specific Institutional Review Board and National Ethics Domain Specific Review Board approval (reference number: 2011/02010), all electronic documentations and hardcopy medical case records were reviewed. The main outcome data collected included patient mortality, relapse of deep infection requiring only further antibiotics, and reoperation for deep spine infection. Patient characteristics and other possible predictors of poor outcome were also collected. They include demographics, comorbidity, clinical presentation, details of spine infection (radiological, laboratory, and microbiological findings), antibiotics treatment, and surgical details (debridement and surgery).

All data were collected by a single doctor and audited by an independent orthopaedic specialist for accuracy. Any doubt in the clinical documentation was clarified with the primary team managing the patient. All radiological images inclusive of X rays, computed tomography, and gadolinium-enhanced MRI were reviewed by two additional orthopaedic surgeons not directly involved in the study. If there was any discrepancy in the interpretation of these images, another musculoskeletal radiologist was consulted and a consensus was reached.

All patients were followed up for the three outcome parameters up to a minimum of 2 years duration or if a positive outcome occurred, whichever was shorter. All patients whose medical records showed a loss to follow-up were also contacted to ensure that they did not visit another hospital for relapse of infection requiring treatment.

Statistical analysis

All information collected was entered into Microsoft Excel Spreadsheet 2011 (version 14.0.4760.1000, 32-bit; Microsoft Corporation, Redmond, WA, USA) and analysed using SPSS version 16 (SPSS Inc., Chicago, IL, USA). Statistical significance was set as p < 0.05 for all computations. Univariate analysis was performed for baseline patient characteristics between those with implant removal and those without using χ^2 and t tests. Multivariable logistic regression modelling was subsequently used to determine the differences in both groups in terms of outcome measures, while adjusting for confounders. Odds ratios (ORs) are represented and significant predictors of each outcome were also identified.

Results

This study included 20 patients who satisfied the inclusion criteria. There were 12 men (60%) and eight women (40%). Their mean age was 52.6 (standard deviation, 18.4) years. Out of the 20 patients, seven had diabetes mellitus (35%) and two had chronic renal failure (10%). At presentation, 13 patients (65%) had significant back pain, seven patients (35%) had persistent fever, and five patients (25%) had new-onset neurological deficits. Table 1 shows the baseline characteristics of the patients who were similar for both groups.

The most commonly involved level of the spine was the lumbar region, specifically at L4 and L5, which was involved in 30% and 35% of the patients, respectively. Multiple vertebral level involvements occurred in 85% of the patients. The prevalence of osteomyelitis/ spondylodiscitis (50%), epidural abscess (45%), and paravertebral/psoas abscess (40%) was similar. There was no significant difference in the mean total white blood cell count, erythrocyte sedimentation rate, and C-reactive protein values for both groups. No patients presented with pathological fractures in our cohort.

Microorganisms were identified in 19 patients (95%). Staphylococcus aureus was the most common causative organism isolated (60%),^{14,15} followed by *Klebsiella pneumoniae* (15%). In our institution, cefazolin is the empirical antibiotic of choice when a deep spine infection is suspected. It is started promptly after cultures have been taken from the patient and changed accordingly based on definitive culture results. All empirical antibiotics started were ultimately effective against the microorganism with the exception of two patients (1 patient from each group). In these patients, the antibiotic was adjusted accordingly soon after culture results were available. Intravenous antibiotics were converted to oral antibiotics for all patients upon reduction of all inflammatory markers (total white blood cell count, erythrocyte sedimentation rate, C-reactive protein) below 50% of the peak values. They were only stopped after 6 weeks to 3 months of treatment if the patient also had resolution of clinical features, and supporting evidence from an interval gadolinium-enhanced MRI if available.

In our series, 17 patients (85%) had one operation and 16 patients (94%) were successfully treated with follow-up antibiotics for a total duration of 3 months. Three patients (15%) required two operations and all were successfully treated with follow-up antibiotics for a total duration of 3 months. No patients required more than two operations. All cases had a low suction drain connected to the surgical site that was only removed when not needed.

When comparing patients between the two groups for outcome variables, there was no difference in mortality, relapse, and reoperation rates. Under multivariate analysis, thoracic spine infection was the single parameter found to be associated with higher relapse (OR = 1.26, 95% confidence interval = 1.097-1.447) and reoperation rates (OR = 1.101, 95% confidence interval = 1.037-1.168). Table 2 shows the results for the three different outcomes in this study

Table 1

Baseline characteristics of study population

Percent propertiesSet of the set of the s		n = 20	<i>n</i> = 13 (retain)	%	<i>n</i> = 7 (remove)	%	р
Mean age (y) 52.6 56.3 45.7 0.232 Sex Aremale 9 male 602 3 male 4.5 0.096 Ithinity (Chinese) 65 7.14 0.317 0.017	Demographics						
Sex 12 male 9 male 602 3 male 42.9 0.006 Ethnictly (Chinese) 65 61.6 71.4 0.317 Comorbidity 65 11 84.6 5 71.4 0.317 ASA 1-2 16 11 84.6 5 71.4 0.292 ASA 3-4 4 2 28.6 0.271 2.26.6 0.271 Charlson 0° 3 1 7.7 2 28.6 0.271 Charlson 1 0 0 0 0 0.0 0.0 Charlson 4 5 2 3.1 2 28.6 0.53 Charlson 4 5 2 3.6 0.53 0.0 0.1 1.3 0.41 0.0 0.147 Neurological symptoms 1 0 0.0 1.43 0.491 0.43 0.491 Neurological symptoms 1 0 0 0 0.379 1.44.3 0.491 Neurologic	Mean age (y)	52.6	56.3		45.7		0.232
Image Ethnicity (binese) ComorbidityIfemale 6661.67.1Ethnicity (binese) Comorbidity63.77.40.317ASA 1-211.48.4.67.40.232ASA 3-3-4421.5.42.2.60.2.7Charlson 100.00.00.00.0Charlson 100.00.00.00.0Charlson 113.2.22.8.60.71Charlson 343.0.800.00.0Charlson 43.0.800.00.0Charlson 5.433.2.12.02.0.6Charlson 5.43.32.3.10.00.00.0Charlson 5.43.32.3.10.00.0Charlson 5.41.30.1477.5.33.8.22.8.60.589LOW/LOA33.2.3.10.00.01.4.30.147Neurological symptoms1.40.43.0.81.4.30.141Neurological deficits5.43.6.22.8.60.589J levels003.8.22.4.20.63Automic involvement5.40.01.4.30.414Neurological deficits1.6.43.6.42.4.22.8.6J levels003.8.22.4.20.6.3J levels1.6.42.4.13.6.40.4.20.4.2J levels1.6.42.4.13.6.40.4.10.4.1J levels0	Sex	12 male	9 male	69.2	3 male	42.9	0.096
Ethnicity (chinese) comorbidity6561.671.40.317Comorbidity161184.6571.40.292ASA 1-216215.4228.60.271Charlson 0'317.7228.60.271Charlson 10000.00.00.0Charlson 25323.1228.6Charlson 34.430.800.00.0Charlson 45215.4342.9Charlson 4538.5228.60.89Charlson 413107.6342.90.063Fever7585.5228.60.899LOWLOA3323.1000.147Neurological symptoms100.0114.30.496No. of spinal level involvement217.7114.30.496No. of spinal level involvement217.7111.430.496Site of infection3323.10011.430.496Vice (x10 ⁴ /1/1)1066.242.10.16110.141.430.496Jevels33323.10011.430.49611.430.49611.430.49611.430.49611.430.49611.43 </td <td></td> <td>8 female</td> <td>4 female</td> <td>30.8</td> <td>4 female</td> <td>57.1</td> <td></td>		8 female	4 female	30.8	4 female	57.1	
Comorbidity Construction Construction </td <td>Ethnicity (Chinese)</td> <td>65</td> <td></td> <td>61.6</td> <td></td> <td>71.4</td> <td>0.317</td>	Ethnicity (Chinese)	65		61.6		71.4	0.317
ASA 1-2 16 11 846 5 71.4 0.225 ASA 3-4 4 2 15.4 2 28.6 0.271 Charlson 0° 3 1 7.7 2 28.6 0.271 Charlson 1 0 0 0.0 0.0 0.0 0.0 Charlson 2 5 3 23.1 2 28.6 0.271 Charlson 4 5 2 15.4 3 42.9 0.03 Charlson 4 5 2 15.4 3 42.9 0.063 Charlson 4 3 0 0.0 0.0 0.0 0.0 Charlson 4 3 3 23.1 0 0.0	Comorbidity						
ASA-4 4 2 15.4 2 28.6 Charlson 0° 3 1 7,7 2 28.6 0.271 Charlson 1 0 0.0 0.0 0.0 0.0 Charlson 2 5 3 23.1 2 28.6 0.00 Charlson 3 4 4 30.8 0.00 0.00 0.00 0.00 Charlson 3 4 3 3 23.1 0.00	ASA 1–2	16	11	84.6	5	71.4	0.292
Charlson 0° 3 1 7.7 2 28.6 0.271 Charlson 1 0 0 0.0 0.0 0.0 0.0 Charlson 2 5 3 23.1 2 28.6 0.0 Charlson 3 4 4 30.8 0 0.0 0.0 Charlson 4 5 2 15.4 3 42.9 0.06 Charlson 4 3 3 23.1 0 0.0 0.0 Clinical presentation 13 10 76.9 3 42.9 0.063 Ecw(LOA) 3 3 23.1 0 0.0 0.1473 Neurological symptoms 1 0 0.0 1 14.3 0.90 Autonomic involvement 2 1 7.7 1 14.3 0.90 Autonomic involvement 2 2 16.5 2 28.6 0.379 1 1evel 3 3 23.1 0	ASA 3-4	4	2	15.4	2	28.6	
Charlson 1 0 0 0.0 0 0.0 0.0 Charlson 2 5 3 23.1 2 28.6 Charlson 3 4 4 30.8 0.0 0.0 Charlson 3 4 30.8 0 0.0 Charlson 3 2 15.4 3 42.9 Charlson 4 3 23.1 0 0.0 Clinical presentation	Charlson 0 ^a	3	1	7.7	2	28.6	0.271
Charlson 2 5 3 23.1 2 28.6 Charlson 3 4 4 30.8 0 0.0 Charlson 4 5 2 15.4 3 42.9 Charlson 4 5 2 15.4 3 42.9 Charlson 4 3 3 23.1 0 0.0 Clinical presentation 13 10 76.9 3 42.9 0.063 Fever 7 5 38.5 2 28.6 0.899 LOW/LOA 3 3 23.1 0 0.0 0.147 Neurological symptoms 1 0 0.0 1.4.3 0.909 Autonomic involvement 2 1 7.7 1 1.4.3 0.909 Autonomic involvement 2 0 0 0 3.231 0 0 3.2429 2 levels 10 8 23.1 0 0 3.2429 3.2429 3.2429 3.2429	Charlson 1	0	0	0.0	0	0.0	
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	Charlson 2	5	3	23.1	2	28.6	
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	Charlson 3	4	4	30.8	2	0.0	
$\begin{array}{c c c c c c c } Charlson >4 & 3 & 2 & 2 & 2 & 0 & 0 & 0 & 0 & 0 & 0 & 0$	Charlson 4	5	2	15.4	3	42.9	
$\begin{array}{c c c c c c } Can constrain a bord bord bord bord bord bord bord bord$	Charlson \4	3	3	23.1	0	0.0	
107.6.9342.90.063Fever7538.5228.60.589LOW/LOA3323.100.00.147Neurological symptoms100.0114.30.141Neurological deficits5430.8114.30.090Autonomic involvement217.7114.30.090O000000.3379I level300342.922 levels10861.5228.63 levels3323.1003Site of infection5861.5228.60.6161Ost complitis/spondyldiscitis10646.24228.60.6161Paravetebral/psoas8753.8228.60.6380.638CRP (mg/L)9.3 (SD 2.4)9.2 (SD 2.8)9.4 (SD 1.2)6.6386.6380.638Laboratory values (at amission)9.4 (SD 30.7)50.3 (SD 20.3)6.6386.386.332.6590.638WBC ($\times 10^9/L$)6.65 (SD 7.1)9.7 (SD 5.3)18.7 (SD 8.4)0.4126.730.412Staphylococcus auruus1296.92342.90.734Kleviella pneunomiae3215.7 (SD 5.3)18.7 (SD 8.4)0.412ESR (mm/h)	Clinical presentation	5	5	23.1	0	0.0	
Date A pain151070.5542.50.003Fever7538.5228.60.589LOW/LOA3323.100.00.147Neurological symptoms100.0114.30.141Neurological deficits5430.8114.30.090Autonomic involvement217.7114.30.090No. of spinal level involvement217.7114.30.0901 level300342.902 levels10861.5228.6103 levels3323.10003 levels4215.4228.616.1Site of infection646.2457.10.425Epidural abscess9646.2228.60.054Laboratory values (at admission)9.3 (SD 2.4)9.2 (SD 2.8)9.4 (SD 1.2)0.875ESR (mm/h)56.8 (SD 31.1)59.4 (SD 20.7)50.3 (SD 20.3)0.638Laboratory values (highest point)103.7 (SD 8.8)78.9 (SD 70.9)18.7 (SD 8.4)0.412WBC (×10 ⁹ /L)16.9 (SD 7.2)15.7 (SD 5.3)18.7 (SD 8.4)0.412CRP (mg/L)16.9 (SD 7.2)15.7 (SD 5.3)18.7 (SD 8.4)0.412Klebiella pneunomice3211.430.412Klebiella pneunomice32 <t< td=""><td>Back pain</td><td>13</td><td>10</td><td>76.0</td><td>3</td><td>12.0</td><td>0.063</td></t<>	Back pain	13	10	76.0	3	12.0	0.063
I eveljj <td>Four</td> <td>7</td> <td>5</td> <td>70.9</td> <td>5 0</td> <td>42.9</td> <td>0.003</td>	Four	7	5	70.9	5 0	42.9	0.003
Low(L)A3525.100.00.141Neurological symptoms100.0114.30.141Neurological deficits5430.8114.30.090Autonomic involvement217.7114.30.090No. of spinal level involvement217.7114.30.0490No. of spinal level involvement000000.3791 level300342.922 levels10861.5228.623 levels3323.10033 levels4215.4228.61Site of infection510646.2457.10.425Paravetebral/psoas8753.8228.60.161Paravetebral/psoas8753.8228.60.613Laboratory values (at admission)92 (SD 2.8)94 (SD 1.2)0.8750.875WBC ($\times 10^9$ [L)60.5 (SD 71.3)49 (SD 26.9)77.6 (SD 10.0)0.468Laboratory values (highest point)16.9 (SD 7.2)15.7 (SD 5.3)18.7 (SD 8.4)0.412ESR (mm/h)86.4 (SD 29.9)79.0 (SD 34.5)98.0 (SD 10.1)0.198Microbiology10.37 (SD 88.8)78.9 (SD 70.9)142.5 (SD 97.2)0.143Microbiology12969.2342.90.734<		2	3	20.5	2	28.0	0.389
Neurological symptoms1000114.30.141Neurological deficits5430.8114.30.090Autonomic involvement217.7114.30.090No. of spinal level involvement000000.037911evel300342.912levels10861.5228.613levels323.100328.61Site of infection646.24228.60.161Paravetebral/psoas8753.8228.60.054Laboratory values (at admission)9.3 (SD 2.4)9.4 (SD 30.7)50.3 (SD 20.3)0.633CRP (mg/L)9.3 (SD 2.4)9.4 (SD 30.7)50.3 (SD 20.3)0.633CRP (mg/L)6.9 (SD 7.1)59.4 (SD 30.7)50.3 (SD 20.3)0.633CRP (mg/L)16.9 (SD 7.2)15.7 (SD 5.3)18.7 (SD 8.4)0.412ESR (mm/h)86.4 (SD 29.9)79.0 (SD 34.5)9.60 (SD 10.1)0.198CRP (mg/L)10.7 (SD 88.8)78.9 (SD 70.9)142.5 (SD 97.2)0.134Microbiology17.7114.314.3Cozquidas-negative staphylococci117.7114.3Cozquidas-negative staphylococci117.700.0Ferkerichin curve17.7114.31.43Cozqu	LOW/LOA	5	3	25.1	0	0.0	0.147
Nethological deficits3430.8114.30.099Autonomic involvement217.7114.30.099No. of spinal level involvement00000.37911300342.92levels10861.5228.63levels3323.100 > 3 levels4215.4228.6Site of infectionOsteomyelitis/spondyldiscitis10646.245.7.10.425Epidural abscess9666.2228.60.054Laboratory values (at admission)53.8228.60.638CRP (mg/L)9.3 (SD 2.4)9.2 (SD 2.8)-9.4 (SD 1.2)0.875ESR (mm/h)56.8 (SD 31.1)59.4 (SD 30.7)50.3 (SD 20.3)0.638CRP (mg/L)16.9 (SD 7.2)15.7 (SD 5.3)18.7 (SD 8.4)0.412Laboratory values (highest point)0.05 (SD 71.3)49 (SD 26.9)9.80 (SD 10.1)0.198CRP (mg/L)10.3.7 (SD 88.8)7.8.9 (SD 70.9)14.2.5 (SD 97.2)0.143MicrobiologyStaphylococcus arerus12969.2342.90.734Klebsiella pneunomiae217.7114.3-Staphylococci117.70 <td>Neurological deficito</td> <td>I F</td> <td>0</td> <td>0.0</td> <td>1</td> <td>14.3</td> <td>0.141</td>	Neurological deficito	I F	0	0.0	1	14.3	0.141
Autonomic involvement217.7114.30.439No. of spinal level involvement00000.3791 level300342.92 levels10861.5228.63 levels323.100-3 levels4215.4228.6Site of infectionOsteomyelitis/spondyldiscitis10646.2228.6Epidural abscess9646.2228.60.614Paravetebral/psoas8753.8228.60.614Laboratory values (at admission)-59.4 (SD 3.7)50.3 (SD 20.3)0.638Laboratory values (highest point)9.3 (SD 2.4)9.2 (SD 2.8)9.4 (SD 1.2)0.875ESR (mm/h)56.8 (SD 31.1)59.4 (SD 30.7)50.3 (SD 20.3)0.638Laboratory values (highest point)0.412WBC (×10 ⁹ /L)16.9 (SD 7.2)15.7 (SD 5.3)18.7 (SD 8.4)0.412ESR (mm/h)86.4 (SD 29.9)79.0 (SD 34.5)98.0 (SD 10.1)0.198MicrobiologyMicrobiologyMicrobiology-17.7114.3Alphylococcus areus117.700.0Staphylococci117.700.0Staphyloco	Neurological delicits	5	4	30.8	1	14.3	0.090
No. of spinal level involvement000000.3791 level300342.92 levels10861.5228.63 levels3323.100>3 levels4215.4228.6Site of infection510646.2457.10.425Spidural abscess9646.2228.60.161Paravetebral/psoas8753.8228.60.161Laboratory values (at admission)9.2 (SD 2.8)9.4 (SD 1.2)0.8756.850.654Laboratory values (bighest point)9.3 (SD 2.4)9.2 (SD 2.8)9.4 (SD 1.2)0.8750.675ESR (mm/h)60.5 (SD 71.3)49 (SD 26.9)77.6 (SD 100.0)0.4680.468Laboratory values (highest point)16.9 (SD 7.2)15.7 (SD 5.3)18.7 (SD 8.4)0.412WBC (×10 ⁹ /L)103.7 (SD 8.8.8)78.9 (SD 70.9)142.5 (SD 97.2)0.143Microbiology12969.2342.90.734Klebsiela pneunomiae3215.4114.314.3Pseudomonas aeruginosa217.7114.35Fscheridir coli17.700.00.01	Autonomic involvement	2	1	7.7	1	14.3	0.496
0 0	No. of spinal level involvement			â	0	0	0.070
1 level 3 0 0 3 42.9 2 levels 10 8 61.5 2 28.6 3 levels 3 23.1 0 0 3 >3 levels 4 2 15.4 2 28.6 Site of infection 5 9 6 46.2 4 57.1 0.425 Epidural absces 9 6 46.2 2 28.6 0.054 Laboratory values (at admission) 7 53.8 2 28.6 0.054 Laboratory values (at admission) 9.3 (SD 2.4) 9.2 (SD 2.8) 9.4 (SD 1.2) 0.875 ESR (mm/h) 56.8 (SD 31.1) 59.4 (SD 30.7) 50.3 (SD 20.3) 0.638 CRP (mg/L) 60.5 (SD 71.3) 49 (SD 26.9) 77.6 (SD 100.0) 0.468 Laboratory values (highest point) 15.7 (SD 5.3) 18.7 (SD 8.4) 0.412 WBC (×10 ⁹ /L) 16.9 (SD 7.2) 15.7 (SD 5.3) 18.7 (SD 8.4) 0.412 LSR (mm/h) 86.4 (SD 29.9) 79.0 (SD 34.5) 98.0 (SD 10.1) 0.198 CRP (mg/L)	0	0	0	0	0	0	0.379
$\begin{array}{cccccc} 2 \mbox{ levels} & 10 & 8 & 61.5 & 2 & 28.6 \\ 3 \mbox{ levels} & 3 & 23.1 & 0 & 0 \\ > 3 \mbox{ levels} & 4 & 2 & 15.4 & 2 & 28.6 \\ \hline Site of infection & & & & & & & & & & & & & & & & & & &$	1 level	3	0	0	3	42.9	
3 levels 3 23.1 0 0 >3 levels 4 2 15.4 2 28.6 Site of infection 5 10 6 46.2 4 57.1 0.425 Epidural abscess 9 6 46.2 2 28.6 0.161 Paravetebral/psoas 8 7 53.8 2 28.6 0.054 Laboratory values (at admission) 9.3 (SD 2.4) 9.2 (SD 2.8) 9.4 (SD 1.2) 0.875 ESR (mm/h) 56.8 (SD 31.1) 59.4 (SD 30.7) 50.3 (SD 20.3) 0.638 CRP (mg/L) 60.5 (SD 71.3) 49 (SD 26.9) 77.6 (SD 100.0) 0.468 Laboratory values (highest point) WBC (× 10 ⁹ /L) 16.9 (SD 7.2) 15.7 (SD 5.3) 18.7 (SD 8.4) 0.412 ESR (mm/h) 86.4 (SD 29.9) 79.0 (SD 34.5) 98.0 (SD 10.1) 0.198 CRP (mg/L) 103.7 (SD 88.8) 78.9 (SD 70.9) 142.5 (SD 97.2) 0.143 Microbiology 1 7.7 1 14.3 Pseudomonas aeruginosa 2 15.4 1 14.3 <td>2 levels</td> <td>10</td> <td>8</td> <td>61.5</td> <td>2</td> <td>28.6</td> <td></td>	2 levels	10	8	61.5	2	28.6	
> 3 levels4215.4228.6Site of infection 0 6 46.2 4 57.1 0.425 Osteomyelitis/spondyldiscitis10 6 46.2 4 57.1 0.425 Epidural abscess9 6 46.2 2 28.6 0.161 Paravetebral/psoas 8 7 53.8 2 28.6 0.054 Laboratory values (at admission) V 9.2 (SD 2.8) 9.4 (SD 1.2) 28.6 0.054 WBC $(\times 10^9/L)$ 9.3 (SD 2.4) 9.2 (SD 2.8) 9.4 (SD 1.2) 0.875 ESR (mm/h) 56.8 (SD 31.1) 59.4 (SD 30.7) 50.3 (SD 20.3) 0.638 CRP (mg/L) 60.5 (SD 71.3) 49 (SD 26.9) 77.6 (SD 100.0) 0.468 Laboratory values (highest point) V V 0.412 ESR (mm/h) 66.4 (SD 29.9) 79.0 (SD 34.5) 98.0 (SD 10.1) 0.412 ESR (mm/h) 86.4 (SD 29.9) 78.9 (SD 70.9) 142.5 (SD 97.2) 0.143 Microbiology V V V 0.74 14.3 Pseudomonas aeruginosa 2 1 7.7 1 14.3 <i>Pseudomonas aeruginosa</i> 2 1 7.7 0 0.0	3 levels	3	3	23.1	0	0	
Site of infection osteomyelitis/spondyldiscitis 10 6 46.2 4 57.1 0.425 Epidural abscess 9 6 46.2 2 28.6 0.161 Paravetebral/psoas 8 7 53.8 2 28.6 0.054 Laboratory values (at admission) 7 53.8 2 28.6 0.054 WBC (×10 ⁹ /L) 9.3 (SD 2.4) 9.2 (SD 2.8) 9.4 (SD 1.2) 0.875 ESR (mm/h) 56.8 (SD 31.1) 59.4 (SD 30.7) 50.3 (SD 20.3) 0.638 CRP (mg/L) 60.5 (SD 71.3) 49 (SD 26.9) 77.6 (SD 100.0) 0.638 Laboratory values (highest point) 9.4 (SD 1.2) 0.412 WBC (×10 ⁹ /L) 16.9 (SD 7.2) 15.7 (SD 5.3) 18.7 (SD 8.4) 0.412 ESR (mm/h) 86.4 (SD 29.9) 79.0 (SD 34.5) 98.0 (SD 10.1) 0.198 CRP (mg/L) 10.37 (SD 88.8) 78.9 (SD 70.9) 14.7 (SD 8.9) 0.143 Microbiology 1 2 1 1	>3 levels	4	2	15.4	2	28.6	
Osteomyelitis/spondyldiscitis10646.2457.10.425Epidural abscess9646.2228.60.161Paravetebral/psoas8753.8228.60.054Laboratory values (at admission)9.3 (SD 2.4)9.2 (SD 2.8)9.4 (SD 1.2)0.875ESR (mm/h)56.8 (SD 31.1)59.4 (SD 30.7)50.3 (SD 20.3)0.638CRP (mg/L)60.5 (SD 71.3)49 (SD 26.9)77.6 (SD 100.0)0.468Laboratory values (highest point)16.9 (SD 7.2)15.7 (SD 5.3)18.7 (SD 8.4)0.412WBC (× 10 ⁹ /L)16.9 (SD 7.2)15.7 (SD 5.3)98.0 (SD 10.1)0.198CRP (mg/L)10.3.7 (SD 88.8)78.9 (SD 70.9)142.5 (SD 97.2)0.143Microbiology5taphylococcus aureus12969.2342.90.734Klebsiella pneunomiae3215.4114.35Pseudomonas aeruginosa217.700.00.0Fsecheripting coil117.700.00.0	Site of infection						
Epidural abscess 9 6 46.2 2 28.6 0.161 Paravetebral/psoas 8 7 53.8 2 28.6 0.054 Laboratory values (at admission) .<	Osteomyelitis/spondyldiscitis	10	6	46.2	4	57.1	0.425
Paravetebral/psoas 8 7 53.8 2 28.6 0.054 Laboratory values (at admission)	Epidural abscess	9	6	46.2	2	28.6	0.161
Laboratory values (at admission)WBC (×10 ⁹ /L)9.3 (SD 2.4)9.2 (SD 2.8)9.4 (SD 1.2)0.875ESR (mm/h)56.8 (SD 31.1)59.4 (SD 30.7)50.3 (SD 20.3)0.638CRP (mg/L)60.5 (SD 71.3)49 (SD 26.9)77.6 (SD 100.0)0.468Laboratory values (highest point)15.7 (SD 5.3)18.7 (SD 8.4)0.412WBC (×10 ⁹ /L)16.9 (SD 7.2)15.7 (SD 5.3)98.0 (SD 10.1)0.198CRP (mg/L)103.7 (SD 88.8)78.9 (SD 70.9)142.5 (SD 97.2)0.143Microbiology54.4 (SD 29.9)969.2342.90.734Klebsiella pneunomiae3215.4114.314.3Pseudomonas aeruginosa217.7114.314.3Coagulase-negative staphylococci117.700.00.0	Paravetebral/psoas	8	7	53.8	2	28.6	0.054
WBC $(\times 10^9/L)$ 9.3 (SD 2.4)9.2 (SD 2.8)9.4 (SD 1.2)0.875ESR (mm/h)56.8 (SD 31.1)59.4 (SD 30.7)50.3 (SD 20.3)0.638CRP (mg/L)60.5 (SD 71.3)49 (SD 26.9)77.6 (SD 10.0)0.468Laboratory values (highest point) $WBC (\times 10^9/L)$ 16.9 (SD 7.2)15.7 (SD 5.3)18.7 (SD 8.4)0.412WBC $(\times 10^9/L)$ 16.9 (SD 7.2)15.7 (SD 5.3)98.0 (SD 10.1)0.198CRP (mg/L)103.7 (SD 88.8)78.9 (SD 70.9)142.5 (SD 97.2)0.143Microbiology5taphylococcus aureus12969.2342.90.734Klebsiella pneunomiae3215.4114.314.3Pseudomonas aeruginosa217.7114.3Coagulase-negative staphylococci117.700.0	Laboratory values (at admission)						
ESR (mm/h) $56.8 (SD 31.1)$ $59.4 (SD 30.7)$ $50.3 (SD 20.3)$ 0.638 CRP (mg/L) $60.5 (SD 71.3)$ $49 (SD 26.9)$ $77.6 (SD 100.0)$ 0.468 Laboratory values (highest point) $WBC (\times 10^9/L)$ $16.9 (SD 7.2)$ $15.7 (SD 5.3)$ $18.7 (SD 8.4)$ 0.412 WBC ($\times 10^9/L)$ $86.4 (SD 29.9)$ $79.0 (SD 34.5)$ $98.0 (SD 10.1)$ 0.198 CRP (mg/L) $103.7 (SD 88.8)$ $78.9 (SD 70.9)$ $142.5 (SD 97.2)$ 0.143 Microbiology 552 15.4 1 14.3 Pseudomonas aeruginosa 2 15.4 1 14.3 Cogulase-negative staphylococci 1 1 7.7 0 0.0	WBC ($\times 10^9/L$)	9.3 (SD 2.4)	9.2 (SD 2.8)		9.4 (SD 1.2)		0.875
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	ESR (mm/h)	56.8 (SD 31.1)	59.4 (SD 30.7)		50.3 (SD 20.3)		0.638
Laboratory values (highest point) 16.9 (SD 7.2) 15.7 (SD 5.3) 18.7 (SD 8.4) 0.412 WBC ($\times 10^9/L$) 86.4 (SD 29.9) 79.0 (SD 34.5) 98.0 (SD 10.1) 0.198 CRP (mg/L) 103.7 (SD 8.8.) 78.9 (SD 70.9) 142.5 (SD 97.2) 0.143 Microbiology 5taphylococcus aureus 12 9 69.2 3 42.9 0.734 Klebsiella pneunomiae 3 2 15.4 1 14.3 Pseudomonas aeruginosa 2 1 7.7 1 14.3 Coagulase-negative staphylococci 1 1 7.7 0 0.0	CRP (mg/L)	60.5 (SD 71.3)	49 (SD 26.9)		77.6 (SD 100.0)		0.468
WBC (×10 ⁹ /L) 16.9 (SD 7.2) 15.7 (SD 5.3) 18.7 (SD 8.4) 0.412 ESR (mm/h) 86.4 (SD 29.9) 79.0 (SD 34.5) 98.0 (SD 10.1) 0.198 CRP (mg/L) 103.7 (SD 88.8) 78.9 (SD 70.9) 142.5 (SD 97.2) 0.143 Microbiology 5taphylococcus aureus 12 9 69.2 3 42.9 0.734 Klebsiella pneunomiae 3 2 15.4 1 14.3 Pseudomonas aeruginosa 2 1 7.7 1 14.3 Coagulase-negative staphylococci 1 7.7 0 0.0	Laboratory values (highest point)						
ESR (mm/h) 86.4 (SD 29.9) 79.0 (SD 34.5) 98.0 (SD 10.1) 0.198 CRP (mg/L) 103.7 (SD 88.8) 78.9 (SD 70.9) 142.5 (SD 97.2) 0.143 Microbiology 5taphylococcus aureus 12 9 69.2 3 42.9 0.734 Klebsiella pneunomiae 3 2 15.4 1 14.3 Pseudomonas aeruginosa 2 1 7.7 1 14.3 Coagulase-negative staphylococci 1 7.7 0 0.0	WBC ($\times 10^9/L$)	16.9 (SD 7.2)	15.7 (SD 5.3)		18.7 (SD 8.4)		0.412
CRP (mg/L) 103.7 (SD 88.8) 78.9 (SD 70.9) 142.5 (SD 97.2) 0.143 Microbiology	ESR (mm/h)	86.4 (SD 29.9)	79.0 (SD 34.5)		98.0 (SD 10.1)		0.198
Microbiology Staphylococcus aureus 12 9 69.2 3 42.9 0.734 Klebsiella pneunomiae 3 2 15.4 1 14.3 Pseudomonas aeruginosa 2 1 7.7 1 14.3 Coagulase-negative staphylococci 1 1 7.7 0 0.0	CRP (mg/L)	103.7 (SD 88.8)	78.9 (SD 70.9)		142.5 (SD 97.2)		0.143
Staphylococcus aureus 12 9 69.2 3 42.9 0.734 Klebsiella pneunomiae 3 2 15.4 1 14.3 Pseudomonas aeruginosa 2 1 7.7 1 14.3 Coagulase-negative staphylococci 1 1 7.7 0 0.0	Microbiology						
Klebsiella pneunomiae 3 2 15.4 1 14.3 Pseudomonas aeruginosa 2 1 7.7 1 14.3 Coagulase-negative staphylococci 1 1 7.7 0 0.0 Escherichia coli 1 7.7 0 0.0	Staphylococcus aureus	12	9	69.2	3	42.9	0.734
Pseudomonas aeruginosa 2 1 7.7 1 14.3 Coagulase-negative staphylococci 1 1 7.7 0 0.0 Escherichia coli 1 1 7.7 0 0.0	Klebsiella pneunomiae	3	2	15.4	1	14.3	
Coagulase-negative staphylococci117.700.0Escherichia coli117.700.0	Pseudomonas aeruginosa	2	1	7.7	1	14.3	
Exclusion 1 1 1 77 0 00	Coagulase-negative staphylococci	1	1	7.7	0	0.0	
	Escherichia coli	1	1	7.7	0	0.0	
Antimicrobials (empirical)	Antimicrobials (empirical)		-		-		
Cefazolin 16 11 84.6 5 71.4 0.253	Cefazolin	16	11	84.6	5	71.4	0.253
Vancomycin 2 1 7.7 1 143	Vancomvcin	2	1	7.7	1	14.3	
Cloxacillin 2 1 7.7 1 14.3	Cloxacillin	2	1	7.7	1	14.3	

ASA = American Society of Anesthesiology; CRP = C-reactive protein; ESR = erythrocyte sedimentation rate; LOA = loss of appetite; LOW = loss of weight; SD = standard deviation; WBC = white blood cell count.

^a Charlson comorbidity score.

arm. Of note, two patients had infection involving the thoracic spine, and one of these had implants retained and the other had implants removed.

Discussion

Deep infection involving the instrumented spine is a serious complication and occurs with an incidence ranging from 0.2% to 6.7%.^{1–4} It results in substantial morbidity to the patient and burdens the healthcare system.¹⁶ Patients with this complication may require prolonged hospital stay for intravenous antibiotics or multiple surgical procedures aimed at radical debridement and washout of infected tissues to control the infective load. However, little is known as to how extensive this infective clearance should be, and whether it should include instrument removal or retention.¹⁷

Our study examined specifically the outcomes of removing or retaining spinal implants in the context of a deep infection of an instrumented spine. We found no difference in terms of mortality, relapse, and reoperation for both groups of patients. These results are supported by earlier studies^{18,19} and suggest that implant removal need not be routine if there is a clinical indication for keeping the implants and a thorough debridement has been performed. The decisions are usually made balancing the benefits of infection eradication and risks of losing spinal stability in the absence of bony fusion.^{20,21} Other studies in the literature that support implant removal have emphasised the need for infection eradication to prevent relapse and reoperation but the evidence for this has been weak and often disputed.^{22,23} In fact, there is increasing awareness of aseptic inflammation from metal corrosion masquerading as culture-positive infection and any microorganism isolated in such cases may be of no pathogenic significance.^{22,24}

Table 2

Mortality, relapse and reoperation rates following implant-retaining or implantremoval surgery

Mortality ($n = 2$)			
Treatment	OR	95% CI	р
Implant retained $(n = 1)$	1	_	_
Implant removed $(n = 1)$	1.021	0.817-1.274	0.857
Parameter	OR	95% CI	р
Age	0.839	0.568-1.238	0.376
ASA	1.149	0.992-1.312	0.063
CCI	1.057	0.924-1.227	0.084
Relapse ($n = 4$)			
Treatment	OR	95% CI	р
Implant retained $(n = 2)$	1	_	_
Implant removed $(n = 2)$	0.983	0.749 - 1.290	0.901
Parameter	OR	95% CI	р
Age	1.056	0.636-1.754	0.833
ASA	1.161	0.972-1.388	0.1
CCI	1.052	0.954 - 1.269	0.275
Thoracic spine infection	1.260	1.097 - 1.447	0.001
Reoperation ($n = 3$)			
Treatment	OR	95% CI	р
Implant retained $(n = 2)$	1	-	—
Implant removed $(n = 1)$	0.977	0.876-1.091	0.681
Parameter	OR	95% CI	р
Age	0.812	0.670-1.252	0.821
ASA	1.113	0.842-1.326	0.538
CCI	1.253	0.934-1.296	0.172
Thoracic spine infection	1.101	1.037-1.168	0.002

ASA = American Society of Anesthesiology; CCI = Charlson Comorbidity Index; CI = confidence interval; OR = odds ratio.

Routine removal of implants in these patients may be unnecessary and potentially harmful.

It has also been shown previously that microbes form a layer of biofilm and adhere to the surface of implants, leading to difficulty in eradication.²⁵ These microbes are embedded within a selfproduced matrix of extracellular polymeric substance compose of extracellular DNA, proteins, and polysaccharides, and is impervious to the usual antibodies in the systemic circulation. Furthermore, they respond to many factors via cellular recognition of specific or nonspecific attachment sites on surfaces and undergo a phenotypic shift in behaviour to enable self-sustainment and resistance to most antibiotics.²⁶ It is therefore suggested that the only way to remove these microbes would require removal of these implants. Although this phenomenon has been demonstrated in arthroplastic surgery,¹⁹ it has not been as well documented in spine surgery. It is believed that a good blood supply at the axial skeleton compared to the appendicular skeleton makes it feasible to at least suppress these infections if not eradicate them.²

Moreover, routine removal of implants would mean subjecting patients who may have spinal instability to potential neurological sequelae.^{28,29} This occurs in a number of scenarios including a fractured spine when bony union has not occurred following fixation; a resected spine tumour when major resection renders instrumentation support mandatory; and deformed surgery following recent curve correction before bony fusion takes place. To address this issue often requires the use of external orthoses or prolonged bed immobilisation³⁰ in exchange for spinal instrumentation, which is not without inherent disadvantages. A balance of the benefits and risks is required in such circumstances. Larger and longer follow-up studies are required to evaluate the pros and cons for each approach before strong recommendations can be made.

When retaining implants is preferred for stability, the literature has conflicting data. Some authors have proposed surgical debridement and spinal stabilization performed as a single stage for a better chance of fusion and avoidance of deformity.^{20,21} They recommend that the wound should be closed over multiple drains to allow subsequent clearance of collection.³¹ However, other

studies have advocate serial wound washouts with retention of implants.^{1,2,17} Yet others propose to treat with long-term appropriate antibiotics as a form of suppressive therapy and delay removal of implants only until fusion has occurred.²⁷ The use of local vancomycin powder in the wound has also be described.³² In our series, 17 patients (85%) had one operation and 16 patients (94%) had good outcomes. We believe that a single, thorough surgical debridement of the infected spine without implant removal is usually sufficient. Proper assessment of the purpose and functionality of the implants at the point of debridement is crucial if removal is contemplated. Indications for removal of implants usually include loosening³³ or if they are no longer necessary in the absence of spinal instability.³⁴

Our multivariate analysis showed that thoracic spine involvement was a predictor of higher relapse and reoperation rates in patients retaining their implants. We believe that this could have been due to compromised initial debridement in an attempt to avoid a cord injury,³⁵ or a poorer blood supply present at the proximal thoracic spine watershed area such that response to antibiotic treatment was suboptimal. This finding has not been shown in the literature and may be worth exploring in future larger studies.

There were several strengths to this study. Although retrospective, it utilized reliable data collected judiciously by two departments via a database over the past 14 years. A minimum 2-year follow-up was also available for all patients who did not have a positive event to allow meaningful statistical calculation. However, we acknowledge that the sample population was small and may not have been powered to show significance in outcome. The retrospective nature of this study, which inherently results in missing data, prohibits the assessment of chronicity of infection when comparing groups. This may be a potential confounding factor. Finally, with the patients managed over a 14-year period, the authors also recognize inevitable time-dependent factors that could have biased the results. Nevertheless, the results shown in this study can still add to the existing evidence if interpreted with caution.

In conclusion, removing spinal implants in the context of an infected spine to better control infection appears to have no additional benefit compared to retaining these implants with respect to mortality, relapse, and reoperation rates. We believe that deep infection of the spine does not necessitate routine removal of existing implants and the decision to remove or retain implants should always consider the best evidence of the benefits and risks for so doing. Larger prospective studies should be conducted to validate this observation.

Conflicts of interest

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Acknowledgements

We would like to acknowledge M. Ramkumar for his assistance in submitting this manuscript. This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors, and no material support of any kind was received.

References

1. Glassman SD, Dimar JR, Puno RM, et al. Salvage of instrumented lumbar fusions complicated by surgical wound infection. *Spine* 1996;**21**:2163–9.

- Levi AD, Dickman CA, Sonntag VK. Management of postoperative infection after spinal instrumentation. J Neurosurg 1997;86:975–80.
- **3.** Richards BS. Delayed infections following posterior spinal instrumentation for the treatment of idiopathic scoliosis. *J Bone Joint Surg Am* 1995;**77-A**: 524–9.
- **4.** Viola RW, King HA, Adler SM, et al. Delayed infection after elective spinal instrumentation and fusion: a retrospective analysis of eight cases. *Spine* 1997;**22**:2444–50.
- Batson OV. The function of the vertebral veins and their role in the spread of metastases. Ann Surg 1940;112:138.
- Darouiche RO. Spinal epidural abscess. *New Engl J Med* 2006;355:2012–20.
 Wiley A, Trueta J. The vascular anatomy of the spine and its relationship to
- pyogenic vertebral osteomyelitis. J Bone Joint Surg Br 1959;**41**:796–809. 8. Karikari IO. Powers Cl. Revnolds RM. et al. Management of spontaneous spinal
- epidural abscess: a single-center 10-year experience. *Neurosurgery* 2009;**65**: 919–24.
- **9.** Brady RA, Leid JG, Calhoun JH, et al. Osteomyelitis and the role of biofilms in chronic infection. *FEMS Immunol Med Microbiol* 2008;**52**:13–22.
- Swanson AN, Pappou IP, Cammisa FP, et al. Chronic infections of the spine: surgical indications and treatments. *Clin Orthop Rel Res* 2006;444:100–6.
- **11.** Dennis HH, Seng TC, Fisher D, et al. Spinal implants can be inserted in patients with deep spine infection-results from a large cohort study. *Spine* 2017;**42**: E490–5.
- **12.** Chew FS, Kline MJ. Diagnostic yield of CT-guided percutaneous aspiration procedures in suspected spontaneous infectious diskitis 1. *Radiology* 2001;**218**: 211–4.
- De Lucas EM, Mandly AG, Gutiérrez A, et al. CT-guided fine-needle aspiration in vertebral osteomyelitis: true usefulness of a common practice. *Clin Rheumatol* 2009;28:315–20.
- Clark C, Shufflebarger H. Late-developing infection in instrumented idiopathic scoliosis. Spine 1999;24:1909–16.
- Schofferman I, Zucherman J, Schofferman J, et al. Diptheroids and associated infections as a cause of failed instrument stabilization procedures in the lumbar spine. Spine 1991;16:356–8.
- 16. Smith JS, Shaffrey CI, Sansur CA, et al. Rates of infection after spine surgery based on 108,419 procedures: a report from the Scoliosis Research Society Morbidity and Mortality Committee. *Spine* 2011;36:556–63.
- Weinstein MA, McCabe JP, Cammisa FP. Postoperative spinal wound infection: a review of 2,391 consecutive index procedures. J Spinal Disord Tech 2000;13: 422-6.
- Rayes M, Colen CB, Bahgat DA, et al. Safety of instrumentation in patients with spinal infection: clinical article. J Neurosurg Spine 2010;12:647–59.

- **19.** Tattevin P, Crémieux AC, Pottier P, et al. Prosthetic joint infection: when can prosthesis salvage be considered? *Clin Infect Dis* 1999;**29**:292–5.
- Ogden A, Kaiser M. Single stage debridement and instrumentation for pyogenic spinal infections. *Neurosurg Focus* 2004;17:E5.
- Pullter AF, Mohamed AS, Skolasky RL, et al. The presentation, incidence, etiology, and treatment of surgical site infections after spinal surgery. Spine 2010;35:1323-8.
- Kowalski TJ, Berbari EF, Huddleston PM, et al. The management and outcome of spinal implant infections: contemporary retrospective cohort study. *Clin Infect Dis* 2007;44:913–20.
- Lazennec JY, Fourniols E, Lenoir T, et al. Infections in the operated spine: update on risk management and therapeutic strategies. *Orthop Traumatol Surg Res* 2011;97:S107–16.
- Savage K, Holtom PD, Zalavras CG. Spinal epidural abscess: early clinical outcome in patients treated medically. *Clin Orthop Rel Res* 2005;439:56–60.
 Vinh DC, Embil JM. Device-related infections: a review. J Long Term Eff Med
- Vinh DC, Embil JM. Device-related infections: a review. J Long Term Eff Med Implants 2005;15:467–88.
- Zimmerli W, Moser C. Pathogenesis and treatment concepts of orthopaedic biofilm infections. *FEMS Immunol Med Microbiol* 2012;65:158–68.
- 27. Dimar JR, Carreon LY, Glassman SD, et al. Treatment of pyogenic vertebral osteomyelitis with anterior debridement and fusion followed by delayed posterior spinal fusion. *Spine* 2004;**29**:326–32.
- Stahl RS, Burstein FD, Lieponis JV, et al. Extensive wounds of the spine: a comprehensive approach to debridement and reconstruction. *Plast Reconstruc* Surg 1990;85:747-53.
- **29.** Carragee EJ. Instrumentation of the infected and unstable spine: a review of 17 cases from the thoracic and lumbar spine with pyogenic infections. *J Spinal Disord* 1997;**10**:317–24.
- Browder J, Meyers R. Pyogenic infections of the spinal epidural space. Surgery 1941;10:296–308.
- Hey HW, Thiam DW, Koh ZS, et al. Is intraoperative local vancomycin powder the answer to surgical site infections in spine surgery? *Spine (Phila Pa 1976)* 2017 Feb 15;42(4):267–74.
- Raney EM, Freccero DM, Dolan LA, et al. Evidence-based analysis of removal of orthopaedic implants in the pediatric population. J Pediatr Orthop 2008;28:701–4.
- An HS, Seldomridge JA. Spinal infections: diagnostic tests and imaging studies. *Clin Orthop Rel Res* 2006;444:27–33.
- **34.** Wagner C, Sauermann R, Joukhadar C. Principles of antibiotic penetration into abscess fluid. *Pharmacology* 2006;**78**:1–10.
- Bamberger DM. Outcome of medical treatment of bacterial abscesses without therapeutic drainage: review of cases reported in the literature. *Clin Infect Dis* 1996;23:592–603.