Professional Athletes Are at Higher Risk of Septic Arthritis After Anterior Cruciate Ligament Reconstruction

An Analysis of 4421 Consecutive Patients Including 265 Elite Athletes From the SANTI Study Group

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Background: Professional athletes are reported to be at greater risk of septic arthritis (SA) after anterior cruciate ligament reconstruction (ACLR) than the nonprofessional population. However, this finding has been controversial, and confusion has arisen in the literature owing to the underpowering of previous studies.

Purpose/Hypothesis: The purpose was to report the differences in the rate of SA after ACLR in a large series of patients and to perform pooled data analysis including previously published studies. The hypothesis was that professional athletes have a significantly higher risk of SA than nonprofessional athletes.

Study Design: Case-control study; Level of evidence, 3.

Methods: A retrospective analysis of prospectively collected data was performed. Patients who underwent ACLR between January 2009 and July 2017 (with a minimum follow-up of 12 months) were considered for study eligibility. The rate of SA was determined, and multivariate analysis was used to evaluate potentially important risk factors, including participation in professional sport. Furthermore, a literature search was performed, and data were extracted from all identified relevant studies. A pooled data analysis was performed to determine differences in the risk of SA between professional and nonprofessional populations.

Results: The current series comprised 4421 anterior cruciate ligament surgical procedures with 265 professional athletes. There were 15 cases of SA diagnosed over the study period (0.34%; 95% CI, 0.19%-0.56%). Ten cases occurred in professional athletes (3.8%; 95% CI, 1.82%-6.83%). The percentage of SA was 0.12% (95% CI, 0.04%-0.28%) in the nonprofessional population. Being a professional athlete was associated with a significantly increased risk of SA after ACLR (odds ratio, 21.038; 95% CI, 6.585-75.789; $P < .0001$). This finding was confirmed in the pooled data analysis comprising 11,416 patients including 1118 professional athletes (odds ratio, 5.03; 95% CI, 1.17-21.61).

Conclusion: Professional athletes are at greater risk of SA after ACLR than nonprofessional athletes. The results of previous studies may have been conflicting owing to underpowering. The current study confirms the elevated risk by using a large clinical series and pooled data analysis to avoid the limitations of previous studies.

Keywords: anterior cruciate ligament; septic arthritis; professional athletes; pooled analysis

Septic arthritis (SA) after anterior cruciate ligament reconstruction (ACLR) is a rare (0.14%-2.6%)[1,5,12,15,18,32] but potentially devastating complication. Consequences can include destruction of articular cartilage and the need to remove the graft and associated implants.[2,18,30,39] Sonnery-Cottet et al[32] and Ristić et al[27] both reported that SA after ACLR was observed with a significantly increased frequency in professional compared with nonprofessional athletes. This finding seems to be logical given the plethora of reports describing sporadic cases and outbreaks of infectious diseases among professional athletes, especially those involved in close physical contact and therefore at an increased risk of traumatic injury to the skin.[14,22,26,32,36] In contrast to these previous reports, 2 recent series did not identify a significant

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difference in the rate of SA between professional and nonprofessional athletes.[3,17] These conflicting findings may have been due to the fact that previous studies were underpowered.

Underpowering is an important problem to consider, especially in the design of studies that investigate diseases that occur with a low incidence. Appropriate methodologies to achieve adequate statistical power include the use of specific statistical models, very large study populations, and/or pooled data analysis.

The primary objective of this study was to report the differences in the rate of SA after ACLR in a large series of consecutive patients including professional athletes. The secondary objective was to perform a pooled data analysis, including previously published studies and the current series, to determine whether there is an overall greater risk of SA in professional athletes. The hypothesis of the current study was that professional athletes have a significantly greater risk of SA than do nonprofessional athletes.

METHODS

Study Design

Institutional review board approval (COS-RGDS-2019-01-001) was granted for the study, and all patients provided informed consent to participate. A retrospective analysis of prospectively collected data from the SANTI Study Group Database was performed. All patients who underwent ACLR by the senior surgeon (B.S.-C.) between January 2009 and July 2017 and had a minimum follow-up of 12 months were considered for study eligibility. Patients were excluded only if they had sustained a multiligament injury or underwent other major concomitant procedures (eg, high tibial osteotomy). To assess the influence of the type of sports activity on the risk of SA, patients were allocated to 1 of 3 groups based on the degree of skin contact expected in participation in their particular sport: no skin contact (eg, swimming, alpine ski), outdoor sports with skin contact (eg, soccer, rugby), and indoor sports with skin contact (eg, wrestling, futsal).

Preoperative Infection Prophylaxis Protocol

The same preoperative infection prophylaxis measures were undertaken for all patients. The affected limb was chemically epilated 1 week before ACLR. At 24 hours before surgery, patients took a shower with povidone-iodine. At the time of induction of anesthesia, a prophylactic intravenous cefazolin bolus of 2 g was administered. In the operating room, the skin was precleaned by brush application of alcoholic betadine. The surgeon then performed final preparation, again with alcoholic betadine, before setting up sterile surgical drapes.

ACLR Techniques

All procedures were performed with the patient under general anesthesia and with a tourniquet. Various types of autografts were utilized. Graft choice was based on patient factors and the evolving indications for performing a concomitant lateral extra-articular tenodesis over the study period. These factors included young age (<20 years old), participation in pivoting sports, high-demand athlete, high-grade pivot shift, lateral femoral notch sign, and Segond fracture.

Bone–Patellar Tendon–Bone Graft. A 2-incision technique was used. A 10 mm–diameter bone–patellar tendon–bone (BPTB) graft[9] was harvested with a 9 to 11 × 25–mm bone wedge from the tibia and a 10 × 15–mm bone plug from the patella. After the femoral and tibial tunnels were drilled with an outside-in technique, the BPTB autograft was passed from the femur to the tibia under direct arthroscopic vision. Press-fit graft fixation was obtained in the femoral tunnel, and tibial fixation was achieved with a 9-mm interference screw (Bio-Interference, Arthrex) placed anterior to the graft with the knee at 20° of flexion. When an associated extra-articular tenodesis was performed (with a BPTB ACL graft), it was done so with a 12 × 75–mm strip of iliotibial band, which remained attached to the Gerdy tubercle.[13]

Quadrupled Semitendinosus Tendon Graft. The semitendinosus tendon was harvested with an open tendon stripper, with the tibial insertion preserved to improve fixation and vascularity of the graft.[21] Femoral and tibial tunnels were drilled with an outside-in technique. Whenever possible, the ACL remnant was preserved. The graft was passed from the tibia to the femur. Tibial fixation was achieved with an interference screw. A TightRope button (Arthrex) secured the graft on the femoral side.

Combined ACL and Anterolateral Ligament Reconstruction With Hamstring Tendon Graft. The semitendinosus and gracilis tendons were harvested with an open tendon stripper. The tibial attachment of the semitendinosus tendon was preserved, but the gracilis was detached. The ACL graft comprised a tripled semitendinosus and a single strand of gracilis, the additional length of which formed the anterolateral ligament (ALL) graft. An outside-in femoral guide (Arthrex) was placed through the anteromedial

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portal, at the native ACL footprint. Externally, the guide was positioned proximal and posterior to the lateral epicondyle (at the femoral origin of the ALL), and a femoral tunnel was drilled over a guide wire. For the tibial part of the ALL reconstruction, two 4.5-mm-diameter sockets were drilled via stab incisions, one just posterior to the Gerdy tubercle and the second just anterior to the fibula head. These were then converted into a single tunnel with a right-angled clamp. A suture was then passed through the tunnel to create a loop for ALL graft passage. The hamstring tendon and ALL grafts were routed from the tibia to the femur through the knee and fixed on the tibial side with an interference screw. The ACL graft was then secured with an outside-in interference screw at 20° of flexion. The ALL graft was passed deep to the iliotibial band from the femur to the tibia and subsequently shuttled through the tibial bony tunnel and back proximally to the femur. On the femoral side, the sutures holding the ACL graft were then tied around the ALL graft, with the knee placed in full extension and neutral rotation.

Diagnosis and Management of Postoperative SA

The main outcome of interest was the occurrence of SA within 12 months after ACLR. All patients with clinical signs and symptoms of a deep infection were admitted to hospital for urgent physical examination and laboratory tests, including assessment of C-reactive protein, leucocyte count, and erythrocyte sedimentation rate. Patients who were suspected to have a deep infection underwent arthroscopic lavage with 9 L of normal saline solution and careful debridement of inflamed soft tissues. All knee compartments were inspected, and an assessment of graft integrity was made. Specimens of intra-articular synovial fluid and debrided tissue were sent for culture and antibiotic sensitivity.

All patients who underwent washout for infection received empirical postoperative antibiotic therapy, including a combination of intravenous penicillin and gentamicin. This was subsequently adapted according to bacterial identification and antibiotic sensitivities. Antibiotics were administered intravenously for 3 days and then given orally for 6 weeks.

Identification of Previous Studies for Pooled Data Analysis

A PubMed Search was conducted on January 31, 2019, with the search terms "septic arthritis," "professional athlete," and "anterior cruciate ligament." The search was limited to articles published in the English language. All relevant clinical studies were included, and the references of these articles were reviewed to identify additional eligible studies.

Statistical Analysis

All analyses were performed with SAS for Windows (v 9.4; SAS Institute Inc), with the level of statistical significance set at $P < .05$. Descriptive data analyses were conducted depending on the nature of the considered criteria. For quantitative data, this included number of observed values (and missing values, if any), mean, SD, median, first and third quartiles, and minimum and maximum. For qualitative data, this included the number of observed and missing values and the number and percentage of patients per class. Comparisons between variables were assessed with chi-square or the Fisher exact test for categorical variables and the Student test or Wilcoxon test for quantitative variables. Normality of variables was graphically confirmed. The primary endpoint was defined as the proportion of patients with SA that occurred within 12 months of ACLR. According to the anticipated low number of infections, the 95% CI was calculated with the binomial exact method. The characteristics of the studied population were described according to the group (professional or nonprofessional).

Multivariate logistic regression with penalized maximum likelihood was used to identify risk factors associated with SA. Factors initially considered were those selected as statistically significant at the 25% threshold or those of clinical interest in a first univariate approach. A stepwise descending strategy was applied from the initial full model to determine the most parsimonious one, by removing step-by-step all the non–statistically significant parameters and keeping the clinically relevant parameters and the confounding factors (if any).

To conduct a pooled data analysis, the incidence and odds ratios (ORs) for SA in professional and nonprofessional athletes were extracted from all included studies. Heterogeneity across publications was assessed with I^2, with substantial heterogeneity defined as I^2 between 60% and 90%. As data were extracted from a number of clinical studies, some differences were expected in the interventions and evaluations of the outcomes. Therefore, as recommended by Cochrane, a random Mantel-Haenszel effect model was conducted, allowing a conservative conclusion.[11] Results were presented by forest plots, and CIs were calculated with the binomial exact distribution.

RESULTS

The study population comprised 4421 ACLRs: 3930 (88.9%) primary reconstructions and 491 (11.1%) revision procedures (Figure 1). The mean delay between ACL rupture and surgery was 2 months in the professional athlete group (n = 265) and 13 months in the nonprofessional group (n = 4156). An extra-articular tenodesis was performed in 69.1% of professional athletes and 40.4% of the nonprofessional group. The mean age of the population was 29.1 years (range, 11-69 years). Tables 1 and 2 report demographic data and details regarding the surgical procedures performed. The distribution of sports commonly practiced by the study population is summarized in Figure 2.

Outcomes of Interest

Fifteen diagnoses of SA were made during the study period (0.34%; 95% CI, 0.19%-0.56%; 13 male, 2 female). Ten patients participated in outdoor sports involving skin

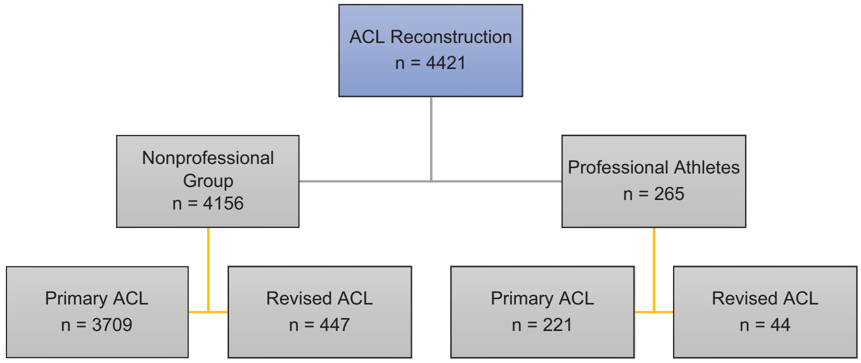

Figure 1. Flowchart. ACL, anterior cruciate ligament.

TABLE 1
Description of the Surgery According to the Group[a]

	Nonprofessional (n = 4156)		Professional (n = 265)		Total (N = 4421)		
	n	%	n	%	n	%	*P* Value
Graft							<.0001
BPTB	653	15.7	79	29.8	732	16.6	
HT	3431	82.6	177	66.8	3608	81.6	
Other	72	1.7	9	3.4	81	1.8	
EAT							<.0001
Isolated ACL	2475	59.6	82	30.9	2557	57.9	
Lemaire	218	5.2	42	15.8	260	5.9	
ALLR	1463	35.2	141	53.2	1604	36.3	
Revised ACL							.0033
No	3709	89.2	221	83.4	3930	88.9	
Yes	447	10.8	44	16.6	491	11.1	

[a]ACL, anterior cruciate ligament; ALLR, anterolateral ligament reconstruction; BPTB, bone–patellar tendon–bone; EAT, extra-articular tenodesis; HT, hamstring tendon.

contact (7 soccer players, 3 rugby players); 2 participated in indoor sports involving skin contact (1 basketball player, 1 wrestler); and 1 was a skier, 1 was a dancer, and 1 did not play sports (no skin contact). Of these 15 infected cases, 4 occurred in patients who had undergone revision ACLRs.

Ten cases of SA occurred in the professional athlete population (3.8%; 95% CI, 1.82%-6.83%). In contrast, the incidence of SA was 0.12% (95% CI, 0.04%-0.28%) in the nonprofessional group. Being a professional player was associated with a significantly increased risk of SA after ACLR (OR, 21.0; 95% CI, 6.6-75.8; $P < .0001$).

In the professional population, of those patients who developed SA, 8 had hamstring tendon autografts, and 2 had BPTB autografts. Ten of the 15 infections occurred in patients who underwent an extra-articular tenodesis as an additional procedure. All characteristics of infected patients are tabulated in Table 3.

Analysis of Potential Risk Factors

Univariate and multivariate analysis demonstrated that participation in outdoor sports involving skin contact did not have a significant influence on the risk of infections ($P = .40$), nor did undergoing a procedure combined with lateral tenodesis ($P = .09$) (Table 4). Other factors evaluated that were not associated with a higher risk of SA included the type of graft ($P = .82$), age ($P = .74$), sex ($P = .28$), body mass index ($P = .71$), and revision ACLR ($P = .06$).

Microbiologic Data of SA

All infections occurred in the first postoperative month (range, 4-28 days), except 1 in which the organism detected was *Propionibacterium acnes* at 149 days (Table 5).

Microorganisms identified in the infected populations were mainly *Staphylococcus* (5 *S caprae*, 6 *S aureus*, 1 *S epidermidis*). A second arthroscopic lavage was undertaken in 4 cases. Graft preservation was achieved in all cases.

Literature Search and Pooled Data Analysis

The PubMed search strategy yielded 4 eligible studies,[3,17,27,32] from which data were extracted and included in the pooled data analysis with the current study. The pooled study population comprised 11,416 patients,

TABLE 2
Patient Demographic Data[a]

	Nonprofessional (n = 4156)	Professional (n = 265)	Total (N = 4421)	*P* Value
Sex, n (%)				.0987
Male	3022 (72.7)	205 (77.4)	3227 (73.0)	
Female	1134 (27.3)	60 (22.6)	1194 (27.0)	
Age, y				<.0001
Mean ± SD	29.4 ± 10.3	23.8 ± 5.2	29.1 ± 10.2	
Median (IQR)	27 (21; 36)	23 (20; 27)	27 (21; 36)	
Range	11-69	13-38	11-69	

[a]IQR, interquartile range.

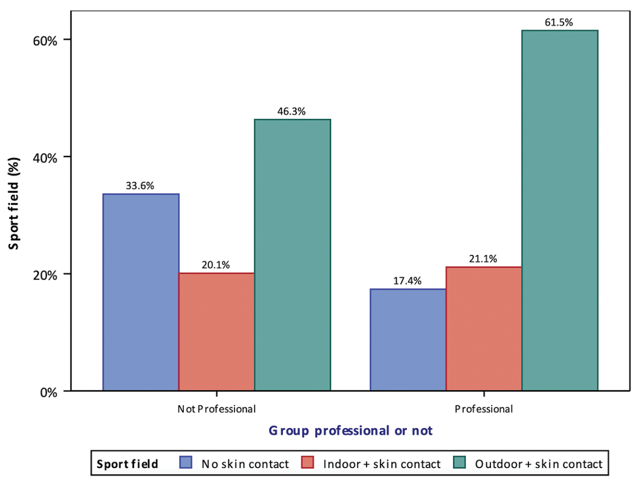

Figure 2. Distribution of patients participating in the defined sports categories.

including 1118 professional athletes. The incidence of SA and the associated CIs for all studies are reported in Table 6, and ORs are reported in Figure 3. Pooled data analysis of all studies demonstrated that professional athletes were at significantly greater risk of SA after ACLR than were nonprofessional athletes (OR, 5.03; 95% CI, 1.17-21.61) (Figure 3).

DISCUSSION

The main findings of this study were a confirmation of a significantly higher risk of SA after ACLR in professional athletes as compared with a nonprofessional population. This finding was observed in the current large clinical series and also the pooled data analysis including all previous relevant studies. Although an increased risk for professional athletes has already been reported, there are conflicting results in the literature.[3,17] The previous literature that supports the current finding of a significantly increased risk of SA in elite athletes includes Sonnery-Cottet et al,[32] who reported that SA after ACLR was observed more frequently in elite athletes (OR = 16, *P* = .0001), and also Ristić et al,[27] who reported a higher rate of SA in professional (1.9%) than nonprofessional (0.8%) athletes.

Bohu et al[3] and Krutsch et al[17] recently reported that there were no significant differences in rates of SA after ACLR between professional and nonprofessional athletes. Bohu et al concluded that this indicated that no special precautions are required in elite athletes undergoing ACLR. However, this conclusion must be interpreted with considerable caution. The authors reported a post hoc calculation demonstrating that the power of their study approached zero.

In the current study, a much larger cohort of professional athletes (n = 265) enabled adequate power to demonstrate a statistically significant increased risk for this population when compared with nonprofessional athletes (OR, 21.038; 95% CI, 6.585-75.789; *P* < .0001). This finding also held true in the pooled data analysis. Pooling of data is an appropriate methodology to achieve adequate statistical power when diseases with low incidence are investigated. With this methodology, a population of 11,416 patients including 1,118 professional athletes was achieved. In this pooled population, the professional athletes had a significantly higher risk of SA (OR, 5.03; 95% CI, 1.17-21.61).

Multivariate analysis of potential risk factors for the development of SA in the current clinical series was undertaken. This did not demonstrate an increased risk for patients undergoing concomitant extra-articular tenodesis. These findings are not consistent with the fact that additional procedures at the time of ACLR are generally reported to increase the infection rate.[15,29,40] Vadalà et al[37] demonstrated in a series of 1423 ACLRs (including 564 McIntosh procedures) that adding a lateral extra-articular tenodesis resulted in a significantly higher rate of infection when compared with an isolated intra-articular procedure (2.1% vs 0.3%, *P* = .03). These contradictory results in a large cohort of patients can be explained by the fact that the majority of extra-articular procedures performed in the current study were ALL reconstructions and that the only study that evaluated the risk of infection rates after ALL reconstruction demonstrated that it is extremely rare.[33] The reduced infection rate is considered to be an advantage of this percutaneous procedure over iliotibial band procedures that require a lateral incision.

Multivariate analysis also demonstrated that neither revision ACLR nor the type of sport was associated with an increased risk of SA. However, there was a nonsignificant trend toward more frequent deep infection in professional outdoor players participating in sports with skin contact than indoor athletes or those participating in sports

TABLE 3
Characteristics of Infected Patients[a]

Patient	Age, y	Sex	Sport	Graft	LT	Add Procedure
1 PA	25	M	Rugby	BPTB	No	No
2 PA	27	M	Soccer	HT	Yes (ITB)	No
3 PA	28	M	Wrestling	HT (R)	Yes (ITB)	MMs/LMs
4 PA	32	M	Soccer	HT	Yes (ALL)	LM-s
5 PA	24	F	Skiing	HT	No	No
6 PA	33	M	Soccer	HT	Yes (ALLR)	No
7 PA	23	M	Soccer	HT (R)	Yes (ALLR)	MM-s
8 PA	33	M	Rugby	HT	Yes (ALLR)	MM-s/LM-s
9 PA	23	M	Rugby	BPTB (R)	Yes (ITB)	No
10 PA	28	M	Basketball	HT (R)	Yes (ALLR)	No
11 NPA	44	M	No sport	HT	Yes (ITB)	No
12 NPA	37	M	Soccer	HT	No	MM-s/LM-r
13 NPA	24	M	Soccer	HT	No	No
14 NPA	28	F	Dance	HT	No	No
15 NPA	18	M	Soccer	HT	Yes (ALLR)	MM-s

[a]ALL, anterolateral ligament; ALLR, anterolateral ligament reconstruction; BPTB, bone–patellar tendon–bone; F, female; HT, hamstring tendon; ITB, iliotibial band; LM, lateral meniscus; LT, lateral tenodesis; M, male; MM, medial meniscus; NPA, nonprofessional athlete; PA, professional athlete; R, revision; r, resection; s, suture.

TABLE 4
Multivariate Analysis of Potential Risk Factors Associated With Septic Arthritis (N = 4338)[a]

Risk Factor	Comparison	Odds Ratio	95% CI	*P* Value
Group	Professional vs nonprofessional	21.038	6.585-75.789	<.0001
Time from injury, mo	$\leq$3 vs >3	1.585	0.452-6.827	.4850
EAT	ALL vs isolated ACL	1.033	0.312-3.532	.0967
	Lemaire vs isolated ACL	3.046	0.747-11.866	.1159
	ALL + Lemaire vs isolated ACL	1.323	0.458-4.225	.6112

[a]This logistic multivariate model with penalized maximum likelihood gives the probability of the risk of having septic arthritis versus not. Variables are included in the initial multivariable model if they are significantly associated with the dependent variable at a significance level of P = .25 or if the prognostic factors are known in the literature. Therefore, time between injury and surgery and whether patients underwent extra-articular tenodesis or revision ACL reconstruction were included. The final model is the result of a manual backward stepwise selection of variables with a significance level of P = .05. Estimation by profile likelihood for Penalized Firth logistic regression (R package logistf). ACL, anterior cruciate ligament; ALL, anterolateral ligament; EAT, extra-articular tenodesis.

without skin contact (of 10 elite athletes who were infected, 7 were outdoor; P = .40). In contrast, Krutsch et al[17] identified that patients performing summer outdoor sports were at significantly higher risk than winter sports athletes (soccer, 1%; skiing, 0%; P = .02). A potential explanation for these findings is the observation that higher skin infection rates are reported in professional athletes involved in frequent skin-to-skin contact sports, such as soccer or rugby.[4,26,36] Skin abrasions and turf burns, often left uncovered, provide an opportunity for pathogenic bacterial colonization.[10,16] This is further evidenced by the fact that other studies demonstrated that athletes in contact sports become more frequently colonized with *S aureus* than do participants in noncontact sports.[14,20] Krutsch et al reported additional potential explanations, including differences in colonization of skin bacteria owing to differences in temperature and excretion of sweat depending on participation in different sports. Further study is required to better understand these complex factors.

SA after ACLR is mainly caused by coagulase-negative *Staphylococcus* organisms.[39] Despite the fact that these organisms produce biofilms, many authors have confirmed successful graft and implant retention after ACLR-associated SA. While the involvement of *S aureus* in common infections in the athletic population is well known[6,14,22,26] and is most frequently identified as the cause of SA after ACLR, *S caprae* graft contamination and deep infection have also been previously reported.[7,23,32] The latter organism is important to note because it is less well studied and the mechanism of biofilm formation is not clearly understood. The clinical importance of this is that Seng et al[31] reported a significantly higher risk of recurrent orthopaedic infection with *S caprae* if implant removal was not performed. However, the study included all types of orthopaedic procedures and did not specifically evaluate SA after ACLR. In the current study, *S caprae* was identified as the infecting organism in 5 of 15 cases. Of these cases, 2 were refractory to a single arthroscopic washout but were successfully managed after a second

TABLE 5
Bacteriological Data and Management[a]

Patient	Time to Presentation, d	Symptoms	CRP, mg/mL	Organism	Arthroscopic Lavage, n	Antibiotic
1 PA	14	T, S	50	SC	1	RIF/OFL
2 PA	149	Chronic S	140	PBA	1	MOX
3 PA	11	T, S	100	SA MS	1	ORB
4 PA	13	T, PF	120	SC	2	RIF/OFL
5 PA	16	T, S, PF	115	SA MS	1	ORB
6 PA	7	T, S, PF	100	SE MS	1	RIF/OFL
7 PA	8	T, S	250	SC	1	RIF/OFL
8 PA	4	T, S	252	SM	1	MOX/BAC
9 PA	12	T, S, PF	280	SC	1	RIF/OFL
10 PA	4	T, S, PF	387	EC	2	CEF/OFL
11 NPA	24	T, PF	150	SA MS	1	RIF/OFL
12 NPA	8	T, S, PF	253	SC + SCA	2	RIF/OFL
13 NPA	15	T, PF	104	SA MS	2	RIF/OFL
14 NPA	17	T, S, PF	184	SA MS	1	OFL/FUS
15 NPA	28	T, S, PF	118	SA MS	1	RIF/OFL

[a]BAC, Bactrim; CEF, Cefepime; CRP, C-reactive protein; EC, *Enterobacter cloacae*; FUS, fusidic acid; MOX, moxifloxacin; MS, methicillin sensitive; NPA, nonprofessional athlete; OFL, ofloxacin; ORB, Cloxacilline; PA, professional athlete; PBA, *Propionibacterium acnes*; PF, painful flexion; RIF, rifampicin; S, swelling; SA, *Staphylococcus aureus*; SC, *Staphylococcus caprae*; SCA, *Staphylococcus capitis*; SE, *Staphylococcus epidermidis*; SM, *Serratia marcescens*; T, temperature >38°C.

TABLE 6
Incidence of Septic Arthritis and Associated 95% CI Derived From Included Studies

	Septic Arthritis, n	Patients, n	95% CI (Septic Arthritis Proportion)
Current study			
Overall population	15	4421	0.19-0.56
Nonprofessional population	5	4156	0.04-0.28
Professional population	10	265	1.82-6.83
Bohu et al[3]			
Overall population	7	1809	0.16-0.80
Nonprofessional population	7	1719	0.16-0.84
Professional population	0	90	0.00-4.02
Krutsch et al[17]			
Overall population	17	1809	0.55-1.50
Nonprofessional population	15	1609	0.52-1.53
Professional population	2	200	0.12-3.57
Ristić et al[27]			
Overall population	17	1425	0.70-1.90
Nonprofessional population	8	950	0.36-1.65
Professional population	9	475	0.87-3.57
Sonnery-Cottet et al[32]			
Overall population	12	1957	0.32-1.07
Nonprofessional population	7	1869	0.15-0.77
Professional population	5	88	1.87-12.76

washout and exchange of tibial screw. Graft retention was achieved in all cases. However, this highlights that further study is required to better understand the role of *S caprae* in SA and to investigate a potentially increased risk of failure of a primary washout when compared with more common infecting organisms.

On the basis of previous studies,[28,34,39] our therapeutic protocol for infection consisted of arthroscopic irrigation, graft preservation, and joint debridement combined with antibiotic therapy. A recent study demonstrated that arthroscopic lavage should be the first-line management of SA rather than open irrigation, owing to the association with a lower rate of bleeding and adverse events.[8] In the current study, a second arthroscopic washout was performed in 4 cases because of persistent infection despite antibiotic treatment. In these patients, tibial screw exchange was also performed because of the possibility of bacterial biofilm formation being responsible for the failure of the first arthroscopy.[35]

On the basis of the potentially devastating effects of SA and the results of the current study, we disagree that special precautions are not required in professional athletes. Since

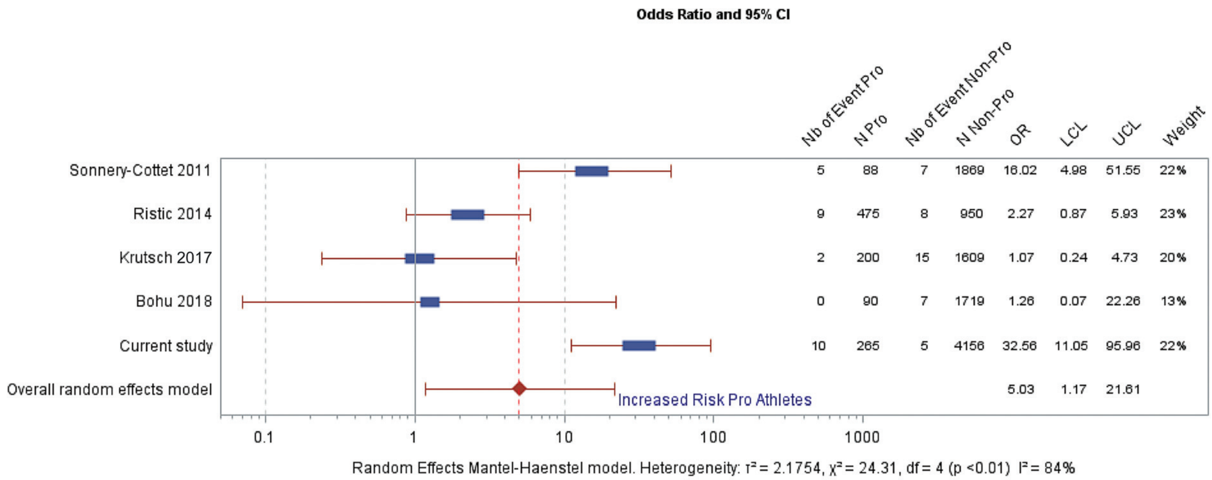

Figure 3. Forest plot demonstrating relative risk of septic arthritis in each study and pooled summary estimate. LCL, lateral collateral ligament; Nb of Event, infection in; OR, odds ratio; Pro, professional athlete; UCL, ulnar collateral ligament.

Sonnery-Cottet et al[32] reported in 2011 an overall SA rate of 0.37% after ACLR and an elevated risk of 5.7% in the professional athlete population, we have continuously sought to reduce the risk of deep infection in our patients. Since July 2017, we have introduced the topical application of vancomycin to the graft. This strategy was demonstrated to be associated with promising outcomes by multiple authors.[19,23-25,38] Pérez-Prieto et al[23,24] did not observe any bacterial growth after using a 5-mg/mL vancomycin solution. These results were supported by Phegan et al,[25] who confirmed that presoaking the autograft during ACLR reduces the postsurgical infection risk. These authors reported that in their consecutive series of 1300 ACLRs, there were no cases of infection with this preventive technique.

Despite the large comparative cohort analysis, there are several limitations to the current study. The retrospective nature of the analysis, combined with the small rate of the primary endpoint, limits the interpretation of the findings. The variability in ACLR techniques used, as well as the different extra-articular procedures and associated injuries (eg, meniscal or articular cartilage), limits the generalizability of the findings. All patients underwent an exclusive preoperative asepsis with alcoholic betadine. Perhaps the use of another substance or a complementary asepsis would affect the results, reducing infection rates. The strengths of this study are a large cohort of ACLRs including a considerable number of professional athletes.

CONCLUSION

Professional athletes are at greater risk of SA after ACLR than are nonprofessional athletes. The results of previous studies may have been conflicting because of underpowering. The current study confirms the elevated risk by using a large clinical series and pooled data analysis to avoid the limitations of previous studies.

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