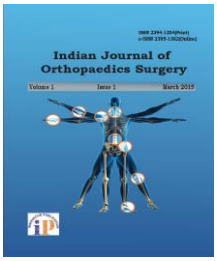

ISSN 2395-1354(Print)
e-ISSN 2395-1362(Online)

Indian Journal of Orthopaedics Surgery

IJOS

POST ANTERIOR CRUCIATE LIGAMENT RECONSTRUCTION INFECTION: REVIEW OF LITERATURE

Sanyam Chaurasia[1,*], Georgios Karnatzikos[2], Alberto Gobbi[3], Vivek Gupta[4]

[1,4]Department of Orthopaedics, Irene Swasthik Hospital, A-9, Raghuvir Enclave, Dhichaun Chowk, Nangloi Stand, Najafgarh, New Delhi, India
[2,3]OASI Bioresearch Foundation,Via Amadeo 24, 20133, Milan, Italy

***Corresponding Author:**
E-mail: knee_shoulderclinic@yahoo.com, sanyamchaurasia85@gmail.com

Abstract
Purpose: *The purpose of this study was to review and summarize the literature and suggest the probable most effective protocol in the management of post anterior cruciate ligament reconstruction (ACLR) infection.*
Methods: *We reviewed 16 studies (Level 1- Level 4) published between January 2000 and December 2013, by a thorough search in PUBMED, MEDLINE and EMBASE databases.*
Results: *There were 246 cases of infection amongst 35,795 ACLR making the proportion of infection 0.68% (range- 0.14-2.6%). The mean time of onset of infection from index surgery was between 7.5 days to 32 days. The most common organism was Coagulase-negative Staphylococci (CNS) followed by Staphylococcus Aureus. Optimal outcome was reported in most studies using serial arthroscopic lavage and intravenous antibiotics with graft retention as a prime protocol.*
Conclusion: *Septic arthritis after ACLR is a rare and disastrous complication which can be successfully managed by early diagnosis and arthroscopic debridement with a proper protocol.*
Level of Evidence: *Review Article; Level 4*.

Introduction

Infection after anterior cruciate ligament reconstruction (ACLR) is relatively uncommon[1-18]. However, it comes as a disaster due to its devastating consequences such as graft failure, arthrofibrosis, articular cartilage loss, or may even require graft removal.[14,19,20] According to an ongoing study in the United States, the incidence of ACL injury is roughly one in 3,000 people per year and an estimated 200,000 ACLR are performed annually.[21] Most of the intra-articular post-ACLR knee infections are acute (< 2 weeks) or sub-acute (> 2 weeks- 2 months).[13] Full-thickness cartilage lesions, diffuse chondral thinning, degenerative arthritis and osteomyelitis are severe sequelae of knee sepsis.[12, 13] While cartilage loses more than half of its glycosaminoglycan and collagen within 7 days from the onset of infection[13] , early diagnosis and prompt aggressive treatment is crucial for optimal outcome.

Present study investigated literature with the aim to provide data on the incidence, risk factors, causes and important investigation parameters such as prognosis and complications after post ACLR infection. In addition, we summarized the ongoing research articles and gave most efficient treatment protocol to enhance in the pool of evidence in this concern; this will encourage more close and appropriate adherence to clinical guidelines to improve quality care for patients and vise versa will reduce the incidence of post-ACLR infection.

Review Material

By a thorough search in PUBMED, MEDLINE and EMBASE databases, we summarized 16 articles between January 2000 and December 2013, on post ACLR

infection. Case reports, animal and/or experimental studies were excluded from this review. Relevant data were tabulated for easier reference. From the included studies, 2 studies[1,3] were level-2, 6 studies[4,5,6,8,11,15] were level-3 and 9 studies[2,7,9,10,12,13,14,15,16] were level-4 studies (Table 1).

Table 1. Characteristics, Data, Incidence and Graft Specific Incidence.

Serial no.	Study	Journal	Y/LOE	Period(years)	ACLR	n/ %	MA	M/F%	Al/H/B%	Rat. H/B
1.	Abdel-Aziz et al[1]	Int.Orth.	2014/L-2	NA	2,560	24/0.94	NA	NA	NA	NA
2.	Claramunt et al[2]	KSSTA	2013/L-4	4	810	15/1.8	33.5	76/24	0/80/20	NA
3.	Maletis et al.[3]	AJSM	2013/L-2	5	10626	51/0.48	29.5	64/36	41/31/28	8.2
4.	Sonnery-Cottet et al.[4]	AJSM	2011/L-3	5	1957	12/0.61	24	100/0	0/34/58	NA
5.	Monaco et al[5]	PSM	2010/L-3	9	1232	12/0.97	NA	NA	NA	NA
6.	Barker et al.[6]	AJSM	2010/L-3	5	3126	18/0.58	34.1	78/22	0.44/1.44/0.49*	3.34
7.	Wang et al.[7]	ARTH	2009/L-4	11	4068	21/0.52	28.6	86/14	1.11/0.57/0*	2.5
8.	Katz et al.[8]	ARTH	2008/L-3	5	801	6/0.75	29	NA	1.2/0.6/0*	NA
9.	Schulz et al.[9]	AJSM	2007/L-4	10	1872	24/0.78	32.5	79/21	0/29/50	NA
10.	Van Tongel et al.[10]	AJSM	2007/L-4	9	1736	15/0.51	31	93/7	0/100/0	NA
11.	Binnet et al.[11]	ARTH	2007/L-3	8	1231	6/0.48	24.5	100/0	0.64/0.29[1]*	NA
12.	Judd et al.[12]	ARTH	2006/L-4	8	1615	11/2.6	28	72/28	0/100/0	NA
13.	Fong et al[13]	AAM	2004/L-4	3	472	7/1	23	100/0	0/100/0	NA
14.	Burks et al.[14]	AJSM	2003/L-4	11	1918	8/0.42	27	75/25	0/88/12	NA
15.	Schollin-Borg et al.[15]	ARTH	2003/L-3	3	575	10/1.7	28.3	80/20	0/40/60	NA
16.	Indelli et al[16]	CORR	2002/L-4	6	3500	6/0.14	32.5	83/17	NA	NA

Abbreviations:Y/LOE, Year/Level of Evidence; ACLR, Number of Anterior Cruciate Ligament. Reconstruction performed; n/%, Number of Infections/Incidence; MA, Mean Age in Years amongst patients with post ACLR infection; M/F, Male/Female amongst patients with post ACLR infection; Al/H/B, Allograft/Hamstring/BPTB graft Amongst Infected Cases; Rat. H/B, Ratio of Incidence of Infection in Hamstring and BPTB graft; Int. Orth, International Orthopaedics; KSSTA, Knee Surgery, Sports Traumatology and Arthroscopy; AJSM, American Journal of Sports Medicine; PSM, Physician and Sports medicine; ARTH, Arthroscopy; AAM, Annals Academy of Medicine; CORR, Clinical Orthopaedic Related Research; NA, Not Available
*Incidence of Infection in Allograft/Hamstring/BPTB grafts, [1]*No Allografts used.

Post ACLR Infection Incidence

There were 246 infections amongst 35,795 ACLR.[1-16] Average incidence was 0.68% (range- 0.14-2.6%), while the reported incidence in 2 systematic reviews was between 0.3 and 1.7%[17] and 0.6%[18], respectively. Average age was between 23-34[1-16]. Incidence of infection in allograft [6,7,8], hamstring and BPTB grafts [6,7,8,11] was between 0.44-1.2%, 0.57-1.44% and 0-0.49% respectively. Hamstring autografts were more frequently infected (29-100%) while BPTB autografts and allografts were found in 0-60% and 0-41% of the total reported cases[1-5,9,10.12-15]. The ratio of incidence of infection after autologous hamstring and BPTB graft was between 2.5-8.2, however it was provided only in 3 studies[3,6,7].

Pathogenesis and Risk Factors

Grafts have been reported in some studies as the nidus for infection because they act as a foreign body[11,12,16], while other studies have attributed hematoma at tibial tunnel end as a origin of infection in sub-acute and late cases (Table 2). [11,12,13,15] The reported risk factors included: high body mass index[3] (BMI) > 30kg\m^2, infection prone hamstring graft[2,3,6,22], previous knee surgery (arthroscopy or open) [12,13,14,23], concomitant surgery[13,15], short hamstring tendon with more suture material[11], post-operative effusion[11], subcutaneous position of the metallic post/washer/braided suture construct[17], different implants[12] and the use of intra-operative/intra-articular steroids[15] (Table 2). Other causes postulated were persistent communication between skin and joint by sutures[11], unsterile metal part and the rubber membranes on the suture clamps which cannot be sterilized satisfactorily despite sterilization performed in certified autoclaves[15], environmental contamination of surgical equipments or hospital stuff in studies[24,25] demonstrating epidemics and adjacent tunnel osteomyelitis in persistent cases[11]. It has been also hypothesized that if microorganism of normal skin flora was cultured from the joint, then inoculation must have occurred at the time of surgery, or shortly thereafter through femoral or tibial site.[12]

Table 2. Risk factors and Pathogenesis

SRN	Risk factors	Pathogenesis	Causes
2,3,6,24	Hamstring Graft	-	Multifilament suture used to prepa harvest site and tunnel site coir tendon is us
3	High BMI	-	-
11,12,16	-	Grafts act as foreign body	-
12	-	-	If normal skin flora cultured, ther occurred at the time of surgery or s femoral or tibia
11,12,13,15	-	Hematoma at tibial tunnel end in subacute and late cases	Meniscal repairs/arrows or sutur material
12,14	-	Postinfectious synovitis and inflammatory mediators causes chondrolysis	-
12,13,14,25	Previous knee surgery(arthroscopy or open) in 53-75% patients	-	-
13,15	Concomitant surgery in 43-50% patients	-	-
10,16	No risk with concomitant surgery	-	-
11	Short hamstring tendon with more suture material; post-op effusion	-	Persistent Communication betv sutures
17	Subcutaneous fixation material (position of the metallic post/ washer/braided suture construct)	-	-
12	Different implants increases risk by 3.2-4.5 times.	-	-
15	Arthroscopy combined with the use of intra-operative, intra-articular steroids	-	Metal part and the rubber membra cannot be sterilized satisfactori performed in certified
26,27	-	-	Environmental conta surgical equipments or hospital epidemic
11	-	-	Adjacent tunnel osteomyelitis

Abbreviations: SRN, study reference numbers; BMI, Body mass index

Clinical Signs and Symptoms

The reported mean time of onset (MTO) of infection from index surgery was between 7.5 to 61.7 days (Table 3). A recent systematic review [18], reported an average of 16.8 ± 10.5 days (114/123, 92.6 %), with most infections in acute or sub-acute phases symptoms. Classical clinical features given were: acutely swollen painful joint, limited range of motion, sudden increase of pulsatile knee pain, rapidly increased and persistent effusion, incision drainage, local erythema, warmth and intermittent fever (usually over 38°C).[7,13,14,16] Other features included hyperemic with serous or purulent discharge[11] and indolent presentation (60% missed on first visit)[15], excluding large hematomas simulating acute (Table 3).

Table 3.Clinical Presentation and Laboratory Values

SR N	Clinical Presentation	MTO Days(range)	Mean investigation values(range) at the time of diagnosis ESR-mm/hr, CRP- mg/L, TLC(× 10^9/L)	Microrganism Cultured- n(%)
1	Classic Features[7,13,14,16] • Acutely swollen painfull joint. • Limited range of motion. • Sudden increase of pulsatile knee pain. • Rapidly increased and persistent effusion. • Incision drainage. • Local erythema. • Warmth. • Intermittent fever usually over 38°C. • Hyperemic with serous or purulent discharge[11]. • Indolent presentation(60% missed on first visit)[15]. • Exclude large hematomas simulating acute symptoms; laboratory investigations needed for confirmation.[12]	12 (5-45)	NA	CNS-7(29), MSSA- 7(29), OS-7(30), NG- 3(12)
2		24(7-35)	NA	CNS-10(66), MSSA-2(19), MRSA-1(7), P-1(7)
3		20(12-30)	NA	Deep- CNS-11(32), SA-8(24),NG -9(26),OS-6(18) Superficial- SA-10(59), CNS-1(6), OS- 5(30), NG- 2(12)
4		15(2-37)	NA	CNS-11(92), P-1(8)
5		NA	ESR-76, CRP-7.4, TLC-8.6	CNS- 11(92), OS- 1(8)
6		32 (5-205)	ESR- 79.8(15–118), CRP - 17.5(4.5–38.1), TLC-9.5(6.0 13.8),TLC(joint) - 11.5	MSSA-6(33), MRSA-4(22),P-2(11), NG-6(33)
7		16(5-32)	ESR-59.7 (9-108), CRP-8.3 (4.1-17.8), TLC-9.1 (4.2-15.2)	CNS-11(52), SA-2(9), OS-3(14), NG-5(25)
8		16(6-33)	ESR-65.7(30-124), CRP-27.7(11.2-70.1), TLC-9.6(7.3-13-7)	CNS-6(75),NG-2(25)
9		61.7 (5–196)	CRP-4.8(0.5-17.4), TLC-9.5 (3.3–17)	CNS-5(21), SA-12(50),OS-3(16), NG- 4(17)
10		10.9(3-1year3months)	ESR- 70.6(40–110), CRP-146.6(12.9–316.3), TLC-8.1(6.0–12.8).	CNS-8(53), MSSA-1(7), OS-6(40), NG-0(0)
11		22(14-35)	ESR- 51(17 to 80). CRP-29.7(17-42), TLC- more than 10.	SA- 3(50), OS-1(17), NG-2(33)
12		22.9(6-45)	ESR-67, CRP-14, TLC-9.8(4.9-17.7),TLC (joint)-5.2	CNS-8(72), SA-1(9),P-1(8),OS-1(8)
13		25(7-35)	ESR-72(10-95),CRP-123(25-215), TLC-11.7(10-16)	SA-4(57), P-3(43)
14		19	NA	NA

Abbreviations: MTO, Mean Time of Onset; ESR, Erythrocyte Sedimentation Rate; CRP, C-Reactive Protein; TLC, Total Leucocyte Count; CNS, Coagulase Negative Staphylococcous; MSSA, Methicillin Sensitive Staphylococcus Aureus; MRSA, Methicillin Resistant Staphylococcus Aureus; SA, Staphylococcus Aureus; P, Propionibacterium; OS, Other Species; NG, No Growth; NA, Not Available

Diagnostic Evaluation

Laboratory investigations are required for infection confirmation.[12] The average erythrocyte sedimentation rate (ESR) ranged between 51 -80 mm/hour and average C-reactive protein(CRP) between 4.8-146.6 mg/l, with total leucocyte count (TLC) in blood between 8.1- 11.7 × 10^9/l.[1,5-13,15] In only 2 studies the average TLC in synovial fluid was provided and was 11.5 and 5.2 × 10^9/l, respectively.[6,12] One systematic review[17] reported that the ESR and CRP were markedly increased in 90% (50–100%) and 95% (67–100%) of patients, respectively, and if the CRP level does not decrease to nearly normal by 2 weeks postoperatively, or there is a secondary rise, infection should be suspected. Complete CRP levels normalization is seen after 2–12 weeks.[10,13] Blood culture is less sensitive in diagnosis.[17] Synovial fluid culture and staining revealed Coagulase Negative Staphylococcus (CNS), Staphylococcus Aureus (SA) and Propionibacterium, in nearly all studies. Among CNS, Staphylococus Epidermidis was the most common pathogen. Other species[1-16] included non/hemolytic Streptococcus, Peptostreptococcus, Klebsiella, Enterobacter species, Erysipelothrix Rhusiopathiae, Fungus, Mycobacterium Tuberculosis and other anaerobic or gram-negative organisms.

Treatment Protocol

Serial arthroscopic lavages and intravenous antibiotics with graft retention, remains the most efficient treatment protocol in most studies.[18] Basic guidelines were as follows:

- *Empirical intravenous antibiotic therapy* at the time of presentation: intravenous (IV) ceftazidime (2g/8hr) and vancomycin (1g/12hr)[2], or Cafazolin[11], or flucloxacillin (6 × 1 g/day) and gentamycin (320 mg/day). [12] *Pathogen-specific antibiotics* after culture; additional cultures during operation(s). IV antibiotics changed to culture sensitive oral antibiotics as soon as the CRP levels had nearly normalized (<1 mg/mL)[7] for 6 weeks or until normalization of clinical and lab parameters. Average duration of antibiotics ranged for IV between 17.3days-6weeks followed by oral up to 3.2months in 9 studies(Table 4a)[1,2,7,10-13,15,16]. Delayed diagnosis of more than 7 days or SA infection required a longer duration of antibiotic therapy and increased the likelihood for graft removal and restricted range of motion.[18]
- *Arthroscopic debridement and lavage:* extensive arthroscopic removal of necrotic tissue with a shaver, as near total synovectomy as possible, debridement of fibrinous exudates of graft's surface, arthroscopic lysis of fibrous adhesions and extensive pulsatile lavage with 10-15 lit of saline. Additional lavage if clinical and laboratory parameters are not satisfactory. Average number of arthroscopic lavage per person ranged between 1 to 3 in 11 studies(Table 4a).[1,2,6,7,9-13,15,16] 66-100% of patients retained graft and graft removal resulted in 0-34% of patients in 12 studies[1,2,5-8,10-13,15,16] excluding the study[9] which dealt with complicated cases(Table 4a). This protocol remained more or less the same in nearly all studies supporting graft retention.[1-8,10-13,15,16]
- *Concomitant open incision and drainage*, through the old scars of arthroscopy and meniscal repair portals, at the same time of the arthroscopic lavage, in cases of complicated or infected wounds, in order to avoid extra-articular fluid collection and to eliminate the infection.[10] The wounds are left open with only a sterile dressing applied, in order to promote secondary wound closure.[9] Continuous irrigation drains in the joint may be used for 2 days.[15] Average hospital stay ranged between 17.3 to 27.2 days in 3 studies(Table 4a)[10,11,13].
- *Immediate graft removal* should be considered if the graft is unstable resulting in nonfunctional ligament during clinical examination and arthroscopic evaluation and causing instability or block. The same if the graft is impregnated by a tenacious, thick purulent exudation which could not be removed without graft damage and the articular cartilage is appeared soft and swollen or possible

bony involvement of the tibia and femur is existed. Furthermore, if the diagnosis is SA and the treatment has been delayed for more than a few days after the onset of the symptoms.[10,16,18] During repeated

- arthroscopic debridement the graft removal is suggested in case of resistant infection or the joint has persistent positive cultures and the clinical response to treatment is poor.[2,14,16] After graft removal, the tunnels should be debrided.[9]

Table 4a. Arthroscopic Lavage and Graft Retention at final follow-up

SRN*	Arthroscopic Lavage (Average No /person)	Graft Retention/removal No (%)
1	3	24(100)/0(0)
2	1.3	14(93)/1(7)
5	NR	14(100)/0(0)
6	1.5	13(72)/5(28)
7	1	21(100)/0(0)
8	NR	5(83)/1(17)
9**	2.2	7(29)/17(71)
10	1.9	11(73)/4(27)
11	2.66	6(100)/0(0)
12	2.4	10(91)/1(9)
13	1.4	4(100)/0(0)
15	1	10(100)/0(0)
16	1.3	4(66)/2(34)

Abbreviations: SRN, Study Reference numbers; NR, not reported; No, number of patients
* Studies 3, 4 and 14 were excluded because they followed different protocols
**dealt with complicated cases

Table 4b. Clinical Outcome

SRN*	MFU[a](months)	Prognosis at final follow-up						
		Lachman (-) (%)	KT-diff, (mm)	Lysholm		IKDC(obj)	TEGNER	
				PRE	FFU		PREI	FFU
1	60	91	5 in 2PT	-	-	-	7	5.5
2	39.3	73.3	1.3	90.7	77.7	**	-	-
5	38	100	2.5	-	96	14 (A/B)	-	-
9	66	-	-	-	65.6	8 (A/B),16 (C/D)	6.1	3.8
10	58	54.5	1	-	83	9 (A/B), 2C	NR	5.6
11	102.5	33(66-G1)*	2.7	-	81.1	-	NR	NR
12	22	-	-	-	71.6	5 (A/B), 4 (C/D)	NR	NR
13	11.7	100	-	-	-	5A,2C	7	5.14
15	36	100	1.35(2.95)	85.4	74.9	-	8.3	5.3
16	36	-	-	-	-	3 (A/B),1C	NA	NA

Abbreviations: SRN, Study reference numbers; MFU, Mean Follow Up; L-neg, Patients with negative Lachman test with retained graft; G, grade of laxity; KT diff, Mean difference by KT-1000 arthrometer between infected and control group; PT, patient; PRE, preoperative score; FFU, final follow up; IKDC(obj.), international knee documentation score (objective scale); PREI, preinjury; N, normal; NN, nearly normal; AB, abnormal; SA, severely abnormal; NA, not available; NR, not reported

*includes studies reporting outcomes with arthroscopic debridement and graft retention as their protocol

**subjective scale 86.6(Control),70.4 (infection)*

Table 4c. Average duration of antibiotics, Average hospital stay and Complications in included studies

SRN*	Average duration of antibiotics	Average hospital stay(days)	Complications					
			Flexion deficit-N,degrees	Extension deficit N,degrees	Graft rupture	Osteoarthritis(Joint space narrowing or osteophytes,Crepitus)	Graft removal (nonviable)	Osteomyelitis
1	NA	NA	5	0	3	2	0	0
2	IV4W,O2-4W	NR	0	0	0	0	1	0
7	IV-19.4	NR	NR	NR	NR	NR	NR	NR
9	NR	NR	24,20	24,3	0	1	17	0
10	IV-24.6D, T-3.2M	27.2	3,6-15	2,3-5	2	3	1	0
11	IV-3W	19.5	6,6	0	0	1	0	1
12	IV-4W,O- 3-4W	NR	2,20-30	0	0	3	1	0
13	IV-17.3D, O-4-6W	17.3	7,20	0	0	0	0	0
15	IV+O- 4-12	NR	0	0	0	5	0	0
16	IV-6W	NR	0	0	0	1	1	0

Abbreviations: SRN, Study Reference numbers; N, Number of patients; NA, not available; NR, not reported; IV, intravenous; O, oral; W, weeks; D, days; M, months; T, total
*includes studies reporting these parameters with arthroscopic debridement and graft retention as their protocol

Outcome and Complications

Ten studies[1,2,5,9-13,15,16] (Table 4b), reported the follow-ups along with the use of more or less the above proposed most effective treatment protocol and gave satisfactory results. Lachman's test was negative in 54.5 - 100% of the patients, at a mean final follow-up between 11.7 - 60 months[1,2,5,10,11,13,15]. Mean KT-1000 arthrometer difference between control and infected group ranged from 1 - 5 mm at an average of 21 - 102.5 months [1,2,5,10,11,15]. Average Lysholm score ranged between 65.6 - 96, at 22 - 102.5 months[1,2,5,9-12,15], while average Tegner score was between 3.8 - 5.6 [1,5,9,10,13,15] and preinjury average Tegner score was 6.1-8.3 [9,13,15]. Out of 75 infected patients, 43 patients had normal or near normal and 25 patients had abnormal or severely abnormal scores in International Knee Documentation Committee (IKDC) subjective scale[5,9,10,12,13,16]. Out of 142 infected patients, 47 patients had a flexion deficit ranging from an average of 6 - 30°, 26 patients had extension deficit ranging from 3-5°.[1,2,5,9,10-13,15,16] Other complications are reported in Table 4c.

Discussion

Infection following ACLR is not common; the reported incidence of infection is between 0.14-2.6%. *Staphylococcus Epidermidis,* a CNS remains the most common species consistent with post ACLR infection (Table 3). Currently, post-ACLR infection is considered multifactorial. As ACL grafts act as a foreign body, pathogenesis behind this factor is universal. Also, we used autologous hamstring tendon as a graft which may be more prone to get infected as compared to BPTB graft as it has 2.5-8.2 times more chances to get infected (Table 1). Additional literature reports more chances of infection during preparation in hamstring grafts.[24] Another proposed predisposing factor is the extended operative time of DB compared to single bundle (SB) ACLR.[25] However, another study[26] showed no significant differences concerning the time for operation between the DB and SB groups and there is no study to our knowledge in literature confirming that rate of septic arthritis is more in DB than in SB groups. Most of the untabled literature[27,28] reports same clinical symptoms and similar range laboratory parameters as reported in Table 3. Apart from risk factors enumerated in Table 2, literature[27,28,29] includes: operative time, tourniquet inflation time, contaminated sterile inflow cannula, concomitant open surgical procedures, increased foreign body load (suture material or hardware), and use of a drain. The most effective treatment protocol proposed was irrigation and debridement with graft retention as infection subsides usually in 1-3 arthroscopic debridements and the 66-100% grafts can be retained (Table 4a). Following the proposed protocol, most studies reported comparable or inferior outcome to control group and associated complications as reported before (Tables 4b and 4c).

Conclusion

Septic arthritis after ACLR is a rare and disastrous complication which can be successfully managed by early diagnosis and arthroscopic debridement with a proper protocol.

References

1. Abdel-Aziz A, Radwan YA, Rizk A. Multiple arthroscopic debridement and graft retention in septic knee arthritis after ACL reconstruction: a prospectivecase-control study. *Int Orthop* 2014;38:73-82.
2. Torres-Claramunt R, Pelfort X, Erquicia J, et al. Knee joint infection after ACL reconstruction: prevalence, management and functional outcomes. *Knee Surg Sports Traumatol Arthrosc* 2013;21:2844-9.
3. Maletis GB, Inacio MC, Reynolds S, Desmond JL, Maletis MM, Funahashi TT. Incidence of postoperative anterior cruciate ligament reconstruction infections: graft choice makes a difference. *Am J Sports Med* 2013;41:1780-5.
4. Sonnery-Cottet B, Archbold P, Zayni R, et al. Prevalence of septic arthritis after anterior cruciate ligament reconstruction among professional athletes. *Am J Sports Med* 2011;39:2371-6.
5. Monaco E, Maestri B, Vadalà A, Iorio R, Ferretti A. Return to sports activity after postoperative septic arthritis in ACL reconstruction. *Phys Sportsmed* 2010;38:69-76.

6. Barker JU, Drakos MC, Maak TG, Warren RF, Williams RJ 3rd, Allen AA. Effect of graft selection on the incidence of postoperative infection in anterior cruciate ligament reconstruction. *Am J Sports Med* 2010;38:281-6.
7. Wang C, Ao Y, Wang J, Hu Y, Cui G, Yu J. Septic arthritis after arthroscopic anterior cruciate ligament reconstruction: a retrospective analysis of incidence,presentation, treatment, and cause. *Arthroscopy* 2009 ;25:243-9.
8. Katz LM, Battaglia TC, Patino P, Reichmann W, Hunter DJ, Richmond JC. A retrospective comparison of the incidence of bacterial infection following anterior cruciate ligament reconstruction with autograft versus allograft. *Arthroscopy* 2008;24:1330-5.
9. Schulz AP, Götze S, Schmidt HG, Jürgens C, Faschingbauer M. Septic arthritis of the knee after anterior cruciate ligament surgery: a stage-adapted treatment regimen. *Am J Sports Med* 2007;35:1064-9.
10. Van Tongel A, Stuyck J, Bellemans J, Vandenneucker H. Septic arthritis after arthroscopic anterior cruciate ligament reconstruction: A retrospective analysis of incidence, management and outcome. *Am J Sports Med* 2007;35:1059-1063.
11. Binnet MS, Başarir K. Risk and outcome of infection after different arthroscopic anterior cruciate ligament reconstruction techniques. *Arthroscopy* 2007;23:862-8.
12. Judd D, Bottoni C, Kim D, Burke M, Hooker S. Infections following arthroscopic anterior cruciate ligament reconstruction. *Arthroscopy* 2006;22:375-84.
13. Fong SY, Tan JL. Septic arthritis after arthroscopic anterior cruciate ligament reconstruction. *Ann Acad Med Singapore* 2004;33:228-34.
14. Burks RT, Friederichs MG, Fink B, Luker MG, West HS, Greis PE. Treatment of postoperative anterior cruciate ligament infections with graft removal and early reimplantation. *Am J Sports Med* 2003;31:414-8.
15. Schollin-Borg M, Michaëlsson K, Rahme H. Presentation, outcome, and cause of septic arthritis after anterior cruciate ligament reconstruction: a case control study. *Arthroscopy* 2003;19:941-7.
16. Indelli PF, Dillingham M, Fanton G, Schurman DJ. Septic arthritis in postoperative anterior cruciate ligament reconstruction. *Clin Orthop Relat Res* 2002;(398):182-8.
17. Mouzopoulos G, Fotopoulos VC, Tzurbakis M. Septic knee arthritis following ACL reconstruction: a systematic review. *Knee Surg Sports Traumatol Arthrosc* 2009;17:1033-42.
18. Wang C, Lee YH, Siebold R. Recommendations for the management of septic arthritis after ACL reconstruction. *Knee Surg Sports Traumatol Arthrosc* 2013. [Epub ahead of print] PubMed PMID: 24061716.
19. Goldenberg DL, Reed JI. Bacterial arthritis. *N Engl J Med* 1985;312:764–771.
20. Hogan CJ, Fang GD, Scheld WM, Linden J, Dibuch DR. Inhibiting the inflammatory response in joint sepsis. *Arthroscopy* 2001;17:311–315.
21. National Institutes of Health (NIH), National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS), Vanderbilt University, United States. Prognosis and Predictors of ACL Reconstruction—A Multicenter Cohort Study. Available at: *http://clinicaltrials.gov/ct2/show/NCT00463099*. Accessed March 1, 2011.
22. Plante MJ, Li X, Scully G, Brown MA, Busconi BD, DeAngelis NA. Evaluation of msterilization methods following contamination of hamstring autograft during anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc* 2013;21:696-701.
23. Parada SA, Grassbaugh JA, Devine JG, Arrington ED. Instrumentation-specific infection after anterior cruciate ligament reconstruction. Sports Health. 2009;1:481-5.
24. Hantes ME, Basdekis GK, Varitimidis SE, Giotikas D, Petinaki E, Malizos KN. Autograft contamination during preparation for anterior cruciate ligament reconstruction. *J Bone Joint Surg Am* 2008;90:760-764.
25. Kondo E, Yasuda K, Azuma H. Prospective clinical comparisons of anatomic double-bundle versus single-bundle anterior cruciate ligament reconstruction procedures in 328 consecutive patients. *Am J Sports Med* 2008;36:1675-87.
26. Yasuda K, Kondo E, Ichiyama H. Clinical evaluation of anatomic double-bundle anterior cruciate ligament reconstruction procedure using hamstring tendon grafts: comparisons among different procedures. *Arthroscopy* 2006;22:240-51.
27. Azar FM, Arthur ST. Complications of anterior cruciate ligament reconstruction. *Tech Knee Surg* 2004;3:238-250.
28. Williams RJ III, Laurencin CT, Warren RF, Speciale AC, Brause BD, O'Brien S. Septic arthritis after arthroscopic anterior cruciate ligament reconstruction: Diagnosis and management. *Am J Sports Med* 1997;25:261-267.
29. Viola R, Marzano N, Vianello R. An unusual epidemic of *Staphylococcus*-negative infections involving anterior cruciate ligament reconstruction with salvage of the graft and function. *Arthroscopy* 2000;16:173-177.
30. Millett PJ, Wickiewicz TL, Warren RF. Motion loss after ligament injuries to the knee. Part I: causes. *Am J Sports Med* 2001;29:664-75.