

# Managing septic arthritis after knee ligament reconstruction

Raúl Torres-Claramunt<sup>1,4</sup> · Pablo Gelber<sup>2</sup> · Xavier Pelfort<sup>3</sup> · Pedro Hinarejos<sup>1,4</sup> · Joan Leal-Blanquet<sup>1</sup> · Daniel Pérez-Prieto<sup>1</sup> · Joan C. Monllau<sup>1,4</sup>

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## Abstract

**Purpose** Joint infection after anterior cruciate ligament (ACL) reconstruction is uncommon but has potentially serious consequences for the graft and articular cartilage. Most recently published series are in agreement that an urgent arthroscopic washout and antibiotic treatment are mandatory to preserve both graft and cartilage. However, several questions have not as yet been touched upon.

**Methods** We performed a literature review to assess the most interesting series published about this issue.

**Results** In this review, a management protocol is first presented that discusses the different diagnostic parameters to consider and surgical and antibiotic treatment suggested according to the literature. Outcomes published in different series are also discussed.

**Keywords** Anterior cruciate ligament · ACL · Infection · Septic arthritis · Joint infection · Knee · Debridement · Antibiotic

## Introduction

The number of anterior cruciate ligament reconstructions (ACLR) performed over recent years has increased and is expected to increase further in coming years. Knee-joint infection following this procedure is an uncommon but serious complication. Therefore, it is important to understand the natural history of this complication and its devastating consequences if not swiftly acted upon. Depending on the time elapsed since apparition to diagnosis of infection and establishment of correct therapy, the infection may have no consequences for the joint or it might imply a loss of the ACL graft viability, injury to the articular cartilage or even early osteoarthritis. In 1998, Matava et al. [1] performed an interesting study in which a questionnaire was directed to different orthopaedic fellowship programme directors that asked about the best way to manage an infected knee after an ACLR. That study highlighted the lack of conclusive evidence, making it difficult to arrive at a consensus with regard to the best way of managing it. In this review, prevalence, aetiopathology, diagnosis, the most accepted treatment method and functional outcomes after this complication are described.

## Prevalence

Several series showing different rates of knee-joint infection following ACLR have been published over recent years. The lowest rate was presented by Indelli et al. [2], who reported a rate of 0.14 % for septic arthritis in a series of 3500 consecutive ACLR patients. On the other hand, the highest published rates are by Torres-Claramunt et al. [3] (1.8 % in a series with 810 consecutive ACLR) and Shollin-Borg et al. [4] (1.7 % in a series with 575 ACLR). Viola et al. [5] reported a retrospective series with 13 patients diagnosed with septic arthritis

✉ Raúl Torres-Claramunt  
rtorresclaramunt@parcdesalutmar.cat

<sup>1</sup> Orthopaedic Department, Hospital del Mar, Universitat Autònoma de Barcelona, Passeig Marítim 25-29, 08003 Barcelona, Spain

<sup>2</sup> Orthopaedic Department, Hospital de la Santa Creu i Sant Pau, Universitat Autònoma de Barcelona, Sant Antoni Maria Claret 167, 08025 Barcelona, Spain

<sup>3</sup> Orthopaedic Department, Consorci Sanitari de l'Anoia, Avinguda Catalunya 11, 08700, Igualada Barcelona, Spain

<sup>4</sup> IMIM Hospital del Mar Medical Research Institute, Barcelona, Spain

following ACLR performed with a bone–patellar tendon–bone (BPTB) autograft. Eleven of 13 of these infections were observed in 70 consecutive ACLRs, and the other two infections were reported amongst 1300 ACLRs. It was possible to identify a contamination source in an inflow cannula in which coagulase-negative *Staphylococcus* (CNS) was seen. When this source of contamination was eradicated, the infection rate decreased dramatically. Sechriest et al. [6] observed a similar situation in their study. They identified a yearly ACLR infection incidence of 4.4 %, which obliged them to temporarily suspend all ACLR procedures. They ultimately observed different shortfalls in the surgical equipment decontamination and sterilisation process that ultimately led to the development of an infection-preventing pathway for patients and providers. Sonnery-Cottet et al. [7] also presented a consecutive series with 1957 ACLRs with an infection rate of 0.61 %. Those authors observed that this rate increased to 5.7 % in a subgroup of professional athletes that included 88 patients. The remaining patients (1869), considered non-professional athletes, had an infection rate of 0.37 %. As 23 % of the professional athlete group had a combined ACLR and lateral tenodesis, the authors concluded that being professional athletes and having an associated lateral tenodesis when an ACLR was performed were risk factors for the development of infection. Judd et al. [8] published a series with 1615 consecutive ACLRs performed over seven consecutive years in which 11 (0.68 %) cases of postoperative knee-joint infections were observed. Those 11 infections occurred in a subgroup of 418 consecutive ACLRs performed over three consecutive years. There were no infections in the remaining four year period (1197 ACLRs). Although the grafts used in these 418 ACLRs were similar, using either a BPTB or a hamstring autograft, all infections occurred with hamstring autografts. Those authors also observed a higher incidence of infection in patients with prior knee surgery [relative risk (RR) 1.9].

In summary, although most series have shown rates of infections <1 % following an ACLR, some series (or different subgroups within those series) reached rates >1.5 %. Moreover, an open surgical procedure associated with ACLR or previous knee surgery might lead to an increase in these rates of infection.

### Aetiopathology

Fong and Tan [9] presented a series with 472 cases of consecutive ACLR in which they observed seven cases of postoperative joint infection. These seven cases were classified with regard to time elapsed from surgical procedure. Patients were classified as acute (<2 weeks), subacute (2 weeks to 2 months) and late (>2 months) infection; none of these infections was classified as late infection. While this classification uses an appropriate two week cutoff point to differentiate between acute and subacute infections, eight weeks might be

excessively long to differentiate between subacute and late infection, considering infection aetiology. In fact, knee-joint infections occurring four or five weeks after ACLR may be considered late. Source of infection and the moment at which microorganisms invaded the joint have not been clearly defined [8–13].

*S. aureus* and CNS (*S. epidermidis* and other coagulase-negative species) are the most common bacteria found in most series [2, 3, 5, 7, 10, 11, 14–16]. Additionally, different methicillin-resistant *S. aureus* or anaerobium microorganisms have also been cultured as the origin of such infections. This fact should be considered when empirical antibiotic treatment is initiated [3, 14]. *Staphylococcus* is a microorganism capable of forming a biofilm within the first few weeks that protects itself from antimicrobials, thus making it difficult to eradicate [17]. It is therefore important to initiate treatment as early as possible to preserve both graft and cartilage [18].

Infection rate following ACLR differs depending on the graft used [8, 14, 19, 20]. Recently, Brophy et al. [21] reported that a patient with diabetes who receives a hamstring auto- or allograft has an increased risk of infection following ACLR. Additional surgical procedures during ACLR are also considered a risk factor [7, 11, 19]. This might not only be due to the need to add an open surgical approach but also due to increased surgical time. Finally, prior surgical procedures in the same knee [4, 8, 22], the use of drains and of different types of graft fixation [8, 23] are considered risk factors for septic arthritis following ACLR.

With regard to new prophylactic measures described to decrease infection rate following ACLR, recent investigations demonstrated that presoaking the autograft in a diluted solution of vancomycin is effective for this purpose [24–26]. Other measures, such as the use of gentamicin irrigation solution, have been shown as less effective [27].

### Diagnosis

Joint aspirate is the main diagnostic test to confirm an infection and is mandatory before starting antimicrobial treatment. In fact, infection is undoubtedly confirmed when a positive culture is obtained from aspirate or when synovial cell count is consistent with this diagnosis [28]. Paci et al. [29] studied postoperative synovial fluid in patients who had undergone ACLR and observed that granulocyte values >16,200 g/mm<sup>3</sup> might be considered a good cutoff point to define this fluid as infected, with a susceptibility of 86 % and a specificity of 92 %. The susceptibility of synovial fluid culture is slightly >80 %. However, Gram-stain susceptibility is <50 %. Thus, a negative result does not preclude the diagnosis of septic arthritis. In most series, most microorganisms were identified [3, 4, 7–9, 11, 13, 14, 30]. However, some series report only 14 % of microorganisms as being identified [14]. On the other hand, it

is important to emphasise that the evolution of testing for C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) values in the postoperative period after ACLR can be helpful to diagnose and monitor an infection [31–34].

To summarised, when septic arthritis after ACLR is suspected, a blood test and synovial fluid aspiration are absolutely mandatory. If the biochemical fluid analysis or the Gram stain is conclusive, it can be diagnosed as septic arthritis. If these values are not conclusive but CRP value and white blood cell count (along with high clinical suspicion) are suggestive of knee-joint infection, the problem should be treated as septic arthritis at least. Due to the deleterious effects of joint infection, treatment must be initiated with haste. In the list below, we suggest a diagnostic protocol to define septic arthritis following an ACLR; one diagnostic parameter or at least two highly suspicious parameters are diagnostic of septic arthritis after ACLR.

### 1. Diagnostic

- Positive culture or positive Gram
- Purulent aspect of the aspirate
- Polymorphonuclear cell percentage >90 %
- Cell count >100,000

### 2. Highly suspicious

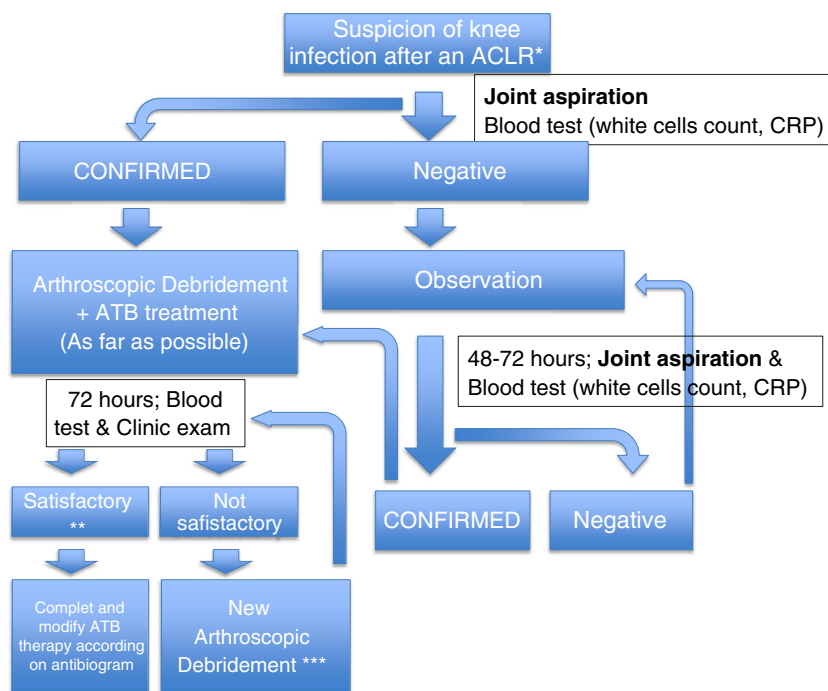
- Turbid aspect
- Polymorphonuclear cell percentage; from 75 % to 90 %

- Cell count; from 20,000 to 100,000
- Glucose: <50 % serum level
- CRP value; >150 mg/dl day 3, or >20 mg/dl day 15

## Treatment

Protecting the articular cartilage and the ACL graft are the two main objectives of treatment. With new knowledge gained over recent years and the lack of standard guidelines for treatment, here we present a new algorithm of treatment for this complication (Fig. 1). Recently, Abdel-Aziz et al. [39] proposed a similar algorithm, which they used in a large series of 2560 ACLRs. They treated 24 cases of infection with this algorithm, obtaining good results. Different authors have used isolated non-operative treatments. Viola et al. [5] chose antibiotic treatment with ciprofloxacin plus amoxicillin/clavulanic acid for a period of 15–90 days; in >40 % of cases, treatment failed, and patients required an arthroscopic lavage. Monaco et al. [40] described antibiotic therapy along with repeated joint irrigation; that treatment failed in 30 % of patients and required an arthroscopic lavage. Contrary to those results, Wang et al. [16] presented a series with 21 cases of infection following an ACLR, some of which were treated conservatively. As the study authors acknowledged, this treatment was chosen because they were short of experience in their early cases. Both treatments, non-operative and operative, were reported as being effective. In the non-operative group, fever decreased at a mean of 9.2 days and antibiotic therapy IV was 27.7 days. In the

**Fig. 1** Management algorithm for knee-joint infection following anterior cruciate ligament repair (ACLR). \*\*Satisfactory is defined as a decrease in C-reactive protein (CRP) values and improvement in clinical signs of infection. \*\*\* In cases in which three arthroscopic lavages are necessary to resolve infection, we recommend removing the graft and hardware if a fourth washout is needed. Table 1 shows the different diagnoses and subsequent treatments performed by different authors.



**Table 1** Literature review

Study	Year of publication	No. knees	Incidence [n (%)]	Graft	Most common pathogens <sup>b</sup>	Days until diagnosis	Empiric antibiotic	Weeks IV/oral (total) <sup>a</sup>	Mean number lavages	Grafts removed
Burks et al. [14]	2003	1918	8 (0.42 %)	7 HT 1 BPTB	3 SA 1 PA	19	Not reported	6/0 (6)	2	7
Barker et al. [35]	2010	3126	18 (0.58 %)	5 HT 7 BPTB 6 Allo	6 SA 4 CNS 2 <i>P. acnes</i> 6 Unknown	32	Not reported	(6)	1.38	5
Fong and Tan [9]	2004	472	7 (1 %)	7 HT	4 SA 3 <i>Pepto</i> 1 <i>Klebsiella</i> 1 <i>Enterobacter</i>	24.5	Not reported	2.5/4 (6.5)	1.4	0
Indelli et al. [2]	2002	3500	6 (0.14 %)	4 BPTB 2 Allo	3 SA 2 CNS 1 <i>Strep</i>	33.5	Not reported	6/6 (6)	2.3	2
Judd et al. [8]	2006	1615	11 (0.68 %)	11 HT	1 SA 8 CNS 1 <i>Enterobacter</i> 1 <i>Propioni</i>	14.2	Cefazolin/ vancomycin	4/4 (4)	2.4	1
Katz et al. [36]	2008	801	6 (0.75 %)	2 HT 4 Allo	1 SA 6 CNS 1 <i>P. acnes</i>	16.4	Not reported	4–6 weeks	–	5
Shollin-Borg et al. [4]	2003	575	10 (1.7 %)	4 HT 6 BPTB	1 SA 6 CNS 1 <i>Propioni</i> 2 Unknown	15.4	Not reported	4–12 weeks	1 (and continuous irrigation)	0
Van Tongel et al. [30]	2007	1736	15 (0.86 %)	12 HT 2 BPTB 1 Allo	1 SA 8 CNS 1 <i>Enterococcus</i> 1 <i>E. cloacae</i> 3 Poly	10.9	Cloxacillin/ gentemycin	4/10 (±14)	1.9	1
Viola et al. [5]	2000	1794	13 (0.78 %)		2 CNS	7.7	Ciprofloxacin/ amoxicillin plus clavulanate	15–90 days	0.46	0
Shulz et al. [13]	2007	3077	24 (0.78 %)	12 BPTB 7 HT 4 Vicryl band 1 Trevira band	12 SA 5 CNS 2 <i>Strep</i> 4 Unknown	61.7	Ampiciline/ Sulbactam	Not reported	3.95	17
Schub et al. [37]	2009	4068	21 (0.52 %)	20 HT 1 Allo	3 SA 12 CNS 1 <i>E. faecalis</i> 1 <i>Coryne</i>	16.4	Vancomycin or cephalosporin	3/3 (3)	0.7	0
William et al. [19]	1997	2500	7 (0.3 %)	3 HT 4 BPTB	6 SA 2 CNS 1 <i>Pepto</i>	21.8	Ceftazidime/ vancomycin	4–6/- (4–6)	1.57	4
Sonnery-Cottet et al. [7]	2011	1956	12 (0.61 %)	7 HT 4 BPTB 1 QT	11 CNS 1 <i>Propioni</i>	15.6	Not reported	Not reported	1.25	0
Torres-Claramunt et al. [3]	2013	810	15 (1.8 %)	11 HT 4 BPTB	3 SA 10 CNS 1 <i>Propioni</i>	23.9	Ceftazidime/ vancomycin	2–3/3 (6)	1.3	1
McAllister et al. [11]	1999	831	4 (0.48 %)	1 HT 3 BPTB	4 SA	11.2	Not reported	4.7/3 (7.7)	2.75	0
Binnet et al. [20]	2007	1231	6 (0.49 %)	4 BPTB 2 HT	3 SA 1 PA	22	Cefazolin	3/- (3)	2.66	0
Sajovic et al. [38]	2009	1283	3 (0.23 %)	3 HT	1 SA 1 CNS 1 Unknown	8	Cloxacillin	2/4 (6)	1	0

**Table 1** (continued)

Study	Year of publication	No. knees	Incidence [n (%)]	Graft	Most common pathogens <sup>b</sup>	Days until diagnosis	Empiric antibiotic	Weeks IV/oral (total) <sup>a</sup>	Mean number lavages	Grafts removed
Abdel-Aziz et al. [39]	2014	2560	24 (0.93 %)	24 HT	7 CNS 7 SA 1 <i>E. faecalis</i> 1 <i>E. coli</i> 2 <i>Propioni</i> 1 <i>Strep</i> 2 <i>Pepto</i> 3 Unknown	12	Cephalosporin or vancomycin EV	4/- (-)	3	3

<sup>a</sup> The total duration of the antibiotic treatment is not well-documented in all studies. Total duration is the summation of IV and oral treatment, although in some cases, it is simply referred to as IV treatment duration

*HT* hamstring, *BPTB* bone–patellar tendon–bone, *allo* allogenic, *SA* *Staphylococcus aureus*, *CNS* coagulase-negative staphylococcus, *Propion* *Propionibacterium*, *Strep* *Streptococcus*, *PA* *Pseudomonas aeruginosa*, *Coryne* *Corynebacterium*, *E. faecalis* *Enterococcus faecalis*, *E. cloacae* *Enterococcus cloacae*, *Pepto* *Peptostreptococcus*, *Poly* polymicrobial

operative group, fever lasted for 1.5 days, and antibiotic therapy IV was stopped after 19 days.

Overall, it is accepted that the treatment of choice is arthroscopic lavage, performed as soon as possible, along with beginning antibiotic treatment IV in most published series [10, 41]. During arthroscopic debridement, it is mandatory to gather samples for culturing before antibiotic administration, and both therapies must be performed as soon as possible. Even if the diagnosis is not completely confirmed, it is always preferable to perform the arthroscopic washout prior to beginning antibiotic treatment; however, if the surgical procedure cannot be performed immediately, antibiotic therapy must still be initiated. Arthroscopy must include extensive lavage with normal serum, debridement of devitalised tissue, removal of fibrin clots and synovectomy. It is also suggested to remove the fibrin layer covering the graft surface [3] and evaluate the macroscopic integrity of the graft as well as its tension. If the graft is not viable, it should be removed, along with fixation hardware. Antibiotic therapy should be initiated immediately after culture samples are taken. A new blood test should be done at 72 hours. If clinical and/or laboratory parameters are not satisfactory (e.g. improving from pre-operative values), a new arthroscopic washout must be performed.

Although most series recommend specific antibiotic treatment in accordance with micro-organism susceptibility, most failed to report whether empirical antibiotic therapy IV was previously initiated. Some authors reported that all patients were empirically treated with cephalosporin or vancomycin when the infection was first diagnosed [8, 16]. Van Tongel et al. [30] preferred to use a combination of cloxacillin and gentamycin. However, in many cases, bacteria were resistant to the empiric antibiotic treatment, and antibiotics must be changed when culture sensitivities are obtained; other series did not mention whether they used empiric antibiotic treatment. Empiric antibiotic treatment should be effective against most

microorganisms described for this infection—not only against the *Staphylococcus* group, including CNS and methicillin-resistant subgroups, but also against Gram-negative microorganisms, as this infection is comparable with a prosthetic joint infection [42]. In our series [3], vancomycin (1 g/12 h) and cefazidime (2 g/8 h) were initiated after obtaining joint aspirate or after the arthroscopic procedure. We propose this combination as empirical antibiotic therapy for this type of complication.

There is also controversy regarding how long the antibiotic should be administered and when to switch from IV to enteral administration. Although the duration of antibiotic treatment can vary between four [36] and 14 [30] weeks, most authors agree that it should be provided for not less than six weeks. There is also consensus that IV administration is preferable for the first two to three weeks [3, 9, 16]. However, the precise duration of IV treatment should be determined based on the microorganism cultured, its susceptibilities and postoperative clinical and laboratory parameters [43]. Thus, the final antibiotic regimen must consider a number of factors.

Post-operative physical therapy is not described in all series. In most series, however, continuous passive motion was initiated in days following debridement, and weight bearing was not initially allowed. While in some series weight bearing was allowed when wounds were clinically healed [19], other series waited until the sixth post-operative week [11, 37]. Our recommendation is to initiate in the firsts post-operative days a graded knee-strengthening programme including quadriceps and hamstrings strength through progressive isometric, isotonic and isokinetic exercises. Moreover, range of motion (ROM) might be progressively increased. All these exercises might be initiated when symptoms suggestive of infection disappear. In our point of view, progressive weight bearing could be also allowed at this time, achieving complete weight bearing by the sixth post-operative week [3]. Figure 1 summarises the management algorithm suggested by us.

## Functional outcomes

A satisfactory result after treatment of an infected ACLR should be defined as good functional scores, preserved articular cartilage architecture, preserved ACL graft, restored full ROM or return to previous level of activity. However, previous series provided only some of these aspects. In addition, most series only reported very short follow-up results [8, 9, 14]. Different authors considered a mean Lysholm score of 65.6 [13] or 69.5 [40] to be satisfactory. More in accordance with current standards of satisfactory results, Lysholm score >90 points [5, 14, 15, 41] has also been reported. In our series [3], a mean Lysholm score of 77.7 and a mean International Knee Documentation Committee (IKDC) score of 70.4 at a mean follow-up of 39.3 months were achieved. Conversely, the control group had a mean Lysholm score of 90.7 and a mean IKDC of 86.6. Surprisingly enough, Monaco et al. [40] reported on a series of patients who were able to return to pre-operative sports at the same level. Shulz et al. [13] reported a 60-day mean time to diagnose the infection, and in one case, a total joint arthroplasty had to be performed five years after ACLR. They concluded that subjective and clinical results after this complication were clearly inferior to those of “unproblematic” ACLR. Boström Windhamre et al. [44] studied functional outcomes in 27 patients after infected ACLRs with 60 months of follow-up, indicating no inferior objective knee function or lower degree of patient satisfaction. However, patients required a longer rehabilitation period, and fewer patients returned to sports. Gille et al. [45] reviewed 31 patients with infection after ACLR from 1993 to 2010; of these 31 patients it was possible to maintain the graft in eight only. They confirmed that patients that had not been diagnosed early presented worst results than those arthroscopically managed in the immediate postoperative period.

With regard to the KT-1000 arthrometer test, we obtained a mean difference of 1.3 mm when the injured knee was compared with the non-injured knee [3]. In fact, in four of 15 patients, this difference was  $\leq 1$  mm. Monaco et al. [40], in a series with 14 infections, observed a mean KT-1000 side-to-side difference of 2.5 mm (follow-up, 38 months) and Binnet et al. [20] observed that this value was 2.7 mm (follow-up, 102 months). It seems that, if the graft can be maintained, the laxity obtained in most cases is similar to those patients who have not had an infection. A combination of glycosaminoglycan and collagen depletion after a joint infection, along with some degree of arthrofibrosis, might help explain these results [46].

Shub et al. [37] published a study with four cases of infections following ACLR with a mean follow-up of 17.9 years and reported that patients had diminished subjective, functional and radiographic outcomes compared with uncomplicated cases. Compared with an earlier functional follow-up, those patients improved in terms of pain and remained stable in

terms of both functional scoring and activity-related subjective scales. Although radiographic studies revealed arthritis progression in all patients and a torn graft in one, no patient required additional surgery at almost 18 years after the infection.

While hamstring autografts are more susceptible to infection, patients with an ACL reconstructed with a BPTB autograft have 2.75 more possibilities of presenting degenerative changes after a knee infection [11, 30]. Scholling-Borg et al. [4] also showed a worse Knee Injury and Osteoarthritis Outcome Score (KOOS) in patients with infection after ACLR with a BPTB autograft than those with a hamstring autograft. However, other independent factors might also contribute to degenerative changes. *S. aureus* infection, two or more debridements and allografts have also been related with these findings [43].

## Summary

Following is a summary of findings from our literature review:

- The rate of joint infection after an ACLR is <to 1 % in most series.
- Concomitant open surgery or previous surgery on the same knee increases the risk of septic arthritis following ACLR.
- Most of these infections are caused by *Staphylococcus*, mainly CNS, and it seems that the infections are introduced during the surgical procedure.
- In most series, infection occurs during the first postoperative month. Close patient monitoring and a high index of diligence are needed for a proper and early diagnosis.
- The most used classification groups such infections into: acute (<2 weeks), subacute (2 weeks–2 months) or late (>2 months). We believe this classification can be improved. However, the two month cutoff point for differentiating between subacute and late infection seems unreasonably long. Instead, four weeks might be a more appropriate cutoff time.
- Presoaking the graft with a vancomycin solution seems a good option for lowering infection rates.
- Joint aspiration and a blood test are essential for diagnosing infection. However, if the infection cannot be formerly diagnosed but analytical parameters and a high clinical suspicion suggest it, it should be treated as a knee-joint infection.
- Treatment must be initiated as early as possible. A combination of arthroscopic debridement and antibiotic therapy are the basis of this treatment. Antibiotic treatment initiated empirically may be agreed upon with the

infectious diseases department, but this initial treatment should be similar to that used in a prosthetic infection.

- If clinical and laboratory parameters do not improve within 48–72 hours, a new arthroscopic lavage should be considered. If the graft is viable, it may be possible to retain it.
- Intravenously administered antibiotic treatment should be maintained until clinical and analytical parameters normalise. Overall, antibiotic treatment is maintained for a minimum of six weeks.
- Function after septic arthritis following ACLR is impaired. While good functional outcomes can be achieved, full return to athletic activities cannot be always accomplished.

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