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Septic Arthritis in Dogs in North Africa: Microbiological Isolation, Histopathological Progression and Therapeutic Efficacy of Platelet-Rich Plasma

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Abstract

Background. Septic arthritis destroys articular cartilage and subchondral bone within days unless treatment begins promptly. The condition is well characterised in European and North American case series, but no North African dataset has combined clinical scoring, imaging, histopathology, culture, susceptibility testing and regenerative therapy in a single cohort.

Methods. We retrospectively reviewed 50 client-owned dogs with naturally occurring septic arthritis presented in eastern Libya between 2017 and 2025. Recorded variables comprised lameness (1–5), joint swelling (0–3), pain on a 0–10 visual analogue scale, orthogonal radiographs, high-resolution B-mode ultrasonography, synovial fluid cytology, aerobic culture, disk-diffusion susceptibility testing to CLSI VET01, and haematoxylin–eosin histopathology graded on a four-tier synovitis scale. Eleven dogs with chronic culture-negative synovitis received a single intra-articular injection of autologous platelet-rich plasma (PRP) after lavage, with follow-up biopsy at four to six weeks.

Results. Males predominated (56%) and the stifle was the joint most often

involved (38%). Five variables predicted high-grade (grade ≥ 3) synovitis: lameness ≥ 4 (odds ratio [OR] 90.3, $p < 0.001$), pain ≥ 7 (OR 22.1, $p < 0.001$), radiographic joint space narrowing (OR 41.8, $p < 0.001$), subchondral osteolysis (OR 37.1, $p < 0.001$) and symptom duration beyond 30 days (OR 9.1, $p = 0.038$). Staphylococcus pseudintermedius was the commonest isolate (28.6%), and meticillin-resistant staphylococci accounted for 16.3% of positive cultures. Amikacin retained the greatest in-vitro activity (92%). Pannus, cartilage erosion and grade-3 synovitis each predicted failure to resolve by 30 days. Post-PRP biopsies showed villous re-epithelialisation, subsynovial fibrosis and angiogenesis without microbiological or histological evidence of recurrent infection.

Conclusions. Canine septic arthritis in eastern Libya is driven predominantly by coagulase-positive staphylococci. Two bedside scores (lameness, pain) and two radiographic signs (joint space narrowing, osteolysis) identify high-risk animals reliably enough to guide urgent management where advanced diagnostics are unavailable. Intra-articular PRP appeared well tolerated in carefully selected post-infectious dogs, although the uncontrolled design of that subgroup precludes any inference of efficacy.

Key words: dog; septic arthritis; synovitis; histopathology; antimicrobial susceptibility; platelet-rich plasma.

1. Introduction

1.1 Pathophysiology and routes of infection

Bacterial colonisation of the synovial space provokes an intense neutrophilic response, and it is the host reaction rather than the organism itself that inflicts most of the damage. Activated proteases and matrix metalloproteinases can degrade hyaline cartilage within 72 hours, which is why septic arthritis is regarded as a surgical emergency rather than a condition that can be managed expectantly (1-3). Organisms reach the joint by several routes: penetrating and bite wounds, peri-operative contamination, intra-articular injection, migrating plant material, haematogenous seeding, and direct extension from adjacent osteomyelitis (4,5). The intra-articular environment then works against recovery. It is hypoxic and acidic, neutrophil-derived enzymes accumulate, and the end results may be fibrous or bony ankylosis or, in severe cases, systemic sepsis (6-8).

1.2 Causative organisms and antimicrobial resistance

Coagulase-positive staphylococci dominate the published literature worldwide, with Staphylococcus pseudintermedius the organism most consistently reported. Meticillin-resistant S. pseudintermedius (MRSP) and meticillin-resistant S. aureus (MRSA) are both increasing in frequency. Streptococci, Escherichia coli, Pasteurella spp., Pseudomonas aeruginosa and mixed infections account for most remaining isolates (9-12). Because multidrug resistance in companion animal orthopaedic infection is now a global concern, current local susceptibility data are needed wherever empirical therapy is prescribed — and such data are largely absent for North Africa (13-15).

1.3 Diagnostic approach

Diagnosis rests on the integration of history, physical examination, synovial fluid analysis, culture, imaging and, where available, synovial biopsy (16-18). Cytological findings typically comprise a total nucleated cell count above 30,000 cells/μL with at least 80% neutrophils, with or without visible intracellular bacteria. Radiographs early in the course show only periarticular soft tissue swelling and displacement of the infrapatellar fat pad; joint space narrowing, subchondral lysis, osteophytosis and periosteal new bone appear later (19,20). Ultrasonography is the more sensitive modality for early effusion, synovial thickening, fibrin septa and villous proliferation, and it can guide arthrocentesis (21-23). Culture, though central, is imperfect: negative results are common, particularly after antimicrobial exposure, and molecular methods have not so far demonstrated superior accuracy (24,25).

Histological assessment of synovium permits direct grading of inflammatory severity and chronicity through synoviocyte hyperplasia, villous hypertrophy, vascular congestion, fibrin deposition, pannus, fibrosis and cartilage erosion. Semi-quantitative scoring is established in equine and human rheumatology (26-28), yet few veterinary studies have correlated histological grade with concurrent clinical, imaging and bacteriological findings in spontaneous canine disease.

1.4 Platelet-rich plasma

Interest in platelet-rich plasma (PRP) as a regenerative adjunct rests on the supraphysiological concentrations of growth factors released by activated platelets — PDGF, TGF-β, VEGF, IGF-1 and

EGF among them — which promote angiogenesis, collagen synthesis and chondrocyte metabolism (29-32). Whether PRP can be given safely after documented bacterial eradication in chronic septic arthritis, and what the synovium looks like histologically afterwards, has not been described in a North African cohort.

1.5 Objectives

This retrospective study was designed to address four questions:-

1- What is the clinical, radiographic, ultrasonographic and histopathological spectrum of naturally occurring canine septic arthritis in eastern Libya?

2-Which bacteria are responsible, and what is their in-vitro susceptibility to commonly used antimicrobials?

3-Which clinical and imaging findings predict severe synovitis, and which predict failure to resolve by 30 days?

4-What is the histological appearance of synovium after joint lavage followed by intra-articular PRP in chronic, culture-negative cases?

2. Materials and Methods

2.1 Study design and setting

We reviewed medical records, digital imaging archives, cytology reports and histopathology files held by the Veterinary Teaching Hospital of the Faculty of Veterinary Medicine, Omar Al-Mukhtar University (Al-Beyda, Libya), together with cases referred from two private specialist orthopaedic centres. All dogs diagnosed with septic arthritis between January 2017 and December 2025 were screened for eligibility. Reporting followed the STROBE-Vet statement.

2.2 Ethical approval

The protocol was approved by the Institutional Animal Care and Use Committee of Omar Al-Mukhtar University (approval OMU-VET-2026-014), and written informed consent was obtained from every owner. Procedures complied with the ICLAS Guidelines for Researchers, European Directive 2010/63/EU and the National Research Council Guide for the Care and Use of Laboratory Animals.

2.3 Case definition

Dogs were enrolled when at least two of the following were satisfied: -

1- Severe or non-weight-bearing lameness with joint effusion, pain, swelling or local heat.

2-Synovial fluid total nucleated cell count above 30,000 cells/μL with ≥ 80% neutrophils, with or without intracellular bacteria.

3-A positive aerobic culture from synovial fluid or synovial membrane.

4-Surgical or histopathological findings consistent with septic arthritis.

Dogs with confirmed immune-mediated polyarthritis, primary degenerative osteoarthritis, joint neoplasia or experimentally induced arthritis were excluded.

2.4 Clinical assessment

For each animal we recorded signalment, affected joint and laterality, duration of clinical signs before presentation, and any history of trauma, previous joint surgery or antimicrobial administration within the preceding four weeks. Lameness was graded 1–5, swelling 0–3 and pain on a 0–10 cm visual analogue scale. Local joint heat was noted and rectal temperature measured.

2.5 Diagnostic imaging

Orthogonal mediolateral and craniocaudal radiographs were acquired at 50–60 kVp and 5–10 mAs and read independently by two observers blinded to clinical data — a board-eligible radiologist and an orthopaedic surgeon — who scored soft tissue swelling or effusion, joint space narrowing, osteophytosis, subchondral osteolysis and periosteal reaction. Disagreements were settled by consensus. B-mode ultrasonography used a 7.5–12 MHz linear transducer with the contralateral healthy joint as an internal reference; effusion, synovial and capsular thickening, fibrin strands, villous proliferation and cartilage surface irregularity were each recorded as present or absent.

2.6 Sampling, cytology and microbiology

Arthrocentesis was performed aseptically under sedation with medetomidine and butorphanol. Fluid was collected into EDTA tubes for cytology and plain sterile tubes for culture, with total protein estimated by refractometry. Where lavage, arthrotomy or arthroscopy formed part of treatment, separate sterile biopsies of synovial membrane, articular cartilage and meniscus were taken.

Samples were plated onto 5% sheep blood agar and MacConkey agar (Oxoid, Basingstoke, UK) and incubated aerobically at 37 °C for 24–48 hours; plates without growth were held a further 72 hours before being reported negative. Isolates were identified by colony morphology, Gram reaction, catalase, coagulase and oxidase tests and standard biochemical panels. Where resources allowed, staphylococci were speciated by the multiplex PCR of Sasaki and colleagues (33). Susceptibility was determined by Kirby–Bauer disk diffusion on Mueller–Hinton agar to CLSI VET01

(15) using ampicillin, amoxicillin–clavulanate, cefazolin, ceftriaxone, oxacillin (staphylococci only), amikacin, gentamicin, marbofloxacin, enrofloxacin, doxycycline, clindamycin, trimethoprim–sulfamethoxazole and vancomycin discs. Multidrug resistance was defined as non-susceptibility to agents of three or more distinct classes.

2.7 Histopathology

Synovial biopsies were fixed immediately in 10% neutral buffered formalin, processed to paraffin, sectioned at 4 µm and stained with haematoxylin and eosin. A single pathologist blinded to all clinical data assigned each section a grade from 0 to 3. Grade 0 denoted normal synovium of one to two cell layers without inflammation. Grade 1 indicated mild synovitis with hyperplasia up to three layers and mild vascular congestion, but no fibrin and no erosion. Grade 2 described moderate disease with four to six cell layers, villous hypertrophy, scattered neutrophils and lymphocytes, perivascular infiltrates and focal fibrin. Grade 3 denoted severe synovitis exceeding six cell layers with marked villous hypertrophy, dense mixed infiltrate, fibrin exudation, pannus and/or cartilage erosion, and fibrosis. Individual features — vascular congestion, hyperplasia, fibrin, fibrosis, villous proliferation, cartilage erosion and pannus — were additionally recorded as present or absent. Scale bars were applied at acquisition: 500 µm at ×40, 100 µm at ×200, 50 µm at ×400 and 20 µm at ×1000.

2.8 Treatment protocol and follow-up

All dogs underwent therapeutic lavage with one to three litres of warm sterile isotonic saline through 14–16 gauge needles, received systemic antimicrobial therapy chosen empirically and then revised according to susceptibility results, analgesia (meloxicam 0.1 mg/kg orally once daily, with or without tramadol 3–4 mg/kg twice daily), and exercise restriction.

Eleven dogs with chronic persistent lameness and synovial thickening that remained culture-negative on two consecutive samples taken at least 72 hours after antimicrobial withdrawal received a single intra-articular injection of 4–6 mL autologous PRP, prepared by a double-spin protocol (150 g for 10 minutes, then 400 g for 10 minutes), immediately after repeat lavage. Ultrasound-guided repeat synovial biopsies were taken four to six weeks later. These dogs were not randomly

allocated, and no parallel control group was available; PRP was offered on clinical grounds alone.

Outcome at 30 days was dichotomized as resolution (no lameness, no pain on manipulation, no palpable effusion, normal temperature) or non-resolution (any residual lameness, pain, effusion, pyrexia or progressive radiographic change).

2.9 Statistical analysis

Data were entered into Microsoft Excel and analysed in SPSS version 26 (IBM, Armonk, NY). Continuous variables are reported as mean ± standard deviation or median with interquartile range (IQR) according to distribution, and categorical variables as counts with percentages. Inter-observer agreement for imaging scores was quantified with Cohen's kappa. Associations with the two binary outcomes — severe (grade ≥ 3) synovitis and non-resolution at 30 days — were examined by univariable binary logistic regression, reported as odds ratios with 95% confidence intervals (CI). Pearson coefficients described correlations between clinical scores and histological grade. Significance was set at $p < 0.05$.

A note on statistical power. With 50 animals and 17 grade-3 cases, the number of events per candidate variable is low. Multivariable modelling was therefore not attempted, and the univariable odds ratios reported below carry wide confidence intervals — in three instances spanning more than two orders of magnitude. These estimates identify the direction and probable importance of an association; they do not establish its magnitude with precision, and they should not be used for individual-level risk prediction.

3. Results

3.1 Study population

Fifty dogs met the inclusion criteria (Table 1). Mean age was 5.0 ± 1.9 years (range 1.5–10) and mean body weight 29.6 ± 6.8 kg. Twenty-eight were male (56%), 20 female (40%), and sex was not recorded in two. Mixed-breed dogs formed the largest group (n = 18), followed by German Shepherds (n = 8), Labrador Retrievers (n = 7), Rottweilers (n = 5), Golden Retrievers (n = 4), Pit Bull-type dogs (n = 4) and other pure breeds (n = 4). The stifle was affected in 19 dogs (38%), the elbow in 16 (32%), the carpus in 8 (16%) and the hock in 7 (14%).

Trauma was reported in 11 animals (22%), prior joint surgery in 15 (30%) and antimicrobial administration within four weeks in

18 (36%). Median duration of lameness before presentation was 62.5 days (IQR 27–107) — a notably chronic profile that reflects the referral nature of the caseload. Mean lameness score was 3.4 ± 1.0, mean swelling 1.9 ± 0.7 and mean pain 6.0 ± 1.5; local heat was palpable in 33 dogs (66%). Inter-observer agreement was very good for both radiographic (κ = 0.82) and ultrasonographic (κ = 0.85) scoring.

Table 1. Demographic and clinical characteristics of 50 dogs with naturally occurring septic arthritis.

Variable	Value
Number of dogs	50
Age (years), mean ± SD (range)	5.0 ± 1.9 (1.5–10)
Body weight (kg), mean ± SD	29.6 ± 6.8
Sex: male / female / not recorded	28 (56%) / 20 (40%) / 2 (4%)
Joint: stifle / elbow / carpus / hock	19 (38%) / 16 (32%) / 8 (16%) / 7 (14%)
Trauma history	11 (22%)
Prior joint surgery	15 (30%)
Prior antimicrobials (previous 4 weeks)	18 (36%)
Duration of lameness (days), median (IQR)	62.5 (27–107)
Lameness score (1–5), mean ± SD	3.4 ± 1.0
Swelling score (0–3), mean ± SD	1.9 ± 0.7
Pain VAS (0–10), mean ± SD	6.0 ± 1.5
Local heat present	33 (66%)

SD = standard deviation; IQR = interquartile range; VAS = visual analogue scale.

3.2 Imaging findings

Soft tissue swelling or fat pad displacement was present radiographically in every dog (100%). Joint space narrowing was seen in 32 animals (64%), subchondral osteolysis in 33 (66%), osteophytes in 27 (54%) and periosteal reaction in 13 (26%). Ultrasonography detected effusion in 44 dogs (88%), synovial or capsular thickening in 38 (76%), fibrin strands or septa in 31 (62%), villous proliferation in 31 (62%) and cartilage surface irregularity in 14 (28%). Representative radiographs from one acute and two chronic cases appear in Figure 1, and representative ultrasonographic images in Figure 2.

3.3 Cytology and bacteriology

Synovial fluid was obtained from 47 dogs. Median total nucleated cell count was 78,500 cells/µL (IQR 47,000–132,000) with a median neutrophil proportion of 90% (IQR 83–94). Intracellular bacteria were visible in 31 of 47 samples (66%), and total protein exceeded 30 g/L in 45 of 47 (96%).

Culture was positive in 49 of 50 dogs (98%), a yield considerably higher than the 50–70% typically reported elsewhere; a single organism was recovered from 47 animals and two yielded polymicrobial growth (Table 2). Coagulase-positive staphylococci accounted for 20 of 49 positive cultures (40.8%), comprising S. pseudintermedius in 14 dogs (28.6%) and S. aureus in six (12.2%). Meticillin-resistant isolates (MRSP n

= 5, MRSA n = 3) represented 8 of 49 positive cultures (16.3%). Streptococci and enterococci were cultured from eight dogs, Gram-negative bacilli accounted for 18 isolates, and Corynebacterium sp. was recovered from one.

Table 2. Bacterial isolates from 49 culture-positive dogs.

Pathogen	n	% of isolates
Staphylococcus pseudintermedius (MSSP)	9	17.6
Staphylococcus pseudintermedius (MRSP)	5	9.8
Staphylococcus aureus (MSSA)	3	5.9
Staphylococcus aureus (MRSA)	3	5.9
Streptococcus canis	5	9.8
Streptococcus dysgalactiae	1	2.0
Enterococcus faecalis	2	3.9
Corynebacterium sp.	1	2.0
Escherichia coli	6	11.8
Pseudomonas aeruginosa	5	9.8
Klebsiella pneumoniae	2	3.9
Proteus mirabilis	2	3.9
Enterobacter cloacae	1	2.0
Pasteurella multocida	2	3.9
Polymicrobial growth	2	3.9
No growth	1	—

MSSP/MRSP = meticillin-susceptible/-resistant S. pseudintermedius; MSSA/MRSA = meticillin-susceptible/-resistant S. aureus. Percentages are calculated over 51 organism entries recovered from 49 culture-positive dogs and therefore exceed the case count because two dogs yielded two isolates each.

3.4 Antimicrobial susceptibility

Susceptibility results for the 37 non-fastidious isolates tested appear in Table 3 and Figure 3. Amikacin was the most active agent, inhibiting 92% of isolates (34/37). Marbofloxacin, enrofloxacin and gentamicin each inhibited 78% (29/37), and trimethoprim–sulfamethoxazole and vancomycin each 73% (27/37). Doxycycline inhibited 68% (25/37) and oxacillin 69% of the 16 staphylococci tested (11/16). Ceftriaxone and clindamycin each inhibited 59% (22/37), cefazolin and amoxicillin–clavulanate each 57% (21/37), and ampicillin only 30% (11/37). Fourteen of 37 isolates (37.8%) were multidrug resistant.

Table 3. In-vitro antimicrobial susceptibility of 37 non-fastidious isolates (CLSI VET01 disk diffusion).

Antimicrobial	n/N susceptible	% susceptible
Amikacin	34/37	92
Marbofloxacin	29/37	78
Enrofloxacin	29/37	78
Gentamicin	29/37	78
Vancomycin	27/37	73
Trimethoprim–sulfamethoxazole	27/37	73
Oxacillin (staphylococci only)	11/16	69
Doxycycline	25/37	68
Ceftriaxone	22/37	59
Clindamycin	22/37	59
Cefazolin	21/37	57
Amoxicillin–clavulanate	21/37	57
Ampicillin	11/37	30

Agents are ordered by descending activity. Vancomycin is included for surveillance purposes only; its use in companion animals is not appropriate outside exceptional circumstances.

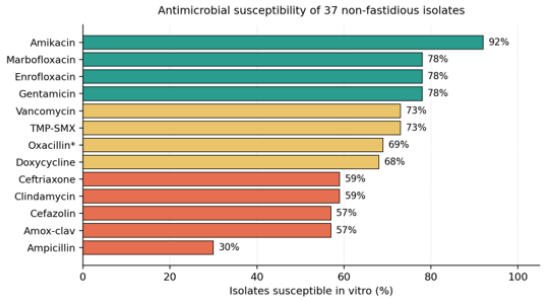

Figure 3. In-vitro susceptibility of 37 non-fastidious isolates. Green denotes ≥ 75% susceptibility, amber 60–74% and red < 60%. Asterisk: oxacillin was tested against the 16 staphylococcal isolates only.

3.5 Histopathology

Synovitis grades among the 50 biopsied dogs were distributed as follows: grade 0 in four dogs (8%), grade 1 in 15 (30%), grade 2 in 14 (28%) and grade 3 in 17 (34%). Individual features comprised vascular congestion in 46 dogs (92%), synoviocyte hyperplasia in 37 (74%), fibrin exudation in 36 (72%), fibrosis in 28 (56%), villous proliferation in 20 (40%), cartilage erosion in 20 (40%) and pannus in 16 (32%). The grading scale is illustrated in Figure 4 and the principal lesions in Figure 5.

3.6 Predictors of severe synovitis

Univariable logistic regression results for grade ≥ 3 synovitis are given in Table 4 and displayed in Figure 6. The strongest association was with a lameness score of 4 or above (OR 90.26, 95% CI 4.92–1655.75, p < 0.001), although the width of that interval warrants emphasis. Pain ≥ 7 (OR 22.14, 95% CI 3.94–124.35, p < 0.001), joint space narrowing (OR 41.77, 95% CI 2.32–752.45, p < 0.001), subchondral osteolysis (OR 37.12, 95% CI 2.06–668.28, p < 0.001) and duration beyond 30 days (OR 9.14, 95% CI 1.07–77.80, p = 0.038) were also significant. Sex, age, body weight, affected joint and prior antimicrobial exposure showed no significant association.

Table 4. Univariable predictors of severe (grade ≥ 3) synovitis.

Predictor	OR	95% CI	p
Lameness score ≥ 4	90.26	4.92–1655.75	< 0.001
Radiographic joint space narrowing	41.77	2.32–752.45	< 0.001
Subchondral osteolysis	37.12	2.06–668.28	< 0.001
Pain VAS ≥ 7	22.14	3.94–124.35	< 0.001
Chronic course > 30 days	9.14	1.07–77.80	0.038
Prior joint surgery	2.20	0.48–10.05	0.31
Prior antimicrobials	2.04	0.61–6.84	0.35
Male sex	0.83	0.26–2.69	0.77

OR = odds ratio; CI = confidence interval. Predictors are ordered by descending effect size. Confidence intervals are wide because of the modest sample; see Section 2.9.

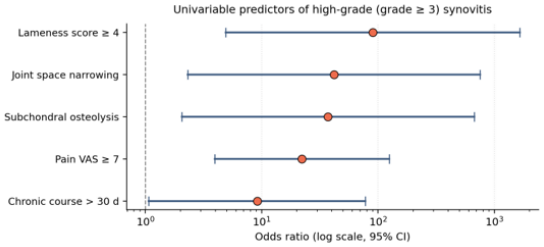

Figure 6. Univariable predictors of high-grade synovitis, plotted on a logarithmic

scale. Point estimates are shown as circles and 95% confidence intervals as horizontal bars; the dashed line marks an odds ratio of 1.

3.7 Predictors of non-resolution at 30 days

Twenty dogs (40%) had resolved completely by 30 days and 30 (60%) had not. Four factors were associated with non-resolution: grade-3 synovitis (OR 21.71, 95% CI 2.57–183.64, p = 0.001), a course longer than 30 days (OR 9.00, 95% CI 2.05–39.55, p = 0.003), pannus (OR 7.88, 95% CI 1.55–40.09, p = 0.012) and cartilage erosion (OR 4.57, 95% CI 1.23–16.94, p = 0.022).

Correlations between clinical scores and histological grade are shown in Table 5. Lameness and pain were the variables most strongly correlated with histological severity (r = +0.69 for both, p < 0.001), followed by swelling (r = +0.46, p < 0.001). Symptom duration showed only a weak, non-significant positive correlation (r = +0.22, p = 0.12) — which sits somewhat awkwardly beside the significant odds ratio for duration in Table 4, and probably reflects a threshold effect at 30 days rather than a continuous relationship.

Table 5. Pearson correlations between clinical scores and histopathological synovitis grade.

Variable	r (vs grade)	p
Lameness score	+0.69	< 0.001
Pain VAS	+0.69	< 0.001
Swelling score	+0.46	< 0.001
Duration of lameness	+0.22	0.12
Lameness vs pain	+0.35	0.01

3.8 Synovial histology after PRP

Eleven dogs with grade 2–3 synovitis and two consecutive negative follow-up cultures received intra-articular autologous PRP. Repeat biopsies at four to six weeks showed regenerative villous re-epithelialisation, a marked reduction in inflammatory infiltrate, mild subsynovial fibrosis, small newly formed capillaries and a smooth intact synovial lining. Fibrin, pannus and intra-epithelial neutrophils were absent in all eleven. No dog developed clinical or bacteriological evidence of recurrent infection during follow-up (Figure 7).

4. Discussion

4.1 Population and disease pattern

This appears to be the first integrated clinical, imaging, bacteriological and histopathological series of naturally occurring canine septic arthritis reported from North Africa. The demographic profile — adult, medium-to-large dogs, males modestly over-represented, stifle most often affected — corresponds closely with series from the United

Kingdom, continental Europe and North America (3,16,34,35). Our figure of 38% stifle involvement sits within the published range; a five-hospital review of 103 dogs reported 40% stifle and 24% elbow involvement (34).

The median duration of signs of 62.5 days is longer than most published series and marks this as a predominantly chronic cohort. That single feature shapes much of what follows: it explains the high prevalence of fibrosis, the substantial proportion of dogs with established radiographic change at first presentation, and, in all likelihood, the relatively low 30-day resolution rate of 40%.

4.2 Microbiology and resistance

Coagulase-positive staphylococci caused more than 40% of culture-positive infections, with S. pseudintermedius predominant, followed by streptococci and a range of Gram-negative rods. This pattern agrees with the wider veterinary literature (3,9-12,35). The 16.3% prevalence of meticillin resistance is a clinically material finding and reinforces the case for culture and susceptibility testing before committing any dog to prolonged therapy.

Two observations deserve caution. The first concerns our 98% culture positivity rate, which is markedly higher than the 50–70% usually reported and higher than would be expected given that 36% of dogs had received antimicrobials beforehand — an exposure repeatedly shown to reduce culture yield (24,34). Sampling technique, immediate plating and the chronic nature of the caseload may all contribute, but selection bias in a retrospective review cannot be excluded, since dogs without a positive culture may have been assigned other diagnoses and thus never entered the dataset. The second concerns therapeutic inference. Amikacin and the fluoroquinolones were the most active agents in vitro, consistent with earlier reports (11,12), but in-vitro activity is not a licence for routine use. Aminoglycoside nephrotoxicity, fluoroquinolone stewardship obligations and the need to preserve these agents for confirmed resistant infection all argue for restraint. The susceptibility of only 30% of isolates to ampicillin is a useful reminder that β-lactamase producers dominate this setting.

Vancomycin merits separate comment. It was included in the panel for surveillance purposes, and its 73% activity should not be read as a treatment option: vancomycin is a

critically important antimicrobial in human medicine and its use in companion animals is difficult to justify outside wholly exceptional circumstances.

4.3 Imaging and clinical scoring

Radiographic and ultrasonographic findings served principally as markers of chronicity. Soft tissue swelling was universal, while joint space narrowing and osteolysis each appeared in roughly two-thirds of cases and both strongly predicted severe histological change. Ultrasonography identified effusion in 88% of dogs and fibrin or villous proliferation in close to two-thirds, supporting its role as a rapid non-invasive screening tool (21-23).

The correlation of lameness and pain scores with histological grade ($r \approx 0.69$ for both) carries the most immediate practical value of any finding in this study. Where advanced imaging and biopsy are unavailable — the situation in much of the region — two simple bedside scores identify the animals most likely to have advanced joint damage and to need urgent lavage and aggressive therapy. It should be said plainly, however, that correlation of this strength describes a useful clinical signal, not a diagnostic test; roughly half the variance in histological grade remains unexplained.

4.4 Histological predictors of failure

Cartilage erosion, pannus and grade-3 synovitis were the only histological features independently predicting failure to resolve by 30 days, supporting the long-held view that these changes mark irreversible damage (26-28). Fibrosis in more than half the cohort is consistent with the chronic presentation already noted. Whether earlier presentation would have altered these outcomes cannot be determined from a retrospective design.

4.5 Platelet-rich plasma

The post-PRP histological appearances are encouraging but must be interpreted with real caution. Eleven dogs received PRP, they were not randomised, there was no parallel control group, and PRP was deliberately reserved for animals in whom infection had already been eradicated. Under those conditions the observed re-epithelialisation, reduced inflammation, subsynovial fibrosis and neoangiogenesis are compatible with the reported anabolic effects of platelet-derived growth factors on synoviocytes and chondrocytes (29-32) — but they are equally compatible with the natural course of a resolving, adequately

lavaged and successfully treated joint. Without a control arm the two explanations cannot be distinguished.

The defensible conclusion is therefore narrow: intra-articular PRP was well tolerated in this selected group, with no evidence of reactivated infection. No claim of efficacy is made or supported. PRP is not a substitute for thorough lavage and appropriate antimicrobial therapy, and randomised controlled trials remain necessary before any clinical recommendation can follow.

4.6 Limitations

Several constraints qualify these findings.

1-The retrospective design spanning eight years produced missing data points and unavoidable referral bias toward chronic and complicated cases.

2-Anaerobic culture was unavailable, so anaerobic pathogens were almost certainly under-detected; the true polymicrobial rate is likely higher than the 3.9% recorded.

3-Molecular speciation was not performed for every staphylococcal isolate, and susceptibility relied on disk diffusion rather than minimum inhibitory concentration.

4-Events per variable were too few for multivariable modelling, so the reported odds ratios are unadjusted and may reflect confounding between correlated predictors such as lameness and pain.

5-The exceptionally high culture yield of 98% raises the possibility of selection bias in case ascertainment.

6-The PRP subgroup was small, uncontrolled and non-randomised, and follow-up beyond six weeks was not standardised.

Notwithstanding these limitations, the data provide the first local information on the aetiology and antimicrobial susceptibility of canine septic arthritis in eastern Libya, and the four-tier grading system used here can be applied in laboratories with modest resources.

5. Conclusions

Canine septic arthritis in eastern Libya is caused predominantly by coagulase-positive staphylococci, with S. pseudintermedius the single commonest pathogen and meticillin-resistant strains accounting for approximately one isolate in six. Two bedside assessments — severe lameness and high pain score — together with two radiographic signs — joint space narrowing and subchondral osteolysis — identify dogs with high-grade synovitis reliably enough to guide urgent management where advanced diagnostics are not available. Amikacin and the fluoroquinolones

retained the greatest in-vitro activity, though stewardship considerations should govern their use. Pannus, cartilage erosion and grade-3 synovitis were each associated with failure to resolve by 30 days. In carefully selected chronic cases in which infection had already been eradicated, intra-articular autologous PRP was well tolerated and accompanied by regenerative histological change; controlled trials are required before efficacy can be claimed.

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*** Authors' Contributions**

K.M. conceived the study, curated the clinical and laboratory dataset, performed the statistical analysis and microbiological work, and drafted the manuscript. H.A. performed the histopathological examinations, contributed laboratory resources and critically revised the manuscript. R.H. undertook the surgical and imaging investigations, supervised clinical management and critically revised the manuscript. All authors read and approved the final version and accept accountability for its content.

*** Declarations**

Ethics approval and consent to participate. Approved by the Institutional Animal Care and Use Committee of Omar Al-Mukhtar University (OMU-VET-2026-014). Written informed consent was obtained from every owner. Procedures complied with European Directive 2010/63/EU, the ICLAS Guidelines and the National Research Council Guide for the Care and Use of Laboratory Animals.

Consent for publication. Owners provided written consent for anonymised clinical data and images to be published.

Availability of data and materials. The dataset supporting the conclusions of this article is provided as a supplementary file.

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Competing interests. The authors declare no competing interests.

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*** Figure Legends**

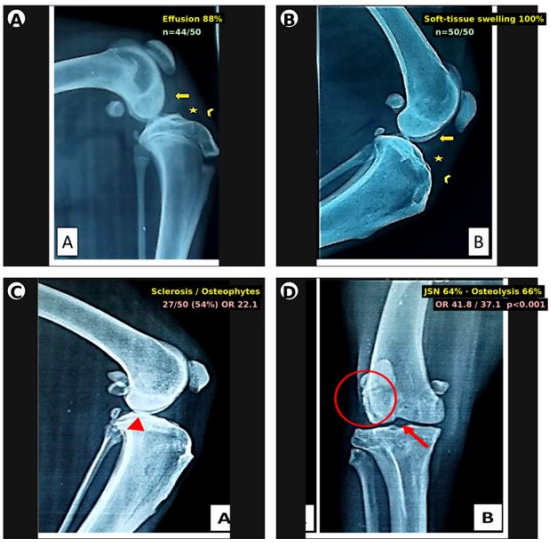

Figure 1. Mediolateral radiographs from three dogs with septic arthritis of the stifle. (A) Acute case with marked soft tissue swelling and effusion displacing the infrapatellar fat pad (arrows). (B) Chronic case at two months showing joint space narrowing, subchondral osteolysis and periosteal reaction. (C) Chronic case at three months with severe joint space loss, large osteophytes and extensive osteolysis.

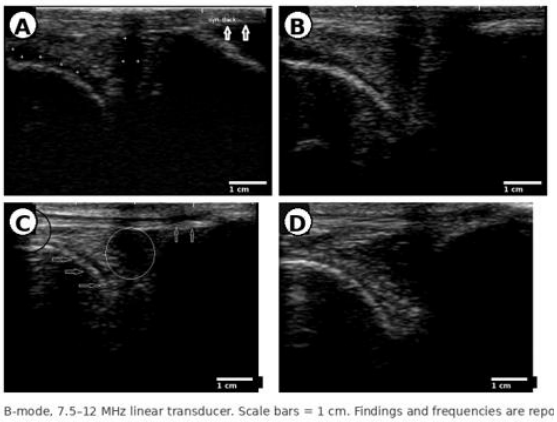

Figure 2. B-mode ultrasonographic images acquired with a 7.5–12 MHz linear transducer. (A) Anechoic effusion displacing the fat pad. (B) Synovial and capsular thickening with a hyperechoic lining. (C) Villous synovial proliferation projecting into the effusion. (D) Hyperechoic fibrin strands and septa.

Figure 3. In-vitro susceptibility of 37 non-fastidious isolates (see Results, Section 3.4).

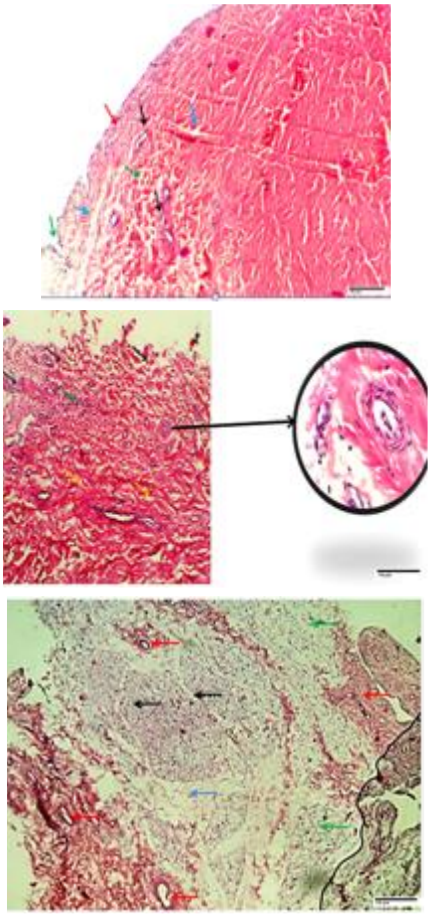

Figure 4. Synovitis grading scale (haematoxylin and eosin). Grade 0: normal lining of one to two cell layers without inflammation. Grade 1: hyperplasia to three layers with vascular congestion. Grade 2: four to six layers with villous hypertrophy, perivascular mononuclear infiltrate and focal fibrin. Grade 3: more than six layers with marked villous

hypertrophy, dense mixed infiltrate, fibrin, pannus and cartilage erosion. Scale bars 100 µm (×200).

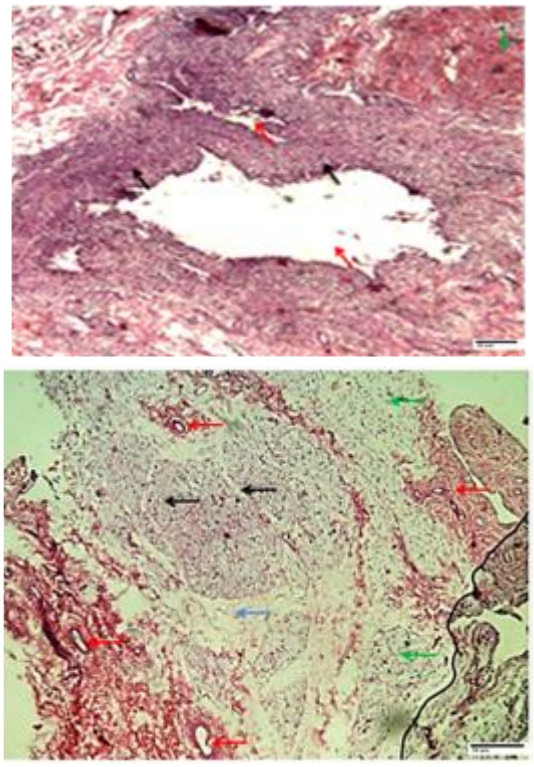

Figure 5. Principal synovial lesions (haematoxylin and eosin). (A) Vascular congestion (×200, bar 100 µm). (B) Synoviocyte hyperplasia with villous hypertrophy (×200). (C) Surface fibrin exudate (×400, bar 50 µm). (D) Subsynovial fibrosis (×100, bar 500 µm). (E) Pannus eroding articular cartilage (×200). (F) Cartilage erosion with neutrophil clusters (×400).

Figure 6. Forest plot of univariable predictors of high-grade synovitis (see Results, Section 3.6).

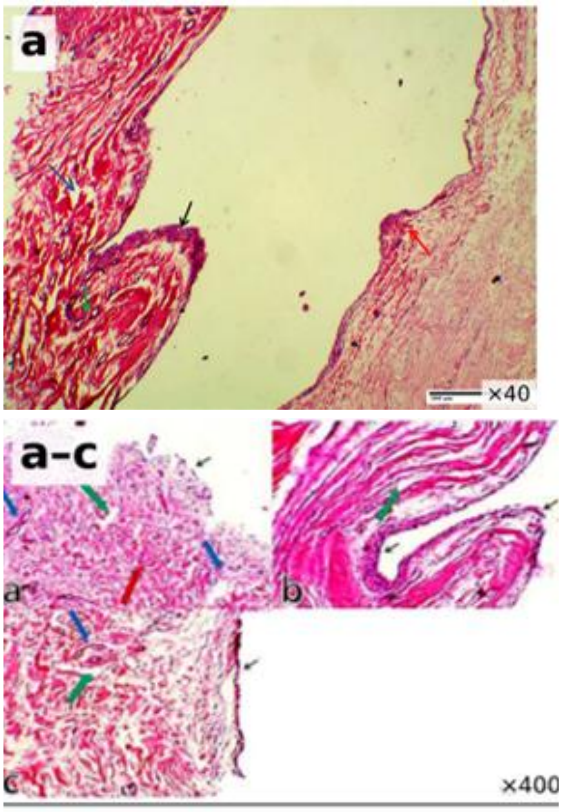

Figure 7. Synovial histology four to six weeks after intra-articular autologous PRP in 11 chronic culture-negative dogs. (A) Regenerative villous re-epithelialisation with a smooth surface (×100, bar 500 µm). (B) Higher magnification (×200) showing mild subsynovial fibrosis, small new capillaries and a sparse lymphoplasmacytic infiltrate, without fibrin, pannus or intra-synovial neutrophils.

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Formatted in Vancouver style. Every DOI and URL below was resolved and verified at the time of writing.

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