Iranian Journal of Microbiology
Volume 18 Number 1 (*February 2026*) *74-81*
DOI: http://doi.org/10.18502/ijm.v18i1.20933

ORIGINAL ARTICLE

The antimicrobial effects of cord blood-derived platelet gel on nosocomial pathogens

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Received: July 2024**, Accepted:** December 2025

ABSTRACT

Background and Objectives: Umbilical cord blood-derived platelet gel (CBPG) is rich in growth factors (GFs) and antimicrobial peptides. This study evaluated its in vitro antibacterial and antifungal activity against dominant nosocomial pathogens.

Materials and Methods: In this experimental study, CB samples were taken from 12 healthy pregnant women post-cesarean at Motahari Hospital, Jahrom. Platelet -rich plasma (PRP) was isolated using a two-step centrifugation protocol (soft-spin: 200×g, 10 min; hard spin:1000×g, 15 min, 22°C) and activated with calcium and human thrombin to form PG. Antimicrobial effect of PG was determined against *Klebsiella pneumoniae*, Methicillin-resistant *Staphylococcus aureus* (MRSA), *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, *Candida albicans*, *Aspergillus* spp., and *Penicillium* spp. using broth microdilution and time-kill assays per CLSI guidelines.

Results: PG exhibited strong bacteriostatic activity against MRSA and *K. pneumoniae* (MIC 2.2–2.8 × 10^8 platelets/mL; 1.7-1.8 $\log_{10}$ reduction at 24 h, $p < 0.001$), while PRP was moderately active and PPP was ineffective. No significant activity was observed against *P. aeruginosa* or *A. baumannii* (p= 0.2). PG showed sustained fungistatic effects (MIC: 1.9-4.2× 10^8 platelets/mL up to 72 h).

Conclusion: CBPG exhibits potent bacteriostatic and fungistatic effects, particularly against MDR Gram-positive bacteria, offering a novel autologous antimicrobial.

Keywords: Antifungal agents; Anti-bacterial agents; Platelet rich plasma; Microbial sensitivity tests; Umbilical cord blood

INTRODUCTION

Platelet gel (PG), also known as PRP gel, consists of concentrated platelets from blood and is widely used in surgical fields to treat tissue defects, particularly chronic non-healing wounds, and to promote bone regeneration (1). PG accelerates endothelial and epithelial regeneration, stimulates angiogenesis, enhances collagen synthesis, counteracts the inhibitory effects of glucocorticosteroids on wound healing, reduces pain and inflammation, and exhibits antimicrobial properties. PG clinical applications include facial plastic surgery, chronic wound management, general surgery, ophthalmology, urology, orthopedics, and burn treatment (2-4).

PG is formed by mixing PRP with thrombin derived from human plasma. PRP is an autologous concentrate of platelets in a small plasma volume.

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Thrombin induces platelet degranulation, releases GFs such as epidermal GF, fibroblast GF, hepatocyte GF, insulin-like GF, platelet-derived GF, and transforming GF-β, and vascular endothelial GF (1-3). These GFs, stored in platelet alpha-granules and produced by endothelial cells and macrophages, promote cell migration to injury sites and stimulate proliferation, and play a critical role in tissue repair (5).

Beyond GFs, platelet granules contain bioactive molecules including catecholamines, serotonin, adenosine triphosphate, von Willebrand factor (vWF), and antimicrobial peptides such as β-defensin-3, connective tissue-activating peptide-3, fibrinopeptides A and B, platelet factor-4, RANTES, and thymosin-β4. Platelets also exhibit antibacterial activity by migrating toward the chemoattractant N-formyl-methionyl-leucyl-phenylalanine (fMLP) and generating reactive oxygen species (hydrogen peroxide and superoxide). In vitro studies have confirmed efficacy of these peptides against *Escherichia coli*, *S. aureus*, *C. albicans*, *Cryptococcus neoformans*, and *P. aeruginosa* (6-8).

Nosocomial infections, acquired in healthcare settings within 48 h of admission, three days post-discharge, or 30 days after surgery, remain a major challenge (9). Common bacterial pathogens include *Pseudomonas*, *Staphylococcus*, and *Enterococcus* species, while *C. albicans* and *Cryptococcus* spp. are the prevalent fungi (10). Given its antimicrobial properties, PG is considered a promising agent for preventing postoperative and burn-related infections (6).

PG can also be derived from the umbilical CB. Studies indicate that CBPG contains higher levels of platelet GFs and demonstrates superior tissue repair efficacy compared to peripheral blood-derived PG (4, 11, 12). Despite this potential, umbilical CB is typically discarded, and limited research exists on the antimicrobial properties of CBPG (12, 13). Given the rising prevalence of multidrug-resistant (MDR) pathogens in infected wounds, together with recent clinical evidence demonstrating accelerated healing of antimony-resistant cutaneous leishmaniasis lesions using autologous PG (14), CBPG may represent a novel, ethically abundant, and biologically enhanced dual-action therapeutic with both regenerative and antimicrobial properties.

This study evaluates the antibacterial and antifungal effects of CBPG against dominant nosocomial pathogens. Nosocomial infections pose a global health threat, particularly with MDR pathogens. PRP and its gel form contain antimicrobial peptides, GFs, and leukocytes, conferring regenerative and microbicidal properties (15, 16). This is the first study to systematically evaluate CBPG as a standalone antimicrobial agent against dominant nosocomial bacteria and fungi, leveraging its higher platelet and GFs content compared to adult peripheral blood.

MATERIALS AND METHODS

Ethics statements. This study was approved by the Ethics Committee of Jahrom University of Medical Sciences under the code of IR.JUMS.REC.1398.014, and was conducted in full compliance with the Declaration of Helsinki. Written informed consent was obtained from all volunteers before enrollment. All samples were anonymized, and no personal identifiers were retained. Laboratory procedures adhered to institutional biosafety guidelines to ensure safe handling, storage, and disposal of biological materials.

Healthy subjects. Twelve healthy pregnant women (aged 20-35 years, full-term pregnancies, undergoing cesarean delivery) were recruited at Motahari Hospital, Jahrom. Inclusion criteria: Normal Complete Blood Count (CBC), hemoglobin level ≥ 11 g/dL, no fever, no antiplatelet and fibrinolytic medications, and no antibiotics 14 days prior. The exclusion criteria consisted of abnormal screening tests (HIV, HBV, HCV, syphilis, coagulation profile), platelet disorders, premature rupture of membranes, maternal infection, and a small volume of CB obtained (Accepted volume: ≥70 mL CB).

Cord blood collection and processing. CB samples were collected immediately post-cesarean section from 12 healthy full-term pregnant women at Motahari Hospital, Jahrom, Iran. CB samples were obtained via gravity drainage from the umbilical vein into 250 mL sterile collection bags pre-filled with 35 mL citrate phosphate dextrose anticoagulant (Pall Corporation, USA) (7). To prevent citrate toxicity due to the low blood volume and maintain a safe blood to CPD ratio of more than 5:1, 25 mL of plasma was added to the collection bag immediately after sampling and before centrifugation. All procedures were performed under aseptic conditions within 2 h of collection at 4°C.

PRP and PG preparation. PRP was prepared using a double-centrifugation protocol as described by Parazzi et al (6). The collected CB was centrifuged in a soft-spin model (200×g, 10 minutes, 22°C). The upper layer containing platelets was then transferred to the side bag, and after re-centrifugation at hard spin (1000×g, 15 minutes, 22°C), the upper layer of the plasma was transferred to the third bag as PPP. The platelet pellet at the bottom of the bag was re-suspended in 5 mL plasma to yield PRP (Fig. 1). To obtain active thrombin, PPP (5 vol) was mixed with 0.2 mol/L calcium gluconate (1 vol) and kept at 22°C for 30 minutes. After clot formation, the sample was centrifuged at 2000×g for 10 minutes, and the thrombin-containing supernatant was collected (14). Upon addition of thrombin (1 vol) and calcium gluconate (1.5 vol) to PRP (3 vol), a thick adhesive gel formed immediately (Fig. 1). The gel was incubated at 37°C for 30 minutes to release GFs (14).

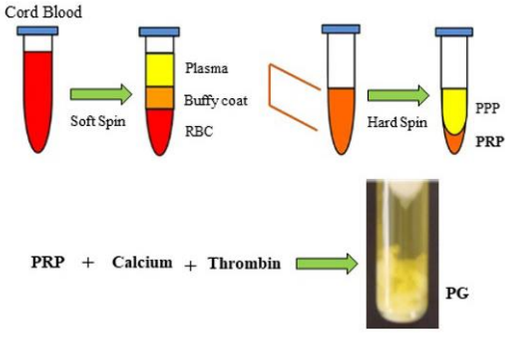

Fig. 1. Preparation of CB-derived PRP and PG by double centrifugation and calcium/thrombin activation.

Antimicrobial assays. MRSA, *K. pneumoniae*, *A. baumannii*, *P. aeruginosa*, *C. albicans*, *Aspergillus* spp., and *Penicillium* spp. were obtained from the Microbiological Laboratory, Jahrom University of Medical Sciences, Jahrom, Iran. Reference strains (MRSA ATCC 43300, *P. aeruginosa* ATCC 27853, *K. pneumoniae* ATCC 10031, *A. baumannii* ATCC 19606, *Candida krusei* ATCC 6258, *Paecilomyces variotii* ATCC 22319, and *Candida parapsilosis* ATCC 22019) were provided by the Pasteur Institute of Iran, Tehran, and used as controls. All bacterial isolates were subcultured on the Mueller-Hinton Agar (MHA) and incubated at 37°C for 24 h before testing.

Time-kill assay. Mid-logarithmic-phase bacteria were adjusted to a final concentration of 1×10^6 CFU/mL in 4 mL Mueller-Hinton broth (MHB). Then, 250 µL of PG, PRP, PPP, or phosphate-buffered saline (PBS; negative control) was added to 250 µL of the inoculated MHB in sterile tubes. A positive control consisted of 1×10^6 CFU/mL bacteria in MHB without any CB-derived component. All assays were performed in duplicate. Culture media were incubated at 37°C with shaking at 200 rpm on an orbital shaker. Aliquots of 50 µL were withdrawn at 0, 4-, 8-, 12-, and 24-h post-inoculation. Each aliquot was serially diluted 10-fold in 0.9% saline, and 10 µL of the appropriate dilutions were spread onto MHA plates. Following overnight incubation at 37°C, viable colonies were enumerated to calculate CFU/mL.

Bacterial log reduction was calculated as: $\log_{10}$ (CFU/mL at time 0) − $\log_{10}$ (CFU/mL at 24 h). Results were compared between test components and the positive control (16).

Broth microdilution assay. The antibacterial activity of CB components was determined using the standard broth microdilution method according to CLSI M07-A10 guidelines. Bacterial strains were subcultured on MHA and incubated for 18 h at 37°C to obtain isolated colonies. A single colony was suspended in sterile saline and adjusted to a 0.5 McFarland standard (approximately 1×10^8 CFU/mL) using a spectrophotometer at 600 nm.

The suspension was further diluted in MHB to achieve a final inoculum concentration of 1×10^6 CFU/mL. PG, PRP, and PPP were serially diluted twofold in MHB. In standard flat-bottom 96-well microtiter plates, 100 µL of diluted CB component was added to each well, followed by 10 µL of the bacterial inoculum.

Positive control wells contained 100 µL MHB and 10 µL inoculum (no CB component). Negative control wells contained 100 µL MHB and 10 µL CB component (no bacteria). Plates were sealed with adhesive film and incubated at 37°C on an orbital shaker (100 rpm for 24 h). Absorbance was read at 600 nm using a microplate reader at 12 and 24 h. The minimum inhibitory concentration (MIC) was recorded as the lowest platelet count of PG, PRP, or PPP showing no visible growth or $OD_{600} < 0.05$ compared to the negative control (17).

Antifungal susceptibility testing. Broth microdilution was used to determine the MIC according to CLSI M27-A3 for yeast strains and M38-A2 for mold isolates. *Candida*, *Aspergillus*, and *Penicillium* were

tested against CB-derived components (PG, PRP, PPP). Test compounds were first dissolved in DMSO and then diluted 50-fold in RPMI-1640 medium (with glutamine and MOPS buffer) to achieve the working concentration. Flat-bottom microdilution plates containing 100 µL of diluted test component were inoculated with 100 µL of fungal suspension prepared from 48-hour Sabouraud dextrose agar cultures. Fungal cells were suspended in sterile distilled water, and turbidity was adjusted spectrophotometrically per CLSI M27-A3 and M38-A2. Quality control strains included *Candida krusei* (ATCC 6258), *Paecilomyces variotii* (ATCC 22319), and *Candida parapsilosis* (ATCC 22019) for each new MIC plate series. Negative controls contained the test component and medium without fungi. Plates were sealed and incubated at 35°C for 60 h. MIC was determined visually as the lowest concentration with no significant growth after 48 h. Susceptibility and resistance of isolates were evaluated based on CLSI M27-A3 guidelines.

Statistical analysis. Statistical analyses were performed using SPSS-26 (IBM, USA). Mean ± standard deviation was reported for all quantitative variables. Repeated-measures analysis of variance (ANOVA) assessed time-dependent changes (e.g., in the time-kill assay). Paired t-test compared treatment groups (PG, PRP, PPP vs. controls). Pearson's correlation coefficient evaluated the relationship between platelet concentration and antimicrobial activity. Graphs were generated using GraphPad prism8 (GraphPad Software, USA). P-values < 0.05 were considered statistically significant.

RESULTS

Sample characteristics. Twelve pregnant donors (mean age ± SD: 25.1 ± 6.4 years) with normal bleeding times participated in this study. Maternal hemoglobin was 11.9 ± 0.9 g/dl, and a baseline platelet count was 240 ± 66 ×10³/µL. The mean collected CB volume was 93.3± 16.8 mL, yielding 35.7 ± 5.2 mL PPP and 15.8 ± 3.1 mL PRP. Leukocyte and platelet concentrations in whole CB, PRP, and PPP are shown in Table 1.

Time-kill assay. Time-kill kinetics revealed that CBPG exerted strong bacteriostatic activity against both MRSA and *K. pneumoniae*, achieving a rapid >1.7 $\log_{10}$ CFU/mL reduction within the first 4 h that was sustained through 24 h ($p < 0.001$). PRP showed moderate inhibition (0.4-0.8 log reduction), whereas PPP had a negligible effect. In contrast, none of the CB components significantly inhibited *P. aeruginosa* (transient effect only at 4-8 h, p= 0.02) or *A. baumannii* (no inhibition at any time point, p= 0.2), with bacterial counts exceeding 8.6 $\log_{10}$ CFU/mL by 24 h in all treatment groups (Fig. 2).

Table 1. Platelet and leukocyte count in CB, PRP, and PPP (Data shown as mean ± SD)

Variable	CB	PRP	PPP
Platelet count (10^6/mL)	250 ± 90	1960 ± 230	20 ± 6
Leukocyte count (10^6/mL)	11 ± 3.7	27 ± 6.2	< 0.1

Comparative analysis between clinical isolates and ATCC standard strains demonstrated highly consistent responses (Table 2). The initial bacterial load was uniformly 6.0 $\log_{10}$ CFU/mL across all strains. No statistically significant differences were observed in MIC values or log reduction at 24 h (1.7-1.9 log for susceptible pathogens; >2.6 log growth for resistant pathogens) or overall killing kinetics ($p > 0.05$, unpaired t-test; Table 2) between clinical and standard strains, indicating reproducible and strain-independent antimicrobial efficacy of PG.

Broth microdilution assay. Broth microdilution assay revealed that CBPG exhibited potent inhibitory activity against MRSA and *K. pneumoniae* with MIC values of 2.2 ± 0.3 and 2.8 ± 0.4 × 10^8 platelets/mL, respectively. PRP showed moderate activity (MIC 4.1–5.3 × 10^8 platelets/mL), whereas PPP was ineffective (MIC > 8.0 × 10^8 platelets/mL). No inhibition was observed against *P. aeruginosa* or *A. baumannii* by any product (MIC > 8.0 × 10^8 platelets/mL) (Table 3).

Antifungal susceptibility testing. The broth microdilution assay demonstrated strong fungistatic activity of PG against all tested fungi. The lowest MIC values were recorded for *C. albicans* (1.9 ± 0.3 × 10^8 platelets/mL at 48 h), followed by *Penicillium* spp. (3.5 ± 0.5 × 10^8 platelets/mL) and *Aspergillus* spp. (4.2 ± 0.6 × 10^8 platelets/mL at 72 h). PRP exhibited moderate and transient inhibition (MIC 3.8–>8.0 × 10^8 platelets/mL), while PPP was ineffective (MIC > 8.0 × 10^8 platelets/mL). PG completely prevented visible growth and regrowth up to 72 h in all cases (Table 3).

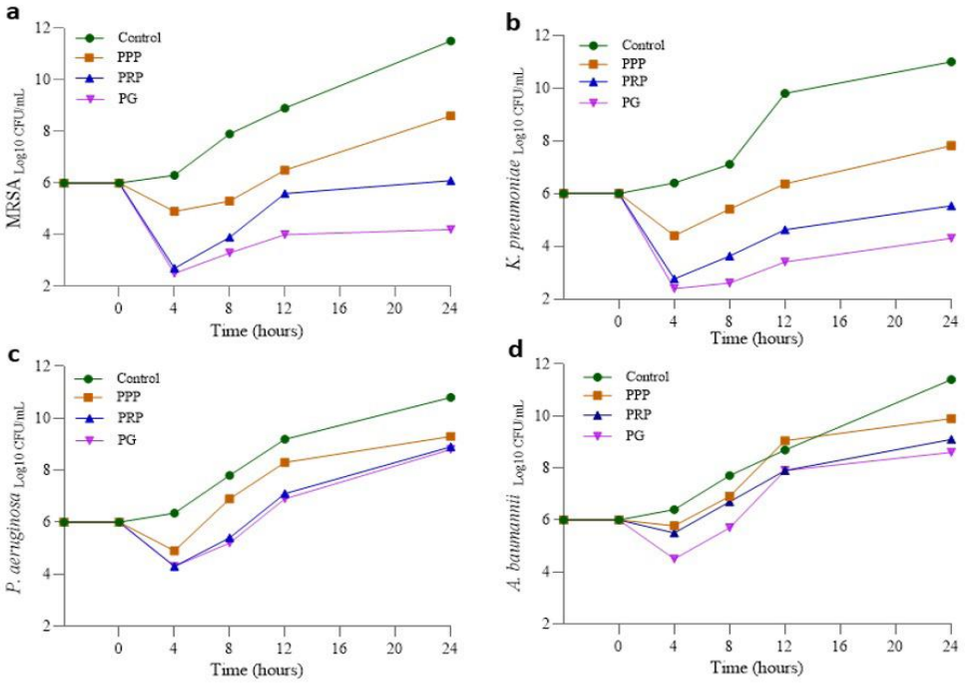

Fig. 2. Time-kill curves of PG, PRP, PPP, and untreated control against MRSA (a), *K. pneumoniae* (b), *P. aeruginosa* (c), and *A. baumannii* (d). Initial inoculum: 1×10^6 CFU/mL (6.0 $\log_{10}$ CFU/mL). Data represent mean ± SD.

Table 2. Antimicrobial activity of CBPG against clinical isolates and ATCC reference strains. Time-kill data ($\log_{10}$ CFU/mL) from an initial inoculum of 1×10^6 CFU/mL (6.0 log). $\Delta Log = \log_0 - \log_{24}$; Data shown as mean ± SD, No significant difference ($p > 0.05$, t-test)

Strain	Type	Log_{10} CFU/mL		ΔLog at 24 h (vs. 0 h)	p-value
		at 0 h	at 24 h		
S. aureus	Clinical	6.0	4.2	+ 1.8 (Reduction)	0.73
	ATCC 43300	6.0	4.3	+1.7	
P. aeruginosa	Clinical	6.0	8.8	- 2.8 (Growth)	0.87
	ATCC 27853	6.0	8.9	- 2.9	
K. pneumoniae	Clinical	6.0	4.3	+ 1.7 (Reduction)	0.78
	ATCC 10031	6.0	4.4	+ 1.6	
A. baumannii	Clinical	6.0	8.6	- 2.6 (Growth)	0.85
	ATCC 19606	6.0	8.8	- 2.8	

DISCUSSION

This investigation represents the first comprehensive in vitro assessment of CBPG as a leukocyte-rich, autologous antimicrobial agent against key nosocomial pathogens, including multidrug-resistant bacteria and fungi, utilizing standardized CLSI broth microdilution and time-kill assays with direct platelet count-based MIC reporting (5). By leveraging the unique bioactive profile of umbilical CB, characterized by 3-5-fold higher concentrations of antimicrobial peptides and GFs than adult peripheral blood, CBPG demonstrated selective bacteriostatic and fungistatic efficacy, offering a promising alternative to conventional antimicrobials in wound management (6, 7).

The time-kill kinetics revealed robust inhibition of MRSA and *K. pneumoniae* by CBPG, with >1.7 $\log_{10}$

Table 3. MIC of CB products against nosocomial pathogens determined by broth microdilution. MIC is expressed as the lowest platelet count (×10^8/mL), preventing visible growth after 24 h (Data shown as mean ± SD).

Strain	Components	MIC (× 10^8 platelets/mL)	Interpretation
S. aureus	PG	2.2 ± 0.3	No growth ≥ 2.2 × 10^8
	PRP	4.1 ± 0.4	No growth ≥ 4.1 × 10^8
	PPP	> 8.0	Growth
P. aeruginosa	PG	> 8.0	Growth
	PRP	> 8.0	Growth
	PPP	> 8.0	Growth
K. pneumoniae	PG	2.8 ± 0.4	No growth ≥ 2.8 × 10^8
	PRP	5.3 ± 0.5	No growth ≥ 5.3 × 10^8
	PPP	> 8.0	Growth
A. baumannii	PG	> 8.0	Growth
	PRP	> 8.0	Growth
	PPP	> 8.0	Growth
C. albicans	PG	1.9 ± 0.3	No growth ≥ 1.9 × 10^8; sustained to 72 h
	PRP	3.8 ± 0.4	Inhibition to 48 h; regrowth at 72 h
	PPP	> 8.0	Growth from 4 h
Aspergillus spp.	PG	4.2 ± 0.6	No conidia/mycelia ≥ 4.2 × 10^8
	PRP	> 8.0	Partial inhibition to 24 h; growth at 72 h
	PPP	> 8.0	Uninhibited growth
Penicillium spp.	PG	3.5 ± 0.5	No growth ≥ 3.5 × 10^8 up to 72 h
	PRP	> 8.0	Growth from 12 h
	PPP	> 8.0	Growth from 4 h

CFU/mL reduction sustained over 24 h, aligning with the bacteriostatic threshold. This effect was dose-dependent, as evidenced by MIC values of 2.2–2.8 × 10^8 platelets/mL, where no visible growth occurred, confirmed by negative subcultures. PRP exhibited moderate activity (0.4–0.8 log reduction; MIC 4.1-5.3 × 10^8 platelets/mL), while PPP failed to inhibit growth, consistent with the critical role of platelet alpha-granule-derived peptides like thrombocidins, β-defensins, and PF-4 in disrupting bacterial membranes (1, 10). The leukocyte-rich composition of our CBPG preparation (27 ± 6.2 × 10^6 leukocytes/mL) further amplified efficacy, as neutrophils and monocytes contribute via phagocytosis and reactive oxygen species, explaining the superior performance relative to leukocyte-depleted PRP in prior studies (12, 13). Notably, no regrowth was observed in susceptible strains, underscoring CBPG's potential for preventing biofilm formation in chronic wounds (16).

In contrast, CBPG was ineffective against Gram-negative *P. aeruginosa* and *A. baumannii* (MIC > 8.0 × 10^8 platelets/mL; log change –2.6 to –2.8, indicating growth), with bacterial counts exceeding 8.6 $\log_{10}$ CFU/mL by 24 h. This Gram-negative selectivity is well-documented, as outer membrane lipopolysaccharide in *P. aeruginosa* and efflux pumps in *A. baumannii* confer resistance to cationic antimicrobial peptides (18, 19). Our results extend these findings by demonstrating that even high platelet concentrations (up to 8 × 10^8/mL) fail to overcome this barrier, highlighting the need for combination therapies, such as CBPG with efflux inhibitors, in polymicrobial infections (15).

Fungistatic evaluation further highlighted CBPG versatility, with MICs of 1.9-4.2 × 10^8 platelets/mL preventing hyphal transition and conidial germination in *C. albicans*, *Aspergillus* spp., and *Penicillium* spp. up to 72 h. PRP provided transient inhibition (MIC > 8.0 × 10^8 platelets/mL for molds), while PPP was inert. This sustained fungistatic effect surpasses previous PRP reports, likely due to synergistic leukocyte-platelet interactions releasing cathelicidins and histatins, which target fungal ergosterol membranes (11, 13). The lower MIC for *C. albicans* (1.9 × 10^8 platelets/mL) indicates that its yeast-like morphology is more susceptible to platelet defensins than

filamentous molds (10).

The consistent efficacy across clinical isolates and ATCC strains validates CBPG reproducibility, independent of resistance profiles, positioning it as a reliable topical agent for nosocomial wound infections where conventional antifungals fail due to biofilm penetration issues (16). Compared to synthetic antimicrobials, CBPG's autologous nature minimizes the development of resistance and immunogenicity, with potential cost savings in burn care (1, 18). However, leukocyte enrichment, while enhancing activity, may introduce pro-inflammatory cytokines, warranting future optimization via differential centrifugation (12).

Although this study demonstrates promising antimicrobial activity, several limitations warrant consideration. CBPG exhibited only bacteriostatic/fungistatic effects without complete pathogen eradication. The in vitro design failed to replicate the complex wound microenvironment, including pH variations, exudate, biofilm formation, and host immune system interactions. Moreover, platelet and leukocyte concentrations were not systematically controlled, which limited the precise assessment of their contribution to the observed efficacy.

CONCLUSION

In conclusion, CBPG proved to be a safe, autologous, and highly effective antimicrobial agent, delivering strong and sustained bacteriostatic activity against MRSA and *K. pneumoniae*, while exhibiting robust fungistatic effects against *C. albicans*, *Aspergillus* spp., and *Penicillium* spp. With its naturally enriched GFs and leukocyte content, CBPG represents an innovative and promising topical therapy for combating MDR bacterial and fungal infections in challenging wound environments. Future clinical trials should explore its efficacy in vivo, optimizing leukocyte content for broader spectrum coverage.

ACKNOWLEDGEMENTS

The authors gratefully acknowledge Jahrom University of Medical Sciences for financial support and sincerely thank the mothers who generously donated cord blood for this research.

REFERENCES

1. Everts P, Onishi K, Jayaram P, Lana JF, Mautner K. Platelet-Rich Plasma: New Performance Understandings and Therapeutic Considerations in 2020. *Int J Mol Sci* 2020; 21: 7794.
2. Hesseler MJ, Shyam N. Platelet-rich plasma and its utility in medical dermatology: a systematic review. *J Am Acad Dermatol* 2019; 81: 834-846.
3. Zheng W, Zhao DL, Zhao YQ, Li ZY. Effectiveness of platelet rich plasma in burn wound healing: a systematic review and meta-analysis. *J Dermatolog Treat* 2022; 33: 131-137.
4. Hasiba-Pappas SK, Tuca AC, Luze H, Nischwitz SP, Zrim R, Geißler JCJ, et al. Platelet-Rich Plasma in Plastic Surgery: A Systematic Review. *Transfus Med Hemother* 2022; 49: 129-142.
5. Yeaman MR. Platelets in defense against bacterial pathogens. *Cell Mol Life Sci* 2010; 67: 525-544.
6. Parazzi V, Lazzari L, Rebulla P. Platelet gel from cord blood: a novel tool for tissue engineering. *Platelets* 2010; 21:549-554.
7. Rebulla P, Pupella S, Santodirocco M, Basso L, Nardini P, Salvadeo A, et al. Multicentre standardisation of a clinical grade procedure for the preparation of allogeneic platelet concentrates from umbilical cord blood. *Blood Transfus* 2016; 14: 73-79.
8. Moojen DJ, Everts PA, Schure RM, Overdevest EP, van Zundert A, Knape JT, et al. Antimicrobial activity of platelet-leukocyte gel against *Staphylococcus aureus. J Orthop Res* 2008; 26: 404-410.
9. Revelas A. Healthcare - associated infections: A public health problem. *Niger Med J* 2012; 53: 59-64.
10. Fabbro MD, Bortolin M, Taschieri S, Ceci C, Weinstein RL. Antimicrobial properties of platelet-rich preparations. A systematic review of the current pre-clinical evidence. *Platelets* 2016; 27: 276-285.
11. Czakai K, Dittrich M, Kaltdorf M, Müller T, Krappmann S, Schedler A, et al. Influence of Platelet-rich Plasma on the immune response of human monocyte-derived dendritic cells and macrophages stimulated with *Aspergillus fumigatus. Int J Med Microbiol* 2017; 307: 95-107.
12. Castro AB, Herrero ER, Slomka V, Pinto N, Teughels W, Quirynen M. Antimicrobial capacity of Leucocyte-and Platelet Rich Fibrin against periodontal pathogens. *Sci Rep* 2019; 9: 8188.
13. Zheng L, Duan Z, Tang D, He Y, Chen X, Chen Q, et al. GP IIb/IIIa-Mediated Platelet Activation and Its Modulation of the Immune Response of Monocytes Against *Candida albicans. Front Cell Infect Microbiol* 2021; 11: 783085.
14. Shadmand E, Solhjoo K, Taghipour A, Tayer AH, Sadeghi F, Meshkin A. Healing effects of autologous

platelet gel and growth factors on cutaneous leishmaniasis wounds in addition to antimony; a self-controlled clinical trial with randomized lesion assignment. *BMC Res Notes* 2023; 16: 200.
15. Nurden AT. Platelets, inflammation and tissue regeneration. *Thromb Haemost* 2011;105 Suppl 1:S13-33.
16. Ladhani HA, Yowler CJ, Claridge JA. Burn wound colonization, infection, and sepsis. *Surg Infect (Larchmt)* 2021; 22: 44-48.
17. Wiegand I, Hilpert K, Hancock REW. Agar and broth dilution methods to determine the minimal inhibitory concentration (MIC) of antimicrobial substances. *Nat Protoc* 2008; 3: 163-175.
18. Tang R, Wang S, Yang J, Wu T, Fei J. Application of platelet-rich plasma in traumatic bone infections. *Expert Rev Anti Infect Ther* 2021; 19: 867-875.
19. Zhang W, Guo Y, Kuss M, Shi W, Aldrich AL, Untrauer J, et al. Platelet-Rich Plasma for the Treatment of Tissue Infection: Preparation and Clinical Evaluation. *Tissue Eng Part B Rev* 2019; 25: 225-236.