



# Extracorporeal hemoperfusion as an adjunctive therapy for liver failure

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## Abstract

Liver failure, whether due to alcoholic liver disease or hepatic malignancy, often leads to acute-on-chronic liver failure (ACLF), necessitating liver transplantation. A major challenge in managing these patients is the acute decompensation phase, during which hepatic toxins and inflammatory mediators accumulate, resulting in multi-system dysfunction. While liver transplantation remains the most effective treatment, artificial liver support systems, particularly extracorporeal blood purification, offer potential benefits. Hemoperfusion, utilizing solute adsorption via a solid agent, provides an alternative to conventional dialysis, which is limited by membrane permeability. In this case series, we report our experience using HA330 hemoperfusion in combination with standard therapy for liver failure patients treated at our single-center hospital from 2023 to 2024. Extracorporeal blood purification with an adsorptive cartridge (HA330, Jaftron, China) was employed as an adjunctive strategy to standard care. Our findings suggest that hemoperfusion may serve as a valuable supportive therapy for liver failure patients requiring transplantation. However, larger cohort studies are necessary to confirm its safety and efficacy.

**Keywords** Hemoperfusion · Liver transplantation · Extracorporeal blood purification · Critical care

## Introduction

Acute-on-chronic liver failure (ACLF) and acute liver failure (ALF) represent critical conditions marked by rapid hepatic decompensation, systemic toxin accumulation, and multi-organ dysfunction. Ammonia, bilirubin, and inflammatory cytokines (e.g., TNF- $\alpha$ , IL-6) drive complications such as hepatic encephalopathy, hepatorenal syndrome, and coagulopathy, with mortality exceeding 50% in patient's

ineligible for liver transplantation (LT) [1–4]. While LT remains definitive therapy, organ shortages and delayed eligibility necessitate bridging strategies to stabilize patients during acute decompensation.

Conventional extracorporeal liver support systems aim to mitigate toxins and inflammation. Albumin-dependent methods like the Molecular Adsorbent Recirculating System (MARS) and Prometheus effectively clear protein-bound toxins but face limitations, including high costs, albumin loss, and inadvertent antibiotic removal [5, 6]. Plasma exchange (PEX) replenishes coagulation factors and modulates immunity but requires substantial resources. Bioartificial liver systems, though innovative in replicating synthetic liver functions, remain experimental due to safety concerns and logistical barriers [7, 8].

Adsorption therapies offer a pragmatic alternative by directly targeting toxins and cytokines via resins or activated carbon, bypassing albumin dependency. Cytosorb, widely used in hyperinflammatory states (e.g., sepsis, COVID-19), reduces IL-6 and myoglobin but faces scrutiny over cost, variable efficacy, and unintended nutrient/antibiotic adsorption [9–11]. Similarly, the HA330 hemoperfusion cartridge, a styrene-based adsorbent, addresses

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both detoxification and immunomodulation by selectively removing bilirubin, ammonia, and cytokines (e.g., IL-6, TNF- $\alpha$ ) [11–17]. Unlike albumin dialysis, HA330 operates without exogenous albumin, minimizing resource strain. However, evidence for HA330 remains sparse compared to established systems, with unresolved challenges in protocol standardization, cost-effectiveness, and therapeutic drug monitoring (18, 19).

This case series evaluates the HA330 hemoperfusion system in ACLF patients, emphasizing its dual capacity to reduce hepatic toxins and attenuate inflammation as an adjunct to standard care. We focused on ACLF due to its characteristic hyperinflammatory pathophysiology, for which cytokine adsorption is particularly suited, and because it represents a common clinical scenario where bridging strategies to transplantation are urgently needed. By examining real-world outcomes, including biochemical improvement and bridging success to LT, we aim to address gaps in evidence for adsorption therapies and refine their role in liver failure management.

## Materials and methods

This retrospective case series evaluated the efficacy of HA330 hemoperfusion as an adjunctive therapy in patients with acute-on-chronic liver failure (ACLF) admitted in Tehran Pars Hospital between 2023 and 2024. Written informed consent was obtained from all patients or their legal representatives.

### Patient selection

Inclusion criteria comprised a diagnosis of ACLF based on EASL-CLIF criteria, severe hepatic decompensation (total bilirubin  $>15$  mg/dL, INR  $>2.0$ ), and at least one extrahepatic organ failure (e.g., renal dysfunction, hepatic encephalopathy). Patients were excluded for active hemorrhage, hemodynamic instability (MAP  $<60$  mmHg despite vaso-pressors), terminal malignancy, or severe cardiopulmonary compromise precluding vascular access.

### Intervention protocol

Hemoperfusion was performed using Jaftron HA330 (cytokine adsorption) and BS330 (broad-spectrum toxin removal) cartridges, selected based on clinical presentation:

- Dual-filter (HA330+BS330): For hyperinflammation (CRP  $>150$  mg/L, ferritin  $>1000$  ng/mL).
- BS330 alone: For predominant toxin accumulation (e.g., severe jaundice).

Vascular access was achieved via a femoral double-lumen Sheldon catheter (12 Fr). Each session lasted 4–6 h, repeated daily until clinical improvement (e.g.,  $\geq 50\%$  bilirubin reduction, improved GCS). Anticoagulation with unfractionated heparin targeted an ACT of 180–220 s.

## Adjunctive therapies

Standard care included lactulose (30 mL q6h), neomycin (500 mg q8h), therapeutic paracentesis (2–4 L/day), and 20% albumin (40 g/day). Organ support measures included CRRT (B-BRAUN Diapact<sup>®</sup>) for hepatorenal syndrome, broad-spectrum antibiotics for infections, and platelet transfusions for severe thrombocytopenia ( $<20,000/\mu\text{L}$ ).

## Data collection and outcomes

Primary outcomes were biochemical improvement ( $\geq 50\%$  reduction in bilirubin/CRP) and successful bridging to liver transplantation. Secondary outcomes included 90-day mortality and adverse events. Clinical (GCS, hemodynamics) and laboratory (bilirubin, INR, CRP, creatinine) parameters were monitored pre- and post-therapy. Safety outcomes encompassed catheter-related complications, bleeding, and hemodynamic instability.

## Statistical analysis

Descriptive statistics (mean, range) summarized demographic and laboratory data. Paired t-tests compared pre- and post-therapy values (GraphPad Prism v9.0). Given the small sample size ( $n=3$ ), results were interpreted as exploratory.

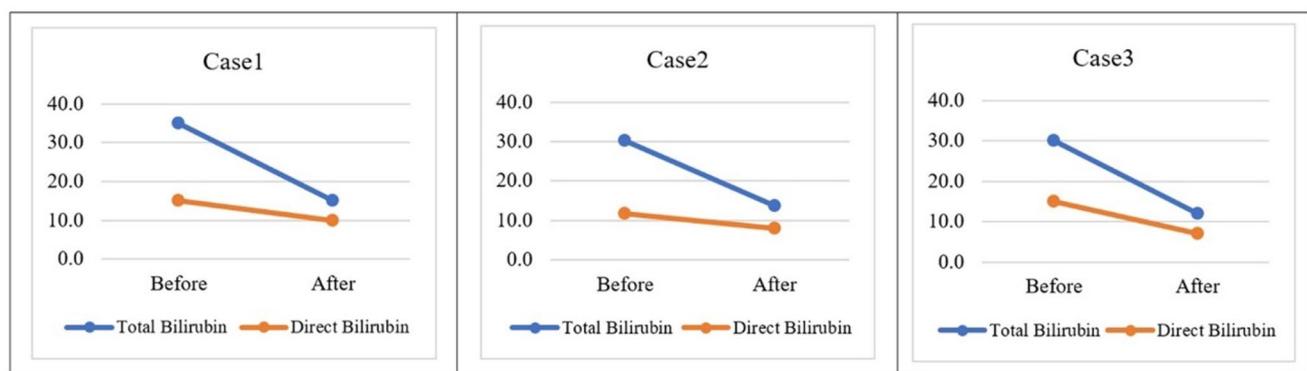
## Results

### Cohort characteristics and clinical presentation

Our study evaluated three male patients (mean age 49.3 years, range 32–66) with acute-on-chronic liver failure treated with hemoperfusion, including two cases of alcoholic cirrhosis and one hepatocellular carcinoma (Table 1). All patients presented with the classic ACLF triad: severe hepatic dysfunction (mean total bilirubin 31.7 mg/dL), profound coagulopathy (INR  $>2.9$ ), and encephalopathy requiring intensive care (Table 1). The admission laboratory profile revealed concurrent multi-organ involvement, with markedly elevated inflammatory markers (CRP  $200\pm 25$  mg/L, ESR  $110\pm 20$  mm/hr) and renal impairment (mean creatinine 1.87 mg/dL).

**Table 1** Laboratory findings in three cases of liver disease with systemic complications

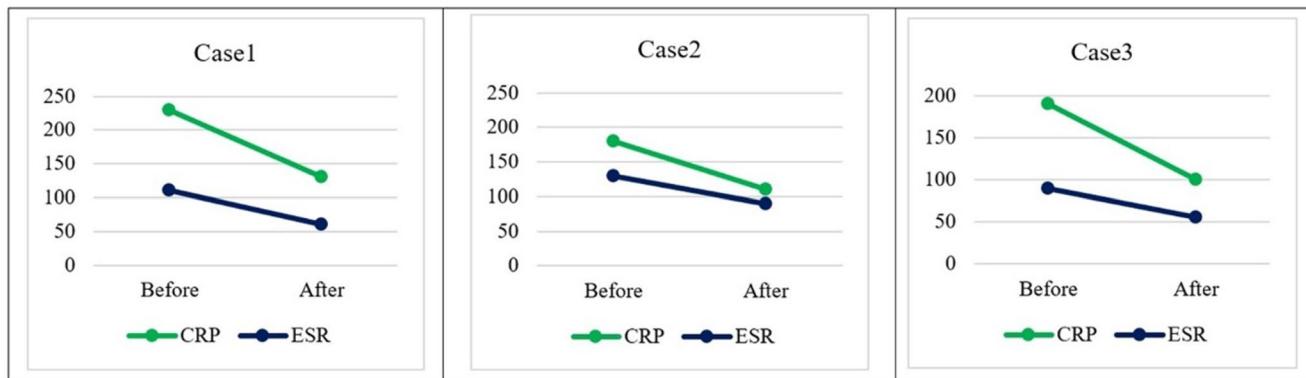
Parameter	Case 1		Case 2		Case 3	
	Before	After	Before	After	Before	After
<i>Demographics</i>						
Age (years)	32	32	66	66	50	50
Gender	Male	Male	Male	Male	Male	Male
History	Alcoholic cirrhosis		Hepatocellular carcinoma		Alcoholic cirrhosis	
Number of HP Sessions	3		4		5	
<i>Liver function</i>						
Total Bilirubin (mg/dL)	35	15	30.1	13.7	30	12
Direct Bilirubin (mg/dL)	15	10	11.7	7.9	15	7
ALT (U/L)	1600	250	25	7	1000	150
AST (U/L)	1800	300	41	11.9	1100	100
INR	>4	2	3	2	1.8	1.4
<i>Inflammation/Infection</i>						
CRP (mg/L)	230	130	180	110	190	100
Procalcitonin (ng/mL)	4	1	2	1	3	0.5
Ferritin (ng/mL)	2100	800	1500	780	1200	400
ESR (mm/hr)	110	60	130	90	90	56
<i>Coagulation/Hematology</i>						
Platelet count ( $\times 10^3/\mu\text{L}$ )	70	60	40	20	120	80
D-dimer (ng/mL)	5600	1800	3700	1600	2500	800
<i>Renal function</i>						
Creatinine (mg/dL)	1.7	1.2	2.0	1.3	1.9	0.9
BUN (mg/dL)	70	50	100	70	130	70
<i>Other</i>						
LDH (U/L)	1400	700	1900	1000	2400	1350
WBC ( $\times 10^3/\mu\text{L}$ )	20	12	1.7	2.1	18	14
APACHE II score	25	—	28	—	30	—

**Fig. 1** Total and Direct Bilirubin in three cases before and after intervention

### Biochemical and inflammatory response to hemoperfusion

Hemoperfusion therapy produced significant multisystem improvements (Figs. 1 and 2). Total bilirubin decreased by 57% (31.7 to 13.6 mg/dL), and direct bilirubin declined by 40% (13.9 to 8.3 mg/dL) across the cohort (Fig. 1). Transaminase levels (ALT/AST) fell by 75–90% in Cases 1 and 3. Inflammatory markers responded robustly, with CRP declining by 43% (200 to 113.3 mg/L) and ESR improving by

38% (110 to 68.7 mm/hr) (Fig. 2). Renal function normalized (creatinine: 1.87 to 1.13 mg/dL; BUN: 100 to 63.3 mg/dL), and coagulopathy improved (mean INR: >2.9 to 1.8).



**Fig. 2** CRP and ESR in three cases before and after intervention

## Clinical outcomes and case highlights

### Case 1: acute-on-chronic liver failure managed with dual hemoperfusion

A 32-year-old male with a history of alcoholic cirrhosis presented to our institution with acute hepatic decompensation manifesting as severe jaundice and progressive encephalopathy requiring emergent intubation. Initial evaluation revealed profound coagulopathy and systemic inflammation, with laboratory markers consistent with acute-on-chronic liver failure (detailed in Table 1). The severity of his presentation, particularly his markedly elevated INR ( $>4$ ) and inflammatory markers, prompted initiation of an innovative dual hemoperfusion strategy combining Jaftron BS330 (for hepatic toxin removal) and HA330 (for cytokine adsorption) filters.

The patient demonstrated a remarkable response to therapy within 48 h, with objective improvement in both biochemical parameters (Table 1) and clinical status. He received a total of 3 hemoperfusion sessions. Most notably, we observed complete resolution of encephalopathy and significant correction of coagulopathy. Concurrent management included judicious administration of fresh frozen plasma, therapeutic paracentesis (removing 2 L of ascitic fluid), and standard medical therapy including lactulose and antibiotics.

This therapeutic approach yielded such significant clinical improvement that the patient regained full neurological function and was ultimately discharged for outpatient liver transplantation evaluation. The case provides compelling clinical evidence for the potential utility of combined BS330/HA330 hemoperfusion in addressing the dual pathology of toxin accumulation and systemic inflammation characteristic of acute-on-chronic liver failure.

The rapid resolution of both biochemical abnormalities and clinical symptoms suggests this approach may offer

advantages over conventional therapies, particularly in bridge-to-transplant scenarios.

### Case 2: hepatocellular carcinoma with acute-on-chronic liver failure and hepatorenal syndrome

A 66-year-old male with hepatocellular carcinoma presented to our ICU with severe hepatic decompensation manifesting as profound encephalopathy requiring intubation and refractory ascites necessitating repeated paracenteses. Initial laboratory evaluation (Table 1) revealed marked hyperbilirubinemia (total bilirubin 30.1 mg/dL), renal dysfunction (creatinine 2.0 mg/dL), and significant thrombocytopenia (40,000/ $\mu$ L), complicating vascular access. The clinical picture was consistent with acute-on-chronic liver failure complicated by hepatorenal syndrome, evidenced by rising creatinine despite maintained urine output.

Given the multisystem involvement, we initiated an aggressive multimodal approach combining continuous renal replacement therapy (B-BRAUN) with Jaftron BS330 hemoperfusion. The treatment protocol included daily therapeutic paracentesis with albumin replacement, high-protein enteral nutrition, and G-CSF administration for concomitant leukopenia. Remarkably, despite the patient's severe thrombocytopenia (which later required platelet transfusion), the 5-hour hemoperfusion sessions were well-tolerated without hemodynamic instability.

Therapeutic response was evidenced by significant improvement in both hepatic (bilirubin decreased to 13.7 mg/dL) and renal parameters, along with successful extubation and reduced ascites accumulation. This patient underwent 4 hemoperfusion sessions. This case highlights the potential of combined CRRT and BS330 hemoperfusion in managing complex hepatorenal syndrome, even in patients with significant thrombocytopenia. Following clinical stabilization, the patient was discharged for liver transplantation evaluation and oncologic follow-up, demonstrating the viability of this approach as a bridge to definitive therapy.

### Case 3: alcoholic cirrhosis with multi-organ failure complicated by influenza myocarditis

A 50-year-old male with alcoholic cirrhosis presented with acute encephalopathy (GCS 9) subsequently complicated by influenza-associated myocarditis and progressive liver failure. Initial evaluation (Table 1) revealed severe hepatocellular injury ( $AST/ALT > 1000$  U/L), marked hyperbilirubinemia (30 mg/dL total), and evidence of systemic inflammation, compounded by cardiac dysfunction requiring inotropic support with dobutamine and norepinephrine.

The complex pathophysiology prompted a comprehensive treatment strategy addressing both viral etiology and end-organ dysfunction. Antiviral therapy with oseltamivir was supplemented by IVIG administration (20 g) for immunomodulation, while dual hemoperfusion (BS330 and HA330 filters with DPMAS) targeted both hepatic toxin clearance and cytokine storm mitigation. This approach was particularly crucial given the concurrent cardiac and hepatic failure, where conventional therapies would have been insufficient.

After one week of intensive therapy, which included a total of 5 hemoperfusion sessions, we observed dramatic improvement across all organ systems: hepatic parameters normalized substantially (bilirubin decreased to 12 mg/dL,  $AST/ALT < 150$  U/L), cardiac function stabilized, and mental status recovered completely. This remarkable recovery underscores the potential of combined hemoperfusion strategies in managing multi-organ failure, even when complicated by viral myocarditis. The successful ICU discharge with near-complete functional recovery suggests such approaches may expand therapeutic options for critically ill patients with limited conventional treatment alternatives.

Two patients (Cases 1 and 3) achieved clinical stability sufficient for transplant evaluation. In contrast, Case 2 (with hepatocellular carcinoma) succumbed to delayed cardiac complications (a fatal arrhythmia) despite initial biochemical stabilization, highlighting the prognostic influence of underlying etiology.

### Adverse events

Therapy was well-tolerated, with no catheter-related complications or hemodynamic instability. Thrombocytopenia (40,000/ $\mu$ L in Case 2) required platelet transfusion but did not preclude hemoperfusion.

These biochemical improvements correlated with clinical recovery in all patients. Renal function normalized (creatinine: 1.87 to 1.13 mg/dL; BUN: 100 to 63.3 mg/dL; Table 1), and coagulopathy improved (mean INR: >2.9 to 1.8). Both cirrhotic patients (Cases 1 & 3) achieved sufficient recovery for transplant evaluation, while the patient

with hepatocellular carcinoma (Case 2) ultimately died from cardiac complications (fatal arrhythmia) despite initial biochemical stabilization (Table 1), highlighting how underlying etiology and comorbidities influence ACLF prognosis even after successful hemoperfusion.

## Discussion

Acute-on-chronic liver failure presents a complex clinical challenge characterized by rapid hepatic decompensation, systemic inflammation, and multi-organ dysfunction. The management of ACLF complicated by hepatorenal syndrome (HRS) remains a formidable challenge, with high mortality rates despite advances in critical care [20]. Our case series contributes to the growing body of evidence supporting extracorporeal hemoperfusion as a viable bridge therapy in this critically ill population. The observed biochemical improvements, particularly the 57% reduction in bilirubin and 43% decline in CRP, demonstrate the dual efficacy of HA330/BS330 hemoperfusion in addressing both hepatic toxin accumulation and systemic inflammation. These findings align with recent studies highlighting the role of adsorption therapies in modulating the hyperinflammatory state of ACLF. Ronco and Bellomo's comprehensive reviews emphasize hemoperfusion's capacity to remove protein-bound toxins and cytokines, a mechanism particularly relevant in liver failure where conventional dialysis often falls short, while their technical insights into modern hemoperfusion systems underscore its evolving role in critical care [21]. Our cases further validate this approach, with two patients achieving sufficient clinical stability for transplant evaluation, while the third—a patient with hepatocellular carcinoma—succumbed to cardiac complications, underscoring the impact of underlying etiology on outcomes. However, hemoperfusion's persistent challenges, including limited adsorption of mid-molecular-weight toxins (e.g., ammonia) and biocompatibility issues, which may explain the partial response observed in our hepatocellular carcinoma case (Case 2), are consistent with broader limitations noted in ICU-based studies [22]. Furthermore, the risk of nonspecific drug adsorption, particularly antibiotics—a concern highlighted in sepsis trials using cytokine adsorption [23]—remains critical for optimizing therapeutic protocols. Prospects such as hybrid systems combining adsorption with dialysis or bioartificial components, as proposed in maintenance hemodialysis populations [24], could address these limitations, potentially enhancing multi-organ support in complex ACLF cases like our influenza myocarditis patient (Case 3).

The observed reductions in CRP and bilirubin in our report parallel findings from studies evaluating combined

hemodialysis and hemoperfusion in multiple organ dysfunction syndrome (MODS), where dual therapy attenuated systemic inflammation by lowering pro-inflammatory cytokines (e.g., IL-6, TNF- $\alpha$ ) and suppressing nuclear transcription factors such as NF- $\kappa$ B and STAT3. These transcription factors regulate downstream inflammatory and apoptotic pathways, and their inhibition has been linked to improved organ function in MODS [22]. In our ACLF patients, HA330/BS330 hemoperfusion likely exerted similar immunomodulatory effects, disrupting the cytokine-driven feedback loops that perpetuate hepatic injury and multi-organ failure. The synergy between toxin adsorption (BS330) and cytokine removal (HA330) mirrors the complementary mechanisms of hemodialysis and hemoperfusion in MODS, where small-molecule clearance and middle/large-molecule adsorption collectively mitigate systemic toxicity [24]. Notably, the rapid resolution of encephalopathy and coagulopathy in our cases may reflect not only toxin removal but also dampened NF- $\kappa$ B/STAT3 signaling, which is implicated in blood-brain barrier disruption and hepatic apoptosis. These parallels underscore the broader applicability of adsorption therapies in hyperinflammatory critical illnesses, as evidenced by cytokine adsorption's efficacy in sepsis and cardiac surgery [23], though further research is needed to elucidate the molecular interplay between extracorporeal support and transcriptional regulation in ACLF.

However, our results must be interpreted in the context of ongoing controversies in artificial liver support systems. Compared to albumin-dependent systems like MARS, hemoperfusion offers potential advantages in cost and simplicity by eliminating the need for exogenous albumin, a benefit also observed in therapeutic plasma exchange (PEX) studies [25]. However, unlike PEX, which requires substantial resources and carries risks of fluid imbalance, hemoperfusion provides targeted detoxification without depleting coagulation factors. The underlying etiology significantly influences outcomes, as seen in our hepatocellular carcinoma case (Case 2) with malignancy-associated ACLF showing less durable responses. The dual-filter approach (HA330 + BS330) appeared particularly effective in hyperinflammatory cases, supporting emerging evidence that combined cytokine and toxin removal may be superior to toxin clearance alone, a strategy increasingly advocated in hybrid extracorporeal therapies [21].

## Conclusion and future directions

This case series demonstrates the potential utility of HA330/BS330 hemoperfusion as an adjunctive therapy for stabilizing patients with ACLF during acute decompensation. The observed biochemical improvements alongside

clinical recovery in two of three patients underscore hemoperfusion's dual capacity to mitigate hepatic toxin accumulation and systemic inflammation. The rapid resolution of encephalopathy and coagulopathy in alcoholic cirrhosis cases highlights its role as a bridging strategy to liver transplantation, particularly in hyperinflammatory states. However, outcomes varied with underlying etiology, as seen in the hepatocellular carcinoma case, emphasizing that hemoperfusion's efficacy may depend on disease-specific pathophysiology. The therapy's favorable safety profile, with no major adverse events, further supports its feasibility in critically ill cohorts.

Future research should prioritize larger, multicenter studies to validate these findings and establish standardized protocols for filter selection, session duration, and anti-coagulation. Hybrid systems combining adsorption with dialysis or bioartificial components could address limitations in mid-molecular-weight toxin clearance and mitigate unintended drug adsorption. Investigations into cost-effectiveness compared to albumin-dependent systems (e.g., MARS) and therapeutic plasma exchange are warranted to optimize resource allocation. Mechanistic studies exploring hemoperfusion's impact on transcriptional regulators (e.g., NF- $\kappa$ B/STAT3) may elucidate its immunomodulatory effects, while randomized trials comparing hemoperfusion to conventional therapies could refine its role in ACLF management. Finally, prognostic biomarkers to identify patients most likely to benefit from adsorption therapies—particularly those with hyperinflammatory phenotypes—are essential for personalized treatment strategies.

In conclusion, extracorporeal hemoperfusion represents a promising adjunct in ACLF care, but its integration into clinical practice requires robust evidence and innovation to overcome existing limitations. Collaborative efforts across critical care, hepatology, and bioengineering disciplines will be pivotal in advancing this field.

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## Declarations

**Conflict of interest** No potential conflict of interest relevant to this article was reported.

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