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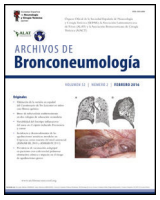

Case Report

Challenges and Limitations of Extracorporeal Membrane Oxygenation Therapy in Severe Paraquat Poisoning: An Analysis of Unsuccessful Cases

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We report a case series of four men aged 21–43, referred to the respiratory intensive care unit for paraquat (PQ) poisoning. The patients ingested 50–70 mL of a 20% PQ solution and presented with symptoms within 2.5–13.5 h, with plasma PQ concentrations ranging from 3.5 mg/L to 11.5 mg/L upon admission (Table 1). They received immediate symptomatic and supportive treatments, including gastric lavage with activated charcoal, antioxidants like vitamin E, and immunosuppressive therapy with cyclophosphamide, methylprednisolone, and dexamethasone. Precautions were taken to protect the stomach and maintain cardiovascular stability. Hemoperfusion therapy was performed until plasma PQ levels were undetectable, followed by at least 12 h of continuous veno-venous hemofiltration therapy for all patients. Despite these measures, all patients developed respiratory failure requiring extracorporeal membrane oxygenation (ECMO).

Following PQ ingestion, the four patients experienced varied clinical courses (Table 1). Patient 1, with advancing pulmonary fibrosis, required intubation and invasive mechanical ventilation (IMV) on day 3, and despite ECMO on day 4, succumbed to cardiac failure on day 19 (Fig. S1). Patient 2, with stable arterial blood gas (ABG) until day 4, received ECMO on day 5, and developed systemic inflammatory response syndrome and hepatic failure, leading to death on day 16 after poisoning (Fig. S2). Patient 3, with severe ABG on day 4, underwent intubation and IMV, then ECMO on day 5, but gastrointestinal hemorrhage ensued, resulting in ECMO removal on day 8 and death on day 10 (Fig. S3). Patient 4, with worsening dyspnea and ABG on day 3, was stabilized initially with ECMO but later developed septic multiorgan dysfunction on day 12, dying from cardiac failure on day 21 (Fig. S4).

PQ poisoning induces systemic toxicity through the generation of reactive oxygen species, leading to multiorgan involvement. Conventional treatments have not been able to halt the lung fibrosis progression after PQ poisoning, death can occur anywhere between 5 and 31 days after lung injury.[1] In large academic centers, ECMO has become a preferred treatment strategy for critically ill poisoned patients where standard resuscitative therapy or antidotes have not been effective, aiming to preserve organ perfusion and allow time for clearance of toxins.[1,2] As lung transplantation (LT) is the only curative treatment for end-stage PQ-induced pulmonary fibrosis, ECMO has been reported to offer a temporary bridge to transplantation.[3–5] However, can the routine implementation of ECMO in the management of PQ poisoning yield therapeutic benefits for patients?

While ECMO can temporarily improve oxygenation, our findings suggest that it does not prevent the progression to organ failure in PQ poisoning (Figs. S1–S4). The uniformly poor outcomes in this series indicate that ECMO may not be beneficial in the setting of progressive multiple organ failure. Overall, considering other reports,[3–5] in cases where other organ failures are manageable and patients are accepted for transplant, ECMO may provide a temporary bridge to transplantation. This is particularly relevant given the resource-intensive nature of ECMO and the need for careful consideration of its use in the context of PQ poisoning.

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Table 1
Clinical Profiles of the Four Patients Undergoing Extracorporeal Membrane Oxygenation Following Fatal Paraquat Poisoning.

	Patient 1	Patient 2	Patient 3	Patient 4
Age (years)	35	21	43	37
Gender	Male	Male	Male	Male
Ingestion amount (mL)	70	50	–	–
Poisoning to admission time (h)	13.5	4	6	2.5
Plasma PQ concentration at admission (mg/L)	3.5	7.3	4.5	11.5
Hospital stays (days)	19	16	10	21
CRRT	HP + CVVH	HP + CVVH	HP + CVVH	HP + CVVH
Gastric lavage	Yes	Yes	Yes	Yes
Intubation and invasive mechanical ventilation	Yes	No	Yes	No
Clinical manifestations before ECMO				
Dyspnea	Yes	Yes	Yes	Yes
Creatinine (μmol/L)	190	121	–	201
PaO_2 (mmHg)	39	56	17	58
$PaCO_2$ (mmHg)	45	37	25	46
pH	7.48	7.42	7.53	7.44
WBC ($\times 10^9$/L)	–	16.63	9.57	19.29
Indications of ECOMO	Respiratory failure	Respiratory failure	Respiratory failure	Respiratory failure
ECOMO characteristics				
Poisoning to ECMO time (days)	4	5	5	3
ECMO pattern	V–V	V–V	V–V	V–V
Bypass	RFV-LFV	RFV-LFV	RFV-LFV	RFV-LFV
Blood flow rate (mL/kg)	60–80	60–80	60–80	60–80
FiO_2	100%	100%	100%	100%
Speed (R/min)	3000	3000	3000	3000
ECMO complications	–	Infection	GIT hemorrhage	Multiorgan dysfunction
ECMO to death time (days)	15	11	3	18
Death reasons	Cardiac failure	SIRS and hepatic failure	GIT hemorrhage	Cardiac failure

CRRT: continuous renal replacement therapy; CVVH: continuous veno-venous hemofiltration; HP: hemoperfusion; ECMO: extracorporeal membrane oxygenation; FiO_2: fraction of inspired oxygen; LFV: left femoral vein; PaO_2: partial pressure of oxygen; $PaCO_2$: partial pressure of carbon dioxide; RFV: right femoral vein; SaO_2: oxygen saturation; WBC: white blood count; V–V: venous–venous; SIRS: systemic inflammatory response syndrome; GIT: gastrointestinal tract.

Conflict of Interests

The authors state that they have no conflict of interests.

Appendix A. Supplementary Data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.arbres.2024.03.004.

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