

Veno-Venous ECMO: An Alternative Strategy for Acute Respiratory Failure After High-Voltage Electrocution. The Utility of Point-of-Care Tests

Annalisa Boscolo^{a, c}, Elisabetta Saraceni^a, Stefano Dal Cin^a, Giulia Sartori^a, Carlo Ori^b, Sandra Rossi^a

Abstract

We report an extraordinary case of a 43-year-old man who sustained high-voltage electrocution injury associated with severe pulmonary damage due to current flow through the tissue. For the first time, a veno-venous extracorporeal membrane oxygenation (ECMO) was successfully used to provide respiratory support during severe hypoxemia without any limitation for surgical wound excision and homologous skin transplantation. To prevent over-bleeding due to surgery, daily medications and heparin infusion aggregometry and thromboelastometry were used as new point-of-care tests. After more than 400 hours, the patient returned to conventional ventilation with a significant improvement of gas exchanges, total pulmonary restore and without thromboembolic complications. Based on our experience, maximum clot firmness (MCF) in FIBTEM is usually high. Our finding shows that fibrinogen deficiency is not a leading mechanism for bleeding in burn patients also during ECMO; they need plasma transfusion preferably. About whole blood impedance aggregometry, thrombin receptor activating peptide 6-test (AUC) is strongly correlated to surgical bleeding and platelet consumption. We suggest that a rapid correction of coagulopathy, using ROTEM and MULTIPATE, helps to minimize allogeneic blood products and to avoid thromboembolic complications during ECMO treatment and surgical burn wound excision.

Keywords: Electrocution; Extracorporeal membrane oxygenation; Thromboelastometry, Aggregometry

Introduction

Electrical injury is a very serious event, accounting for ap-

proximately 5% of admissions to major burn centers. The incidence of electrical injuries increases as adults enter the workplace. More than 90% of victims are men. Factors that determine the damage depend on magnitude of energy, type of current and duration of contact. The classic injury pattern develops when the body becomes part of a circuit and is usually associated with entrance and exit wounds. The clinical manifestations of electrical injuries range from mild superficial skin burns to severe multiorgan dysfunction and death [1]. Visceral damage only depends on the duration of exposure to current flow, its pathway through the tissue and the resistivity of the organ [2]. We report an exceptional case of an electrical burn that affected the lung parenchyma mainly and the skin only marginally. The main complication was a severe hypoxemic and hypercapnic respiratory failure which did not respond to conventional ventilation. A veno-venous extracorporeal membrane oxygenation (ECMO) became necessary to provide respiratory support, adequate oxygenation and carbon dioxide removal for more than 400 h, until the total pulmonary restore. Severe hemostatic alterations, due to burn wounds and heparin infusion (correlated to ECMO), were managed using point-of-care thromboelastometry and platelet aggregometry, minimizing unnecessary pro-coagulant interventions as recommended by Cochrane analysis and the updated European guideline for trauma [3, 4].

Case Report

A 43-year-old male sustained high-voltage electrocution injury associated with a pulmonary electrocution during a work accident. He was intubated at the referring hospital because of difficult breathing and transferred to the Burn Center at the Hospital of Padua.

Patient's burn wounds were cleaned and dressed in operative room (OR) before the patient was transferred to the intensive care unit (ICU). He was resuscitated based on the Parkland formula (4 mL/kg/% total body surface area (TBSA) burn of a crystalloid solution) and was given an infusion of C vitamin during the first 24 h [1, 2, 5, 6]. The hemodynamic monitoring was guaranteed by PICCO[®] (Pulsion Medical-Systems, Germany) to allow adequate fluid resuscitation after the first 24 h post-burn and overcome hypovolemia, to replace the massive intravascular volume loss due to thermal injury

Manuscript accepted for publication October 30, 2015

^aUOC Anesthesia and Intensive Care Unit, Hospital of Padova, 2, Giustiniani St., 35128 Padova, Italy

^bDepartment of Medicine-DIMED, UO Anesthesia and Intensive Care Unit, Padova University Hospital, 267, C. Battisti St., 35128 Padova, Italy

^cCorresponding Author: Annalisa Boscolo, UOC Anesthesia and Intensive Care Unit, Hospital of Padova, 2, Giustiniani St., 35128 Padova, Italy. Email: annalisa.boscolo@gmail.com

doi: <http://dx.doi.org/10.14740/jmc2380e>

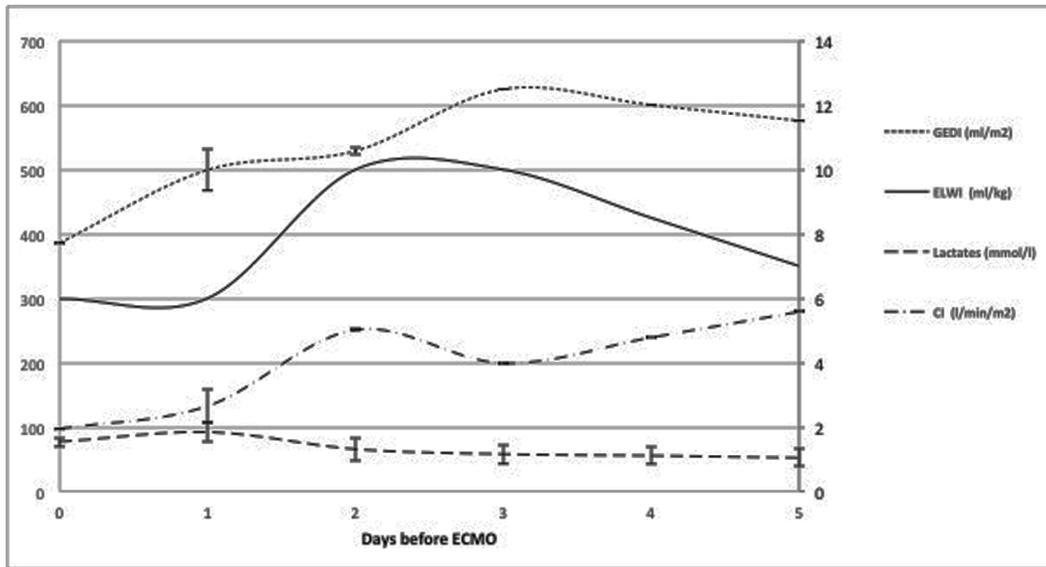


Figure 1. TV, PEEP, PaO₂/FiO₂ and mean airway pressure before ECMO. Description of ventilator parameters and oxygenation before ECMO. TV: tidal volume; PEEP: positive end-expiratory pressure; PaO₂/FiO₂: ratio of arterial oxygen partial pressure to fractional inspired oxygen.

and prevent fluid creep. The volume delivery was guided by mean blood pressure ≥ 65 mm Hg, lactate ≤ 2 mmol/L, urinary output between 0.5 and 1 mL/kg/h and invasive hemodynamic parameters as cardiac index (CI ≥ 2.5 L/min/m²), global end diastolic index (GEDI was maintained close to 680 mL/m²) and extralung water index (ELWI increased from ≤ 6 mL/m² at admission to over 10 mL/m² 5 days later, Fig. 1) according to

Sanchez-Sanchez and colleagues [6, 7]. An echocardiography excluded cardiac pathologies and shunts. At the admission in ICU PaO₂/FiO₂ ratio was 280, and the arterial CO₂ level was normal. The bronchoscopy did not reveal signs of airway damage. His respiratory system was managed with conventional ventilator methods but 2 days later, the patient suffered a progressive hypoxia (PaO₂/FiO₂ < 100) and hypercapnia (PaCO₂

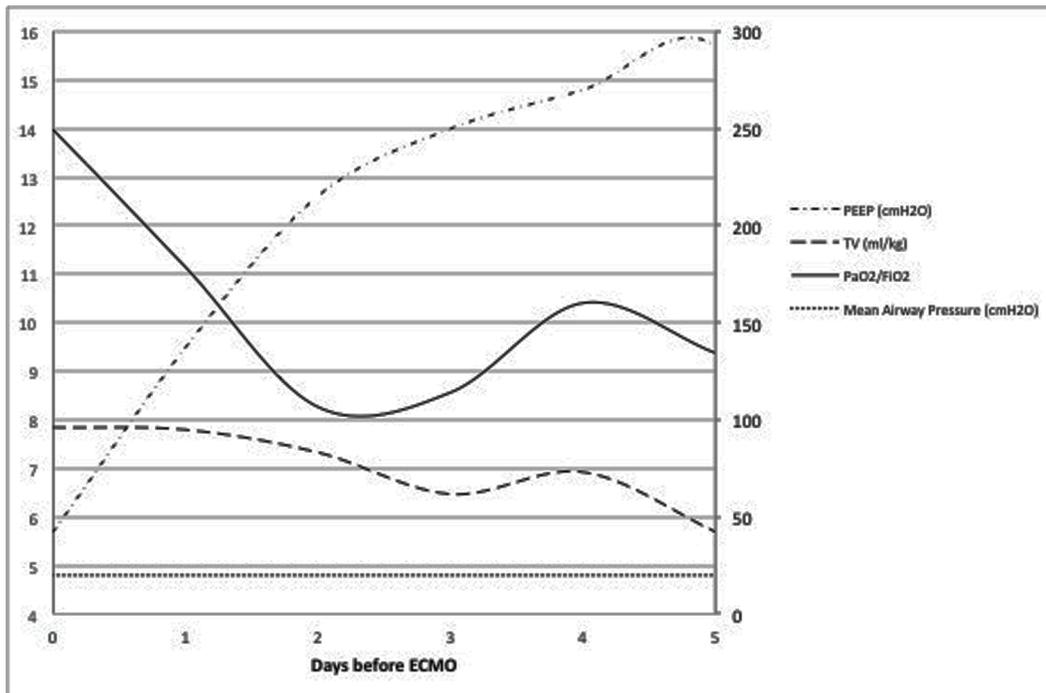


Figure 2. Hemodynamic parameters based on PICCO before ECMO. Lactates were stable while GEDI and ELWI increased gradually before ECMO (mean \pm SD). GEDI: global end diastolic index; ELWI: extralung water index; CI: cardiac index.

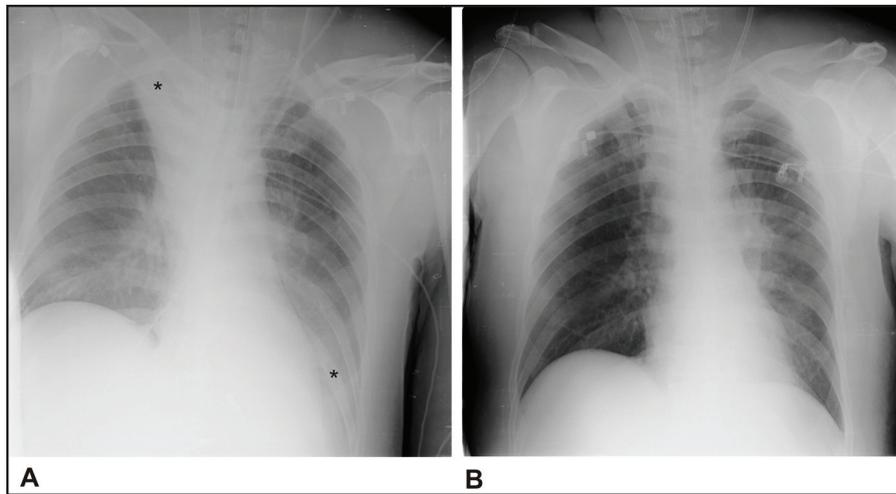


Figure 3. (A) Antero-posterior chest radiograph before ECMO. *Severe bilateral injuries. (B) Antero-posterior chest radiograph after ECMO. Regular chest XR.

high > 60 mm Hg) (Fig. 2) and an infusion of cisatracurium was started to achieve neuromuscular blockade.

Despite a protective ventilator method (ventilator setting was: $\text{FiO}_2 = 1.00$, respiratory rates = $18 \pm 2/\text{min}$, positive end-expiratory pressure (PEEP) between 10 and 15 $\text{cm H}_2\text{O}$, pressure control ventilation 22 ± 2 $\text{cm H}_2\text{O}$, VT = 6 mL/kg , peak airway pressure and mean airway pressure around 30 - 35 $\text{cm H}_2\text{O}$ and 20 $\text{cm H}_2\text{O}$ respectively), his arterial O_2 and CO_2 levels became very difficult to control (pH was 7.27, PaCO_2 was 77 mm Hg, $\text{PaO}_2/\text{FiO}_2 < 100$) (Fig. 2) [8, 9].

On post-burn day (PBD) 3, he was ventilated in prone position without any benefit and his chest X-ray and CT scan showed bilateral opacities due to the passage of electrical current (Fig. 3A, 4).

Bronchoalveolar lavage (BAL) and bacterial cultures were sterile.

Multiple bronchoscopies and recruitment maneuvers were carried out to guarantee clearance of bronchial secretions from lung but without benefits.

The patient was placed on veno-venous ECMO on PBD 5

(right internal jugular vein/femoral vein) according to CESAR trial and Extracorporeal Life Support Organization registry general guidelines [10]. The ECMO flow was maintained between 4 and 5 L/min ; gas flow was adjusted to obtain adequate PaCO_2 and PaO_2 . The ventilator setting was reduced to minimum to allow lung rest (pressure control ventilation was 14 ± 3 $\text{cm H}_2\text{O}$, TV: 6 ± 1 mL/kg , PEEP: 15 ± 2 $\text{cm H}_2\text{O}$, peak airway pressure: 28 ± 2 $\text{cm H}_2\text{O}$, mean airway pressure: 17 ± 1.5 $\text{cm H}_2\text{O}$).

To check the efficacy of heparin infusion, to prevent an over-bleeding during surgical excision of burn wounds and guide hemostatic therapy, we used aggregometry (MULTIPLATE[®]) and thromboelastometry (ROTEM[®])-based management algorithm (Fig. 5), as suggested by Schaden et al, by Cochrane analysis and the updated European guidelines for trauma [3, 4, 11-13].

In our case, a majority of viscoelastic values were within the normal range during recovery, except for maximum clot firmness (MCF) in FIBTEM usually high (MCF between 35 and 40 mm) and clot formation time (CFT) lower in INTEM

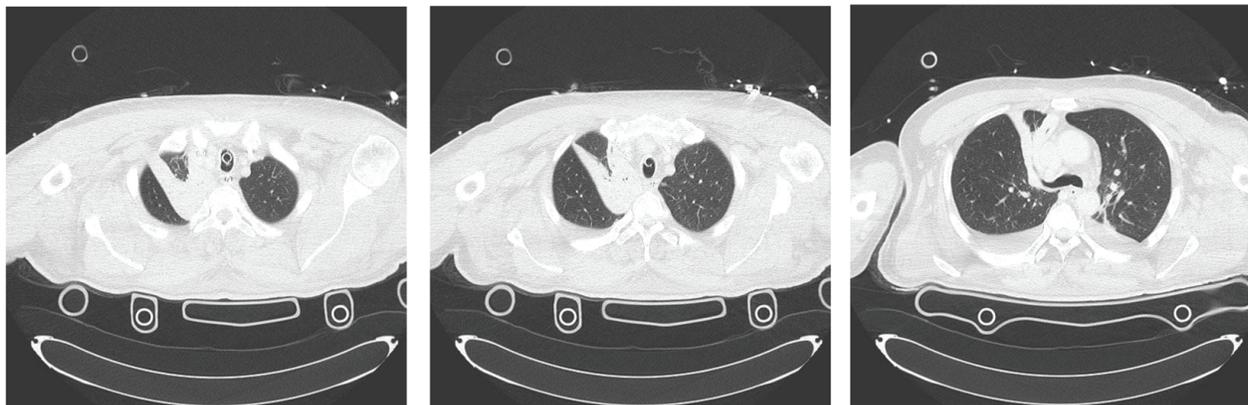


Figure 4. Three consecutive computed tomography scans show a severe lung injury due to the passage of electrical current. Entrance site was patient's right arm while and exit site was his left leg.

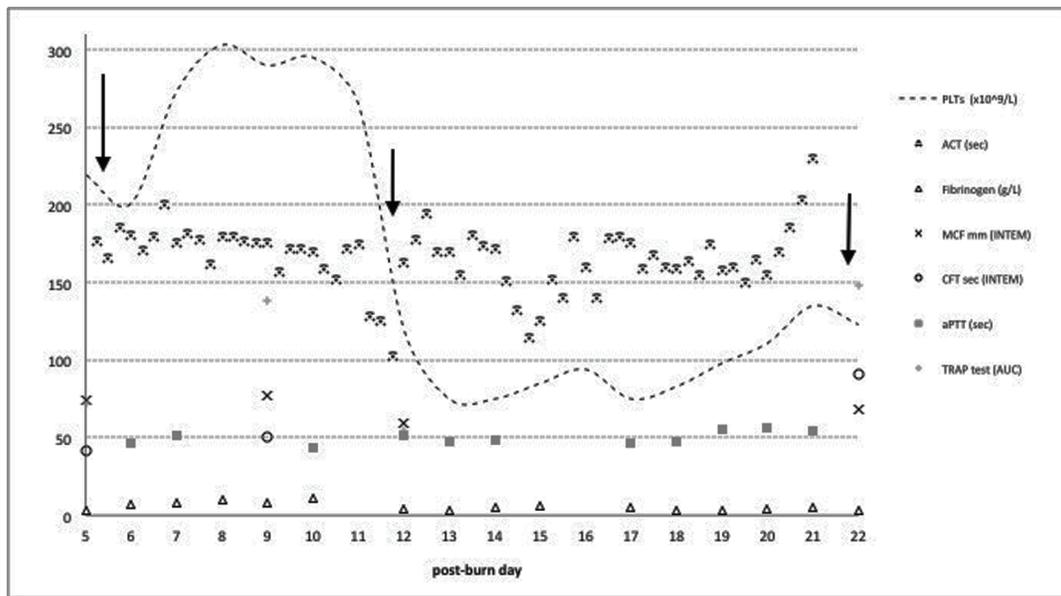


Figure 5. Conventional coagulation tests, ROTEM and MULTIPLATE during ECMO and surgery. From left to right: ↓, on PBD 5, the patient was placed on ECMO. ↓, on PBD 12, he went to OR. ↓, on PBD 22, he was decannulated. Strong correlations between MCF, CFT, ACT and aPTT. Fibrinogen was normal. TRAP test value decreased especially after surgery as well as platelet count. ECMO: extracorporeal membrane oxygenator; ACT: activated clotting time; OR: operative room; aPTT: activated partial thromboplastin time; MCF: maximum clot firmness; CFT: clot formation time; INTEM: intrinsic elastometry; TRAP: thrombin receptor activating peptide-6.

and EXTEM (CFT between 42 - 50 s and 45 - 49 s respectively) both correlated to a high plasmatic fibrinogen level (> 5 g/L). For this reason, in case of bleeding and worsening of INTEM and EXTEM values, we administered fresh frozen plasma preferably to normalize coagulation and not fibrinogen concentrate.

With respect to platelet aggregometry, thrombin receptor activating peptide-6 (TRAP) test was approximately in range during ECMO period and the area under the curve (AUC) increased significantly after decannulation (from 72 AUC with $201 \times 10^9/L$ platelets up to 148 AUC with $208 \times 10^9/L$ platelets), presumably because platelet function was affected by ECMO filter and heparin administration (10 ± 2 U/kg/h, Fig. 5) [13].

By ECMO day 7, the patient was conducted with his oxygenator in the OR to allow cleaning of wounds and homologous skin transplantation.

INTEM, EXTEM and FIBTEM were used also in OR to minimize the therapy with allogenic products and a strong correlation was noticed between TRAP test and surgical bleeding due to platelets consumption (AUC decreased to 54 for $90 \times 10^9/L$ platelets after OR, Fig. 5).

By ECMO day 17, the oxygenator setting was FiO_2 0.3, 1735 rates per minute, blood flow 1.96 L/min, gas flow 2 L/min; the ventilator was set in BI-level (high PEEP 28 cm H_2O , low PEEP 15 cm H_2O , FiO_2 0.8 and 16 rates/min), pH and $PaCO_2$ were in range and $PaO_2/FiO_2 \geq 200$. The patient was taken off extracorporeal support and returned to conventional ventilation after 405 h (Fig. 3B). The tracheotomy was placed percutaneously (Ciaglia Blue Rhino) after 25 days of hospitalization.

He was transferred to the non-intensive burns unit after 43 days and discharged from the hospital after 2 months of convalescences.

Discussion

The cause of electrocution may be due to direct contact with a vehicle or any other equipment with power lines. Electrical burns are classified as either high voltage ($\geq 1,000$ V) or low voltage ($< 1,000$ V).

In literature, three major mechanisms were correlated to electricity-induced injury.

First is direct damage to tissue and cell membrane resting potential. Transmembrane protein molecules contain polar amino acid residues that may alter their orientation in response to the passage of an electrical current. This electro-conformational denaturation of membrane proteins is usually irreversible (poration). A second mechanism is based on conversion of electricity into thermal energy, which generates heat according to Joule's law: heat (Joule) = I^2 (current) + R (resistance) [1, 2]. Finally, massive tissue destruction and coagulative necrosis may occur, normally due to high-voltage injuries (Joule heating).

With regard to pulmonary electrical injury, Masanes and Robin confirmed the presence of lung infarction and coagulation necrosis related to poration during a histological examination of some resected lungs after electrocution. Suppurative bronchiolitis and lesional edema were similar to inhalation injuries and both correlated to Joule heating [1, 2, 14].

Some data were published reviewing the level of evidence

for the use of ECMO in hypoxemic respiratory failure resulting from burn and smoke inhalation injury. In a recent systemic review, some authors analyzed in total 18 clinical cases where 16 survived and two died but the age range was broad, specifically from 5 weeks to 42 years, where no differences were made between A-V and V-V extracorporeal support but pulmonary damage due to electrocution has been never described.

The final results of their meta-analysis revealed: 1) a tendency of higher survival of burn patients suffering respiratory failure and placed on ECMO; 2) an ECMO run time of less than 200 h correlates with higher survival; 3) scald burns show a tendency of higher survival in contrast to flame burns; 4) no difference on mortality between patients who have PaO₂/FiO₂ ratios of more or less than 60 at the time point of ECMO initiation [15].

We present a case of severe electrocution successfully treated with V-V ECMO in place for more than 400 h without major complications. The patient was a victim of a work-related accident and the major damages were localized in patient's right arm, left leg and lung (throughout the apical right lung to left base) (Fig. 3A, 4).

In V-V ECMO, the oxygen blood was pumped to the right atrium and pulmonary circulation. The action provided a pre-pulmonary blood oxygenation and allowed us to adopt "lung protective ventilation", to rest patient's lung and to bypass his refractory respiratory failure for more than 400 h [9, 16-18].

About the use of an ROTEM and MULTIPLATE-based algorithm tailoring coagulation therapy, Schaden et al suggested a specific perioperative algorithm for bleeding burn patients but no data have been published previously about the use of ECMO for this kind of injured patients [11].

As described in most recent literature, ROTEM and MULTIPLATE promptly evaluate the entire clotting process and, compared to conventional coagulation tests (as PT, aPTT, ACT, etc.), which have static endpoints and are usually measured in plasma and not in whole blood, they give us more information about the quality of clot and dynamics of its formation [3, 4, 11, 19, 20].

We applied the same guidelines, suggested by recent Cochrane analysis and the updated European guideline in trauma, for our burn patient not only during ECMO treatment but for the entire recovery and our major aim was minimizing blood and plasma transfusions [3, 4, 19].

Based on our experience, MCF in FIBTEM was usually high (MCF between 35 and 40 mm) and CFT was lower in INTEM and EXTEM (CFT between 42 - 50 s and 45 - 49 s respectively), both correlated to a high plasmatic fibrinogen level (> 5 g/L) (Fig. 5).

We confirmed that fibrinogen deficiency is not a leading mechanism for bleeding in burn patients also during ECMO, at least if severe dilutional coagulopathy is avoided by an individualized and restrictive fluid management approach as we did using PICCO monitoring (Fig. 1) [20].

As expected, our patient never needed administration of fibrinogen concentrate and he recorded high base-line levels of fibrinogen before and after burn wound excision. For this reason, we normalize coagulation using fresh frozen plasma preferably.

With respect to platelet aggregometry, MULTIPLATE was

really useful to exclude any congenital or acquired platelet dysfunction. During ECMO period, TRAP was approximately in range and when the patient was conducted with his oxygenator in OR, we noticed a strong correlation between TRAP test and surgical bleeding, due to platelets consumption (Fig. 5).

The efficacy of the use of MULTIPLATE in burn patients or during V-V ECMO treatment is still unproven but based on our experience, it could be useful to identify any platelet abnormality and to complete the study of clotting process at bedside.

We aimed our therapy on these new point-of-care tests to minimize transfusion requirements, transfusion-associated adverse events and to avoid thromboembolic complications.

ROTEM and MULTIPLATE provide specific, reliable and timely information during bleeding and daily practice, but it is important to validate this as point-of-care tests in large multi-center, well-controlled, prospective designed trials.

Conclusion

Presented here is a rare clinical case describing the successful use of ECMO as a rescue therapy for severe acute respiratory failure following electrocution burn injury.

The high risk of bleeding and/or thrombosis due to surgery, heparin infusion and daily medications was presumably minimized by the use of ROTEM and MULTIPLATE.

Their use as new point-of-care tests was inspired by international protocols and guidelines already published for bleeding trauma.

Further studies are needed to confirm our observations in burn victims and especially to elaborate targeted therapy algorithms for these severe and complicated patients to allow improvement in outcomes, reduce costs and target heparin therapy.

Acknowledgement

We thank the entire ICU team of the Hospital of Padua for their assistance with this case.

Abbreviations

ECMO: extracorporeal membrane oxygenator; TBSA: total body surface area; ICU: intensive care unit; OR: operative room; CI: cardiac index; GEDI: global end diastolic index; ELWI: extralung water index; TPTD: transpulmonary thermodilutions; PBD: post-burn day; BAL: bronchoalveolar lavage; XR: X-ray; CT: computed tomography; aPTT: activated partial thromboplastin time; MCF: maximum clot firmness; CT: clotting time; CVVH: continuous veno-venous hemofiltration; ACT: activated clotting time; TV: tidal volume; PEEP: positive end-expiratory pressure; POC: point-of-care; DVT: deep vein thrombosis; SAPS II: simplified acute physiology score II; SOFA: sequential organ failure assessment; TRAP test: thrombin receptor activating peptide-6 test; ROTEM:

rotational thromboelastometry; MULTIPLATE: multiple electrode platelet aggregometry

References

1. Ungureanu M. Electrocutions--treatment strategy (case presentation). *J Med Life*. 2014;7(4):623-626.
2. Michiue T, Ishikawa T, Zhao D, Kamikodai Y, Zhu BL, Maeda H. Pathological and biochemical analysis of the pathophysiology of fatal electrocution in five autopsy cases. *Leg Med (Tokyo)*. 2009;11(Suppl 1):S549-552.
3. Afshari A, Wikkelso A, Brok J, Moller AM, Wetterslev J. Thrombelastography (TEG) or thromboelastometry (ROTEM) to monitor haemotherapy versus usual care in patients with massive transfusion. *Cochrane Database Syst Rev*. 2011;(3):CD007871.
4. Rossaint R, Bouillon B, Cerny V, Coats TJ, Duranteau J, Fernandez-Mondejar E, Hunt BJ, et al. Management of bleeding following major trauma: an updated European guideline. *Crit Care*. 2010;14(2):R52.
5. Kahn SA, Beers RJ, Lentz CW. Resuscitation after severe burn injury using high-dose ascorbic acid: a retrospective review. *J Burn Care Res*. 2011;32(1):110-117.
6. Alvarado R, Chung KK, Cancio LC, Wolf SE. Burn resuscitation. *Burns*. 2009;35(1):4-14.
7. Sanchez M, Garcia-de-Lorenzo A, Herrero E, Lopez T, Galvan B, Asensio M, Cachafeiro L, et al. A protocol for resuscitation of severe burn patients guided by transpulmonary thermodilution and lactate levels: a 3-year prospective cohort study. *Crit Care*. 2013;17(4):R176.
8. Berger MM, Que YA. A protocol guided by transpulmonary thermodilution and lactate levels for resuscitation of patients with severe burns. *Crit Care*. 2013;17(5):195.
9. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. The Acute Respiratory Distress Syndrome Network. *N Engl J Med*. 2000;342(18):1301-1308.
10. Peek GJ, Clemens F, Elbourne D, Firmin R, Hardy P, Hibbert C, Killer H, et al. CESAR: conventional ventilatory support vs extracorporeal membrane oxygenation for severe adult respiratory failure. *BMC Health Serv Res*. 2006;6:163.
11. Schaden E, Kimberger O, Kraincuk P, Baron DM, Metnitz PG, Kozek-Langenecker S. Perioperative treatment algorithm for bleeding burn patients reduces allogeneic blood product requirements. *Br J Anaesth*. 2012;109(3):376-381.
12. Weingart C, Lubnow M, Philipp A, Bein T, Camboni D, Muller T. Comparison of Coagulation Parameters, Anticoagulation, and Need for Transfusion in Patients on Interventional Lung Assist or Venovenous Extracorporeal Membrane Oxygenation. *Artif Organs*. 2015;39(9):765-773.
13. Nair P, Hoechter DJ, Buscher H, Venkatesh K, Whittam S, Joseph J, Jansz P. Prospective observational study of hemostatic alterations during adult extracorporeal membrane oxygenation (ECMO) using point-of-care thromboelastometry and platelet aggregometry. *J Cardiothorac Vasc Anesth*. 2015;29(2):288-296.
14. Masanes MJ, Gourbiere E, Prudent J, Lioret N, Febvre M, Prevot S, Lebeau B. A high voltage electrical burn of lung parenchyma. *Burns*. 2000;26(7):659-663.
15. Marques EG, Junior GA, Neto BF, Freitas RA, Yaegashi LB, Almeida CE, Junior JA. Visceral injury in electrical shock trauma: proposed guideline for the management of abdominal electrocution and literature review. *Int J Burns Trauma*. 2014;4(1):1-6.
16. Tseng YH, Wu TI, Liu YC, Lin PJ, Wu MY. Venoarterial extracorporeal life support in post-traumatic shock and cardiac arrest: lessons learned. *Scand J Trauma Resusc Emerg Med*. 2014;22:12.
17. Asmussen S, Maybauer DM, Fraser JF, Jennings K, George S, Keiralla A, Maybauer MO. Extracorporeal membrane oxygenation in burn and smoke inhalation injury. *Burns*. 2013;39(3):429-435.
18. Brodie D, Bacchetta M. Extracorporeal membrane oxygenation for ARDS in adults. *N Engl J Med*. 2011;365(20):1905-1914.
19. Da Luz LT, Nascimento B, Shankarakutty AK, Rizoli S, Adhikari NK. Effect of thromboelastography (TEG(R)) and rotational thromboelastometry (ROTEM(R)) on diagnosis of coagulopathy, transfusion guidance and mortality in trauma: descriptive systematic review. *Crit Care*. 2014;18(5):518.
20. Fodor L, Fodor A, Ramon Y, Shoshani O, Rissin Y, Ullmann Y. Controversies in fluid resuscitation for burn management: literature review and our experience. *Injury*. 2006;37(5):374-379.