Editorial

Neurological complications during veno-venous extracorporeal membrane oxygenation

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Lorusso *et al.* studied 4,988 adult patients who underwent veno-venous extracorporeal membrane oxygenation (VV ECMO) using Extracorporeal Life Support Organization (ELSO) database to evaluate the incidence of neurological complication (1). They found that the neurological complications rate on VV ECMO was 7.1%. The majorities of these neurological complications were intracranial bleed (42.5%), brain death (23.5%), and ischemic stroke (19.9%). The incidence of neurological complications while on VV ECMO decreased over time from 9.5–14.3% in the 1990s to 6.7–7.9% from the year 2000 forwards. The pre-ECMO risk factors for neurological complications during VV ECMO were reported to be cardiac arrest, continuous renal replacement therapy (CRRT) and hyperbilirubinemia. Once neurological complications occurred during VV ECMO, less than 25% of patients survived to discharge (mortality rate 75.8% in patients with neurological injury), while those who without neurological complication had a discharge rate from hospital of 63%.

The studied population was primarily patients with severe acute respiratory failure since VV ECMO provides oxygenation and CO_2 clearance. Because the mechanism of the VV ECMO is depending on the intact native circulatory functioning, the patients on VV ECMO are less likely to have primary cardiac problems or general perfusion issues at the time of VV ECMO cannulation (2). In VV ECMO, the cerebral perfusion is always pulsatile with ante-grade blood flow, while cerebral perfusion in VA ECMO is more likely non-pulsatile and retrograde flow although depending on location of the arterial cannula and cardiac function. VV ECMO patients are less likely to develop emboli from the oxygenator or circuit since the inflow cannula is always located in the right side of the heart, unless the patient has patent foramen ovale.

The neurological injury in VV ECMO may occur by two different mechanisms. One is the preexisting neurological injury before ECMO, including anoxic brain injury or hypoperfusion related brain injury. The physician may have placed VV ECMO with minimum knowledge of the patient neurological status due to the presence of neuromuscular blocking agents or deep sedation to control the ventilation and oxygenation before ECMO. The patients that experienced cardiac arrest due to hypoxia carried the highest risks of the anoxic brain injury as described in Lorusso report. Those who had brain death on the Lorusso study (23.5% of the patients) were more likely evolving at the time of cannulation but it was not recognized until appropriate work-up after placement of ECMO, since the patient was not have stable to transport for imaging modalities before ECMO. The second mechanism of the neurological complication during VV ECMO could be related to anticoagulation. In early era of the VV ECMO, anticoagulation was controlled by ACT 180–240 sec, which often over-estimated the level of the anticoagulation (3). Modern ECMO system using heparin-bounded tubing, a low-pressure oxygenator and centrifugal pump allows the maintenance anticoagulation to be lower without thrombotic complications. Thus, anticoagulation controlled to be PTT

45–55 sec may be enough to maintain VV ECMO (4). This current ECMO devices system and need for lower anticoagulation therapy may have contributed to the lower incidence of neurological complication after year of 2000.

Patient selection is important for improvement of the ECMO survival. If the hypoxic brain injury is suspected at the time of the ECMO cannulation due to pre-ECMO cardiac arrest or per-ECMO prolonged hypoxia, induced hypothermia may protect brain (5). Quick and effective hypothermia can be achieved with conjunctions of heater-cooler from ECMO circuit and Arctic-Sun (Bard, Louisville, CO, USA) or other external devices (6). Head CT should be done if the patient remained comatose after completion of hypothermia protocol and appropriate sedation vacation period. If CT suggests diffuse anoxic brain injury and the patient remained coma, cerebral perfusion study may be necessary to determine brain death.

Early detection of stroke and intracranial bleed is important. Cerebral oximetry using near-infrared spectroscopy may play a role for early detection (7). Unilateral drop of the cerebral oximetry could be highly suspicious intracranial event while bilateral drop could be related with low oxygen delivery such as low perfusion pressure, low hemoglobin, or low oxygen saturation.

Use of CRRT and hyperbilirubinemia were major risk factors of neurological injury according to Lorusso study. Pre-operative renal and liver failure reflects the general sickness of the patient prior to ECMO. Severe irreversible multi-organ failure could be relative contraindication for ECMO. The major indication for VV ECMO is acute isolated respiratory failure without other major organ injury. If renal and liver dysfunction is observed at the time of the cannulation, VV ECMO support may not be adequate and VA ECMO may be necessary to perfuse kidney, liver and other major organs adequately. Once the hepatorenal syndrome occurs, it may lead to disseminated intravascular coagulation (DIC) and it may further contribute the risk of intracranial bleed. Early institution of CRRT for renal failure and MARS dialysis for liver failure is to avoid persistent multi-organ failure (8).

Neurological injury occurring VV ECMO could be a devastating complication and major cause of death during ECMO. Appropriate pre-ECMO patient selection, brain protection using hypothermia for high-risk patients, and routine cerebral oximetry may improve the outcomes.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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