Residência RP Pediátrica
Publicação Oficial da Sociedade Brasileira de Pediatria
ISSN-Online: 2236-6814

Submitted on: 02/07/2019
Approved on: 03/11/2019

CASE REPORT

Oxygenation by extracorphorus membrane (ECMO) installed during cardiorrespiratory stop in lung with acute respiratory anguish syndrome: a case report

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Keywords:
Extracorporeal Membrane Oxygenation, Adult Respiratory Discomfort Syndrome, Heart Arrest

Abstract

Extracorporeal membrane oxygenation (ECMO) provides a better oxygen supply to patients with severe respiratory and cardiac diseases refractory to conventional therapy. ECMO installed during cardiorespiratory arrest (CPR) is called ECPR, which has been improved, presenting favorable results. The present article is an observational study, whose material was the medical record of a patient with a diagnosis of acute respiratory distress syndrome (ARDS) where ECMO was performed during a successful cardiorespiratory arrest. Clinical, laboratory and radiographic data were evaluated, which reflect and corroborate the pathophysiology of the disease and the structural alteration of the respiratory system. This paper aims to describe a clinical case of successful ECPR in a 2-year-old female patient with hospital admission for extensive pneumonia who progressed with ARDS and cardiac arrest refractory to conventional treatment. The use of ECMO deserves to be considered in patients with cardiac arrest, since it demonstrated significant benefits, allowing effective treatment and survival without sequelae.

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Residência Pediátrica; 2020: Ahead of Print.
DOI: 10.25060/residpediatr-2020.v10n3-95

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INTRODUCTION

ECMO (extracorporeal membrane oxygenation) provides better oxygen supply (O2) to the tissues of patients with severe respiratory or cardiac diseases. It is an accepted modality for the support of children with potentially reversible respiratory or cardiac failure in which conventional therapy has failed. As for the type, ECMO is classified as venous (VV) or venoarterial (VA). The VA support offers hemodynamic support, in addition to gas exchange. The central venous circulation serves as a preload for the circuit pump, while the blood returns through a cannula located in an artery.

There is an arteriovenous modality that is performed without the need for a pump. The blood is diverted to a membrane, where gas exchange occurs, and is returned to the patient's circulation. This modality depends on the ability of the heart to support this blood diversion; it is not possible to provide full support, since the blood cannot be totally diverted from the circulation. It is not very much used today.

ECMO VV can be used in acute respiratory failure (ARF), in which blood is drained from the patient's venous circulation to the extracorporeal support circuit, pumped through an oxygenation membrane and returned to the patient's venous system. This support increases available O2 for coronary arteries and can indirectly contribute to the improvement of cardiac function, in addition, the improvement in oxygenation decreases pulmonary vascular resistance (PVR), improving the afterload to the right ventricle (RV). Therefore, one checks VV support assessment with an increase in O_2 content, which can be better evaluated by SaO_2, which must be maintained between 80-90%, or by PaO_2.

VA support offers hemodynamic support, in addition to gas exchange. The central venous circulation serves as a preload for the circuit pump, while the blood returns via a cannula located in an artery. Unlike cardiopulmonary bypass (CPB) performed in the operating room, the VA support circuit does not capture all venous return from the right heart. Only part of the venous return is drained into the circuit according to the pump speed. The venous blood that remains in the right heart follows the common route through the lungs to the left heart, and then to the aorta.

ECLS is a therapeutic option for cases of respiratory or heart failure in which conventional therapies have been exhausted. ECMO VV or VA can be performed, and the choice of method must take into account the advantages and disadvantages of each modality. The understanding of the concepts of oxygenation, recirculation and presenting an adequate monitoring are paramount for the decision of the chosen modality.

Currently, the rapid growth of ECMO use has been used to provide support to patients in cardiac arrest (CA) after failure of conventional support to provide a spontaneous return of circulation. CAEC provides a higher level of cardiac support when compared to conventional CPR.

CASE PRESENTATION

C.O.S, female, 2 years old, presented with unchecked fever and productive cough on 07/19/2017, being taken to the emergency room. During treatment, she was diagnosed with pneumonia, and was prescribed Amoxicillin (50mg/kg/day) and acetylcysteine. Her mother reported that she used the medications correctly until 07/22/2017, when she returned to the emergency room because there was no improvement in her fever and cough. On 07/24/2017, the patient presented worsening of her general condition, and her chest X-ray showed bilateral pneumonia. They decided for hospitalization and initiation of antibiotic therapy with Ceftriaxone (dose 100mg/kg/day). Without response to clinical treatment, her chest USG showed a 5mm pleural effusion to the right. She remained hospitalized until 07/28/17, when the mother decided to remove her from the service because there was no resolution of her condition. On the night of 07/29/17, the patient was referred to our service in a regular general condition, prostrated, irritated, flushed and hydrated, with 80% oxygen saturation. On pulmonary auscultation, she had vesicular murmur present and decreased on the right with diffuse subcrepitant rales and sparse wheezing. She received non-reinhalant O2 at 5L/min, nebulization with SF0.9% with Fenoterol and Ipratropium, 10mg methylprednisolone and was referred for hospitalization. Her urine 1 test showed significant proteinuria (4+/4+) and massive leukocyturia, and her blood count showed increased inflammatory activity (CRP: 12.2), serum cholesterol within the normal range, as well as serum complements and basal electrolytes.

Her admission diagnosis was extensive pneumonia associated with severe bronchospasm and laryngospasm.

Upon admission, she was started on antibiotic therapy with Ceftriaxone (dose 100mg/kg/day) and Oxacillin (dose 200mg/kg/day). Upon examination, she was in REG, hydrated, flushed, crying and touchy, with edema on her face and lower limbs +/4+. Upon pulmonary auscultation: lung sounds present, decreased on the right with crackle sounds, and wheezing. Abdominal exam: distended abdomen, tense and painful on palpation. Furosemide 1mg/kg was introduced. We ordered a chest x-ray (PA + SIDE + LAURELL), (Figures 1 - 2) below.

In the early hours of 08/02/17, the patient progressed to severe irritability, tiredness, desaturation (75-85%), tachy/dyspnea and agitation. In the pulmonary exam, her lung sounds decreased globally with a crackle sound throughout the lung projection associated with wheezing and snoring. Tachycardia with acrocyanosis was present. She progressed to 60% saturation, requiring orotracheal intubation with a 5 cuffed cannula and referred to the pediatric ICU, where she received Meropenem 40mg/kg/day and Vancomycin 40mg/kg/day 6h; Dobutamine 15mcg/kg/min; Midazolam 0.5mg/kg/hour; Fentanyl 2mcg/kg/h; Dopamine 10mcg/kg/min, OTI maintaining Sat 70-80% with PEEP 15, I: E 1: 1.8. She was then

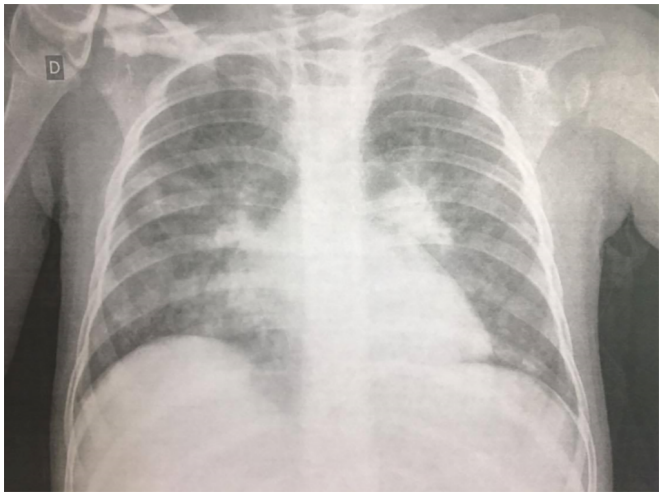


Figure 1. Chest X-ray in PA: areas of heterogeneous opacities are evident, with blurred cardiac silhouette, elevation of the diaphragmatic dome on the right without signs of pleural effusion, thickening of the bilateral pulmonary hilum.

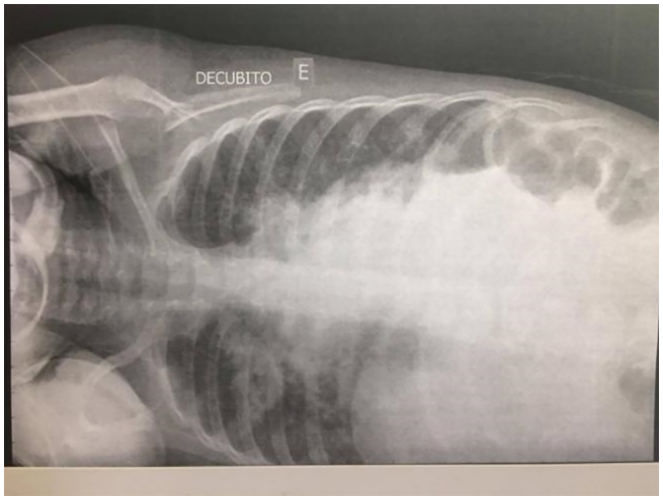


Figure 2. Chest radiography in the right lateral decubitus position: presence of atelectasis in the upper third of the right lung, with heterogeneous opacities, bilateral pulmonary hilus thickening, blurring of the cardiac silhouette, with no pleural effusion.



Figure 3. Rectification of ribs and diaphragm, air bronchograms, areas of atelectasis interspersed with areas of diffuse homogeneous opacification, blurring of the cardiac silhouette, with loss of delimitation of pulmonary vascular network, diffuse hypotransparency.

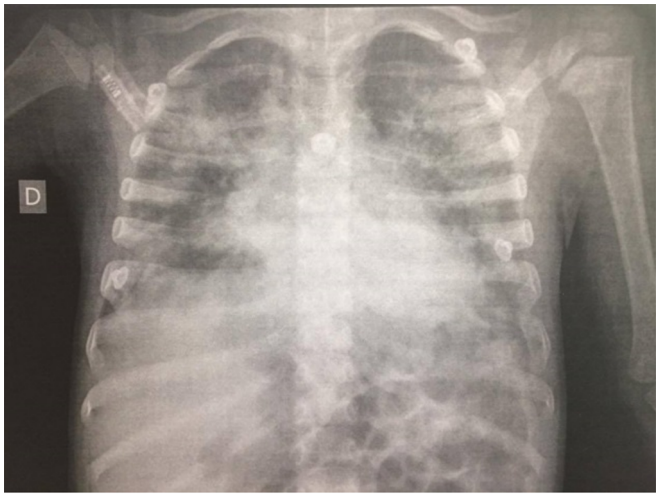


Figure 4. Rectification of ribs, loss of diaphragm delimitation due to diffuse bilateral opacity, bilateral air bronchograms, areas of atelectasis, blurring of the cardiac silhouette, with loss of delimitation of the pulmonary vascular network.

I critical condition, with high MPV parameters, maintaining 70-80% saturation, using inotropes, but with ventilatory and hemodynamic instability, with edema 2+/4+. Chest radiography showing diffuse pulmonary hypo-transparency (Figures 3 - 4).

On 08/03/2017, the patient progressed with decreased peripheral perfusion, thin pulses, saturating 53%, tachycardia and swelling. We decided to increase the MPV parameters (Pinsp 43, PEEP 12), introduce Terbutaline, Adrenaline and Nitrous Oxide by 35 ppm, in addition to 20% 5ml/kg albumin infusion. The patient deteriorated on her clinical picture, and we discussed with the Hospital Infection Committee concerning the replacement of her antimicrobial therapy, and then, opted for the introduction of Clarithromycin to cover for atypical germs and Oseltamivir to cover for influenza, in addition to collecting swab samples for culture. Due to a refractory hypoxemia (less than 70%) in association with respiratory acidosis seen on her arterial blood gases (pH 7.1, pco_2 115, po_2 36, $HCO_3$0, BE 0 Lactate 6), we then added 8.5% 10ml HCO_3.

We discussed her case PUC-Campinas Hospital, and decided to put her on ECMO due to severe refractory hypoxemia (08/05/17). Upon admission to the other service, she had bradycardia, central cyanosis, hypothermia, with thin pulses and slowed capillary perfusion, low saturation and little diuresis by the bladder catheter present. The cardiac surgery team installed the veno-arterial ECMO (from 08/05/17 to 08/11/17), switching to veno-arterial-venous (from 08/11 to 08/14) and subsequently

to veno-venous (from 08/14 to 08/25), when it was successfully interrupted. During the hospitalization period, she required continuous sedation, peritoneal dialysis and transfusions. He received Clarithromycin, Vancomycin, Meropenem, Fluconazole and oseltamivir. Due to oxygen demand difficulties, the patient required a tracheostomy (9/14), progressing with improved breathing pattern and sedation weaning.

The patient returned to our service on 9/19/2017, brought by the EMT, to complete her treatment for ARDS and Respiratory Failure. Upon admission, she was pale +/4+, hydrated, afebrile, anicteric, acyanotic, active and contacting. Pulmonary exam: diffuse snores, and with an ulcer in her right hallux.

Diagnostic hypothesis upon admission: Acute respiratory failure, ARDS, Septic Shock, ECMO, Lung Injury (atelectasis and fibrosis), Hydrocephalus, Corneal Ulcer, Decubitus Ulcer and Treatment Withdrawal Syndrome.

Her anal swab was positive for Enterobacter aerogenes and Klebsiella pneumoniae, positive nasal swab for Klebsiella pneumoniae, negative axillary swab, and positive blood culture for Staphylococcus aureus in two samples (MSD and MSE).

The patient progressed well and was discharged from the ICU on 10/04/17. The otorhinolaryngology team assessed the patient on 10/6, and was started on an oral diet, which she accepted well, without choking.

With good progress, she was discharged on 10/20/17.

DISCUSSION

It is difficult to measure the incidence rate of ARDS due to problems associated with its definition and failure in therapeutic tests.[1] However, if we take into account ALI/ARDS, the incidence is frequent, estimated at 79 cases per 100 thousand inhabitants per year, most common during the winter (data related to the incidence in the USA).[2]

The mortality rate is high, ranging from 35% to 60%. Surviving patients have a prolonged stay in the intensive care unit (ICU) and have important limitations to their usual functionality, affecting her muscle activity.[3,4]

ARDS is the most serious spectrum of acute lung injury (ALI), pathologically characterized by diffuse alveolar damage with the development of non-cardiogenic pulmonary edema due to increased permeability of the pulmonary alveolar-capillary membrane.[5] For such a mechanism to occur, there must be damage to the alveolar epithelium and capillary endothelium, by different pro-inflammatory mediators, released in response to a wide variety of precipitants (direct or indirect lung injury).[6] The lungs are vulnerable to inflammatory injuries. In this way, the barriers usually responsible for preventing alveolar edema are lost, with the escape of proteins from the intra-vascular space towards the interstitial space, causing interstitial and alveolar edema. The influx of protein-rich liquid into the alveoli alters pulmonary surfactant integrity, with additional damage to the lung tissue and alveolar collapse. Its clinical expression is hypoxemic respiratory failure and bilateral pulmonary infiltrate on chest radiography in patients with pulmonary and/or extrapulmonary risk.[7]

The factors associated with the syndrome development can be divided between direct lung injury and indirect lung injury.[8] Among the pulmonary causes; we list, in decreasing order: Pneumonia, Aspiration of gastric contents, near-drowning, pulmonary contusion, Fat embolism and pulmonary reperfusion edema. Of the extra-pulmonary causes, the following prevail: sepsis, severe non-thoracic trauma with shock, hyper transfusion for emergency resuscitation, cardiopulmonary bypass, drug overdose, acute pancreatitis and disseminated intravascular coagulation.[9,10,11,12,13] The presence of multiple risk factors substantially increases the risk of developing ARDS.

In the case presented, the patient underwent ECMO (oxygenation by extracorporeal membrane), which allows better oxygen supply (O_2) to the tissues of patients with severe respiratory or cardiac diseases. It is an accepted modality for the support of children with potentially reversible respiratory or cardiac failure, in whom conventional therapy has failed. The patient was submitted to CREC (cardiopulmonary resuscitation with extracorporeal membrane), using the ECMO installation technique during cardiorespiratory arrest. CAEC success depends on early onset in a patient with potential for myocardial recovery, as it provides a higher level of cardiac support when compared to conventional cardiopulmonary resuscitation. Its advantage is the high success rate in the return of spontaneous cardiac circulation, support after resuscitation from cardiogenic shock during coronary performance intervention and maintaining organ perfusion during native cardiac recovery.

CAEC is defined as the application of VA (veno-arterial) ECMO in patients in cardiac arrest who do not achieve spontaneous cardiac return, or fail to have spontaneous return of circulation during resuscitation. Observational studies suggest that survival with good neurological recovery may be better with CAEC than with conventional CPR, but we still lack prospective controlled studies. CAEC consideration is given to individuals with cardiac arrest in an in-hospital or out-of-hospital setting who received good quality effective chest compressions within 5 minutes of arrest and in whom the return to normal circulation does not return within 15 to 30 minutes.

Both children and adults are candidates for CAEC. However, further studies are needed to better define its role and application; however, now, CAEC should be considered in patients with the correct indication and in hospitals with existing ECLS programs.

CONCLUSION

The indications for the use of ECMO have advanced considerably in recent years and deserve to be considered in patients with cardiac arrest, because it has shown significant benefits, enabling an effective treatment, with improvement in neurological function and survival without sequelae.

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