86673_Auto_Edited.docx

WORD COUNT 2289
TIME SUBMITTED 05-JAN-2024 03:50PM
PAPER ID 105939715

2
Name of Journal: *World Journal of Clinical Cases*

Manuscript NO: 86673

Manuscript Type: CASE REPORT

Left ventricular thrombosis caused cerebral embolism during Veno-arterial extracorporeal membrane oxygenation support: A case report

Left ventricular thrombosis caused cerebral embolism

Yaobang BAI, Feng Zhao, Zhenhua WU, Guoning SHI, Nan JIANG

Abstract

BACKGROUND

Venoarterial extracorporeal membrane oxygenation (VA-ECMO), an effective short-term circulatory support method for refractory cardiogenic shock, is widely applied.However, retrospective analyses showed that the VA-ECMO-assisted cases were associated with a relatively high mortality rate at about 60%. The embolization in important organs caused by complications of left ventricular thrombosis (LVT) during VA-ECMO is also an important reason. Although the incidence of LVT during VA-ECMO is not high, the consequences of embolization will be disastrous.

CASE SUMMARY

A 37-year-old female patient was admitted with 4 days of fever and 3 days of palpitations. After excluding the diagnosis of coronary heart disease, we established a diagnosis of “clinically explosive myocarditis”. The patient still had unstable hemodynamics after drug treatment was supported by VA-ECMO, with ordinary heparin for anticoagulation. On day 4 of ECMO support, a left ventricular thrombus attached to the papillary muscle root of mitral valve was found by transthoracic echocardiography. After that, left ventricular decompression was performed and the

patient was successfully removed from ECMO, but eventually died from multiple cerebral embolism.

CONCLUSION

LVT with high mobility during VA-ECMO may cause important organs Embolism. Therefore, we shouldn't take a "wait-and-see" attitude.

Key Words: Venoarterial extracorporeal membrane oxygenation; Left ventricular thrombosis; Cerebral embolism; Magnetic Resonance Imaging; Therapy; Case report

BAI Y, Zhao F, WU Z, SHI G, JIANG N. Left Ventricular Thrombosis caused Cerebral Embolism During Veno-arterial Extracorporeal Membrane Oxygenation Support : A case report. *World J Clin Cases* 2023; In press

Core Tip: The embolism of vital organs (brain, mesenteric artery, *etc.*) caused by detachment of the LVT can lead to catastrophic consequences. We report a case of explosive myocarditis in which a LVT attached to the papillary muscle root of mitral valve, which resulted in massive cerebral embolism. Although we can adopt a "wait and see" attitude considering the autolytic rate of LVT and the fatal complications associated with thrombolysis and surgical thrombectomy. We should adopt more aggressive treatment methods for left ventricular thrombi with high mobility, such as transcatheter left ventricular thrombolysis or surgical thrombectomy.

INTRODUCTION

VA-ECMO has been widely performed in short-term circulation support for refractory cardiogenic shock, due to its economical price and mature catheterization and management compared with other mechanical circulation assist devices [1]. In the past decades, the number of VA-ECMO applications both in China and aboard has shown explosive growth [2,3]. However, the clinical results of VA-ECMO application are

disappointing, with the overall mortality rate supported by VA-ECMO in refractory cardiogenic shock reported to be 60% [4]. Although the underlying diseases leading to cardiogenic shock are serious and are the main cause of failure, some complications during the application of VA-ECMO (e.g., fetal hemorrhage, thromboembolism of vital organs, severe hemolysis, infection, *etc.*) can also lead to the failure. And embolism of vital organs has become one of the most frightening complications during VA-ECMO support. The thrombosis may occur in the circuit, oxygenator, pump and ventricle, with the incidence reported ranging from 3% to 12%. Despite the low incidence of LVT, the embolism of brain caused by detachment of the thrombosis can lead to catastrophic consequences [9]. It is reported that mortality in cardioembolic stroke is the highest as compared with other ischemic stroke subtypes [10,11].

We report a case of explosive myocarditis in which a left ventricular thrombus attached to the papillary muscle root of mitral valve, which resulted in massive cerebral embolism during the VA-ECMO support.

CASE PRESENTATION

Chief complaints

The 37-year-old female patient was admitted to the hospital mainly for "fever for 4 days and palpitation for 3 days.

History of present illness

The patient developed a fever 4 days before admission and continued to have intermittent fever after symptomatic treatment. 3 days before admission, the patient presented palpitations accompanied by chest tightness, fatigue and were admitted to the emergency department of our hospital.

History of past illness

The patient had a history of hyperthyroidism and was treated with iodine-131, and currently was treated with oral levothyroxine tablets for hypothyroidism.

Personal and family history

The patient denied any family history of cardiac disease.

Physical examination

On physical examination, the vital signs were as follows: Body temperature, 37.1℃; Blood pressure, 90/71 mmHg; Heart rate, 95 beats per min; Respiratory rate, 14 breaths per min. Cardiac Auscultation (Arrhythmia, decreased heart sound, no heart murmur heard in auscultation areas)

Laboratory examinations

Myocardium Zymogram Items(Creatine kinase 466U/L, creatine kinase isoenzyme 41U/L, TroponinT 2.66ug/L); N-terminal pro b-type natriuretic peptide 6599pg/mL; Thyroid Function Tests (Free Triiodothyronine 2.13 pmol/L, Free Tetraiodothyronine 15.91 pmol/L, Thyroid Stimulating Hormone 3.88 uIU/mL).

No abnormality was found in routine blood and urine analyses.

Imaging examinations

Cardiac ultrasound in emergency room: LA 32mm, LV 47m, RA 38mm, RV 16mm, PAP 30mmHg, LVEF 62%, and the contraction and diastolic function of left heart were normal.

Re-examination of bedside ultrasound: LA 33 mm, LV 46 mm, LVEF 35-39%, left ventricular wall thickening, extensive myocardial motility reduction, and reduced left heart function.

FINAL DIAGNOSIS

Combined with the patient's medical history and Laboratory examinations, the final diagnosis was explosive myocarditis, arrhythmias.

TREATMENT

After admission, he was given myocardial nutrition, volume supplementation, dopamine cardiac strengthening, and norepinephrine vasopressor therapy. The patient's condition progressed rapidly, and after 13 h of admission, he developed a third-degree AV block with ventricular rate of about 60 beats/minute, blood pressure of 75/52 mmHg, and distal dampness. Rehydration fluids and high-dose vasoactive drugs continued to maintain circulation (vasoactive drug score: 30), and blood gas analysis showed metabolic acidosis combined with respiratory alkalosis. Blood lactic acid is 2.5 mmol/L. With the assistance of an emergency endotracheal intubation ventilator, percutaneous ECMO implantation was performed at the bedside, using VA- mode, flow rate 3.5 L/min. Heparin anticoagulation was applied during ECMO operation to maintain ACT 180-200s; chest X-ray and echocardiography were monitored daily. After the implantation of ECMO, the patient had a heart rhythm of third-degree atrioventricular block, with a ventricular of about 50 times, occasional ventricular tachycardia and ventricular fibrillation. The cardiologist was contacted for temporary pacemaker support. Echocardiography results after 4 days of ECMO support: a moderate intensity echoic mass of 2.4cm*1.5cm was observed in the left ventricular thrombus attached to papillary muscle root of mitral valve at the flow rate of 0.5m/s (Figure 1); chest X-ray showed increased pulmonary edema. At this time, the patient was considered to have developed a hemodynamic change specific to peripheral VA-ECMO support: left ventricular dilation. Accordingly, the therapeutic strategies were given: reducing the auxiliary flow to 3 L/min, maintaining the negative balance of the inflow and outflow, adding epinephrine to strengthen the heart, increasing PEEP to improve right ventricular drainage, and giving IABP support to promote aortic valve opening. The patient's cardiac function gradually improved, and the pulmonary edema gradually subsides. On the 7th day of ECMO support, bedside ultrasound showed: LA 32 mm, LV 55 mm, LVEF 30%, the thrombus sound shadow in the heart was not obvious (Figure 2), and there was no abnormality in the patient's neurological examination at this time.

OUTCOME AND FOLLOW-UP

On the 9th day of ECMO support, the autonomic rhythm was recovered, cardiac function continued to improve, pulmonary edema was further reduced, and no abnormalities in the neurological examination, so ECMO support was removed. Since then, the patient has recovered smoothly. 12 days after admission, the patient suddenly lost consciousness, and CT examination showed multiple cerebral embolism (Figure 3). The patient's family members gave up treatment and the patient was discharged from hospital.

DISCUSSION

There is limited data on LVT in patients requiring VA- ECMO. One case showed a series of patients (n = 11) developed LVT due to ischemic cardiomyopathy with cardiogenic shock, accounted for 3.1% of the center's total VA- ECMO experience [12]. LVT is a terrible complication for VA- ECMO. Embolization of vital organs such as the brain, kidneys, and mesentery caused by thrombectomy will have fatal consequences, leading to the failure of ECMO support [13,14]. The pathophysiology of LVT formation during VA- ECMO support is complex and is a result of multiple factors. Severely impaired cardiac function, left ventricular dilation induced by VA- ECMO, and left ventricular blood stasis are the dominant factors for thrombosis. The hypercoagulable state of patients and the inadequacy of current anticoagulation therapy also play an important role in thrombosis.

Although transthoracic echocardiogram (TTE) has been widely used in the diagnosis of LVT, it is greatly affected by patient's acoustic window (small intercostal space, large body size, chest deformities, or lung disease) and position [15]. In this case, the initial ultrasound images suggested that the thrombus was attached to the mitral valve, but after repeated multi-sectional examinations, the thrombus was eventually found to be attached to the root of the papillary muscle. Possible reason is that

transthoracic ultrasound is a two-dimensional image and the patient was in supine position, so the judgement of the overall morphology of the thrombus was poor.

Currently, there are no guidelines or expert consensus recommendations for ECMO support for LVT treatment in patients [16,17]. Some therapeutic options reported include improving anticoagulant strength, surgical thrombectomy and thrombolytic therapy. **Heparin:** according to most reports [18,19], anticoagulation by heparin can reduce the incidence of LVT, but has no effect on thrombolysis. **Surgery:** Surgical resection of the LVT is an option when undergoing other open-heart surgeries or transitioning from peripheral VA-ECMO intubation to central intubation [20]. However, the risk-benefit ratio should also be considered, as most patients with LVT have a severely reduced left ventricular ejection fraction, which has higher perioperative complications and mortality if they undergo thrombectomy. Therefore, in the absence of other indications emergency surgeries, surgical thrombectomy should be carefully considered, as the risks for patients far outweigh the benefits [21]. **Thrombolysis:** multiple studies have shown that fibrinolytic solvents can dissolve LVT, but the risk of this treatment is too high (thrombosis fall off can lead to embolism [22,23]). In one study, four patients with left ventricular thrombosis were given intravenous fibrinolytic drugs, and after 8 to 12 h, the size of the thrombus was significantly reduced, and the thrombus disappeared completely in 2 of the patients; but the mobility of the thrombus also increased significantly. And the last 2 patients in the study developed a severe systemic thromboembolic event. Simultaneous administration of thrombolytics increases the risk of bleeding [19].

In addition to thrombosis, bleeding at the puncture site or surgical site is also a common cause of death in VA-ECMO patients, and thrombolysis for VA-ECMO patients is a big challenge [24]. Fabio S [25] et al. reported a new approach for LVT, in which the patients' LVT was completely dissolved 24 h after catheterized injection of tenecteplase into the left ventricle, and only moderate bleeding occurred. However, a case report can provide us with new ideas for the treatment of LVT during VA- ECMO

support, but the therapeutic effect and related complications need to be studied in large-scale clinical trials.

It is reported that approximately 20-40% of LVTs resolve spontaneously without anticoagulation with the restoration of cardiac function [26,27]. Pratic [28] et al. showed that the morphological, size and mobility of LVT would change, and there was no obvious correlation between the morphological characteristics and the occurrence of thromboembolism. Anne-Iris L [21] et al. believed that a "wait-and-see" attitude seems to be a safe and reasonable management plan for LVT in patients with heart failure. Therefore, we took active conservative treatment measures: (1) to improve the strength of anticoagulation; (2) to promote the development of aortic valves and improve blood stasis by giving positive inotropic drugs and reducing support flow. At the same time, we took a "wait-and-see" attitude and insisted on daily TTE monitoring.

In this case, the patient still had thromboembolism even though ultrasound suggested thrombolysis. This may be due to poor sensitivity of conventional ultrasound to LVT, and there should be thrombus not found in routine examination. Therefore, such patients should be examined by MRI after ECMO removal [29] to exclude the existence of thrombosis, and regular anticoagulation should be given according to relevant guidelines if thrombosis is found in the examination [30].

The limitations of this study are as follows: First, no MRI examination was performed after the removal of ECMO support to confirm the complete disappearance of left ventricular thrombus. Second, no laboratory tests for hematologic diseases was conducted to rule out stroke caused by such diseases. Because, Arboix A and his colleagues reported that Hematological disorders are one of the easily overlooked cause of acute stroke[31].

Future research should focus on the overall prognosis and treatment of patients with left ventricular thrombus during ECMO support, and develop relevant treatment guidelines or expert consensus to improve the outcome of ECMO support.

CONCLUSION

The occurrence of LVT during VA- ECMO is the result of a multifactorial combination and has a higher mortality rate in patients after its occurrence. Management of LVT after its occurrence is a major challenge for clinicians. Although we can adopt a "wait and see" attitude considering the autolytic rate of LVT and the fatal complications associated with thrombolysis and surgical thrombectomy. We should adopt more aggressive treatment methods for left ventricular thrombi with high mobility, such as transcatheter left ventricular thrombolysis or surgical thrombectomy. In clinical work, we should pay attention to the monitoring and management during operation, and actively prevent and treat the left ventricular blood stasis. Continuously improve the device to improve biocompatibility and reduce the activation of coagulation and inflammatory reactions. Only when every element of ECMO operation is optimized can the prognosis of patients ultimately be improved.

86673_Auto_Edited.docx

ORIGINALITY REPORT

4%
SIMILARITY INDEX

PRIMARY SOURCES

1	www.wjgnet.com Internet	67 words — 3%
2	f6publishing.blob.core.windows.net Internet	14 words — 1%
3	pubmed.ncbi.nlm.nih.gov Internet	13 words — 1%
4	www.f6publishing.com Internet	12 words — 1%

EXCLUDE QUOTES	ON	EXCLUDE SOURCES	< 12 WORDS
EXCLUDE BIBLIOGRAPHY	ON	EXCLUDE MATCHES	< 12 WORDS