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CASE REPORTS

Splanchnic ultrasound to guide unloading in VA-ECMO

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ABSTRACT

Patients supported with veno-arterial extracorporeal membrane oxygenation (VA-ECMO) may require left ventricular (LV) unloading to prevent pulmonary congestion and adverse hemodynamic interactions. However, defining the need and timing of unloading remains challenging, as current approaches rely predominantly on cardiac and pulmonary parameters, often neglecting systemic and organ-level congestion. We report the case of a 53-year-old woman admitted with cardiogenic shock following pericardial drainage and mediastinal mass biopsy. Due to rapid hemodynamic deterioration, VA-ECMO was initiated, resulting in stabilization. Early echocardiographic assessment showed severe biventricular dysfunction but evidence of partial aortic valve opening without LV distension. To further characterize the hemodynamic profile, splanchnic Doppler ultrasound was performed, demonstrating preserved renal arterial flow (resistive index <0.72), continuous intrarenal venous flow, and low portal vein pulsatility (<30%), consistent with a non-congestive phenotype. Based on this integrated assessment, LV unloading was deferred. Subsequent pulmonary artery catheterization confirmed low filling pressures despite reduced cardiac output. A diagnosis of stress-induced cardiomyopathy was suspected, and levosimendan was administered, leading to rapid improvement in cardiac function and successful ECMO weaning. Final pathology revealed a thymic neuroendocrine carcinoma. This case highlights the limitations of relying solely on cardiac indices to guide LV unloading decisions during VA-ECMO. Splanchnic Doppler provided a non-invasive, real-time evaluation of the perfusion–congestion balance at the organ level, complementing echocardiographic and invasive hemodynamic data. The integration of multimodal monitoring may allow a more comprehensive understanding of patient–device interaction and support individualized management strategies. Further research is needed to validate the role of

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splanchnic ultrasound in guiding unloading decisions and optimizing outcomes in patients with cardiogenic shock supported by VA-ECMO.

Key words: Cardiogenic shock, unloading, cardiac and abdominal ultrasound

Introduction

Patient's undertaking peripheral veno-arterial extracorporeal membrane oxygenation (V-A ECMO) may require further unloading to resolve the increased ventricular pressure related to both poor cardiac performance and backward flow from the arterial cannula. Although V-A ECMO favour perfusion of the splanchnic organs it can lead to eventually increased ventricular stress blood volume maldistribution favouring organ congestion and refractory pulmonary oedema. This can be prevent/treated by unloading the ventricle when there are clinical and ultrasound signs of congestion (i.e. closed aortic valve, intraventricular/aortic smoke, PCWP >18 mmHg, refractory pulmonary edema and arrhythmias) (1). However, the definition of the unloading need and remains challenging if straightforward criteria are not present. Pulmonary artery catheter is considered as the reference for the hemodynamic evaluation. Although, it may not be readily inserted at the first medical contact and its correlation with end-organ congestion, rather than cardiac/pulmonary, has not been established yet.

Case presentation

A 53-year-old woman was admitted to the emergency room due to worsening dyspnea over 24-hours (respiratory rate 30, oxygen saturation 92% in room air), with progressive symptoms, including weight loss and night sweats, lasting four weeks. A computed tomography scan revealed a circumferential pericardial effusion with a large mediastinal mass infiltrating the pericardium. She underwent urgent pericardial drainage (700 ml) and mass biopsy in cardiac surgery theatre on awake sedation. The procedure was complicated by overt respiratory failure with severe hypertension (175/90 mmHg) and hypoxemia requiring oro-tracheal intubation and post operative intensive care (ICU) admission. Upon arrival she was in cardiogenic shock (heart rate 140 bpm; blood pressure 96/75/65 mmHg on epinephrine 0.05 mcg/Kg/min, norepinephrine 0.2 mcg/Kg/min and vasopressin 0.03 UI/min; arterial lactate 5 mmol/dL – SCAI stage C) further worsening within 30 minutes (lactate 9 mmol/L, BP 85/65/55 mmHg, HR 140 bpm – SCAI D). Echocardiography showed severe biventricular dysfunction (LV velocity time integral 7.5 cm, LVEF 12%, total isovolumic time 19 sec/minute, MAPSE 7mm with post ejectional shortening, TAPSE 8 mm) with an apical ballooning phenotype (Figure 1). VA-ECMO was instituted with discontinuation of adrenergic drugs and haemodynamic stabilization within 6 hours. Echocardiography monitoring of patient-device interaction soon after V-A ECMO initiation showed biventricular unloading with partial but continuous aortic valve opening at each heartbeat (LV velocity time integral 9 cm, mild – to- moderate mitral regurgitation; PaO_2/FiO_2 250 with B lines < 50% over the lung fields). Abdominal assessment of splanchnic Doppler demonstrated Renal Doppler Resistive Index <0.72, continuous renal venous flow pattern, and a portal flow characterized by a pulsatility <30% (Figure 1) Renal Doppler resistive index (RDRI). The RDRI is calculated through the measurement of the peak systolic velocity (V_S) and the tele-diastolic velocity (V_d), according to the formula $(V_S - V_d)/V_S$. Pulsed-wave Doppler waveform analysis measures blood flow velocities, reflecting renal vascular resistance and compliance, therefore overall organ perfusion. Intra-renal venous Doppler is normally a continuous and not pulsatile. As venous congestion worsens, it becomes interrupted and pulsatile (biphasic or monophasic pattern corresponding to increasing severity)(2). Similarly, portal vein pulsatility may reflect an exhaustion of liver compliance in case of fluid overload or due to RV dysfunction (RV pressure overload/

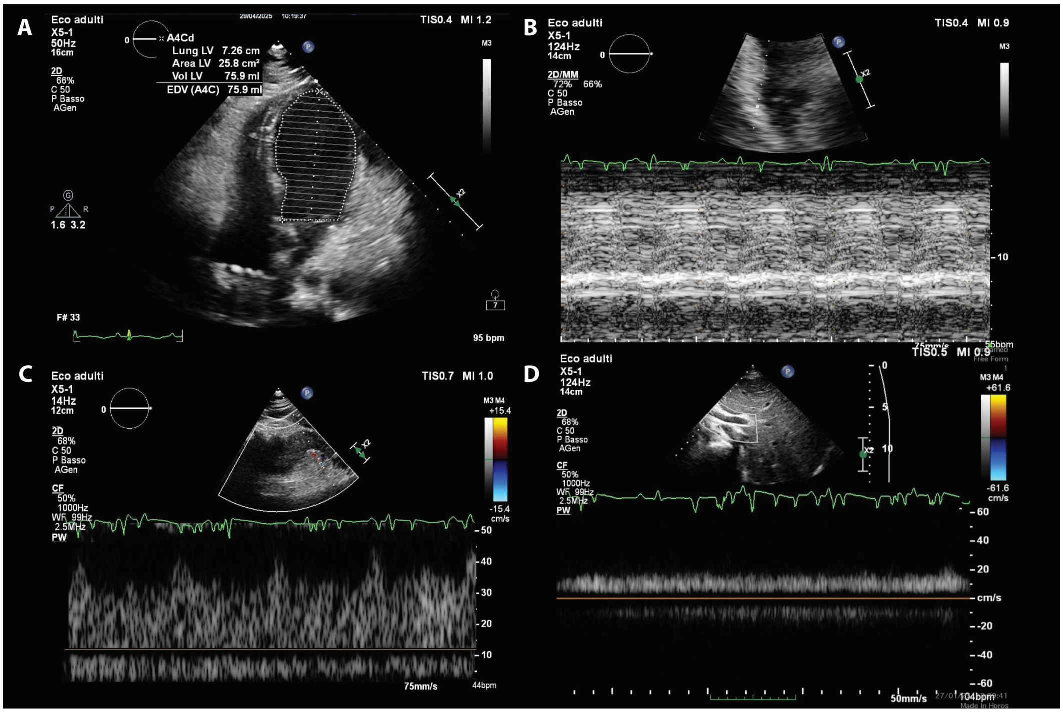

Figure 1. The ultrasound examination after the V-A cannulation. a) the left ventricular end diastolic volume with the ventricular shape recalling the apical ballooning phenotype. b) TAPSE (8 mm) with V-A ECMO flow 3.8 L/Min. c) Doppler kidney ultrasound with low resistive index and continuous venous Doppler flow and d) portal vein continuous pattern.

RV compliance dysfunction). Based on the cardiac and splanchnic Doppler findings, unloading was not deemed necessary. Two hours later, a pulmonary artery catheter confirmed the non-congestive phenotype: right atrial and wedge pressures were low (respectively, 7 and 12 mmHg), though cardiac output was severely reduced (Cardiac index 1.4 L/min/m^2, pulmonary artery pulsatility index - PAPi - 0.7). Troponin and BNP were elevated (peaked at 8000 ng/L and 1100 pg/mL respectively), and ECG showed precordial T-wave inversions. Levosimendan infusion was started on the putative diagnosis of biventricular CS due to stress related cardiomyopathy considering the context and the echo/ECG findings. After 12 hours since levosimendan cycle end, cardiac function significantly improved: LV VTI of 16 cm, total isovolumic time 13 second/min, MAPSE and TAPSE respectively > 10 and 16 mm with no post ejectional shortening; CO 2.9 L/min, PAPi 2). The patient was successfully weaned from VA-ECMO. Pathology revealed a small-cell neuroendocrine thymic carcinoma, and the patient was transferred to oncology ward four days later.

Discussion

Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) provides combined circulatory and respiratory support in patients with severe cardiogenic shock. In selected populations—such as stress-induced cardiomyopathy—outcomes may be favorable due to the potential for rapid myocardial recovery and short duration of support.(3). Establish the need, right timing and kind of unloading support in V-A ECMO is crucial. V-A ECMO by increasing afterload can induce a ventricular/ pulmonary congestion in severe LV failure and therefore to a maldistribution of the blood stress volume (BSV) at the organ level which may contribute to multi-organ dysfunction. Unloading devices are meant to either directly reduce cardiac workload (micro-axial flow pump) or reduce arterial elastance (intra-aortic balloon pump) thus improving the ventriculo-arterial coupling. Both, if appropriately targeted to the patient feature and need, may provide additional support leading to a reduction of filling pressures and therefore re-distribution of BSV. Although

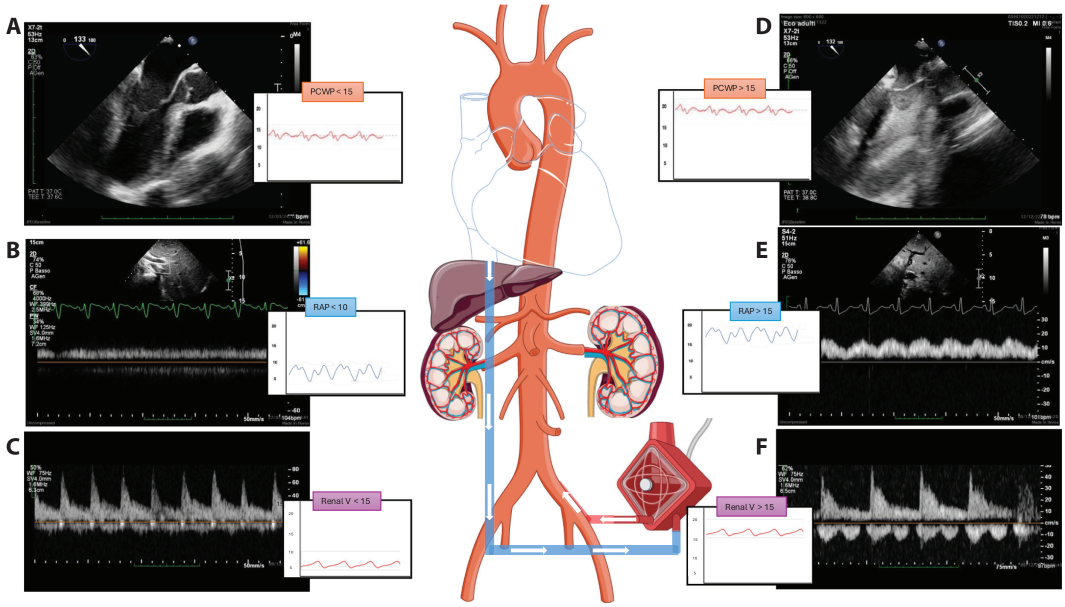

Figure 2. Side by side comparison of two AMI related CS on V-A ECMO with a non-congestive physiology on the left characterized by unloaded LV with normal aortic valve opening at each heart-beat and low pulmonary capillary wedge pressure (PCWP - A); continuous portal vein pulsatility corresponding to effective offloading of RV and lower right atrial pressure (RAP - B) and continuous renal venous flow pattern and good kidney perfusion (renal venous pressure < 15 mmHg- Renal Doppler Resistive Index < 0.7 - C). At the opposite, on the right side the case of severe LV dysfunction with overladed LV and stasis at ventricular and aortic level (PCWP > 20 mmHg); high (>50%) portal pulsatility index with high RAP and congested renal flow (biphasic) with high renal venous pressure (> 15 mmHg) leading to impair perfusion of the renal artery despite the continuous V-A ECMO flow (RDRi > 0.7).

the right timing for the unloading is yet unclear, some studies have found that early unload is associated with better outcome (4). However, current criteria for unloading selection focus almost exclusively on cardiac indices and neglect the systemic congestion (1). While, laboratory markers of end-organ dysfunction are late indicators, renal and portal Doppler provides a real-time, non-invasive assessment of perfusion-to-congestion coupling (5) (Figure 2).

Pulmonary congestion and LV unloading in VA-ECMO

Traditionally, the need for LV unloading has been guided by a combination of: echocardiographic findings (e.g., absent aortic valve opening, LV distension, spontaneous echo contrast), hemodynamic parameters (e.g., elevated pulmonary capillary wedge pressure) and clinical signs of pulmonary edema or refractory arrhythmias (1). Recent data suggest that the relationship between ECMO flow and LV loading conditions is not uniform (6, 7). The effect of increasing ECMO flow on LV filling pressures likely depends on multiple factors, including: ventricular systolic and diastolic function, ventricular geometry (acute vs chronic remodeling), ventricular interdependence, right ventricular contribution to preload. As a result, severe V-A ECMO may coexist with normal or only mildly elevated filling pressures, as observed in our case. This highlights the limitation of relying solely on ventricular function and static indices to guide unloading decisions.

Systemic (venous) congestion: the missing dimension in MCS evaluation

Increased CVP represents one of the three key hemodynamic determinants of splanchnic perfusion, together with splanchnic arterial inflow and venous vascular resistance. Yet, there is limited evidence regarding the ability of autoregulatory mechanisms to compensate for elevated CVP. Elevated right-sided filling pressures increase CVP, thereby raising splanchnic afterload and parenchymal wedging pressures within abdominal organs, particularly the kidney (8). Importantly, the phenotype of CS plays a crucial role in defining the aetiology of congestion. In the ischemic phenotype (AMI CS) hypoperfusion is primarily driven by reduced cardiac output, with congestion developing later as a consequence of compensatory vasoconstriction (veno and arterial). In contrast, in heart failure-related cardiogenic shock, congestion may represent the primary insult, leading to hypoperfusion through activation of the spleno-renal axis (9). In this setting, compensatory mechanisms—including increases in arterial pressure or splanchnic vasodilation—appear insufficient to preserve organ perfusion under conditions of venous congestion (8). This conceptual framework may be extended to VA-ECMO patients. Retrograde arterial flow from the peripheral cannula increases arterial pressure at the level of the femoral–iliac axis and, in the presence of severe biventricular dysfunction and variable watershed positioning, may contribute to increased splanchnic venous pressure despite ongoing venous drainage (Figure 2). Splanchnic venous hypertension occurs when CVP approaches or exceeds glomerular filtration pressure, resulting in a reduction or inversion of the perfusion gradient between systemic and organ venous compartments—a condition that can be conceptualized as a “splanchnic tamponade” (10). This leads to impaired regional blood flow, with perfusion limitation driven by congestion rather than arterial hypoperfusion. In this context, Doppler assessment of renal, hepatic, and portal circulation may allow early identification of distinct hemodynamic phenotypes, providing an integrated, organ-level perspective of cardio-circulatory interaction and patient–device interaction.

Role of splanchnic ultrasound in ECMO physiology

In this context, Doppler assessment of the splanchnic circulation provides a non-invasive, real-time evaluation of the perfusion–congestion balance at the organ level. Parameters such as:Renal Doppler resistive index, Intrarenal venous flow pattern, Portal vein pulsatility reflect the interaction between arterial inflow and venous congestion, and may serve as early indicators of hemodynamic maladaptation.In our case, the absence of abnormal splanchnic Doppler findings suggested a non-congestive phenotype, which was subsequently confirmed by invasive hemodynamic assessment. This concordance supports the potential role of splanchnic ultrasound as an adjunctive tool to refine clinical decision-making.Importantly, we emphasize that the absence of splanchnic congestion does not exclude the need for LV unloading, nor does it guarantee adequate perfusion. Rather, it provides a complementary perspective on the current hemodynamic state, which should be interpreted within a broader clinical context.

The role of complementary monitoring

A key message of this report is that no single modality or parameter is sufficient to guide management in VA-ECMO, thereby the complementary use of pulmonary artery catheter and ultrasound should be the preffered approach. Pulmonary artery catheterization provides quantitative hemodynamic measurements (gives numbers), echocardiography provides mechanistic and structural insights (explain the reason of the numbers), splanchnic ultrasound provides organ-level functional assessment (expaning the reason of numbers to the cardio- circulatory system). While pulmonary artery catheterization remains the reference standard for invasive hemodynamic evaluation, it may not be immediately available and does not directly capture organ-level consequences of congestion. Conversely, ultrasound techniques offer dynamic and bedside assessment, but should not be used in isolation to guide high-stakes decisions such as LV unloading.

Clinical implications and future directions

This case highlights the importance of moving beyond a cardiac-centric view, toward a more integrated assessment of cardio-circulatory interactions and end-organ perfusion. Splanchnic ultrasound may: help identify distinct hemodynamic CS phenotypes;

support interpretation of discordant findings between cardiac function and filling pressures; potentially assist in monitoring response to ECMO flow adjustments and therapeutic interventions. In addition, its role may extend beyond the initiation phase to support weaning trials, where early detection of maladaptation during the preload increase can inform regarding additional hemodynamization and exit strategies. These hypothesis are generated on the pathphysiological background and potential integration of the Doopler teqhiniques in framing congestion/pefusion profiles at organ level, proposing an holistic multimodal approach. However, the proof of it is still yet to be demonstrated. Further studies are needed to validate splanchnic Doppler parameters integrating integrate these measures into standardized multimodal monitoring protocols in different clinical scenarios of CS patients (heart lung interaction, response to inotropes and volume, different MCS) and define their role in guiding unloading strategies and weaning. We also believe that, in this exploratory phase, it is essential to analyze individual Doppler patterns in isolation. The mechanisms underlying a given pattern may be multiple—particularly in the setting of profound hemodynamic derangement such as biventricular failure. Therefore, each pattern should be described and interpreted individually to better elucidate the specific pathophysiological drivers.

Conclusion

In patient's supported with VA-ECMO, the assessment of congestion should not be limited to cardiac and pulmonary parameters alone. Integrating splanchnic ultrasound with echocardiography and invasive hemodynamics may provide a more comprehensive understanding of patient–device interaction, enabling more individualized and physiologically informed management.

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