ECRONICON OPEN ACCESS

EC EMERGENCY MEDICINE AND CRITICAL CARE
Case Report

The Value of Monitoring of Vital Organs Blood Flow by Full Doppler Study During Shock

Walid Shibl*

QCH, Ministry of Health, Saudi Arabia

***Corresponding Author**: Walid Shibl, ICU Consultant, QCH, Ministry of Health, Saudi Arabia.

Received: October 04, 2019; **Published:** October 17, 2019

Abstract

Background: The perfusion of the vital organs during shock state is the main goal of management of shock and may be as important as the monitoring of stroke volume, as we rely on LVOT VTI as a surrogate of stroke volume, we can consider the spectral arterial Doppler of vital organs a surrogate of blood flow to these organs, the study of blood flow in vital organs during shock by full Doppler study can give us a very important and rapid information about the ongoing perfusion which can help a lot during the golden hours of management.

Methods: We did complete Advanced Critical Care U/S in a severe obstructive shock patient, we used phase array 3.2 MH for Echocardiography and decrease to 2 MH to assess TCD, curvilinear abdominal probe to do full Doppler study of the renal artery branches, high frequency probe for DVT studies.

Case Report: a 40-year-old SCD male patient developed severe obstructive shock due the Systemic Fat Embolization Syndrome leading to acute cor-pulmonale, while he was shocked with BP 83/55 ON 20 mic/min levophed, we did Advanced Critical Care U/S which revealed weak dilated RT ventricle with a TAPSE 1.1 cm, compressing LT ventricle with a very low stroke volume [LVOT VTI 6 cm], at the same time we did full Doppler study of the LT MCA, RT interlobar renal artery, hepatic artery, we found that the TCD MCA and hepatic artery were normal despite shock state, on the other hand, the flow in the renal artery branch RT interlobar artery revealed a low flow state during the shock.

After improvement of hemodynamic with treatment, BP improved and stroke volume improved [LVOT VTI increase to 10.3], renal artery branch interlobar Doppler flow improved but revealed high resistance pattern with high RI 0.75.

Conclusion: Study of blood flow in the vital organs during shock state by arterial Doppler study revealed that the renal blood flow suffer first, this appear by the following changes of RT interlobar artery Doppler study, first, increase of RI denoting high resistance pattern, second, and with more deterioration of shock state interlobar renal artery branch Doppler revealed decrease both systolic and diastolic flow, these changes were correlated with BP and LVOT VTI. At the same time and to the degree of our patient shock the LT MCA TCD, Hepatic artery Doppler were normal while renal flow was impaired.

Recommendation: We need a big study to examine not only the macro circulation as stroke volume and cardiac output but we need also to examine the vital organ blood flow by full arterial Doppler during various degree of shock because the perfusion of vital organs is our main goal during management of shock.

Keywords: *RI Renal Artery Branches; MCA TCD; LVOT VTI; Microcirculation in Shock*

Abbreviations

RI: Resistivity Index; PI: Pulsitility Index; MCA: Middle Cerebral Artery; LVOT VTI: Left Ventricular Outflow Tract Velocity Time Integral; TAPSE: Tricuspid Annulus Plane Systolic Excursion; SCD: Sickle Cell Disease

Introduction

The cardiovascular system is an elaborate transport system whose major function is the supply of oxygen to metabolizing tissues, Systemic perfusion pressure is a key element of the macro circulatory system but is strongly influenced by vascular tone within the microcirculation. Individual organs adjust their microcirculatory perfusion to regulate the local supply of oxygen in order to meet their metabolic needs. In times of pathological and physiological stress, the microcirculation is likely to be a key player in the development of critical illness.

Clinicians are hindered by their inability to assess the microcirculation and the balance of metabolic supply and demand at the bedside. Hence, more readily available macro circulatory measures, such as cardiac output, mean arterial pressure, central venous pressure, serum lactate, and mixed venous oxygen saturation, are used as surrogates, with the necessary assumption that microcirculatory perfusion is coupled to the macro circulation. In shock states, however, this relationship is disrupted such that microcirculatory organ perfusion may be abnormal despite restitution of seemingly adequate macro circulatory parameters. Some authorities suggest that disordered perfusion alone is sufficient in itself to play an important role in critical illness and trigger the development of multi-organ failure [1,2].

The microcirculation and macrocirculation are uncoupled early after the onset of sepsis [3] and adequate early microcirculatory resuscitation may improve patient outcomes [4].

Hence, integrating a measure of microcirculatory function into resuscitation protocols may be desirable. There is currently a small suite of bedside tools available for microcirculatory analysis, of which side stream dark-field imaging is the most promising. Unfortunately, none has yet been validated.

Doppler imaging identifies changes in blood flow at microvascular level, it is a noninvasive, radiation free, non-expensive and repeatable, so, it could be a promising tool in examine the vital organs in shock.

We present a case of 40-year-old SCD male patient who developed severe obstructive shock due to systemic fat embolization syndrome and acute corpulmonale, we examine the blood flow to the vital organs during his shock state by full Doppler study of hepatic artery, LT MCA, and RT interlobar renal artery branch. During his hemodynamic deterioration with a very low stroke volume[LVOT VTI 6 cm] and BP [83/55] on levophed 20 mic/min, we found normal LT MCA and hepatic artery Doppler while there was a very bad RT interlobar artery blood flow with a PSV of 30 cm/sec and almost no diastolic flow, moreover, after an improvement of patient hemodynamic with management [increase of LVOT VTI to 10.2 cm and BP 110/60] RT interlobar renal artery branch blood flow improved but with high RI of 0.75 denoting a still renal hypo perfusion.

Case Report

40-year-old male patient K/C of SCD with infrequent admission was admitted to ER because of severe painful crisis and mild acute chest syndrome with O_2 saturation 92% on room air, HR 90/min, BP 130/80, Temp 37, RR 18/min, Chest, HT, Abdomen Exam: unremarkable.

He was treated with exchange transfusion, 1/2NS IVI, morphine injection.

Patient gradually deteriorated over the next 10 hours with increasing pain, dyspnea and O_2 desaturation with increase oxygen requirement and connected to mechanical ventilation.

I saw the patient after connecting to mechanical ventilation.

He was fully sedated with 200 mic fentanyl/hr, 3mg midazolam/hr, rocuronium 25 mg/hr, hemodynamically unstable with HR 155/min sinus rhythm, BP 83/55 on levophed 20 mic/min.

Chest Exam: bilateral basal crackles.

HT: Sinus tachycardia.

Abdomen: Distended with audible sounds.

Ventilator setting: TV 430, RR 26/min, I:E 1:1.7, FIO_2 100%, PEEP 12 [plateau pressure 33].

ABG on this setting: PH 7.18, PCO_2 62 mmgh, PO_2 94 mmgh, O_2 saturation 97%, HCO_3 19 mmgh.

All chemistry were markedly deteriorated compared with the results one day before admission.

ALP 145---697 U/L, ALT 53---446 U/L, AST 126---818 U/L, TOT BILI 32---41 mg/dl, DIR BILI 14---17 mg/dl, GGT 45---52 U/L, CK 126---1456 U/L, CK-MB 23---136 U/L, CREATININE 41---190 micmol/L, LDH 267---5137 U/L.

Haematology

WBC 6.7---14.7, HB 10---9.3 gm/dl, Plat 216---46.

PTT 42, PT 16, INR 1.61.

X-ray chest before intubation: basal infiltrate.

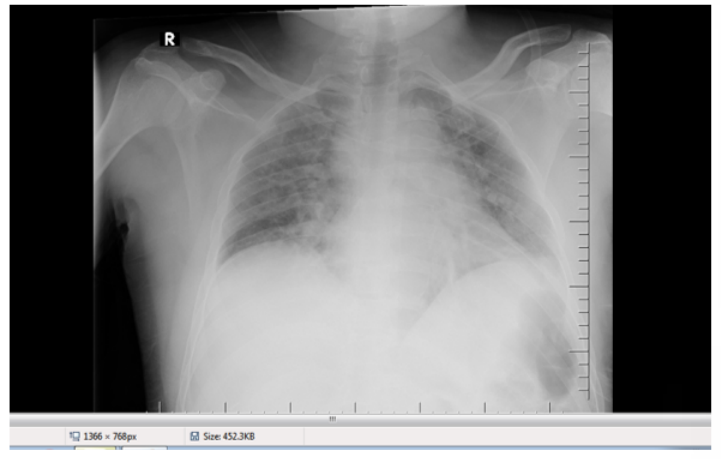

Figure 1: *Bilateral basal infiltrate, decreased lung volume.*

We used to do Advanced Critical Care U/S for admitted patient with acute chest syndrome on admission and with any deterioration.

On admission there was good LT ventricular function EF 55%, LVOT VTI 18.3CM, Good RT ventricular function and normal size.

Lung U/S slight scattered B- Lines on both lung bases.

We repeat Advanced Critical Care U/S with deterioration.

IVC: dilated non distensible.

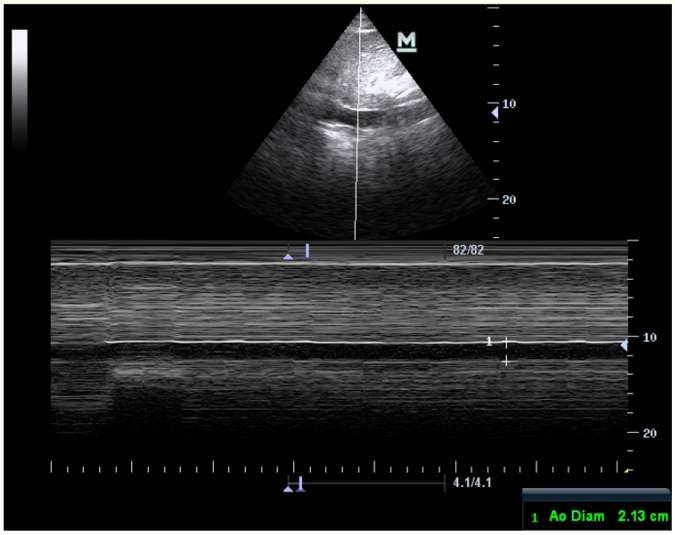

Figure 2A: *IVC dilated mildly distensible, going with CVP 15mmgh.*

RT ventricle dilatation with a diameter more than LT ventricle with poor contractility TAPSE 1.1 cm, compressing the LT side with weak LT side EF 20 - 30%, very low LVOT VTI 6 cm very low compared with value on admission 18.3 cm, mild TR, no pulmonary HTN, thin wall RT ventricle, all findings are going with severe obstructive shock due to acute corpulmonale.

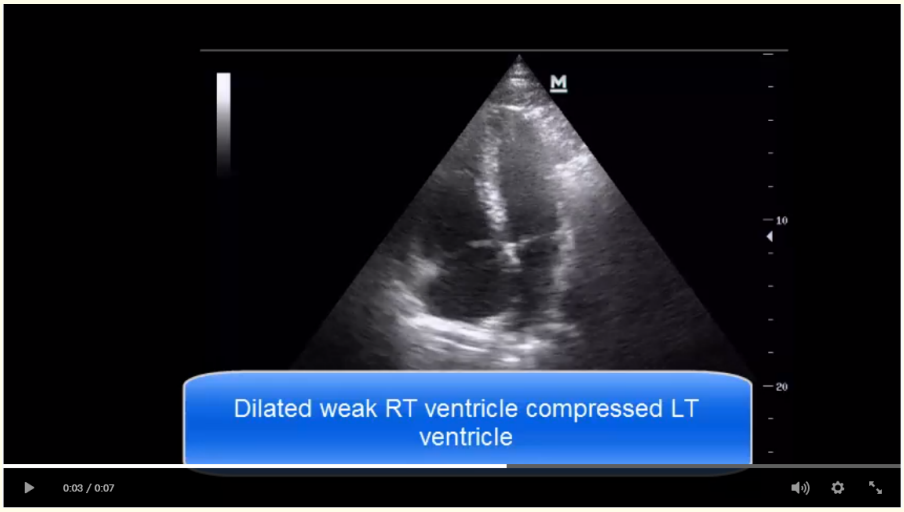

Figure 2B: *Video revealing dilated weak RT ventricle and RT atrium compressing the LT side.*

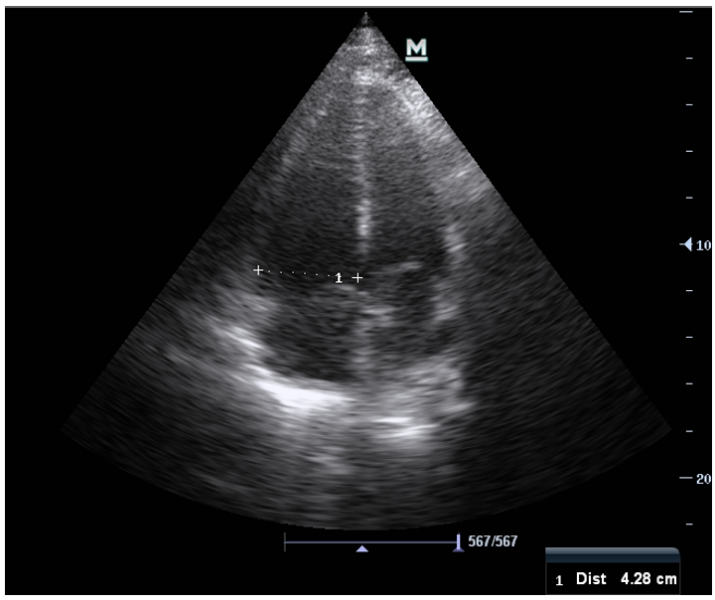

Figure 3: *Dilated RT atrium and ventricle more than LT side.*

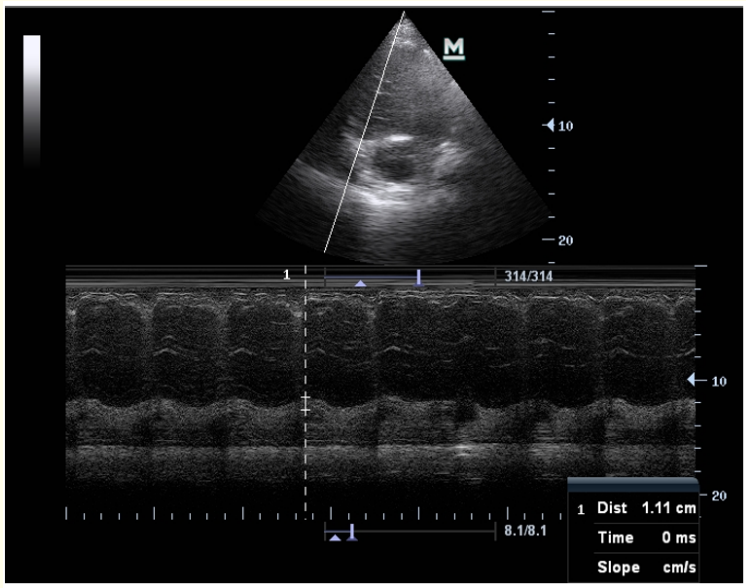

Figure 4: *Low TAPSE [Tricuspid annulus plane systolic excursion].*

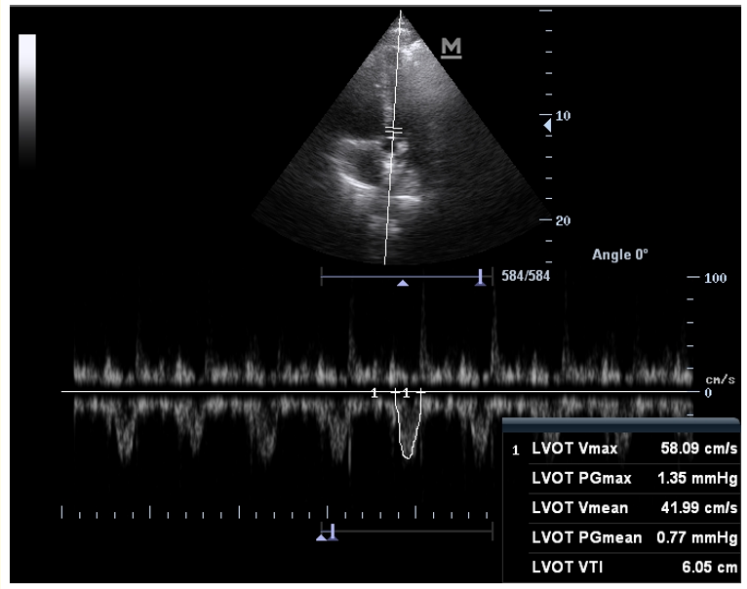

Figure 5: *Low LVOT VTI 6cm compared with 18.3cm on admission.*

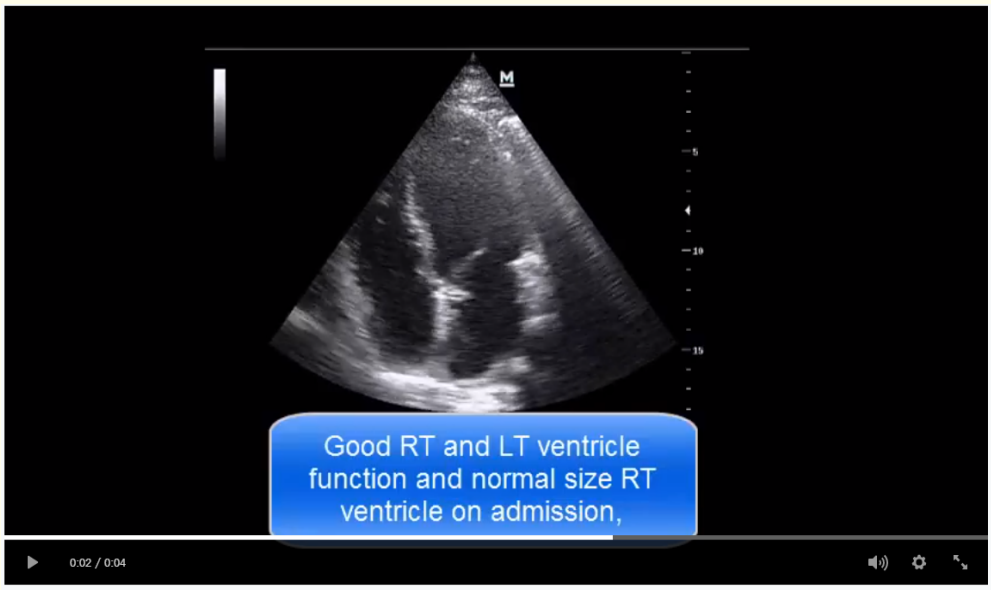

Figure 6: *Video of normal heart on admission.*

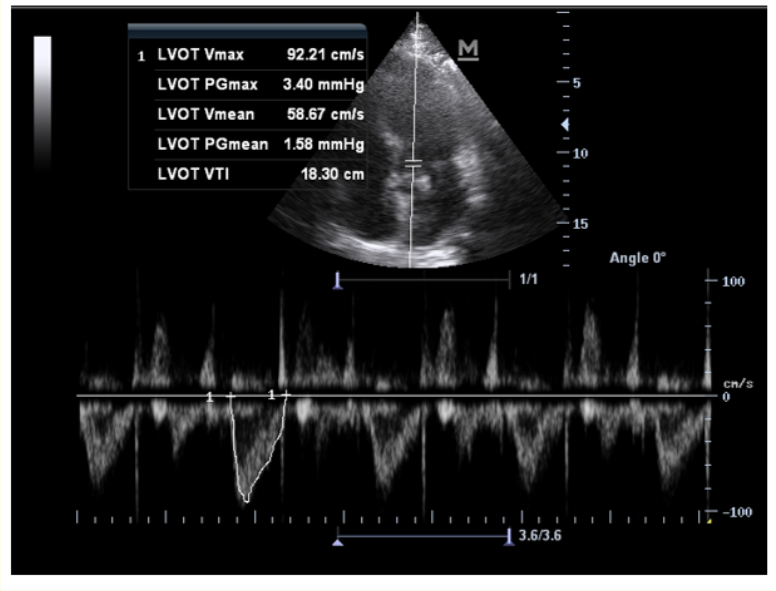

Figure 7: *LVOT VTI 18.3 on admission.*

No DVT on both femoral and popliteal veins by focused DVT study.

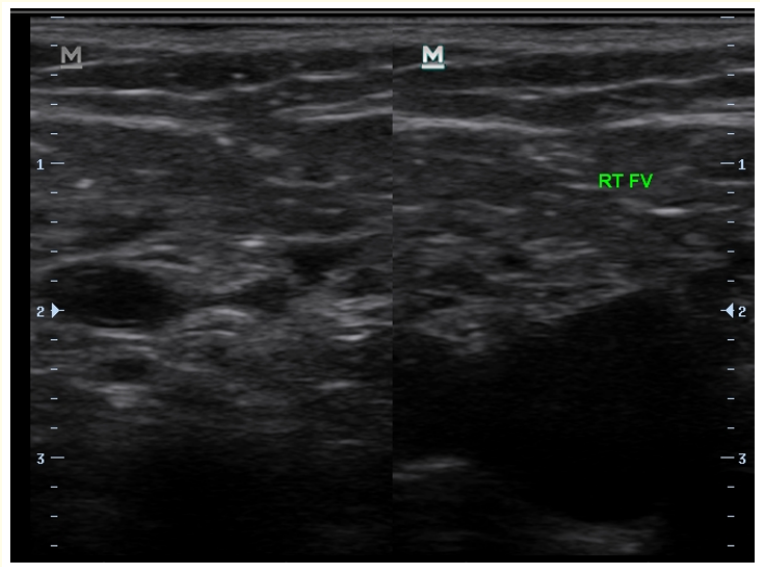

Figure 8: *No DVT RT FV.*

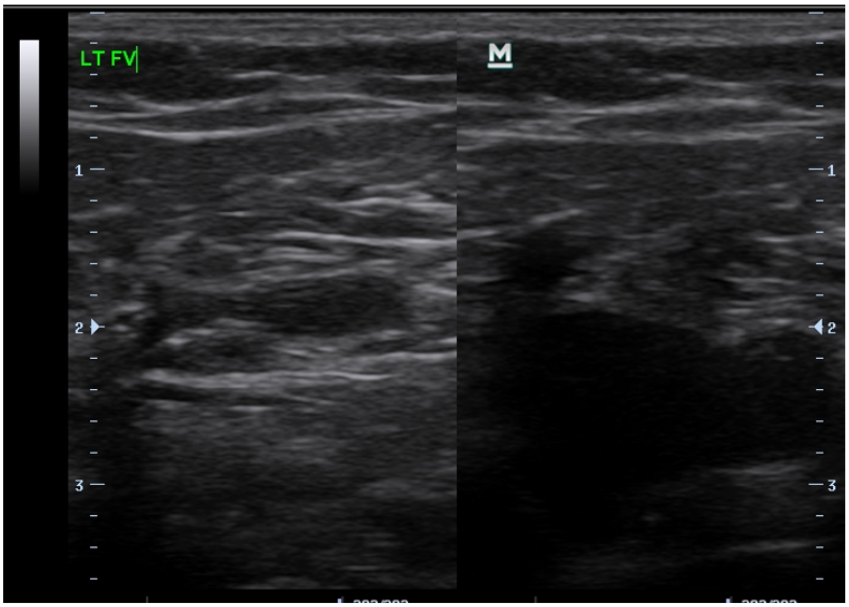

Figure 9: *No DVT LT FV.*

Lung U/S: Bilateral small basal consolidation and scattered B-lines both upper zones.

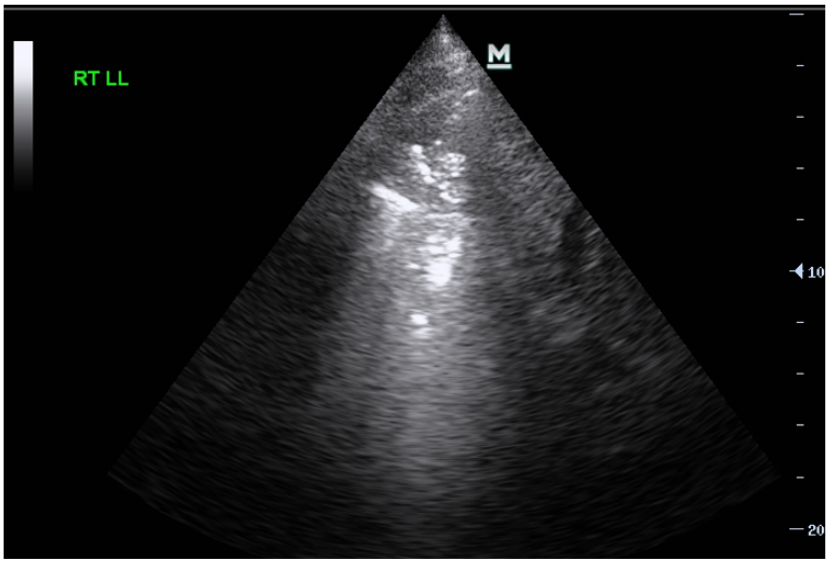

Figure 10: *RT basal consolidation.*

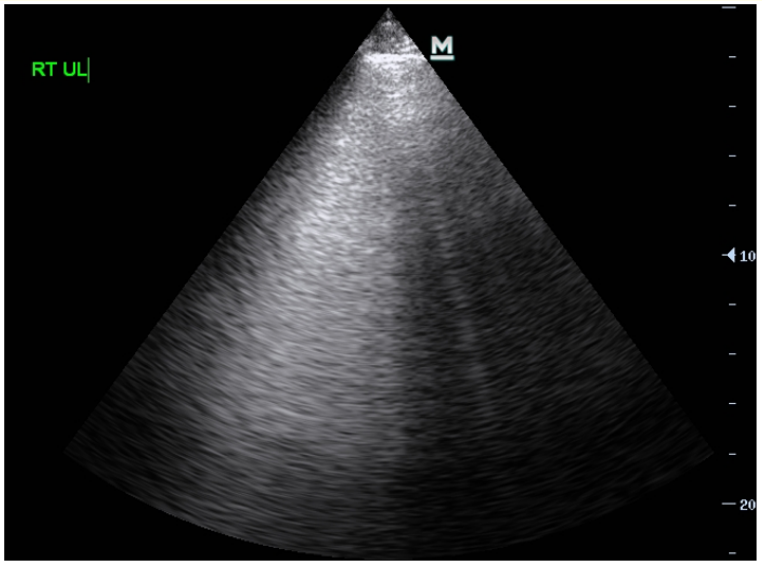

Figure 11: *B-lines.*

In all shocked patient, we used to do a full Doppler study of Blood flow of the vital organs [brain by TCD, renal, and hepatic].

First pan-Doppler study revealed as we always see good blood flow of the MCAs, hepatic artery, but poor flow of the renal blood flow, we always notice that the kidney is the first vital organ to be deprived of blood in shock.

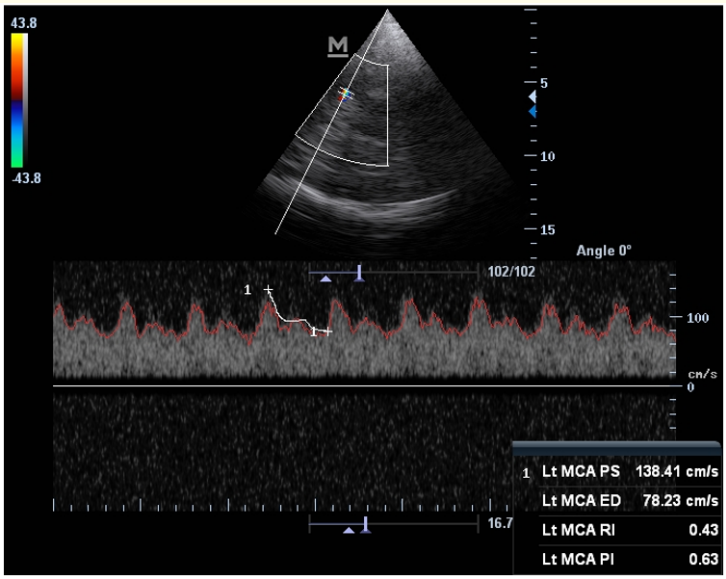

Figure 12: *Normal LT MCA blood flow.*

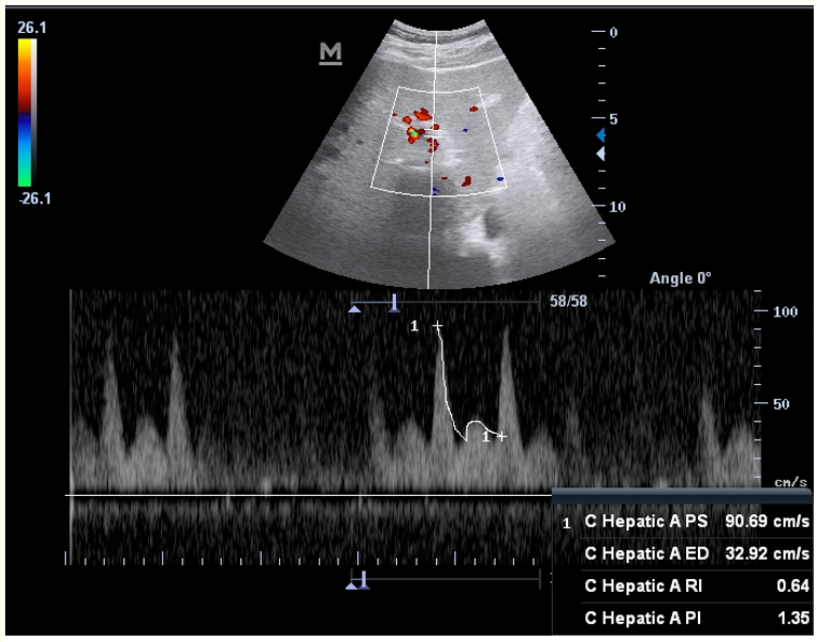

Figure 13: *Normal hepatic artery RI.*

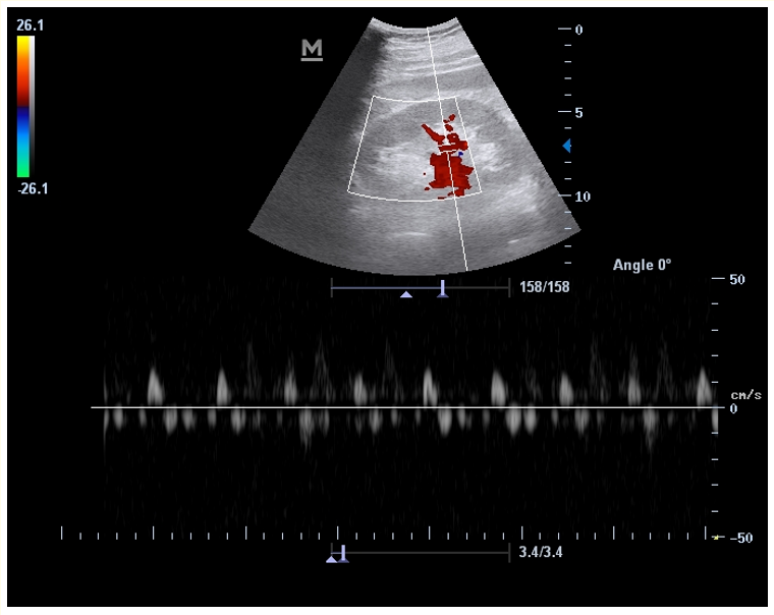

Figure 14: *Poor renal blood flow.*

Considering all these data, our diagnosis was systemic fat embolization syndrome due to marrow infarction in a sickle cell disease leading to severe obstructive shock and acute respiratory failure due to acute cor-pulmonale.

We urgently call a higher center for VA ECMO and planning for full conservative medical management with exchange transfusion, hold IVF because of dilated RT side, and adjusting ventilator setting to decrease Plateau pressure below 28 and wash CO_2 to decrease RT ventricle afterload, we increase RR from 26 to 33/min, decrease PEEP from 9to 7, and decrease TV from 420 to 350 ml and this lead to drop of plateau pressure from 32 to 27 cmH_2O and next ABG PCO_2 drop from 62 to 51 mmgh, as a consequence RT ventricle function improved with TAPSE 1.4 and LVOT VTI increase from 6 to 10.2 cm and BP increased to 110/60.

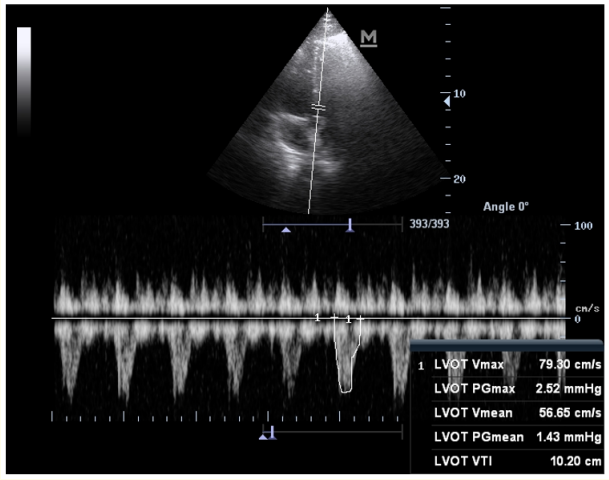

Figure 15: *Improved LVOT VTI from 6 to 10.2 cm.*

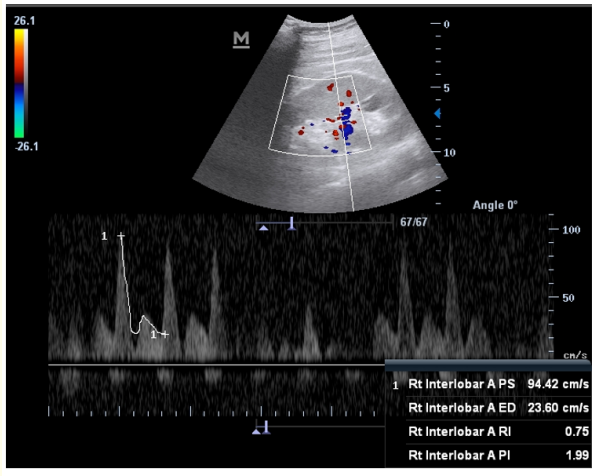

Figure 16: *TAPSE improving from 1.1 to 1.41 cm.*

Amazing, repeating the Pan-Doppler studies of vital organs after improvement of hemodynamics improved BP, TAPSE, LVOT VTI revealed improvement of renal blood flow but still with high resistance flow and high RI 0.75.

Unfortunately, Patient died before ECMO by sudden cardiac arrest.

Discussion

Cardiac output, i.e. the quantification of the blood flow that is generated by the heart, is by far the most important macro hemodynamic variable in the assessment of hemodynamic instable, critically ill patient, its assessment frequently determines and changes the direction of therapeutic interventions, there are a various methods to calculate and monitor the cardiac output in ICU, like, indicator dilution, pulse wave analysis, bio impedance and bio reactance as well as Echocardiography [5].

Signs and symptoms of shock, which is syndrome, are related to the different organ-specific response to hypoperfusion in a clinical progression based on an 'inverse priority pattern' in the body economy for importance of functions (skin first, visceral organs to follow, and the noble organs of heart and brain as last) [6,7].

Microcirculatory perfusion is subject to myogenic, metabolic, and neurohumoral mechanisms that control locoregional blood flow [8].

We present a case of a 40-year-old SCD male patient who developed severe obstructive shock due to systemic fat embolization syndrome, his BP 83/55, HR 155/min on noradrenaline 20 mic/min, dilated RT atrium and the RT ventricle diameter was more than 1.5 LT ventricle and compressing it with RT ventricle failure and TAPSE 1.1 cm, global LT ventricle EF by visual assessment 20 - 30% with septal dyskinesia, very low stroke volume as evidenced by LVOT VTI 6 cm, all these happened over 24 hrs and his Echocardiogram was normal 24 hrs ago.

We assessed the blood flow to his vital organs through full Doppler study of the LT MCA, Hepatic artery and RT renal interlobar branch inside the kidney at the same time of shock state, LT MCA Doppler study [PSV 138 cm/sec, EDV 78 cm/sec, PI 0.63] was normal, and hepatic artery Doppler [PSV 90 cm/sec, EDV 32 cm/sec, RI 0.64] was normal, in contrast, there was a bad flow of renal artery flow Doppler [PSV 30 cm/sec, with almost absent of diastolic flow].

Amazingly, after adjustment of ventilator setting to decrease plateau pressure from 32 to 27 cmH_2O and decrease of PCO_2 from 62 to 51 mmgh, the RT ventricle systolic overload decrease and function improved, TAPSE increase from 1.1 to 1.4 cm, LVOT VTI increase from 6 cm to 10.2 cm. We repeat Doppler study of all vital organs again, both LT MCA, Hepatic artery Doppler were normal, RT side interlobar renal artery showing improved Doppler flow with PSV 94 cm/sec, EDV 23 cm/sec but RI revealed high resistance circulation with a value of 0.75 denoting the continuous impairment of renal arterial circulation.

So in this case report, Doppler study of arterial circulation of vital organs during shock state revealed that not all arterial vascular beds of vital organs behave the same, at the stage of shock of our patient while renal circulation was impaired significantly, the hepatic and cerebral circulation were normal, moreover, renal circulation improved with improvement of BP and LVOT VTI.

Other studies revealed the differences in the microcirculation of different vital organs, arteriolar responses vary within different tissues; for example, cerebral perfusion is preserved given its unique metabolic requirements and fundamental importance to survival [9].

Conversely, perfusion of the splanchnic and cutaneous circulations is partly sacrificed [10].

This difference is mediated by variable expression of adrenergic receptor subtypes.

The microcirculation and macrocirculation are uncoupled early after the onset of sepsis [3] and adequate early microcirculatory resuscitation may improve patient outcomes [4].

Hence, integrating a measure of microcirculatory function into resuscitation protocols may be desirable.

There is currently a small suite of bedside tools available for microcirculatory analysis, of which side stream dark-field imaging is the most promising. Unfortunately, none has yet been validated.

Doppler imaging identifies changes in blood flow at microvascular level, it is a noninvasive, radiation free, non-expensive and repeatable, so, it could be a promising tool to examine the vital organs in shock.

Increase of RI of the renal artery branches was found to correlate with AKI in shock due to trauma, sepsis, and post cardiac surgery.

Moreover, In studies of RI in prerenal azotemia versus intrinsic AKI, an RI > 0.75 correlated well with the diagnosis of ATN, while prerenal azotemia was typically associated with an RI < 0.71.

In our case report, with severe hemodynamic deterioration with LVOT VTI 6 cm and BP 83/55 on 20 mic levophed/min, the blood flow to the kidney was very poor with RT interlobar artery peak systolic velocity 30 cm/sec and almost absent diastolic flow, but, with improved hemodynamic and increase of LVOT VTI TO 10.2 cm and BP to 110/60 the RT interlobar artery flow improved with PSV 94 cm/sec, and EDV 23 cm/sec but the RI was high 0.75 denoting renal arteriolar vasoconstriction and still renal hypo perfusion, so, according to our case kidney is the first vital organ to suffer hypo perfusion in shock and the derangement of blood flow is proportional to shock severity with the increase of RI in early stages and with more worsening of shock state there will be decrease of VTI of renal artery branches.

In our case report, blood flow to the brain and liver as represented by RT MCA and hepatic artery was preserved despite hemodynamic deterioration with LVOT VTI 6 cm and BP 83/55, RT MCA PSV 138, EDV 78 CM/sec with PI 0.63 and hepatic artery PSV 90, EDV 32 cm/sec with RI 0.64 which was normal.

To my knowledge no study investigate the MCA and Hepatic artery Doppler in shock state [11-16].

Conclusion

Doppler study of the vital organs arteries during shock along with monitoring the macro circulation through stroke volume could be an important and promising tool in management of shock.

Bibliography

1. Abraham E and Singer M. "Mechanisms of sepsis-induced organ dysfunction". *Critical Care Medicine* 35.10 (2007): 2408-2416.
2. Inca C. "The microcirculation is the motor of sepsis". *Critical Care* 9.4 (2005): S13-S19.
3. De Backer D., *et al*. "Coupling microcirculation to systemic hemodynamics". *Current Opinion in Critical Care* 16.3 (2010): 250-254.
4. Trzeciak S., *et al*. "Early increases in microcirculatory perfusion during protocol-directed resuscitation are associated with reduced multi-organ failure at 24 h in patients with sepsis". *Intensive Care Medicine* 34.12 (2008): 2210-2217.
5. Funcke S., *et al*. "Practice of hemodynamic monitoring and management in German, Austrian, and Swiss intensive care units: the multicenter cross-sectional ICU-CardioMan Study". *Annals of Intensive Care* 6.1 (2016): 49.
6. Haljamäe H. "Microcirculation and hemorrhagic shock". *American Journal of Emergency Medicine* 2.1 (1984): 100-107.
7. Nakai K., *et al*. "The distribution of adrenergic receptors in cerebral vessels: an autoradiographic study". *Brain Research* 381.1 (1986): 148-152.
8. Davis MJ. "Perspective: physiological role(s) of the vascular myogenic response". *Microcirculation* 19.2 (2012): 99-114.

9. Wan Z., *et al*. "The cerebral microcirculation is protected during experimental hemorrhagic shock". *Critical Care Medicine* 38.3 (2010): 928-932.

10. Reilly PM., *et al*. "The mesenteric hemodynamic response to circulatory shock: an overview". *Shock* 15.5 (2001): 329-343.

11. Bossard G., *et al*. "Early detection of postoperative acute kidney injury by Doppler renal resistive index in cardiac surgery with cardiopulmonary bypass". *British Journal of Anaesthesia* 107.6 (2011): 891-898.

12. Lerolle N., *et al*. "Renal failure in septic shock: Predictive value of Doppler-based renal arterial resistive index". *Intensive Care Medicine* 32.10 (2006): 1553-1559.

13. Schnell D., *et al*. "Renal resistive index better predicts the occurrence of acute kidney injury than cystatin C". *Shock* 38.6 (2012): 592-597.

14. Platt JF., *et al*. "Acute renal failure: possible role of duplex Doppler US in distinction between acute prerenal failure and acute tubular necrosis". *Radiology* 179.2 (1991): 419-423.

15. Gheisari A and Haghighi M. "Diagnostic value of Doppler ultrasound in differentiating prerenal azotemia from acute tubular necrosis in children". *Saudi Journal of Kidney Disease and Transplantation* 17.2 (2006): 168-170.

16. Izumi M., *et al*. "Differential diagnosis of prerenal azotemia from acute tubular necrosis and prediction of recovery by Doppler ultrasound". *American Journal of Kidney Diseases* 35.4 (2000): 713-719.

Volume 3 Issue 11 November 2019