Case Report

The role of side stream dark field microvasculature imaging in a rare case of vancomycin-resistant enterococcal endocarditis complicated by heparin-induced thrombocytopenia

Janak Bechar, Luigi Polesello, Maria Lombrano, Giampaolo Martinelli, Heyman Luckraz
Cardiothoracic Unit, Heart and Lung Centre, Wolverhampton, WV10 0QP, United Kingdom

ABSTRACT

Sidestream dark field (SDF) imaging allows direct visualization of microvascular architecture and function. We examine the role of an SDF imaging device in visualizing the sub-lingual microvasculature as a surrogate for splanchnic microperfusion. We demonstrate good correlation between current monitoring techniques and the SDF imaging device in a rare case of vancomycin-resistant enterococcal (VRE) sepsis along with heparin-induced thrombocytopenia (HIT). To the best of our knowledge, VRE endocarditis with concurrent HIT has not been described in literature. The role of SDF imaging may predict the earlier need for escalation of care, improving morbidity and mortality.

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INTRODUCTION

Assessment of hemodynamic status is often limited to global parameters and ignores the microvascular perfusion.[1] Sidestream dark field imaging (SDF) allows the direct visualization of the microcirculation.[2] The SDF device (handheld) uses a polarized green light (3 mm penetration), measuring scatter of hemoglobin containing red blood cells to visualize the microvasculature of human organs in real time.[3] Microvessels smaller than 20 μm are detected.

Three video time points of 20 s duration were recorded to map the sub-lingual microvasculature. Using software AVA 3.0, vessel density can be interrogated and perfusion quality averaged over three video samples. Parameters of vessel density are: Total vascular density (TVD), perfused small vessel density (PVD) and the De Backer score which is derived from the SDF device videos to describe the vessel density more reliably.[1,4] The score is also a function of the flow through the microvasculature and the proportion of perfused vessels. The proportion of perfused small vessels (PPV), microvascular flow index (MFI), and flow heterogeneity index (FHI) describe the perfusion quality of

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Address for correspondence: Dr. Janak Bechar, New Queen Elizabeth Hospital, Edgbaston, B15 2WB, United Kingdom.
E-mail: janakashwin.bechar@nhs.net

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microvasculature. The PPV is simply the percentage of vessels perfused as a function of the total number of vessels. The MFI describes the predominant type of flow in the microvasculature (0 = absent, 1 = intermittent, 2 = sluggish, 3 = normal). The FHI is calculated as the difference between the maximum and the minimum MFI of the different sub-lingual sites examined, divided by the average of all the MFI obtained. The methodology for all indices is detailed in De Backer *et al*.[1] and Koning *et al*.[5]

Recent clinical studies have demonstrated that alterations in microcirculation are key in the development of sepsis,[3,6,7] needing monitoring and early intervention.[8] We report a rare case of a septic patient with heparin induced thrombocytopenia (HIT), assessing the microcirculation using SDF.

CASE REPORT

A 63-year-old man was admitted to his local general hospital after feeling unwell for a fortnight. A blood culture confirmed the presence of vancomycin resistant enterococcal (VRE). Transthoracic esophageal echocardiogram confirmed dehiscence of the aortic valve with a root abscess, mitral valve vegetations, patent foramen ovale (PFO), and an inferior vena cava Eustachian valve. On transfer to our institution, he was in liver and renal failure (bilirubin 54 mg/dL, alkaline phosphatase 222 IU/L and alanine aminotransferase 1211IU/L), the latter being managed with continuous veno-venous hemofiltration (CVVHF) with heparin as the anticoagulant. Initial blood tests revealed a platelet count of 179 × 10^3 per mm.[3] During hospitalization, he developed a significant drop in platelet count to 35 × 10^3 per mm^3 on day 3 of CVVHF and HIT was confirmed by enzyme-linked immunosorbent assay serotonin release assay. Heparin was thus stopped, using Danaparoid as hemofiltration anticoagulation. While being investigated for HIT, platelet counts dropped to 16 × 10^3 per mm^3 having been off heparin for over a week.

The patient previously underwent an aortic valve replacement for aortic stenosis, 4 months prior to this acute presentation. His regular medication was as follows: Aspirin, bisoprolol, perindopril and atorvastatin. On his acute presentation, the patient underwent redo-aortic valve replacement (size 23 Perimount Magna tissue valve), mitral valve repair (size 28 mm St. Jude Medical saddle ring), closure of the PFO (4/0 Proline), repair of the aortic root abscess and excision of the Eustachian valve (due to possible endocarditis). The patient made slow progress on the Cardiac Intensive Care Unit (CICU) due to his ventilator dependence needing tracheostomy and his renal failure needing renal replacement therapy. In total, the patient was on cardiovascular support for 22 days (maximal of milrinone 10 ml/h, noradrenaline 20 ml/h and vasopressin 2 IU/h). After appropriate weaning, he was discharged after 6 months with further rehabilitation.

During his CICU stay, SDF device was used to examine the sub-lingual microvasculature as a surrogate of splanchnic cardiac perfusion at 4 times points postsurgery. There are currently no other means of assessing splanchnic circulation. The time points were early postoperative (48, 60 and 90 h) when the patient was still displaying signs of sepsis (pyrexia, low systemic vascular resistance, increased inflammatory markers, and increased inotropic-vasoconstrictor support) along with HIT being present and later on when sepsis was well-controlled, and his platelet count had recovered (47 days post postoperative). SDF data were not recorded between 90 h and 47 days at the patient was clinically stable. Table 1 displays global cardiovascular data from these time points and the data gathered from the SDF device for vessel density and perfusion quality.

DISCUSSION

Gilliam *et al*.[8] reported 18 cases of VRE endocarditis in literature. Clinical studies have shown microcirculatory alterations are key in sepsis development.[3,6-9] However, there are no reports in the literature assessing the microcirculation in a septic environment with the added insult of HIT. The implications of VRE endocarditis and HIT results in very high-risk surgery if undertaken. Our case report as demonstrates that surgery can be done when appropriate measures are taken. The SDF device was used at time points when the patient's condition changed clinically. Figure 1 shows a representative image of the microvasculature seen by the SDF device. Table 1 confirmed that the vessel density (TVD, PVD and De Backer Score) and perfusion quality (PPV, MFI and FHI) were at their lowest at time point 1 (48 h). PPV was very low at time point 1 (83.96), suggesting intermittent microvascular flow, despite a good cardiac index (CI) (3 L/min/m^2). CI in this case was, thus, not representative of microvascular perfusion. Furthermore, the microperfusion was intermittent, despite the low

Table 1: Microvascular and global monitoring data

Time point (postsurgery)	Vessel density			Perfusion quality			Cardiovascular variables					Inotropes and vasopressors	
	TVD	PVD	De Backer score	PPV	MFI small	FHI	CI	MAP	SVRI	pH	Lactate	NA	VA
1 (48 h)	20.35	17.25	11.66	83.69	1	0	3.7	66	1038	7.42	4.9	0.3	1
2 (60 h)	37.21	37.21	21.37	100	3.5	0.28	2.9	55	1178	7.36	6.9	0.2	1
3 (90 h)	35.78	35.78	22.54	100	3	0	2.8	74	1885	7.2	6.6	0.26	4
4 (47 days)	36.86	36.86	22.39	100	3	0	2.8	52	1715	7.45	1.1	0.06	None

Working, normal values and further information on the SDF device is described in Koning *et al*.[5] TVD: Total vascular density, PVD: Perfused small vessel density, PPV: Proportion of perfused small vessels, MFI: Microvascular flow index, FHI: Flow heterogeneity index. Calculated as the difference between the maximum and minimum MFI of different sub-lingual sites, divided by the average of all MFI obtained. CI: Cardiac index (L/min/m^2), MAP: Mean arterial pressure (mmHg), SVRI: Systemic vascular resistance index (dynes×s/cm^5/m^2), NA: Noradrenaline (mcg/kg/min), VA: Vasopressin (UI/h), SDF: Side stream dark field

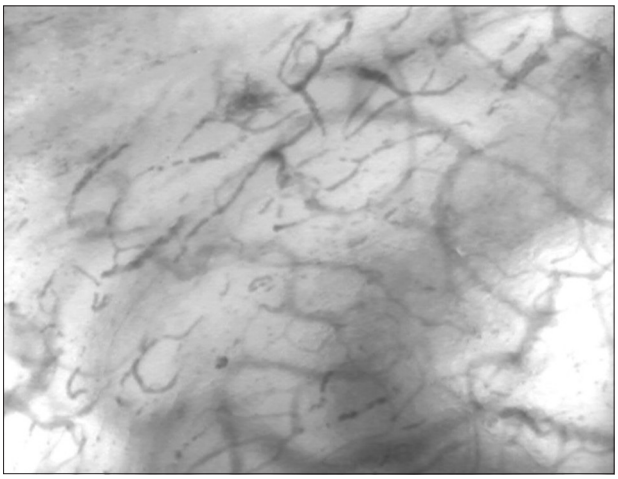

Figure 1: A representative image from the side stream dark field device. Capillaries (with red cells) and arterioles visualized

systemic vascular resistance index (SVRI) at time point 1 (1038).

Timepoint 2 (60 h) heralded a peak in the vessel density scores of TVD and PVD, as well as MFI. This, perhaps, suggests a microvascular hyperemia (MFI 3.5). A FHI of 0.28 could also signal patchy and irregular microvasculature, suggesting that some areas may be re-perfused (hyperemic) with persistent areas of ischemia. Moreover, according to Trzeciak *et al*. a heterogeneous flow (FHI = 0.28) is more often linked with lower mean arterial pressure (MAP) in septic patients compared with controls.[10] These patients have a higher chance of not surviving.

Not surprisingly, lactate was also markedly elevated at time point 2 (6.9 mmol/l), suggesting previous anaerobic respiration and metabolic stress. Interestingly, this time point also shows a decrease in MAP 55 mmHg, which supports the increase in lactic acid. This is also probably due to decreased inotrope and vasopressor administration.

De Backer score (vessel density) was elevated in comparison to time point 1 (21.37 vs. 11.66) but lower than time points 3 and 4 (22.54 and 22.39 respectively). The fluctuation of this score at time points 3 and 4 may be a hallmark of the vessel density being preserved, but not homogenously. The fall in MAP seen in time point 2 may be due to decreased inotrope and vasopressor administration.

The perfusion quality (PPV and MFI) plateaus from time point 3 (90 h). CI falls and plateaus at time point 3, inversely to perfusion quality in this instance. Lactate was also high (6.6 mmol/l) and pH low (7.2) despite a reasonable SVRI (1715 dynes × s/cm^5/m^2) and good microcirculation. This increased metabolic stress may be attributed to the high dose of vasopressin used (4 UI/h), with hypothesized subsequent splanchnic vasoconstriction due to an episode of sepsis requiring increased inotropic support. This, however, was not mirrored in the sub-lingual microcirculation. MAP was much better than other time points at 70 mmHg, once more suggesting that MAP is not a good indicator of microvascular perfusion. This suggests that microvascular monitoring in critically ill patients has benefit and is partially independent of global changes. This has also been demonstrated in literature.[1] While the SDF device did not alter the course of this case (as there is no case for comparison), it provided information about the microcirculation, which could be reflective of the splanchnic circulation. While no gold standard for measuring microperfusion exists, the SDF device shows promise in measuring such parameters.

CONCLUSION

To the best of our knowledge, VRE endocarditis with concurrent HIT has not been described in literature. The possible role of SDF imaging in the assessment of

microvascular tissue well-being is important, especially when there are fluctuating global cardiovascular characteristics. This technology may prove vital in predicting the earlier need for escalation of care, improving morbidity and mortality in complex cases.

Working, normal values and further information on the SDF device is described in Koning *et al*.[5]

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. De Backer D, Hollenberg S, Boerma C, Goedhart P, Büchele G, Ospina-Tascon G, *et al.* How to evaluate the microcirculation: Report of a round table conference. Crit Care 2007;11:R101.
2. Slaaf DW, Tangelder GJ, Reneman RS, Jäger K, Bollinger A. A versatile incident illuminator for intravital microscopy. Int J Microcirc Clin Exp 1987;6:391-7.
3. Boerma EC, Mathura KR, van der Voort PH, Spronk PE, Ince C. Quantifying bedside-derived imaging of microcirculatory abnormalities in septic patients: A prospective validation study. Crit Care 2005;9:R601-6.
4. De Backer D, Creteur J, Preiser JC, Dubois MJ, Vincent JL. Microvascular blood flow is altered in patients with sepsis. Am J Respir Crit Care Med 2002;166:98-104.
5. Koning NJ, Atasever B, Vonk AB, Boer C. Changes in microcirculatory perfusion and oxygenation during cardiac surgery with or without cardiopulmonary bypass. J Cardiothorac Vasc Anesth 2014;28:1331-40.
6. Sakr Y, Dubois MJ, De Backer D, Creteur J, Vincent JL. Persistent microcirculatory alterations are associated with organ failure and death in patients with septic shock. Crit Care Med 2004;32:1825-31.
7. Ince C. Microcirculation in distress: A new resuscitation end point? Crit Care Med 2004;32:1963-4.
8. Forrest GN, Arnold RS, Gammie JS, Gilliam BL. Single center experience of a vancomycin resistant enterococcal endocarditis cohort. J Infect 2011;63:420-8.
9. McDonald JR, Olaison L, Anderson DJ, Hoen B, Miro JM, Eykyn S, *et al.* Enterococcal endocarditis: 107 cases from the international collaboration on endocarditis merged database. Am J Med 2005;118:759-66.
10. Trzeciak S, Dellinger RP, Parrillo JE, Guglielmi M, Bajaj J, Abate NL, *et al.* Early microcirculatory perfusion derangements in patients with severe sepsis and septic shock: Relationship to hemodynamics, oxygen transport, and survival. Ann Emerg Med 2007;49:88-98, 98.e1-2.