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CASE REPORT
Other

A case report: use of cerebral oximetry in the early detection of cerebral hypoperfusion in a post-cardiac arrest patient during targeted temperature management

Shonda Ng and Yew Woon Chia *

Department of Cardiology, Tan Tock Seng Hospital, 11 Jalan Tan Tock Seng, Singapore 308433, Singapore

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Background We present a patient who received cerebral oximetry monitoring during targeted temperature management (TTM) post-cardiac arrest and discuss its potential in the early detection of cerebral hypoperfusion and implications on haemodynamics and ventilatory management.

Case summary A 60-year-old Chinese male was admitted for acute pulmonary oedema with Type 2 respiratory failure. He failed an initial trial of non-invasive ventilation and was planned for intubation and mechanical ventilation. However, the patient suffered a pulseless electrical activity cardiac arrest peri-intubation. He was started on our institution's protocolized post-cardiac arrest care bundle, which included cerebral regional oxygen saturation (rSO_2) monitoring and TTM. Initial arterial blood gas (ABG) post-return of spontaneous circulation showed severe respiratory acidosis, and the patient was sedated, paralyzed, and ventilator settings optimized. Repeat ABG showed resolution of respiratory acidosis. However, a drop in rSO_2 to 35% was subsequently noted. Ventilator settings were quickly adjusted, and dobutamine was started to improve global and cerebral perfusion. These measures improved cerebral rSO_2 to more than 50%. Patient was cooled for 24 h and gradually rewarmed. He was later extubated with a cerebral performance category of 1 and is now on outpatient follow-up.

Discussion During post-cardiac arrest care, there are many factors which can contribute to a decrease in cerebral blood flow. Therapeutic hypothermia and ventilation strategies, including the use of neuromuscular blocking agents, can both reduce pCO_2 which is a major regulator of cerebrovascular tone. Accidental hypocapnia can lead to adverse cerebral vasoconstriction and hypoperfusion. Without cerebral oximetry, cerebral ischaemia may not be detected early and can potentially result in secondary brain injury.

Keywords Case report • Cerebral oximetry • Hypocapnia • Post-cardiac arrest care • Targeted temperature management

* Corresponding author. Tel: +65 6357 7831, Email: yew_woon_chia@ttsh.com.sg
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Learning points

- During post-cardiac arrest care, haemodynamic changes, ventilation strategies, and therapeutic hypothermia may lead to a decrease in cerebral blood flow.
- Cerebral oximetry offers real-time monitoring of brain tissue oxygenation and serves as a safety measure during targeted temperature management and haemodynamic and ventilator titration.
- Cerebral oximetry can help in the early detection of cerebral hypoperfusion and prevent secondary brain injury.

Introduction

Cerebral oxygenation can be monitored non-invasively with the use of near-infrared spectroscopy (NIRS) to determine brain regional oxygen saturation (rSO_2). In post-cardiac arrest patients, there is often global cerebral ischaemia contributing to neurological dysfunction. Targeted temperature management (TTM) is often implemented to limit neurological injury caused by the hypoxia during cardiac arrest and help improve survival, as shown in the Hypothermia after Cardiac Arrest Study Group trial.[1] During TTM post-cardiac arrest, it is crucial to optimize cerebral perfusion to achieve good neurological recovery. We present a case where the use of cerebral oximetry monitoring post-cardiac arrest led to the early detection of cerebral hypoperfusion and discuss its implications on management and outcome.

Timeline

Case presentation

A 60-year-old Chinese male presented to the hospital with acute shortness of breath and lower limb swelling. He has a past medical history of stroke disease, chronic kidney disease, and ischaemic cardiomyopathy with a mildly reduced left ventricular ejection fraction of 45%. His cardiovascular risk factors include diabetes mellitus, hypertension, and hyperlipidaemia.

Clinical examination revealed bibasal crepitations with bilateral pitting oedema. The blood pressure on admission was 150/87 mmHg with sinus tachycardia with a heart rate of 129 beats per minute.

Initial arterial blood gas (ABG) on admission (*Table 1*) revealed Type 2 respiratory failure, while chest X-ray done showed bilateral pulmonary congestion.

He was initially started on non-invasive ventilation and intravenous frusemide and glyceryl trinitrate infusion. Despite initial clinical improvement, the patient became increasingly restless with

Clinical events	
Admission	Patient admitted for acute pulmonary oedema with Type 2 respiratory failure and started on trial of non-invasive ventilation
Intubation	Intubated in view of worsening hypoxaemia
Pulseless electrical activity (PEA) collapse	PEA collapse peri-intubation Total downtime 20 min
Return of spontaneous circulation (ROSC)	Initial arterial blood gas (ABG) post-ROSC: pH 7.14, pCO_2 54, pO_2 110, HCO_3 18, and SaO_2 97% Mean arterial pressure: 102 mmHg not requiring inotropic support Started on regional cerebral oxygen saturation (rSO_2) monitoring: 66% (left) and 67% (right)
Measures	Started on targeted temperature management (TTM) Ventilation rate increased to 28 breaths per min Tidal volume increased to 7–8 mL/kg predicted body weight
Drop in rSO_2	rSO_2 dropped to 33% (left) and 35% (right) Repeat ABG: pH 7.3, pCO_2 33, pO_2 138, HCO_3 16, and SaO_2 99% Central venous oxygen saturation ($ScvO_2$) 62% reflecting systemic oxygen delivery/consumption mismatch Pcv-aCO_2 gap 14 mmHg suggesting a low flow state
Measures	Ventilation rate decreased to target pCO_2 50–55 mmHg PEEP cut to lowest level which can maintain SaO_2 94–98% Dobutamine started to improve perfusion
Increase in rSO_2	rSO_2 improved to 55% (left) and 51% (right)
TTM complete	Cooled for total 24 h, then gradually rewarmed
Outcome	Extubated successfully with good neurological recovery Cerebral performance category 1 On outpatient follow-up

worsening hypoxaemia and decision was made for intubation and mechanical ventilation. However, he suffered a pulseless electrical activity cardiac arrest peri-intubation with a low-flow time of 20 min before return of spontaneous circulation (ROSC).

He was started on our institution's protocolized post-cardiac arrest care bundle (*Figure 1*) which included continuous rSO_2 monitoring and TTM with mild induced hypothermia at 33°C. An oesophageal probe was inserted for core body temperature monitoring. Initial ABG post-ROSC revealed a Type 2 respiratory failure with pH 7.14, pCO_2 54, pO_2 110, HCO_3 18, and SaO_2 97%. Patient was sedated and paralyzed and ventilator settings were optimized to correct the respiratory acidosis. Patient was ventilated with a tidal volume of 7 mL/kg predicted body weight and at a ventilator rate of 28 breaths per minute. Initial cerebral oximetry revealed an rSO_2 66% (left) and 67% (right).

Table 1 Investigations on admission

ABG	pH 7.12
	pCO_2 51 mmHg
	pO_2 45 mmHg
	HCO_3 17 mmol/L
	SaO_2 64%
Troponin	47 ng/L
Creatinine	206 µmol/L
Haemoglobin	8.9 g/dL

Post-resuscitation SpO_2 was maintained above 94% and mean arterial pressure (MAP) was constantly above 80 mmHg without the need for inotropes or vasopressors. However, it was subsequently noted that the cerebral rSO_2 had decreased to 33% (left) and 35% (right) and a repeat ABG showed resolution of the respiratory acidosis with pH 7.3, pCO_2 33, pO_2 138, HCO_3 16, and SaO_2 99%.

Ventilator settings were immediately adjusted and the minute ventilation reduced by lowering the ventilator rate to aim for mild therapeutic hypercapnia with a target pCO_2 of 50–55 mmHg.[2] PEEP was reduced from 12 cmH_2O to avoid an excessively high intrathoracic pressure which may impede cerebral venous drainage. Patient was also started on dobutamine infusion to improve the global and cerebral perfusion as the central venous oxygen saturation ($ScvO_2$) of 62% reflected a systemic oxygen delivery/consumption mismatch, while a Pcv-aCO_2 gap of 14 mmHg suggested a low-flow state. The above interventions resulted in an improvement in cerebral rSO_2 to 55% (left) and 51% (right) (*Table 2*).

Patient was cooled for 24 h and then gradually rewarmed. A repeat transthoracic echocardiogram revealed severe global hypokinesia with a left ventricular ejection fraction of 26%.

He was subsequently extubated after 6 days in the cardiac intensive care unit with improvement in Glasgow coma scale score and underwent rehabilitation in the general ward. On discharge, he had a cerebral performance category of 1 and is now on follow-up in the outpatient clinic.

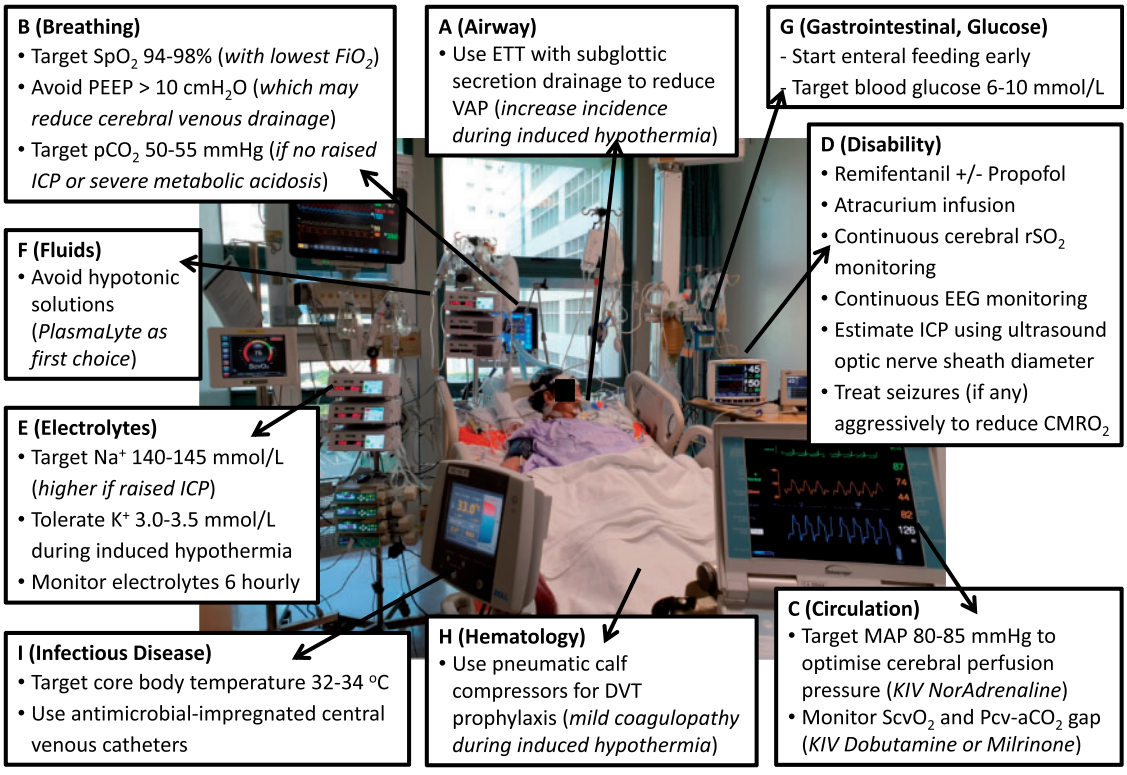


Figure 1 Cardiac intensive care unit management of post-cardiac arrest syndrome (the 'A to I' approach).

Table 2 **Parameters charted during intensive care unit stay**

Date	19 April 2018	19 April 2018	20 April 2018	20 April 2018	20 April 2018	20 April 2018	20 April 2018	20 April 2018	20 April 2018	20 April 2018
Time	2000	2300 Prior to collapse	0000 Post-intubation	0300	0600	0800 After correction of respiratory acidosis	0900	1100	1300 After allowing for permissive hypercarbia and increasing perfusion	1500
Temperature (°C)	37.1 Tympanic		36.9 Oesophageal	33.0 Oesophageal	33.0 Oesophageal	33.0 Oesophageal	33.0 Oesophageal	33.0 Oesophageal	33.0 Oesophageal	33.0 Oesophageal
Heart rate (b.p.m.)	128	161	130	106	96	88	86	88	89	92
Blood pressure (mmHg)	152/78 (99) NIBP	204/145 (169) ABP	162/78 (102) ABP	127/66 (84) ABP	112/68 (81) ABP	125/76 (93) ABP	113/98 (105) ABP	128/72 (90) ABP	134/70 (91) ABP	168/87 (116) ABP
Respiratory rate	31	38	24	28	28	28	28	22	20	20
Oxygen saturation (%)	88	80	98	100	98	98	99	97	97	98
End-tidal CO_2 (mmHg)			31	30	23	24	25	30	34	25
$ScvO_2$ (%)				65	40	30	61	65	74	81
rSO_2 (%)			Left 66 Right 67	Left 67 Right 67	Left 53 Right 45	Left 41 Right 40	Left 33 Right 36	Left 40 Right 41	Left 66 Right 67	Left 66 Right 67

ABP, arterial blood pressure; NIBP, non-invasive blood pressure.

Discussion

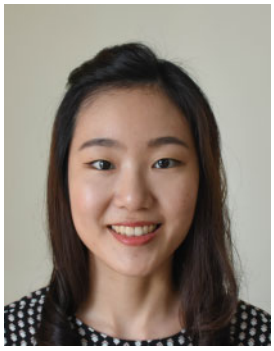
It is important to optimize cerebral perfusion after ROSC to improve the number of neurologically intact survivors. There have been studies looking at the correlation of cerebral rSO_2 during resuscitation and successful ROSC and it has also been shown that a high rSO_2 on arrival at the hospital predicted good 90-day neurologic outcomes.[1,3,4] However, equally important to a successful ROSC is the care the patient receives after admission to the intensive care unit. There have been limited tools to guide optimization of cerebral oxygenation during post-cardiac arrest care.

Targeted temperature management is an essential part of the post-ROSC care in cardiac arrest patients and it has been shown to increase the chance of survival with favourable neurological outcomes.[1] However, during post-cardiac arrest care, there may be a drop in mean arterial pressure and cardiac output leading to a drop in cerebral blood flow.[5] Ventilation strategies including the use of neuromuscular blocking agents together with the reduction of metabolism during therapeutic hypothermia[6] also reduce pCO_2 which is a major regulator of cerebrovascular tone. Accidental hypocapnia can occur and result in adverse cerebral vasoconstriction and cerebral ischaemia.[7,8] This may not be detected early as neurological function is difficult to assess clinically when the patient is sedated and paralyzed during TTM.

Conclusion

Cerebral oximetry offers real-time and dynamic monitoring of brain tissue oxygenation and can potentially serve as a safety measure during the hemodynamic and ventilatory management of patients post-cardiac arrest.[9] Cerebral oximetry may be the answer to assessing cerebral perfusion during the crucial period of TTM and aid in preventing secondary iatrogenic brain injury. More studies can be done to assess the morbidity and mortality benefit of cerebral rSO_2 monitoring during post-cardiac arrest care, its accuracy in assessing cerebral ischaemia and the cut-off values which would signify sufficient cerebral perfusion to impact good neurological recovery.

Lead author biography



Shonda Ng graduated from the Yong Loo Lin School of Medicine, National University of Singapore (NUS) in 2014. She obtained her Master of Medicine and MRCP (UK) in 2016. She completed her internal medicine residency training in 2017 and is currently a third year senior resident in the National Healthcare Group Cardiology residency programme. She is also an active clinical tutor for medical students from Yong Loo Lin School of Medicine and Lee Kong Chian School of Medicine.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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