Pericardial Injury Following Severe Sepsis from Faecal Peritonitis —A Case Report on the Use of Continuous Cardiac Output Monitoring

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Abstract

We report on a case of a 43-year-old man who developed reversible myocardial depression and pericarditis related to severe sepsis secondary to rectosigmoid colonic perforation. The management of this patient was aided by the use of a continuous thermodilution cardiac output catheter and monitor, recently introduced in clinical practice.

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Key words: Pericarditis, Pulmonary artery catheter, Thermodilution

Introduction

The intermittent manual bolus thermodilution method is the most common means of determining cardiac output in critically ill patients. Although widely used, there are many factors which may affect the accuracy of this technique such as the volume, temperature and timing of the injectate.[1] Recent technological advances have led to the development of continuous cardiac output (CCO) monitoring systems. In this case, modified pulmonary artery catheters have a thermal filament, which is positioned in the right ventricle. Without the need for fluid injectate, the filament continually transfers a level of heat directly into the blood according to a pseudorandom binary sequence. The resulting temperature change is detected downstream in the pulmonary artery by thermistor and cross-correlated with the input sequence to produce a thermodilution 'washout' curve. Both laboratory[2] and clinical[3-5] evaluation of this method of cardiac output measurement have validated the results obtained. It has been predicted that in clinical practice, CCO measurement will replace the intermittent thermodilution method.[6]

We present a case in whom a continuous cardiac output catheter and monitor proved both beneficial and convenient.

Case Report

A previously well 43-year-old Chinese man was admitted with a 24-hour history of abdominal pain and vomiting. He had constipation for three days and had used laxatives to no effect. On further questioning, he admitted to attempting to relieve his constipation with a garden hose, which he had inserted per rectum one day previously. On initial examination, he was found to have a rigid, tender abdomen, a temperature of 37.3°C, heart rate of 145.min^{-1}, blood pressure 140/90 mmHg and respiratory rate of 21.min^{-1}. Chest radiograph showed gas under the diaphragm and emergency laparotomy was scheduled for suspected large bowel perforation. Preoperatively the patient's blood pressure fell to 70/50 mmHg which responded to volume replacement with 1 L gelofusine and crystalloid. Haemoglobin was 18.5 g/dL, white cell count 1.02 x 10^9/L and platelets 239 x 10^9/L.

Laparotomy revealed perforation of the rectosigmoid colon with gross faecal peritonitis. A Hartmann's procedure and peritoneal lavage were performed. Intraoperatively, the patient required direct arterial and central venous pressure (cvp) monitoring. Dopamine 10 μg.kg^{-1}.min^{-1} and noradrenaline 0.1 μg.kg^{-1}.min^{-1} were required for inotropic support. Crystalloids 3 L, colloids 2.5 L and blood 2 units were administered. Intraoperative blood gas analysis showed pH 7.254, PO_2 88 mmHg (FiO_2 0.4) and base excess -11.9. Urine output was 375 ml.

Postoperatively, the patient was admitted to the surgical intensive care unit (SICU) where a pulmonary artery catheter with continuous cardiac output monitor (Abbott TDG) was inserted. On dopamine 8 μg.kg^{-1}.min^{-1} and noradrenaline 0.08 μg.kg^{-1}.min^{-1}, his mean arterial pressure (MAP) was 65 mmHg, cardiac output 6.2 L.min^{-1} and systemic vascular resistance (SVR) 653 dyne.s.cm^{-5} (Table I). He was sedated with morphine 1 mg.h^{-1} and

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TABLE I: HAEMODYNAMIC VARIABLES IN SURGICAL INTENSIVE CARE—DAYS 1 TO 4

Day number in SICU	Day 1	Day 1	Day 2	Day 2	Day 2	Day 3	Day 3	Day 3	Day 4
Time	12:50	20:30	08:00	10:00	20:00	08:00	10:25	20:00	10:30
Dopamine $\mu g.kg^{-1}.min^{-1}$	8	8	8	8	4	4		30	26
Adrenaline $\mu g.kg^{-1}.min^{-1}$		0.16							
Noradrenaline $\mu g.kg^{-1}.min^{-1}$	0.08	0.1	0.2	0.32	0.30	0.18	0.12		
Heart rate min^{-1}	149	160	160	155	109	126	126	123	131
Mean arterial pressure mmHg	65	75	65		80	78	88	81	74
Mean pulmonary artery pressure mmHg	20	20	18		26	26		36	32
Central venous pressure mmHg	20	20	18		11	10	17	24	20
Pulmonary artery wedge pressure mmHg	21	17	16		17	17		33	21
Intermittent bolus cardiac output $L.min^{-1}$	6.2	11	9		5.4	6.0		3.2	4.9
Continuous cardiac output $L.min^{-1}$	6	10			5	6.1		3.1	
Cardiac index $L.min^{-1}.m^{-2}$	5.4	5.6				3.3		2.8	2.7
Systemic vascular resistance $dyne.s.cm^{-5}$	653	445	550		1110			1011	899
Systemic vascular resistance index $dyne.s.cm^{-5}.m^{-2}$	1182	650						1862	1655

midazolam 0.5 $mg.h^{-1}$. Ceftriazone 2 g OM, metronidazole 500 mg 8 h and gentamicin 120 mg OM were started. Optimization of filling pressures to a pulmonary artery wedge pressure of 17 mmHg and addition of adrenaline 0.16 $\mu g.kg^{-1}.min^{-1}$ resulted in increased cardiac output to 11 $L.min^{-1}$ and fall in SVR to 445 $dyne.s.cm^{-5}$.

He remained on synchronized intermittent mandatory ventilatory support using FiO_2 0.5. The next day, he developed ST segment elevation on the ECG which responded to glyceryl trinitrate infusion 4 $mg.h^{-1}$. Cardiac enzymes sent off on the subsequent three days were markedly elevated—aspartate aminotransferase (AST) (15 to 33) was 149 and 154 u/L, and lactate dehydrogenase (LDH) (180 to 380) was 568 and 922 u/L on day 2 and 3, respectively. Creatine kinase (CK) (40 to 210) was up to 8854, 1447 and 1497 u/L on days 2, 3 and 4. Creatine kinase MB isoenzyme (CKMB) (0.5 to 5.0) was 33.4 on day 3 and 6.5 on day 4. This may have been related to gut perforation and resection. Twelve-lead ECG showed low voltage complexes consistent with pericarditis but no evidence of myocardial infarction. Cardiac output fell to 5.4 $L.min^{-1}$ despite continuing inotropic support. Cardiac output on day 3 fell to 3.2 $L.min^{-1}$ despite tailing down noradrenaline to recover to 4.9 $L.min^{-1}$ on the next day on (Table I) dopamine 26 $\mu g.kg^{-1}.min^{-1}$. The pulmonary artery catheter was removed on day 5 when a bedside echocardiogram confirmed recovery of myocardial function.

The patient required repeated platelet transfusions over nine days to maintain platelet count above 25×10^9/L. He was extubated on day 6 but required repeat laparotomy for drainage of paracolic and pelvic collections on day 11. He was weaned off the ventilator on day 14 and enteral feeding was established. His recovery was complicated by methicillin-resistant *Staphylococcus aureus* (MRSA) and Enterobacter septicaemia for which he was treated with vancomycin 500 mg 6 h and gentamicin 120 mg OM. He was discharged to the ward after 20 days on the SICU and went home two weeks later.

Discussion

In sepsis, the usual clinical picture is that of increased cardiac output and decreased systemic vascular resistance. The treatment is directed towards aggressive fluid

resuscitation and antibiotic therapy with the use of inotropes to improve oxygen delivery while maintaining an adequate perfusion pressure to the vital organs. Reversible myocardial depression due to humoral mediators has been shown both clinically[7] and experimentally[8] in sepsis. However, we are not aware of any previous reports of transient pericarditis in cases with unrelated sepsis.

We feel that the management of this patient was helped by the use of the pulmonary artery catheter with CCO measurements. In particular, the unexpected gradual decrease in cardiac output was graphically displayed and allowed prompt intervention. Subjectively, the cardiac output readings obtained by CCO varied by less than 10% from those measured by the bolus thermodilution technique (Table I). This is supported by previous prospective clinical investigations[5,9] which demonstrate clinically acceptable agreement between the continuous and intermittent bolus method when cardiac output is in the range 2.5 to 12 $L.min^{-1}$. Whilst all the information given above could have been obtained using a conventional pulmonary artery catheter and cardiac output determined by the bolus thermodilution method, the use of a CCO catheter removes some of the errors inherent in the method, as well as allowing easy recognition of unexpected changes. In addition response to therapeutic manoeuvres can be readily obtained by looking at the continuous trend readout rather than doing repeated measurements at arbitrary intervals. The savings in terms of reduced manpower costs and nursing time can be offset against the higher price of the modified pulmonary artery catheter.[9,10] Furthermore microbial colonization is more likely in an open when compared to a closed delivery system[11] for thermodilution such as the CCO system. There is evidence to suggest that continuous cardiac output measurements are less accurate at body temperature exceeding 38.5°C.[12]

While there is now a sufficient body of evidence to support the use of CCO monitoring in clinical practice, wider experience will be required to determine which patients would best benefit from this technology.

REFERENCES

1. Stetz C W, Miller R G, Kelly G E, Raffin T A. Reliability of the thermodilution method in the determination of cardiac output in clinical practice. Am Rev Respir Dis 1982; 126:1001-4.
2. Mihaljevic T, Von Segesser L K, Tonz M, Leskosek B, Seifert B, Jenni R, et al. Continuous versus bolus thermodilution cardiac output measurement: A comparative study. Crit Care Med 1995; 23:944-9.
3. Boldt J, Menges T, Wollbruck M, Hammermann H, Hempelmann G. Is continuous cardiac output measurement using thermodilution reliable in critically ill patients? Crit Care Med 1994; 22:1913-8.
4. Haller M, Zollner C, Briegel J, Forst H. Evaluation of a new continuous thermodilution cardiac output monitor in critically ill patients: A prospective criterion standard study. Crit Care Med 1995; 23:860-6.
5. Burchell S A, Yu M, Takiguchi S A, Ohta R M, Myers S A. Evaluation of continuous cardiac output and mixed venous oxygen saturation catheter in critically ill surgical patients. Crit Care Med 1997; 25:388-91.
6. Edwards D. Continuous thermodilution cardiac output: A significant step forward in hemodynamic monitoring. Crit Care Med 1997; 25:381-2.
7. Siegel J H, Greenspan M, Del Guercio L R M. Abnormal vascular tone, defective organ transport and myocardial failure in human septic shock. Ann Surg 1967; 165:504-17.
8. Kumar A, Thota V, Dee L, Olson J, Uretz E, Parrillo J E. Tumor necrosis factor alpha and interleukin beta are responsible for in vitro myocardial cell depression induced by human septic shock serum. J Exp Med 1996; 1893:949-58.
9. Ditmyer C E, Shively M, Burns D B, Reichman R T. Comparison of continuous with intermittent bolus thermodilution cardiac output measurements. Am J Crit Care 1995; 4:460-5.
10. Boldt J, Heesen M, Muller M, Hempelmann G. Continuous monitoring of critical patients with a newly developed pulmonary arterial catheter. A cost analysis. Anaesthetist 1995; 44:423-8.
11. Nelson L D, Martinez O V, Anderson H B. Incidence of microbial colonization in open versus closed delivery systems for thermodilution injectate. Crit Care Med 1986; 14:291-3.
12. Luchette F, Johannigman J, Branson R, Porembka D, Davis Jr K. Effects of body temperature on accuracy of continuous cardiac output measurements. Crit Care Med 1995; 23:A137.