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CASE REPORT

Haemostatic management for aortic valve replacement in a patient with advanced liver disease

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Abstract

Redo-sternotomy and aortic valve replacement in patients with advanced liver disease is rare and associated with a prohibitive morbidity and mortality. Refractory coagulopathy is common and a consequence of intense activation of the coagulation system that can be triggered by contact of blood with the cardiopulmonary bypass circuitry, bypass-induced fibrinolysis, platelet activation and dysfunction, haemodilution, surgical trauma, hepatic decompensation and hypothermia. Management can be further complicated by right heart dysfunction, porto-pulmonary hypertension, poor myocardial protection, and hepato-renal syndrome. Complex interactions between coagulation/fibrinolysis and systemic inflammatory response syndrome reactions like "post-perfusion-syndrome" also compound haemostatic failure. Given the limited information available for the specific management and prevention of cardiopulmonary bypass-induced haemostatic failure, this report serves to guide the anaesthesia and medical management of future cases of a similar kind. We discuss our multimodal management of haemostatic failure using pharmacological strategies, thromboelastography, continuous cerebral and liver oximetry, and continuous cardiac output monitoring.

Key words: Cardiac surgery; Liver failure; Coagulopathy; Cardiopulmonary bypass

Core tip: Cardiac surgery in patients with advanced liver disease is associated with significant morbidity and mortality. Refractory coagulopathy is common and requires a proactive multidisciplinary haemostatic management strategy. Given the limited information available for the specific management and prevention of cardiopulmonary bypass induced haemostatic failure, this report serves to guide the anaesthesia and medical management of future cases of a similar kind. We discuss our multimodal management of haemostatic failure using pharmacological strategies, thromboelastography, continuous cerebral and liver oximetry, and continuous cardiac output monitoring.

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INTRODUCTION

Redo-sternotomy and aortic valve replacement (AVR) in patients with advanced liver disease is rare and associated with a prohibitive morbidity and mortality. Refractory coagulopathy is common and a consequence of intense activation of the coagulation system that can be triggered by contact of blood with the cardiopulmonary bypass (CPB) circuitry, CPB-induced fibrinolysis, platelet activation and dysfunction, haemodilution, surgical trauma, hepatic decompensation and hypothermia. Management can be further complicated by right heart dysfunction, porto-pulmonary hypertension, poor myocardial protection, and hepato-renal syndrome. Complex interactions between coagulation/fibrinolysis and systemic inflammatory response syndrome (SIRS) reactions like "post-perfusion-syndrome" also compound haemostatic failure.

We present a patient with critical aortic stenosis who underwent redo-sternotomy and AVR prior to being listed for orthotopic liver transplantation. In this context, there is little information on the specific management of CPB-induced haemostatic failure. Therefore, we discuss our multimodal management of haemostatic failure using pharmacological strategies, thromboelastography (TEG), continuous cerebral and liver oximetry, and continuous cardiac output monitoring.

CASE REPORT

A 46-year-old male (weight 68 kg, height 183 cm) presented to our institution with acute pulmonary oedema secondary to severe aortic stenosis. The patient consented for a redo-sternotomy and AVR, with an estimated perioperative mortality of 50%. Previous cardiac history included open valvotomy *via* median sternotomy for a congenital calcified bicuspid aortic valve at age 6. The patient had a 10-year history of chronic liver disease secondary to alcohol abuse, with a Child Pugh Score of 8 (Child Class B), and a Model for End-Stage Liver Disease (MELD) Score of 12. The liver disease was further complicated by severe portal hypertension with ascites, thrombocytopaenia, oesophageal varices and portal hypertensive gastropathy. Two years prior, he underwent an emergency laparotomy for bleeding umbilical varices, which required intensive care unit (ICU) admission and an 8-unit red blood cell transfusion for hemorrhagic shock.

On this admission a transthoracic echocardiogram revealed preserved systolic left and right ventricular function, a severely calcified bicuspid valve (aortic valve area: 0.7 cm^2; mean aortic valve pressure gradient of 60 mmHg), moderate aortic regurgitation, mild mitral regurgitation, moderate pulmonary hypertension and a dilated ascending aorta (5.4 cm). Other cardiovascular risk factors included IgA nephropathy (creatinine 110 μmol/L, estimated glomerular filtration rate 73 mL/min per 1.73 m^2). There was no history of smoking or diabetes. The pulmonary oedema settled with conservative medical therapy. A coronary angiogram and right heart catheter study revealed no occlusive coronary artery disease, with a cardiac index of 3.4 L/min per square meter and a pulmonary artery pressure of 71/30 mmHg (mean 33 mmHg). Preoperative investigations including TEG are summarised in Tables 1-3. A detailed perioperative haemostatic coagulation strategy was formulated by a team composed of anaesthetist, haematologist, cardiac surgeon and intensivist.

The day before surgery, terlipressin [1 mg intravenous (*iv*) every 6 h] was commenced. Prior to induction of anaesthesia, an 8-French Rapid Infuser Catheter (Arrow) was inserted into each arm. Invasive monitoring included a 20 Gauge arterial line, 4-lumen central venous catheter, continuous cardiac output and continuous mixed venous oximetry measured with a fiberoptic pulmonary artery catheter (Edwards Lifesciences, Irvine CA) (Figure 1). External defibrillator pads were applied as a safety precaution. Bispectral index monitoring and cerebral and hepatic tissue oxygenation (Invos, Somanetics®) were measured with a cerebral/somatic oximeter, by placing disposable transducers over the right and left forehead (Figure 1), and on the skin overlying the lower right costal margin (Figure 2). The oximeters provided real-time monitoring of brain and liver oxygen saturations, measuring oxygen consumption and delivery. This allowed for detection and correction of cerebral and hepatic oxygen desaturation to optimise haemodynamic intervention. Tranexamic acid (1 g *iv* load then 500 g/h infusion) was commenced to minimize fibrinolysis during and after CPB. Octreotide (100 mcg bolus, then 25 mcg/h) was commenced to control portal hypertension and minimise hepatic ischaemia reperfusion injury from CPB. Vancomycin (1 g *iv*) and ceftriaxone (1 g *iv*) were administered for antimicrobial prophylaxis.

Redo-sternotomy was performed using an oscillating saw while lifting up the sternal wires. Dense adhesions of the right ventricle and posterior table of the sternum precluded access to the heart for central venous cannulation. Consequently, the femoral artery and vein were cannulated and the venous cannula carefully positioned using transoesophageal echocardiography guidance in the right atrium. After careful dissection around the heart and full heparinisation, the standard on-pump AVR technique was applied. A second venous cannula was added *via* the superior vena cava to the venous circuit to allow venous drainage, further minimizing hepatic congestion. After aortic cross clamp, pulsatile CPB was established, complete with haemofiltration to prevent fluid overload and maintain electrolyte neutrality. During CPB the patient was severely vasoplegic requiring escalating doses of noradrenaline (20 μg/min *iv*) and vasopressin (0.4 IU/min *iv*) to maintain a mean arterial pressure of 50 mmHg. Optimal pump flow rates and vasopressor use were

Table 1 Perioperative laboratory values and heparinase thromboelastography results

	Ref. ranges	Pre-operative	Pre-CPB	Rewarming Haemostatic intervention: FFP 15 mg/kg	Immediately post separation from CPB Haemostatic intervention: Protamine 500 mg; DDAVP 0.3 mg/kg; Concentrated fibrinogen 4 g; Platelets 2 pooled doses; 2 units packed RBC	15 min post separation from CPB Haemostatic intervention: Prothrombine × 1000 IU; then continuous infusion at 100 IU /h	30-min post separation from CPB Haemostatic intervention: Nil	Arrival intensive care unit
R (min)	4-8	6.4	5.0	6.7	6.9	28.9	7.8	6.7
K (min)	0-4	2.0	1.8	1.8	1.8	4.9	1.5	1.6
Angle (deg)	47-74	62.7	63.5	65.6	65.2	49.6	68.5	65.5
MA (mm)	54-72	53.7	61.9	61.3	57.6	71.8	69.0	70.1
LY30 (%)	0-8	0.7	0.3	0.0	0.0	1.4	0.0	0.0
INR	-	1.3	1.2	1.4	1.7	1.5	1.4	1.4
PT	11-15 s	13 s	14 s	15 s	19 h	17 h	16 h	16 h
APPT	22-38 s	36 s	39 h	> 200 h	49 h	50 h	45 h	38 s
Fib clauss	2.0-4.0 g/L		2.3 g/L	1.3 L	2.6 g/L	2.0 g/L	1.6 L	1.8 g/L
D-dimer	< 0.23 mg/L	1.05 h	Not measured	0.94 h		1.15 mg/L	Not measured	Not measured
Hb	130-180/L	90 L	83 L	73 L	55 L	85 L	Not measured	71 L
WBC	4.0-11.0 × 10^9	3.9 L	4.2 × 10^9	10.8 × 10^9	8.2 × 10^9	11.7 h	Not measured	9.4 × 10^9
Platelets	150-400 × 10^9	76 L	64 L	57 L	140 L	120 L	Not measured	73 L

The thromboelastography was performed after each of haemostatic interventions described. CPB: Cardiopulmonary bypass; FFP: Fresh frozen plasma; DDAVP: Desmopressin acetate; INR: International normalised ratio; PT: Prothrombin time; APPT: Activated partial prothrombin time; WBC: White blood cells; RBC: Red blood cells; MA: Maximum amplitude; K: Clot formation time; R: Reaction time.

Table 2 Perioperative arterial blood gases

	Ref. ranges	Pre-operation	Pre-cardiopulmonary bypass	Rewarming	Post separation	Closure	Post-op day 1	Post-op day 2 (venous)
pH	7.35-7.45	7.34[1]	7.32[1]	7.37[1]	7.24[1]	7.34[1]	7.34[1]	7.33[1]
pCO_2 (mmHg)	35-45	37	38	35	50[2]	42	39	45
pO_2 (mmHg)	80-110	105	230[2]	388[2]	385[2]	404[2]	111[2]	33[1]
HCO_3^- (mmol/L)	-	19	19	20	20	22	20	23
Base excess (mmol/L)	-3/+3	-6[1]	-6[1]	-4[1]	-6[1]	-3	-5[1]	-2
O_2 sat (%)	> 94	98	100	100	100	100	100	56[1]
Na^+ (mmol/L)	135-148	132[1]	132[2]	136	138	138	138	130[1]
K^+ (mmol/L)	3.5-5.3	4.0	4.0	4.9	3.9	3.9	4.6	4.5
Cl^- (mmol/L)	95-106	107[1]	107[2]	106	108[2]	108[2]	106	99
Ionised Ca^{2+} (mmol/L)	1.13-1.32	1.16	1.07[1]	0.92[1]	0.83[1]	1.06[1]	1.10[1]	1.10[1]
Haemoglobin (g/L)	120-180	80[1]	78[1]	71[1]	77[1]	86[1]	71[1]	63[1]
Glucose (mmol/L)	0.0-5.0	5.9[2]	6.3[2]	8.3[2]	6.2[2]	3.8[1]	8.0[2]	8.9[2]
Lactate (mmol/L)	3.9-5.8	0.9	0.6	4.0[2]	2.9[2]	2.0[2]	1.5	1.3

[1]Value below reference range; [2]Value above reference range.

Table 3 Perioperative renal function and liver function tests

	Ref. ranges	Pre-op	Arrival in ICU	Day 1 post op
Urea (mmol/L)	3.2-7.3	8.7[2]	6.3	7.6[2]
Creatinine (μmol/L)	62-106	110[2]	91	125[2]
Albumin (g/L)	35-52	31	25[1]	36
Globulins (g/L)	25-35	46[2]	19[1]	20[1]
Bilirubin (μmol/L)	< 18	40	35[2]	47[2]
ALP (IU)	40-130	99	51	49
ALT (IU)	< 41	30	21	22
GGT (U/L)	< 60	100[2]	40	40

[1]Value below reference range; [2]Value above reference range. ICU: Intensive care unit; ALP: Alkaline phosphatase; ALT: Alanine aminotransferase; GGT: Gamma-glutamyl transpeptidase.

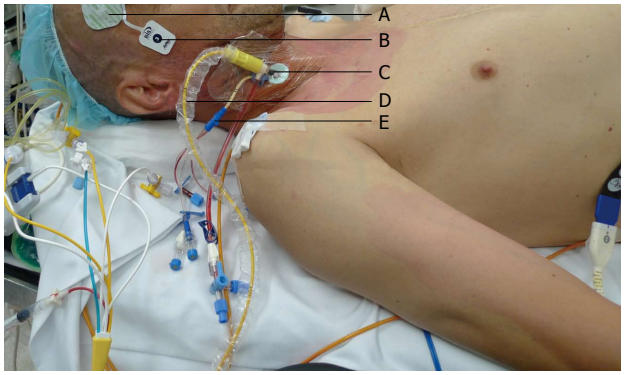


Figure 1 Monitoring used for redo-stenotomy and aortic valve replacement. A: Cerebral oximeter (Invos, Somanetics®); B: Bispectral index; C: 9 French Internal jugular sheath; D: Continuous cardiac output and mixed venous oximetry measured with a fiberoptic pulmonary artery catheter (Edwards Lifesciences, Irvine CA); E: 4-lumen central venous catheter.

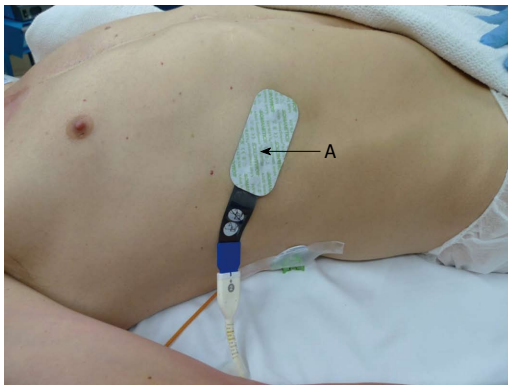


Figure 2 Hepatic tissue oxygenation measured by positioning an oximetry disposable transducer between the ribs and over the liver. A: Hepatic oximeter (Invos, Somanetics).

guided by cerebral and liver oximetry. There was excellent correlation between cardiac output, mixed venous saturations and cerebral and liver oximetry throughout the case (Figures 3 and 4). In response to progressive refractory vasoplegia, methylene blue (1 mg/kg *iv*) was administered, which rapidly re-established an acceptable mean arterial pressure. The noradrenaline and vasopressin requirements were weaned to 3 μg/min and 0.05 IU/min

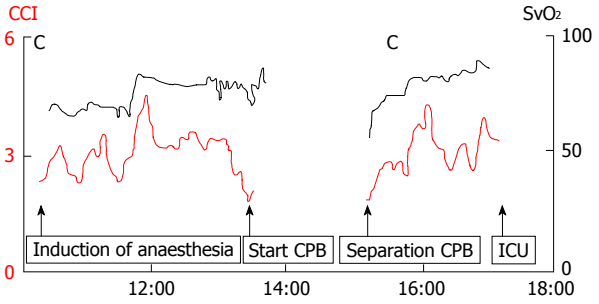


Figure 3 Intraoperative continuous cardiac index and mixed venous oxygenations tracings measured from the pulmonary artery catheter displayed on a Vigilance™ Monitor (Edwards Lifesciences, Irvine CA). ICU: Intensive care unit; CPB: Cardiopulmonary bypass; CCI: Continuous cardiac index.

respectively. A 25 mm Mitroflow® aortic pericardial valve (Sorin, Milan, Italy) was inserted without complication.

Prior to separation from CPB, a rewarming heparinase TEG was performed (Table 1, Figure 5). Desmopressin acetate (0.3 mcg/kg *iv* over 20 min) was administered to increase the plasma levels of factor Ⅷ and von Willebrand factor to minimize post-operative blood loss. Based on the rewarming TEG (Table 2 and Figure 5), fresh frozen plasma (15 mg/kg) was added to the CPB circuit to avoid volume overload and right ventricular distension during bypass separation. Glyceryl trinitrate (5 μg/min *iv*), and frusemide (20 mg *iv*) were administered to further reduce right ventricular preload and hepatic congestion.

After successful separation from CPB, haemostatic management focused on minimizing intraoperative bleeding and maintaining normothermia. Protamine (500 mg *iv*) was given to reverse the effects of heparin and correct activated clotting time to baseline values, followed by two bags of pooled platelets and *iv* administration of concentrated fibrinogen (4 g *iv*) (Riastap®, *iv* Behring, Australia). The dose of fibrinogen was calculated according to the patient's body weight (68 kg) and his estimated blood volume (4.8 L). Based on a preoperative haemoglobin of 9.8 g/L, a haematocrit of 30% of plasma volume (3.4 L), and a preoperative fibrinogen level of 2.5 g/L, we calculated that a dose of 4 g of fibrinogen would be needed to increase plasma fibrinogen levels by an estimated 1.2 g/L. With haemodilution on bypass, we expected the fibrinogen to fall by approximately 1-1.5 g/L. Following administration of concentrated fibrinogen, a heparinase TEG revealed significant prolongation of the R-time, confirming an underlying coagulopathy (Figure 5). Human prothrombin complex® (500 IU *iv* bolus, then 100 IU/h *iv* infusion) (CSL Behring, Australia) was administered, which corrected the R-time and improved haemostasis (Figure 5). Calcium chloride (1-2 g *iv*) was also administered. The total CPB time was 141 min and aortic cross-clamp time 61 min. Temporary epicardial pacing wires were not used to avoid the small risk of cardiac bleeding on wire removal additional. A topical haemostatic matrix (FLOSEAL™, Baxter, Pty) was used to control bleeding

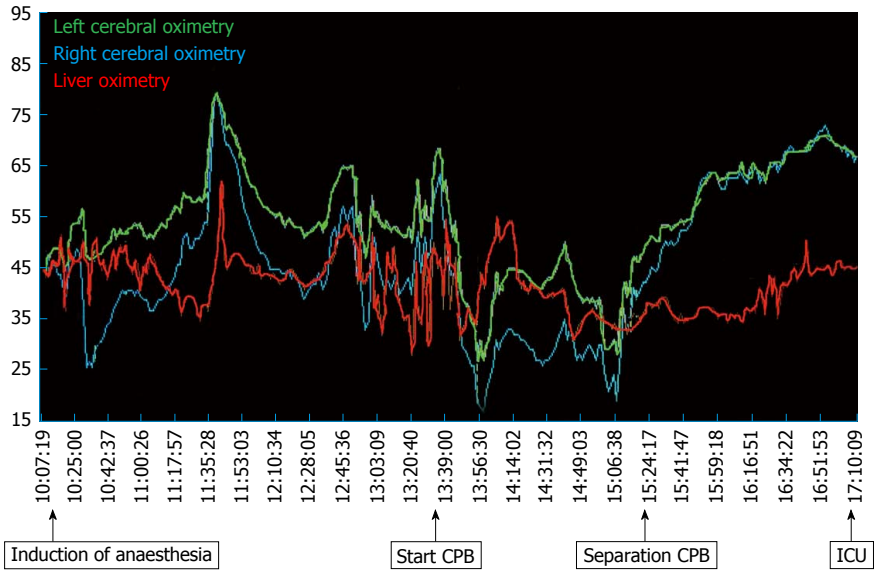


Figure 4 Cerebral and hepatic tissue oxygenation tracings measured with a cerebral/somatic oximeter (Invos Somanetics®) throughout the surgery and during cardiopulmonary bypass. ICU: Intensive care unit; CPB: Cardiopulmonary bypass.

from the suture lines, and thrombin dried powder (GELFOAM®, Baxter, Pty) was applied to the bone marrow of the sternum, which allowed sternal closure with minimal bleeding. Mediastinal and bilateral pleural drains were placed so that volume losses could be measured in ICU, and collection of blood around the heart avoided during the postoperative period.

In ICU, the octreotide (25 mcg/h *iv*) and prothrombinex (100 IU/h *iv*) infusions were continued for 8 h. Haemodynamic stability was maintained and the noradrenaline and vasopressin infusions weaned after 6 h, and the patient was extubated. Terlipressin (1 mg *iv*) was continued every 6 h for a further 24 h. The patient was transferred to the ward the following day, and discharged home ten days later without complications. There were no further requirements for coagulation or blood product intervention. Postoperatively, renal, haematological and liver function tests remained stable, and are summarised in Tables 1-3.

Three months post discharge, and at the time of writing, the patient continues to make satisfactory cardiac progress and is currently awaiting liver transplantation.

DISCUSSION

This case illustrates that redo-sternotomy and aortic valve AVR in the setting of advanced liver disease is feasible but requires careful planning. The central management decisions for such cases include to either (1) replace the valve first and then proceed with liver transplantation at a later date; (2) offer a combined procedure, *i.e.*, AVR and liver transplantation simultaneously; and (3) proceed with liver transplantation first, and then replace the valve at a later stage. In the case described here, after extensive multidisciplinary discussion a consensus was reached that the risks of liver transplantation in the setting of uncorrected symptomatic severe aortic stenosis were prohibitive. In view of the bicuspid valve and dilated ascending aorta, a transcatheter AVR was not a consideration. A combined AVR and liver transplant was considered but there were concerns that there may be further cardiac decompensation during the waiting period. Given that the patient was progressively symptomatic, a redo-sternotomy and AVR was considered to afford the best chance of survival, with activation for liver transplantation initiated at a later stage if the outcome was successful.

As shown in this case, redo cardiac surgery provides several technical challenges that distinguish it from primary cardiac surgery. These obstacles include repeat sternotomy, injury to the heart during dissection, quality and availability of conduits if required, a calcified ascending aorta, and more-advanced coronary disease involving the native vessels. As a result, operative mortality in most reoperations is 3 to 5 times that for a primary AVR. Adding in the ensuing complications of advanced liver disease, perioperative mortality increases with an estimated perioperative risk of mortality of 50%[1]. Each patient's condition and presentation is unique, and thus requires individualized management delivered by a multidisciplinary team. Consideration must be given to the sequence of procedures, cardiac surgical technique, and management of anticoagulation.

Combined cardiac and liver transplantation was first reported by Starzl *et al*[2] in 1984, but has remained uncommon because of the unique medical and surgical challenges it poses . In two descriptive reports of outcomes in patients with advanced liver cirrhosis undergoing cardiac surgery[3,4], hepatic decompensation, respiratory and renal failure, gastrointestinal haemorrhagic events, sepsis and mediastinitis were among the most common postoperative complications. The association of MELD scores and Child-Turcotte-Pugh classification, and adverse outcomes is less clear. In the study by Filsoufi *et al*[4], mortality rate increased significantly according to the

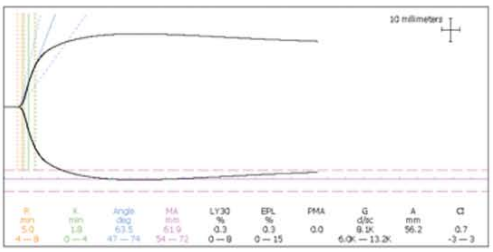
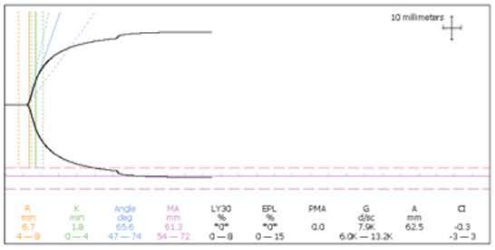
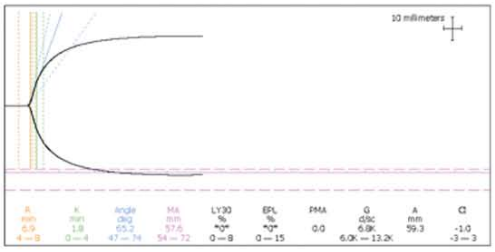
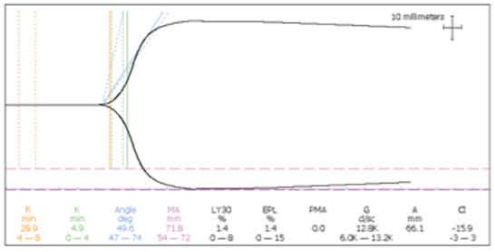
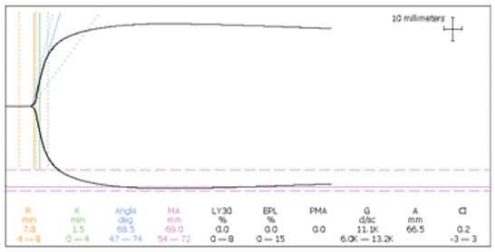
Pre-induction of anaesthesia	(1) Pre-med: morphine intramuscular 15 mg (2) 3 L central venous catheter, continuous pulmonary artery catheter, 8-f rapid infuser catheter peripheral access (3) Tranexamic acid *iv*: 1 g load then 500 g/h infusion to stop at surgical closure (4) Octreotide *iv* 100 mg bolus intravenous, followed after 2 h by a continuous infusion of 25 mg/h for 12 h (5) Terlipressin *iv* 1-2 mg administered every 6 h for 12 (6) Vancomycin *iv* 1 g/ceftriaxone 1 g *iv* antibiotic prophylaxis	
During CPB	(1) TOE guidance of venous lines to ensure excellent drainage and minimal hepatic congestion (2) Pulsatile flow (3) Haemofiltration (4) Noradrenalin commenced for vasoplegia (5) Vasopressin *iv* 0.1-0.4 IU/min commenced for vasoplegia (6) Methylene blue *iv* 1.5 mg/kg administered for refractory vasoplegia unresponsive to above	
Rewarming	(1) DDAVP *iv*: 0.3 μg/kg over 20 min (2) 4 unit FFP directly into the CPB circuit ensuring ACT > 600 s haemofiltering and removing fluid to minimise increasing portal pressures and unnecessary volume load when separating from CPB (3) Glyceryltrinitrate *iv* 5 μg/min to reduce preload to right heart pressures and minimise hepatic congestion (4) Calcium chloride 1-2 g	
After separation from CPB	(1) Protamine *iv* 500 mg, guided by ACT (2) Concentrated fibrinogen: 4 g (3) Prothrombinex *iv* 500 IU load followed by 100 IU/h infusion (4) Platelets: 2 pooled bags (10 units) (5) Frusemide *iv* 20 mg	
Prior to transfer to ICU	(1) Octeotide *iv* 25 mg/h for 6-12 h (2) Terlipressin *iv* 1-2 mg 4-h for 12-24 h (3) Prothrombinex at 100 IU/h for 6-12 h If bleeding still refractory: consideration of: (1) ± Cryoprecipitate 1 bag per 10 kg body weight (2) ± Recombinant activated factor Ⅶ (45-90 μg/kg) if ongoing haemostatic failure observed (3) Additional products based on clinical picture, TEG and laboratory coagulation results	

Figure 5 Perioperative thromboelastrography tracings observed in this case with corresponding haemostatic management action or planned strategy. DDAVP: Desmopressin acetate; FFP: Fresh frozen plasma; CPB: Cardiopulmonary bypass; TEG: Thromboelastography; TOE: Transoesophageal echocardiography; *IV*: Intravenous; ACT: Activated clotting time.

Child-Turcotte-Pugh classification (class A, 10%; B, 18%; and C, 67%). The reported mortality of redo cardiac surgery was approximately 50%[3]. Similarly, the rate of complications was higher in class B (50%) and C (100%) compared to class A (20%). Suman *et al*[5] reported that a cutoff Child-Pugh score > 7 had a sensitivity and specificity of 86% and 92% for mortality, although there was no association between mortality and MELD scores. In contrast, Morimoto *et al*[3] reported that Child-Pugh class score did not correlate with hospital mortality, although MELD score was significantly higher in patients who died immediately post cardiac surgery. To date, there have been several reports of combined AVR and liver transplantation[6]. Postoperative outcomes are variable; the majority of cases have been successful however, mortality due to clotting disturbances has also been reported. As a result, careful preoperative preparation must be conducted in such highly complex cases to prevent catastrophic

outcomes.

As seen in this case, a common yet serious complication of CPB is vasoplegic syndrome, a post-perfusion syndrome characterised by low systemic vascular resistance, significant hypotension, and a high cardiac output. It has an incidence of 5%-25% and a mortality rate as high as 25%[7]. In this case, we used the standard first line vasoactive treatment (noradrenaline and vasopressin) to maintain a mean arterial pressure of 50 mmHg[8]. However methylene blue was required during CPB to reduce the severity of vasoplegia. Use of methylene blue had been used effectively for the treatment of refractory vasoplegia in two randomised control trials[9,10], acting through its inhibitory effect on cyclic guanosine 3',5'-monophosphate-mediated vasodilatation. Despite restoring vascular tone intraoperatively, discontinuation of vasopressin has been associated with postoperative refractory vasoplegia, therefore in the case described here, vasopressin was for continued for 6 h postoperatively.

AVR performed in the context of advanced liver disease added an additional layer of complexity in preventing further decline of hepatic and renal function. The patient's history of significant portal hypertension justified the use of both terlipressin and octreotide to prevent variceal beeding[11-13]. Terlipressin has also been shown to improve hepatorenal syndrome, thought to be due to arteriolar vasoconstriction in the splanchnic area, which was an important consideration for the patient's underlying IgA nephropathy. Its effects are mediated *via* V1 receptors on vascular smooth muscle[13]. In animal models, octreotide has been shown to improve hepatic ischaemia-reperfusion injury by down-regulating inflammatory cytokines (tumor necrosis factor alpha and interleukin-1 beta) and inhibition of hepatocellular apoptosis[14], and in this case, served an added benefit when separating from bypass.

Coagulopathy is a frequent occurrence during CPB and is due to a number of factors including excessive fibrinolysis, platelet dysfunction, coagulation factor consumption, and coagulation factor dilution from intravascular volume replacement. We used a variety of multimodal pharmacological agents to prevent intraoperative and postoperative bleeding. Hypofibrinogenemia is common in cardiac surgery, which was minimized preoperatively with tranexamic acid, and intraoperatively with concentrated fibrinogen[15,16]. Desmopressin acetate[17,18], Human Prothrombin-X complex[19] and protamine were also implemented as described previously.

In this case, we employed several haemostatic and haemodynamic monitoring methods to guide our management. TEG, commonly used in cardiac surgery, is a useful tool in denoting a patient's clotting profile at landmark time points to influence specific pharmacologic decisions[20,21]. Figure 5 summarizes consecutive TEG readings and the subsequent interventions undertaken. It should also be noted that TEG requires trained personnel to operate and therefore poses as a limiting factor for its use[20]. Additional haemodynamic monitoring included pulmonary artery catheter sampling of mixed venous blood (SvO_2) and tissue oximetry. Intraoperatively, we were primarily concerned about the key factors that influence oxygen delivery, namely haemoglobin, oxygenation, and cardiac output. Continuous liver, cerebral and mixed venous oximetry enabled immediate detection of adverse changes, prompting correction and subsequent visualization of improvements of haemodynamic trends. Continuous recordings of cardiac output and global oxygenation status are presented in Figure 3. The brain and liver tissue oxygenation tracings are shown in Figure 4. In the context of low liver oximetry, in addition to the aforementioned factors that influence tissue oxygenation, hepatic congestion secondary to the outflow obstruction was also carefully monitored. Then, depending on the determined underlying cause, suitable corrections were made in the form of red cell transfusion, adjustment pump flow rates, and ensuring adequate venous drainage at all times. Although hepatic oximetry is predominantly used in the paediatric setting[22,23], we justified its use to intensively monitor the already compromised liver, and guide therapy as above. Interestingly, the liver oximeter tracing tracked the cerebral oximeter tracing very accurately (Figure 4), providing reassurance of continual intact hepatic perfusion.

In conclusion, we report a case of AVR in a patient with advanced liver disease. Given the limited information available for specific management and prevention of haemostatic failure, this report serves to guide future cases of a similar kind.

COMMENTS

Case characteristics

A 46-year-old male with a history of chronic liver disease secondary to alcohol abuse, presents with acute pulmonary oedema secondary to left ventricular failure.

Clinical diagnosis

Severe aortic stenosis.

Differential diagnosis

Non cardiogenic causes of pulmonary oedema include pulmonary contusion, acute respiratory distress syndrome, transfusion-related acute lung injury, aspiration, hypertensive crisis, upper airway obstruction, and neurogenic causes (seizures, intracranial haemorrhage).

Laboratory diagnosis

Plasma creatinine 110 μmol/L; albumin 31 g/L; bilirubin 40 μmol/L; haemoglobin 90 g/L; prothrombin time 1.3 s; platelets 76 ($\times 10^9$).

Imaging diagnosis

Transthoracic echocardiogram a severely calcified bicuspid valve, with an aortic valve area of 0.7 cm^2; mean aortic valve pressure gradient of 60 mmHg with moderate pulmonary hypertension.

Treatment

The patients underwent redo-aortic valve replacement requiring aggressive haemostatic therapy for coagulopathy and refractory vasoplegia.

Related reports

Combined cardiac surgery in patients with advanced liver disease.

Experiences and lessons

Cardiac surgery in patients with advanced liver disease is associated with significant morbidity and mortality. Refractory coagulopathy is common and requires a proactive multidisciplinary haemostatic management strategy.

Peer review

Weinberg *et al* present an interesting and complex case report of a patient with critical aortic stenosis and advanced liver disease who underwent redo-

sternotomy and aortic valve replacement prior to being listed for orthotopic liver transplantation.

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