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Emergency Valve Replacement Under Minimal Cardiopulmonary Bypass for a Patient With Infective Endocarditis and Large Brain Hematoma: A Case Report

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A 19-year-old man with mitral valve endocarditis and prolapse, intracerebral and intracerebellar hematoma, and a mycotic cerebral aneurysm underwent emergency mitral valve replacement during minimal cardiopulmonary bypass (total priming volume, 800 mL; autologous retropriming, activated clotting time <300 seconds) 1 day after undergoing endovascular coil embolization of the aneurysm. Postoperatively, there were no extensions of the intracerebral and intracerebellar hematoma. After intensive rehabilitation therapy, the patient recovered fully except for residual bilateral claudication because of preoperative bilateral embolism to both superficial femoral arteries. (A&A Practice. 2018;10:144–7.)

The rate of cerebrovascular accident with mitral valve endocarditis is reported to be at 35.5%. Between 10% and 40 % of left-sided infective endocarditis is accompanied with neurological dysfunction.[1] Literature concludes that rapid intervention with early valve surgery, even within 72 hours of the intracranial hemorrhage (ICH), is neither associated with increased mortality nor associated with higher neurological morbidity, regardless of the size of the hemorrhagic stroke preoperatively.[2]

To our knowledge, this is the first report of using minimal cardiopulmonary bypass (MCPB) for valve surgery in infective endocarditis patients with ICH. MCPB allows lower priming volume compared to conventional cardiopulmonary bypass (CCPB) and allows a decrease in heparin dose: 150–200 IU/kg for MCPB vs 300 IU/kg for CCPB.[3]

Written consent was obtained from the patient before publication.

CASE DESCRIPTION

A 19-year-old man, 48 kg, 172 cm, with a background of mild asthma, was scratched by a cat and developed fever with deep venous thrombosis in his left leg. He was treated with 2 mg fondaparinux subcutaneously twice daily for 7 days and then 5 mg apixaban orally twice daily. He consulted his family physician for macroscopic hematuria, leg edema, asthenia, and persistent fever. In hospital, the patient was lethargic with a Glasgow Coma Scale of 15/15, blood pressure of 100/ 66 mm Hg, and sinus tachycardia at 120 beats per minute with a mitral murmur. Hemocultures were taken, and piperacillin was started and then changed to ceftriaxone/gentamicin after blood cultures revealed *Haemophilus parainfluenzae*. Hemoglobin (Hb) was 6.3 g/dL, and 4 units of red blood cells were transfused.

Anterograde amnesia prompted a cerebral computed tomography and nuclear magnetic resonance scan revealing a large hematoma in the left occipitoparietal region (46 mm × 28 mm) with multiple bilateral microhemorrhage, a hematoma next to the right cerebellum and the right occipital region. Multiple ischemic microlesions were present with a left cerebellar ischemic stroke. Further, an intracerebral mycotic aneurysm of the left posterior cerebral artery, responsible for the left occipitoparietal ICH, was embolized (Figures 1–3) without complication.

Transthoracic echocardiography revealed a large vegetation of 20 mm with 2 mobile echo dense masses (>15 mm) on the mitral valve with significant mitral valve prolapse. After admission, the patient went into septic shock. He was intubated and sedated, and norepinephrine and dobutamine were started. After a wake-up test, the patient was obeying commands and did not reveal any neurological deficits.

Two days after admission, emergency surgery was performed with minimal cardiopulmonary bypass (MCBP). The patient had an in situ right radial arterial line and a left internal jugular central venous line. He was already intubated, ventilated, and sedated with propofol and sufentanil infusions and hemodynamically supported with norepinephrine infusion. Tranexamic acid 1 g was given intravenously followed by an infusion of 10 mg/kg/h.

MCPB, equipped with a centrifugal pump and a whole circuit tubing system containing a phosphorylcholine inert surface, was primed with 200 mL of lactated Ringer's solution.

To keep total priming volume (800 mL) and heparin use as low as possible, a retrograde priming technique with the patient's own blood via venous and arterial cannulae was utilized. During MCPB, Hb dropped to 7.8 (hematocrit, 23%),

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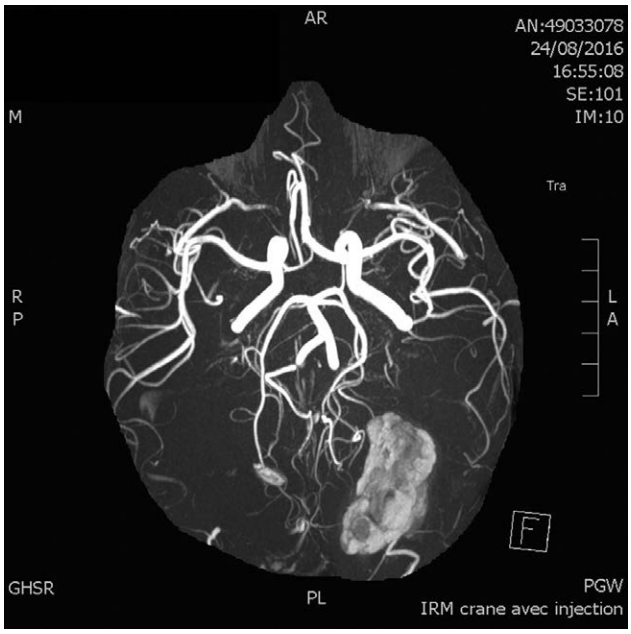

Figure 1. Magnetic resonance (MR) imaging. Technique: angio-MR 3-dimensional time of flight of the polygon of Willis. A large hematoma (46 mm × 28 mm) is located in the left occipitoparietal region.

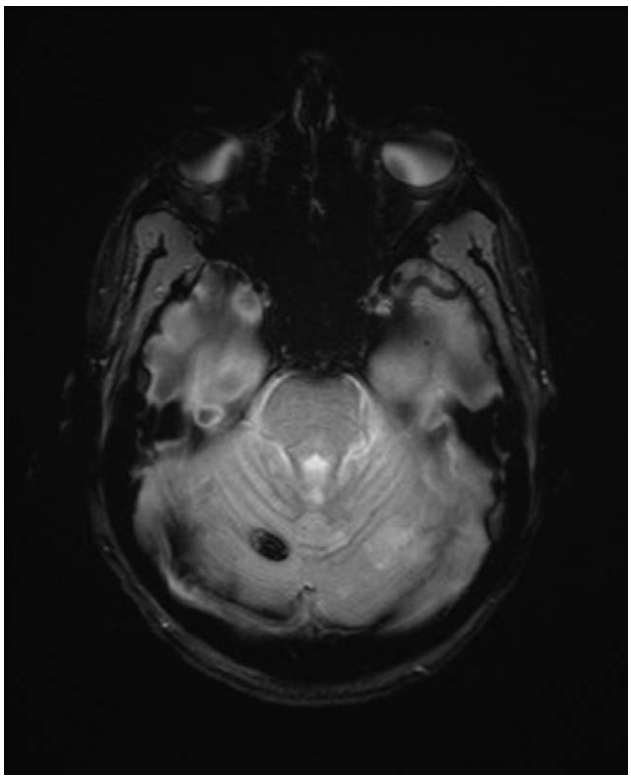

Figure 2. Magnetic resonance imaging. Technique: axial T2 gradient echo at the level of the central pons with a hematoma located in the right cerebellum.

and 2 units of red blood cells were given to obtain an Hb of 8.5–8.8 g/dL (hematocrit, 25%–26%). Mean arterial blood pressure (MAP) was maintained above 70 mm Hg (range, 60–93 mm Hg) with norepinephrine (peak infusion rate, 1 mg/h). Anterograde and retrograde cold (20°C) cardioplegia was

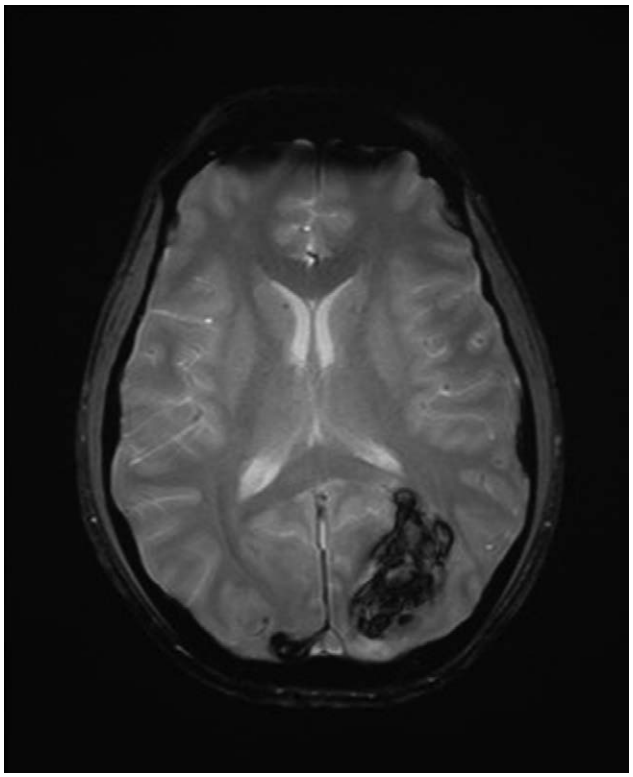

Figure 3. Magnetic resonance imaging. Technique: axial T2 gradient echo at the level of the splenium of the corpus callosum. A large hematoma is located in the left occipitoparietal region, measuring 46 mm × 28 mm.

administered every 15 minutes. Central body temperature, measured in the bladder, was cooled down to 33°C, and at the end of cardiopulmonary bypass (CPB), temperature increased to 36.8°C. Transesophageal echocardiography confirmed the presence of large vegetations and damage of the mitral annulus (Figure 4). Surgical time was kept to a minimum by proceeding directly to replacing the entire valve by a biological mitral valve. Nonin cerebral oximeter (Nonin Medical Inc, Plymouth, MN) for cerebral perfusion monitoring displayed values for tissue oxygen saturation of both hemispheres above 60 with its lowest value at 53 in the right hemisphere at the end of surgery.

Activated clotting time (ACT) before heparin administration was 133 seconds. In total, 10,000 units of heparin were given. After a peak of 401 seconds, on controls, every 30 min, ACT remained at 283 and 289 seconds. After MCBP, based on a homeostasis management system, 100 mg protamine was given and ACT normalized at 126 seconds.

MCBP time was 74 minutes, aortic clamping time 56 minutes and MCBP weaning lasted for 18 minutes. Atrioventricular pacemaker stimulation was temporarily used at a rate of 90/min.

Four units of fresh frozen plasma and 1 unit of platelets were transfused.

Postoperatively, the patient was transferred to the intensive care unit with norepinephrine at 0.032 mg/h. The 18-day intensive care unit stay were characterized by a reoperation to remove thromboemboli from the left popliteal artery, ventilation-acquired pneumonia followed by tracheostomy,

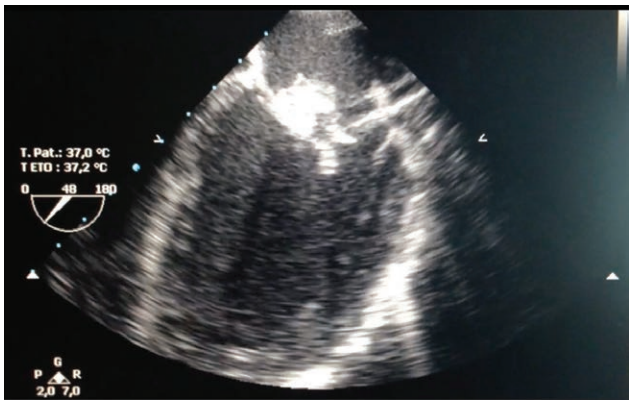

Figure 4. Perioperative transesophageal echocardiography. Midesophageal 2-chamber view at 40° showing a large vegetation with partial damage of the posterior mitral annulus.

and percutaneous gastric feeding. After 4 weeks in rehabilitation medicine, he had good neurological recovery, no loss in memory capacity, no signs of neurological deficits, and was discharged home. Due to bilateral emboli into the legs, the patient kept residual bilateral claudication. On follow-up, a control nuclear magnetic resonance showed resorption of the occipital and left cerebellar hematoma.

DISCUSSION

Low postoperative incidence of hemorrhagic stroke, even in case of decompressive craniotomy, is a strong argument to surgically remove the embolic source rapidly and thereby minimizing the risk of secondary hemorrhage.[4] Given the serious state of our patient, rapid surgical intervention under CPB with heparin was justified even 1 day after hospital admission.

Before cardiac surgery, the patient underwent coil embolization of the mycotic aneurysm. Lin et al[5] reported that early clipping or endovascular treatment with antibiotic therapy of a ruptured mycotic cerebral aneurysm can reduce mortality by up to 11%, decreasing the risk of ICH during CPB.

In a case of a 12-year-old boy with ICH secondary to infective endocarditis and aortic regurgitation undergoing aortic root replacement 5 days after his hemorrhagic stroke, a priming volume of 1000 mL was used.[6] In our case, priming was 800 mL for a young adult. Growing evidence points toward favorable neurological outcome and attenuation of postoperative neurocognitive impairment with MCPB after coronary artery bypass grafting.[7] In contrast to CCPB, the use of MCPB reduces hemolysis, hemodilution, and blood loss for coronary arterial bypass surgery.[8] However, consequences of air entering venous lines, thereby stopping the pump or inducing embolization requires the absence of atrial septal defect for MCPB use.[9] To reduce dilution of coagulation factors, we also applied retrograde priming via the venous cannula of the MCPB circuit by using the patient's own blood, which is proven to be a safe method.[10] As anemia constitutes a risk factor for stroke, we aimed our Hb level >8.5 g/dL and even a Hb level >9.2 g/dL has been recommended.[11]

An ACT of 250 seconds during MCPB was described in the literature to reduce blood transfusions, and based on experience, we considered an ACT level of 300 seconds sufficient for MCPB with a priming volume as low as 800 mL.[12] We used phosphorylcholine-coated CPB tubings to reduce systemic dose of heparin during CPB, but other CPB tubings also exist such as tubings coated with heparin and nafamostat mesilate.[13]

Currently, literature does not provide any recommendations on MAP in patients with ICH during CPB. In case of vasospasm, a higher MAP should be targeted.[5] Comparing pump MAP of 80–100 mm Hg and 50–60 mm Hg, literature describes a significantly higher incidence of neurologic morbidity in the low MAP group, and more infarcts are reported with 10 mm Hg decrease in MAP from preoperative baseline during CPB.[14] In absence of further evidence, we suggest that the average preoperative MAP of the awake patient could serve as a reference point for a MAP used during CPB.

In a case report using hypothermic CPB of 21°C for infective endocarditis valve surgery with ICH, MAP was maintained at 75–90 mm Hg.[4] However, although hypothermic CPB could contribute to coagulopathy increasing ICH, it would also reduce or eliminate the use of systemic heparin for CPB.[15] Considering European resuscitation guidelines 2015, deep hypothermia during surgery should be avoided in patients with ICH.[16]

CONCLUSIONS

Threat of reembolization justifies rapid surgical intervention under CPB with heparin even within 72 hours of the event of ICH. Preoperative embolization of ruptured mycotic cerebral aneurysm reduces mortality and the risk of ICH during CPB. MCBP with retrograde blood priming allows decrease of heparin dose, reduces blood loss, and favors neurological outcome and neurocognitive function in patients with ICH. Hb levels >92 g/dL should be aimed for with a MAP > 75 mm Hg with preferably normothermic CPB to reduce the occurrence of intraoperative stroke.

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DISCLOSURES

Name: Gabor Kiss, MD.
Contribution: This author helped search the literature, and prepare and edit the manuscript.
Name: Eric Braunberger, MD.
Contribution: This author helped edit the manuscript.
This manuscript was handled by: Raymond C. Roy, MD.

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