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Case Report

Cardiopulmonary Bypass and Malaria Relapse

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CASES OF MALARIA have been described after surgery.[1,2] This subject is highly important both in endemic areas and globally with the increasing number of travelers. The pathophysiology of malaria is complex and still not entirely understood. The occurrence of malaria after cardiac surgery is not exceptional, but the literature on this subject is scarce.[3] Blood transfusions have been described as being the cause in cases reports,[4] but in most cases, the etiology remains unknown. The authors present a case of malaria after cardiac surgery, including a brief review of the literature and some hypotheses for the pathophysiologic mechanism.

Case Report

A 17-year-old female patient was admitted for surgical repair of mitral valvular insufficiency. She had been transferred from Burkina-Faso to France by the humanitarian association Mécénat Chirurgie Cardiaque, which financially supports patients from developing countries in undergoing cardiac surgery. This patient had dilated cardiomyopathy (end-diastolic left ventricular diameter = 66 mm) associated with a grade IV mitral insufficiency (regurgitant volume = 130 mL, regurgitant orifice area = 73 mm^2), preserved left ventricular function, tricuspid grade III insufficiency with right ventricular hypertrophy, and pulmonary arterial hypertension (PAH) (systolic pulmonary artery pressure = 110 mmHg at right-sided cardiac catheterization). These lesions were the late consequence of an acute articular rheumatism. Preoperative biology showed no anemia and no other pathology except chronic hepatitis C with a very low viral load (2.34 log IU/mL).

Mitral valvuloplasty and tricuspid annuloplasty were performed in 151 minutes under normothermic cardiopulmonary bypass (CPB) with a cold blood cardioplegia. A hollow fiber membrane oxygenator (QUADROX-I, Maquet Gentinge Group, Orléans, France), a heparin-coated circuit with a roller pump, and intraoperative blood salvage were used. Two red blood cell concentrates (RBCs) were transfused for the priming of the CPB. Perioperative hemodynamic monitoring was performed by a pulmonary artery catheter with continuous cardiac output monitoring (Edwards Lifesciences, Guyancourt, France) inserted into the right internal jugular vein and a radial arterial catheter. Hemofiltration during CPB was used to adjust blood volume rapidly. Transesophageal echocardiography performed at the end of surgery showed residual grade I-II/IV mitral valvular insufficiency, septal dyskinesia, and an altered visual left ventricular ejection fraction. Hypotension occurred at CPB weaning and required norepinephrine. An inotropic agent was added due to hypokinesia. Hemodynamic variations are presented in Table 1. Inhaled nitrite oxide (NO) was provided to attenuate PAH at weaning of CPB.

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Table 1
Hemodynamic Report

	Preoperative	Pre-CPB	Post-CPB	Postoperative (H+1)	H+12	H+20	D+1	D+2
Pulse rate (beats/min)	110	75	101	120	110	124	85	114
Arterial pressure (mmHg)	145/100 (115)	84/45 (61)	88/53 (67)	98/57 (71)	86/37 (53)	103/62 (76)	115/64 (81)	134/72 (93)
Pulmonary artery pressure (mmHg)	112/35 (68)	46/16 (27)	59/20 (31)	-/- (35)	-/-(25)	-/- (23)	-/- (31)	-/- (42)
Wedge pressure (mmHg)	20	15	11	-	-	-	-	-
Cardiac index (L/min/m^3)	3.0	2.6	2.8	2.3	2.0	1.7	2.6	3.1
NO (ppm)	0	0	5	5	5	3	3	1
Norepinephrine (μg/kg/min)	0	0	0.6	0.9	1.1	1.4	0.6	0.2
Dobutamine (μg/kg/min)	0	0	10	10	1.1	0	0	0
Volemic expansion (mL)	0	800	0	1,300*	400	100	500	850
Urinary output (mL)	-	-	730	820	575	470	380	955
Body weight (kg)	37.0	-	-	-	-	36.5	-	37.5
Temperature (°C)	-	36.9	36.5	39.0	39.8	40.4	37.3	37.5

NOTE. Artemisinin was started at H+20. Volemic expansion include isotonic crystalloids and colloids administered (not the maintenance IV fluid solution).
Abbreviations: CPB, cardiopulmonary bypass; NO, nitric oxide; -, not available.
*Integrates CPB priming and hemofiltration balance. The volume of fluids and diuresis indicated are measured since the previous summary.

Postoperatively, the patient was admitted to the intensive care unit. Dobutamine was weaned rapidly after good myocardial kinetics at echocardiography were observed. However, vasoplegia required an increase in norepinephrine to 1.5 μg/kg/min. At the same time, the patient developed a fever of 39°C that sharply rose to 40°C (Table 1). The presence of trophozoites of *Plasmodium falciparum* and *Plasmodium ovale* was observed incidentally by the biologist on the blood smear after detection of an unusually severe thrombocytopenia. In the context of a postoperative state of persistent hemodynamic shock despite adequate fluid resuscitation and vasopressors, the authors initiated treatment with intravenous artemisinin. Apyrexia was obtained promptly and the hemodynamic condition improved, allowing the weaning of norepinephrine 48 hours postoperatively.

The patient was extubated on the third postoperative day, and inhaled NO was relayed by oral sildenafil to maintain PAH control. Table 2 shows the biological evolution. The patient received 2 RBCs 4 days after the surgery. The transfusion threshold was chosen at 9.0 g/dL due to the severe cardiomyopathy. Transfer in the cardiac surgery ward was possible on the sixth postoperative day. Artemisinin was relayed by atovaquone proguanil orally after 5 days of intravenous treatment. The patient felt well and traveled back to her home country a few days later.

Discussion

In some postoperative malaria cases, transfusion is highly suspected as being responsible for infection.[4] However, in France, exclusion of international travelers from blood donation and systematic serology for all donors significantly lowers the risk.

The pathophysiology of malaria relapse after cardiac surgery was considered by Purohit et al[5] more than 10 years ago. Recently, sequestrations, inflammatory, and immunologic responses to malaria have been further explored. Relapse of malaria occurring by activation of latent forms (hypnozoites) is known to occur with *P vivax* and *P ovale* but not with *P falciparum*. These relapses can be triggered by a stimulus such as various infections or surgery.[6]

In the case presented here, parasitemia had remained below the currently accepted threshold of positivity (> 1,000 parasitized erythrocytes per mm^3).[7] The chronology and the rapid response to antimalarial treatment are strongly evocative of severe malaria.[8] In patients living in endemic areas, malaria diagnosis cannot be made with a sole blood smear. Indeed,

Table 2
Biological Evolution in the Intensive Care Unit

Time of Sampling	Preoperative	Postoperative	D+1	D+2	D+3	D+4	D+5
Leucocyte (G/L)	9.08	8.85	11.43	13.46	8.99	8.34	7.76
Hemoglobin (g/dL)	13.4	13.4	14.7	11.0	9.3	8.9	11.9
Platelet count (g/L)	223	53	88	91	66	78	100
Protein (g/L)	95	42	55	51	54	61	64
Glycemia (mmol/L)	-	6.2	6.3	7.2	6.0	5.7	5.9
Haptoglobin (g/L)	-	-	-	< 0.2	-	-	-
Parasitemia (% of parasitized erythrocytes)	-	0.2	-	-	-	-	-

NOTE. Transfusion of 2 erythrocyte concentrates is performed at D+4 due to anemia. The maintenance intravenous fluid solution was 5% dextrose hypotonic solution. Insulin infusion was not required.
Abbreviation: -, not available.

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