Anaesthesia in a patient with univentricular heart for emergency craniotomy: A case report

Sir,

Patients with single ventricle physiology pose a challenge to the anaesthesiologist for both cardiac and noncardiac surgeries. Abnormal parallel circulation associated with ventricular dysfunction, chronic hypoxia, polycythaemia, and infective endocarditis contributes to increased mortality. We report a patient with single ventricle physiology who underwent noncardiac surgery.

A 22-year-old girl (40 kg) presented to emergency department with headache, vomiting, fever, diplopia, and blurring of vision for 10 days along with dyspnoea on exertion (Grade II) for last 2 months. She was diagnosed as a case of congenital cyanotic heart disease at age of 5 years. Patient was fully conscious, oriented but had cyanosis. Ejection systolic murmur was heard in the pulmonary area, and room air saturation was 85%.

Routine investigations showed haemoglobin - 16 mg/dL, total leukocyte count - 18,000/μL and platelet count was 40,000/μL (which increased to 1.5 lakhs/μL after platelet transfusion). Electrocardiogram showed broad and bifid P wave in all limb leads and V1, q wave in L2, L3 and a VF, dominant R wave in V1, V3-V5, large equiphasic RS complex in V2, dominant S wave in V6. Cardiomegaly and scoliosis were seen on chest X-ray.

Echocardiography showed situs solitus, a single ventricle of left ventricular morphology, supramitral ring with severe mitral inflow obstruction, moderate tricuspid regurgitation, both aorta and pulmonary artery taking origin from single left ventricle, malpositioned great arteries with side by side relation and valvular pulmonary stenosis. Cardiac catheterisation showed a left-sided (inverted) outlet chamber giving origin to aorta. There were no major aorto-pulmonary collaterals. Computerised tomography and magnetic resonance imaging scan of the brain showed ring-enhancing lesion in left temporal lobe with perilesional oedema suggestive of abscess.

Emergency craniotomy and drainage for left temporal abscess was planned under general anaesthesia. She had baseline SpO_2 85%, heart rate - 72/min and blood pressure - 120/60 mmHg. Intravenous (i.v) antibiotic prophylaxis with i.v cefuroxime - 1.5 g was given. Under local anaesthesia and sedation with i.v 3 mg morphine, left radial arterial cannula, and right subclavian central line was inserted because of poor peripheral access. Induction of anaesthesia was performed with 6 mg morphine, 100 μg fentanyl and 50 mg thiopentone. 50 μg phenylephrine bolus was used to maintain blood pressure. Endotracheal intubation was performed after achieving muscle relaxation with vecuronium 4 mg. Anaesthesia was maintained with oxygen, air and isoflurane. Ventilation was maintained with tidal volume 400 ml, respiratory rate 10/min, I:E ratio 1:2. Arterial blood gas analysis during surgery showed pH - 7.44, PaO_2 - 107 mmHg, PCO_2-36 mmHg, base excess 0.6 mmol/L, haematocrit - 40% and electrolytes were within normal range. Total urine output was 200 ml during 2 h of surgery. After completion of surgery, patient was shifted to neurosurgery intensive care unit on a ventilator and was extubated uneventfully after 6 h of surgery.

Congenital heart defects are the most common birth defects. Survival to the adulthood has increased to 85% with the advancement in surgical and medical management.[1] The aim of anaesthetic management in such patients is to prevent myocardial depression, to maintain adequate saturation, to prevent the occurrence of air embolism and to prevent other complications such as thrombosis, haemorrhage, infective endocarditis, and paradoxical embolism.

The ventricular interdependence is lost in univentricular hearts, resulting in abnormal systolic and diastolic function of a single ventricle.[2-4] and is more likely to fail under the stress of surgery and anaesthesia, for which the myocardial depressant drugs should be avoided.[5] We used opioids and isoflurane, which are safe in these cardiac patients. These patients are at increased risk of desaturation due to associated pulmonary stenosis and moderately elevated tidal volume (10-15 mL/kg), low respiratory rate (10/min), reduced inspiratory time and avoidance of a rise in positive end-expiratory pressure were the strategies in this patient.[5] Nitrous oxide was avoided to prevent fall in saturation and adequate muscle relaxation prevented the rise in intracranial pressure, cerebral hypoxia and fall in cerebral blood flow.

Intermittent bolus injections of 50 μg of phenylephrine intravenously helped to maintain vascular resistance, blood pressure and thus the systemic oxygen saturation.

Chronic hypoxia results in polycythaemia thus increasing the risk of thrombosis and stroke in these patients.[6] We corrected the platelet deficit, and adequate hydration was maintained to avoid problems associated with polycythaemia. These patients are at increased risk of infective endocarditis and antibiotic prophylaxis was provided. Full care was taken to avoid air in the i.v line keeping in mind the possibility of paradoxical embolism.

The present case exemplifies how by understanding pathophysiology, anticipating possible complications and taking simple preventive measures for the same, a seemingly formidable case can be managed.

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