Journal of Pediatric Sciences

Anaesthetic management of ventricular septal defect closure under cardiopulmonary bypass in a child with recently diagnosed hepatitis A

Sathyanarayan J, Praveen Kumar BA, Shio priye,
Bhaskar B.V, Durgaprasad Reddy

Journal of Pediatric Sciences 2013;5:e186

How to cite this article:

Sathyanarayan J, Praveen Kumar BA, Shio priye, Bhaskar B.V, Durgaprasad Reddy. Anaesthetic management of ventricular septal defect closure under cardiopulmonary bypass in a child with recently diagnosed hepatitis A. Journal of Pediatric Sciences. 2013;5:e186

CASE REPORT

Anaesthetic management of ventricular septal defect closure under cardiopulmonary bypass in a child with recently diagnosed hepatitis A

Sathyanarayan J[1], Praveenkumar BA[2], Shio priye[2], Bhaskar B.V[2], Durgaprasad reddy[2]

[1]Dept of CTVS,VIMS&RC,Whitefield,Bangalore,Karnataka, India
[2]Dept of Community Medicine, PESIMSR, Kuppam, AP, India

Abstract:
Hepatitis A is highly contagious and is spread largely by the fecal-oral route, more commonly in areas of overcrowding and poor sanitation. Infection is common in children but often asymptomatic. Acute liver failure complicates acute hepatitis A in only 0.1% of cases and chronic infection does not occur. A 2-yr-old female baby was operated for Ventricular Septal Defect (VSD) closure under cardiopulmonary bypass. She was diagnosed to have acute hepatitis A while preoperative evaluation and was decided to operate once the acute phase resolves as indicated by liver enzymes touching baseline. General anesthesia was induced with morphine, atracurium and maintained with morphine, propofol and atracurium. No complications were encountered during perioperative period. We concluded that following acute hepatitis, surgery can be done on cardiopulmonary bypass as early as the acute phase resolves using anesthetic agents that are least hepatotoxic and metabolism of which is less affected by liver disease. Additionally insult from cardiopulmonary bypass can be minimized by reducing bypass time, non pulsatile flows and normothermia. By close follow-up of patients clinically and biochemically, it is possible to reduce the complication rates to a minimum.

Keywords: *Hepatitis A, general anaesthesia, cardiopulmonary bypass, ventricular septal defect*
Corresponding author: *Sathyanarayan. J; Department of CTVS, Vydehi Institute of Medical Sciences and Research Center, Whitefield, Bangalore - 560066, India*
Telephone : +917204222070
e-mail: sathyaforall2005@gmail.com

Introduction

Hepatitis A is caused by contaminated food or water or contact with an infected person and rarely progresses beyond the acute illness [1]. Virtually all previously healthy patients with hepatitis A recover completely from their illness with no clinical sequelae. A small proportion of patients with hepatitis A experience relapsing hepatitis weeks to months after apparent recovery from acute hepatitis. The case-fatality rate in hepatitis A is very low [2]. There are limited studies regarding how early a patient can be taken for cardiac surgery after acute hepatitis, so we report anaesthetic management of a case of ventricular septal defect (VSD) closure with recent hepatitis A taken as early as acute phase is resolved.

Case report

A 2-yr-old female baby weighting 10kgs presented with history of recurrent respiratory

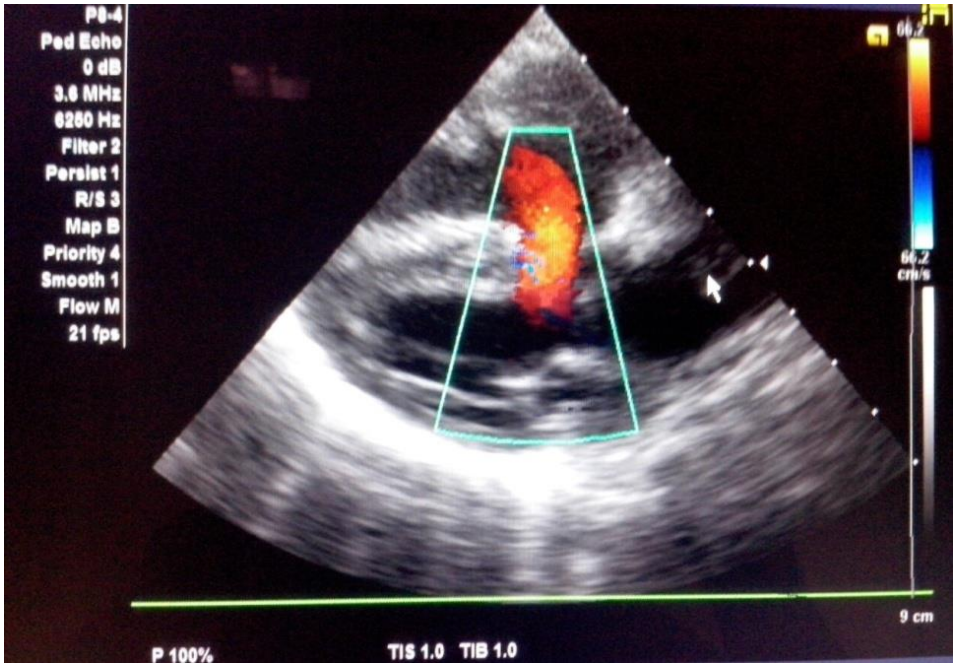

Figure 1. Echocardiography showing ventricular septal defect (VSD)

tract infection, fever and breathing difficulty since birth and clinical examination revealed icterus and pansystolic murmur. Echo-cardiography confirmed 10 mm sub-aortic ventricular septal defect (VSD) with left to right shunt and gradient of 85 mmHg (Figure 1). Liver function revealed total bilirubin (TBR): 4.0mg/dl, direct bilirubin (DBR): 2.0 mg/dl, indirect bilirubin (IBR): 2.0 mg/dl, serum aspartate amino-transferase (AST): 1166 IU/L, serum alanine aminotransferase (ALT): 1004 IU/L, serum alkaline phosphatase (ALP): 489 IU/L, prothrombin time (PT): 16.7 sec, international normalized ratio (INR): 1.08, seronegative for HBsAg, HIV and HCV. Further evaluation revealed positive for anti HAV IgM, indicating acute hepatitis A but there was no positive history. Baby was treated with syrup Hepamerz (Merz pharmaceuticals GmbH, Germany) 2.5 ml TDS and vitamin K 2 mg OD. Enzymes showed a down trend. Since baby was repeatedly getting admitted for respiratory infection, VSD closure was planned after 10 days when liver function was TBR: 0.6 mg/dl, DBR: 0.3 mg/dl, IBR: 0.3 mg/dl, AST: 58 IU/L, ALT: 63 IU/L, ALP: 282 IU/L, PT: 15.4s, INR: 0.99.

After taking informed written consent baby was shifted to operating room and monitoring was established with pulse oxymetry, electro-cardiogram, temperature, capnography, invasive blood pressure and central venous pressure monitor.

Baby was induced with intravenous inj morphine 0.2 mg/kg and inj atracurium 0.5 mg/kg and intubated with 5.0 uncuffed ET tube. Mechanical ventilation was instituted with O2 and air mixture to maintain EtCO2 of 32-34. As remifentanil is not available in India, anesthesia was maintained with inj morphine 0.2mg/kg boluses before bypass, on bypass and post bypass. Infusion of atracurium 5ug/kg/min and propofol 100ug/kg/min were maintained. Direct closure of VSD was done through right atrial approach under cardiopulmonary bypass with 2.4 L • min−1 • m−2 non pulsatile flows and normothermia. Aortic cross clamp time was 15 minutes and total cardiopulmonary bypass time 39 minutes. Hemodynamics was maintained within normal limits. Baby was shifted to ICU with inj adrenaline 0.01 mg/kg/h and inj nitroglycerin 0.5 mg/kg/h infusion. Perioperative period was uneventful.

In the ICU baby was on inj meropenam 200 mg tds, inj cefotaxime 250 mg qid, syrup hepamerz 2.5ml tds, and tramadol suppository. Baby was extubated after 6 hours. 0' postoperative day liver function was TBR: 2.0 mg/dl, DBR: 0.5 mg/dl, IBR: 1.5 mg/dl, AST: 76 IU/L, ALT: 90 IU/L, ALP: 224 IU/L, PT: 15.4s, INR: 0.99 indicating perioperative insult. But serial postoperative liver function from 1st postoperative day revealed a downtrend. Postoperative period remained uneventful. Baby was discharged from hospital after 7 days.

Discussion

Hepatitis A is caused by contaminated food or water or contact with an infected person and rarely progresses beyond the acute illness. A remote history of hepatitis A has no significance perioperatively [1].

Although hepatitis A is rarely blood borne, several outbreaks have been recognized in recipients of clotting factor concentrates. Complete clinical and biochemical recovery is to be expected 1–2 months after all cases of hepatitis A. A small proportion of patients with hepatitis A experience relapsing hepatitis weeks to months after apparent recovery from acute hepatitis. The case-fatality rate in hepatitis A and B is very low (~0.1%) but is increased by advanced age and underlying debilitating disorders. Fulminant hepatitis is primarily seen in hepatitis B and D, as well as hepatitis E, but rare fulminant cases of hepatitis A occur primarily in older adults and in persons with underlying chronic liver disease [2].

Antibodies to HAV (anti-HAV) can be detected during acute illness. This early antibody response is predominantly of the IgM class and persists for several months, rarely for 6–12 months. The diagnosis of hepatitis A is made during acute illness by demonstrating anti-HAV of the IgM class [2].

Delaying elective surgery until after an acute episode of hepatitis or an exacerbation of chronic disease has resolved or until a diagnosis is established if hepatic dysfunction is newly detected is appropriate [3]. Elective surgery is contraindicated in patients with acute or fulminant liver disease, including alcoholic (mortality >55%), viral (mortality >10%), or undefined hepatitis [1]. Consensus opinion, based largely on data derived from older, predominantly retrospective studies, is that acute hepatitis, whether viral, alcohol, or drug induced, is a risk for the development of hepatic failure or death after elective surgery [4].

The goals of perioperative management are to avoid or minimize hepatotoxic drugs, minimize cardiopulmonary bypass time and avoid hypotension or low cardiac output.

The influence of volatile anesthetics on hepatic blood flow and function is complex and related not only to features unique to the anesthetic itself but also to other patient-related variables such as the severity of underlying liver dysfunction, the presence of advanced age, and the impact of surgical stress and intra-abdominal surgical manipulation [5]. Decrease in total hepatic blood flow which occurs normally during general anesthesia, the effects of anesthetics that are toxic to the liver, decreased blood pressure during anesthesia and decrease in tissue perfusion as a result of surgery may further disrupt hepatic function.

Propofol probably has a more favorable splanchnic and hepatic oxygen delivery balance than halothane [6]. Based on limited clinical and experimental data, intravenous anesthetics have only a modest impact on hepatic blood flow and no meaningful adverse influence on postoperative liver function when arterial blood pressure is adequately maintained [5]. The pharmacokinetics of morphine is relatively unchanged by developing liver disease, such as liver cirrhosis and hepatic carcinoma, because of the substantial compensatory extra hepatic metabolism of morphine. Remifentanil is a synthetic opioid with an ester linkage that allows for rapid hydrolysis by blood and tissue esterases; such hydrolysis leads to high clearance, rapid elimination, and recovery that is

almost independent of the dose or duration of infusions [7]. Atracurium and cisatracurium are not dependent on hepatic elimination and can be used without modification of dosing in patients with end-stage liver disease [5].

Cardiothoracic surgery is associated with a high mortality rate in patients with preexisting liver disease [4]. Hepatic dysfunction is often exacerbated by cardiopulmonary bypass (CPB), although the precise mechanism of this dysfunction has not been clearly elucidated. In a canine model of hypothermic nonpulsatile CPB, Koizumi and colleagues [8] noted a decrease in PBF and HABF without a concomitant change in hepatic oxygen metabolism. Normothermic CPB was associated with a similar decline in PBF, but HABF was maintained. Increasing the dose of fentanyl from 10 to 50 μg/kg/hr significantly suppressed HABF and impaired hepatic oxygen metabolism in normothermic and hypothermic CPB, suggesting the possibility of fentanyl-mediated peripheral venous pooling and subsequent lowering of cardiac output [8].

Hepatic dysfunction following cardiopulmonary bypass (CPB) is a relatively frequent finding, and jaundice occurring after CPB is associated with an increased mortality rate. Post-CPB jaundice may be a consequence of inadequate liver perfusion during CPB. During CPB, effective hepatic blood flow was consistently reduced by an average of 19%. Although for most patients this reduction seems well tolerated, in a minority of patients it may contribute to postoperative hepatic dysfunction [9].

During cardiopulmonary bypass, hepatic blood flow is better maintained by high pump flow than by low pump flow rates. Hypothermic cardiopulmonary bypass may benefit the hepatic circulation, although the additional advantages usually gained by the use of pulsatile perfusion may be partly lost when hypothermia is combined with a high pump flow rate [10].

Mathie and et al. concluded that hepatic blood flow is well maintained by both pulsatile and nonpulsatile flow during normothermic CPB at 2.4 L • min−1 • m−2. However, a decrease in liver blood flow during hypothermic perfusion is associated more with pulsatile than with nonpulsatile flow. Their findings thus support the view that an optimum CPB protocol for preserving the hepatic circulation requires a high perfusion rate with pulsatile or nonpulsatile flow at 37° C; if hypothermia is used, a nonpulsatile protocol appears to be preferable to a pulsatile one[11].

In addition to the possible effects of hepatic artery and portal venous perfusion, other potential determinants of hepatic dysfunction after CPB include hypotension, low cardiac output syndrome, hypoxemia, micro embolism or macro embolism, cytokine and oxygen-free radical formation, and the influence of vasoactive and anesthetic drugs[5].

Conclusion

General anesthesia can be successfully achieved in an asymptomatic, pediatric case of hepatitis A virus at the earliest when liver enzymes touches the baseline using agents that are least toxic to the liver. Cardiopulmonary bypass poses additional problem, which can be reduced by reducing bypass time, nonpulsatile flows and normothermia. Preventive measures and meticulous observation and follow-up in the perioperative periods would minimize the complication rates and secure a successful outcome in such cases.

References

1. Stephen P, Fischer, Angela M, Bader, BobbieJean Sweitzer. Preoperative Evaluation. In: Miller RD, Editor. Miller's anesthesia. 7th ed. Philadelphia: Churchill Livingstone 2010, p1026-1028.
2. Jules L Dienstag. Acute Viral Hepatitis. In: T.R.Harrison, Editor. Harrison's principles of internal medicine. 17th Ed. The McGraw-Hill Companies, Inc; 2008, p1932-1939.

3. Befeler AS, Palmer DE, Hoffman M, Longo W, Solomon H, Di Bisceglie AM. The safety of intra-abdominal surgery in patients with cirrhosis: Model for End-Stage Liver Disease score is superior to Child-Turcotte-Pugh classification in predicting outcome. Arch Surg 2005; 140:650-654.
4. Ziser, Avishai, Plevak et al. Morbidity and mortality in cirrhotic patients undergoing anesthesia and surgery. Anesthesiology 1999; 90:42-53.
5. Rothenberg DM, O'Connor CJ, Tuman KJ. Anesthesia and the Hepatobiliary System in: Miller RD, Editor. Miller's anesthesia. 7th edition. Philadelphia; Churchill Livingstone 2010, p2139-2140.
6. Christiansen CL, Ahlburg P, Jakobsen C, Andresen EB, Paulsen PK. The influence of propofol and midazolam/ halothane anesthesia on hepatic and gastric mucosal pH during cardiopulmonary bypass. J Cardiothorac Vasc Anesth 1998; 12:418-421.
7. Tegeder I, Lötsch J, Geisslinger G. Pharmacokinetics of opioids in liver disease. Clin Pharmacokinet 1999;37:17-40.
8. Koizumi M, Matsumoto N, Uede K. Influences of cardiopulmonary bypass and fentanyl anesthesia on hepatic circulation and oxygen metabolism in beagles. Anesth Analg 1998; 96:1177-1187.
9. Hampton WW, Townsend MC, Schirmer WJ, Haybron DM, Fry DE. Effective hepatic blood flow during cardiopulmonary bypass. Arch Surg. 1989;124:458-9.
10. Mathie RT. Hepatic blood flow during cardiopulmonary bypass. Crit Care Med. 1993;21:S72-6.
11. Mathie RT, Ohri SK, Batten JJ, Peters AM, Keogh BE. Hepatic blood flow during cardiopulmonary bypass operations: The effect of temperature and pulsatility. J Thorac Cardiovasc Surg 1997; 114:292-3.