Venous air embolism during orthotopic liver transplantation in a child

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Purpose: A first case of massive venous air embolism is reported as a complication of orthotopic liver transplantation in a 17-month-old child during the dissection phase.

Clinical features: During the hepatic dissection phase, perforation of suprahepatic veins was responsible for an air embolism with a decrease of $P_{ET}CO_2$ (27 to 6 mmHg), hypoxaemia (SpO_2 decreased from 100 to 88%), and haemodynamic instability (systolic blood pressure decreased from 85 to 50 mmHg, central venous pressure increased from 6 to 10 mmHg). Central venous aspiration of air was unsuccessful but immediate resolution occurred and neurological outcome was normal.

Conclusion: Venous air embolism during the dissection phase of liver transplantation in children is a risk that should be considered.

Objectif : Rapporter une première observation d'embolie aérienne survenant à la phase de dissection du foie chez un enfant de 17 mois pendant une transplantation hépatique orthoptique.

Éléments cliniques : Pendant la phase de dissection du foie, une perforation des veines sus-hépatiques a provoqué une embolie aérienne avec une chute de la $P_{ET}CO_2$ (27 à 6 mmHg), une hypoxémie (une chute de la SpO_2 de 100 à 88%), de l'instabilité hémodynamique (la pression artérielle systolique s'abaissant de 85 à 50 mmHg, la tension veineuse centrale augmentant de 6 à 10 mmHg). L'aspiration centrale d'air a été un échec mais la situation s'est rétablie immédiatement et l'évolution neurologique a été normale.

Conclusion : Pendant la phase de dissection de la transplantation hépatique, il faut tenir compte du risque d'embolie gazeuse survenant par la voie veineuse.

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Accepted for publication July 21, 1997.

VENOUS air embolism is a well-known complication of orthotopic liver transplantation in adults after unclamping of vessels.[1] This case is the first report of a venous air embolus occuring in a child during the dissection phase of liver transplantation.

Clinical report

A 17-month-old 11.2-kg-boy was admitted for liver transplantation. He had a long history of congenital biliary atresia. At the age of two months, he had undergone a Kasaï portoenterostomy which had failed. Before transplantation, he presented in end-stage cirrhosis with ascites and several episodes of bleeding from oesophageal varicose veins. Biochemical investigations revealed haemoglobin of 10 $g \cdot dl^{-1}$, factor V of % and a prothrombin time of 64%.

Prior to transplantation, arterial blood pressure was 80/45 mmHg, heart rate was 110 bpm and cardiopulmonary examination, including transthoracic echocardiography, was normal.

During surgery, heart rate (HR), arterial blood pressure (ABP), central venous pressure (CVP), pulse oximetry (SpO_2), capnography, inhaled and expired gas, and temperature were continuously assessed and recorded on a computer (Idacare Hermès Systems®). After administration of 0.2 mg atropine, anaesthesia was induced with 100 mg thiopentone, 20 mg fentanyl followed by 2 mg pancuronium. The trachea was intubated and the lungs were mechanically ventilated with a mixture of 50% air/oxygen without N_2O: PEEP was not used. Two large peripheric venous catheters (20 G) connected to a rapid infusion system were inserted, a central venous catheter was placed in the right internal jugular vein (18 G bi-lumen), and a radial artery catheter (22 G) was used to monitor arterial pressure continuously and to withdraw blood samples. Correct positioning of the endotracheal tube and the central venous catheter (at the junction of the right atrium and the superior vena cava) were confirmed by a chest radiograph prior to incision. Intraoperative blood salvage was started from the beginning of the operation. Anaesthesia was maintained with continuous infusions of midazolam (0.8 $mg \cdot hr^{-1}$) and fentanyl (0.2 $mg \cdot hr^{-1}$) and muscle relaxation was obtained by an infusion of pancuronium (0.4 $mg \cdot hr^{-1}$).

The dissection phase was long and difficult because of multiple adhesions. Fresh frozen plasma was infused at the rate of 50 $ml \cdot hr^{-1}$ to maintain central filling pressures. The blood losses necessitated both autologous transfusion and continuous infusion of aprotinin (1000 $UI \cdot kg^{-1} \cdot hr^{-1}$). However the haemodynamic status was stable during this phase and CVP was low. The rapid infusion system was not connected to the patient during the first stage of the transplantation. Suddenly, the surgeon informed the anaesthesia team of accidental perforation of the suprahepatic veins but with no evidence of bleeding. Immediatly, $P_{ET}CO_2$ decreased from 27 to 6 mmHg; SpO_2 decreased from 100 to 88%, systolic ABP from 85 to 50 mmHg, CVP increased from 6 to 10 mmHg and HR from 130 to 155 bpm (Figure). Cardiopulmonary ressucitation was not necessary. During this period, arterial blood was not withdrawn. The FiO_2 was increased to 1.0 and the patient was placed in the Trendelenburg position. Attempts to aspirate air from the internal jugular vein were unsuccessful. The surgeon rapidly clamped the suprahepatic veins. Approximately 20 min later, all variables had returned to normal, and the FiO_2 was decreased to 0.6 but, a moderate decrease of SpO_2 to 95% required a second increase of FiO_2 to 1.0 for a few minutes.

No further problems were encountered even after unclamping. Neurological outcome was normal.

Discussion

Although venous air embolism is commonly cited as a complication of liver transplantation,[1] there are very few case reports in adults,[2-4] and none in chidren. All those reported occurred at the unclamping of vessels, but not during the hepatic dissection phase. The present case was well documented by computerized monitoring (Figure).

In two of the previous cases, air embolism occured after unclamping (in one despite adequate flushing of the liver).[2,4] Venous air embolism has also been reported in relation to dysfunction of veno-venous bypass.[3] The present complication has never been described during the dissection phase in either the paediatric or adult population although dissection is known to be difficult and haemorrhagic in children with biliary atresia because of the previous surgery, e.g., Kasaï portoenterostomy. Our surgical team is experienced in hepatic surgery and has performed more than 180 liver transplantations over five years. Such an accident has never been previously encountered.

The first sign of venous air embolism was the decrease in $P_{ET}CO_2$ (Figure). Increase of physiological deadspace causes ventilation/perfusion mismatch, and subsequent hypocapnia and hypoxaemia. The degree and the duration of $P_{ET}CO_2$ decrease provides a semi-quantitative estimation of air embolism.[5] Other variables (i.e., tachycardia, hypotension, increase in CVP) are less sensitive, but attest to the severity of venous air embolism.[5] We do not routinely use transoesophageal echocardiography or precordial Doppler ultrasound to detect venous air embolism, although they are probably the most sensitive

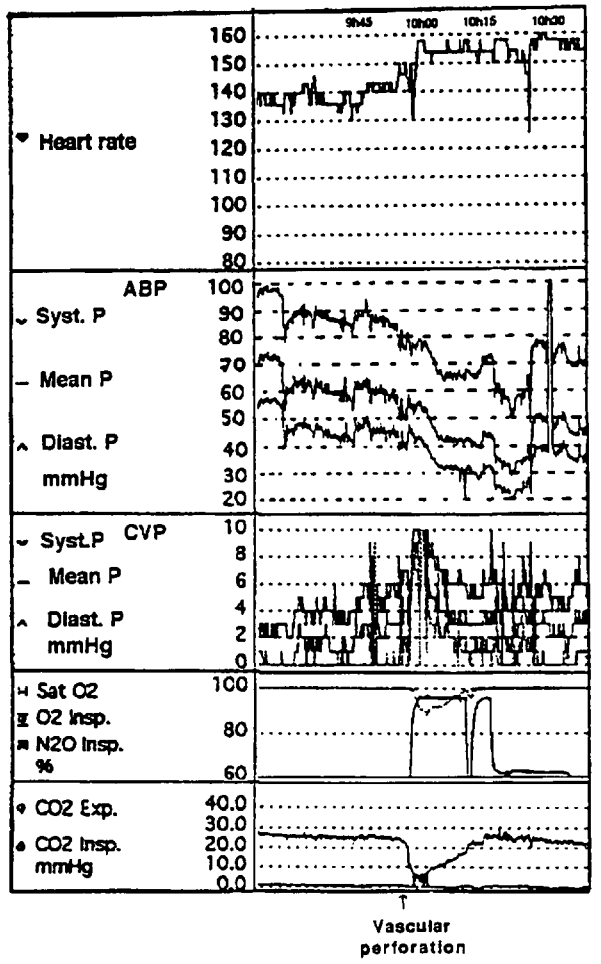

FIGURE Haemodynamic and respiratory changes during the episode of venous air embolism.

monitors.[5] In this case, transoesophageal echocardiography and the use of an oesophageal stethoscope were contraindicated due to presence of oesophageal varices and the risk of bleeding. End-tidal nitrogen was not monitored during this event although it may provide a quantitative measure of entrained venous air.[4] In our institution, pulmonary artery catheters are not commonly used in liver transplantation in children weighing < 20 kg. As with $P_{ET}CO_2$, an increase of pulmonary artery pressure has been shown to be proportional to the severity of air embolism.[6] During neurosurgery in children, some authors consider that a right atrial catheter is a useful adjunct to diagnosis and treatment of massive air embolism,[6] but others disagree.[7] Commercial anaesthesia record keepers decrease the workload of the anaesthesia team during liver transplantation. They also provide accurate and detailed information in case of complications.

The acute changes in respiratory and cardiovascular variables observed during this event very strongly suggested that air emboli was related to vascular perforation. However, other sources of venous air embolism must be discussed: dysfunction of veno-venous bypass or rapid infusion sets or tubing, and vascular unclamping. In children weighing < 20 kg, veno-venous bypass is not used by our team because of the good haemodynamic tolerance of clamping in this population.[1] A rapid infusion set was not used during the preanhepatic phase of transplantation. All the tubing was checked during the event and was free from air bubbles. No vessel was clamped before this episode. Because of muscle relaxation, there was no spontaneous breathing effort and there was no leak in the anaesthesia circuit.[4] Haemorrhagic shock may also produce acute hypocapnia but can be excluded in this case because of the absence of sudden bleeding during the episode, the increase of CVP and the immediate profond decrease of end tidal CO_2.

Treatment of massive venous air embolism includes immediate flooding of the surgical field with some fluid, air aspiration from a central venous or a pulmonary-artery catheter,[6] Trendelenburg positioning, ventilation with FiO_2 1.0 and vasopressor(s) in the presence of prolonged haemodynamic deterioration. In our case, attempts to aspirate air from the internal jugular catheter were unsuccessful.

The volume of air bubbles may be increased by the diffusion of N_2O.[3] However, during liver transplantation, our protocol does not include administration of N_2O since it increases bowel distension in the surgical field.

Previously, venous air embolism was thought to occur only after unclamping even if perfusion of the donor liver was adequate,[2,4] or after malfunction of veno-venous bypass.[3] This report describes another, previously unreported, mechanism of air embolism occuring during the dissection phase of liver transplantation. Children with biliary atresia and previous surgery may be at particular risk of this complication.

References

1 *Carton EG, Plevak DJ, Kranner PW, Rettke SR, Geiger J, Coursin DB.* Perioperative care of the liver transplant patient: part 2. Anesth Analg 1994; 78: 382–99.

2 *Kutt JL, Gelb AW.* Air embolism during liver transplantation (Letter). Can Anaesth Soc J 1984; 31: 713–5.

3 *Khoury GF, Mann ME, Porot MJ, Abdul-Rasool IH, Busuttil RW.* Air embolism associated with veno-venous bypass during orthotopic liver transplantation. Anesthesiology 1987; 67: 848–51.

4 *Prager MC, Gregory GA, Ascher NL, Roberts JP.* Massive venous air embolism during orthotopic liver transplantation. Anesthesiology 1990; 72: 198–200.

5 *Shapiro HM, Drummond JC.* Neurosurgical anesthesia. *In*: Miller RD (Ed.). Anesthesia, 4th ed. New York: Churchill Livingstone, 1994: 1897–946.
6 *Cucchiara RF, Bowers B.* Air embolism in chidren undergoing suboccipital craniotomy. Anesthesiology 1982; 57: 338–9.
7 *Marshall WK, Bedford RF.* Use of a pulmonary-artery catheter for detection and treatment of venous air embolism: a prospective study in man. Anesthesiology 1980; 52: 131–4.