Effect of high central venous pressure on the incidence of air embolism in patients receiving posterior lumber interbody fusion

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Received November 9, 2015; Accepted April 5, 2016; Epub May 15, 2016; Published May 30, 2016

Abstract: Background: Venous air embolism (VAE) can lead to severe clinical events and takes place at higher frequency in spine surgery recently. In this study, we applied transesophageal echocardiography (TEE) to observe the effect of high central venous pressure (CVP) on the incidence of air embolism in patients. Method: Eighty-two posterior lumber interbody fusion (PLIF) patients that would receive PLIF under general anesthesia were randomly divided into two groups, control group (C group, n=40) and high CVP group (HCVP group, n=42). These patients were monitored by TEE during surgery. After surgery, two cardiac anesthesiologists reviewed TEE images and assessed the grades of VAE. Results: In C group, VAE was not detected in 17.5% of the patients; grade I VAE was detected in 22.5% of patients, grade II in 37.5% of patients, grade III in 17.5% of patients, and grade IV in 5% of patients. For HCVP group, VAE was not detected in 50% of the patients; grade I VAE was detected in 23.8% of patients, grade II in 16.7% of patients, grade III in 9.5% of patients, and grade IV in none of the patients. The grade and incidence of VAE were obviously lower in HCVP group than C group (P<0.001). Conclusion: For patients receiving PLIF, maintaining HCVP can effectively reduce the incidence of VAE and therefore enhance the safety of surgery.

Keywords: Venous air embolism, central venous pressure, transesophageal, echocardiography

Introduction

Venous air embolism (VAE) is a rare but fatal surgical complication caused by the entry of air from the surgical site to the venous system. VAE has been reported in neurosurgery, bone surgery, urological surgery, laparoscopy, cesarean section and removal of central venous catheter [1, 2]. VAE during spine surgery is described in literature with high mortality [3-6], but it is mostly presented as case report rather than in prospective large-sample trial. Since VAE may be asymptomatic, undiagnosed or unreported, its incidence is hard to estimate and no preventive measures have been established yet. To gain more understanding of VAE, we observed the incidence of VAE and the effect of high central venous pressure (HCVP) on the incidence by applying transesophageal echocardiography (TEE).

Materials and method

Patient recruitment

As shown in Figure 1, 82 patients were recruited. The experiment was approved by the Ethics Committee of Qinghai University Affiliated Hospital. Informed consent was obtained from 82 patients that would receive PLIF. The patients were aged 30-50 years and classified as ASA-I-II. The history of abnormal cardiopulmonary function, congenital heart disease and gastric and esophageal disease was excluded. Using random number generator, 82 patients were divided into two groups: odd numbers for control group (C group, n=40) and even numbers for high CVP group (HCVP group, n=42). The CVP value high than 15 cmH₂O was considered HCVP. All surgeries were performed by one surgeon. VAE was monitored intraoperatively and graded postoperatively using TEE.
The patients were fasted for 8 h before surgery. Intramuscular injection of 0.1 g Luminal and 0.3 mg scopolamine was performed 30 min before surgery. Upper extremity vascular access was established in supine position. ECG, non-invasive blood pressure and \( \text{SpO}_2 \) were monitored, and radial artery puncture and catheterization were performed under local anesthesia with invasive blood pressure measurement. Anesthesia was induced by intravenous injection of sufentanil (0.3 \( \mu \text{g/kg} \)), propofol (2 \( \text{mg/kg} \)) and vecuronium bromide (0.12 \( \text{mg/kg} \)). After endotracheal intubation, right central venous cannulation was performed to monitor CVP. Anesthesia was maintained with sevoflurane (1.5-3 vol%) and remifentanil (0.1 \( \mu \text{g/kg/min} \)). Muscular relaxation was maintained by intermittent intravenous injection of vecuronium bromide (0.03 \( \text{mg/kg} \)) every 30 min. Mechanical ventilation was adopted using 50% oxygen/50% air (2 L), end tidal carbon dioxide tension (\( \text{PET-CO}_2 \)) was maintained at 35-40 mmHg. Before the patient was turned over, 5.0-MHz ultrasound transducer (SONOS 4500, Philips, Boeblingen, Germany) was inserted into the esophagus (Figure 2). After turning over, the zero point of the transducer was leveled using a U-shaped tube. For HCVP group, the point of spinous process was located, and the pressure difference between this point and the zero point was defined as the target value of CVP. The patient was infused with 0.9% lactated Ringer’s injection and 6% hydroxyethyl starch 130/0.4 (Voluven, Fresenius Kabi, Germany). Hydroxyethyl starch was intended to supplement the lost blood volume (1:1). Amount of infusion in C group = compensatory expansion of vascular volume + deficient volume + maintenance volume + lost volume + third-space deficient volume. Before the destruction of bone structure in HCVP group, 0.9% lactated Ringer’s injection was infused to achieve the target value of CVP. The amount and rate of fluid infusion were adjusted such that the target value of CVP was maintained. The position of transducer and the scan angle were adjusted for the imaging of the mid esophageal four-chamber view (Figure 3); the right atrium was observed. TEE monitoring was performed throughout the surgery, and the time of onset of VAE was recorded. When the air bubbles were detected in the right atrium (RA), the transducer was turned right; the scan angle was adjusted to observe the superior and inferior vena cava and to confirm whether the air bubbles entered from the inferior vena cava. Then the scan angle was adjusted again to observe the right ventricle (RV) and the right ventricular outflow tract (RVOT). When the air bubbles accumulated to over one half of RA, RV and RVOT, the functions of left and right ventricles were assessed from four-chamber view.
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The sample size was estimated by Power Analysis Software (Power and Sample Size Calculation, Version 3.0; Vanderbilt University, Nashville, TN). The assumptions made for the estimation were as follows: 1) the incidence of VAE (above grade II) was about 60% in patients receiving PLIF in C group (obtained by pre-experiment in 15 patients); 2) the incidence of VAE (above grade II) was about 30% in patients receiving PLIF in HCPV group; 3) α=0.05, (1-β)=0.8. Thus, according to the estimate, the sample size of each group should be no less than 40.

Statistical analyses were performed using SAS software (version 6.12, SAS Cary Academy, North Carolina, USA). The means of baseline data of patients in the two groups were compared with t-test. The means of vital signs were compared with analysis of variance. Bonferroni test was adopted for post-hoc analysis. Chi-square test and Fisher’s exact test were used for the difference in the incidence of VAE between the two groups. P<0.05 was considered as significant difference.

Results

Full data were collected from all 82 patients. The baseline data of the two groups did not show significant difference (Table 1), and the intraoperative vital signs manifested similar variations in two groups except CVP (Table 2).

As shown in Figure 4, TEE images indicated that VAE did not occur in 17.5% (7/40) of the patients in C group; grade I VAE was detected in 22.5% (9/40) of patients, grade II in 37.5% of patients, grade III in 17.5% of patients, and grade IV in 5% of patients. For HCVP group, VAE was not detected in 50% of the patients; grade I VAE was detected in 23.8% (9/40) of patients, grade II in 37.5% of patients, grade III in 17.5% of patients, and grade IV in 5% of patients. For HCVP group, VAE was not detected in 50% of the patients; grade I VAE was detected in 23.8% of patients, grade II in 16.7% of patients, grade III in 9.5% of patients, and grade IV in none of the patients. HCVP group had lower grades and incidence of VAE as compared with C group (P<0.001).

Grade IV VAE was detected in 2 patients of C group, with a sudden decline of PETCO₂ by over 2 mmHg but no obvious hemodynamic and ECG changes. The time of onset of VAE was from the damage of vertebral lamina to suturing of muscles. All patients were discharged from hospital after recovery without VAE-related neurological complications.

Postoperative neurological complications were observed until discharge from hospital, including the clinical signs of focal cerebral infarction and psychiatric symptoms.

Figure 3. A. Mid-esophageal four-chamber view. B. Bicaval view. RA, right atrium; RV, right ventricle; LA, left atrium; LV, left ventricle.
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complications, such as neurological dysfunction, altered mental state and coma.

Discussion

Spine surgery will inevitably cause damage to the vertebral lamina, vertebral bodies and vertebral pedicle. Subsequently the air may enter the venous system via the exposed cancellous bone [9], leading to VAE. To lower the inferior vena cava pressure (IVCP) and hence the intraoperative bleeding, a frame is usually used to elevate the abdomen. However, the decreased blood pressure gradient between the operated site and the right atrium adds the risk of air entering the blood circulation [10]. It is reported that when the blood pressure gradient decreases to 5 cm, the air may enter the veins [11]. We infer that the incidence of VAE can be reduced by increasing the blood pressure gradient through the increase of CVP (i.e., right atrium pressure), and this is exactly what was done during surgery. Results showed that the incidence of VAE in HCVP group was significantly lower than that in C group.

Vertebral lamina decompression in prone position is unable to fully expose the chest, which restricts the manipulation of transducer. TEE is the most sensitive method to monitor VAE and also free from such constraint for this type of surgery [12]. Although TEE is able to detect air at the amount of 0.02 ml/kg in a single injection [13], it is an invasive technique not yet popularized in department of anesthesia. In this study, intravenous fluid infusion was mainly performed via the peripheral vein using the infusion pump. The infusion rate was less than 50 mL/min to avoid infusion via the central venous catheter and rapid peripheral vein infusion that would generate the interfering air emboli on TEE images. TEE monitoring was stopped during intravenous injection until the disturbance to TEE images disappeared [14].

To adjust CVP for HCVP group, the amount of fluid infusion was varied in this study. Rapid fluid infusion was performed during the period from establishing venous access to the damage of bone structure to achieve the target value of CVP. According to the definition of CVP in the experimental section, CVP was affected by the changes of arterial pressure and heart rate. To control for the changes of arterial pressure and heart rate, the depth of anesthesia was adjusted properly and the vasoactive drugs were administered, including norepinephrine, ephedrine, atropine, trinitroglycerol and Esmolol, so that the fluctuation would be less than 20% from the baseline.

In case of severe VAE, central venous catheter can be used to remove the air entering the atria, and the multi-hole central venous catheter is more effective than single-hole central venous catheter [15, 16]. Although only about 50% of the air will be removed, the fatal events can be avoided [17]. The degree of damage caused by VAE is determined by amount, rate and type of the entering air and the position of the patient [18]. Large dose of air rapidly entering the veins and the pulmonary vessels will induce vascular endothelial injury [19, 20]. This will cause gaps between endothelial cells and pulmonary edema [21], severely affecting pulmonary gas exchange function, increasing pulmonary circulation resistance and pulmonary arterial pressure. VAE may also lead to decreased cardiac output, arrhythmia, right heart damage and finally heart failure and death [22-24]. On ECG, the manifestations include ST-T changes and the subsequent supraventricular and ventricular arrhythmia [25]. Even small dose of air entering the arterial circulation via the ventricular septal defect or atrial septal defect such as patent foramen ovale (PFO) will result in paradoxical embolism and severe clinical events. The risk is especially high when the air emboli accumulate, thereby increasing the pulmonary arterial pressure. Autopsies show that the incidence of VAE in adults with PFO is 20%-35% [26]. Physicians should be fully aware of the risk of right-to-left shunting which may lead to air embolism in heart and brain.

### Table 1. Patient characteristics

<table>
<thead>
<tr>
<th></th>
<th>Control group (n=40)</th>
<th>High central venous group (n=42)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>40.2±7.34</td>
<td>41.6±6.9</td>
<td>0.315</td>
</tr>
<tr>
<td>Sex ratio (M/F)</td>
<td>19/21</td>
<td>20/22</td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>65.9±9.8</td>
<td>64.4±8.7</td>
<td>0.736</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>165.0±9.3</td>
<td>167.7±11.2</td>
<td>0.511</td>
</tr>
<tr>
<td>Operative time (min)</td>
<td>168.5±34.4</td>
<td>176.1±40.4</td>
<td>0.508</td>
</tr>
<tr>
<td>Blood loss</td>
<td>381.2±98.0</td>
<td>481.2±120.0</td>
<td>0.08</td>
</tr>
</tbody>
</table>

The values are reported as mean ± SD.
Among all cases, two had grade III VAE, and the surgery was stopped immediately. The surgical site was applied with normal saline and the head was placed higher than the feet [27] so as to avoid severe VAE. However, no air was aspirated from the central venous catheter; there was neither instability of circulations, nor ECG changes such as ST-T changes or supraventricular and ventricular arrhythmia. These findings agreed with the TEE monitoring of VAE in other studies [7, 12, 14, 28] due to small dose of air entering the heart.

To conclude, the incidence and grades of VAE were lower in HCVP group than in C group. Maintaining HCVP is beneficial for reducing the incidence of VAE. Although intraoperative bleeding will increase due to HCVP, this defect can be resolved by large-dose fluid infusion and the use of blood recovery unit, especially for high-risk population such as patients with PFO. According to case report, severe VAE is most frequent in patients with normal heart function [3-5], which may be attributed to lower CVP in these patients. Therefore, the changes of CVP in patients with normal heart function deserve extra attention for reducing the incidence of VAE and increasing the safety of surgery.

Disclosure of conflict of interest

None.

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References

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Table 2. Changes in vital signs

<table>
<thead>
<tr>
<th>Group</th>
<th>Before induction</th>
<th>5 minutes after Bone destruction</th>
<th>30 minutes after Bone destruction</th>
<th>60 minutes after Bone destruction</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>SBP</td>
<td>DBP</td>
<td>MBP</td>
<td>PR</td>
</tr>
<tr>
<td>Control</td>
<td>132.3±18.8</td>
<td>84.7±15.4</td>
<td>100.7±18.3</td>
<td>74.2±13.6</td>
</tr>
<tr>
<td>HCVP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

SBP, systolic blood pressure; DBP, diastolic blood pressure; MBP, mean blood pressure; PR, pulse rate per minute; PETCO₂, end-tidalCO₂ partial pressure; PaO₂, pulse oximetric saturation. CVP, central venous pressure. The values are reported as mean ± SD. There was no significant difference between the two group (P>0.05). *The CVP in HCVP group were significantly higher than those in the Control group (P<0.001).
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