Air Embolism during Insertion of Central Venous Catheters

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PURPOSE: To determine the clinical consequences of air embolism occurring during insertion of central venous catheters.

MATERIALS AND METHODS: A computer search of our interventional radiology database revealed that 11,583 central venous catheters were inserted between January 1, 1995 and August 1, 2000: 7,178 were nontunneled and 4,404 were tunneled. Air embolism was reported to have occurred in 15 patients. Air embolization was recognized by audible air aspiration during catheter insertion or by fluoroscopic visualization of air within the right atrium or pulmonary artery. The radiology reports and medical records of these 15 patients were reviewed.

RESULTS: All 15 patients had an air embolism occur during insertion of a tunneled central venous catheter. These included eight Ash catheters, five chest wall ports, one Tesio catheter, and one Pheres-Flow catheter. Four patients remained asymptomatic. Six patients had mild symptoms that quickly resolved with supplemental oxygen. Four had moderate symptoms that also resolved with supplemental oxygen. One patient died acutely as a result of the air embolism.

CONCLUSION: Air embolism is a rare but potentially fatal complication of central venous catheter procedures. In our series, all occurred during insertion of a tunneled catheter through a peel-away sheath. The administration of supplemental oxygen was an effective treatment in the majority of patients.

Index terms: Catheters and catheterization, complications • Central venous access • Embolism

THE insertion of a central venous catheter is a procedure that is commonly performed by many interventional radiologists. By using ultrasound (US) and fluoroscopic guidance, radiologists have improved the safety and proficiency of this procedure when compared to bedside or surgical placement (1–3). Although uncommon, problems and complications can still occur during central venous access procedures. One of the most serious complications is an air embolism, a potentially fatal event caused by the sudden entry of air into the heart or pulmonary artery.

The true incidence of this complication is unknown. However, the frequency of air embolism during central venous access procedures is probably underestimated because the signs and symptoms are nonspecific and may be transient (4).

In our interventional radiology practice, we have observed multiple occurrences of air embolism during the insertion of central venous catheters. To determine the clinical consequences of this complication, a retrospective study of our patient population was performed.

MATERIALS AND METHODS

A computer search of our interventional radiology database revealed that 11,583 central venous catheters were inserted between January 1, 1995, and August 1, 2000, at our institution. These included 7,179 nontunneled catheters and 4,404 tunneled catheters. A refined computer search was performed to identify patients who were reported to have experienced an air embolism during insertion of a central venous catheter.

An air embolism was reported to have occurred in 15 patients. Air embolization was documented by audible air aspiration (“sucking sound”) in four patients, fluoroscopic visualization of air within the heart or pulmonary artery in seven patients, and both in four patients. The radiology procedural reports and medical records of these 15 patients were reviewed.

The median age of these 15 patients was 58.5 years (range, 23–90 y) and eight were women. Heart rate and rhythm and peripheral oxygen saturation were continuously monitored in all patients undergoing a central venous catheter insertion procedure. The majority of patients received mild conscious sedation with intravenous anxi-
Information for Each Patient Who Had a Catheter-related Air Embolus

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>Age (y)</th>
<th>Catheter Type</th>
<th>Sheath Size (F)</th>
<th>Signs and Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>54</td>
<td>Port</td>
<td>10</td>
<td>Brief decrease in (O_2) saturation</td>
<td>Left lateral decubitus position; face mask with 100% (O_2)</td>
</tr>
<tr>
<td>2</td>
<td>73</td>
<td>Port</td>
<td>10</td>
<td>Asymptomatic</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>52</td>
<td>Tesio</td>
<td>11</td>
<td>Severe dyspnea, death</td>
<td>Left lateral decubitus position; full code</td>
</tr>
<tr>
<td>4</td>
<td>56</td>
<td>Ash</td>
<td>16</td>
<td>Asymptomatic</td>
<td>None</td>
</tr>
<tr>
<td>5</td>
<td>23</td>
<td>Ash</td>
<td>16</td>
<td>(O_2) saturation decreased to 80%; dyspnea; tachycardia</td>
<td>Left lateral decubitus position; face mask with 100% (O_2); resolved in 5-10 minutes</td>
</tr>
<tr>
<td>6</td>
<td>47</td>
<td>Port</td>
<td>10</td>
<td>Brief chest pain; (O_2) saturation decreased to 75%</td>
<td>Face mask with 100% (O_2)</td>
</tr>
<tr>
<td>7</td>
<td>60</td>
<td>Neostar</td>
<td>13</td>
<td>Transient (&gt;60 sec) hypoxia</td>
<td>None</td>
</tr>
<tr>
<td>8</td>
<td>61</td>
<td>Ash</td>
<td>14</td>
<td>Asymptomatic; (O_2) saturation decreased to 85%</td>
<td>Left lateral decubitus position; 100% (O_2) via nasal cannula</td>
</tr>
<tr>
<td>9</td>
<td>90</td>
<td>Port</td>
<td>10</td>
<td>Asymptomatic</td>
<td>None</td>
</tr>
<tr>
<td>10</td>
<td>70</td>
<td>Ash</td>
<td>16</td>
<td>Tachycardia; hypotension; unresponsive</td>
<td>100% (O_2) via nasal cannula; Narcan; intravenous fluid bolus</td>
</tr>
<tr>
<td>11</td>
<td>75</td>
<td>Ash</td>
<td>16</td>
<td>Asymptomatic</td>
<td>None</td>
</tr>
<tr>
<td>12</td>
<td>59</td>
<td>Port</td>
<td>9, 11, 13</td>
<td>(O_2) saturation decreased to 70%; hypotensive</td>
<td>Left lateral decubitus position, 100% (O_2) via nasal cannula</td>
</tr>
<tr>
<td>13</td>
<td>80</td>
<td>Ash</td>
<td>16</td>
<td>Mild dyspnea, mild hypotension</td>
<td>Left lateral decubitus position; 100% (O_2) via nasal cannula</td>
</tr>
<tr>
<td>14</td>
<td>41</td>
<td>Ash</td>
<td>16</td>
<td>Hypotension; dyspnea</td>
<td>Left lateral decubitus position; 100% (O_2) via face mask</td>
</tr>
<tr>
<td>15</td>
<td>37</td>
<td>Ash</td>
<td>16</td>
<td>Tachycardia; chest pain</td>
<td>100% (O_2) via face mask</td>
</tr>
</tbody>
</table>

Oxytocic and analgesic medications. The standard sedation regimen consisted of 1 mg of midazolam (Abbott Laboratories, North Chicago, IL) and 50 \(\mu\)g of fentanyl (Abbott Laboratories).

RESULTS

Fifteen patients had an air embolism during insertion of a tunneled central venous catheter (Table). These 15 catheters included eight Ash Split catheters (Medcomp, Harleysville, PA), five chest ports (CR Bard, Salt Lake City, UT), one Tesio Twin catheter (Medcomp) and one Pheres-Flow catheter (Neostar, Manchester, GA). Eight catheters were inserted into the internal jugular vein and seven into the subclavian vein. Thirteen were right-sided placements and two were left-sided. Despite the air embolism, 14 (93%) of these patients had successful insertion of the central venous catheter.

Symptoms of the air embolus were reported in 11 of these 15 patients, whereas four remained asymptomatic. Thirteen of the 15 patients were lying flat on an angiography table at the time of the air embolization event. One patient, who had severe dyspnea before the procedure, was positioned with his head and upper chest elevated (at approximately 30°) during the procedure. Another procedure was performed on a tilting fluoroscopy table with the patient placed in a head-down position at the time of catheter insertion.

All 15 air embolization events were reported to have occurred during the time when the catheter was inserted through the introducer (peel-away) sheath. Patient participation maneuvers to prevent air embolism, such as the Valsalva maneuver, respiratory expiration, or humming, are not routinely performed during catheter insertion procedures at our institution. Our standard practice is to immediately pinch and occlude the introducer sheath after removal of the inner dilator and guide wire. The catheter is then quickly inserted into the introducer sheath while the pinched occlusion is maintained. The pinch is then carefully released as the catheter is rapidly advanced through the introducer sheath. After the catheter has been advanced into its final position, the introducer sheath is peeled away in the standard fashion. Review of the radiology procedural reports of the 15 study patients revealed that one patient was asked to perform the Valsalva maneuver and one patient was asked to suspend respiration while the catheter was advanced through the introducer sheath.

All of these central venous access procedures were directly supervised by an attending interventional radiologist. The primary operator during catheter insertion was a radiology resident in three procedures, an interventional radiology fellow in 10 procedures, and a staff interventional radiologist in two procedures.

As mentioned, all 15 air embolus events occurred while the catheter was advanced through the introducer sheath. One patient coughed as the catheter was inserted into the sheath. The patient who was asked to perform the Valsalva maneuver inadvertently took a large breath as the catheter was inserted into the introducer sheath. In two other patients, the operator physician had difficulty advancing the catheter through a kinked introducer sheath. In one of these patients, three different sheaths (9-, 11-, and 13-F) were used before the catheter was finally inserted.

Four of the patients who experi-
enced an air embolus remained asymptomatic and no treatment was given. Six patients had mild symptoms, such as brief chest discomfort or dyspnea, accompanied by a transient decrease in peripheral oxygen saturation. All six of these mildly symptomatic patients had rapid resolution of their clinical symptoms after the administration of supplemental oxygen. In addition, two of these patients were turned to a left lateral decubitus position immediately after the air embolization.

Four patients had moderate or severe symptoms as a result of the air embolus. These symptoms included chest pain, dyspnea, coughing, peripheral numbness, or a sense of impending death. One patient became unresponsive for a period of approximately 5 minutes. These symptoms were accompanied by cyanosis, hypotension, tachycardia, and decreased peripheral oxygen saturation. Supplemental oxygen, via nasal cannula or face mask, was administered to all four of these patients. Two patients were placed into the left lateral decubitus position. One patient, the patient who became unresponsive, was also given naloxone. All of the patients stabilized with treatment and were further observed for 30 to 90 minutes. All had complete resolution of their symptoms and returned to their baseline vital signs at the time of discharge from the radiology department.

One patient died during the procedure as the result of a massive air embolus. This patient had Wegener’s granulomatosis with extensive pulmonary involvement. The patient had severe dyspnea before and during the catheter placement procedure. For this reason, the patient was positioned in a semi-sitting position with his head and upper chest elevated with several pillows. Supplemental oxygen was administered via nasal cannula during the procedure. An audible air embolus occurred during insertion of the catheter through the introducer sheath and the patient reported a worsening shortness of breath. Fluoroscopy revealed a large amount of air within the cardiac silhouette. The patient was immediately rolled into a left lateral decubitus position. The patient had an acute cardiac arrest, cardiopulmonary resuscitation was initiated, and the code team was called. Fluoroscopy demonstrated both lungs to be fully inflated with no evidence of a pneumothorax. Despite resuscitative efforts, the patient died in the angiography suite.

DISCUSSION

The majority of published reports describing catheter-related venous air embolism have been case reports or small series with few patients. Feli- ciano et al (5) reviewed the major complications that occurred during placement of “over 1,500 subclavian vein venipunctures” and reported that two patients (0.13%) experienced air embolus (5). In another report, a review of 170 catheter-related complications that occurred in 185 patients, only four patients had an air embolus (6).

In our study group of 15 patients, all of the air embolization events occurred while the catheter was advanced through the introducer sheath into the superior vena cava or right atrium. Morello et al (7) described three cases of air embolism that occurred during placement of tunneled catheters in children. They also reported that all three events occurred as the catheter was advanced through the introducer (peel-away) sheath (7). Perini et al (8) reported a series of Tesio catheter placements in 71 patients, one of whom had a fatal air embolus. The event was reported to have occurred during insertion of the introducer sheath.

However, the majority of catheter-related air embolization events that have been reported have not occurred during the catheter insertion procedure (9–13). This complication has been attributed to multiple other causes, including detachment of the intravenous tubing from the catheter hub, failure to close the hub, a fractured catheter, or air entering a persistent subcutaneous tunnel after catheter removal.

Air will enter the venous system when there is an open communication between atmospheric air and the central veins, and there is a simultaneous decrease in intravenous pressure, as occurs during inspiration. Significant contributing factors include the patient’s body position and hydration status (14–16). An upright position will accentuate the normal decrease in intravenous pressure that occurs within the central veins during inspiration. Traditional training has dictated that patients be placed in a head-down position during insertion of central venous catheter (17–19). This position helps to maintain a positive central venous pressure, thereby decreasing the likelihood of an air embolus.

Hypovolemia can cause a low intravascular or intracardiac pressure and accentuate the entry of air into the central venous system during catheter placement.

The sudden entry of a large bolus of air into the right ventricle is thought to act as an “air lock,” a mechanical obstruction of the right ventricular outflow tract (14–19). This abrupt increase in resistance to right ventricular outflow has several effects, including increasing right heart pressures, decreasing pulmonary venous return, decreasing left ventricular preload, and decreasing systemic arterial pressure. The immediate cardiac response to an air embolus is tachycardia that changes to bradycardia after 1–3 minutes. There is also an immediate increase in pulmonary arterial pressure and a decrease in mean arterial (systemic) pressure. These hemodynamic effects will diminish cardiac output, decrease coronary artery perfusion, and eventually lead to cardiovascular collapse.

The increased right heart pressures can also cause an arterial air embolism through a right-to-left shunt, such as a patent foramen ovale (“paradoxic” air embolism) (20,21).

Patients who have an air embolus may remain asymptomatic or experience only mild symptoms and the event goes unnoticed. As noted in this study, four (26.7%) of 15 patients remained completely asymptomatic.

The clinical symptoms of venous air embolism are nonspecific. The patient’s response to an air embolus is dependent on several variables, including the volume of air, the rapidity with which it enters the venous circulation, and the cardiovascular status of the patient. Patients may report chest pain, air hunger, dizziness, or a feeling of impending death. Several reports have mentioned that the patient may gasp or cough immediately after entry of air into the central venous circulation (12,15,22). All of these symptoms
were experienced by at least one of the 15 patients in our study group.

In animals, it has been shown that a volume of >1.8 mL/kg of air is often fatal when delivered as a sudden bolus. In humans, the fatal dose is unknown but estimates vary between 300 and 500 mL of air at 100 mL/sec (14,16).

The classic treatment for an acute venous air embolism is to immediately move the patient into a left lateral decubitus position (“Durant’s position”) (17–19). In the left lateral decubitus position, the right ventricular outflow tract is inferior to the body of the right ventricle. In this position, air will be displaced away from the right ventricular pulmonary outflow tract, thereby relieving the mechanical obstruction or “air trapping” phenomenon. In this series, seven of 15 patients were rolled into a left lateral decubitus position immediately after the air embolism was identified (Table). It is not known if this maneuver was beneficial. Anecdotally, in the majority of patients who had an air embolus visualized fluoroscopically, the air was seen within the pulmonary artery and not in the right ventricle. The left lateral decubitus position would probably not be helpful when the air embolus is within the main pulmonary artery or segmental branches.

Animal studies of echocardiography to study the effects of body positioning during experimental venous air embolization have refuted this traditional theory. Geissler et al (17) reported that there was no difference in the hemodynamic response to an air embolus when the animal was rolled into the left lateral decubitus position. However, when the animal was in the left lateral decubitus position, the air within the right atrium was persistent for a longer duration than when in the supine position. In addition, there was an acute increase in the diameter of the right ventricle caused by the air embolism and this increase was greater when the animal was in the left lateral decubitus position. Because of these detrimental effects, Geissler et al (17) recommend leaving the patient in the supine position.

The administration of supplemental oxygen can be one of the most effective treatments that can be easily performed in the angiography suite. The administration of 100% oxygen can decrease the size of the air embolus by causing nitrogen to diffuse out of the air bubbles and into the alveoli, a phenomenon referred to as nitrogen washout (14–16,21).

Successful aspiration of an air embolus with use of a central venous catheter has been reported (23,24). However, in most cases, the amount of air that was aspirated was minimal and probably did not represent a significant factor in improving the patient’s condition. As stated by Orebaugh (14), if a central venous catheter is present it, may be useful to attempt to aspirate air from the right atrium, but the low success rate does not justify the time and effort necessary to insert a new catheter in the acute situation.

In this study, despite the acute air embolus, 14 of 15 patients had successful insertion of the central venous catheter. Aspiration of the air embolus was not attempted. As previously mentioned, when identified fluoroscopically, the air embolus was usually in the main pulmonary artery or segmental branches. The newly inserted catheter, with the tip positioned at the junction of the superior vena cava and right atrium, would not be useful for aspirating air in this situation. If the air embolus were within the right atrium or pulmonary outflow tract, turning the patient into a left lateral decubitus position would cause the air embolus to rise into the right atrium, an advantageous position for aspiration via the catheter.

Although it is important to know how to correctly treat a patient who has experienced an acute air embolus, it is more useful to focus on prevention of this complication. The use of introducer sheaths, which are an open communication between atmospheric air and the central venous circulation, continue to be the most common method for insertion of large-diameter central venous catheters. Interventional radiologists often occlude the sheath by pinching it after removal of the inner dilator. But, as reported in this study, this technique is fallible. Several other techniques that have been used for the prevention of air embolization attempt to increase the central venous pressure during the catheter insertion procedure. These methods include placing the patient in a head-down position, elevating the patient’s legs, inserting the catheter during respiratory expiration, or having the patient perform the Valsalva maneuver. In our experience, patient participation maneuvers, such as having the patient exhale while inserting the catheter, can be incorrectly performed and may increase the risk of an air embolism.

Another technique involves a long introducer sheath advanced through the right atrium into the inferior vena cava. The intravascular pressure within the inferior vena cava is always higher than atmospheric pressure, so there is no danger of air embolism while the sheath is in this position (25). The central venous catheter is advanced through the sheath, which is then peeled away in the standard fashion. There is a slight risk of air embolism as the distal end of the sheath is retracted through the right atrium and superior vena cava, but this is minimized if the sheath is removed quickly.

A more recently reported technique eliminates the use of the introducer sheath (26). After subcutaneous tunneling of the catheter and dilation of the venous entry site, large-diameter catheters can be inserted into the central veins with use of one or two stiff guide wires. Although this technique requires some practice, it can be successfully performed in many patients.

Hypovolemia is a significant contributing factor for a venous air embolus. The patient should be well hydrated to maintain a high right atrial pressure (15). This will optimize cardiac output and arterial pressure, and can also decrease the risk of a paradoxical air embolism by reducing the right-to-left atrial pressure gradient.

Finally, redesigning the introducer sheath could reduce the risk of air embolism during catheter insertion. An iris-type or spline valve positioned within the introducer could provide a tight seal around the catheter, thereby preventing air entry.

In conclusion, air embolism is an uncommon complication of central venous access procedures. The majority of patients in this study group were effectively treated with the administration of 100% oxygen. Although it is useful to know how to correctly treat a patient who has sustained an acute air embolus, it is more important to focus on the prevention of this complication.
References