Case Report

Uncommon Occurrence of an Air Embolism during the Preanhepatic Phase of an Orthotopic Liver Transplant

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1. Introduction

Vascular air embolism (VAE) commonly occurs in liver transplantation while performing vascular anastomosis and during the liver reperfusion phase (inadequate hepatic wash out and anastomotic leaks) [1]. This phenomenon typically develops due to the entrapment of air from the operative field into the venous circulation or from intrahepatic air contained in the donor liver [2]. Air emboli have the potential to enter the right side of the heart and eventually the pulmonary circulation. Air emboli also have the potential to enter the arterial circulation through intracardiac and intrapulmonary shunts which can result in devastating cardiac or neurological complications. We present the case of a VAE during the preanhepatic phase of an orthotopic liver transplant (OLT) due to a large defect in the vena cava.

2. Case Description

A 52-year-old patient with a past medical history of decompensated liver cirrhosis secondary to alcohol use underwent an OLT. The patient’s symptoms prior to admission included chronic hyponatremia, dyspnea, ascites (requiring multiple paracentesis), bilateral peripheral edema, and altered mental status. The patient had no prior history of cardiovascular or pulmonary comorbidities. Preoperative transthoracic echocardiogram (TTE), electrocardiogram (ECG), and myocardial perfusion imaging were normal. The patient’s Model for End Stage Liver Disease-Sodium (MELD-Na) score was 30 and Maddrey’s Discriminant Function Score was 67.8 (creatinine, 0.95 mg/dl; bilirubin, 13.1 mg/dl; international normalized ratio, 2.11; sodium, 123 mg/dl; and prothrombin time 24.4 seconds).

Anesthesia was induced with fentanyl (250 mcg), propofol (150 mg), and maintained with sevoflurane. Muscle relaxation was achieved using rocuronium and ventilation was mechanically controlled to maintain an end-tidal carbon dioxide pressure between 30 and 35 mm Hg along with a positive end-expiratory pressure of 5 cm H2O. Intraoperative monitors comprised standard American Society of Anesthesiologists monitors and a 20-gauge right radial artery invasive monitor. Pulmonary artery (PA) and central venous pressure (CVP) measurements were obtained continuously via a Swan-Ganz catheter placed through a 9 French multilumen sheath introducer in the right internal jugular vein.

Consistent with our institution’s practice, the OLT was done by piggyback technique with the preservation of the recipient’s vena cava. After the liver was mobilized in the preanhepatic phase, the short hepatic veins of the liver were
identified. A low-normal CVP was maintained to decrease hepatic congestion as well as bleeding from the liver sinu-
soids and hepatic veins. However, during ligation of these
vessels, there was significant bleeding which was controlled
by suture ligation. The surgical team anticipated further
bleed, and as a result, the native liver heptectomy was
completed on veno-venous bypass at a flow of 3 liters per
minute. This required cannulation of the left femoral vein
and portal veins providing venous return to the left axillary
vein. About three hours into the start of the surgery, during
the preanhepatic phase, an inadvertent injury to the inferior
vena cava was made. A massive transfusion protocol was
initiated as per the hospital’s protocol, and the patient was
resuscitated and the defect was repaired.

During this time, there was a sudden decline in the
patient’s end-tidal carbon dioxide from 31 to 11 mmHg. The
CVP immediately before this event was 6 mmHg. The
pulmonary arterial pressure increased from 25/15 to 43/25
and hypotension ensued. The blood pressure dropped to 67/
31, oxygen saturation was 88%, peak airway pressure in-
creased, and sinus tachycardia developed at a rate of 110
beats per minute. An acute pulmonary air embolism was
suspected, the operating surgeon was immediately notified,
and the field was flooded with normal saline. The fraction
of inspired oxygen (FiO2) was increased to 100 percent, and the
patient required hemodynamic support with 8 mcg/minute
of norepinephrine, 2.4 units/hour of vasopressin, and 3 mcg/
minute of epinephrine infusions. The patient was placed in
reverse Trendelenburg position, and approximately 7 ml of
air was aspirated from all 3 ports of the Swan-Ganz catheter.

Over the next 10 minutes, there was gradual improve-
ment in his PA pressure to 18/11, end-tidal carbon dioxide
increased to 25 mmHg, oxygen saturation increased to 99%,
and the patient became normotensive. ABG revealed a pH of
7.30 along with partial pressures of carbon dioxide and
oxygen of 39 mmHg and 253 mmHg, respectively. The pa-
patient remained hemodynamically stable thereafter. The pa-
tient’s total blood loss was 20 liters throughout the case
requiring 14 pack red blood cells, 14 units of FFP, 35 units of
cryoprecipitate, and 18 units of platelets. The blood product
resuscitation was guided by rotational thromboelastometry
(ROTEM), estimated blood loss, and hemoglobin levels. At
the conclusion of the case, the patient remained intubated
and was transferred to the intensive care unit. The patient
was extubated on postoperative day (POD) 2, and the
postoperative course was complicated by acute renal failure
and a small occipital intraparenchymal hemorrhage. On
POD16, he was transferred to an acute rehabilitation facility.

3. Discussion

There are three main phases that occur during liver trans-
plants: the preanhepatic, anhepatic, and neohepatic phase.
Most causes of air embolism thus far that have been reported
occur during the surgical dissection phase and during the
reperfusion phase. This case report describes the unique
development of a pulmonary air embolism during the
preanhepatic phase due to a large iatrogenic defect in the
vena cava. The preanhepatic phase starts from the initial
surgical incision and ends with cross clamping of the portal
vein, the inferior vena cava, and the hepatic artery [3].
During the anhepatic and neohepatic phases, air entrain-
ment can occur, while performing surgical anastomosis,
during venous bypass, or during liver reperfusion (insuffi-
cient hepatic wash out or leaks in vascular anastomoses) [1].
The air that enters the liver graft can cause obstruction to the
hepatic microcirculation, obstruction to cardiac output, and
paradoxical air embolism producing potential life-threaten-
ing systemic effects.

The volume of air as well as rate at which the air is
entrained usually determines the morbidity and mortality of
air embolisms [4]. This is dependent on a pressure gradient
and the caliber of the vessel that entrains the air. The CVP
was intentionally kept low-normal during the preanhepatic
phase which may have facilitated the air entrainment.
Typically, when the air is suctioned by a gravitational gra-
dient, the rate and volume of air entrained are affected by the
position of the patient and height of the vein with respect to
the right side of the heart. In adults, lethal volume is between
200 and 300 ml, or 3–5 ml/kg. If a large amount of air enters
the right side of the heart, an air-lock mechanism may occur,
leading to RV outflow obstruction.

There have been numerous reports of large air embolism
that had no clinical repercussions or were resuscitated
successfully. However, air embolisms usually trigger dif-
f erent cardiovascular, pulmonary, and neurological prob-
lems. Electrocardiogram (ECG) abnormalities such as
tachyarrhythmias and ST and T wave changes can occur. Our
patient initially had a tachycardic response possibly due to
right heart strain from the air embolism and hypotension.
Additionally, the blood pressure dropped possibly due to
the reduction of cardiac output. The PA pressure increased due
to both a reduction in cardiac output and increased filling
pressures [3]. This sudden decrease in cardiac output causes
a ventilation to perfusion mismatch, precipitating a drop in
the patient’s end-tidal carbon dioxide, arterial oxygen ten-
sion and saturation. Transesophageal echocardiography is
considered to be the most sensitive monitor for air embo-

lism. TEE may be useful in identifying the size and location
of the air embolism as well as intracardiac shunts that allow
passage of the air into the arterial circulation [5]. This
modality was not used during this case.

Management of venous air embolism focuses on pre-
vention of further air entrainment, reduction of the air
entrained and hemodynamic support [4]. We promptly
notified the surgeon, and the surgical field was flooded with
saline to prevent further entrainment and the defect in the
vena cava was also immediately repaired. During that time,
we supported the patient’s blood pressure with vasopressors.
In an effort to reduce the volume of air entrained we as-
spirated air from the Swan-Ganz catheter. However, both
multilumen catheters and Swan-Ganz catheters have a
success rate of aspirating air between 6 and 16 percent [4].
These strategies resulted in prompt improvement in the
patient’s hemodynamics. We suspected that the renal failure
that developed was possibly caused by a combination of
hypovolemia and venous congestion from the venous air
embolism.
In summary, this case report highlights the occurrence of a venous air embolism during an OLT causing hemodynamic instability. Our treatment focused on identifying and repairing the source of entrainment, reduction of volume of air in the right heart through aspiration, and hemodynamic support.

Conflicts of Interest
The authors declare that there are no conflicts of interest.

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