

# Occult Hemothorax after Transcatheter Aortic Valve Implantation (TAVI)

Kenneth D. Eichenbaum, Bryan Noorda, Walter Bethune, Dennis E. Feierman\*

Department of Anesthesiology, Maimonides Medical Center, New York, USA.  
Email: \*dfeierman@maimonidesmed.org

Received May 18<sup>th</sup>, 2013; revised June 19<sup>th</sup>, 2013; accepted July 5<sup>th</sup>, 2013

Copyright © 2013 Kenneth D. Eichenbaum *et al.* This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

## ABSTRACT

We present a case of occult hemothorax, a rare but dangerous complication resulting from cannulation of the internal jugular vein. To date we are not aware of any case reports of bleeding sequelae resulting from direct parenchymal lung injury. The insidious nature of this complication, in which the clinical presentation occurred several hours after central venous cannulation, provides an important reminder for clinicians to follow up central line placement with imaging studies. In this case, the latent period prior to the appearance of clinical manifestations of hemorrhage along with the patient's subsequent acute decompensation raises questions as to the nature of the underlying injury and mechanisms of both detection and prevention. The patient underwent two right video-assisted thoracoscopic surgical explorations, the first revealed ongoing venous bleeding from within the parenchyma of the right upper lobe which was controlled by a wedge resection. She was ultimately discharged home and, as of the time of this writing, appears to have recovered completely, suffering no long-term sequelae as a result of this complication.

**Keywords:** Central Line Complication; Hemothorax; TAVI

## 1. Introduction

An 82-year-old female presented with severe, symptomatic aortic stenosis. Her symptoms included dyspnea with minimal exertion and decreased exercise tolerance, with limited functional capacity: only able to walk half a block. The patient was not a candidate for valve replacement by conventional open-heart surgery due to age, compromised left ventricular function, pulmonary hypertension and diffuse general atherosclerosis. The patient was therefore scheduled for trans-catheter aortic valve implantation. Past medical history was significant for coronary artery disease requiring percutaneous coronary stent placement, severe aortic stenosis, with a valve area 0.9 cm<sup>2</sup>, dyslipidemia, chronic anemia secondary to a myelodysplastic syndrome, gout, and splenomegaly. Her surgical history only included her percutaneous coronary intervention. EKG was significant for left ventricular hypertrophy and frequent premature atrial complexes. Trans-thoracic echocardiogram showed: 1) a severely calcific stenotic aortic valve with aortic valve area of 0.9 cm<sup>2</sup>, mean gradient 40 mm Hg, peak velocity/gradient 4.3 msec<sup>-1</sup>/74 mm Hg, respectively, with a left ven-

tricular ejection fraction of 30%, 2) trace aortic insufficiency, trace mitral regurgitation, trace pulmonic insufficiency, and 3) moderate left ventricular hypertrophy. Cardiac catheterization revealed <40% stenosis in multiple coronary arteries. Her medications included metoprolol, plavix, aspirin, crestor, allopurinol, colchicine, and omeprazole.

## 2. Case

Peripheral intravenous and left radial arterial access was established. ASA standard monitors were placed and general anesthesia was slowly induced using 100 mg of propofol, 75 mcg of fentanyl and 6 mg of vecuronium for muscle relaxation. Anesthesia maintenance included 0.2% - 1% isoflurane and an additional dose of 25 mcg fentanyl. Approximately 90 minutes into the case a propofol infusion @ 80 mcg/min/kg was started and continued until the end of the procedure.

Using a glidescope, the airway was secured with a 7.0 endotracheal tube on the 2nd attempt. After sterile prep and drape, right internal jugular (IJ) vein was cannulated with a 9 Fr double-lumen introducer and a pulmonary artery catheter was inserted. Of note, the internal jugular

\*Corresponding author.

vein was visualized by “static” ultrasound and its course mapped on the overlying skin with a felt pen prior to sterile prep and drape. The right internal jugular was then accessed with a 22 g finder needle followed by an unsuccessful pass with an 18 g needle/catheter. Dynamic ultrasound was then used to visualize the right internal jugular vein with another unsuccessful 18 g needle pass. On the third attempt the internal jugular was successfully cannulated followed by the insertion of a guide-wire and the introducer was subsequently placed into the vein by Seldinger’s technique. The pulmonary artery catheter was inserted uneventfully, properly positioned (confirmed by pressure transduction) and confirmed to be in the left pulmonary artery by both transesophageal echocardiography and fluoroscopy. The patient was positioned and prepped, followed by commencement of the TAVI procedure. The case proceeded uneventfully: there were neither gas exchange abnormalities nor unexplained hemodynamic instability during the procedure. The total procedure took 210 minutes. During that period, the patient received 2.8 liters of normal saline and had a total of 1.3 liters of urine output.

After conclusion of the procedure, despite reasonable respiratory mechanics, she did not appropriately follow commands. She became increasingly hypercarbic (peak  $\text{ETCO}_2$  was approximately 60 mm Hg), and a decision was made to leave her intubated and have her transferred to cardiothoracic intensive care unit for post operative care. Just prior to transport, a slow air leak from the ETT cuff was discovered, prompting exchange for a new 7.0 ETT over a gum-elastic bougie. This proceeded uneventfully and the patient was transferred to an ICU bed and transported to CTICU.

During transport and over the first 2 hours in the ICU, the patient’s hemodynamic profile steadily deteriorated, requiring fluid/blood product resuscitation coupled with increasing vasopressor support. Initially only a few bolus doses of phenylephrine and ephedrine were required, but subsequently she required infusions of phenylephrine and norepinephrine in order to maintain adequate blood pressure. Bedside transesophageal echocardiography revealed a normal functioning prosthetic aortic valve and no obvious vascular injury. Chest X-ray revealed white-out of the right lung. A right chest tube was placed and approximately 2.5 liters of bloody chest tube output was recorded. This prompted video-assisted thoracoscopic surgical exploration.

Right video-assisted thoracoscopic surgical exploration revealed ongoing venous bleeding from within the parenchyma of the right upper lobe, which was controlled by a wedge resection. On post-operative day 1, continued venous bleeding was noted. The patient underwent a repeat VATS exploration and ligation of a bleeding chest wall vein. The patient remained hemodynamically

stable with no further bleed. Ventilator support and sedation were gradually weaned as tolerated and the patient was extubated on post-operative day 5. She was ultimately discharged to home and, as of the time of this writing, she appears to have recovered completely, suffering no long-term sequelae as a result of this complication.

### 3. Discussion

We present a case of hemothorax resulting from parenchymal injury during central venous cannulation. While hemothorax is a rare, but known complication of internal jugular cannulation [1], the mechanism of injury has often been attributed to cardiac tamponade or vascular injury, carotid puncture, catheter embolus, or pneumothorax. Several clinicians have also reported this complication resulting from laceration of the subclavian artery [2-4]. There is also one report of this complication resulting from right brachiocephalic vein injury [5].

Data from the ASA Closed Claims Project found the complication rate of central venous catheterization increased from 13.5% to 16.5% between 1990 and 2004 with associated deaths occurring in up to 47% of cases [1]. The incorporation of ultrasound imaging, pressure wave transduction, and post placement chest radiograph have been advocated as measures to improve patient safety. It is felt that these measures may have helped prevent close to half the reported central line related claims. The utility of ultrasound guided central line placement is now generally recognized and was recently incorporated as an SCA guideline [6,7].

Of note, the protracted delay in both the presentation and subsequent detection of the pulmonary insult prompt questions about the course of the injury. The injury is presumed to have occurred during central vein cannulation at the beginning of the procedure. However, the patient remained hemodynamically stable during the operative portion of the case, which lasted over 5 hours. This observation raises the following important questions: First, how did the patient compensate for this injury for over 5 hours during a complex procedure? Second, why did the patient decompensate so rapidly at the end of the procedure?

We suggest three explanations for the mechanics of this physiologic compensation and subsequent rapid decompensation. First and most obviously, the patient had been taking anticoagulant medications which predisposed her to bleeding complications. Second, during this case, positive pressure ventilation with PEEP was utilized, likely resulting in tamponade at the site of bleeding near the apex of the lung. The apparent pulmonary arterial injury was sufficiently small and distal to prevent the intra-vascular pressure from exceeding the constant positive pressure being applied to the lungs by this mode of ventilation. At the end of the case, the patient was wean-

ed from mechanical ventilation, gradually resuming spontaneous respiration. Associated with this transition was a switch from positive to negative pleural pressures.

We believe that the relatively fragile clot that would have existed at the injury site at this point could not withstand the negative intrapleural pressures, resulting in blood being drawn into the pleural cavity. The distinct physiological difference between mechanical (positive pressure) ventilation and spontaneous (negative pressure) ventilation greatly increased the patient's propensity for hemorrhage into the pleural space by removing the mechanisms that had limited bleeding. As the responsibility for breathing was shifted from the ventilator to the patient, she also gradually became hypercarbic. Hypercarbia is known to be associated with increased PA pressure [8,9]. The patient's previous history of pulmonary hypertension, coupled with this acute change may have increased intravascular pulmonary pressures that were too high for the fragile clot to withstand, thus resulting in hemorrhage into the pleural cavity. It is interesting, that the complication was a vascular injury leading to a hemothorax, instead of a pneumothorax. It is likely that all three of the proposed mechanisms played a role in the specific timing and severity of hemodynamic decompensation in this case.

## REFERENCES

- [1] K. B. Domino, T. A. Bowdle, K. L. Posner, *et al.*, "Injuries and Liability Related to Central Vascular Catheters: A Closed Claims Analysis," *Anesthesiology*, Vol. 100, No. 6, 2004, pp. 1411-1418. [doi:10.1097/00000542-200406000-00013](https://doi.org/10.1097/00000542-200406000-00013)
- [2] N. Kulvatunyou, S. O. Heard and P. E. Bankey, "A Subclavian Artery Injury, Secondary to Internal Jugular Vein Cannulation, Is a Predictable Right-Sided Phenomenon," *Anesthesia and Analgesia*, Vol. 95, No. 3, 2002, pp. 564-566.
- [3] Y. Beilin, D. Bronheim and C. Mandelbaum, "Hemothorax and Subclavian Artery Laceration during 'J' Wire Change of a Right Internal Jugular Vein Catheter," *Anesthesiology*, Vol. 88, No. 5, 1998, pp. 1399-1400. [doi:10.1097/00000542-199805000-00034](https://doi.org/10.1097/00000542-199805000-00034)
- [4] U. Jain, K. B. Shah, R. J. Belusko, *et al.*, "Subclavian Artery Laceration and Acute Hemothorax on Attempted Internal Jugular Vein Cannulation," *Journal of Cardiothoracic and Vascular Anesthesia*, Vol. 5, No. 6, 1991, pp. 608-610. [doi:10.1016/1053-0770\(91\)90017-N](https://doi.org/10.1016/1053-0770(91)90017-N)
- [5] Y. Innami, T. Oyaizu, T. Ouchi, *et al.*, "Life-Threatening Hemothorax Resulting from Right Brachiocephalic Vein Perforation during Right Internal Jugular Vein Catheterization," *Journal of Anesthesia*, Vol. 23, No. 1, 2009, pp. 135-138. [doi:10.1007/s00540-008-0696-1](https://doi.org/10.1007/s00540-008-0696-1)
- [6] C. A. Troianos, G. S. Hartman, K. E. Glas, *et al.*, "Special articles: Guidelines for Performing Ultrasound Guided Vascular Cannulation: Recommendations of the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists," *Anesthesia and Analgesia*, Vol. 114, No. 1, 2012, pp. 46-72. [doi:10.1213/ANE.0b013e3182407cd8](https://doi.org/10.1213/ANE.0b013e3182407cd8)
- [7] T. J. Milling Jr., J. Rose, W. M. Briggs, *et al.*, "Randomized, Controlled Clinical Trial of Point-of-Care Limited Ultrasonography Assistance of Central Venous Cannulation: the Third Sonography Outcomes Assessment Program (SOAP-3) Trial," *Critical Care Medicine*, Vol. 33, No. 8, 2005, pp. 1764-1769. [doi:10.1097/01.CCM.0000171533.92856.E5](https://doi.org/10.1097/01.CCM.0000171533.92856.E5)
- [8] K. Subramaniam and J. P. Yared, "Management of Pulmonary Hypertension in the Operating Room," *Seminars in Cardiothoracic and Vascular Anesthesia*, Vol. 11, No. 2, 2007, pp. 119-136. [doi:10.1177/1089253207301733](https://doi.org/10.1177/1089253207301733)
- [9] G. M. Balanos, N. P. Talbot, K. L. Dorrington, *et al.*, "Human Pulmonary Vascular Response to 4 h of Hypercapnia and Hypocapnia Measured Using Doppler Echocardiography," *Journal of Applied Physiology*, Vol. 94, No. 4, 2003, pp. 1543-1551.