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Case Report

Pericardiocentesis in an Ambulance: A Case Report and Lessons Learned

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A B S T R A C T

There are few procedures performed in the prehospital setting as intimidating as pericardiocentesis. We report a case in which lifesaving pericardiocentesis was performed in the back of an ambulance after temporizing measures of volume resuscitation and vasopressor therapy failed.

Fluid accumulation within the pericardial sac can increase pressures around the heart and lead to cardiac tamponade. Helicopter emergency medical service crews may be called to transport patients with cardiac tamponade physiology to definitive care where removal of the pericardial fluid can be achieved. Pericardiocentesis is indicated as an emergency procedure in patients with hemodynamic compromise secondary to cardiac tamponade.[1] Because most HEMS crews do not routinely perform pericardiocentesis because of the rare need or crew scope of practice limitations, the general approach to management in the prehospital setting is volume resuscitation, to overcome decreased preload, and vasopressor support. Here, we report a case in which lifesaving pericardiocentesis was performed in a ground ambulance after temporizing measures of volume resuscitation and vasopressor therapy failed.

Case Report

A 21-year-old man presented to a small rural hospital for complaints of sharp chest pain since the day prior. The emergency department administered ketorolac, and with improvement of his pain, the patient was discharged home on naproxen. Two days later, the patient saw his primary care provider for the same symptoms and was prescribed omeprazole for possible gastroesophageal reflux disease. The following day, 5 days after the initial symptoms, he awoke, acting delirious, with severe chest pain. His mother took him back to the small hospital, where he was found to be hypotensive, hypoxic, and agitated. A computed tomographic scan revealed a moderate pericardial effusion, and a follow-up transthoracic echocardiogram was ordered. As a result, air medical transport, based 31 miles away, was requested for transfer to definitive care, but because of inclement weather, the patient was transported the 71 miles via ground ambulance.

Upon the transport team's arrival, the patient's chest pain was still present. The patient was alert and mildly agitated. He had been given 3 L 0.9% normal saline and was on a norepinephrine (NE) infusion at 20 μg/min. His heart rate (HR) was in the 90s, his systolic blood pressure (SBP) was near the low 100s, and his peripheral capillary oxygen saturation (SpO_2) was in the mid 90s on a nasal cannula. Patient monitoring was continued, including HR, blood pressure, SpO_2, and end-tidal carbon dioxide ($ETCO_2$) via nasal capnography; the NE infusion was continued; and the 71-mile ground transport was initiated.

Significant pericardial effusion was confirmed with transport ultrasonography in the parasternal long-axis view shortly after departure (Fig. 1). We could not appreciate right ventricular collapse in the subxiphoid view (not shown). Pericardial effusions in adults are commonly classified according to the size, including trivial (seen only in systole), mild (< 10 mm), moderate (10-20 mm), and severe (> 20 mm).[2] The effusion appeared to be larger than 20 mm at its largest point. Although we would have liked to have obtained higher-quality images, at the time of the transport, our focus was only to quantify the size and significance of the effusion. Unfortunately, we could not capture ideal images (Fig. 1).

Approximately 30 to 35 minutes from the receiving facility, the patient's SBP dropped to the low 70s, the NE infusion rate was increased, and a lactated Ringer (LR) bolus was given to correct hypotension. The total volume infused to this point was 4 to 5 L. While attempting to place an arterial line en route, the patient became less arousable, and the procedure was aborted to start a dobutamine infusion. The receiving cardiologist was notified via mobile phone, and

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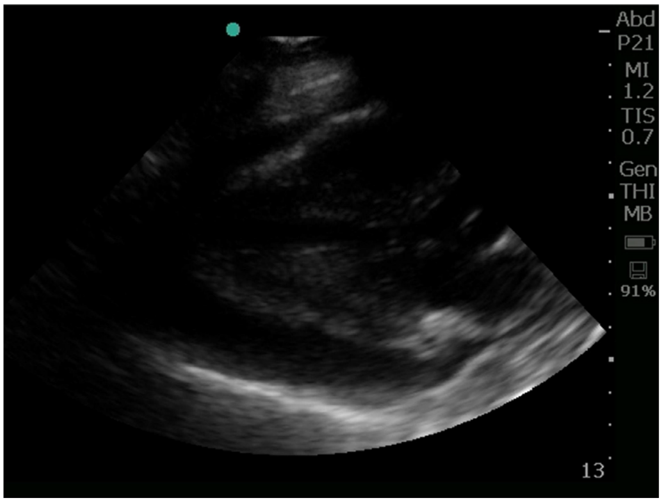

Figure 1. The parasternal long-axis view obtained shortly after departure.

the case was discussed. The crew was advised by the cardiologist to continue the NE infusion and to initiate an epinephrine infusion, instead of the dobutamine. The cardiologist and transport crew agreed that if the patient continued to deteriorate, pericardiocentesis would be necessary.

Over the next several minutes, the patient became increasingly agitated and combative, posing a danger to himself and the crew. Therefore, a small dose of intravenous ketamine (10 mg) was given for mild sedation. A nonrebreather mask was applied at a fraction of inspired oxygen at 100% for denitrogenation/preoxygenation because impending intubation seemed likely. Additionally, nasal capnography had trended from the mid 30s to the low 20s. The decision was made to intubate the patient for airway protection and to provide more reliable capnography to better gauge cardiac output.

The crew was concerned that additional intrathoracic pressures created from the transition to positive-pressure ventilation would further reduce preload and that rapid sequence induction might worsen his hypotension. Therefore, the NE infusion was increased to 30 μg/min. A reduced dose of ketamine (30 mg) followed by an increased dose of rocuronium (150 mg) was given, and the patient was intubated successfully on the first attempt without difficulty.

Endotracheal tube (ETT) placement was confirmed using capnography, bilateral breath sounds, and condensation in the ETT. Bag valve ventilation via the ETT with 15 L oxygen was used to ventilate the patient at a rate of 12 to 18 times per minute. Shortly after intubation, the patient's HR trended from the upper 90s to the mid 40s, $ETCO_2$ dropped to the single digits, and SpO_2 was unattainable.

In an effort to increase the patient's HR, he was given atropine and then epinephrine in 0.5-mg doses. This improved his HR initially. However, his HR then decreased to the mid 20s. At this point, 1 provider performed cardiopulmonary resuscitation while another maneuvered to perform emergent pericardiocentesis. Our clinical judgment was that a HR in the mid 20s, in combination with single-digit $ETCO_2$ readings, warranted chest compressions to support forward cardiac flow. Once the equipment was ready for the emergent pericardiocentesis, compressions were paused briefly, and pericardiocentesis was performed with the subxiphoid approach with a 60-mL syringe connected to a spinal needle. After filling one 60-mL syringe with serous (yellow, straw-colored) fluid, a second 60-mL syringe was applied and another 20 mL aspirated, totaling 80 mL.

Minutes after the pericardiocentesis, the patient's HR improved to the low 100s, and SBP increased to the 180s. By this time, the crew had arrived at the receiving facility. Another ultrasound of the heart was performed by the crew, noting a significant reduction in the pericardial effusion size. The patient was then taken directly to the catheterization laboratory where an additional 120 mL pericardial fluid was removed, and a pericardial drain was placed. The patient had a full recovery. Endocrinology service was consulted during his admission, and the pathologic cause of the pericardial effusion was determined to be either primary adrenal insufficiency or hypothyroidism.

Discussion

Cardiac tamponade results from an accumulation of pericardial fluid and compression of the cardiac chambers, leading to impaired cardiac filling (reduced preload) and hemodynamic compromise. The classic patient presentation of pericardial tamponade includes the Beck triad (ie, jugular venous distention from elevated systemic venous pressure, distant heart sounds, and hypotension).[3] Of these, we noted only hypotension. Distant heart sounds may have been present but are difficult to appreciate in a loud emergency department, ambulance, or aircraft. Regardless, the diagnosis had already been determined by the referring physician and radiologist. Indicators of our patient's poor cardiac output included an altered mental status, agitation, cool extremities, and decreased $ETCO_2$ levels. $ETCO_2$ detection requires ventilation (specifically exhalation), cellular production of CO_2, and pulmonary blood flow for the excretion of CO_2. A decrease in $ETCO_2$ can result from diminished perfusion and subsequent metabolic (lactic) acidosis signaling impaired cardiac output. With complete cardiac standstill, blood movement ceases throughout the pulmonary vasculature and $ETCO_2$ abruptly declines.[4]

One might argue that the pericardiocentesis should have been performed either before the patient's transportation or at any time before the intubation. Given the invasiveness of the pericardiocentesis and limited experience of the available providers in performing the procedure, we were hesitant to perform the procedure until absolutely necessary. Therefore, we elected to continue with volume resuscitation and vasopressor support because hemodynamics were acceptable upon initial contact with our patient.

Volume Resuscitation

Our approach to management was initially focused at overcoming decreased preload by giving intravascular volume. Because the patient had been given 3 L 0.9% normal saline before our care, we elected to give subsequent fluid boluses of LR to limit the potential for hyperchloremic metabolic acidosis. Although the benefit of LR in patients with tamponade physiology might not be well studied, balanced resuscitative fluids have been associated with a decreased incidence of hyperchloremic metabolic acidosis in other adult populations.[5]

Vasopressors

We chose to continue the NE infusion primarily because its mechanism of action supported its use, and it was already infusing. NE stimulates α1, α2, and β1 adrenoceptors.[6] Stimulation of α-adrenoceptor receptors results in vasoconstriction, thereby increasing systolic and diastolic blood pressures. The β1-adrenoceptor effects cause an increased HR and force of contraction.[6] We elected to add the dobutamine infusion after reaching high doses of NE. The rationale for adding this medication was primarily because of its immediate availability, and the emergent need for additional hemodynamic support. However, the cardiologist preferred epinephrine over dobutamine.

Dobutamine is generally considered a relatively selective β1-adrenoreceptor agonist, which is most often used to increase cardiac contractility and augment stroke volume. However, dobutamine has the capacity to stimulate β1, β2, and α1 adrenoceptors.[6] Theoretically, dobutamine may cause a decrease in peripheral vascular resistance either via a reflex withdrawal of sympathetic tone to the vasculature or through stimulation of β2 receptors.[7] Epinephrine stimulates α1, α2, β1, and β2 adrenoceptors. It is a powerful vasoconstrictor, positive inotrope, and a positive chronotrope with vasoconstrictor effects that become more apparent at increased doses.[6] The theoretical rationale for choosing epinephrine over dobutamine is to decrease the likelihood of peripheral vasodilatation. As we were preparing the epinephrine infusion, the patient decompensated. We were forced to intubate and then perform the pericardiocentesis, so it remains unknown whether this would have helped our patient.

Our Approach to Intubation

Dr. Scott Weingart coined the HOP mnemonic for considerations before intubation. This mnemonic allows one to contemplate the conditions of hypotension, oxygenation, and pH before induction and intubation, alternating the approach accordingly.[8] We anticipated and prepared for postintubation hypotension. The addition of positive-pressure ventilation, whether via intubation, continuous positive airway pressure, or BiLevel positive airway pressure, can aggravate hypotension, especially in preload-dependent conditions such as cardiac tamponade or hypovolemia.[9] We chose ketamine as our induction agent given its preferable hemodynamic profile, and we reduced our induction dose in an attempt to maintain the patient's intrinsic catecholamine response. Fluid boluses before intubation and restricting the induction agent to hemodynamically stable drugs, such as ketamine or etomidate at a reduced dose, have been suggested to prevent postintubation hypotension.[10] In addition, we increased the NE infusion, hoping to avoid postintubation hypotension. Perhaps giving additional fluid boluses or starting the epinephrine infusion was warranted; however, at this point, we were prepared to treat the underlying cause and remove the pericardial fluid via pericardiocentesis should further patient deterioration occur.

Causes of Bradycardia After Intubation

Bradycardia or asystole after intubation can occur from several mechanisms including hypoxemia, a strong vagal reflex, suspension laryngoscopy, vagotonic drugs (ie, fentanyl), and insertion of a laryngoscope or ETT causing stimulation of the larynx and trachea.[11,12] We presume our patient's bradycardia was attributed to relative hypoxemia caused by a decreased cardiac output and reduced preload. We briefly tried rectifying this by giving atropine and epinephrine. Although epinephrine 0.5 mg is not considered typical management for bradycardia, an ampule is readily available, and we hoped to provide brief support before eventually moving forward with the pericardiocentesis and attempting to fix the underlying problem.

Performing the Pericardiocentesis

Several approaches to emergent pericardiocentesis have been described. Ultrasound-guided pericardiocentesis has been reported to improve safety and success rates and is the preferred technique for performing pericardiocentesis.[1,13,14] An electrocardiographic electrode attached to a needle via an alligator clip followed by watching for ST elevation has also been suggested.[1] For those not accustomed to these techniques, the addition of an ultrasound machine or alligator clip might complicate an already stressful situation. Additionally, older ultrasound machines can take 30 seconds or longer to boot—time not available during cardiac arrest. Although our crew was skilled at obtaining ultrasonic views of the heart, we had not trained for ultrasound-guided pericardiocentesis nor was an alligator clip readily available. In this situation, we reverted to our prior training and used a blind subxiphoid approach, with a spinal needle directed toward the left midclavicular point at a 30° inclination. In simpler terms, the needle was directly beneath the xiphoid process and pointed toward the left scapula.

The textbook approach to blind subxiphoid pericardiocentesis is summarized as follows: 1) begin just below the xiphoid process and the left costal margin, 2) insert a spinal needle directed toward the left midclavicular point at a 30° inclination with the stylet in place to prevent dermal tissue from plugging the needle (if a needle with a stylet is not available, an alternative technique is to nick the skin with a scalpel before inserting the needle), and 3) once the needle has punctured the skin, remove the stylet and attach a 3-way stopcock and a 20-mL syringe. Then, advance the needle toward the left shoulder while aspirating continuously.[1] We used a 60-mL syringe attached to a spinal needle, omitted the 3-way stopcock, and forgot to first puncture the skin with the stylet or nick the skin with a scalpel. Once we filled our 60-mL syringe with pericardial fluid, we attached a second 60-mL syringe and aspirated as much as possible. Our technique was certainly not considered textbook. The most difficult part of the procedure was making the decision to perform it.

There are many potential complications of pericardiocentesis including mortality, cardiac arrest, cardiac perforation, cardiac chamber laceration, injury to an intercostal vessel, pneumothorax, arrhythmias, hepatic injury, pulmonary edema, and infection.[15] By the time we elected to perform the procedure, the risk benefit clearly favored emergent pericardiocentesis. At the time, we did not consider pulling over and stopping the ambulance to limit movement. Should the ambulance have encountered a large bump, perforation of the ventricle may have occurred. In retrospect, having the ambulance driver pull to the side of the road seems prudent.

Minutes after the pericardiocentesis, the patient's HR improved to the low 100s and SBPs were in the 180s; this was likely the result of catecholamine infusions, as well as those given during cardiopulmonary resuscitation. We were fortunate enough to have arrived at the accepting facility shortly after performing the pericardiocentesis. Although performing the procedure had likely rectified the underlying problem, at least temporarily, we were hesitant to down titrate our vasopressors and left this decision to the accepting facility.

It is important to note that our patient's tamponade physiology (pericardial effusion) might differ from what other transport crews encounter. There is controversy as to whether emergent pericardiocentesis is warranted in cases such as proximal aortic dissection in which rapid and aggressive drainage of pericardial blood might precipitate a worsening leak from the aorta into the pericardium.[14,16]

Supplementary materials

Supplementary material associated with this article can be found in the online version at https://doi.org/10.1016/j.amj.2019.07.008.

References

1. Fitch MT, Nicks BA, Pariyadath M, McGinnis HD, Manthey DE. Videos in clinical medicine. Emergency pericardiocentesis. *N Engl J Med*. 2012;366:e17.
2. Pérez-Casares A, Cesar S, Brunet-Garcia L, Sanchez-de-Toledo J. Echocardiographic evaluation of pericardial effusion and cardiac tamponade. *Front Pediatr*. 2017;5:79.
3. Beck CS. Two cardiac compression triads. *J Am Med Assoc*. 1935;104:714–716.
4. Aminiahidashti H, Shafiee S, Zamani Kiasari A, Sazgar M. Applications of end-tidal carbon dioxide (ETCO2) monitoring in emergency department: a narrative review. *Emerg (Tehran)*. 2018;6:e5.
5. Sethi M, Owyang CG, Meyers C, Parekh R, Shah KH, Manini AF. Choice of resuscitative fluids and mortality in emergency department patients with sepsis. *Am J Emerg Med*. 2018;36:625–629.
6. Kee VR. Hemodynamic pharmacology of intravenous vasopressors. *Crit Care Nurse*. 2003;23:79–82.
7. Ruffolo RR Jr. The pharmacology of dobutamine. *Am J Med Sci*. 1987;294:244–248.
8. Weingart S. The HOP Mnemonic and AirwayWorld.com Next Week. *EMCrit Blog*. June 21, 2012. https://emcrit.org/emcrit/hop-mnemonic/. Accessed March 23, 2019.
9. Shafi S, Gentilello L. Pre-hospital endotracheal intubation and positive pressure ventilation is associated with hypotension and decreased survival in hypovolemic trauma patients: an analysis of the national trauma data bank. *J Trauma*. 2005;59:1140–1145. discussion 1145-1147.
10. Althunayyan SM. Shock index as a predictor of post-intubation hypotension and cardiac arrest; a review of the current evidence. *Bull Emerg Trauma*. 2019;7:21–27.
11. Jones P, Dauger S, Peters MJ. Bradycardia during critical care intubation: mechanisms, significance and atropine. *Arch Dis Child*. 2012;97:139–144.
12. Glassman SH, Green MS, Brodsky M. Asystole following reintubation during suspension laryngoscopy. *Case Rep Anesthesiol*. 2012;2012:916306.

13. Chetrit M, Lipes J, Mardigyan V. A practical approach to pericardiocentesis with periprocedural use of ultrasound training initiative. *Can J Cardiol*. 2018;34:1229–1232.
14. Pavie A, Brucato A, Ristić AD, et al. 2015 ESC guidelines for the diagnosis and management of pericardial diseases: the Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC)Endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J*. 2015;36:2921–2964.
15. Kumar R, Sinha A, Lin MJ, et al. Complications of pericardiocentesis: a clinical synopsis. *Int J Crit Illn Inj Sci*. 2015;5:206–212.
16. Isselbacher EM, Cigarroa JE, Eagle KA. Cardiac tamponade complicating proximal aortic dissection. Is pericardiocentesis harmful? *Circulation*. 1994;90:2375–2378.