Case Studies 3 & 4

Kimberley Roberts

Wright State University

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Case Study 3

1. What is the differential diagnosis of this patient's clinical deterioration and why?

Acute respiratory distress syndrome (ARDS) is the likely differential diagnosis based on the clinical picture and rapid respiratory deterioration depicted in this scenario. Additional differential diagnoses to further rule out include; acute lung injury (ALI), pulmonary embolus (PE), atelectasis, pneumothorax (PTX), and pneumonia. The American-European Consensus Conference (AECC) defines ARDS as a ratio of partial pressure of arterial oxygen to the fraction of inspired oxygen (PaO2/FIO2) less than 200 mmHg with bilateral infiltrates on a chest-x-ray (CXR), acute onset, pulmonary artery wedge pressure (PAWP) less than 18 mmHg or no evidence of left atrial hypertension (The ARDS Definition Task Force, 2012). A PaO2/FIO2 ratio of less than 300 mmHg indicates ALI, a less severe hypoxemia which may progress to ARDS. The Berlin definition of ARDS has since been constructed as a result of inconsistencies evolved over time and research revealing data supporting other factors. The Berlin definition of ARDS is described as an onset of respiratory symptoms within one week of insult, bilateral opacities noted on a CXR, respiratory failure not explained by fluid overload or cardiac dysfunction, and a PaO2/FIO2 ratio of less than 200 mm Hg with a positive end expiratory pressure (PEEP) of less than five cm of H2O (The ARDS Definition Task Force, 2012). The definition also clearly delineates mild, moderate, and severe ARDS. In this scenario, the patients peak airway pressures rising to 60 cm from 20 cm of H2O is suggestive of either an airway obstruction or decreased compliance and further determined by changes in plateau pressures. However, the significant decrease in PO2 to 39 mm Hg implies a ventilation/perfusion mismatch and a right to left shunt often seen with ARDS. More so, the calculated PaO2/FIO2 ratio initially

is 147.5 mmHg and progresses to 97.5 mmHg on 40% FIO2 granted no changes had been made with ventilator settings after first arterial blood gas (ABG) was obtained. ARDS is a lung compliance problem stemming from an insult causing increased permeability of the pulmonary vasculature developing into interstitial and alveolar edema. The alveoli are adequately perfused with inadequate ventilation thus causing an intrapulmonary shunt. Eventually if not corrected fibrotic changes in the lung parenchyma ensues. Direct and indirect lung insults consist of infectious agents, aspiration, inhalation injury, sepsis, blood transfusion ALI (TRALI), multiple fractures, and pulmonary contusions (Li et al., 2011). Presenting signs and symptoms in this case include; tachypnea, increased minute ventilation, agitation, initial right lower lobe infiltrate progressing to diffuse airspace pattern, and early respiratory alkalosis. The diffuse airspace pattern on the CXR and onset of symptoms described in this scenario likely depicts the exudative phase of ARDS. Cardiogenic pulmonary edema can be ruled out with an echo showing no evidence of left atrial hypertension, or a swan ganz catheter revealing normal pulmonary artery wedge pressures (PAWP). No cardiomegaly on a CXR with bilateral infiltrates is sometimes a clue, but not always indicative of ARDS. ARDS is highly probable due to the patient's presenting injuries of pulmonary contusions, tension pneumothorax, hemothorax, multiple orthopedic fractures leading to hemodynamic instability requiring immediate interventions (chest tube insertion, intubation and aggressive resuscitation).

Although a PE is possible given this patient has sustained multiple orthopedic fractures and is a trauma victim, the increased PIP suggests another process is occurring. With a PE, acute respiratory deterioration often occurs (hypoxemia); however the PIP would remain unchanged (no increase or decrease). The PaO2 levels from a PE generally respond to increased levels of oxygen (FIO2) as this is a perfusion problem. An increased amount of dead space occurs

secondary to lack of perfusion caused with a PE. Ventilation does not pose a problem in the occurrence of a PE. Also, there are very little changes viewed on a CXR indicating another reason for hypoxemia. In this scenario, the CXR showed diffuse airspace patterns signifying another diagnosis is likely (Chesnutt, Prendergast, & Tavan, 2013).

Atelectasis is airway collapse occurring secondarily to a number of issues. One major cause of this is improper head of bed positioning. The head of bed should always be at thirty degrees unless contraindicated (i.e. spinal injury, post procedure). This patient's bed initially was probably flat due to unknown spinal clearance after a motorcycle accident. Overtime this can increase risk for ventilator assisted pneumonia and alveoli collapse (atelectasis). Atelectasis commonly caused by poor pain relief may lead to insufficient coughing and deep breathing. Inadequate pain control especially in the circumstance of multiple rib fractures and pulmonary contusions can limit pulmonary toilet activities. Atelectasis is a likely contributory factor of ARDS in this scenario (Mercat et al., 2008).

Pneumonia is a possible diagnosis that may be inferred from CXR results of a right lower lobe infiltrate. This causative lung insult can progress to ARDS without proper diagnosis and treatment. Pneumonia would need to be further ruled out in this case as there is no further suggestive evidence other than a CXR pointing to this diagnosis. Unilateral infiltrates causes include; pneumonia, aspiration of gastric contents, pulmonary edema, pleural effusion, and atelectasis. Aspiration can cause pneumonia from gastric bacteria entering the lungs and causing an inflammatory response. Aspiration is a possibility in this case as the patient was minimally responsive at the scene and was intubated in the ED for airway protection. Ventilator associated pneumonia usually occurs after forty-eight hours of intubation and is unlikely for this patient since his respiratory deterioration started within twelve hours of admission (Chesnutt,

Prendergast, & Tavan, 2013).

2. What are the risk factors that put this patient at risk for ARDS? Provide rationale.

This patient is at risk for ARDS secondary to rib fractures, tension pneumothorax, pulmonary contusions, advanced age, inadequate ventilator settings, multiple blood transfusions and orthopedic fractures. In this scenario, rib fractures are the likely cause of pulmonary contusions, hemothorax, and tension pneumothorax. Rib fractures are painful and tend to become an issue related to inadequate pulmonary toilet. Pulmonary contusions generally blossom around forty eight to seventy two hours and can hinder ventilation. During this peak, the patient is at increased risk for atelectasis, pneumonia, and ALI leading to ARDS. Diminished physiologic response occurs with advanced age greater than sixty years old, increasing risk for ARDS among polytrauma victims (Chesnutt, Prendergast, & Tavan, 2013). The numerous orthopedic fractures sustained as a result of the accident place this patient at an elevated risk for fat emboli, PE and prolonged immobility potentially leading to further respiratory compromise.

Throughout mechanical ventilation, appropriate ventilator settings should be maintained. In this scenario, the ventilator settings are inadequate to maintain appropriate ventilation, specifically minute ventilation. This patient is likely sedated post operatively and would require a higher respiratory rate to improve oxygenation and gas exchange. Also, the set tidal volume of 90 ml is substantially low. Normal tidal volumes calculated with ideal body weight are to be maintained between six to eight milliliters per kilogram. Unless the patient weighs 15 kg, this tidal volume is unacceptable potentially leading to fatal consequences if not corrected immediately. Inadequate tidal volumes and low respiratory rates increased the patient's risk for ARDS significantly secondary to ineffective ventilation and other contributing factors (Mercat et al., 2008).

Another risk factor involves the multiple blood transfusions delivered. Blood transfusions of more than fifteen units in a twenty four hour period increase the risk for TRALI which can lead to ARDS (Li et al., 2011). In this scenario, the patient received a total of twenty one units of blood products. TRALI results in an inflammatory process from release of cytokines and inflammatory mediators causing pulmonary vasculature permeability perpetuating fluid accumulation within the alveoli. TRALI generally occurs within the first six hours of receiving a blood transfusion. Symptoms of dyspnea, and increased oxygen requirements related to hypoxemia during or within hours after blood transfusion often are early indications. Further workup with a CXR may reveal diffuse pulmonary infiltrates. In contrast from other lung insults, TRALI usually resolves within a week with early detection and treatment (Li et al., 2011).

3. What are specific considerations for managing an elderly trauma victim? Provide rationale.

Considerations include the fragility of advanced age. Elderly individuals take a longer time to recover from multi-trauma. Elderly trauma victims have a six times higher mortality rate when compared to younger individuals (Dimitriou, Calori, & Giannoudis, 2011). Physiological changes with aging and co-morbidities significantly affect trauma mortality rates in the elderly population (Schroeder, Weiss, & Moschieff, 2009). Co-morbidities must be included in the treatment regimen as this may present future problems if not addressed. Elderly trauma victims may not have the same presenting symptoms as a younger individual. This can skew the clinical picture and often poses an obstacle for many healthcare professionals. In regards to not having a clear presentation, elderly may not respond as well to treatments as others. An example of this is when younger individuals may tolerate low hemoglobin levels, whereas severe anemia can cause serious compromise in the elderly. Gathering information about past medical history (PMH) and

current pharmacological treatment can be extremely helpful when treating elderly trauma victims. Although PMH cannot always be easily accessible, understanding general aging physiology is important when considering appropriate treatments. Volume of distribution changes during the aging process and can effect different drug actions good or bad. Therefore, careful consideration is necessary prior to prescribing medications with elderly individuals due to an increased risk for drug accumulation potentially leading to toxicities (Dimitriou, Calori, & Giannoudis, 2011).

Elderly individuals have limited physiologic reserve, understanding this may further guide more aggressive treatment and identify early prevention strategies. Thermoregulation responses decrease with aging and can potentiate hypercoagulable states when combined with a traumatic injury. Specifically, emphasis on avoiding hypothermia immediately after the traumatic injury, thus decreasing coagulopathy in the elderly population is critical. Preventing hypothermia by applying warm blankets and/or bare hugger, warming intravenous fluids and blood products during administration, and increasing room temperatures are basic ways to increase the patient's core temperature. Applying supplemental oxygen initially to prevent hypoxemia as elders tend to have a lower threshold for hypoxemia may be beneficial. Using advanced trauma life support (ATLS) core interventions should still be the focus with extra attention focusing on age related concerns (Dimitriou, Calori, & Giannoudis, 2011). Prevention of trauma triad of death (hypothermia, hypercoagulable, and acidosis) is included with ATLS and may be difficult to control in the geriatric population.

4. How would you manage this patient's hypoxemia? Provide rationale.

Initial management begins with identifying and treating the underlying cause. To start, increasing the FIO2 to 100% and wean as tolerated to maintain pulse oximetry above 88 to

95% and PaO2 levels between 60 to 80 mmHg. A shortened time period of FIO2 greater than 60% remains important to prevent oxygen toxicity (Chesnutt, Prendergast, & Tavan, 2013). In ARDS the oxygen saturation may be refractory to increased levels of oxygen at any point during the different stages, thus additional ventilatory interventions are often necessary (Petrucci & Iacovelli, 2007). The next step would be to maximize lung ventilation by optimizing adequate tidal volumes at six milliliters per kilogram utilizing this patient's ideal body weight. Research has shown that maintaining tidal volumes on the lower side of normal (six to eight ml/kg) results in less volutrama (Brower et al., 2000). Once correcting the tidal volumes, then increasing PEEP (above five cm H2O) will enhance gas exchange within alveoli and assist with oxygenation. Recruitment maneuvers (increasing PEEP to a higher amount with inspiratory holds) prior to increasing the PEEP help prevent recurrent collapsing of alveoli, and improve ventilation (Mercat et al., 2008). Higher PEEP levels have shown to augment alveoli patency and total lung compliance (Mercat et al., 2008). Avoidance of high plateau pressures greater than 30 cm H2O remains important with increases in PEEP to reduce intrapulmonary shunting. Throughout ventilator changes, ensuring adequate sedation can decrease asynchrony and improve ventilation especially if the patient becomes agitated. Neuromuscular blockade may be indicated if no improvement is noted with prior interventions and is a grade C intervention (Yegeswaran & Murugan, 2011). Chemical paralysis will allow for complete control of breathing, thus eliminating asynchrony, fighting the ventilator, increased work of breathing and will assist with ventilation. Sedation must be adequate prior to initiating neuromuscular blockade. Train of four testing can help reach goals directed for the neuromuscular blockade agent. Once asynchrony/spontaneous respirations are eliminated, the neuromuscular blockade drip should remain at that rate and slowly weaned off when ventilation improvements are met.

Ventilator changes may also include different modes of ventilation such as; pressure control ventilation (PCV) or airway pressure release ventilation (APRV). PCV can maintain a constant pressure during inspiration and expiration, possibly improving ventilation of the lungs and limit peak airway pressures (Lanken, 2005). APRV essentially helps inverse the inspiratory/expiratory ratio to allow for longer inspiration time with at a constant pressure in hopes of opening the stiff alveoli and improving gas exchange (Lanken, 2005). Lung protective ventilation is crucial during the treatment of ARDS. Lung protective measure preservation with PIP less than 30 mmHg and tidal volumes between six to eight ml/kg proves beneficial during the treatment of ARDS (Mercat et al., 2008).

Early proning has come to the forefront in recent studies to improve outcomes of ARDS. Prompt prone positioning in a recent randomized control trial showed a significant decrease in twenty-eight and ninety-day mortality (16% in the prone group versus 32.8% in the supine group) with severe ARDS (Guerin et al., 2013). Proning this patient may be beneficial to improve oxygenation and ventilation since this is likely the early phase of ARDS. If no changes are obtained from direct interventions of increasing PEEP, increasing tidal volumes, prone positioning should be considered. Lastly, if the patient does not respond to previous mentioned interventions (no changes in PaO2, continues to decompensate and hemodynamically unstable despite efforts), starting inhaled nitric oxide may be used as a last stitch attempt to vasodilate the pulmonary artery tree in hopes of improving oxygen delivery.

Throughout these interventions to improve ventilation, treating the underlying cause is essential. Maintaining adequate pain control for rib fractures will promote healing and limit agitation if secondary to pain. Optimizing fluid status to a euvolemic state will help decrease the risk for hypervolemia potentially worsening pulmonary edema (Murphy et al., 2009). Preventing

further damage to the lungs will also be crucial during course of treatment. The injuries sustained in this scenario (multiple orthopedic fractures) combined with initial coagulopathy and potential prolonged immobilization places him in a high risk category for venous thromboembolism (VTE). All the components of Virchows triad (venous stasis, endothelial injury, and hypercoagulability) are encountered in this scenario. VTE prevention with anticoagulants (subcutaneous heparin or low molecular weight heparin) and sequential compression devices should begin as soon as possible unless contraindicated (Schroeder, Weiss, & Moschieff, 2009). Early nutritional support is considered beneficial to decrease the catabolic state if applicable (Doig, Heighes, Sweetman, & Davies, 2009). Enteral nutrition is indicated if the patient has a functional gut without other contraindications and may prevent gastric ulcer occurrence and reduce risk of bacteria formation in the stomach.

5. What are the problems associated with PEEP?

High PEEP levels can cause barotrauma, tension pneumothorax, and hemodynamic compromise. Barotrauma results from too much PEEP causing hyperinflation of alveoli (Mercat et al., 2008). Tension pneumothorax may develop from barotrauma if not corrected causing this medical emergency. Hemodynamic compromise occurs from rising intrathoracic pressures from high levels of PEEP, thus reducing ventricular preload and cardiac output potentially resulting in hypotension (Girgis, Hamed, Khater, & Kacmarek, 2006). Contrastingly, low levels of PEEP may lead to inadequate ventilation especially in patients with pre-existing lung diseases.

6. What is the mortality rate associated with ARDS?

Although mortality rates linked with ARDS have decreased over the decades, mortality still poses a problem. Crude mortality rates from a recent cohort study revealed 26% during 2004 to 2005 compared to 35% from 1996 to 1997 (Erickson, Martin, Davis, Matthay, & Eisner,

2009). The trending decline in mortality rates with ARDS is likely due to improvement of early detection and interventions based on reported studies and guidelines for treatment. A systematic review showed a significant decrease in twenty eight-day mortality with lung protective measures (CI 0.61-0.88) (Petrucci & Iacovelli, 2007). Also, the ARDS- net trial studied lower tidal volumes of six ml/kg versus higher tidal volumes of twelve ml/kg and showed significant reduced mortality rates (22%) in the group treated with lower tidal volumes (Brower et al., 2000). Another caveat to mortality rates are contributory mechanisms (sepsis, blood transfusions, surgery, inflammatory processes, etc.) potentially leading to higher morbidity and mortality. Once an organ system is failing, morbidity and mortality risk increases. As multiple organ systems fail (kidneys, liver, heart), risk of mortality substantially rises. Sepsis can be a huge offender of this if not controlled or treated in a timely manner leading to multiple organ dysfunction. Co-morbidities and age also play a role when analyzing mortality rates. Patients with pre-existing conditions are becoming increasingly common and often potentiate recovery from organ dysfunction thus contributing to mortality risk. Though mortality rates associated with ARDS have declined over the decades from research providing new treatment options and early prevention, ARDS still remains a multi-factorial syndrome that requires aggressive treatment to prevent further deterioration leading to late stage disease where outcomes remain poor.

Case Study 4

A 68 year old male presented to the emergency room with shortness of breath and complaints of chest pain. He is clenching his chest and describing his chest pain as heaviness and pressure. He denies pain radiation and states the pain started a couple of hours ago and woke him up from sleeping. Upon arrival his vital signs: T. 98.7 ˚F, P. 115bpm, R. 26bpm, BP. 115/74, SpO2. 96% on room air. His systolic blood pressure decreased to 95/74 during inspiration when obtaining the BP manually. He is pale and diaphoretic. Cardiac examination revealed muffled heart sounds without rubs, clicks or gallops. Lungs are clear to auscultation. Initial electrocardiogram (ECG) revealed electrical alternans. The patient reports a history of hypertension, diabetes and atrial fibrillation. The patient's wife revealed that the patient had a cardiac ablation yesterday morning for re-occurring atrial fibrillation and has been sleeping since he got home. Thirty minutes after arrival during workup, the patient states "I'm nauseous and don't feel so good". His blood pressure plummets to 70/50, respirations increase to 35bpm and pulse increases to 140bpm. Two large bore intravenous accesses were placed and the patient was subsequently given intravenous fluids as a bolus for resuscitation efforts.

1. What differential diagnoses are associated with this patient's clinical picture?

Differential diagnoses to consider include; pericardial tamponade, acute myocardial infarction, and pulmonary embolus. Chest pain can point to several diagnoses and necessitates further workup. Any of these differentials are likely based on the clinical picture depicted in this scenario, all of which require prompt treatment once established as the official diagnosis.

Pericardial tamponade stems from a rapid accumulation of fluid in the pericardial sac characterized by an intrapericardial pressure greater than 15 mmHg (Bashore, Granger, Hranitzky, & Patel, 2013). The influx of fluid reduces diastolic filling and venous return in the

heart leading to hemodynamic compromise if not corrected promptly. Decreased cardiac output proceeds as fluid accumulates compressing the heart. Blood pressure may be affected as cardiac output diminishes. Specifically pulses paradoxus can be observed during inspiration as negative intrathoracic pressure rises further impeding left ventricular stroke volume. The rapidity of effusion accumulation within the pericardium determines the physiologic response. Slow accumulation often seen with chronic effusions may be tolerated and not alter hemodynamics. Risk factors include malignancy, pericarditis, trauma, and cardiac surgery. The probability of pericardial tamponade is much higher given this clinical picture versus other differentials from the history obtained about recent cardiac ablation and symptomology. Atheroablative therapy has a higher incidence of cardiac tamponade compared with other cardiac surgeries (Hsu et al., 2005).

Acute myocardial infarction (AMI) is always a concern with complaints of chest pain and requires ruling out as a differential diagnosis. Chest pain radiation to the jaw, arm, and shoulders are specific associated symptoms frequently characteristic of an AMI. The treatment is different for AMI and could subsequently be detrimental if ignored. AMI risk increases with obtained PMH in this case of hypertension, atrial fibrillation, and diabetes. Hypotension described in this scenario can be a sign of right ventricular infarction (Bashore, Granger, Hranitzky, & Patel, 2013). A right sided ECG would be helpful to rule this out during resuscitation efforts and eliminate this as the diagnosis. The lack of ST changes on initial ECG and presenting symptomology suggests a different diagnosis and is unlikely an AMI. A full cardiac workup would further eliminate this diagnosis.

Pulmonary embolus (PE) may be considered as a differential to rule out with his symptoms of acute onset of chest pain and history of atrial fibrillation. Pulsus paradoxus can be

observed with a massive PE as result of increased intrathoracic pressure (Chesnutt, Prendergast, & Tavan, 2013). Additional PE workup is necessary if suspected. A chest computed tomography (CT) with contrast, transesophageal echocardiogram (TEE), and transthoracic echocardiogram may aid in determining a PE. Other diagnostic testing should transpire first prior to ordering a CT due to radiographic contrast exposure. Given this patient's symptomology and lack of hypoxemia, other idealistic diagnoses would prevail. More importantly, quickly identifying the problem causing hemodynamic compromise remains priority.

2. Describe the clinical findings associated with cardiac tamponade.

The three components of becks triad; hypotension, jugular venous distension and distant heart sounds are signs suggestive of cardiac tamponade (Hoit, 2011). Dullness with auscultation of lungs under the left scapula also known as Ewart's sign may be observed with assessment. Dyspnea on exertion, tachypnea, and cough with clear lung fields are common respiratory symptoms associated with cardiac tamponade. Pericardial dullness noted with percussion and auscultated pericardial friction rub may be present. Hypotension is indicative of worsened hemodynamic compromise and considered a late symptom. Pulses paradoxus (decrease in SBP with inspiration by 10 mm Hg) has 82% specificity with cardiac tamponade (Synovitz & Brown, 2011). This change in systolic blood pressure (SBP) may initially be noticed when feeling for a radial or brachial pulse noting an absence of pulse with inspiration thus triggering further manual blood pressure assessment to note this abnormality (Hoit, 2011).

3. What tests will assist in diagnosing this patient? Give rationale

Several diagnostic tests are essential for prompt diagnosis. Complaints of chest pain warrant an initial ECG to reveal any ST changes. If pericardial tamponade develops in response to pericarditis, probable diffuse ST segment changes displayed on an ECG appear. Typically, an

ECG showing electrical alternans indicates cardiac tamponade with sizeable pericardial fluid accumulation (Maisch et al., 2004). ECG results revealing bradycardia is generally observed in end stage tamponade potentially leading to cardiac arrest. Regardless of positive or negative ECG findings, further diagnostic test examination is necessary. Preliminary laboratory tests are essential to provide baseline results for interpretation and may guide treatment. Labs to obtain include; complete blood count (CBC), renal panel, coagulation studies (PT/INR & PTT), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and cardiac enzymes (CK-MB and troponin). The CBC may eliminate possible pericarditis related to infectious processes with normal white blood cell count and bands. Hemoglobin and hematocrit levels can help determine if anemia is present and exclude blood loss. A renal panel may not lead to the actual diagnosis, but is beneficial for baseline analysis of kidney function and electrolytes prior to proceeding with additional testing and treatment. Coagulation values are important to consider with any surgical or procedural intervention as elevation of levels may increase risk for bleeding. Cardiac enzymes can be valuable information in distinguishing the cause of chest pain. Significant elevations in troponin levels suggest myocardial injury and may assist with differentiation of diagnoses. CRP and ESR are inflammatory markers that will help eliminate pericarditis as the cause. Mainly laboratory tests uncover underlying causes. A CXR might expose a water bottle heart silhouette with a chronic pericardial effusion, but will often not show specific heart changes in rapid fluid accumulation as seen in tamponade (Maisch et al., 2004). CT and magnetic resonant imaging (MRI) of the heart can detect pericardial effusions. However, time is of the essence with hemodynamic instability and initial pursuance with a CT and/or MRI may be detrimental for the patient portrayed in this scenario. A transthoracic echocardiogram is the preferred diagnostic test with clinical findings suggestive of pericardial tamponade (Hoit, 2011). In the presence of

cardiac tamponade, an echo will likely show the accumulation of fluid in the pericardial sac, collapsed right ventricle in diastole, right atrial systolic collapse and a swinging heart appearance (Maisch et al., 2004). A transesophageal echocardiogram helps with viewing loculated effusions or clots in the pericardium often associated with open cardiothoracic surgery (Hoit, 2011). Right heart catheterization (RHC) is performed to confirm pericardial tamponade. The RHC will expose results of reduced cardiac output, increased right atrial pressures secondary to compression, and attenuated pulmonary artery wedge pressures relatively similar to right atrial pressures (Maisch et al., 2004). Hemodynamic instability may not allow for time to prepare for a RHC in which an echocardiogram is sufficient for diagnostic purposes and immediate intervention.

4. What are the treatment options for this diagnosis?

The treatment depends on the severity of pericardial tamponade from mild to severe. In this scenario the hemodynamic instability depicts a medical emergency and urgent pericardiocentesis is indicated. The European Society of Cardiology guidelines suggest a pericardiocentesis as level of evidence B, class I indication in the presence of cardiac tamponade with hemodynamic compromise (Maisch et al., 2004). A pericardiocentesis for pericardial pressures greater than 20 mmHg without hemodynamic compromise is considered level of evidence B, class IIa indication (Maisch et al., 2004). Aspiration of a small amount of fluid generally improves hemodynamics greatly. Occasionally a needle aspiration is not sufficient as fluid can re-accumulate and a temporary pigtail catheter is left in place to decrease pericardial tamponade reoccurrence. Pericardiocentesis guided by an echocardiogram is safer versus a blind approach and should be considered in most circumstances to prevent further complications. Frequently a RHC is performed simultaneously with a pericardiocentesis to monitor

hemodynamic improvement once effusion is drained (Maisch et al., 2004). Pericardial fluid aspirate should be sent to the laboratory for culture, gram stain, cytology, glucose and hematocrit (Maisch et al., 2004). In the case of suspected malignancy and/or other etiologies as causative factors, pericardial fluid aspiration would require further testing. If unable to remove fluid during a pericardiocentesis, surgical management should be considered. A pericardial window is a surgical procedure done to decompress the pericardial sac and prevent re-accumulation of fluid. A thoracotomy with pericardiostomy is a more invasive surgical approach and should only be considered if all other interventions have failed. Percutaneous balloon pericardiotomy is another alternative to drain fluid accumulation which can be done in the cardiac catheterization lab requiring minimal sedation. This procedure has a 0 to 6% chance of reoccurring fluid accumulation and often is the treatment intervention of choice with effusions caused by malignancies (Bashore, Granger, Hranitzky, & Patel, 2013).

Intravenous fluid resuscitation with 0.9% sodium chloride is indicated in this scenario with hemodynamic instability. Initial fluid bolus challenge of 500 ml to evaluate blood pressure and heart rate response is necessary to determine volume status. Repeating fluid challenge may be necessary if hypotension does not resolve or the SBP drops below 90 mmHg later on in course of treatment. Administering large amounts of volume in a euvolemic or hypervolemic patient can be detrimental especially in the presence of cardiogenic shock. Increasing right heart filling pressures with intravenous fluid can improve cardiac output and hypotension (Bashore, Granger, Hranitzky, & Patel, 2013). Administering fluid is a temporary fix while attempting to correct the actual problem during pericardiocentesis. Provided hemodynamic stability, pain management should also be considered if not relieved with removal of fluid.

5. Does this diagnosis require hospital admission?

This patient would require admission to the cardiac intensive care unit where he can be monitored for further complications and improvement post pericardiocentesis and possible RHC. Continuous cardiac monitoring and frequent vital sign measurement (HR, BP, and O2sat) is essential post procedure. If RHC is performed, frequent assessment includes; monitoring insertion site for hematoma and bruits, checking distal pulses and capillary refill, and evaluating sensation of effected extremity. Furthermore, the patient's primary cardiologist should be notified of the patient's admission and current condition. Provided a temporary pericardial pigtail is left in place post procedure, observation of fluid characteristics and patency are necessary. Generally the pigtail catheter remains in place for up to three days and then removed (Maisch et al., 2004). Throughout the observational period it is important to re-assess for any reoccurring signs and symptoms (chest pain, heaviness, dizziness, hypotension, ECG ST segment changes, and rhythm abnormalities) and encourage the nurse caring for the patient to notify the practitioner immediately if observed. A follow up echocardiogram should be done within 48 to 72 hours to evaluate cardiac function and for reoccurring fluid. Once the pericardial catheter is removed and hemodynamic stability is attained for up to 24 hours the patient can be transferred out of the cardiac intensive care unit to a cardiac floor. Patient and family teaching remains important throughout hospital stay explaining medical course, treatment, limitations, and encouraging questions.

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