EMERGENT MANAGEMENT FOR A CRASHING PATIENT WITH CRITICAL AORTIC STENOSIS IN HUE CARDIOVASCULAR CENTER

Nguyen Tat Dung[1], Ho Anh Binh[1], Duong Dang Hoa[1],
Pham Van Hue[1], Nguyen Huy Thong[1]

DOI: 10.38103/jcmhch.2020.62.14

ABSTRACT

Critical aortic stenosis (AS) is the single most problematic valvular disease we encounter in the ICU. Patients with critical AS have a fixed cardiac output and cannot meaningfully increase cardiac output to meet the physiologic demands of critical illness. In this article, we present the case of a 53-year-old man with severe AS and pulmonary hypertension who developed hypotension during waiting for an elective aortic valve replacement. We discuss practical considerations and emergent treatment for the hemodynamically unstable patient with critical AS.

I. INTRODUCTION

Aortic stenosis is a common condition among older adults that can be associated with dangerous outcomes, due to both the disease itself and its influence on other conditions [1]. The prevalence in the general population is 0.4%, but increases to 9.8% in octogenarians, with an overall prevalence of 2.8% in adults older than 75 years of age [2,3]. Valve replacement, either surgical or catheter directed (ie, transcatheter aortic valve replacement, or TAVR), is the mainstay of treatment for advanced disease.

In a normal adult, the aortic valve area measures 2.6 to 3.5 cm^2. AS becomes hemodynamically significant when aortic valve area approaches <1 cm^2. As the valve becomes tighter, the pressure gradient across the valve increases. A pressure gradient >50 mmHg indicates severe disease [4]. However, it is important to note that a substantial proportion of patients with severe and critical AS have a low gradient, which most frequently results from the decreased stroke volume associated with advanced disease.

Hemodynamically significant AS must be on the differential in the undifferentiated patient presenting with acute pulmonary edema, syncope, or cardiogenic shock, particularly if they are elderly. In addition to the identification of a systolic ejection murmur, bedside echocardiography can help screen patients. In fact, qualitative assessment of the aortic valve from the parasternal long and short axis views has been shown to be 75% sensitive and 93% specific for the diagnosis of severe AS among trained emergency medicine providers [5]. Patients with severe aortic stenosis (AS) often present acutely with decompensated heart failure, cardiogenic shock or cardiac arrest requiring immediate intervention. We reported a case of a

1. Hue Central Hospital

Corresponding author: Nguyen Tat Dung
Email: ngtatdung@hotmail.com
Received: 9/5/2020; **Revised:** 17/5/2020
Accepted: 20/6/2020

hemodynamically unstable patient with critical AS during medical treatment while waiting for an urgent aortic valve replacement.

II. CASE PRESENTATION

A 53-year-old man with a past medical history of severe aortic stenosis, heart failure with reduced ejection fraction, and chronic kidney disease (stage 3 to 4) presents with shortness of breath and bilateral lower extremity edema. ECG showed sinus rhythm with RBBB. Echocardiography showed a critical calcified aortic stenosis (Figure 1) (Aortic annulus, 33mm; aortic valve area, 0.64 cm^2; a peak pressure gradient (PPG) of 137 mmHg; aortic valve velocity, 5.86 m/s) with a hypertrophic and dilated left ventricle and reduced systolic function: EF 34% with PAPs 75 mmHg. It also revealed moderate aortic, mitral and tricuspid valve regurgitation. Left atrial diameter was 50 mm, LVDd of 63 mm, LVDs of 52mm. He was admitted to a cardiology service on a monitored bed. Cardiac enzymes were negative for myocardial infarction and the patient remained hemodynamically stable. Figrue 2 shows the chest X-ray of the patient.

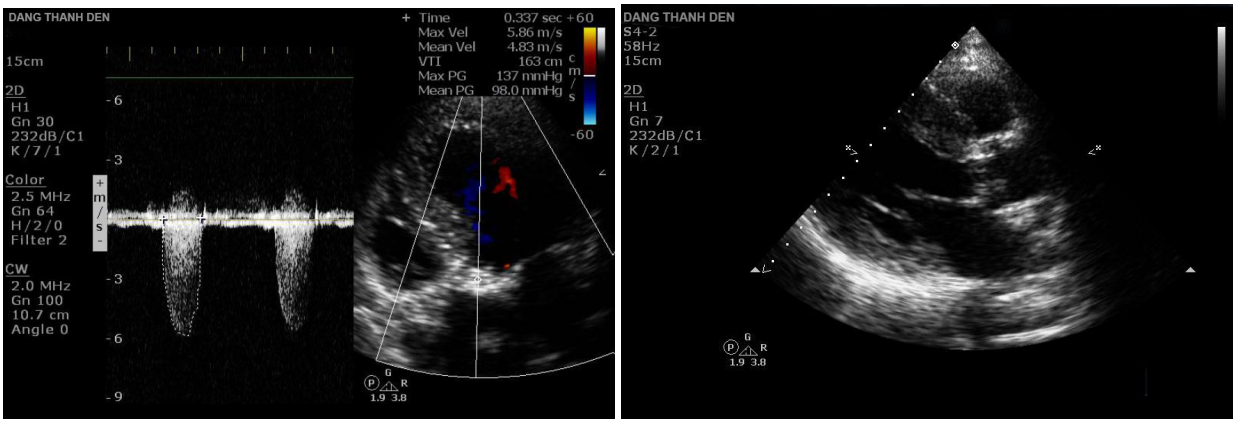

Figure 1: Echocardiography showed a critical calcified aortic stenosis

He experienced no chest pain or dysrhythmia. Coronary angiography revealed no coronary artery disease. Aortic valve replacement surgery was recommended. When the procedure for surgery was described, he became very anxious and refused surgery. His relatives also refused the procedure. He was admitted to the ICU due to suddenly severe chest pain and abruptly multifocal ventricular ectopics. Eight hours after ICU admission the patient's condition deteriorated gradually with pulmonary edema. Echocardiography showed severely reduced left ventricular systolic function with severe global hypokinesis of the left ventricle and a measured ejection fraction of 32%. There was no pericardial effusion.

He was hypotensive 50/35 mmHg. He was intravenously pretreated with phenylephrine 50 mcg and placed on a infusion of 2 mcg/kg/min. Preload with was optimized prior to intubation and etomidate was used for anesthetic induction. Dobutamine of 6 mcg/kg /min was infused.

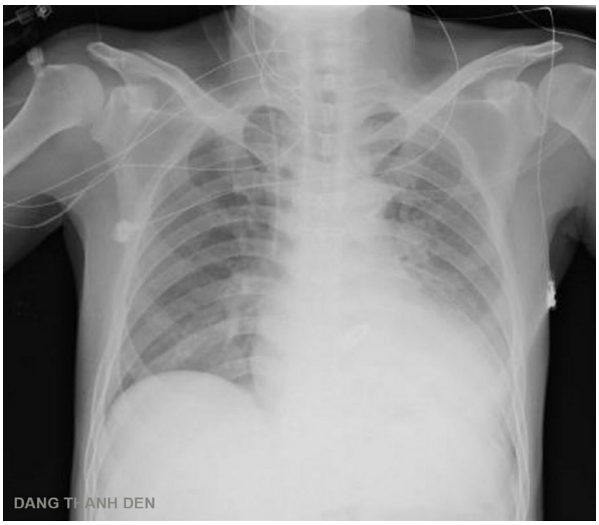

Figure 2: Chest X-ray of the patients

He responded well to medical management and ventilatory support with PEEP of 10 cm H_2O. His logistic EuroSCORE II value was 32.74.

After a written agreement of the patient and his family members, he, who was hemodynamically stable, underwent a full sternotomy, mild hypothermic CPB, and antegrade and retrograde warm cardioplegia. After annular decalcification, a ATS 18- Medtronic valve was implanted. The operation was performed successfully with an Aortic cross-clamp time was 65 minutes and CPB time was 82 minutes.

Significant inotropic support and dialysis were required in the postoperative period, and the patient was discharged from intensive care on day 5.

III. DISCUSSION

3.1. Physiology Primer

Aortic stenosis (AS) obstructs forward blood flow from the left ventricle (LV) to the aorta, causing a systolic gradient to develop between the LV and the aorta. In AS, intracavitary LV pressure must exceed aortic pressure to produce forward flow across the stenotic valve and produce acceptable downstream pressure. There is a geometric progression in the magnitude of the gradient as the valve area narrows. Pressure overload on the LV is compensated by LV hypertrophy. As the disease advances, reduced coronary flow reserve causes myocardial ischemia; in addition, the hypertrophic LV and excessive afterload lead to systolic and diastolic LV dysfunction [6].

Systemic hypotension reduces coronary perfusion pressure, and excessive tachycardia increases myocardial oxygen demand; both contribute to a self-perpetuating cycle of ischemia and cardiogenic shock [7].

The left ventricle hypertrophies in critical AS in response to chronically increased afterload. A stiff, hypertrophied left ventricle requires high filling pressures, and the “atrial kick” of sinus rhythm to fill in diastole. Hypovolemia and supraventricular tachyarrhythmias (eg, atrial fibrillation) dramatically reduce left ventricular preload and are tolerated poorly in this patient population. Excessive tachycardia not only promotes ischemia, but also reduces time spent in diastole for left ventricular filling.

3.2. Vasopressor Management

If the patient is hypotensive and vasopressors are necessary, these agents should be utilized at the lowest dose and for the shortest interval necessary until definitive management can be performed. Vasopressor agents can increase myocardial oxygen demand, leading to decreased contractility, as well as worsen obstruction. Additionally, these agents have the potential to cause dysrhythmias, which can further worsen cardiac function and may lead to cardiovascular collapse [1]. However, it is important to ensure that patients maintain sufficient blood pressure to maintain perfusion, with one study finding that patients with AS had cardiovascular collapse at a much higher mean arterial blood pressure than other critically ill patients (i.e., 51 mm Hg vs 35 mm Hg) [8].

Phenylephrine is the vasopressor of choice in treating the hypotensive patient with AS. Using an agent that solely increases afterload is initially counterintuitive. It is important to recognize that the massive afterload of aortic stenosis is at the level of the aortic valve, with little contribution from the systemic vasculature. As a pure alpha-1 agonist, phenylephrine increases diastolic blood pressure and thus improves coronary perfusion. Phenylephrine also may result in a reflex bradycardia - a favorable pharmacodynamic property for its use in aortic stenosis [9]. Norepinephrine is, similarly, a reasonable choice. Avoid epinephrine as a first line agent given its strong beta - 1 agonism and propensity to promote tachycardia and increase myocardial oxygen demand [7].

3.3. Fluid Management

The tight aortic valve increases left-sided pressures and can lead to pulmonary congestion. However, patients with AS also have diastolic dysfunction and depend highly on preload to fill the left ventricle and maintain cardiac output [7]. Therefore, it is essential to quickly restore the intravascular volume in hypotensive patients [1,10], especially in the hemodynamically unstable patient with critical AS who you are preparing to intubate. Any concern regarding pulmonary congestion is superseded by the need to optimize preload prior to induction. Assuming the patient is not on the tail end of the Frank-Starling curve, temporarily infusing crystalloids to optimize preload and stave off peri-intubation hypotension is a good idea.

3.4. Optimizing preload:

A determination of where the ventricles reside on their pressure stroke volume curves is essential for determining the optimal ventricular filling pressure (central venous or right atrial and left atrial pressures). It is important to note that a given atrial pressure does not correlate with ventricular volume or stroke volume due to alterations in ventricular compliance. Further, in the setting of cardiopulmonary disease there is no correlation between right atrial and left atrial pressures, making it even more challenging to determine the optimal filling pressure for the LV. Administering volume and objectively assessing the response provides some indication of where the ventricles reside on their pressure stroke volume curve. A prompt decrease in heart rate, or increase in venous oxygen saturations or invasive blood pressure immediately following volume administration indicates that preload reserve is present, and that the ventricles are operating on the ascending portion of their pressure stroke volume curves. The lack of a response suggests that the ventricles are residing on the flat portion of their function curves. In this case, preload reserve is exhausted and inotropic and or afterload reducing agents are indicated to improve stroke volume and cardiac output. Additional volume expansion will only increase ventricular filling pressures, increasing myocardial oxygen demand and the formation of pulmonary edema [11].

This is most easily performed with intravenous fluids, though no specific fluid (eg, normal saline, lactated Ringer's, albumin) has been demonstrated to be superior to others in this population. Normal saline should be considered first line, as it is easily available and less expensive than the alternate options.

3.5. Intubation

Patients with critical AS depend on adequate left ventricular preload to maintain cardiac output. Both induction agents and positive pressure ventilation acutely drop left ventricular preload, and place patients with critical AS at risk for peri-intubation hemodynamic collapse. Hemodynamic optimization prior to induction, adequate monitoring, and selection of an induction agent with a favorable hemodynamic profile are the mainstays of safe intubation.

As you prepare to intubate, have push-dose phenylephrine at the bedside (or infusing). Prior to induction, the patient should be preload optimized. Even short periods of systemic hypotension can be devastating and are easily missed by a periodically cycling non-invasive blood pressure cuff. If time allows (eg, the urgent-not-emergent intubation), consider placing an arterial line prior to induction to decrease response time to systemic hypotension.

Etomidate is the RSI induction agent of choice in patients with AS, because it is both hemodynamically stable and comes with a generally favorable side effect profile. Propofol is a profound vasodilator and can acutely drop preload and promote hemodynamic collapse. Ketamine promotes tachycardia - an unfavorable property in patients with AS [7].

3.6. Heart rhythm

Patients with critical AS depend on the atrial

kick of sinus rhythm for diastolic filling [7]. It is also important to ensure a normal heart rate, as patients with severe AS do not tolerate bradycardia well. If bradycardia is present, dobutamine should be strongly considered, as it provides a reduction in afterload, as well as an increase in heart rate and contractility. However, patients with AS can also decompensate with profound tachycardia, as the noncompliant, hypertrophic ventricle requires sufficient time for adequate filling. Moreover, prolonged tachycardia can result in reduced coronary perfusion, further worsening contractility and cardiac output [12,13]. Therefore, if tachycardia is present, rate-reducing agents (e.g., calcium channel blockers, beta-blockers, digoxin) should be considered, while being careful to avoid bradycardia. Unstable patients should be electrically cardioverted. If preparing to intubate a hemodynamically tenuous patient in new atrial fibrillation (or any SVT for that matter), consider cardioversion prior to induction [7].

3.7. Percutaneous aortic balloon dilatation and aortic valve replacement

Aortic Stenosis patients who present with cardiogenic shock can be resuscitated with intravenous fluids, as they are preload dependent. Inotropic medications such as dopamine and dobutamine can also be used in patients in shock, but definitively, these patients will need aortic valve replacement. Severe aortic stenosis patients who are unstable in cardiogenic shock or acute pulmonary edema should be considered for percutaneous aortic balloon dilatation for stabilization, acting as a bridge to valve replacement.

IV. CONCLUSION

The management of the critically ill aortic stenosis patient can be very challenging. These patients need a valve replacement, so consulting cardiothoracic surgery as soon as possible is prudent. Percutaneous aortic balloon dilatation serves as a bridge to surgery. The crashing aortic stenosis patient in cardiogenic shock should be resuscitated with optimized fluids and inotropic medications such as dopamine and dobutamine. Phenylephrine is the vasopressor of choice in treating the hypotensive patient with AS. Avoiding systemic hypotension, maintaining sinus rhythm, and avoiding excessive tachycardia are therefore the cornerstones of resuscitation.

REFERENCES

1. Tewelde SZ, Liu SS, Winters MEJCc. Cardiogenic shock. 2018; 36: 53 - 61.
2. Eveborn GW, Schirmer H, Heggelund G, Lunde P, Rasmussen KJH. The evolving epidemiology of valvular aortic stenosis. the Tromsø study. 2013; 99: 396 - 400.
3. Lindman BR, Clavel M-A, Mathieu P, Iung B, Lancellotti P, Otto CM, et al. Calcific aortic stenosis. 2016; 2: 1 - 28.
4. Kennedy KD, Nishimura RA, Holmes DR, Bailey KRJJotACoC. Natural history of moderate aortic stenosis. 1991; 17: 313 - 319.
5. Alzahrani H, Woo MY, Johnson C, Pageau P, Millington S, Thiruganasambandamoorthy VJCuj. Can severe aortic stenosis be identified by emergency physicians when interpreting a simplified two-view echocardiogram obtained by trained echocardiographers? 2015; 7: 5.
6. Huang J, Chen P, Hu X, Tang J, Fang ZJM. Aortic stenosis complicated by cardiogenic shock treated by transcatheter aortic valve replacement with extracorporeal membrane oxygenation: A case report. 2018; 97.
7. Andreae M, Parekh R. The Crashing Patient with Critical Aortic Stenosis. EMRA - Critical Care 2017; Available from: https://www.emra.org/emresident/article/the - crashing - patient - with - critical - aortic - stenosis/.

8. Brunauer A, Koköfer A, Bataar O, Gradwohl-Matis I, Dankl D, Dünser MWJCC. The arterial blood pressure associated with terminal cardiovascular collapse in critically ill patients: a retrospective cohort study. 2014; 18: 719.
9. Goertz AW, Lindner KH, Schültz W, Schirmer U, Beyer M, Georgieff MJATJotASoA. Influence of phenylephrine bolus administration on left ventricular filling dynamics in patients with coronary artery disease and patients with valvular aortic stenosis. 1994; 18: 49 58.
10. Chen RS, Bivens MJ, Grossman SAJEMC. Diagnosis and management of valvular heart disease in emergency medicine. 2011; 29: 801 - 810.
11. K Chandler H, Kirsch RJCcr. Management of the low cardiac output syndrome following surgery for congenital heart disease. 2016; 12: 107 - 111.
12. Gottlieb M, Long B, Koyfman AJTJoem. Evaluation and management of aortic stenosis for the emergency clinician: an evidence-based review of the literature. 2018; 55: 34 - 41.
13. Tyagaraj K, Gutman DA, Belliveau L, Sadiq A, Bhutada A, Feierman DEJCria. A multidisciplinary approach to anesthetic management of a parturient with severe aortic stenosis. 2015; 2015.