ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 13, Issue, 01, pp.15519-15523, January, 2021

DOI: https://doi.org/10.24941/ijcr.40482.01.2021

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

RESEARCH ARTICLE

BALLOON VALVULOPLASTY OF CONGENITAL PULMONARY VALVULAR STENOSIS IN AN ADULT HOSPITALIZED DUE TO ANASARCA

Nikola Malešević*[1,3] Goran Bokan[2,3] and Saša Lončar[1,3]

[1]Department of Cardiology, University Clinical Centre of the Republic of Srpska, Banja Luka, Bosnia and Herzegovina
[2]Department of Gastroenterology and hepatology, Internal medicine clinic, University Clinical Centre of the Republic of Srpska, Banja Luka, Bosnia and Herzegovina
[3]Faculty of Medicine, University of Banja Luka, Bosnia and Herzegovina

ARTICLE INFO

Article History:
Received 03rd October, 2020
Received in revised form
16th November, 2020
Accepted 11th December, 2020
Published online 30th January, 2021

Key Words:
Pulmonary valvular stenosis,
Congenital heart disease,
Balloon valvuloplasty, Case reports.

ABSTRACT

Congenital stenosis of the pulmonary valve usually occurs in younger people in two forms: isolated and more often, associated with other heart defects. The spectrum of clinical manifestations is variable and usually manifests at a younger age. Cases of previously undiagnosed congenital pulmonary valve stenosis in adults are rare in the literature. This paper presents a case of a patient who was initially hospitalized for anasarca, and in the background there was a congenital stenosis of the pulmonary valve that was treated with balloon valvuloplasty.

Citation: ***Nikola Malešević, Goran Bokan and Saša Lončar.*** "Balloon valvuloplasty of congenital pulmonary valvular stenosis in an adult hospitalized due to anasarca", *International Journal of Current Research*, 13, (01), 15519-15523.

INTRODUCTION

Pulmonary valvular stenosis (PVS) is almost always a congenital heart abnormality and is the result of a commissural fission of thin and pliable cusps. [1] It is one of the most common congenital defects, occurring in about 10% of all congenital heart defects. [2] In a higher percentage of cases, it occurs associated with other heart defects, most often associated with tetralogy of Fallot and transposition of large blood vessels. [3]Also, there is a clear connection between this defect and some genetic syndromes, as well as a higher frequency on certain continents (the largest in Asia). [4] Diagnosis and treatment of congenital pulmonary valvular stenosis is usually associated with childhood, and the disease itself in childhood does not manifest itself with pronounced symptoms. Several cases of congenital PVS in adults who had a much more severe clinical picture and fatal outcome in some instances have been described in the literature. [5] Treatment of PVS in children and adults is either surgical or percutaneous balloon pulmonary valvuloplasty (BPV). [6]In our paper, we present the case of an elderly person who had congenital pulmonary valvular stenosis, previously undiagnosed, and the primary presentation along with anasarca, which was the first reason for hospitalization.

Corresponding author: Nikola Malešević,
Department of Cardiology, University Clinical Centre of the Republic of Srpska, Banja Luka, Bosnia and Herzegovina

Case report

A 60-year-old patient was hospitalized in a local general hospital because of examination of anasarca. He complained that he had been feeling weak and tired in the past two/three months. He also noticed that his legs and arms were swollen. In addition, his medical history reports heart murmurs since childhood. There wasn't any past disease observed. The father died at the age of 55 due to heart disease, without information on the exact cause.

By clinical examination noted: subictericscleras; systolic murmur over the precordium with a punctum maximum over the pulmonary valve; blood pressure 150/90 mmHg; subduedbasal respiratory noise bilaterally; suspected ascites and edema of both extremities. Laboratory findings: normal. Electrocardiogram recording: atrial fibrillation, frequencies around 90/min, IRBBB, secondary changes D3, aVF, V1-V5.X-ray lung and heart: bilateral basal smaller pleural effusions, more pronounced on the right. The cardiovascular silhouette rests on the diaphragm with a wide base. Abdominal ultrasound: pronounced vascular structures of the liver parenchyma; enlarged spleen; free perisplenic and perihepatic fluid. Table I contains the diameters of heart cavities and blood vessels measured by transthoracic echocardiography.

Table 1. Transthoracic echocardiography measurements

Transthoracic echocardiography		Diameters
Aorta	Root	35 mm
	Sinus	41 mm
	Septum	20 mm
	ASCA	36 mm
Right pulmonary artery (RPA)		14 mm
Left pulmonary artery (LPA)		14 mm
Left atrium (LA)		63 mm (47x68 mm)
Right atrium (RA)		73x76 mm
Right ventricle (RV)		41 mm
Left ventricle (LV)		60x43 mm
Intraventricular septum (IVS)		10 mm
Ejection fraction of left ventricle	40-45 %	

The left atrium is larger, no foreign masses or spontaneous contrast are visible. Mitral valve (MV) is fibrously altered, preserved separations and coaptations; mitral regurgitation (MR) 1-2 +. The interatrial septum (IAS) is bulging in LA due to the hemodynamic load of the right heart. LV is of enlarged dimensions, of orderly wall thickness, diffusely hypocontractile at the time of examination with paradoxical movements of the septum, slightly reduced global systolic function, EF 40-45%.

The aorta is regular in dimensions in root, ascending part, arch and visible descending segment, easily to moderately sclerotic walls. Aortic valve (AV) is trifoliate, moderately sclerotic velum, preserved separation and coaptation. RV and RA are more voluminous, tricuspid annular plane systolic excursion (TAPSE) 15mm. PV is sclerotic, with a gradient of 67/34 mmHg, pulmonary regurgitation (PR) 1-2 +. The tricuspidal valve (TV) looks neat, tricuspidal regurgitation (TR) 2-3 +, systolic pressure of right ventricle (SPRV) 85mmHg (70mmHg + central venous pressure-CVP) is recorded. PA is of neat dimensions as are its branches. The pericardium has minimal effusion around the right heart. The vena cava inferior (VCI) is larger in diameter, poorly collapsible. Smaller amounts of pleural effusion are suspected. Initial therapy: furosemide; spironolactone; β-blocker; ACE inhibitor; diet with reduced salt intake. On the eighth day of hospitalization in the local general hospital, the patient was presented to the council of interventional cardiologists and transferred to the University Clinical Centre of the Republic of Srpska for the continuation of diagnostic-therapeutic treatment. Transesophageal echocardiography: cusps of the pulmonary valve altered with sclerotic areas, reduced separation, Doppler registers severe pulmonary stenosis.

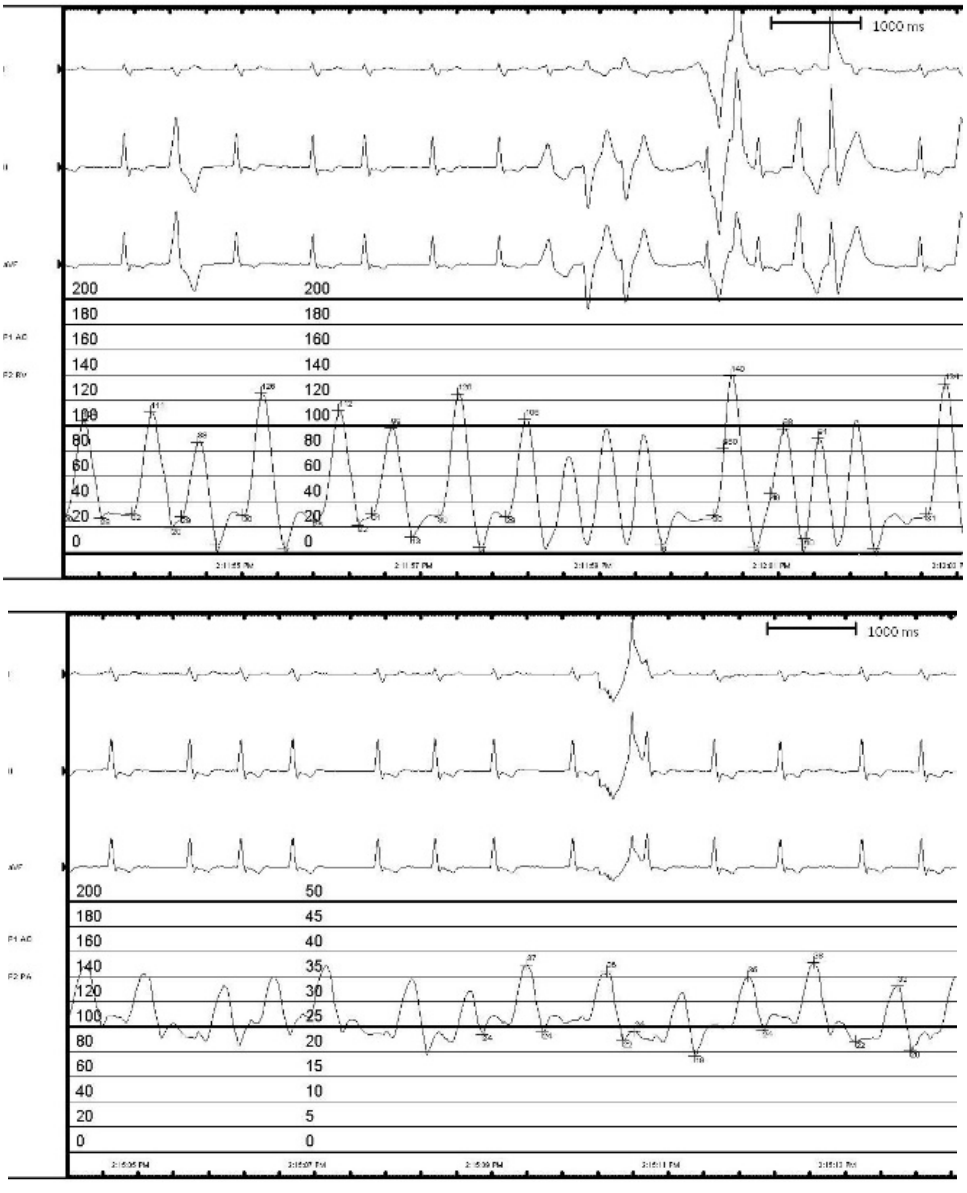

Figure 1. Systolic pressure in RV and PA

Dilated right heart cavities. The cusps of the mitral valve are altered presenting fibrous tracts, hemodynamically insignificant MR is registered. Aortic valve three-leafed, cusps marginally sclerotic modified, trace AR. Computed tomography (CT) of the thorax to exclude pulmonary thromboembolism (PTE): diameters in the axial plane: trees PA about 22mm - ostial margin highly calcified, right branches PA about 21mm, left branches PA about 38mm. Postcontrast, there are certainly no clear defects in the contrast representation along the PA tree or the main and initial parts of the PA branches on both sides that would indicate PTE.

coronarography: severe pulmonary stenosis with a gradient of 85mmHg, pressure in the right ventricle of 112mmHg is observed. Normal findingsconcerning carotid arteries. Balloon valvuloplasty of PVS is advised. Two months after right and left cardiac catheterization, balloon valvuloplasty of the pulmonary valve is performed with a right femoral approach. Initial hemodynamics: systolic pressure in RV 120mmHg, gradient over PV 85mmHg (Figure I). A balloon of 20 mm diameter is inserted on PV and inflated (Figure II). Post procedural hemodynamics: systolic pressure in RV 50mmHg and gradient over PV 15mmHg.

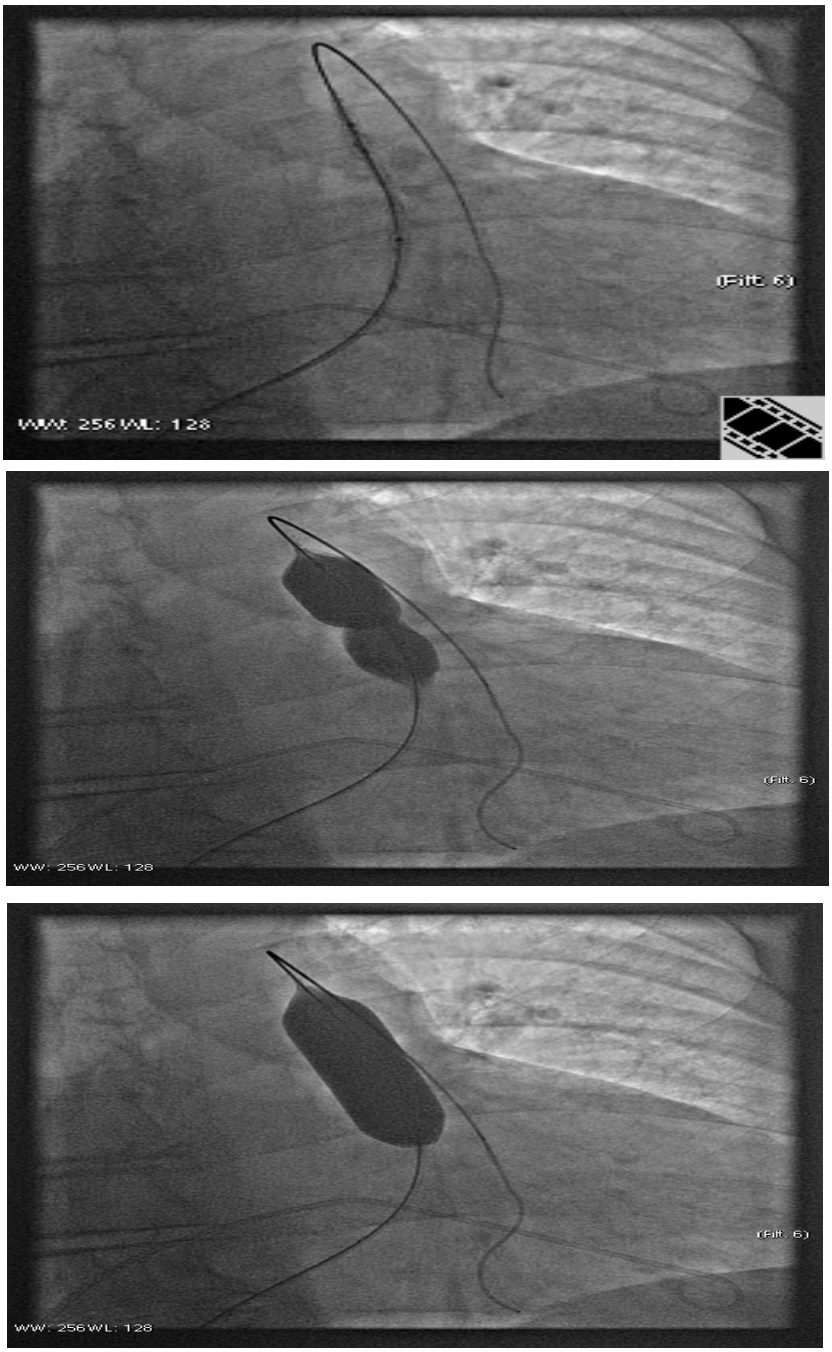

Figure 2. Balloon pulmonary valvuloplasty

Lobar, segmental, subsegmental branches are notanalyzed. Cardiovascular massif globally voluminous with minor pericardial effusion up to about 6 mm thick with DPK and about 8 mm in pericardial recessions. There is no consolidation or infiltration in the lung parenchyma. No pleural effusion. Right and left cardiac catheterization and

The procedure ended without complications (Figure III). First control examination two months after BAV PVS: gradient over PV 25mmHg, TR gradient 35mmHg, TA 150/90 mmHg. Subjectively, the patient reports no problems. Introduced non-vitamin K anatagonist oral anticoagulants (NOAC) due to atrial fibrillation (AF).

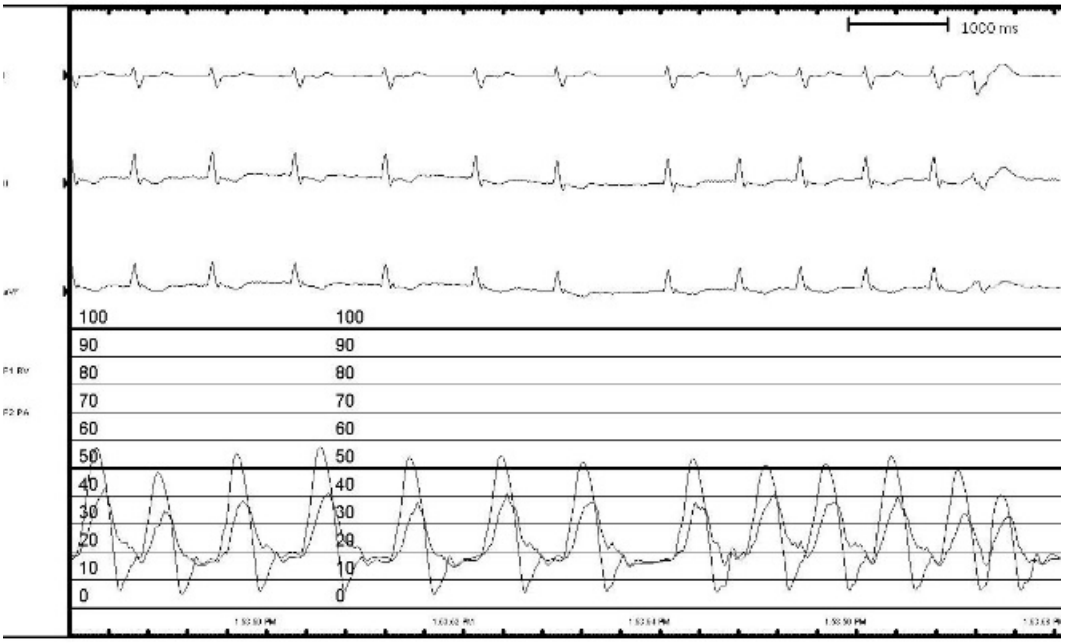

Figure 3. Postprocedural hemodynamics

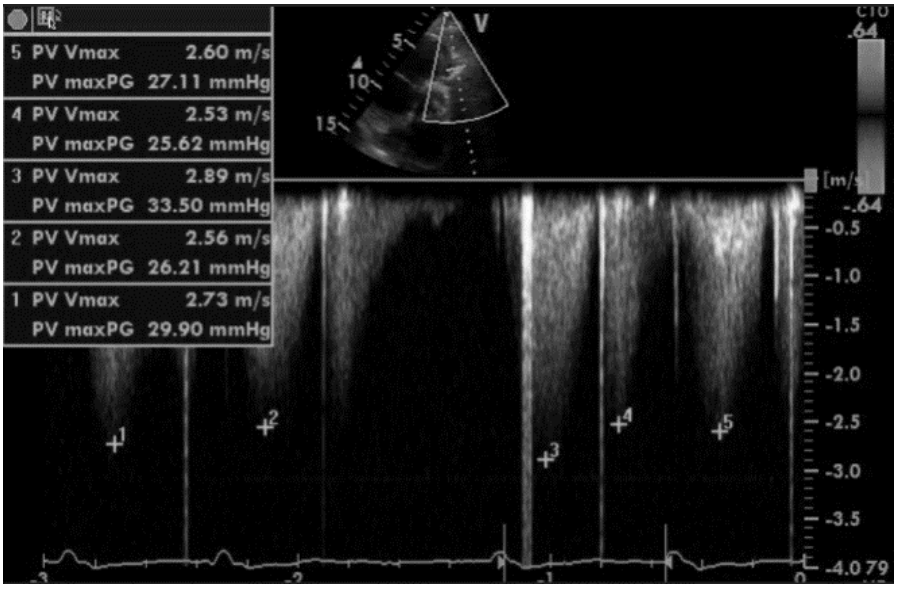

Figure 4. PV gradient 30 months after BPV

Second check-up six months after BAV PVS: gradient over PVS 30mmHg, TR gradient 30mmHg, TA 160/90 mmHg. Mild pretibial edema. Switch NOAC to oral anticoagulants (OAC).Third control examination one year after BAV PVS: gradient over PVS 30mmHg, TR gradient 27mmHg, TA 180 / 100mmHg. Mild pretibial edema. Almost two and a half years hospitalizations due to right-sided cardiac decompensation, also the first decompensation after BAV PVS. Then, aultra sound exam is performed and measureda gradient over PV 25mmHg, TR gradient 27 mmHg; Laboratory observed hypoalbuminemia, 25g / L (RV: 35-52gr / L).After intensified parenteral therapy with two different diuretic drugs, the patient is discharged from the hospital with recommendationsfor therapy with furosemide, spironolactone, β-blocker, ACE inhibitor, PPI and OAC.

DISCUSSION

Pulmonary valvular stenosis (PVS) is one of the most common congenital heart defects, usually occurring in about 10% of cases in relation to all cardiac congenital defects. [1] Acquired cases of PVS are found in other diseases such as infectious endocarditis, carcinoid syndrome. [1] It can occur as an isolated case or associated with other heart defects as part of the tetralogy of Fallot, ventricular septal defect (VSD), transposition of large blood vessels. [2] Also, PVS can be diagnosed at all ages, although it is most often detected in childhood through the observation of systolemurmor in the precordial area, which is mainly the reason for further diagnostic processing. [7, 8] Authors Hardy [9] and Sherman [10] were the first to document cases of congenital pulmonary stenosis in adults. Since 1982, percutaneous balloon pulmonary valvuloplasty has replaced surgical valvulotomy in the treatment of congenital and acquired pulmonary valvular stenosis. [6] Short-term results after the procedure were shown to be very good with low restenosis rates of 4.8%. [11] Similar results were obtained when observing the long-term prognosis. McCrindle [12] presented the results of 533 patients from 22 healthcare institutions averaging 8.7 years after initial BPV and monitored the long-term outcome and concluded that BPV is a useful and reliable procedure whose long-term outcome

depends on independent predictors such as dimension and valvular morphology. A group of scientists, Kaul et al. [13], analyzed the results of BPV performed in forty patients aged 18-56 years and concluded that BPV is a safe and effective procedure for the treatment of pulmonary stenosis in adults, with regression of the initially high RV pressure gradient.Our case is a rare isolated congenital defect, a severe pulmonary valvular stenosis diagnosed in a 60-year-old patient, initially hospitalized for anasarca, weakness, weakness, suffocation, and shortness of breath. Electrocardiographically previously verified permanent atrial fibrillation. Sonographic, transesophageal echocardiography (TEE) visualizes severe pulmonary stenosis and dilatation of the right heart cavities. Invasive cardiac treatment, i.e.right and left cardiac catheterization, shows the systolic pressure of 120 mmHg in the RV, and 85 mmHg above the pulmonary valve, BPV is then performed. Postprocedural hemodynamics values were satisfactory, assystolic pressure in RV was 50 mmHg and gradient over PV was 15 mmHg. The procedure ended without complications. Control inspections were conducted at intervals after one month, two, six months and after one year; the measured parameters presented no significant alterations in the values of pressures. After almost two and a half years, the first right-sided cardiac decompensation occurs, which was successfully remedied by the application of intensive parenteral diuretic therapy. Indications of attenuation of synthetic liver function were noted in the laboratory findings. Ayad et al. [14] in their paper presented the case of a 77-year-old patient who was successfully treated with percutaneous balloon valvuloplasty, with systolic pressure values, i.e. a pressure gradient of RV to pulmonary trunk 114 mmHg. Kilic et al. [6] also describe the case of a 54-year-old patient with severe PVP-treated PV stenosis, with a gradient of RV in the pulmonary trunk reaching 114 mmHg. In his 2019 paper, Liau [15] presented a 73-year-old patient treated for BPV with a positive BPV effect after a six-year follow-up.

Conclusion: Balloon valvuloplasty is the method of choice in the treatment of congenital pulmonary valve stenosis. Reliability and effectiveness have been proven after short-term and long-term follow-up with minimal occurrence of new stenoses in both children and adults.

Conflict of Interest: None.

Acknowledgement: None.

REFERENCES

Ayad RF, Johnston SB, Grayburn PA, et al. Congenital pulmonic stenosis in a 77-year-old woman successfully treated with percutaneous balloon valvuloplasty. *Proc (Bayl Univ Med Cent)*. 2010;23(1):21-23. doi:10.1080/ 08998280.2010.11928575

Hardy WE, Gynoj J, Ayres SM, et al. Pulmonic stenosis and associated atrial septal defects in older patients. Report of three cases, including one with calcific pulmonic stenosis.

Inglessis I, Landzberg MJ. Interventional catheterisation in adult congenital heart disease. Circulation. 2007; 115 (12): 1622-33

Jarrar M, Betbout F, Farhat MB, et al. Long-term invasive and noninvasive results of percutaneous balloon pulmonary valvuloplasty in children, adolescents, and adults. Am Heart J. 1999 Nov;138(5 Pt 1):950-4. doi: 10.1016/s0002-8703(99)70022-0. PMID: 10539828.

Kaul UA, Balbir S, Sanjay T, et al. Long-term results after balloon pulmonary valvuloplasty in adults. Am Heart J. 1993 Vol 126, Issue 5:1152-1155. https://doi.org/ 10.1016/0002-8703(93)90668-Y

Kilic T, Sahin T, Karauzum K, et al. New Pulmonary Valvuloplasty Technique by Use of an Hourglass-Shaped Balloon in 3 Adults with Severe Pulmonary Valve Stenosis. *Tex Heart Inst J*. 2018;45(2):76-83. Published 2018 Apr 7. doi:10.14503/THIJ-16-6018

Kopecky SL, Gersh BJ, McGoon MD, et al. Long-term outcome of patients undergoing surgical repair of isolated pulmonary valve stenosis follow-up at 20-30 years. *Circulation* 1988;78:1150-6.

Liau CS. Congenital Pulmonary Valvular Stenosis Treated with Balloon Valvuloplasty in a Woman with 73 Years of Age. *Acta Cardiol Sin*. 2019;35(3):345-348. doi:10.6515/ACS.201905_35(3).20190123A

McCrindle BW. Independent predictors of long-term results after balloon pulmonary valvuloplasty. Valvuloplasty and Angioplasty of Congenital Anomalies (VACA) Registry Investigators. Circulation. 1994 Apr;89(4):1751-9. doi: 10.1161/01.cir.89.4.1751. PMID: 8149541.

Mitchell, Belinda J.; Mhlongo, M. The diagnosis and management of congenital pulmonary valve stenosis. SA Heart J 2018 Vol 15, No 1 https://doi.org/10.24170/15-1-2903

Nugent EW, Freedom RM, Nora JA, et al. Clinical course inpulmonary stenosis. *Circulation* 1977;56:I38-47.

Otto CM. Valvular Heart Disease, 2nd ed., Sanders, Philadelphia, Right-sided valve disease, 2004;424.

Sherman W, Hershman R, Alexopoulos D, et al. Pulmonic balloon valvuloplasty in adults. *Am Heart J* 1990; 119:186-90

Stout KK, Daniel CJ, Aboulhosn JA, et. al. 2018 AHA/ACC Guideline for the management of adults with congenital heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol* 2019; 73:e81-192

Van der Linde D, Konings EE, Slager MA, et al. Birth prevalence of congenital heart disease worldwide: a systematic review and meta-analysis. J Am Coll Cardiol 2011; 58:2241-7.

****