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Aortic valve stenosis assessment by using the acceleration time, ejection time and ratio of acceleration time to ejection time of the aortic valve

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Abstract

Background:
The aortic valve (AV) stenosis is a common vascular disease of adults. New diagnostics markers and imaging modalities are explored for early detection of AV stenosis and categorization of its severity.
Objective :
To evaluate the role of acceleration time, ejection time and acceleration to ejection time ratio in diagnosis of aortic valve stenosis.
Patients and methods:
This study is a cross sectional study conducted in Baghdad Teaching Hospital, Iraqi Cardiac Center and Ibin Al Bitar Hospital in Baghdad city during the period from 1st of March, 2018 to 31st of May, 2019 on 35 patients with AV stenosis. A sample of 35 healthy controls was selected from relatives of patients in these centers. All hemodynamic measures were assessed
Results:
Acceleration time, ejection time and acceleration time to ejection time ratio of AV stenosis patients were significantly higher than controls. The acceptable cutoff value of acceleration time to ejection time ratio was 0.31 with an appropriate validity results (88.6% sensitivity, 100% specificity and accuracy 94%).
Conclusion:
Acceleration time and Acceleration time to ejection time ratio assessed by two dimensional transthoracic echocardiographic and Doppler studies is helpful in diagnosis of aortic valve stenosis.

Keywords: Aortic valve stenosis, Acceleration time, Ejection time, Acceleration time to ejection.

Introduction

Aortic valve (AV) stenosis is one of the most common valvular diseases and is the third most common cardiovascular disease in developed countries. It occurs in ≈2.8% of patients ≥75 years of age and can occur because of degenerative calcification and congenital valvular defects such as bicuspid AVs or rheumatic disease [1]. Calcific aortic stenosis (AS) is associated with increased leaflet stiffness and a narrowed AV orifice, resulting in increased pressure gradients across the valve. The presence of a bicuspid AV significantly increases the risk of AS. The natural history of AS is a prolonged asymptomatic period, with progressive reduction of the AV orifice area due to sclerosis initially, culminating in calcific AS. This is accompanied by a corresponding increase in the transaortic pressure gradient (ΔP) and myocardial pressure overload. Through the preload reserve, the left ventricle (LV) compensates for the increased workload until the sarcomeres stretch to their maximum diastolic length. Once the preload reserve is exhausted, increases in afterload are accompanied by a reduction in stroke volume (SV), resulting in afterload mismatch. Ultimately, this causes LV hypertrophy, associated with an enlargement of cardiac myocytes and increased LV wall thickness [2].

Initial diagnosis of AS typically occurs during routine physical examination with the presence of a heart murmur, click, or other abnormal sounds, but undiagnosed patients may experience the onset of severe symptoms such as angina, syncope, and heart failure. Without intervention, patient mortality typically occurs within 5 years of the onset of symptoms [3]. Multiple studies and reviews have focused on the clinical aspects of this disease, including disease progression, markers of disease severity, treatment guidelines, and outcomes [2, 4]. Very few reviews have focused on the hemodynamic principles underlying AS and on comparing data obtained across different imaging modalities [5].

Hemodynamics of AS

Flow through the AV is pulsatile in nature and directly depends on multiple factors, including LV systolic and diastolic function, aortic pressure and compliance, leaflet mobility, and LV geometry and chronotropy. When ventricular pressure exceeds aortic pressure at the start of ventricular systole, the AV leaflets open to permit flow through the valve. Cardiac output (CO) increases until peak systole, beyond which it starts to decrease. ΔP and flow rate through the valve vary with the time point in the cardiac cycle. In AS, this temporal variability may play a key role in disease diagnosis [2].

Flow through a stenotic AV is well approximated by flow through a convergent orifice. The narrowed AV orifice and restricted leaflet opening create a hemodynamic nozzle, causing acceleration of blood through the valve—from a low velocity (V1 < 1 m/s) in the LV outflow tract (LVOT) to the maximum velocity (V2 > 1 m/s) at the vena contracta (VC) of the jet.

The area formed by the free edges of the AV leaflets is known as the geometric orifice area (GOA) of the valve, whereas the area of the flow jet at the VC is known as the effective orifice area (EOA). The pressure difference between the LVOT and EOA is referred to as ΔPmax. Fluid mechanics theory shows that GOA is always greater than or equal to EOA (they are equal when GOA and LVOT area are equal) [5, 6]. The ratio of the EOA to the GOA is known as the contraction coefficient (cc). The contraction coefficient depends on the 3-dimensional shape of the valve leaflets, where cc is significantly lower for flat valves than for doming bicuspid valves. Also, the VC always occurs downstream of the valve orifice [6].

Further into the ascending aorta (AAo), some amount of the kinetic energy of the blood is converted back to potential energy, resulting in an increase in the local pressure, and this is known as the pressure recovery effect [5, 7]. The pressure difference between the LVOT and AAo is referred to as ΔPrec. Thus, ΔPmax > ΔPrec owing to pressure recovery. Many in vitro studies have also investigated the effects of pressure recovery, aiming to resolve the discrepancies between catheter and Doppler measurements [8]. These studies clearly demonstrated the role of pressure recovery in the underestimation of the severity of AS by catheterization in comparison with Doppler measurements. In vitro studies suggest that the recovered pressure drop correlates directly to the ventricular workload, but the physiological impact of this is still unclear[8].

Role of Echocardiography

The severity of AS can be assessed with the use of Doppler echocardiography by measuring AS jet velocity, and the AV area can be assessed by use of the continuity equation. ΔPmean can be assessed by use of the Bernoulli equation [9]. Early in vitro studies verified the use of the Bernoulli equation against gold

standard catheterization data to assess ΔP^2. Accurate data recording also requires multiple acoustic windows to determine the highest AS jet velocity. Apical (5-chamber view), suprasternal, or right parasternal views most frequently yield the highest velocity. It was shown that flow characteristics through stenotic AVs by using flow visualization, laser Doppler anemometry, continuous-wave Doppler ultrasound, and color Doppler ultrasound to analyze jet characteristics, turbulent intensities, and peak velocities, and showed that Doppler measurements may be needed in multiple directions to accurately assess the severity of AS [2].

Transthoracic echocardiography is useful in determining AV morphology, concomitant aortic regurgitation, LV function, aortic pathologies, and other valvular abnorm-alities. Transthoracic echocardiogra-phy may distinguish between stenosis caused by hypertrophic cardiomyopathy, valvular or subvalvular stenosis, but in some cases, transesophageal echocardiography may be needed. Dobutamine stress echocardiography is appropriate for patients with low-flow, low-gradient AS with low LV ejection fraction (EF) and has received a Class IIa (Level of Evidence: B) recommendation in the American College of Cardiology/American Heart Association-European Society of Cardiology/ European Association for Cardio Thoracic Surgery guidelines [10, 11].

The AS jet velocity can be directly measured from continuous-wave Doppler tracings through the AV. ΔPmean and ΔPmax can be calculated by using the simplified Bernoulli equation, which assumes a proximal velocity V1 < 1 m/s. ΔPmean must be computed from instantaneous ΔP after using the Bernoulli equation because of the square term in this equation. EOA is calculated by using the continuity equation, because the volume of blood passing through the LVOT must equal the volume of blood ejected at the EOA. The LVOT diameter is measured from a parasternal long-axis view of the LVOT, and the LVOT velocity time integral (VTI) is obtained using a pulsed-wave Doppler signal. From these, the EOA can be calculated as the product of LVOT cross-sectional area and LVOT VTI divided by the continuous-wave Doppler VTI. This is the fluid volume at the VC, because continuous wave measures the highest velocity in the line of interrogation. Other hemodynamic measurements of AS such as energy-loss index, AV resistance, valvuloarterial impedance, and LV stroke loss may also be calculated from the acquired data [12].

Role of Cardiac Catheterization

In the 1950s and 1960s, invasive hemodynamic studies were essential for understanding the physiology and pathophysiology of valvular heart disease. With the advent of echocardiography in the 1980s and 1990s and the evolution of percutaneous coronary intervention, the role of the cardiac catheterization laboratory slowly shifted to diagnosing and treating coronary artery disease. However, in the past few years, the development of percutaneous approaches to valvular heart disease has led to a renaissance of invasive hemodynamic studies [10]. According to the American College of Cardiology/ American Heart Association Guidelines for the management of patients with valvular heart disease, coronary angiography may be sufficient before valve replacement if clinical and echocardiographic data consistently indicate severe AS10. On the other hand, any discrepancies between these must be reconciled by using cardiac catheterization so that the patient is not deprived of the potential benefit of aortic valve replacement (AVR) for severe symptomatic AS. Additionally, catheterization with dobutamine infusion may be used in patients with low-flow, low-gradient AS and LV dysfunction [13].

Typically, ΔP is measured between the LVOT and the AAo by using double-lumen fluid-filled catheters for simultaneous LV and aortic pressure measurements. Micromanometer-tipped catheters may be considered when extensive artifacts degrade the quality of tracings from the fluid-filled catheters or when additional precision is necessary for research. Pullback gradients are inaccurate for diagnostic purposes. CO is assessed in the cardiac catheterization laboratory by [2] principal methods: Fick and thermodilution. The Fick method relies on obtaining arterial and mixed venous saturations, hemoglobin level, and oxygen consumption. The thermodilution method relies on injecting cold or room-temperature saline and measuring the change in temperature as this passes from the injection port to the thermistor on the Swan-Ganz catheter. Once ΔP and CO are obtained, the Gorlin equation is used to calculate the EOA [14]. However, this area differs from the corresponding echocardiographic measurement owing to the difficulty in precisely positioning the aortic side catheter at the VC of the flow jet. Hence, catheterization ΔP is equivalent to ΔPrec. Additionally, ΔPmean and ΔPpeak may be measured, whereas only the mean CO is available for calculation. A detailed description of the potential errors

associated with these measurements is presented later. Despite the potential for inaccuracies, it is recommended that the operator perform a quick on-the-fly calculation of EOA by using the simplified Hakki equation [2].

Low-Flow, Low-Gradient

AS with Reduced EF Approximately 5% to 10% of patients who have severe AS present with low CO and ΔPmean<40 mmHg, with decreased LVEF (<40%) and pose a therapeutic dilemma, because it is complex to determine whether these patients have true severe AS or pseudostenosis due to cardiomyopathy. In patients with pseudostenosis, the AVs are moderately diseased and leaflet opening is impaired because of a diseased and weak LV [2].

Although patients with pseudostenosis may have a high mortality risk approaching 50% with surgical AVR and may benefit more from evidence-based heart failure medical therapy, true AS patients would benefit more from AVR [15]. However, it must be noted that these findings are typically from small patient populations, because patients with pseudostenosis are typically not operated on [16]. Dobutamine infusion remains the primary method of distinguishing pseudostenosis from true AS, both with echocardiography and catheterization. The inotropy provided by dobutamine increases the SV so that patients with true severe AS will increase the ΔP and velocity with minimal change in EOA. On the other hand, in patients with pseudostenosis, the increase in SV will open the AV further and cause minimal increase in ΔP and velocity, but will increase the calculated valve area confirming moderate AS. An accurate measurement of gradients and EOA in these patients is critical to ensure optimal treatment strategies [16].

Paradoxical Low-Flow, Low-Gradient AS Low-flow gradient AS with preserved EF, also known as paradoxical low-flow, low-gradient AS is a newly recognized and appreciated entity where patients with severe AS but apparently good ventricular function present with lower than expected ΔP on the basis of generally accepted guidelines [17]. Patients with paradoxical low-flow low-gradient AS may have significant LV concentric remodeling with a small LV cavity, impaired LV filling, and reduced systolic longitudinal myocardial shortening. These abnormalities result in a low SV with ΔP<40 mmHg despite a preserved EF, which may explain discrepancies in diagnosis while using EOA and ΔP, as noted by Minners and colleagues. Eighty-nine Recent clinical studies have shown that patients with paradoxical low-flow low-gradient AS have a worse prognosis than patients with normal-flow, low-gradient AS 18. This disease pattern, in which the low gradient is interpreted as moderate AS may lead to underestimation of stenosis severity and thus cause an inappropriate delay in surgical intervention. It is important to note that other reasons can cause this hemodynamic situation with a lower than expected gradient. It can occur from a small LV that correlates with a small body size, yielding a lower than normal SV; measurement errors in determining SV and EOA by Doppler echocardiography; systemic hypertension that can influence the estimation of the gradient by Doppler; and an inconsistency in the definition of severe AS by the current guidelines relating to cutoffs of valve area in relation to those of jet velocity and gradient [14]. Whether intervention in these patients with EOA<1.0 cm2 improves outcomes remains to be established and reproduced in future prospective studies. It must be noted that the recent European Society of Cardiology/European Association for Cardio-Thoracic Surgery guidelines have a Class IIa recommendation for AVR in these patients [11]. However, it is clear that an integrated assessment of these patients, including physical examination and CT assessment for characterizing the extent of AVC may lessen the potential for misdiagnosis[11]. However, peak velocity and mean gradient are flow dependent and may be unrepresentative of the grade of AS at extremes of physiologic flow. Effective orifice area estimated by continuity equation may be limited when left ventricular systolic function is severely impaired5; in addition, the measurement of left ventricular tract outflow diameter is the greatest potential source of error in the continuity equation [19].

Guidelines suggest that the aortic waveform shape may be helpful 20. Nevertheless, few studies have evaluated ejection dynamics parameters in native aortic valve disease 21.

Patients and Methods

This study is a case control study conducted in Baghdad Teaching Hospital, Iraqi Cardiac Center and Ibin Al Bitar Hospital in Baghdad city during the period from 1st of March, 2018 to 31st of May, 2019.

A sample size of 35 patients with aortic valve (AV) stenosis was selected. A sample of 35 healthy controls was selected from relatives of patients in these centers.

Inclusion criteria: Adults (age ≥18 years). Valvular aortic stenosis. Aortic stenosis peak velocity > 2 m/s.

Exclusion criteria : Younger age, Suboptimal acoustic window.Concomitant moderate or severe AR (aortic regurgitation). Moderate or severe Mitral or tricuspid valvular disease. Subvalvular or supravalvular AS. Refuse to participate.Anemia. Heart rate and blood pressure.

The data was collected by researcher from study participants directly and filled in a prepared questionnaire. The questionnaire included Demographic characteristics of AV stenosis patients, Age and gender,Vital signs of AV stenosis patients: Blood pressure and heart rate,Acceleration time to ejection time (AT/ET) of patients with AV stenosis, Hemodynamic measurements of AV stenosis patients: Velocity, mean of gradient, effective orifice area, ejection time, acceleration time and ejection fraction.

After taking full history and examination of suspected patients by two physicians, the confirmed diagnosis of AV stenosis was accomplished according to: Severe AS EROA, 1cm2, mean gradient >40 mm Hg, mild AS EROA=1.5cm2, moderate AS EROA between 1 and 1.5 cm2. The two dimensional transthoracic echocardiographic and Doppler studies were done by clinical US machine equipment (GE vivid 9) with 2.5 to 3.5-MHz transducer. These measures are the average of 3 cardiac cycle in sinus rhythm. Doppler record was performed at the sweep speed of 150 mm /sec. The parasternal long-axis view zoom was used to measure aortic annulus diameter in early systole. Using pulse Doppler in the LVOT, placing the sample volume 1 cm below the aortic valve, the time velocity integral was obtained. Stroke volume was then calculated assuming a circular shape of LVOT. Continuous wave Doppler recording of flow through the valve was performed from the five chamber and right parasternal window to record maximal instantaneous and mean pressure gradient across the aortic valve. EOA was calculated using the continuity equation. Mean transvalvular pressure gradient was obtained using modified Bernoulli equation. All measurement represent average of three cardiac cycles for patients in sinus rhythm and at least six cycles if the patients was in rhythm other than sinus. The systolic time intervals of flow by the aortic valve were measured using the velocity curve from the continuous wave Doppler recording in the apical view, ET was measured at the time from onset to end of systolic flow .AT was defined as the time interval between the beginning of systolic flow to its peak velocity, the AT/ET ratio was then calculated accordingly.

The Interobserver variability of systolic time interval were evaluated by second experienced cardiologist .

Results

A total of 35 patients with aortic valve (AV) stenosis were included in present study with mean age of 73.4±4.3 years, 17.1% of them were less than 70 years age, 80% of them were in age group 70-79 years and 2.9% of them were 80 years age and more. Female AV stenosis patients were more than males with female to male ratio as 1.18:1. All these finding were shown in table 1.

Table 1: Demographic characteristics of AV stenosis patients.

Variable	No.	%
Age Mean±SD (73.4±4.3 years)		
<70 years	6	17.1
70-79 years	28	80.0
≥80 years	1	2.9
Total	35	100.0
Gender		
Male	16	45.7
Female	19	54.3
Total	35	100.0

Mean velocity of AV stenosis patients was (4.2 m/s) and mean gradient of them was (44.9 mmHg). Mean effective orifice area (EOA) of AV stenosis patients was (1.0 cm2) and mean acceleration time (AT) of AV stenosis patients was (116.5 ms) while mean ejection time (ET) of them was (314.3 ms) and mean AT/ET of AV stenosis patients was (0.37). Mean ejection fraction of AV stenosis patients was (57.5%). Mean blood pressure of AV stenosis patients was (140.5/85.1 mmHg) and mean heart rate of them was (79.5 b/m). All these findings were shown in table 2.

Table 2:Hemodynamic measurements and vital signs of AV stenosis patients.

Variable	Mean±SD
Velocity (m/s)	4.2±1.0
Mean gradient (mmHg)	44.9±15.1
EOA (cm2)	1.0±0.4
AT (ms)	116.5±22.2
ET (ms)	314.3±31.7
AT/ET	0.37±0.05
EF (%)	57.5±6.2
BP (mmHg)	140.5/85.1±15.7/7.7
HR (b/m)	79.5±7.4

No significant differences were observed between AV stenosis patients and healthy controls regarding age (p=0.6) and gender (p=0.6). All these findings were shown in table 3.

Table 3: Distribution of demographic characteristics according to AV stenosis cases and controls.

Variable	AV		Controls		P
	No.	%	No.	%	
Age					0.6*^{NS}
<70	6	17.1	9	25.7	
70-79	28	80.0	25	71.4	
≥80	1	2.9	1	2.9	
Gender					0.6**^{NS}
Male	16	45.7	18	51.4	
Female	19	54.3	17	48.6	

*Fishers exact test, **Chi-square test,

Mean velocity of patients with AV stenosis was significantly higher than velocity of healthy controls (p<0.001). Mean gradient of AV stenosis patients was significantly higher than mean gradient of healthy controls (p<0.001). Mean EOA of patients with AV stenosis was significantly lower than EOA mean of healthy controls (p<0.001). Mean AT of AV stenosis patients was significantly higher than mean AT of healthy controls (p<0.001). Mean ET of patients with AV stenosis was significantly higher than ET mean of healthy controls (p<0.001). Mean AT/ET of AV stenosis patients was significantly higher than mean AT/ET of healthy controls (p<0.001). Mean ejection fraction of AV stenosis patients was significantly lower than mean ejection fraction of healthy controls (p=0.003). All these findings were shown in table 4.

Table 4: Distribution of hemodynamic measurements means according to AV stenosis cases and controls.

Variable	AV	Controls	P *
	Mean±SD	Mean±SD	
Velocity	4.2±1	1.89±0.1	**<0.001**
Mean	44.9±15.1	12.5±1.7	**<0.001**
EOA	1±0.4	2.2±0.1	**<0.001**
AT	116.5±22.	70.5±3.5	**<0.001**
ET (ms)	314.3±31.	243.8±10.	**<0.001**
AT/ET	0.37±0.05	0.28±0.01	**<0.001**
EF (%)	57.5±6.2	61.1±3.1	**0.003**

*Independent sample t-test, S=Significant.

The acceptable cut off points and the corresponding validity values for velocity level in prediction of AV stenosis was shown in figure 1, cutoff velocity level of 2.2 m/s had acceptable validity results (100% sensitivity, 100% specificity, 100% PPV, 100% NPV and accuracy 100%).

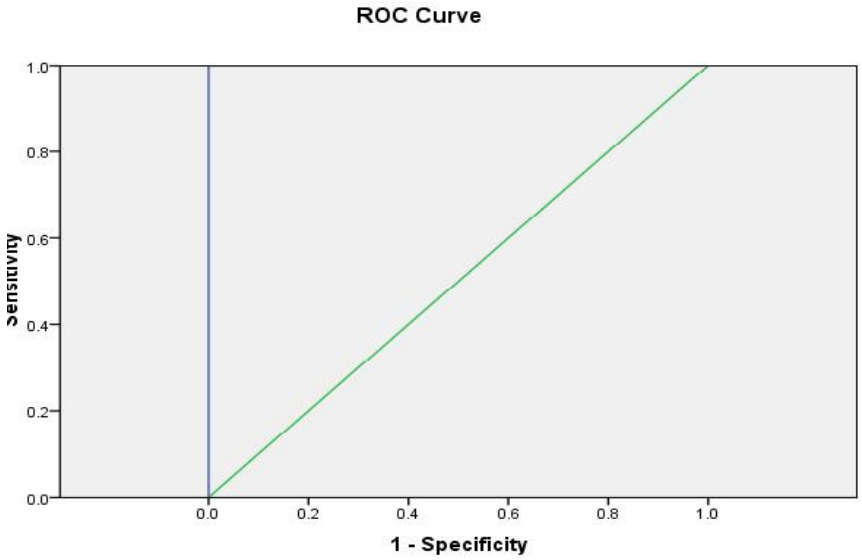

Figure 1: ROC for velocity prediction of AV stenosis (AUC=1.0).

The acceptable cut off points and the corresponding validity values for AT level in prediction of AV stenosis was shown in figure 2, cutoff AT level of 76 ms had acceptable validity results (97.1% sensitivity, 91.4% specificity, 94.3% PPV, 96.2% NPV and accuracy 95%).

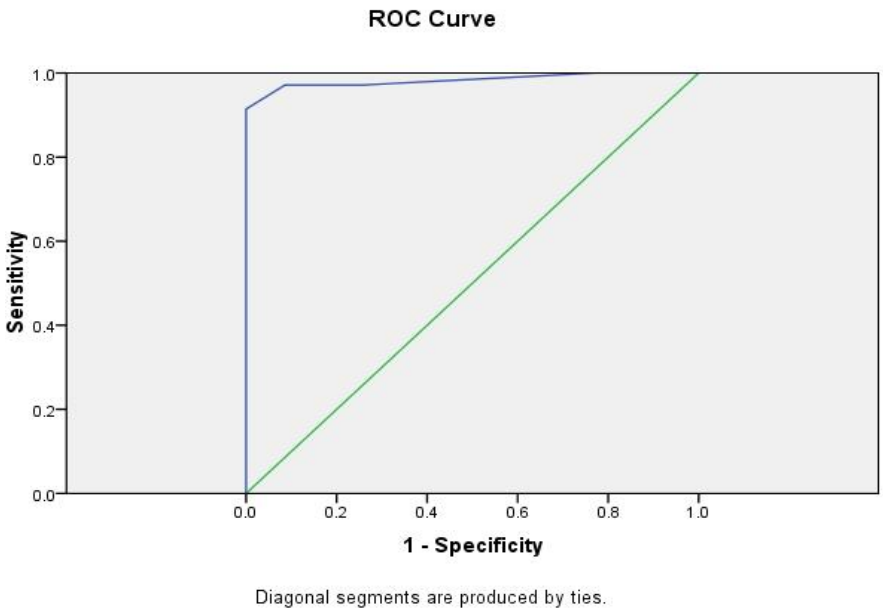

Figure 2: ROC for AT prediction of AV stenosis (AUC=0.98).

The acceptable cut off points and the corresponding validity values for ET level in prediction of AV stenosis was shown in figure 3, cutoff ET level of 263 ms had acceptable validity results (100% sensitivity, 100% specificity, 100% PPV, 100% NPV and accuracy 100%).

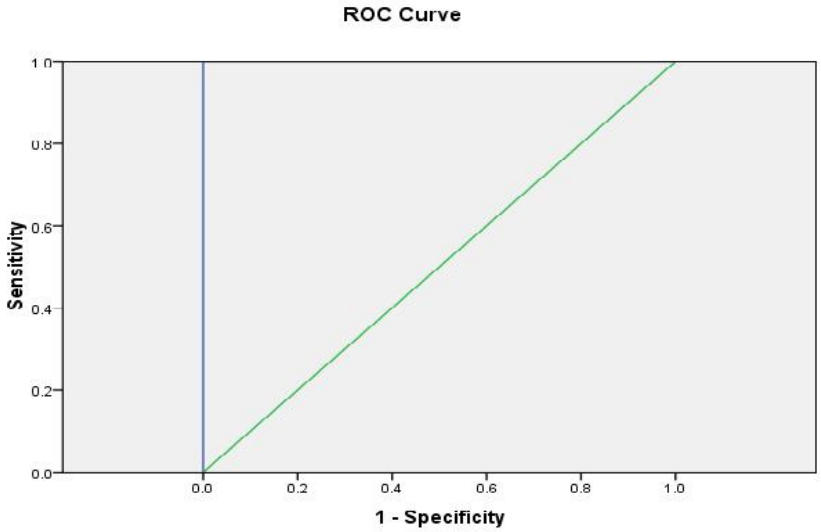

Figure 3: ROC for ET prediction of AV stenosis (AUC=1.0).

The acceptable cut off points and the corresponding validity values for AT/ET ratio in prediction of AV stenosis was shown figure 4, cutoff AT/ET level of 0.31 had acceptable validity results (88.6% sensitivity, 100% specificity, 100% PPV, 95% NPV and accuracy 94%).

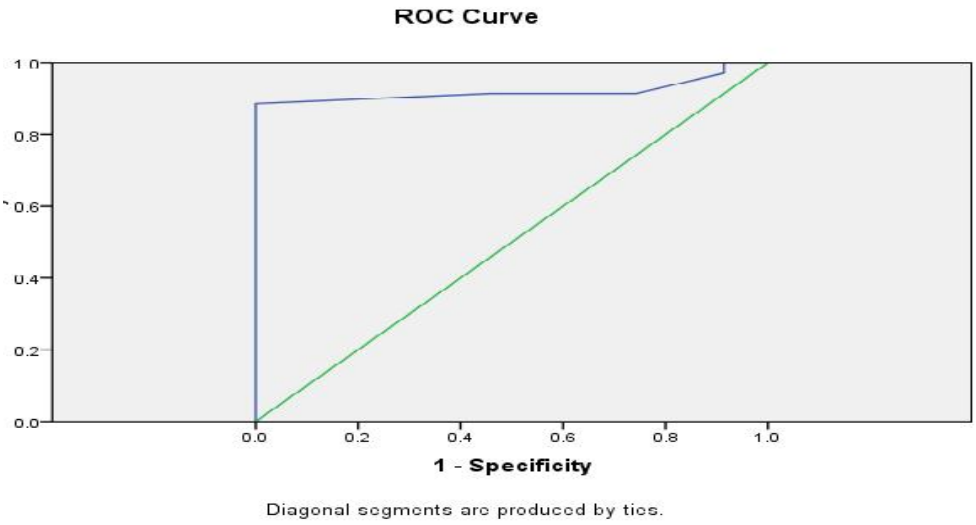

Figure 4: ROC for AT/ET ratio prediction of AV stenosis (AUC=0.92).

Discussion

The aortic stenosis is a highly predominant valvular disease that treated surgically in developed countries[44]. Delay in diagnosis and management of aortic stenosis is accompanied with high risk of sudden cardiac death specifically among asymptomatic cases. So, early detection of aortic stenosis is fundamental for management 45. In current study, mean velocity of patients with AV stenosis was significantly higher than velocity of healthy controls ($p<0.001$). This finding is consistent with results of Lancellotti et al 46 study conducted in multiple centers in Europe and USA which found that increased velocity is diagnostic of AV stenosis and velocity of 5 m/s is indicating of severe AV stenosis. Our study showed that mean gradient of AV stenosis patients was significantly higher than mean gradient of healthy controls ($p<0.001$). This finding is similar to results of McCarthy et al [47] study in Ireland which stated that higher mean gradient of AV stenosis patients is predictive for severity of AV stenosis. Current study also found that mean EOA of patients with AV stenosis was significantly lower than EOA mean of healthy controls ($p<0.001$). This finding coincides with results of Naseem et al [48] study in USA which revealed that lower EOA is predictive for AV stenosis. Many literatures indicated that EOA is an independent diagnostic parameter for AV stenosis and EOA in combination with velocity and mean gradient were applied in detection of AV stenosis severity 49. Present study revealed that Mean AT of AV stenosis patients was significantly higher than mean AT of healthy controls ($p<0.001$).

This finding is similar to results of Sordelli et al [50] study in Italy which reported that mean AT is increased among patients with aortic valve stenosis as compared to healthy individuals. Our study also showed that mean ET of patients with AV stenosis was significantly higher than ET mean of healthy controls ($p<0.001$). This finding is consistent with reports of Clavel et al [51] study in France which stated that ejection time is commonly increased among patients with AV stenosis. For that, our study showed an increased in mean AT/ET ratio among patients with AV stenosis in comparison to healthy controls ($p<0.001$). This finding is similar to results of many literatures like Ben Zekri et al [52] study in Italy and Gamaza-Culián et al [53] study in Spain which reported higher AT/ET ratio among patients with AV stenosis in comparison to healthy controls. In current study, mean ejection fraction of AV stenosis patients was significantly lower than mean ejection fraction of healthy controls ($p=0.003$). Recent study in USA by Ito et al [54] found that decreasing of left ventricular ejection fraction is a predictor of poor prognosis.

Present study showed that appropriate cutoff value of AT/ET ratio in prediction of AV stenosis was 0.31 with validity findings of (88.6% sensitivity, 100% specificity and 94% accuracy). Our study findings are also close to results of Samiei et al 55 study in Iran which included 80 patients with AV stenosis and found that best cutoff value for AT/ET in echocardiographic diagnosis of severe AV stenosis was 0.36 with validity findings of (90% sensitivity, 100% specificity and 96% accuracy). Our study findings revealed lower sensitivity with higher specificity of AT/ET ratio in diagnosis of AV stenosis.

This finding is consistent with results of Gamaza-Culián et al [43] study in Spain which revealed that appropriate cutoff value for AT/ET ratio in diagnosing AV stenosis was 0.35 with validity findings of 77% sensitivity and 100% specificity. Our study showed that appropriate cutoff value of AT in prediction of AV stenosis was 76 ms with validity findings of (97.1% sensitivity, 91.4% specificity and 95% accuracy). A study carried out in Mexico by Miramontes-Espino et al [55] on 75 patients with AV stenosis, found that appropriate cutoff value for AT in diagnosis of severe AV stenosis was 104.5 ms with 92.2% sensitivity and 83.3% specificity, while for ET, the best cutoff value was 323.5 ms in diagnosis of severe AV stenosis with 80.4% sensitivity and 70.8% specificity and for AT/ET ratio, the best cutoff value in diagnosis of severe AV stenosis was 0.34 with sensitivity of 84.3% and specificity of 91.7%. In present study, the appropriate cutoff value of ET in diagnosis of AV stenosis was 263 ms with (100% sensitivity, 100% specificity and 100% accuracy). Our study also showed that mean velocity cutoff value for prediction of AV stenosis was 2.2 m/s with validity findings (100% sensitivity, 100% specificity and 100% accuracy). This finding is consistent with results of Otto study in USA which revealed that peak velocity is predictable for aortic valve stenosis [56]. In general, our study showed that AT/ET ratio is important in confirming diagnosis of AV stenosis as it was associated with 100% specificity and AT is associated with higher sensitivity which is helpful for screening, while ET is accompanied by high sensitivity and specificity rates that help in screening and diagnosis of AV stenosis. All these findings are similar to results of Gamaza-Culián et al 52 study in Spain which documented that AT, ET and AT/ET ratio are helpful in diagnosis and evaluation of AV stenosis severity.

Current study showed that mean blood pressure of patients with AV stenosis was significantly higher than blood pressure of healthy controls (p=0.03). Similarly, Aronow study in USA reported that hypertension prevalence is increased among patients with AV stenosis and this hypertension increased chance of left ventricular hypertrophy [57]. Our study also showed that mean heart rate of AV stenosis patients was significantly higher than mean heart rate of healthy controls (p<0.001). This finding is in agreement with results of O'Sullivan et al [58] study in Switzerland which stated that high resting heart rate is accompanying AV stenosis. Vahanian et al [59] reported that increased cardiac output represented by high blood pressure and heart rate is indicating severe aortic valve stenosis.

Conclusion

Acceleration time and ejection time measurement of two dimensional transthoracic echocardiographic and Doppler studies are helpful in screening and diagnosis of aortic valve stenosis.

Acceleration time to ejection time ratio assessed by two dimensional transthoracic echocardiographic and Doppler studies is helpful in diagnosis of aortic valve stenosis.

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