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Mini Review

Elevated Blood Pressure and Risk of Mitral Regurgitation

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ABSTRACT

Introduction: People with high systolic blood pressure but no known cardiovascular disease is at risk for developing mitral regurgitation (MR), a longitudinal study suggests.

Objectives: The main objective of the study is to find the relation of elevated blood pressure and risk of mitral regurgitation.

Material and methods: This correlational study was conducted in Nishtar Medical College, Multan during 2020. The data was collected from both genders and age range was 30 to 60 years. We exclude all the patients who were at baseline and had a previous history of CVD. Our primary outcome was incident reports of mitral regurgitation, which were identified from hospital discharge reports, death registers, or primary care records.

Results: The data was collected from 50 patients of both genders. The mean age of the patients was 55.67 ± 5.87 years. The baseline values are presented in Table 1.

Conclusion: It is concluded that long-term exposure to elevated BP across its whole spectrum is associated with an increased risk of primary and secondary mitral regurgitation.

Key words: Adequate surgery, Adequate margins, Carcinomas, Head and neck cancers, Perineural invasion

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INTRODUCTION

People with high systolic blood pressure but no known cardiovascular disease are at risk for developing mitral regurgitation (MR), a longitudinal study suggests. When the mitral valve becomes leaky, it's called mitral valve regurgitation. It's also known as mitral insufficiency. The mitral valve is one of the heart's 4 valves. These valves help the blood flow through the heart's 4 chambers and out to the body (Nishimura RA, *et al.*, 2017). The mitral valve lies between the left atrium and the left ventricle. Normally, the mitral valve prevents blood flowing back into the left atrium from the left ventricle. In mitral valve regurgitation, however, some blood leaks back through the valve. It doesn't just flow forward into the ventricle the way it should. Because of this, the heart has to work harder than it should to get blood out to the body. If the regurgitation gets worse, some blood may start to back up into the lungs. A very small amount of mitral regurgitation is common. But some people have severe mitral valve regurgitation (Iung B and Vahanian A, 2011).

Mitral valve regurgitation can be acute or chronic. With the acute condition, the valve suddenly becomes leaky. In this case, the heart doesn't have time to adapt to the leak in the valve. Symptoms with acute mitral regurgitation are often severe. In the chronic form, the valve gradually becomes leakier over time. This gives the heart time to adapt to the leak. With chronic mitral regurgitation, the symptoms may range from mild to severe (Rahimi K, *et al.*, 2015).

Despite substantial progress in our understanding of the pathophysiology of mitral regurgitation and advances in surgical and interventional valve replacement therapies, there are no established preventive strategies (Singh JP, *et al.*, 1999). Causes of mitral regurgitation are categorised as "primary" when due to abnormalities of the valve leaflets and chordae or as "secondary" when due to distortion of ventricular shape related to ischaemic heart disease (IHD) or a cardiomyopathy. However, the distinction between these subtypes is not always obvious and about two-thirds of all mitral regurgitation cases are classified as degenerative, implying that they are a natural consequence of ageing with no possibility of altering their course (Emdin CA, *et al.*, 2015).

Elevated blood pressure (BP) is a strong risk factor for a range of cardiovascular conditions. Given that increased BP correlates with higher left ventricular pressure, and this, in turn, exposes the mitral valve to higher physical stress, it seems plausible that long-term exposure to higher BP could also lead to structural and functional changes of the mitral valve. A cross-sectional analysis of the Framingham study showed a positive association between hypertension and mitral regurgitation (Prospective Studies Collaboration, 2002).

Objectives

The main objective of the study is to find the relation of Elevated blood pressure and risk of mitral regurgitation.

MATERIAL and METHODS

TThis correlational study was conducted in Nishtar Medical College, Multan during 2020. The data was collected from both genders and age range was 30 to 60 years. We exclude all the patients who were at baseline and had a previous history of CVD. Our primary outcome was incident reports of mitral regurgitation, which were identified from hospital discharge reports, death registers, or primary care records. The demographic data were also collected by using a questionnaire and SBP were measured in all selected patients. However, in additional prespecified analyses, we chose DBP and pulse pressure (PP; defined as SBP-DBP) as alternative exposure variables to investigate any differential effect of BP indices on risk and to enable comparison of our findings with epidemiological studies of BP associations with other outcomes. To investigate the extent to which the potential association between BP and mitral regurgitation is mediated by incident MI, IHD, heart failure, or cardiomyopathy, which are established causes of secondary mitral regurgitation, we performed time-varying adjustments for these events during the follow-up.

Statistical analysis

The data was collected and analysed using SPSS version 19. All the values were expressed in mean and standard deviation.

RESULTS

The data was collected from 50 patients of both genders. The mean age of the patients was 55.67 ± 5.87 years. The baseline values are presented in *(Table 1)*.

Table 1: Baseline characteristics and outcome rates

Baseline characteristics	<121 mmHg	121-140 mmHg	141-160 mm Hg	>160 mmHg	Total
Mitral valve disease, n(%)					
Mitral regurgitation	26(26%)	18(18%)	7(7%)	6(6%)	57
Mitral stenosis	12(12%)	7(7%)	2(2%)	22(22%)	43
Age categories, years, n	Retrospective	Retrospective	Retrospective	Retrospective	Retrospective
Age, media(IQI)	32(30,40)	39(33,52)	60(49,70)	67(57,74)	39(33,52)
BMI (kg/m2) categories, n	Retrospective	Retrospective	Retrospective	Retrospective	Retrospective
BMI, median(IQI)	23(21,26)	25(23, 28)	27(24, 31)	27(24,31)	25(22,28)
Cholesterol(mmol/L), median (IQI)	Retrospective	Retrospective	Retrospective	Retrospective	Retrospective
Total	4.0(4.3,5.7)	3.3(4.6,6.1)	4.6(4.8, 6.3)	7.6(4.8,6.4)	5.3(4.6,6.1)
LDL	2.9(2.4,3.7)	2.2(2.6,3.9)	4.3(2.6, 4.0)	2.3(2.6,4.1)	3.2(2.6,3.9)
HDL	1.4(1.2,1.7)	1.3(1.1,1.6)	1.3(1.1,1.6)	1.39(1.1,1.7)	1.3(1.1,1.6)
Intermediary conditions, n(%)	22(22%)	7(7%)	8(8%)	8(8%)	45
Follow-up(months), median (IQI)	9(9%)	10(10%)	12(12%)	24(24%)	55

DISCUSSION

Mitral regurgitation is a heterogeneous condition that can be caused by primary structural abnormalities of the valve apparatus (primary mitral regurgitation) or diseases of the left ventricle leading to incomplete closure of a structurally normal valve (secondary mitral regurgitation). The main diseases of the left ventricle that are commonly considered as more proximate causes of secondary mitral regurgitation are MI, IHD, heart failure, and cardiomyopathy (Emdin CA, *et al.*, 2016). Because elevated BP is a known risk factor for these conditions, it is possible that the observed association between BP and mitral regurgitation is entirely or largely a reflection of such indirect effects. However, accounting for the possible intermediary effect of diseases of the left ventricle had little impact on the observed associations between elevated BP and risk of mitral regurgitation (Honaker J, *et al.*, 2011). In confounder-and mediator-adjusted models, which in essence provide an estimation of the association between BP and primary mitral regurgitation, the overall HR was only slightly attenuated and only 13% of the excess risk could be attributed to the intermediary outcomes (Loardi C, *et al.*, 2011) *(Table 2).*

Table 2: Predictors of outcomes in patients with mitral regurgitation undergoing percutaneous valve repair

Predictors of outcomes	All(n=100)	Organic MR(n=41)	Functional MR(n = 59)	P
MR grade 4+, n(%)	76(76%)	3(3%)	21(21%)	0.961
Effective regurgitant orifice area, mm2 ± SD	31 ± 11	27 ± 15	30 ± 11	0.229
Left ventricular end diastolic volume /BSA ml/m2 ± SD	96 ± 27	80 ± 24	103 ± 25	< 0.001
Left ventricular ejection fraction, % ± SD	39 ± 9	51 ± 6.9	41 ± 8	< 0.001
Left atrium volume /BSA, ml/m2 ± SD	52 ± 11	59 ± 20	51 ± 11	0.065
sPAP, mmHg ± SD	43 ± 9	39 ± 15	44 ± 12	0.07

Therefore, it seems plausible that the mechanical stress caused by the elevated BP will lead to gradual structural changes of the valve apparatus (Tobin MD, *et al.*, 2005). Future studies could explore these and alternative mechanisms further. Such studies, ideally when repeated cardiac imaging is available, might also be able to investigate whether the observed associations differ by presence or type of underlying valve pathologies, such as Barlow disease or mitral valve prolapse (Basso C, *et al.*, 2004; Movahed MR, *et al.*, 2006).

CONCLUSION

It is concluded that long-term exposure to elevated BP across its whole spectrum is associated with an increased risk of primary and secondary mitral regurgitation. These findings suggest that BP control may be of importance in the prevention of mitral regurgitation.

REFERENCES

1. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwinlll JP, Fleisher LA, *et al.* 2017 AHA/ACC focused update of the 2014 AHA/ACC guideline for the management of patients with valvular heart disease. J Am Coll Cardiol. 2017; 135(25): e1159-1195.
2. Iung B, Vahanian A. Epidemiology of valvular heart disease in the adult. Nat Rev Cardiol Nature. 2011; 8: 162-172.
3. Rahimi K, Emdin CA, MacMahon S. The epidemiology of blood pressure and its worldwide management. Circ Res. 2015; 116(6): 925-36.
4. Singh JP, Evans JC, Levy D, Larson MG, Freed LA, Fuller DL, *et al.* Prevalence and clinical determinants of mitral, tricuspid, and aortic regurgitation (the Framingham Heart Study). Am J Cardiol. 1999; 83(6): 897-902.
5. Emdin CA, Anderson SG, Woodward M, Rahimi K. Usual blood pressure and risk of new-onset diabetes: Evidence from 4.1 million adults and a meta-analysis of prospective studies. J Am Coll Cardiol. 2015; 66(14): 1552-1562.
6. Age-specific relevance of usual blood pressure to vascular mortality: A meta-analysis of individual data for one million adults in 61 prospective studies. Prospective Studies Collaboration. Lancet. 2002; 360(9349): 1903-1913.
7. Emdin CA, Anderson SG, Salimi-Khorshidi G, Woodward M, MacMahon S, Dwyer T, *et al.* Usual blood pressure, atrial fibrillation and vascular risk: evidence from 4.3 million adults. Int J Epidemiol. 2016; 46(1): 162-172.
8. Honaker J, King G, Blackwell M. Amelia II: A Program for Missing Data. J Stat Softw. 2011; 45(7): 1-47.

9. Loardi C, Alamanni F, Trezzi M, Kassem S, Cavallotti L, Tremoli E, *et al.* Biology of mitral valve prolapse: The harvest is big, but the workers are few. Int J Cardiol. 2011; 151(2): 129-35.

10. Tobin MD, Sheehan NA, Scurrah KJ, Burton PR. Adjusting for treatment effects in studies of quantitative traits: Antihypertensive therapy and systolic blood pressure. Stat Med. 2005; 24(19): 2911-2935.

11. Basso C, Boschello M, Perrone C, Mecenero A, Cera A, Bicego D, *et al.* An echocardiographic survey of primary school children for bicuspid aortic valve. Am J Cardiol 2004; 93(5): 661-663.

12. Movahed MR, Hepner AD, Kashani AM. Echocardiographic prevalence of bicuspid aortic valve in the population. Heart Lung Circ 2006; 15(5): 297-299.