

Diagnosis of valvular heart disease in primary care

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Abstract:

In this review we discuss importance of early detection of valvular heart disease by primary care physicians and diagnostic test methods in primary care. This narrative review was performed using electronic medical databases; PubMed, Embase, and Google scholar, searched was targeting relevant studies concerned with management of valvular heart disease in primary care published up to 2017. Presented the high prevalence of valvular heart illness, primary care physicians have to be familiar with the most common valvular heart illness and their clinical manifestations. Knowledge of, the natural history of the most usual valvular heart diseases is essential because the onset of signs usually is the point at which intervention comes to be necessary. Most valvular heart diseases are amenable to surgical treatment, which can manage a symptom-free and relatively normal lifespan. For that reason, primary care physicians must be familiar with the indications for therapeutic interventions and one of the most proper treatments currently recommended.

Introduction:

Cardiovascular disease (CVD) is the leading killer worldwide and valvular problems one of them. 80% of all CVD cases occur in developing nations [16], causing more than 19% of deaths –more

than war and malaria combined [17]. Primary care physicians have to be familiar with the diagnosis and treatment for the most likely valvular diseases to be encountered [1]. Although coronary illness often progresses in spite of proper medical treatment (specifically HMG-CoA reductase inhibitors, aspirin, and cigarette smoking cessation) or surgical treatment, proper and timely treatment in valvular heart illness usually causes a symptom free and normal lifespan [2], [3].

Two significant questions must be responded to in the management of every case of valvular disease: Is the extent of condition adequate to cause morbidity or mortality for which restorative intervention may be required? If so, just what is the best medical or surgical treatment to reduce or eliminate this morbidity or death? [3].

In this review we discuss importance of early detection of valvular heart disease by primary care physicians and diagnostic test methods in primary care.

Methodology:

This narrative review was performed using electronic medical databases; PubMed, Embase, and Google scholar, searched was targeting relevant studies concerned with management of valvular heart disease in primary care published up to 2017. Search strategy restricted to only English language articles and restriction to human subjects studies. More search was performed through the references list of the included articles.

Discussion:

• **CARDIAC MURMURS: A CLUE TO VALVULAR HEART DISEASE**

Cardiac murmurs commonly are uncovered on routine physical evaluation in individuals with and without frank cardiac illness, or by history when the patients show that an additional service provider formerly valued, a cardiac murmur. A murmur heard during cardiac auscultation might be of no pathologic relevance or it might be the very first hint to the visibility of valvular heart problem [5]. The conventional auscultatory technique of assessing cardiac murmurs is based upon their timing, configuration, pitch, intensity, period, and their relationship to pulse and high blood pressure [4]. This information is after that used to establish the need for antibiotic prophylaxis for endocarditis or the prevention of reoccurring rheumatic fever, the necessity of limiting exercise, and the need for additional cardiac examination utilizing noninvasive or intrusive strategy [4]. Cardiac whisperings can arise from three significant mechanisms: high blood circulation rate with normal or uncommon orifices; typical circulation via a narrowed orifice or right into a dilated chamber; and regurgitant circulation with an inexperienced valve or orifice [6].

The intensity of murmurs is rated from I to VI (Table 1). A grade I murmur is barely audible, whereas a grade II murmur is heard quickly. A grade III murmur is loud however without an apparent thrill, and a grade IV murmur is loud however with an excitement. A grade V murmur is incredibly loud and could be heard with only the edge of the stethoscope in contact with the chest. A quality VI murmur is audible with the stethoscope slightly eliminated from contact with the upper body. Murmurs usually are intermediate and rated I-II or II-III. Systolic whisperings of grade III or greater are more probable to be hemodynamically substantial and brought on by

cardiac valvular condition [5]. The regularity or pitch of a murmur is mainly the outcome of the velocity of blood circulation at the site of the murmur's beginning [7]. The murmur of aortic stenosis and aortic sclerosis tends to be higher pitched at the apex compared to the base. The murmur of aortic regurgitation is high pitched because of the large diastolic gradient. The murmur of mitral stenosis is a low-pitched, rumbling murmur due to low speed circulation triggered by a little pressure slope. The holosystolic murmur of mitral regurgitation is created since of the high pressure slope between the left ventricle and left atrium throughout systole. The musical late-systolic whoop or honk of mitral prolapse associates with the timing of the changes in pressure gradient [8].

The timing and arrangement of a heart murmur are necessary considerations in specifying the cause (Table 2). Typically, it is simple to differentiate systolic from diastolic murmurs. With quick tachycardias, nevertheless, it is required to all at once palpate the carotid artery to differentiate systole from diastole (diastole starts following S2) [4]. The configuration of a murmur could be crescendo, decrescendo, diamond shaped (crescendo decrescendo), or plateau. Systolic murmurs could be diamond shaped (aortic stenosis) or plateau (mitral regurgitation) or late-systolic (mitral prolapse). Diastolic murmurs can be decrescendo and high pitched (aortic regurgitation) or mid-diastolic, low-pitched rolls (mitral stenosis) (Table 2).

Table 1. GRADING OF HEART MURMURS

Grade	Definition
I	Barely audible
II	Easily audible
III	Loud but without palpable thrill
IV	Loud and with palpable thrill
V	Audible with stethoscope barely touching chest
VI	Audible with stethoscope off chest wall

Table 2. CHARACTERISTICS OF COMMON VALVULAR LESIONS

	Murmur location	Radiation	Timing	Character
Aortic Stenosis	Right sternal border	Carotids	Systolic; Ends before S ₂	Harsh; Diamond shaped
Aortic Regurg	Left sternal border	Apex	Early diastolic	Blowing whiff
Mitral Regurg	Apex	Axilla	Pansystolic and captures S ₂	Harsh
Mitral Prolapse	Apex	Precordial	Late systolic follows click	Absent or whoop
Mitral Stenosis	Apex	Left sternal border	Diastolic; Follows opening snap	Low-pitched rumble
Tricuspid Regurg	Left sternal border	Apex	Pansystolic	Coarse or blowing

• AORTIC STENOSIS

The Classic Triad of Aortic Stenosis

The traditional triad of three of signs and symptoms for aortic stenosis are angina, syncope, and congestive heart failure symptoms. Angina is a result of decreased coronary blood flow and increased myocardial oxygen need triggered by high afterload; approximately 50% of patients with aortic stenosis likewise have concomitant coronary artery condition [10], [9]. The origin of exertional syncope in aortic stenosis is debatable. Concepts include an exercise caused decrease in peripheral resistance that is unremunerated as a result of a decreased cardiac output with the stenotic valve, and the rainfall of a vasodepressor feedback [11]. The congestive heart failure could be triggered by diastolic dysfunction from boosted left ventricular wall thickness or systolic dysfunction from excess afterload or reduced contractility [3].

Physical Findings in Aortic Stenosis

The most common physical finding is a systolic crescendo-decrescendo (diamond-shaped) cardiac murmur ideally heard over the aortic area and radiating to the neck [4] (Table 2). It additionally could be listened to over the precordium (and not listened to over the breast bone), consequently imitating mitral regurgitation, so-called "Gallavardin's phenomenon" [11], [13]. The carotid upstroke could reduce in amplitude and delay with enhancing severity of stenosis, the

classical pulsus parvus et tardus. The S2 could become softer and single as the A2 diminishes and ultimately goes away as the aortic valve ends up being boosting nonmotile.

Diagnostic Evaluation in Aortic Stenosis

Echocardiography with Doppler examination of the aortic valve provides a reasonably precise noninvasive evaluation of the transvalvular slope and valve orificial location. If the optimal slope is less compared to 50 mmHg or the valve area is estimated to be even more than 0.8 cm² and the patient is asymptomatic (no angina, syncope, or heart disease), the patient could be adhered to securely on a yearly basis [12]. Asymptomatic patients with peak gradients going beyond 50 mmHg or valve locations less compared to 0.8 cm² should be followed a minimum of every 6 months [3], [11]. A cardiology specialist might be useful in adhering to asymptomatic people.

- **AORTIC REGURGITATION**

Clinical Features of Aortic Regurgitation

The clinical signs of aortic regurgitation are the outcome of the huge total stroke quantity and boosted pulse pressure. The cardiac murmur of aortic regurgitation is a blowing diastolic murmur finest heard along the left sternal border and highlighted with a full expiration, with the patient leaning onward [5]. Besides the regular murmur of aortic regurgitation, a diastolic rumble (Austin-Hint murmur) simulating mitral stenosis could additionally be heard over the pinnacle. The Austin-Hint murmur most likely is created by the aortic regurgitant jet impinging on the mitral valve, triggering it to vibrate along with compelling a loved one closure of the valve and a physiologic a stenosis [2], [3]. The peripheral signs of a hyperdynamic circulation likewise are a sign of severe illness. Consisted of amongst the peripheral signs of aortic regurgitation are Quincke's pulse (systolic plethora and diastolic blanching of the nail beds, compared to the

contemporary pulse oximeter), Corrigan's pulse (the water-hammer bounding pulse with a quick downstroke appreciable in the carotid or brachial arteries), Musset's indication (head bobbing or titubation), the pistol shot indication (when a stethoscope is held gently over the femoral artery), Doroziev's indication (a to-and-fro murmur when a stethoscope is pushed over the femoral artery), and Hill's indication (a systolic differential above 30 mmHg between the leg and arm) [1], [13]. Electrocardiogram may be normal or could disclose left ventricular hypertrophy and nonspecific ST-T adjustments. Echocardiogram with Doppler evaluation of the aortic valve might disclose the extent of the aortic regurgitation. End diastolic left ventricular diameter associates with severity of illness. Left ventricular ejection portion typically is normal at rest yet diminished left ventricular ejection portion with physical effort is worrisome. Angiography during cardiac catheterization before surgery also can validate the intensity of illness.

- **MITRAL REGURGITATION**

Mitral regurgitation normally is triggered by infective endocarditis, burst chordae tendineae and papillary muscle dysfunction, collagen vascular condition (lupus), and rheumatic fever [1], [14]. It may likewise be the outcome of annular dilatation from a dilated cardiomyopathy. Myxomatous deterioration and mitral valve prolapse is covered in the area that complies with. The pathophysiologic phases of mitral regurgitation commonly proceed from acute mitral regurgitation to chronic made up mitral regurgitation and finally to chronic decompensated mitral regurgitation [1], [3]. Throughout acute mitral regurgitation, signs of left-sided congestive cardiac decompensation (dyspnea on exertion, orthopnea, and paroxysmal nighttime dyspnea) may exist. The patient might end up being asymptomatic as chronic made up mitral regurgitation supervenes. If chronic decompensated mitral regurgitation creates, the patient could once more materialize dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea [1], [3]. Acute

mitral regurgitation is normally symptomatic, whereas chronic mitral regurgitation could continue to be asymptomatic for years. Chronic mitral regurgitation usually is compensated by the growth of left ventricular hypertrophy. The characteristic murmur is a holosystolic apical murmur radiating to the axilla yet transferred throughout the precordium. The loudness of the murmur does not necessarily correlate with intensity of disease. An S₃ gallop recommends that left ventricular hypertrophy is existing and the illness is severe. In enhancement, if a diastolic element to the murmur is heard (relative mitral stenosis) severe condition is suggested. Electrocardiography generally is typical yet sometimes could expose nonspecific findings such as left ventricular hypertrophy or pressure, bundle branch block, left atrial augmentation, or atrial fibrillation [14]. Chest radio graph is commonly normal but might expose left ventricular predominance, left atrial augmentation, or mitral annular calcification [14]. Echocardiography and color circulation Doppler verify the augmentation of the left ventricle and the pattern of disrupted flow triggered by regurgitation throughout the mitral valve. Doppler could likewise grade severity by showing just how much right into the left room the regurgitant jet goes. Similarly left atrial inner diameter supplies a clue about seriousness (atrial fibrillation generally supervenes when left atrial interior diameter surpasses 40 mm). Unlike aortic and mitral stenosis, mitral regurgitation could progress insidiously with marked left ventricular damages existing before the start of sign [2], [3]. For that reason, also if the patient is asymptomatic, surgery must be considered as soon as evidence of left ventricular dysfunction shows up. Cardiology consultation may be practical in adhering to patients that are clinically asymptomatic. Preload is boosted in mitral regurgitation, whereas afterload generally is regular and even decreased. In the existence of normal muscular tissue function, as a result, the ejection fraction usually is supernormal in mitral regurgitation. As soon as the ejection portion drops listed below 60%, the prognosis worsens [2].

- **MITRAL VALVE PROLAPSE**

Mitral valve prolapse refers to a team of conditions in which the mitral valve leaflet(s) prolapse into the left room throughout systole. Several or else healthy and balanced and asymptomatic females may manifest this phenomenon? The most usual pathologic cause is myxomatous degeneration of the valve [2], [14].Other pathologic causes include Marfan's disorder, collagen vascular disorders, and coronary artery condition [2], [14]. Most of patients with mitral valve prolapse are asymptomatic. Some might experience atypical chest pain, palpitations, light-headedness, or simple fatigability. On exam, a mid-systolic click and late systolic murmur could be listened to [4].The majority of young, otherwise healthy and balanced, and asymptomatic females may only materialize a midsystolic click without a murmur. The murmur might have a music quality and has been otherwise called a whoop or honk [9].The electrocardiogram in mitral valve prolapse usually is typical, but may have nonspecific ST and T wave abnormalities, especially in the substandard leads. Echocardiography is the analysis modality of option if one prefers to verify mitral valve prolapse. Once again, a young otherwise healthy and balanced and asymptomatic lady with a midsystolic click does not require an echocardiogram. The echocardiographic medical diagnosis is based upon the appearance of one or both leaflets above the annular plane throughout systole. If the mitral valve is misshapen, repetitive, and enlarged, difficulties are more suitable to occur. Aberrant shutoffs are extra common in older guys; aberrant valves and problems are rare in asymptomatic ladies. Doppler might additionally estimate the degree of frank mitral regurgitation. Most of patients with mitral valve prolapse are asymptomatic and require no therapy. Endocarditis treatment is indicated for those that have a murmur or frank mitral regurgitation on echocardiography [2], [15].Beta blockers, diltiazem or verapamil might be helpful in those patients with chest pain or palpitations [2].Beta blockers have

the advantage of opposing the sympathetic tone, which typically is enhanced in these individuals. When mitral valve prolapse has led to serious mitral regurgitation, after that the regurgitation is managed making use of the principle detailed above for mitral regurgitation.

- **MITRAL STENOSIS**

Mitral stenosis is most commonly a sequela of rheumatic heart problem and even more common in women. The occurrence of rheumatic high temperature and for that reason mitral stenosis has declined gradually over the past numerous years in established countries, but both rheumatic fever and mitral stenosis continue to be usual in developing nations [3]. The symptoms of mitral stenosis include dyspnea on effort, orthopnea, and paroxysmal nighttime dyspnea. A later and less frequent sign, hemoptysis, might be viewed as a result of pulmonary hypertension [14]. If an otherwise asymptomatic lady with mitral stenosis conceives, then decompensation can take place as a result of volume shifts [14]. The signs and symptoms originate from increase in left atrial pressure and decreased cardiac output. Although the symptoms are those of left ventricular failure, left ventricular contractility generally is normal in mitral stenosis. Since the right ventricle eventually carries the ball of the afterload troubled it by high left atrial pressure, secondary pulmonary hypertension (and hemoptysis) could be seen [1].

Physical Findings in Mitral Stenosis

A malar flush (mitral facies) may be seen [14], [13]. Famous jugular a waves may be seen if the patient is in regular sinus rhythm [14]. Mitral stenosis is thought on auscultation due to a diastolic reduced pitched rumble that follows an opening breeze. The very first heart sound is commonly loud due to the fact that the force of ventricular systole compels the valve shut [13]. The diastolic roar later has a presystolic accent most likely owing to atrial tightening till atrial fibrillation

supervenes. The presence of a loud P2, ideal ventricular lift, jugular venous distension, or peripheral edema are ominous indicators suggesting that pulmonary hypertension has supervened.

- **TRICUSPID REGURGITATION**

Diagnostic Tests in Tricuspid Regurgitation

The electrocardiogram generally is nonspecific however may expose a rightward QRS axis and proof of ideal ventricular hypertrophy or strain. Right ventricular augmentation might appear on chest radiograph as obliteration of the retrosternal air space. The echocardiogram could show both right atrial and right ventricular augmentation and evidence of dilated cardiomyopathy. Doppler assessment of the tricuspid valve usually reveals retrograde systolic blood circulation. Vegetations could be imaged if infective endocarditis is the reason of the tricuspid regurgitation but a transesophageal echocardiogram commonly is needed to far better picture vegetations [15].



Conclusion:

Presented the high prevalence of valvular heart illness, primary care physicians have to be familiar with the most common valvular heart illness and their clinical manifestations. Knowledge of, the natural history of the most usual valvular heart diseases is essential because the onset of signs usually is the point at which intervention comes to be necessary. Most valvular heart diseases are amenable to surgical treatment, which can manage a symptom-free and relatively normal lifespan. For that reason, primary care physicians must be familiar with the indications for therapeutic interventions and one of the most proper treatments currently recommended.

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