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Clinical and Echocardiographic Correlation of the Mitral L-Wave: An Observational Study

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Abstract

Background: The mitral L-wave is a mid-diastolic transmitral Doppler flow signal that is often underrecognized in routine echocardiography despite its diagnostic and prognostic importance. This observational study evaluated the clinical and echocardiographic correlates of the mitral L-wave among patients attending a tertiary care general medicine department. **Methods:** Fifty-three patients with an echocardiographically confirmed mitral L-wave were studied over 6 months (April 2024–September 2024) at the Department of General Medicine, L.N. Medical College and J.K. Hospital, Bhopal. Clinical presentation, comorbidity profile, and the response of the L-wave to the Valsalva maneuver were recorded and analyzed descriptively. **Results:** A mitral L-wave was frequently observed in patients with coronary artery disease and chronic kidney disease (50.9% each), and hypertension and type 2 diabetes mellitus (49.1% each), consistent with elevated left ventricular filling pressures. Dyspnea on exertion (62.2%) was the most common presenting symptom, and females constituted a slightly higher proportion (52.8%) of the cohort. Eight patients (15.1%) showed a preload-dependent change in L-wave amplitude during the Valsalva maneuver. **Conclusion:** The mitral L-wave is a reproducible, load-sensitive Doppler finding associated with diastolic burden that may assist in the early detection and management of heart failure with preserved ejection fraction.

Keywords: Echocardiographic, Mitral, Clinical

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Introduction

The mitral L-wave is a mid-diastolic transmitral inflow velocity that occurs between the E- and A-waves on pulsed-wave Doppler, and may be accompanied by concordant mid-diastolic leaflet motion on M-mode. It reflects continuing left atrium-to-left ventricle (LA→LV) flow during diastasis and is typically recognized when the mid-diastolic velocity "bump" is discrete and ≥20 cm/s.[1] Color M-mode echocardiography has been used to directly visualize this phenomenon, demonstrating that the L-wave corresponds to a slowly propagating column of blood re-entering the ventricle from a distended, non-compliant left atrium during diastasis – a mechanism that has been documented even in the presence of significant mitral regurgitation, confirming that the L-wave is a genuine forward-flow event rather than a Doppler artefact.[2] Because it is easily overlooked on a standard transmitral trace unless specifically sought, its clinical importance is likely under-appreciated relative to its potential diagnostic yield in routine practice [Figure 1].

Hemodynamic Basis

During early diastole, the E-wave is driven by the atrioventricular pressure gradient created by left ventricle (LV) relaxation and restoring forces. In some patients, pressure crossover reverses and then re-establishes a positive LA→LV gradient during diastasis, producing a second inflow wave – the L-wave. Proposed mechanisms include delayed ("two-step") LV relaxation, elevated left atrial pressure (LAP)/LV filling pressure, slow heart rates that prolong diastasis, and mechanical dyssynchrony.[3] These mechanisms are not mutually exclusive, and more than one may coexist in an individual patient, which likely contributes to the variability in L-wave amplitude observed even within the same recording.

- Diastolic function is routinely assessed with transmitral spectral Doppler (E/A ratio, deceleration time) to estimate LV relaxation and filling pressures – the framework within which L-wave physiology is interpreted.[4]
- Classic descriptions of M-mode/Doppler timing of diastole, and the value of M-mode for capturing mid-diastolic leaflet motion, underpin recognition of mid-diastolic events such as the L-wave.[5] The 2016 American Society of Echocardiography/ European Association of Cardiovascular Imaging diastology recommendations remain the practical reference standard for interpreting transmitral inflow/tissue Doppler imaging (TDI) and estimating LAP.[1]

The two-step relaxation hypothesis proposes that left ventricular relaxation in some patients does not proceed as a single smooth pressure decline but instead pauses or transiently reverses during diastasis, allowing LAP to once again exceed left ventricular pressure for a brief interval and drive a second, smaller volume of forward flow across the mitral valve before atrial contraction produces the A-wave. Because the L-wave depends on this secondary pressure crossover rather than on the primary relaxation gradient, its amplitude tends to be smaller and more variable than that of the E-wave, and it can be intermittently present or absent within the same patient depending on momentary loading conditions, heart rate, and rhythm.

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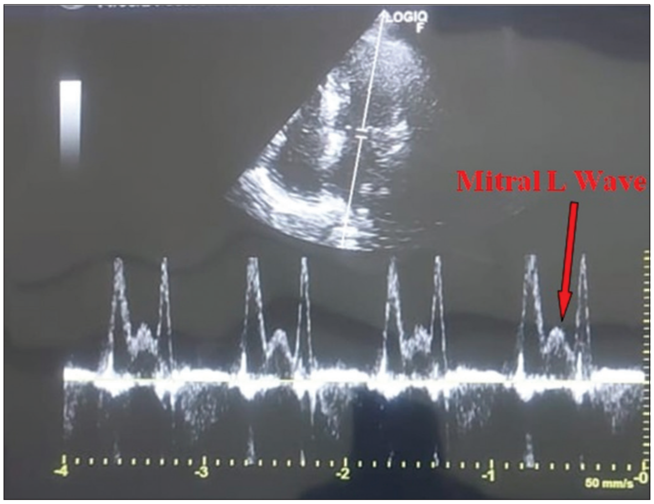

Figure 1: Pulsed-wave Doppler tracing of transmitral inflow showing the mid-diastolic mitral L-wave (between the E and A waves)

Slow heart rates are particularly permissive of L-wave expression because they prolong the diastasis period during which the secondary pressure crossover can occur; conversely, at faster heart rates the diastasis interval shortens and may disappear altogether, fusing the L-wave with the A-wave. Mechanical dyssynchrony between different segments of the LV, whether from conduction abnormalities, prior myocardial infarction, or ventricular pacing, has similarly been proposed to create localized pressure gradients capable of generating a discrete mid-diastolic inflow signal, offering a further explanation for why the L-wave has been documented across such a broad range of underlying cardiac pathologies.

Evaluation of Diastolic Dysfunction

Diastolic dysfunction is evaluated using:

- Color Doppler echocardiography and TDI;
- Strain analysis from cardiac magnetic resonance imaging;
- Strain analysis from speckle-tracking echocardiography.

Its diagnostic framework includes signs and symptoms of heart failure, a left ventricular ejection fraction (LVEF) ≥50%, and evidence of increased LV end-diastolic pressure (LVEDP), impaired filling, delayed isovolumic relaxation, or increased LV stiffness.[6] These criteria are typically applied in combination rather than in isolation, since no single parameter reliably distinguishes preserved from impaired diastolic function across the full spectrum of clinical presentations.

The value of assessing the L wave alongside other diastolic indices, rather than in isolation, is illustrated by case reports, in which a mitral L-wave on pulsed-wave Doppler was accompanied by a corresponding L′-wave on medial mitral annular TDI and a raised E/E′ ratio in a patient presenting with progressive dyspnea; together, these concordant findings confirmed advanced diastolic dysfunction with elevated filling pressures that would otherwise have been easy to underestimate from the transmitral trace alone.[7]

Within the broader framework of diastolic dysfunction grading, the L-wave does not currently form part of the standard grading algorithm recommended by major echocardiography societies, which instead relies primarily on septal and lateral e′ velocities, the E/e′ ratio, left atrial volume index (LAVI), and peak tricuspid regurgitation (TR) velocity. The L-wave should therefore be regarded as a supplementary, corroborative sign rather than a replacement for these validated parameters; its principal clinical value lies in flagging cases where the standard indices may be borderline, technically difficult to obtain, or discordant with the clinical picture, prompting a more detailed diastolic work-up.

Differentiating the L-Wave from Mimics

Distinguishing a true mitral L-wave from a summated E- and A-wave in sinus tachycardia, or from a prominent A-wave in first-degree atrioventricular block, requires careful attention to timing: The L-wave characteristically follows complete deceleration of the E-wave to baseline and remains distinct from the subsequent A-wave, whereas fused E-A waves in tachycardia do not show this intervening return to baseline. Reported prevalence of the L-wave varies widely across populations – from roughly one in 20 to over one in three patients – depending on the underlying cardiac substrate studied, reflecting differences in heart rate, LAP, and the sensitivity of the imaging protocol used to detect it. This variability underscores the importance of a standardized, deliberate search for the L-wave rather than incidental detection, particularly in general medicine settings where echocardiography is often performed primarily to assess ejection fraction and valvular structure rather than diastolic filling in detail.

Clinical Significance of the Mitral L-Wave

- Marker of advanced diastolic dysfunction: Across reviews and case series, the mitral L-wave correlates with larger left atrial volume, and worse diastolic grades, and is considered a marker of elevated LV filling pressures rather than a benign finding.[8]
- Heart failure (heart failure with preserved ejection fraction [HFpEF]/heart failure with reduced ejection fraction [HFrEF]): Mitral L-wave can be observed in both HFpEF and HFrEF and it is associated with poorer prognosis independent of LVEF.[9]
- Atrial fibrillation (AF): The mitral L-wave is not rare in AF, where it indicates advanced diastolic dysfunction and elevated LV filling pressures.[10] It also carries prognostic value for rhythm outcomes after intervention – patients with a mitral L-wave documented before radiofrequency catheter ablation for AF have shown significantly higher rates of late arrhythmia recurrence than those without, suggesting that pre-existing diastolic burden may work against durable maintenance of sinus rhythm.[11]
- Volume-overload states/Chronic kidney disease (CKD): Dedicated studies of the L-wave specifically in CKD remain limited, but volume overload and elevated LAP are well documented in CKD populations, and studies of left ventricular diastolic dysfunction in patients with pre-dialysis CKD show that fluid overload, hypoalbuminemia, and metabolic derangement together promote the same hemodynamic substrate that favors L-wave expression.[12] It should therefore be assessed alongside standard diastolic indices (E/A, LA volume, and TR velocity) in this population.
- The mitral L-wave has also been described as an isolated finding in some otherwise healthy individuals, particularly women and those with sinus bradycardia, in whom it may

not necessarily denote pathological diastolic burden. Its clinical significance should therefore always be interpreted alongside the patient's overall hemodynamic, structural, and symptomatic profile rather than as an isolated Doppler observation.

- Historical perspective: The phenomenon was first described by Keren *et al*. in 1986, who named it the "L-wave" because it follows the "J" and "K" pulmonary vein flow waves corresponding to the systolic and diastolic phases, respectively.[13] It is often observed in sinus bradycardia, AF, and more commonly in females; in HFpEF, it denotes raised LVEDP.
- Prognostic implications: Acute, reversible L-waves may occur with a sudden rise in LV filling pressures, and their resolution can reflect therapeutic response to diuretics – a pattern documented serially in a heart failure patient in whom a giant L-wave diminished and eventually disappeared as congestion improved with treatment.[14] The presence of an L-wave has more broadly been linked to an increased risk of heart failure events and all-cause mortality.[8,9]

Methodology

An observational study was conducted in the Department of General Medicine, J.K. Hospital, Bhopal, over a period of 6 months, from April 2024 to September 2024.

Inclusion and Exclusion Criteria

- Inclusion criteria: Patients aged ≥18 years demonstrating a clearly visualized mid-diastolic L-wave on transmitral pulsed-wave Doppler echocardiography
- Exclusion criteria: Patients aged <18 years.

Echocardiographic Assessment

All patients underwent standard transthoracic echocardiography, with pulsed-wave Doppler interrogation of transmitral inflow performed from the apical four-chamber view with the sample volume positioned at the tips of the mitral leaflets. The mitral L-wave was identified as a discrete, reproducible mid-diastolic forward-flow velocity of ≥20 cm/s occurring after complete deceleration of the E-wave and before the onset of the A-wave, in keeping with previously described criteria.[1] Demographic details, presenting symptoms, and major comorbidities – hypertension, type 2 diabetes mellitus, and coronary artery disease (CKD) – were recorded from clinical history, physical examination, and review of case records for each patient.

In a subset of patients, the behavior of the L-wave and the concurrent E-wave velocity during the Valsalva maneuver was recorded to assess the preload dependence of the finding. The maneuver was performed using the standard forced expiratory technique against a closed glottis, sustained for approximately 10 s while continuous pulsed-wave Doppler recording of transmitral inflow was maintained. Reduction in venous return during the strain phase transiently lowers left ventricular preload, allowing pseudonormalized filling patterns to unmask the underlying degree of diastolic impairment; any consequent change in E-wave and L-wave amplitude was recorded qualitatively as present or absent. This maneuver was performed only in patients who were hemodynamically stable and able to cooperate with the required breath-hold, which is one reason the response could be assessed in only a subset of the overall cohort.

Statistical Analysis

Data were summarized as frequencies and percentages for categorical variables. Results are descriptive in nature; no comparative statistical testing (e.g., Chi-square analysis against a non-L-wave comparator group) was performed, and the associations reported here should be interpreted as observed patterns within this L-wave-positive cohort rather than statistically tested correlations. Categorical variables are reported as counts with percentages throughout, and no imputation was performed for any missing data field.

Ethical Considerations

This study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all patients before inclusion.

Results

Demographics and Comorbidity Distribution

A review of 53 patient cases revealed the following distribution of gender [Table 1] and major comorbidities [Table 2 and Figure 2].

Clinical Presentation

The most frequent presenting symptom was dyspnea on exertion (62.2%), followed by chest pain (39.6%). A subset of patients also exhibited giddiness, palpitations, and bradycardia [Table 3]. These presenting complaints were broadly consistent with those expected in a general medicine population being evaluated for possible cardiac causes of breathlessness or chest discomfort [Figure 3].

Table 1: Gender distribution of the study population (*n*=53)

Gender	*Number*	*Percentage*
Females	28	52.8
Males	25	47.2
Total	53	100

Table 2: Comorbidity distribution among the study population (*n*=53)

Comorbidity	*Patients (n=53)*	*Percentage*
Coronary artery disease	27	50.9
Chronic kidney disease	27	50.9
Hypertension	26	49.1
Type 2 diabetes mellitus	26	49.1

Table 3: Frequency of presenting symptoms among the study population (*n*=53)

Symptom	*Frequency (n=53)*	*Percentage*
Dyspnea on exertion	33	62.2
Chest pain	21	39.6
Giddiness	12	22.6
Palpitations	9	17.0
Bradycardia	5	9.4
Syncope	3	5.6

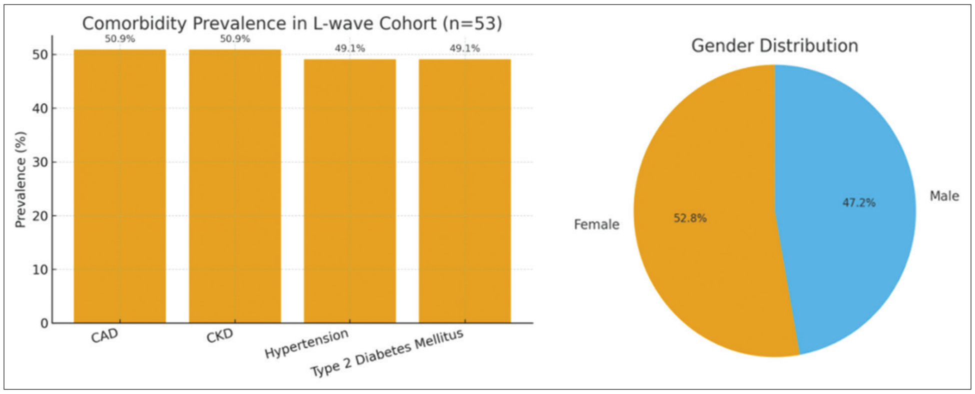

Figure 2: Comorbidity prevalence and gender distribution among the 53 patients with a mitral L-wave

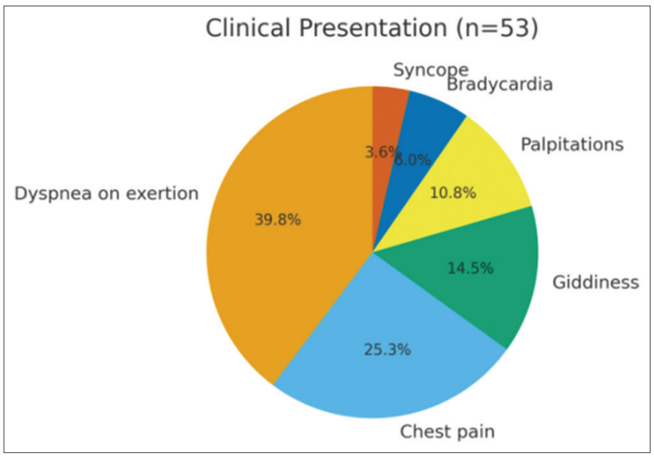

Figure 3: Distribution of presenting symptoms, expressed as a percentage of total symptom occurrences (33 + 21 + 12 + 9 + 5 + 3 = 83 total occurrences); as symptoms were not mutually exclusive, these values differ from the per-patient percentages of $n = 53$ shown in Table 3

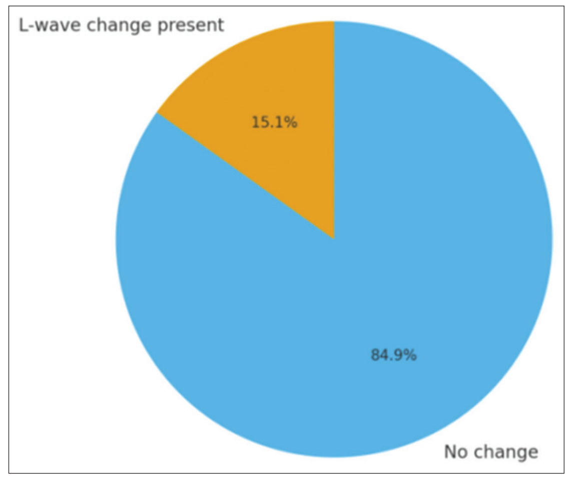

Figure 4: Proportion of patients showing a preload-dependent mitral L-wave response to the Valsalva maneuver ($n = 53$)

Valsalva Maneuver Response

Among the 53 patients, 8 (15.1%) demonstrated preload-dependent changes in the mid-diastolic mitral L-wave during the Valsalva maneuver. In these patients, E-wave amplitude showed diminution (a pseudonormalization pattern), accompanied by a change in amplitude of the mitral L-wave. This hemodynamic response indicates that the mitral L-wave in these cases was sensitive to changes in the left ventricular filling pressures, suggesting a load-dependent component to its generation. The remaining 45 patients (84.9%) did not show significant variation in the L-wave pattern during the maneuver [Figures 4 and 5].

Discussion

The high prevalence of the mitral L-wave among patients with hypertension, diabetes, CAD, and CKD in this cohort supports its use as a marker of elevated filling pressures and diastolic dysfunction, echoing findings reported across diverse clinical settings ranging from systolic and diastolic heart failure[8] to CKD, where volume overload and impaired albumin and glucose homeostasis similarly predispose to a raised diastolic burden.[12]

Unlike traditional metrics that may fluctuate, the L-wave reflects mechanical interplay during diastasis and may help bridge the gap between echocardiographic findings and patient symptoms in the setting of preserved ejection fraction. Its disappearance following decongestive therapy in prior reports highlights its dynamic nature and potential use as a non-invasive marker of therapeutic response – in one documented case, a giant L-wave present at admission with acute decompensated heart failure progressively diminished and eventually disappeared as congestion resolved with treatment, reappearing when the underlying rhythm and rate changed, underscoring how closely the finding tracks real-time loading conditions rather than fixed structural disease.[14]

Females represented a slightly higher proportion of the study population (52.8%), consistent with prior literature indicating a greater prevalence of the mitral L-wave among women, a pattern that has also been noted more broadly in HFpEF, where women are disproportionately represented and often carry a distinct risk-factor profile dominated by hypertension and obesity rather than ischemic heart disease.[15] This sex distribution is a useful reminder that HFpEF and its associated Doppler findings should not be overlooked in women presenting with breathlessness, even in the absence of classical ischemic risk factors.

An L-wave ≥ ~20 cm/s together with an enlarged LAVI supports elevated LAP and advanced diastolic dysfunction, particularly in HFpEF.[16] In the present cohort, the L-wave was most frequently observed alongside comorbidities – CAD, CKD,

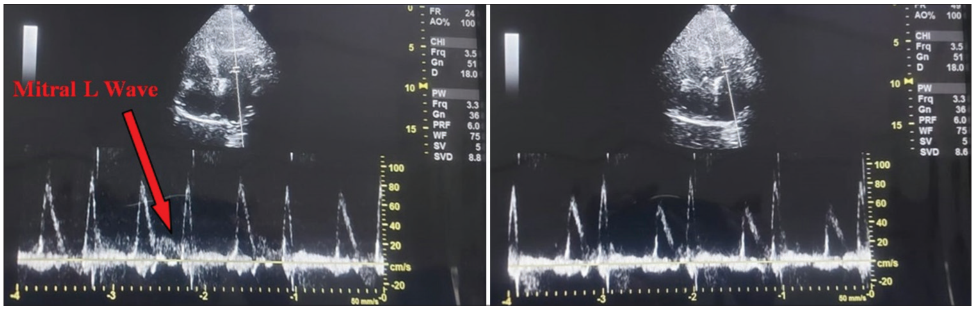

Figure 5: Mitral L-wave at baseline (left) and during the Valsalva maneuver (right), showing diminished amplitude – an example of a preload-dependent response

hypertension, and type 2 diabetes – that are individually well established contributors to elevated LV filling pressure, raising the possibility that L-wave positivity in a patient with several of these comorbidities together may flag a subgroup at particularly high risk of clinically unrecognized diastolic dysfunction or evolving HFpEF. Where LAVI measurement is not routinely available, as in many general medicine settings, the L-wave may offer a simpler bedside surrogate worth incorporating into everyday reporting practice.

The coexistence of hypertension, CAD, and CKD within the same patient, which was common in this cohort, likely reflects shared upstream drivers such as chronic pressure overload, myocardial fibrosis, and impaired renal sodium handling, all of which converge on the same final pathway of elevated left ventricular filling pressure that the L-wave is thought to signal. This overlap makes it difficult, in a purely observational design such as this one, to attribute the presence of an L-wave to any single comorbidity in isolation, and it is more appropriate to interpret the L-wave as a marker of cumulative cardiometabolic and renal burden rather than as being specifically caused by any one condition. This is best regarded as a hypothesis-generating observation, to be tested formally in a study designed with an adequate comparator group.

In the subset of patients undergoing the Valsalva maneuver, both diminution and augmentation of L-wave amplitude were observed alongside E-wave pseudonormalization, reinforcing that L-wave expression is, at least in part, preload-dependent rather than a fixed structural finding.[3] This preload sensitivity has practical implications: a single, isolated measurement of the L-wave under resting conditions may under- or overestimate the severity of underlying diastolic dysfunction if it is not interpreted alongside a dynamic maneuver such as Valsalva, particularly in patients being considered for reclassification of diastolic grade or for a trial of decongestive therapy.

The comorbidity pattern observed in this cohort broadly parallels that described in prior L-wave literature, where hypertension, diabetes, CAD, and impaired renal function recur as the dominant clinical backdrop against which the L-wave is detected, whether the index population was drawn from a dedicated heart failure clinic, an electrophysiology laboratory performing pre-ablation assessment, or, as here, a general medicine ward. In a country such as India, where hypertension, type 2 diabetes, CAD, and CKD collectively constitute a substantial and growing share of the non-communicable disease burden encountered in general medicine practice, a simple, reproducible Doppler sign that can be obtained during a routine transthoracic study without additional cost or specialized equipment may have particular value as a low-resource screening adjunct for diastolic dysfunction and early HFpEF.

From a practical standpoint, the findings of this study suggest that clinicians in general and internal medicine settings, who often request echocardiography primarily to exclude structural heart disease or to quantify ejection fraction, should specifically ask the reporting sonographer or cardiologist to comment on the presence or absence of a mitral L-wave when a patient has hypertension, diabetes, CAD, or CKD and presents with unexplained dyspnea, since this single additional observation may meaningfully raise or lower the pre-test probability of clinically significant diastolic dysfunction without requiring any additional imaging time, equipment, or cost.

Interpreting Tables 1-3 together, the demographic and symptom profile of this L-wave-positive cohort – a slight female predominance, a high burden of cardiometabolic and renal comorbidity, and dyspnea on exertion as the leading presenting complaint – is broadly consistent with a population at risk of, or already progressing toward, HFpEF, even though a formal HFpEF diagnosis using the full ejection fraction, natriuretic peptide, and structural criteria recommended by current guidelines was not separately adjudicated for each patient in this analysis.

Taken together, these observations support incorporating a deliberate search for the mitral L-wave into routine transmitral Doppler assessment, particularly in patients with hypertension, diabetes, CAD, or CKD who present with unexplained dyspnea, since its presence may prompt earlier consideration of HFpEF even when LVEF and standard E/A indices appear reassuring. Wider recognition of the L-wave could also inform decisions around diuretic titration and rate control, given its documented responsiveness to changes in volume status and heart rate.[11,14] Larger, prospective, multicenter studies incorporating a matched non-L-wave comparator group, natriuretic peptide correlation, and longitudinal outcome data are needed to establish the independent prognostic weight of the L-wave in general medicine populations such as the one described here, rather than in the more selected heart failure and electrophysiology cohorts in which most existing evidence has been generated.

Limitations

This was a cross-sectional observational study conducted over a limited period of 6 months, without a comparator group, so the associations described reflect the composition of this L-wave-positive cohort rather than a tested statistical correlation with comorbidities.

Conclusion

In this observational study of 53 patients, the mitral L-wave emerged as a reproducible, load-sensitive Doppler finding associated with diastolic burden in patients with hypertension, diabetes, CAD, and CKD. Its detection may aid the early recognition of HFpEF and help guide tailored management strategies. Routine evaluation for the L-wave as part of standard echocardiographic protocols – particularly in patients with these comorbidities presenting with unexplained dyspnea – may improve early detection of diastolic dysfunction and thereby enhance patient care. Larger prospective studies with a comparator arm and longitudinal follow-up are warranted to define its independent prognostic value. Wider awareness of this simple Doppler sign among treating physicians, not only cardiologists, may be an important first step toward realizing this potential in everyday clinical practice.

Conflicts of Interest

The authors declare no potential conflicts of interest with respect to research, authorship, and/or publication of this article.

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