How to Do Echo in Septic Cardiomyopathy: A Consensus Statement of the Italian Society of Echocardiography and Cardiovascular Imaging

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Abstract

Septic cardiomyopathy (SCM) is an acute, reversible myocardial dysfunction occurring in the context of sepsis, independent of ischemic heart disease. Despite its frequent occurrence in critically ill patients, SCM remains poorly defined and underdiagnosed. This article provides clinicians with a practical guide for the recognition and management of SCM, with particular attention to the role of bedside echocardiography. Echocardiography is emphasized as both a diagnostic and hemodynamic monitoring tool to optimize treatment strategies in patients with sepsis and septic shock. Advanced techniques such as speckle-tracking echocardiography enhance sensitivity for detecting subclinical myocardial impairment and support differential diagnosis. Real-time echocardiographic assessment allows tailored therapy and may improve patient outcomes. Key elements include the recognition of characteristic echocardiographic patterns, integration of global longitudinal strain analysis, and the use of echocardiographic findings to guide hemodynamic management decisions.

Keywords: Echocardiography, monitoring, myocardial dysfunction, myocardial strain, physiologic, sepsis, septic, shock

Introduction

Sepsis and septic shock are major causes of morbidity and mortality in intensive care unit (ICU) worldwide.[1] Among the several complications of sepsis, cardiac dysfunction plays a crucial role in determining patient outcomes. In 1921, E. Romberg first described septic cardiomyopathy (SCM) as "septic acute myocarditis."[2] In 1967, McLean *et al*. described diagnostic criteria for detecting heart failure as part of sepsis included a low cardiac index.[3] In 1984, Parker *et al*., using radionuclide angiography, defined SCM as reversible myocardial depression due to sepsis and septic shock, defined as a reduced left ventricular ejection fraction (LVEF <40%) and an increase in mean end-diastolic volume, excluding acute coronary events, that reversed to normal within two weeks.[4] Prevalence rates vary widely, from 20% to over 50%, depending on the diagnostic criteria used and timing of assessment.[5-7] The risk factors that stand out are male gender, age, high lactate levels at admission, and pre-existing heart diseases.[8] In addition, SCM has been associated with increased mortality, especially when not promptly identified and managed.[2,9]

Definition and Diagnosis

SCM is generally defined as a reversible cardiac dysfunction occurring during sepsis, characterized by ventricular dilation

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with increased ventricular compliance and normal to low filling pressures, in contrast to the pattern of cardiogenic shock, where ventricular pressures are elevated. SCM has also been described as a systolic and/or diastolic impairment of the left and/or right ventricle, in the absence of pre-existing cardiac disease. This condition is usually transient, with recovery observed within 7 to 10 days.[10]

Concerning its diagnosis, serum biomarker dosage is advised. Indeed, N-terminal pro-B-type natriuretic peptide (NT-proBNP) and cardiac Troponin are elevated in 97.4% and 84.5% of patients, respectively, and this increase correlates with a greater degree of LV dysfunction, disease severity, and mortality.[11] Echocardiography has emerged as a cornerstone as both a diagnostic and therapeutic monitoring tool, enabling tailored hemodynamic support.[12,13] Cardiac magnetic resonance imaging (MRI) is also a very useful technique since it shows changes suggestive myocardial edema and inflammation without the presence of focal fibrosis on late gadolinium sequences that are the leading features of SCM.[7] Nonetheless, the leading limiting factor for the application of cardiac MRI in daily practice is the duration of the examination, especially when dealing with hemodynamically unstable patients. In contrast, echocardiography provides several possibilities for the diagnosis of SCM. The method is simple, available, cheap, easily repeatable, and it can be performed "at the bedside" even in critically ill patients.

Currently, five echocardiographic patterns of different hemodynamic phenotypes of SCM can be identified, and they have similar prevalence.[14]

- Cluster 1: Absence of cardiac dysfunction (16.9%)
- Cluster 2: Left ventricular systolic dysfunction (17.7%)
- Cluster 3: Hyperkinetic profile with preserved or supernormal LV systolic function with elevated aortic blood flow velocities (23.3%)
- Cluster 4: Right ventricular (RV) failure (22.5%)
- Cluster 5: Persistent hypovolemia (19.4%).

The highest mortality was recorded in cluster 4 (34% in the intensive care unit, and 22% of 7-day mortality).[15]

Understanding differences between cardiac responses to sepsis and how to best treat them may help inform a more individualized hemodynamic resuscitation. For instance, limiting fluids in patients with LV systolic dysfunction and RV dysfunction, using inotropes only for those patients with systolic dysfunction, prioritizing vasopressin in RV dysfunction, and using beta blockers and ensuring adequate fluid resuscitation for hyperdynamic function and left ventricular outflow tract obstruction (LVOTO), are all theoretical ways to optimize cardiac function in sepsis, but more data are needed.

Recent literature proposes different ways of phenotyping cardiovascular presentations in septic shock, aiming to guide personalized management. Messina and Vieillard-Baron suggest three profiles ("good," "bad," and "ugly").[16] Other studies describe up to four hemodynamic profiles that can be referred to sepsis and septic shock.[17] These classifications overlap in many aspects, highlighting both similarities and nuanced distinctions.

- The "Good" profile aligns with the hyperkinetic profile ("Profile 1"), where left ventricular (LV) function is preserved and CO is maintained or elevated despite reduced systemic vascular resistance. Both groups correspond to the "classic" septic shock phenotype, usually requiring careful titration of fluids, vasopressors, and antibiotics
- The "Bad" profile encompasses patients with either acute LV systolic dysfunction or marked hyperkinesia. This partially overlaps with:
    - Profile 2 (Isolated LV systolic dysfunction), where new-onset SCM leads to decreased cardiac output and elevated LV filling pressures, with high risk of cardiogenic shock
    - Some cases of Profile 1 (Hyperkinetic), when profound vasoplegia persists despite vasopressors, representing fluid-refractory shock with poor prognosis.
- The "Ugly" profile corresponds broadly to Profile 3 (Septic shock with pre-existing cardiac dysfunction), where patients have chronic cardiac disease and may experience overlapping acute and chronic abnormalities. In both schemes, the challenge lies in distinguishing baseline dysfunction from sepsis-induced changes
- Primary cardiogenic shock with superimposed sepsis (Profile 4). This pattern involves primary cardiogenic shock that is subsequently complicated by an infectious process leading to septic shock. Pathophysiology and outcomes of patients in this group can differ significantly.

The "good-bad-ugly" model offers a pragmatic, echocardiography-driven bedside approach, whereas the four-pattern classification of Sato provides a higher level of detail, especially in distinguishing SCM and primary cardiogenic shock syndromes. Combining both frameworks may improve diagnostic precision, clarify therapeutic priorities (fluids, vasopressors, inotropes, and mechanical support), and ultimately enhance personalization of care in septic shock with cardiovascular involvement.

By integrating the conceptual framework proposed by Messina *et al*., with the clinical patterns outlined by Sato *et al*., we developed a summary table that combines echocardiographic features, pathophysiological mechanisms, and therapeutic implications of the main cardiovascular profiles observed frequently in septic shock. This integrated approach aims to provide clinicians with an immediate and practical tool to support early recognition of hemodynamic phenotypes and guide the initial management of critically ill patients, when timely and tailored decisions are crucial for improving outcomes [Table 1].

Table 1: Echocardiographic profiles and management strategies in septic shock and SCM

Integrated echocardiographic profiles and management strategies in SCM			
Profile	**Key echo/ hemodynamic features**	**Pathophysiology**	**Primary bedside therapy**
Hyperdynamic vasoplegic septic shock(“Good” ↔ Profile 1)	Preserved LV systolic function; CO normal/ high; low SVR	Normal systolic function preserved contractility, EF normal; CO may still be inadequate due to vasoplegia	Antibiotics Vasopressors Cautious fluids with small boluses and reassessment.
Hyperkinetic with vasoplegia normal LVED size (“kissing LV”) (subset of “Bad”)	LV hyperkinesia; vasoplegia persists; LV end-diastolic size normal	LV hyperdynamic function hypercontractility, ↓LVESV, CO ↑ if preload normal; pseudohypertrophy; may reflect vasoplegia	Antibiotics Vasopressors Stop fluids
Under-resuscitated hyperkinetic small LV (subset of “Bad”)	Hyperkinesia with small LVED size (±LVOT obstruction)	LV hyperdynamic with LVOT obstruction: anterior mitral motion, outflow obstruction, severely ↓CO despite ↑EF	Antibiotics Fluids carefully titrated Vasopressors as needed.
SCM(“Bad” depressed LV ↔ Profile 2)	New/worsened LV dysfunction; ↓CI; ↑filling pressures; high lactate	LV systolic dysfunction ↓contractility, ↑LVESV, ↓CO; EF may appear normal if low afterload; LV dilation possible	Antibiotics Vasopressors Consider inotropes (dobutamine) if low CI persistsearly echo + PAC.
LV diastolic dysfunction in sepsis(may overlap with all patterns)	Impaired relaxation, reduced LV filling	LV diastolic dysfunction ↓relaxation, ↓LVEDV, ↓CO	Antibiotics Vasopressors Avoid fluid overload (CCE to guide preload).
Septic shock with pre-existing LV dysfunction (“Ugly” or Profile 3)	Chronic cardiomyopathy/ Valvular disease; acute versus chronic overlap	Mix of chronic LV systolic/diastolic dysfunction EF often reduced, variable CO	Antibiotics Vasopressors Restrictive fluids with congestion monitoring
RV dysfunction in sepsis (may overlap with all patterns)	↓RV contractility, ↑RVESV, RV dilation; ↓LV filling by ventricular interdependence	RV dysfunction ↓contractility, RV dilation, ↓LVEDV, ↓CO	Antibiotics Vasopressors Optimize RV preload/afterload Consider pulmonary vasodilators if severe.
Primary cardiogenic shock + superimposed sepsis (Profile 4)	Primary pump failure + infection	As per LV systolic ± RV dysfunction, often severe	Infection source control Vasopressors/inotropes Consider MCS early

LV=Left ventricular, RV=Right ventricular, CCE=Critical care echocardiography, LVED=LV end-diastolic, LVEDV=LV end-diastolic volume, LVESV=LV end-systolic volume, MCS=Mechanical circulatory support, PAC=Pulmonary artery catheter, RVESV=RV end-systolic volume, SVR=Systemic vascular resistance, EF=Ejection fraction

What Key Features to Look For?

Echocardiography plays a central role in identifying SCM.[18,19] [Figure 1]. Most recent studies use echocardiography to evaluate LV systolic function, using <40%–50% as a definition for decreased LVEF. Studies have yielded mixed results with respect to the relationship between LVEF value and outcomes. A 2013 meta-analysis showed no difference in survival based on presence of low LVEF or LV dilation.[20] A challenge with using LVEF is its dependence on preload and afterload conditions. A patient with significant contractile dysfunction can appear to have a normal LVEF in a setting of hypovolemia or low mean arterial pressure, as is often the case in sepsis. LVEF can change rapidly and significantly depending on arterial pressure and volume status, without reflecting the change in true contractility of the myocardium.[21] Thus, it is not surprising that studies based on LVEF have shown such variability in prognostication.

Reduced S′ on tissue Doppler imaging (TDI) is suggestive of systolic dysfunction. It can be abnormal even when EF is preserved; therefore, this parameter helps to detect early or subclinical dysfunction.

Advanced techniques, over TDI, such as speckle tracking echocardiography (STE), further enhance sensitivity for detecting myocardial impairment since it is less dependent on loading conditions and is potentially more reflective of intrinsic myocardial function.[7] STE is an advanced echocardiographic technique that analyzes the motion of natural acoustic markers, called “speckles”, within the myocardium on standard 2D gray-scale images. These speckles, which represent unique interference patterns created by ultrasound scattering, are tracked frame-by-frame throughout the cardiac cycle to assess myocardial deformation in multiple directions: longitudinal, radial, and circumferential.[22,23]

Global longitudinal strain (GLS) is the most commonly used parameter.[24] Normal GLS for LV is more than –18% and for RV, more than –22%. GLS assessment is certainly desirable in subclinical, early assessment of myocardial damage as part of sepsis; the main disadvantage of such a sophisticated method is the difficult availability in the ICU. GLS demonstrated its efficacy in the early identification of cardiac injury, even in patients with mild-to-moderate COVID-19, despite preserved LVEF.[25] Moreover, studies have shown that worse GLS (less negative) is associated with higher mortality in patients with

sepsis, and the same relationship was not established between mortality and LVEF.[26] A specific finding in some patients with hyperdynamic cardiac function is dynamic LVOTO, where blood rapidly flowing through an underfilled ventricle causes anterior motion of the mitral valve and obstruction of flow through the outflow tract, similar to the obstruction seen in hypertrophic obstructive cardiomyopathy. LVOTO is observed in 22%–30% of septic shock patients and can occur due to high catecholamine states, inotrope administration, and hypovolemia, and typically indicates fluid responsiveness; studies show an association between LVOTO and mortality.[26]

The assessment of diastolic function is another important issue in the context of SCM to have an indirect estimation of LV filling pressure (increased E/E'). Moreover, E′ may be reduced due to inflammation and myocardial stiffness. However, since these are patients who are mechanically ventilated, it has been demonstrated that the influence of ventilation on echocardiographic parameters of systolic and diastolic function. Moreover, the failure of weaning from mechanical ventilation is associated with worse diastolic function and increased LV filling pressure.[27]

Finally, it is estimated that RV dysfunction is present in 50–55% of cases, whereas isolated RV dysfunction is present in 47% of cases.[28] RV dysfunction is defined according to the values of tricuspid annular plane systolic excursion (TAPSE), tricuspid lateral annular systolic velocity (TDI S' wave), and RV fractional area change (FAC).[29] Lanspa *et al*. have shown in their research that half of patients with sepsis have RV dysfunction, and it is associated with three times higher 28-day mortality.[30]

Overall, echocardiographic hallmarks of SCM include:

- Left ventricular dilatation with normal or low filling pressures
- Depressed LV and RVEF (not necessarily indicating poor prognosis). Generally, it is a reversible condition
- Absence of regional wall motion abnormalities (RWMAs), differentiating SCM from takotsubo syndrome (TTS)

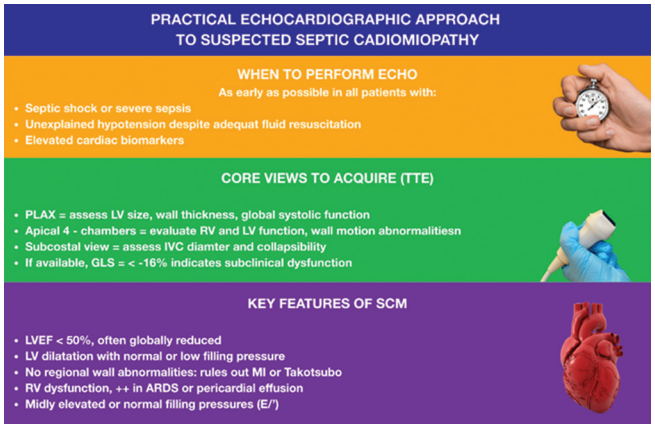

Figure 1: Practical echocardiographic algorithm for suspected septic cardiomyopathy, outlining indications for echocardiography, essential transthoracic views, and characteristic diagnostic features, including left ventricular (LV) ejection fraction, LV and right ventricular function, and GLS thresholds for early detection of subclinical dysfunction

- Global systolic dysfunction rather than focal changes
- Increased end-diastolic volume suggesting elevated ventricular compliance
- RV dysfunction may also be seen, particularly in patients with ARDS or sepsis-induced pulmonary hypertension
- LVOT obstruction (in hyperkinetic profile).

The methodological approach for performing bedside echocardiography, together with the key diagnostic features to be identified and their corresponding cut-off values, is listed in Table 2.

How to Interpret the Findings and How these Findings Can Guide Therapeutic Decisions?

In all patients with septic shock, an early bedside echocardiogram should be performed to evaluate biventricular function, filling pressures, and volume status. If echocardiography suggests cardiac dysfunction, further

Table 2: Standardized protocol and diagnostic thresholds for bedside echocardiographic assessment

How to perform bedside echocadiography	
Parameters	**Cut - off**
Patient position	Supine or 30° semi-recumbent position
Probe	Use low-frequency phased array probe (2–5 MHz)
Views to acquire	PLAX
	PSAX
	A4C
	Subcostal 4 Chamber (if other views are difficult)
	IVC View (Subcostal Long Axis of IVC)
LV systolic function	Visual EF ("eyeballing")
	EPSS >7 mm = ↓ EF[31]
	Fractional shortening: <25% may indicate systolic dysfunction, >45% is rare and could reflect hyperdynamic states (e.g., early sepsis, anemia, thyrotoxicosis) [32]
	Reduced S′ (< 6 cm/s)
	GLS less negative than –18% (e.g., –14%, –12%)
LV Diastolic dysfunction[33]	Left atrium volume (>34 mL/m^2)
	Tricuspid regurgitation velocity >2.8 m/s
	TDI E' velocity <10 cm/s in the lateral annulus and <7 cm/s in the septal annulus
	E/e' ratio >13 in the lateral annulus and >15 in the septal annulus.[6]
RV function[34]	TAPSE < 16 mm
	RV FAC <35%
	RV: LV ratio (> 1 = RV dilatation)
	(TDI S' wave) <15 cm/s
Volume status (Preload), IVC dia/ collapse	>2.1 cm and < 50% collapse=high RA pressure
	<2.1 cm and > 50% collapse=low RA pressure
Pericardial effusion	Echo free space around heart
LVOT VTI[35,36]	LVOT- VTI < 14 c is suggestive of low cardiac output

LV=Left ventricular, PLAX= Parasternal Long Axis, PSAX=Parasternal Short Axis, A4C=Apical 4 Chamber , LVOT=LV outflow tract, EPSS=E – Point Septal Separation, VTI=Velocity time integral, RV=Right ventricular

evaluation should exclude acute coronary syndrome and stress cardiomyopathy. Serial echocardiograms should be used to monitor response to therapy and adjust treatment dynamically.[37] [Figure 2].

Tailored hemodynamic support depending on the echocardiographic profile

- LV systolic dysfunction: Consider inotropes if hypoperfusion persists despite adequate volume resuscitation. There is an important clinical implication, as catecholamine administration typically worsens the obstruction, resulting in unintended worsening of hemodynamic status. Vasopressin instead of adrenergic agents could be used in these patients. Beta-blockers are also a plausible treatment for this patient group, as they are recommended for the treatment of LVOTO in other conditions
- RV dysfunction: Optimize preload and avoid excessive fluid administration; consider pulmonary vasodilators in selected cases
- Normal systolic function with vasoplegia: Prioritize vasopressor support and limit inotropes.

Interpret and integrate data

In addition to guiding therapy, echocardiographic findings should be interpreted in the context of the differential diagnosis:

- Exclude ischemia: RWMAs confined to a coronary artery territory, in a patient with cardiovascular risk factors and/or typical symptoms, supported by electrocardiogram changes and elevated troponin, suggest ischemic cardiomyopathy (ICM) rather than SCM. In this setting, segmental impairment of strain parameters, with non-uniform reduction of GLS, reinforces the suspicion of ischemia[38,39]
- Exclude TTS: Stress-induced cardiomyopathy should be considered when RWMAs extend beyond a single coronary distribution, typically with apical ballooning and relative basal hyperkinesis. On strain analysis, GLS shows marked apical dysfunction with preserved basal segments, distinguishing TTS from the globally depressed strain pattern seen in SCM[40,41]
- Assess volume status to guide resuscitation
- Tailor therapy according to the dominant pathophysiological profile.

Repeat scans

- Perform follow-up echocardiograms whenever clinical status changes
- Document reversibility, typically within 7–10 days, which is a hallmark of SCM and TTS, but less characteristic of ischemic injury[34,42]
- Track response to therapy and adjust hemodynamic support accordingly.

How to Deal with Diagnostic Uncertainty in Septic Cardiomyopathy?

In patients with sepsis, bedside echocardiography is an essential tool for differentiating SCM, stress-induced cardiomyopathy, or TTS, and ICM. Although all these conditions may present with acute left ventricular dysfunction, their echocardiographic patterns and clinical implications differ significantly.

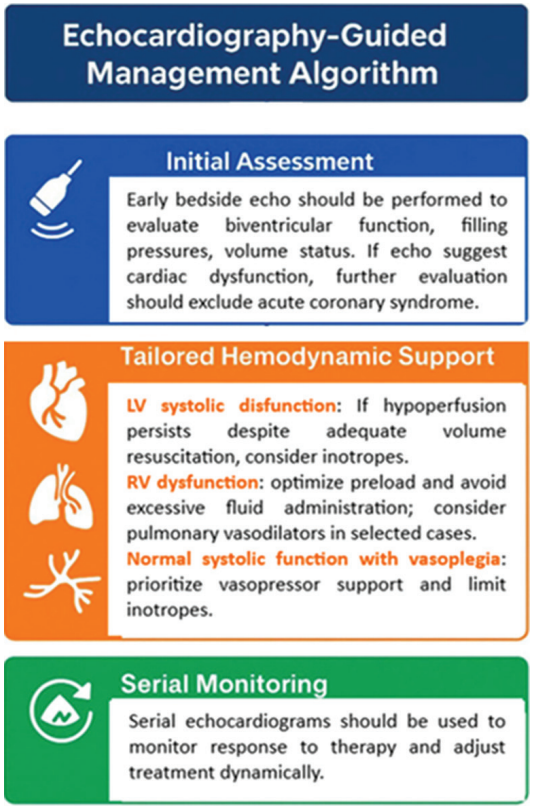

Figure 2: Echocardiography-guided management pathway for septic cardiomyopathy, emphasizing early evaluation of biventricular function, individualized hemodynamic interventions according to systolic dysfunction or vasoplegia, and repeated echocardiographic monitoring to adapt therapy over time

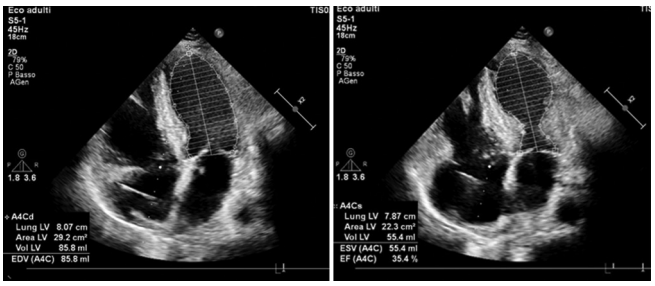

Figure 3: Takotsubo syndrome: typical apical ballooning

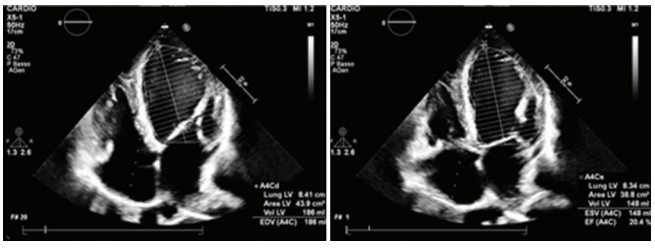

Figure 4: Left ventricular dysfunction in septic cardiomyopathy

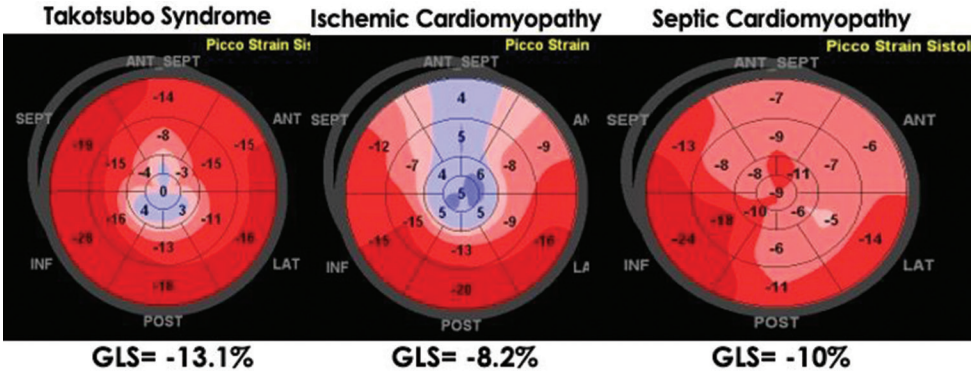

Figure 5: The global longitudinal strain bull's eyes show the typical circumferential pattern of left ventricular strain in takotsubo syndrome (right). This pattern is markedly different from the recognized pattern in anterior AMI (center) where strain alterations reflect site of coronary occlusion and from septic cardiomyopathy (left) showing diffuse reduction of longitudinal strain

Classical echocardiographic hallmarks of takotsubo syndrome

- Presence of RWMAs that extend beyond a single coronary artery territory, most commonly manifesting as apical ballooning [Figure 3 and Video 1]
- Akinesis or hypokinesis of the mid-to-apical segments of the left ventricle with preserved or hypercontractile basal segment[41] whereas in SCM ventricular function is globally depressed [Figure 4 and Video 2]
- RV function is often preserved in TTS, distinguishing it from SCM, where biventricular dysfunction is more frequent.[43]

Even in this clinical scenario, STE adds diagnostic value. In TTS, GLS typically reveals a distinctive reduction in apical strain with relative preservation of basal function, unlike the globally depressed strain seen in SCM.[40,41] SCM is characterized by diffuse, reversible myocardial dysfunction with impairment of GLS across all segments, without respect for coronary distributions.[42] ICM is usually associated with RWMAs confined to a coronary artery territory, and segmental strain values reflect the distribution of ischemia. GLS is reduced in a non-uniform manner, mirroring regional involvement, whereas in SCM the impairment is global and homogeneous.[38,39] This distinction is crucial, as ischemic injury implies underlying coronary pathology requiring specific therapeutic interventions, whereas SCM and TTS are generally transient and reversible conditions [Figure 5].

Conclusion

SCM remains a challenging and often underrecognized complication of sepsis. Echocardiography, particularly at the bedside, plays a central role in identifying SCM and guiding hemodynamic management, permitting a targeted therapy. Its noninvasive nature, ease of use, and ability to guide hemodynamic management (e.g. fluid response, inotropes) make it a first-line modality. Transthoracic echocardiography is not only a reliable snapshot tool but also effective in monitoring cardiac output trends over time in critically ill patients, especially valuable when pulmonary artery catheter placement is contraindicated or not feasible. Standardized use of echocardiographic algorithms may enhance diagnostic accuracy, enable individualized therapy, improving outcomes in this critically ill population.

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Conflicts of interest

There are no conflicts of interest.

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