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A Review of the Role of Transthoracic and Transesophageal Echocardiography, Computed Tomography, and Magnetic Resonance Imaging in Cardioembolic Stroke

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Stroke is a major source of morbidity and mortality worldwide, accounting for the second largest cause of mortality and the third greatest cause of disability. Stroke is frequently preceded by a transient ischemic attack (TIA). The etiologies of 20-30% of ischemic strokes are unknown, and thus are termed "cryptogenic strokes". About 25% of ischemic strokes are cardioembolic. Strokes occur at a rate of around 2% per year in individuals with heart failure with reduced ejection fraction (HFrEF), with a strong correlation between stroke risk and the degree of ventricular impairment. Furthermore, stroke risk is augmented in the absence of anticoagulation therapy. Cardioembolic strokes, when treated inadequately, have a greater predilection for recurrences than atherothrombotic strokes, both early and late in life. The role of a patent foramen ovale in strokes, specifically in "cryptogenic strokes", is a matter of concern that deserves due attention. The use of tissue-engineered heart valves and aspirin for minimizing the risk of stroke is recommended.

Transthoracic echocardiography (TTE) is advantageous for assessing heart function in the acute phase of ischemic stroke. Transesophageal echocardiography (TEE) is considered the criterion standard procedure for detecting LAA thrombi. Computed tomography (CT) scans are good imaging modalities for identifying and excluding bleeding. Magnetic resonance imaging (MRI) images are by far the most effective imaging technique available for assessing the brain parenchymal state.

We conducted a thorough review of the literature on the use of imaging modalities, highlighting the important contribution of TTE, TEE, CT, and MRI in the evaluation of cardioembolic stroke.

Keywords: Atrial Fibrillation • Embolic Stroke

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Background

Stroke is a major source of morbidity and mortality worldwide, accounting for the second largest cause of death and the third greatest cause of disability-adjusted life years (DALYs), as shown in the World Health Organization (WHO) Report on Mortality and Global Health estimates [1]. Frequently, it is preceded by transient ischemic attacks (TIAs). Since the etiologies of 20-30% of ischemic strokes are unknown, they are classified as cryptogenic [2]. However, using novel diagnostic procedures, the cause of 30% of the cryptogenic strokes can be attributed to the cardioembolic events of silent atrial fibrillation (AF) [3]. The most common sustained cardiac arrhythmia is AF, with a prevalence of 2-4% in adults and 5-15% in the elderly [3]. The yearly absolute risk of occurrence of stroke in AF is 3-4% [4-7]. This risk is amplified in patients with asymptomatic and paroxysmal atrial fibrillation (PAF), as these patients are vulnerable due to inadequate use of anticoagulants. Around 15% of TIAs can be attributed to silent PAF [2]. Although Holter monitoring is commonly used for diagnosing PAF, its efficiency is limited by the sporadic nature of PAF and the inability of patients to identify it. Long-term monitoring is only feasible by the use of insertable loop recorders, but their invasive implantation is expensive and thus cannot be achieved in most stroke patients [8]. Situations become even more complex when cardioembolic stroke is distinguished from extracranial and intracranial atherosclerotic strokes, which can be caused by a variety of diverse processes, including in situ thromboembolic obstruction, artery-to-artery embolism, branch occlusion, and circulatory insufficiency [9].

About 25% of ischemic strokes are cardioembolic. Strokes caused by cardioembolic thrombosis are typically more severe than those caused by atherothrombotic thrombosis. Furthermore, they have a greater proclivity for relapses, both earlier and late in life. Therefore, we conducted this review to elucidate the role of transthoracic echocardiography (TTE), transesophageal echocardiography (TEE), magnetic resonance imaging (MRI), and computed tomography (CT) in diagnosing cardioembolic stroke. The following cardioembolic stroke risk factors are among the most common ones.

Cardioembolic Stroke Risk Factors

Atrial Factors

Arrhythmias:

a) *Atrial fibrillation (AF):* It is believed that non-valvular AF is the major cause of cardioembolic stroke [2,3]. It affects about 5% of persons aged 65 and older, with a 17-times greater risk of stroke. In people with AF, the CHADS2 hazard assessment approach is widely used to determine the risk of stroke. Those with valvular AF face a 2- to 7-fold greater risk [4-6,8,10,11].
b) *Sick sinus syndrome*: It is sometimes referred to as bradycardia-tachycardia condition. It has been linked with an increased incidence of stroke as well.

Structural diseases:

a) *Patent foramen ovale:* The role of a patent foramen ovale in strokes, especially in "cryptogenic strokes," is a contentious issue at the moment. Due to the dearth of relevant data, it is difficult to determine whether the foramen promotes the formation of thrombus or whether it merely acts as a conduit for paradox emboli [4,12].
b) *Valvular heart disease*: It amplifies the risk of stroke even in the absence of arrhythmias. These include the following:

Rheumatic valvular disease: The most frequent type of rheumatic valvular disease is rheumatic mitral stenosis; stroke risk is quite high in the absence of anticoagulation.

Endocarditis:

a) *Infective endocarditis (IE)*: According to the 2015 European Society of Cardiology (ESC) Guidelines for the management of infective endocarditis [13] and the and the 2021 ESC Guidelines for the management of valvular heart disease [14], roughly 10% of individuals with IE experience embolic strokes. Stroke risk is greatest before the initiation and during the first 2 weeks of antibiotic medication. Anticoagulation is initiated 7 days following a stroke. Emboli are one of the most frequently occurring complications of IE. Ischemic stroke occurs at a rate of 15-20% in patients with IE, with the risk being highest in the first 7-10 days after presentation. The risk for the development of stroke is greater with the involvement of mitral valve vs the aortic valve. Embolic risk is further increased by the mobility, uniformity, spread, and size of vegetations. Transthoracic echocardiography (TTE) must be performed first, however, owing to its low image resolution, and its sensitivity for identifying vegetation is 62-79%. Transesophageal echocardiography (TEE) is required in situations of questionable TTE and in the presence of an abscess. TEE enhances the detection rate of vegetation to 85-90%. TEE becomes even more crucial for studying prosthetic valve endocarditis and its complications, such as leaflet tears and abscesses (**Figure 1**). TTE has a sensitivity of 20-40% for mitral and aortic valve prosthesis, while TEE has a sensitivity of 80-90%. For abscess diagnosis, TTE has a sensitivity and specificity of 28% and 98%, respectively, compared to 87% and 95% for TEE. Additionally, increased vegetation growth after antibiotic therapy has been linked with an increased incidence of

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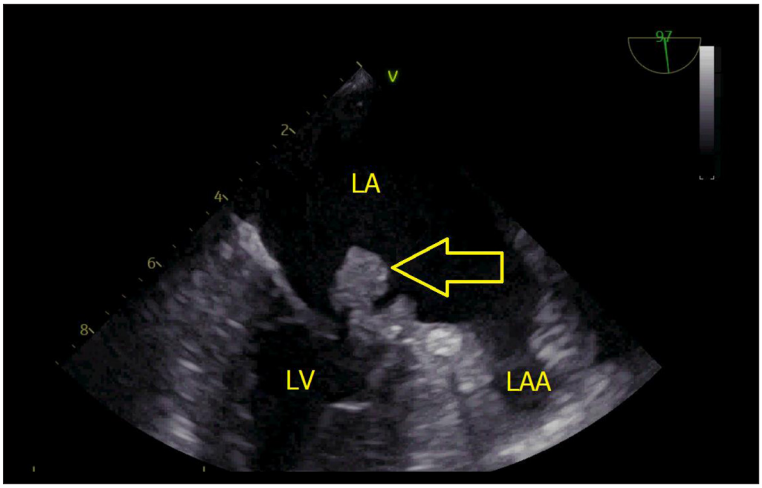

Figure 1. Transesophageal echocardiography. Vegetations of the mitral valve (indicated by yellow arrow). The image was acquired using a VIVID5S, General Electrics phased array ultrasonoscope (Tirat Carmel, Israel). LV – left ventricle; LA – left atrium.

embolism [13]. Treatment must be targeted at the source of the problem. IE necessitates systemic antibiotics and, if necessary, surgical intervention. It is permissible to temporarily cease anticoagulation in patients with IE and signs of cerebral embolism or stroke, independent of the other criteria for anticoagulation. Early surgery (at first hospitalization and or before the completion of one full therapeutic course of antibiotics) is also an option in IE patients with recurring emboli and persistent vegetations despite proper antibiotic treatment. A case series demonstrated a significantly increased rate of embolic incidents with premature anticoagulation. Anticoagulation was an important risk factor for all neurological problems, with a hazard ratio (HR) of 1.31, but was especially linked with a higher rate of hemorrhagic incidents (HR 2.71). The precise timing of anticoagulant use is unknown, as it is dependent on a variety of circumstances (infection is under control, no indication for urgent heart surgery, no large ischemic stroke or cerebral hemorrhage) [15-17].

b) *Marantic endocarditis (ME):* It is a noninfectious endocarditis characterized by the presence of noninfectious valvular vegetations and is observed in patients with neoplasms or other disabling conditions. It carries a significant risk of embolism [18,19]. ME can be effectively treated with antitumor, antiviral, or immunosuppressive medicine in conjunction with a systemic anticoagulant, especially heparin-based anticoagulation.

Native valvular calcifications: Cardioembolic stroke is considerably increased by heart valve disease, particularly mitral valve disease. Calcification of the mitral annulus is associated with a 2.1-fold higher risk of embolic stroke [18].

Mechanical prosthetic heart valves: Even patients on oral anticoagulation have a stroke rate of 2-4%. With an INR of 2.5-3.5, anticoagulation is recommended on a permanent basis. Tissue-engineered heart valves minimize the risk of stroke, and aspirin is recommended unless the affected person has atrial fibrillation [18].

Mitral valve prolapse (MVP): There is a modest risk of stroke associated with MVP; therefore, anticoagulation is not indicated. However, it is advisable to use aspirin on a long-term basis [18].

Ventricular Factors

Ventricular structural and functional diseases:

a) *Aneurysms of the interventricular septum.*

b) *Stroke in patients with heart failure with reduced ejection fraction (HFrEF):* According to the 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure, the annual stroke rate in HFrEF is around 2%, with a strong correlation between the risk of stroke and the degree of ventricular impairment [19].

c) *Myocardial infarction:* Left ventricular dysfunction, ventricular aneurysm and mural thrombus are considered to be risk predictors of stroke. Over 4 weeks of ischemia, approximately 2.5 percent of individuals will get a stroke, and nearly 10% will experience this within the following 6 years [20].

Clinical Characteristics of Cardioembolic Stroke

Cardioembolic stroke is characterized by the following characteristics: sudden deterioration of mental state, presence of neurological impairments, and modification of level of consciousness. Cardiac abnormalities can manifest themselves in the following ways: In the recent past, the patient may have had an arrhythmia (atrial fibrillation), a cardiac murmur, signs of congestive heart failure, acute myocardial infarction, or acute infective endocarditis.

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Cardiovascular Assessment Methods

The first cardiac assessment should focus on both the electrophysiologic and anatomical and functional condition of the heart. As such, all patients must undergo a 12-lead ECG and TTE as part of their workup [4].

Electrophysiologic Evaluation

Twelve-lead electrocardiogram: It is only effective for detecting continuing arrhythmias. Additionally, it can offer important information on the myocardial state of the ventricles (evidence of ventricular hypertrophy) and previous cardiac ischemia events. However, transitory arrhythmias can be overlooked, most notably paroxysmal atrial fibrillation.

Holter monitoring: It is now routinely used on all suspected cardioembolic stroke patients. While it is fundamentally identical to an electrocardiogram, it has the limitation of examining the conduction system for just 24 h.

Implantable loop recorders (ILRs): They can maintain a 3-year record of activities. As a result, their clinical value continues to grow, and their use has aided identification of numerous cases of "missing" atrial fibrillation.

Imaging Methods for Assessment of Structural and Functional Cardiac Abnormalities

Transthoracic echocardiography (TTE): This is advantageous for assessing heart function in the acute phase of ischemic stroke [21]. However, TEE is considered the criterion standard procedure for detecting LAA thrombi. Left atrial (LA) volume enlargement, an elevated pulsed-wave Doppler TMF index, and early diastolic Doppler (E)/late diastolic Doppler (A) are associated with an increased risk of atrial fibrillation (AF) [22,23]. However, several parameters may be different during the acute stroke period when compared to normal circumstances.

Transesophageal Echocardiography (TEE): The transesophageal method enables use of higher-frequency probes with substantially improved imaging resolution, allowing visibility of extremely tiny diseases such as intra-valvar abscesses. Its value as a major cardiac imaging modality is constrained by a higher learning curve and a lack of wide availability [24]. Echocardiography can be used to diagnose the following conditions:

Risk Evaluation and Treatment

Defects of the atrial septum

a) *Patent foramen ovale (PFO)*: This is a remnant of an embryologic circulatory bypass conduit and is seen in roughly 20-30% of autopsy subjects [25]. A study of the connection between PFO and cryptogenic stroke [26,27] revealed a considerably greater incidence of PFO in young individuals with unexplained stroke. The PFO in Cryptogenic Stroke Study evaluated a treatment method for preventing recurrent stroke in individuals with cryptogenic stroke (PICSS). In patients receiving medical therapy (warfarin or aspirin), the presence of PFO had no effect on the risk of adverse events. This is a clinically ambiguous topic that is the subject of ongoing discussion [28].
b) *Atrial septal aneurysm (ASA):* This is diagnosed when the atrial septum is displaced or deviated 10 mm from the midline toward the right atrium or left atrium. There is evidence that ASA can increase the risk of ischemic stroke. A study reported that ASA was much more common in individuals with cryptogenic stroke than in controls (15% vs 4%) [29]. However, individuals with ASA frequently have a coexisting PFO (60-70% of instances), and their PFOs are typically larger. As a result, the increased risk of stroke can reflect the presence of a PFO. The coexistence of the 2 disorders (PFO and ASA) may, however, be a greater risk factor for stroke than either condition alone [30]. TTE and TEE are the most often utilized diagnostic imaging procedures for PFO identification, both of which can be performed with intravenous contrast agent injection to diagnose right-to-left shunting [31]. It is critical to determine whether the interatrial septum is deviated to the LA side, confirming the elevation of RA pressure. When shaken saline contrast is detected in the left atrium 3 cardiac cycles following complete opacification of the right atrial, a PFO is likely. If microbubbles develop later in life, it is hypothesized that they are caused by intrapulmonary shunting. TEE is the criterion standard for diagnosis due to its greater diagnostic accuracy, but because it is semi-invasive, it is frequently reserved for cases needing an exact architectural delineation of the atrial septum. TEE offers direct visualization of the PFO opening and division of the septum primum and secundum, allowing for estimation of the PFO's size. Blood shunting over the PFO can frequently be detected using color flow Doppler [31].

Thrombus Formation

a) *Thrombosis of the atrium*: It occurs more frequently in the left atrial appendage (LAA). It is the most common cause of cardioembolic events and is often linked with atrial fibrillation (AF). Although the risk of thromboembolism is equal in paroxysmal and chronic AF, it is substantially influenced

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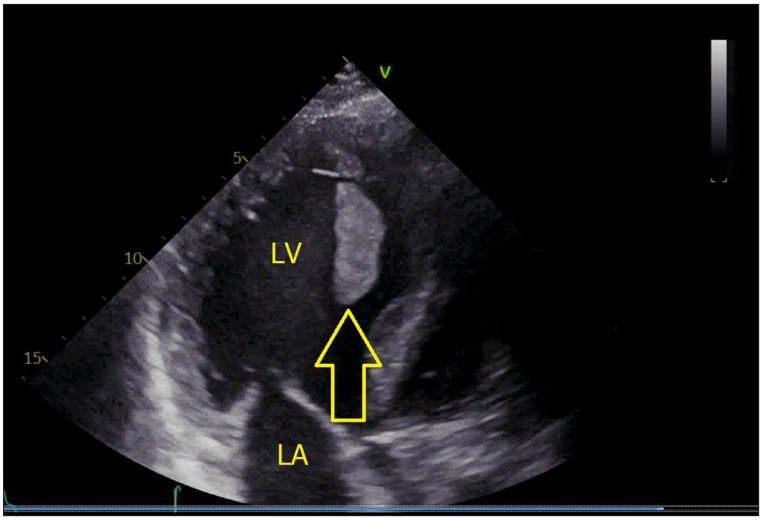

Figure 2. Transthoracic echocardiography. Thrombus in the left ventricle (indicated by yellow arrow). The image was acquired using a VIVID5S, General Electrics phased array ultrasonoscope (Tirat Carmel, Israel). LV – left ventricle.

by concomitant cardiovascular risk factors [4]. The severity and length of these risk factors affects the size of the LA and, subsequently, the chance of AF-associated thrombus development [4]. Clinical prediction tools such as the CHADS2 and CHA2DS2-VASc scores can be used to stratify stroke risk [7,32]. Nowadays, the most effective prophylactic therapy available for persons with atrial fibrillation who are at higher risk for thromboembolic events is oral anticoagulation. However, this therapy significantly increases the risk of serious bleeding, resulting in a high incidence of discontinuation and decreased quality of life [4]. For stroke prevention in patients with AF, new surgical or percutaneous procedures (WATCHMAN, LARIAT, and Amplatzer devices) have been developed [33]. These devices are primarily utilized in patients with atrial fibrillation who are at high risk of thromboembolic events and are unable to tolerate extended anticoagulant medication. While these therapies have been demonstrated to be feasible, their long-term superiority over medical therapy remains debatable. The imaging method employed in patients with atrial fibrillation is centered on evaluating the underlying cardiac causes of the AF, which include valvular heart disease, ventricular geometry, and function. Loss of LA function is related with an increased risk of thromboembolism, difficulty maintaining sinus rhythm, and a poor prognosis [24]. The size of the LA can be reported as either the antero-posterior LA diameter or the LA area, as defined by the American Society of Echocardiography's chamber quantification guidelines [24]. Previous investigations have demonstrated the superiority of LA volume and LA volume indexed by body size as a more accurate measurement [34]. Recent research indicated that real-time 3D echography (RT3DE) is more accurate than two-dimensional TTE for predicting LA volume [35].

b) *Thrombosis of the left ventricle:* MI and dilated cardiomyopathy (DCM) are frequently associated with formation of left ventricular (LV) thrombus as a result of localized or global myocardial failure. Around 1-2.5% of persons with acute MI have a stroke over the first 4 weeks, with half occurring within the first 5 days. Subjects with 40% ejection ratios, anterior wall MI, or LV aneurysm were more likely to develop LV thrombus [15]. The LV thrombus is not restricted to the LV apex; it can occur in a variety of sites across the left ventricle, such as the infero-posterior and septal walls, albeit in a minority of individuals. DCM is also linked to thrombus development in the left ventricle (**Figure 2**). The annual risk of embolization is estimated to be 1-3%, and is proportional to the degree of systolic dysfunction [19]. While patients with AF who have decreased systolic function should be treated according to current guidelines, the stroke prevention strategy for persons with sinus rhythm has been a point of controversy. Contrast agents for LV echocardiography have been utilized to improve detection accuracy, particularly in individuals with challenging acoustic windows [36]. An LV thrombus is defined as a discrete mass in the LV with well-defined boundaries that are different from the endocardium and that is visible during systole and diastole in an area with associated LV wall motion abnormalities on TTE evaluation.

c) *Thrombosis of the aortic arch:* Aortic atherosclerosis worsens with age and is connected with traditional cardiovascular risk factors such as high blood pressure, hypertriglyceridemia, diabetes, and smoking. Numerous studies have established a causal link between massive aortic atheroma and ischemic stroke. The French Aortic Plaque in Stroke (FAPS) group evaluated the thickness of the plaque in a significant number of patients and found those with a plaque thickness of 4 mm had an elevated risk of recurrent ischemic stroke [37]. On the other hand, it is well documented that the ulcerated or mobile nature of the plaque increases the risk of embolism. In the elderly, we discovered that the intricacy of aortic atheroma, rather than its size, was strongly associated with ischemic stroke [38]. Males had significantly

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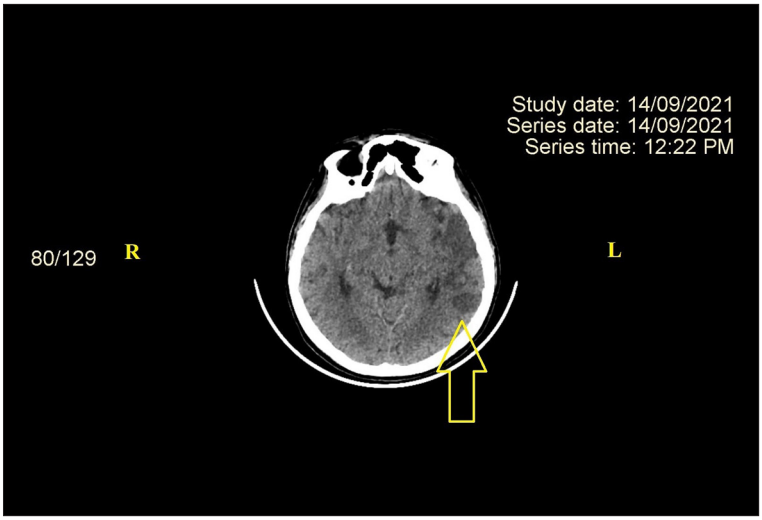

Figure 3. CT scan. Ischemic stroke in left superficial MCA territory (indicated by yellow arrow). The image was acquired using GE REVOLUTION EVO 128 SLICE computed tomograph produced by General Electric Healthcare Japan Corporation – Japan. CT – computed tomography; MCA – middle cerebral artery, R – right; L – left.

more aortic plaques larger than 4 mm in diameter than females (31.5% vs 20.3%) and were associated with ischemic stroke, including both men and women. On the other hand, plaques 3-3.9 mm in thickness were related with stroke in women but not in men [39]. The ideal treatment strategy for people with aortic plaque is unknown. According to the American Heart Association, clopidogrel and warfarin are both effective therapies for individuals with ischemic stroke and plaque in the thoracic aorta more than 4 mm [40]. A study has found no significant difference between the 2 treatments for the composite endpoint of cerebral infarction [41]. Antiplatelet treatment and statin therapy are indicated in the current guideline for individuals who have had an ischemic stroke or transient ischemic attack and have evidence of aortic arch atheroma [42]. The aortic root and proximal ascending aorta are typically seen during TTE. The aortic arch can sometimes be visible via the suprasternal notch, and the descending aorta can be viewed through that window and on apical views [43]. TEE is more accurate than TTE for determining the mobility and thickness of plaques, and can also be used to analyze their calcium content. TEE allows estimating the overall plaque burden by measuring plaque load at various elevations in the aorta. This helps assess risk and track treatment response. TEE technology now allows for three-dimensional reconstruction of the aorta (RT3DE), which can help locate and quantify the atherosclerotic plaque load [44].

LA enlargement is a strong predictor of newly diagnosed atrial fibrillation and embolic events in individuals with atrial fibrillation. Echocardiographic abnormalities, such as LA indexed volume, are related to the diagnosis of AF in patients with cardiac embolic stroke of unknown cause [22,45,46].

The pulsed-wave Doppler transmitral inflow (TMF) pattern is another previously described predictor of AF. TMF is composed of 2 components: early diastole filling induced by the left atrioventricular pressure gradient (E wave) and late diastole filling induced by atrial contraction (A wave). Around the age of 60 years, a decline in E velocity occurs, followed by an increase in compensatory age-related A velocity due to left ventricular diastolic failure, and reversal of the E/A ratio. This change happened regardless of age in response to an increase in blood pressure, even during the acute phase of stroke. Individuals with AF did not exhibit an increase in the velocity of their A-waves [47,48]. Meanwhile, LA stunning is another factor that contributes to patients with atrial fibrillation having a low A wave velocity [49]. Anticoagulation for subclinical device-detected AF is debatable, as short-term AF has not been demonstrated to warrant it [4].

Neurological Imaging Methods

While the imaging modality of choice will depend on the patient's presentation time (ie, within or outside the window period), the following criteria govern the evaluation process [50]:

Evaluation of the Cerebral Parenchyma

Computed tomography (CT) scans: This is a good imaging modality for identifying and excluding bleeding. Rapid imaging and simplicity of reporting, especially in environments with minimal information, continue to be its main benefits. Nonetheless, it demonstrates low sensitivity for detecting infarcts early in their evolution. Subtle symptoms such as sulcal effacement or "the hyperdense middle cerebral artery (MCA) sign" can be present in large infarcts, although they are inconsistent and often ignored (**Figure 3**).

Magnetic resonance imaging (MRI) scans: This is by far the best imaging modality available for assessing the brain parenchymal state. Numerous imaging sequences aid in not only

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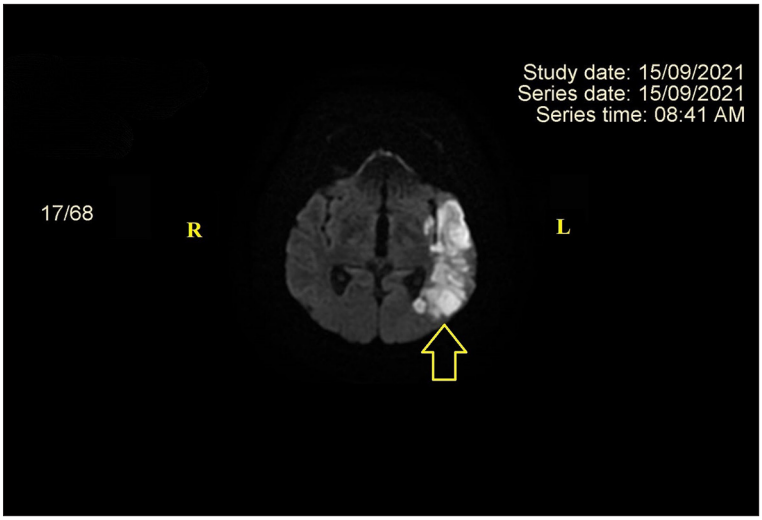

Figure 4. MRI in DWI. Recent ischemic stroke in superficial left MCA (indicated by yellow arrow). The image was acquired using GE SIGNA EXPLORER 1.5T magnetic resonance imaging device produced by General Electric Healthcare Japan Corporation – Japan. MRI – magnetic resonance imaging; DWI – diffusion-weighted imaging; R – right; L – left.

delineating the infarcted region but also in providing a chronological context in cases when the history is not well known. By selecting the right b-value, diffusion-weighted imaging (DWI) (**Figure 4**), MRI can assist in identifying even hyper-acute infarcts. Cortical stroke in various vascular areas raises the possibility of a cardioembolic origin. The presence of an infarct in the fluid-attenuated inversion recovery (FLAIR) sequence implies that the infarct formed partially over 6 h ago. Using susceptibility-weighted imaging (SWI), MRI scans can also correctly detect hemorrhagic infarcts.

Evaluation of Vascularization

Magnetic resonance angiography: This is an advantageous imaging modality, especially in patients with renal impairment, because it does not involve administration of intravenous contrast medium. A disadvantage is that it is susceptible to a variety of errors, including overestimation of stenotic lesions.

CT angiography: This is the criterion standard for examining the cerebral vasculature, but has the disadvantage of not being suitable in individuals with renal impairment.

Complications of Cardioembolic Strokes

Cardioembolic strokes, when not treated adequately, have a greater proclivity for early and late recurrences than athero-thrombotic strokes. Hemorrhagic events, both spontaneous and as a result of anticoagulant treatment, are potentially fatal complications of this illness. Long-term impairment and consequences such as internal cranial hypertension can emerge, although their degree and extent of deterioration corresponds to the severity and depth of the neuro-deficiency [51].

Conclusions

Due to the high likelihood of recurrence and death associated with cardioembolic stroke, cardioembolic embolism should be suspected and evaluated in all patients presenting with stroke. Owing mostly to the increasing prevalence of AF with age, the incidence of cardioembolic stroke is predicted to rise in the future. Both 2-D TTE and 2-D TEE are critical in detecting cardioembolic causes of stroke. These imaging modalities give critical and additional data that can be utilized for secondary prevention and determining the therapeutic plan for stroke patients. However, the novel technological innovation, known as the RT3DE, provides better insight into the pathogenesis of cardioembolic stroke.

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