Cardiac Assessment in Acute Stroke

Understanding how cardiac conditions and stroke are related is essential to the evaluation of patients with acute stroke. The authors present a plan to help you make this critical connection.

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A variety of cardiac conditions may predispose individuals to stroke, and conversely, acute stroke may affect cardiac function. Understanding these relationships is important when evaluating patients who present with an acute ischemic stroke. For example, identifying potential sources of cardiogenic embolism not only helps guide treatments aimed at reducing the risk of a recurrent stroke but also may lead to the diagnosis of previously undetected cardiac disease. Knowing that stroke itself may affect the heart can lead to interventions that limit cardiac complications.

This article focuses on the cardiac evaluation of patients with acute stroke. Although much of this evaluation occurs during the patient's hospitalization, many studies can be performed in the emergency department. Patients with minor symptoms or transient ischemic attacks (TIAs) are sometimes evaluated and managed in emergency department observation units for up to a day before being discharged with referral to primary or specialist care.

KEYS TO HISTORY-TAKING AND PHYSICAL ASSESSMENT

The urgent nature of acute stroke management, especially in patients who are candidates for thrombolytic therapy, leaves only a small window for obtaining the medical history. This challenge is in-

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tensified when linguistic or cognitive impairment is part of the presentation.

In the emergency department, where history-taking is aimed primarily at establishing a diagnosis and eliciting enough information to guide therapy toward the goal of stabilization, the cardiac history is extremely important. Chest pain and diaphoresis suggest acute cardiac ischemia. Palpitations, light-headedness, and syncopal episodes suggest cardiac dysrhythmia. Unexplained fever in a patient with an acute stroke raises concern for endocarditis with septic embolization. A history of cardiac arrhythmias (particularly atrial fibrillation), valvular heart disease, connective tissue disorders, and coagulopathy also increases the likelihood of embolic disease.

Left ventricular mural thrombus is one of the major complications of acute myocardial infarction (AMI), especially in patients with anterior Q-wave AMI. Mural thrombus most often occurs within the first two weeks after AMI.[1] Since the advent of acute reperfusion therapy, the overall incidence of mural thrombus has decreased from 20% to 5%, but it may still be as high as 10% in patients with anterior AMI.[2]

Further history from a focused review of systems may reveal episodes, such as exertional dyspnea, lower extremity edema, and syncope, that are possible indicators of previously undetected cardiac disease, especially if the patient also has vascular risk factors such as hypertension, diabetes, and smoking. For young (especially female) stroke patients, a family history of cardiac disease and coagulopathies can be significant. And, as with all patients, obtaining a list of medications is paramount, noting any history of treatment with vasoactive (antihypertensive, beta-blocking, antianginal, anticoagulant, antiplatelet or antiarrhythmic) medications.

In addition to a neurologic examination, it is also important to perform a general physical examination and a cardiovascular assessment. Cardiac auscultation is critical, as it provides valuable information immediately. An irregular heart rhythm can indicate atrial fibrillation or flutter. Auscultation of heart sounds may reveal murmurs suggesting valvular heart disease, gallops consistent with congestive heart failure (CHF), or rubs that could indicate pericarditis. If endocarditis is suspected, the patient should also be checked for other signs of embolic disease, such as Janeway lesions, Osler nodes, and subungual splinter hemorrhages. Pulmonary auscultation may provide evidence of CHF, and peripheral pulse, skin temperature, and skin color may indicate peripheral vascular disease. Unequal radial pulses and upper extremity blood pressures could signal aortic dissection or subclavian disease.

INTERPRETING THE ECG

Standard 12-lead electrocardiography should be performed routinely in acute stroke. This simple, rapid diagnostic tool may reveal acute myocardial ischemia or infarction, evidence of previous MI, important arrhythmias (especially atrial fibrillation), or left ventricular hypertrophy. If possible, the ECG should be compared with previous ECGs to detect interval changes.

Besides those associated with primary cardiac conditions, other ECG abnormalities commonly occur in stroke patients related to the acute neurologic insult itself. These abnormalities may be quite profound at times, mimicking ECG changes seen during cardiac ischemia. They are found in 60% to 70% of patients with intracerebral hemorrhage, 40% to 70% of patients with subarachnoid hemorrhage, and 15% to 40% of patients with ischemic stroke.[3]
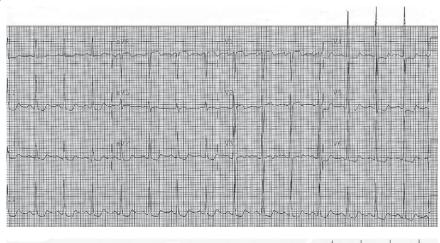
The most common stroke-related ECG abnormality is QT prolongation (Figure 1). Large inverted T waves and large U waves can also be seen, with the most pronounced changes occurring in the anterior precordial leads. New Q waves are identified in about 10% of patients with acute ischemic or hemorrhagic stroke.[4] While isolated ECG abnormalities may not require specific treatment, serial ECG monitoring and assays for cardiac biomarkers, as well as measurement of serum electrolytes (magnesium and potassium), are often necessary.

Cardiac arrhythmias occur in 20% to 40% of patients with ischemic stroke or intracerebral hemorrhage and in nearly 100% of patients with subarachnoid hemorrhage.[3] Atrial fibrillation is the most common, especially in ischemic stroke with underlying cardiac disease, but bradycardia, supraventricular tachycardia, atrial flutter, ectopic ventricular beats, multifocal ventricular tachycardia, torsades de pointes, or ventricular fibrillation also may
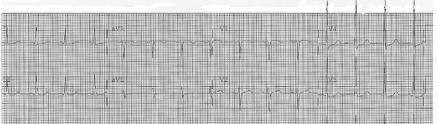
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FIGURE 1. Transient stroke-related abnormalities: The top tracing was obtained on presentation from a 65-year-old patient with acute subarachnoid hemorrhage and no history of coronary artery disease. It shows QT interval prolongation (QT$_c$ 492 ms) and diffuse ST-segment depression, most prominent in the inferior and lateral leads. Some T-wave inversion (V1-V3) is also shown. All of these abnormalities are absent from the bottom tracing, obtained 24 hours later. Cardiac biomarkers were normal in the interval between ECGs.

be seen. The arrhythmias occur most often within the first week after stroke.

Cardiac arrhythmias can lead to stroke, but stroke can also cause cardiac arrhythmias. Brain stem hemorrhages are associated with atrial fibrillation, whereas sinus bradycardia and supraventricular tachycardias are more commonly associated with frontal hemorrhage.[3] The insula is strongly associated with cardiac function. Strokes affecting the right insula are more likely to cause bradycardia and hypotension. Strokes with left insular involvement are associated with tachycardia and hypertension. Some arrhythmias can be life-threatening, so continuous cardiac telemetry and periodic ECG monitoring are recommended

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during the initial management of patients with these problems.

PROBING FURTHER

A routine chest radiograph is not necessary as part of the emergent evaluation of most patients with acute stroke. However, it can be useful in patients with a clinical suspicion of aortic dissection or acute cardiac, airway, or pulmonary disease (including suspected aspiration). Other considerations for diagnostic testing related to the possibility of underlying heart disease are discussed below.

Cardiac biomarkers. The use of cardiac biomarkers to detect myocardial necrosis has been well established in the emergency department. Cardiac troponins I and T are the most widely used. Creatine kinase MB (CK-MB) is an acceptable alternative, but it is less specific than troponins. While potentially useful, total CK, lactate dehydrogenase, and aspartate aminotransferase levels are not primarily used for the diagnosis of MI.[5] Cardiac biomarker testing is warranted in patients with any cardiac symptoms, and the physician should keep in mind that stroke itself may impair a patient's ability to relate those symptoms. Testing should also be considered in patients with known coronary artery disease (CAD), subarachnoid hemorrhage, infarctions involving the insula, and possible ECG abnormalities.

Cardiac troponin levels increase 3 to 4 hours after the onset of myocardial injury. Troponin I can remain elevated for 4 to 7 days, and troponin T can remain high for 10 to 14 days. The level of CK-MB also rises after myocardial injury, with a time course similar to that of troponin, and then falls to normal ranges within 48 to 72 hours. Levels of CK-MB may also be helpful as a marker of reinfarction. Biomarkers of myocardial necrosis may not be detectable within 6 hours of symptom onset, so in patients who tested negative at presentation, testing should be repeated 6 to 9 hours later and again in 4

to 6 hours if they have symptoms or ECG abnormalities that raise concern for myocardial ischemia.[5]

Elevated troponin I predicts a poor outcome independent of patient age and stroke severity.[6] An increase in catecholamine levels caused by acute stroke, particularly intracranial hemorrhage, can lead to cardiac necrosis, which may result in elevation of troponin.[7] In the setting of acute stroke, the diagnosis of AMI requires a typical rise and/or fall in cardiac biomarkers along with an abnormal ECG.[5] When cardiac biomarkers are increased without clinical evidence of MI, a thorough search for other causes of cardiac damage should be undertaken.

Cardiac monitoring. One in six ischemic strokes is due to cardiogenic embolism, which is associated with nonvalvular atrial fibrillation or flutter (AF) in almost half of cases. Atrial fibrillation or flutter increases stroke risk by five times, while ischemic heart disease increases stroke risk by two-and-a-half times.[10] After AF has been identified, oral anticoagulant therapy such as warfarin provides a 48% reduction in the risk of recurrent stroke, compared with antiplatelet therapy.[8]

An initial ECG can identify chronic atrial fibrillation, but it does not exclude paroxysmal AF, which carries a risk of ischemic stroke comparable to chronic AF.[9] To detect paroxysmal AF, it may be necessary to track the patient's cardiac rhythm over a prolonged period using a Holter monitor or one of the newer noninvasive systems. For example, patient-activated transtelephonic event recorders capture just a few minutes of ECG when the patient puts on a
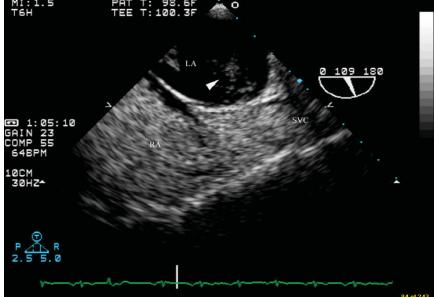

FIGURE 2. **Patent foramen ovale:** The top image is a bicaval transesophageal echocardiographic view showing the left atrium at the top of the image, the right atrium at the bottom of the sector image, and the superior vena cava to the right. Doppler color flow imaging demonstrates flow through the patent foramen ovale (arrow). Note that the blue color, which indicates a small amount of blood flow from the left atrium to the right atrium, is tangential to the plane defined by the atrial septum. The bottom image is the same view with microcavitation study. Note the echodensity in the right atrium and superior vena cava due to the small air bubbles that form as agitated saline is injected intravenously. These microbubbles can be seen traversing the atrial septum across the patent foramen ovale and entering the left atrium (arrow head).

bracelet or presses the device against the chest at the onset of symptoms. Some systems use a recorder that is implanted under the skin, which is useful for diagnosing infrequent rhythm disturbances associated with incapacitating symptoms, such as syncope.

Echocardiography. Echocardiography is useful for detecting abnormalities of cardiac morphology or function that can lead to embolic stroke, including atrial myxoma, mitral stenosis and regurgitation, valvular vegetations, left ventricular or left atrial thrombus, cardiomyopathy, atrial septal aneurysm, and patent foramen ovale or atrial septal defect (Figure 2).

Although echocardiography can usually reveal cardiac abnormalities associated with stroke, its emergent use must be tempered by other available clinical information. For example, routine echocardiography is usually not necessary in patients with previously known cardiac disease, such as atrial fibrillation, who already have an indication for anticoagulant treatment. In addition, it is usually unnecessary when the information obtained would not alter therapy, such as in patients at risk of falling or with other absolute contraindications to anticoagulant therapy.

Patients with acute stroke and a history of heart disease are at higher risk of developing cardiac emboli and are more likely to have an abnormal echocardiogram. A patent foramen ovale or an atrial septal defect is present in 40% of young stroke patients—a much higher rate than their prevalence of 10% to 15% in the general population.[11] Thus, echocardiography may be especially useful in younger patients with an otherwise unexplained ischemic stroke or TIA. Currently, the American College of Cardiology and the American Heart Association recommend echocardiography for patients with embolic stroke and clinical evidence of cardiac disease, as well as in patients with stroke who are less than 45 years old. Routine echocardiography is not recommended in patients older than age 45 who do not have clinical cardiac disease if the cause of the stroke is apparent.[11]

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A patent foramen ovale or an atrial septal defect is present in 40% of young stroke patients.

As confirmed by surgery or autopsy, transthoracic echocardiography has a sensitivity and specificity of 86% to 95% for the detection of left ventricular thrombi. However, it has a sensitivity of only 39% to 63% for the detection of left atrial thrombi and less than a 50% sensitivity for detecting patent foramen ovale.[12] Properly conducted transthoracic echocardiography with saline contrast (the bubble test, with and without Valsalva maneuver) can increase the sensitivity and specificity of chest wall echocardiography for patent foramen ovale to levels approaching 90%.

Transthoracic echocardiography images may be affected by chest wall configuration, lung disease, body habitus, or patient age. In such cases, the use of transesophageal echocardiography is helpful if clinical indications are present. While it offers no superiority in the detection of left ventricular thrombus, it is more than 90% sensitive and specific for left atrial thrombus. For patent foramen ovale, it is 89% sensitive and 100% specific.[12]

Unfortunately, transesophageal echocardiography is associated with traumatic, cardiac, pulmonary, and bleeding complications in 0.18% of patients.[12] Currently, the American Heart Association does not recommend this procedure in unselected patients and its cost-effectiveness is also uncertain. It should only be considered when there is a high suspicion of embolism but no source of embolism has been identified on ECGs or with cardiac monitoring, or when the image quality of transthoracic echocardiography is inadequate. As with all diagnostic procedures, transesophageal echocardiography is never indicated if its results would not affect patient management.

Cardiology referral. Twenty-five percent to 60% of patients with carotid artery disease have myocardial ischemia or severe CAD on provocative cardiac testing, despite the absence of cardiac-related symptoms.[13] Patients with stroke or TIA who have high CAD risk factor scores (based on Framingham algorithms) and/or significant carotid disease should be referred to a cardiologist for further evaluation, including provocative cardiac testing.[13]

STAYING FOCUSED

Stroke patients frequently have cardiac disease. Therefore, initial cardiac evaluation of stroke patients in the emergency department should focus on detecting concomitant AMI and arrhythmias, and further cardiac evaluation should focus on detecting potential sources of cardiogenic embolism. Selected stroke patients should be referred to a cardiologist for further evaluation. □

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