Patient Handouts

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Transient Ischemic Attack (TIA)

Introduction

TIA is short for a "transient ischemic attack." Very few people have an adequate understanding of what it means to have a stroke or a transient ischemic attack (often called a "mini stroke"). Indeed, there are some who confuse the term "stroke" with a heart attack. A stroke, however, causes permanent damage to the brain, not the heart. For this reason, there is a push to rename strokes and call them "brain attacks." In this handout, the term brain attack will be used in place of the old concept of "stroke."

Almost everyone understands the concepts of heart attack and angina. We all appreciate that people who suffer from angina usually have hardening of the arteries to the heart, with rust-like deposits of cholesterol, calcium, and clotted blood on the inner surface of the artery, which gradually causes the "pipe" to silt up. Statistically, we know that attacks of angina indicate a greater risk of the arteries suddenly blocking completely, causing a major heart attack.

In much the same way people who suffer minor warning strokes (TIA's) are also at increased risk of having a fully developed and irreversible brain attack (stroke).

Hardening of the arteries, leading to heart and/or brain attacks, is a major cause of disability and death especially in the western, industrialized world, and much research is being done to try and reduce this. In an ideal world it would be best to prevent these attacks before they happened, which is called primary prevention. If, however, someone has already had a TIA, what can be done to prevent any more, or secondary prevention? This patient handout will attempt to answer these questions.

Definition

A transient ischemic attack is simply a brain attack (stroke) that usually lasts less than 30 minutes. By definition, there must be no permanent disability and the vast majority of TIA's resolve with an hour. It also seems reasonable that there also must be no evidence of permanent damage to the brain on investigations such as an Magnetic Resonance Imaging (MRI) scan.

Until recently the definition of a TIA included the concept that the attack may last up to 24 hours, but there is now reasonable agreement that the duration of the attack is, in the vast majority of patients, less than 30 minutes. Discussion is continuing among neurologists and stroke specialists on an acceptable definition of a transient ischemic attack.

Although this seems to be straightforward, in practice the diagnosis is often extremely difficult and there are other neurologic conditions that may mimic TIA, especially in the younger patient, including migraine with aura and a type of partial seizure.

Mechanisms

• *Stenosis of an artery*

Almost invariably, a TIA occurs when a small particle of the "rust" that we described above, breaks off from the inner wall of an artery and is carried along in the blood flow and lodges in a small blood vessel of the brain. This is called an embolism. It then rapidly breaks up into smaller particles and dissolves. Hence the attack is only transient. The most common site of this build up of fat, blood clot, and calcium

is in the carotid arteries in the neck. You can usually feel the normal carotid artery pulsating in time with your heart beat, at the angle of the jaw in the neck on each side of the body.

There are other sites of atheroma build up, that may be the "source" of this debris that breaks off, including the major blood vessel from the heart and the smaller arteries within the skull, but to find these lesions requires often specialized and complex investigations.

• *Atrial fibrillation*
In a small percentage of cases, the particle that is dislodged comes from the heart chambers and in particular, when there is a specific disturbance of the heart beat, is called atrial fibrillation. In this situation, the heart beat is very irregular and often fast, and in small corners of the heart stagnant pools of blood form, leading to blood clots called thrombus. It is these clots that may dislodge and cause a TIA, or even worse, block a major brain artery permanently, causing a full blown brain attack (stroke).

• *Cryptogenic*
In a minority of patients who suffer a TIA, there is no obvious explanation, these are called "cryptogenic," but your neurologist would certainly order tests to look for atrial fibrillation or a narrowing (stenosis) of the carotid artery in the neck. These 2 possible causes must be excluded, because there are successful treatments for both. In the case of a severe narrowing of the carotid artery, the cleaning out of the carotid artery, by an experienced surgeon, will reduce the risk of a brain attack significantly compared to leaving it alone. In the case of atrial fibrillation, thinning the blood to prevent clots from forming is also of proven benefit.

It must be remembered that in both cases, the treatment options are not without risk.

The rest of the handout will concentrate on the majority of patients who have a TIA for which there is no obvious reason found after extensive investigation.

Risk of brain attack after a TIA

It has, until recently, been very difficult to give meaningful and confident advice about the subsequent risk of another TIA or a brain attack to patients after their first episode. A major reason for this was that neurologists often disagreed on the definition of a TIA and on how long they lasted. This is being rectified at this very time by an independent worldwide committee of leaders in research in TIA and brain attacks.

Secondly, it is also sometimes not easy for professional people to be able to explain rather complex statistics in a meaningful and practical way to a patient sitting across the consulting desk. When discussing the following statistics, which discuss percentages (%), it may be easier to understand if one starts with the concept that we are talking about a theoretical group of 100 patients who have ALL had a TIA at about the same time.

With this concept in mind, we are now able to provide some meaningful information about the risk of brain attack (stroke) after a transient ischemic attack (TIA):

• 10% risk within the first week (i.e., 10 patients in every 100 TIA patients). If the TIA is the consequence of a critical stenosis of the carotid artery in the neck, then the risk of a brain attack may be as high as 30% in the next week (3).

• 18% risk within the first 3 months (i.e., 18 out of 100 TIA patients) (1,2).

• Thereafter, the risk of stroke falls and plateaus at about 4% to 5% per year (4,5).

• By 5 years, almost 35 of the 100 would have suffered a brain attack.

An obvious question from these statistics is: why is there so much variation? Much of the spread in range can be explained by other health issues that we now know influence the risk of a TIA or brain attack (or even a heart attack). These include:

• Age
• Male gender
• Hypertension
• Diabetes
• Tobacco use
• Lack of physical activity and obesity
• Cholesterol (lipid) readings
• Genetics

The theory is that these factors all contribute to atherosclerosis, or the premature hardening of the inner wall of the arteries of the body; to the heart, the brain, the legs, etc.

Genetics, gender, and age

A brief word about the influence of your genes seems in order. If there is a strong family history of premature hardening of the arteries which leads to early heart attacks or brain attacks or even sudden death, then it is logical to anticipate that this tendency may be passed on. Indeed, genetics probably plays a far greater overall part in determining risk than the lifestyle risks listed above. And Although we can't change our genes, or our gender or age, we can modify the other risk factors.

Risk factors that we can influence

So in partnership with your neurologist and family physician, a patient who has had a TIA should:

• stop smoking
• exercise moderately every day for at least 30 minutes (enough to increase the heart rate)
• control blood pressure smoothly
• control diabetes meticulously
• ensure that serum lipids are normal
• take medication to reduce the stickiness of the blood and platelets (aspirin therapy)

It is almost impossible to break each of these risks down in terms of how much influence a single risk factor has. To make the statistics meaningful, we need to provide an estimation of the risk of a brain attack in the normal population.

In the elderly (greater than 65) population, in those who do smoke or have hypertension or diabetes, the risk of a stroke is about 6 in every 1000 persons per year (0.6%); and each of the risk factors above probably multiplies the risk by between 2 and 5 times. As an illustration, consider this hypothetical elderly male (age greater than 65 years)

Risk factors	**Risk of TIA / brain attack**
none	6 per 1000 males each year

smokes	12 to 30 per 1000 males each year
hypertension	12 to 30 per 1000 males each year
smokes AND hypertension	24 to 150 per 1000 males each year

(In other words the combined risks are multiplied rather than simply added together!)

Alcohol

There is some rather conflicting information about alcohol. Too much alcohol, especially if taken as a binge, intermittently, damages the body, especially the liver, and may contribute to chronic changes in lipid levels and increase the risk of obesity, diabetes, and high blood pressure. However, large community studies suggest that taken in moderation, alcohol may reduce slightly the rate of atherosclerosis.

Cholesterol and lipid drugs

Despite the intense fixation of the public on "cholesterol levels," of all the risk factors listed, this is not the single most important in terms of reducing a brain attack. Granted, it may be slightly more important in terms of risk reduction for a heart attack. However, there has been some interesting suggestions that the drugs used to lower cholesterol, called "statins" are useful, not because they lower cholesterol, but because they may reduce the inflammation associated with the buildup the atherosclerosis in the blood vessels. Recent evidence has revealed that in a patient who has had a brain attack (stroke), taking a "statin" drug reduces the risk of further stroke or even a heart attack subsequently, by about 25%. Remember that this effect is not the most important consideration when we are talking about risk reduction, but one of several risk reduction steps.

Antiplatelet agents

The original (and some say still the best) drug to reduce the "stickiness" of the platelets that cause thrombosis formation is aspirin. Although there are more modern agents these are NOT spectacularly better than aspirin, are much more expensive than aspirin, and some have more troublesome side effects. The newer drugs include clopidogrel, dipyridamole, and ticlopidine. The present guidelines for drug selection are:

• the first line drug should be aspirin
• other modern drugs are acceptable as first line, especially if there are problems with sensitivity to aspirin
• the combination of aspirin and dipyridamole may be better then each alone
• the dose of aspirin need only be small, 30 to 100 mg a day. In fact, higher doses do not appear to add any benefit but may cause unacceptable side effects.

Mutual obligation

Your neurologist will do his or her best to help reduce the risk of a TIA by treating your blood pressure and diabetes and by prescribing drugs to lower cholesterol together with aspirin. But if you were to continue to smoke and/or remain obese and physically inactive, then the anticipated risk reduction is thwarted.

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