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Brain Attack: New Concepts

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Abstract

Stroke is the third leading cause of death in North America. It has also been the major lethal disease that has been most ignored by modern medicine. The reason for this is not difficult to understand. Stroke has been easy to ignore, because it has been untreatable.

Fortunately for all of us, stroke is no longer an untreatable disease. The landmark NIH-tissue plasminogen activator (tPA) trial, published in December 1995, proved that stroke is treatable. In an attempt to emphasize this fact, stroke is now sometimes referred to as a "brain attack", hopefully analogous in the minds of the public to a heart attack.

Current treatment of stroke is complex. However, because stroke is such an important disorder, all physicians should be generally familiar with the approach to an acute stroke patient during the first few hours after stroke onset. The approach to the hyper-acute stroke patient can be compartmentalized in the following manner:

1. Identification of the candidate for thrombolytic therapy
2. Review of head CT findings
3. Management of hypertension

I. Identification of the candidate for thrombolytic therapy

A. Time of onset

Stroke is a disorder in which symptoms develop with extreme rapidity, with peak symptomatology often occurring nearly instantaneously. Nevertheless, patients frequently seek medical care in a surprisingly leisurely fashion. It is not uncommon for patient to wait one or two days prior to seeking medical attention. There are several reasons for this. The lay public is remarkably ignorant of stroke and stroke symptoms. Moreover, patients are frequently confused during the early period of stroke, and often deny their illness for both neurological and psychological reasons.

Countless experimental stroke studies have established the existence of a "therapeutic window" for treating stroke. The window refers to the interval between the onset of the stroke and the initiation of therapy. The upper limits of the therapeutic window for tPA in acute stroke have not been established, but may be no more than three hours. Likewise, the NIH-tPA trial demonstrated that the therapeutic window extends to three hours. Additional studies have yet to extend this window.

Thus, patients with stroke of three hours or less in duration are, lacking exclusion factors discussed below, potential candidates for tPA. Stroke often occurs in the early morning hours, and it is important to remember that patients with stroke present upon awakening are considered as having their stroke at the time they were last awake and neurologically normal, i.e., when they went to sleep. tPA is given at a dose of 0.9mg/kg (maximum 90mg), with 10% given as a bolus over one minute and the remainder given as a piggy back over one hour.

B. Stroke subtypes

Stroke is a generic term, and the type of stroke that is treatable by tPA is ischemic stroke; this represents approximately 85% of all strokes, the remainder being hemorrhagic. The distinction between ischemic and hemorrhagic stroke is made by CT scan (see below). Most of the time, it is not difficult to make this distinction, but there are instances when calcifications can be mistaken for blood, and vice versa. Therefore, it is a good idea for physicians to become familiar with the landmarks of a normal head CT scan.

Treatment of acute stroke with tPA increases the risk for intracranial hemorrhage. Symptomatic intracranial hemorrhage occurs in approximately 6% of acute ischemic stroke patients treated with tPA, compared with 1 % without tPA treatment. Despite this difference, outcome is improved wifh tPA treatment: approximately one patient in eight will have a neurological "cure" when properly treated.

II. Review of head CT findings

As noted above, the primary issue is the determination of intracranial hemorrhage, an absolute contraindication for treatment with tPA. Hemorrhage is hyperdense on head CT scans, and is almost always asymmetrically placed. Calcifications tend to be punctuate and symmetrical. There is usually little difficulty in making the distinction between hemorrhage and calcification.

The second major issue is the delineation of ischemic changes on CT. The two most important findings are hypodensities indicative of infarction, and mass effect indicative of early edema. Chronic infarction on CT appears isodense with cerebrospinal fluid, while acute infarction has an appearance between the density of brain parenchyma and cerebrospinal fluid. CT evidence of acute infarction, when present, is not necessarily a contraindication for tPA. However, CT evidence of large infarcts, i.e., more than 113 the territory of the middle cerebral artery, is a relative contraindication for tPA, due to the risk of hemorrhage. Moreover, the presence of edema, i.e., with mass effect or shift seen on CT, is also a contraindication for tPA treatment; once again, the risk of hemorrhage is increased in this setting.

III. Management of hypertension

Improper management of hypertension in acute stroke is an all-too-common occurrence. Under normal circumstance, autoregulation maintains cerebral blood flow at stable levels, relatively independent of changes in blood pressure. However, following a stroke, autoregulation is lost; this results in a more linear relationship between blood pressure and cerebral blood flow. Thus, lowering blood pressure in the acute stroke patient will produce reduction in cerebral blood flow, sometimes with disastrous clinical consequences. A mild neurological deficit can quickly be transformed into a major deficit by improper lowering of blood pressure.

As a general rule, systolic blood pressure of less than 185 and/or diastolic pressure of less than 110 does not require treatment during the acute period. If treatment with tPA is contemplated, blood pressure above this range is best treated with labetalol at a dose of 10 mg IV over 1-2 minutes. This can be repeated or doubled until a total dose of 300 mg has been so administered. Treatment with one or two doses is usually effective, and can be safely followed with treatment with tPA.