Lesions masquerading acute stroke

Acute stroke is in most cases a straightforward diagnosis with acute, sudden onset of lost or reduced neurological function. In most situations this also represents an acute embolic ischemic event, which is one of the leading causes of death and disability in our society. Ischemic stroke, which accounts for about 80% of all clinical strokes, is nowadays potentially treatable with thrombolytic drugs and/or by mechanical removal if detected early on. It is thus more important than in the past to establish a correct diagnosis as early as possible to get the treatment started and reduce the amount of lost brain tissue “save neuron.”

There are however other causes for acute stroke that can result in a similar deficit and should be treated differently. One group, which for apparent reasons has to be excluded, is intracranial hemorrhages. This group represents the second most common cause for acute stroke and includes non-traumatic intracranial hemorrhage, subarachnoid hemorrhage and hemorrhage due to venous thrombosis. These lesions, should in most cases, be excluded on a non-enhanced brain CT and the continued diagnostic work-up will then be steered accordingly to find, and if possible treat the cause of the hemorrhage.

Although less common, there are several other important causes for acute clinical stroke that can mimic an ischemic stroke. Some of these, such as hypoglycemia and hyperglycemia can by themselves cause a stroke like clinical picture and they may also provoke or exaggerate a true ischemic stroke. However, among all misdiagnosis for stroke, seizure is probably the most common with symptoms ranging from mental status changes to sensory deficits and hemiparesis (Todd’s paralysis). Moreover, some of these patients have a history of past ischemic stroke, which further complicates the diagnostic picture.

Due to the urgency in starting stroke treatment and limit the extent of infarction, a non-enhanced CT is often the only imaging technique available for radiological diagnosis. This examination will allow for the detection of most hemorrhages, but there are some other, true mimickers which are difficult to detect, both clinically and on plain CT alone. The increased use of perfusion-CT and CT-angiography is helpful, in particular when
thrombectomy is contemplated, but their increased use (some claim over-use) has raised concerns regarding radiation dose to the patient. In many instances, MRI with a diffusion-weighted sequence would be the best and most helpful technique. Some institutions also use MRI as the primary diagnostic tool in acute stroke; however, for most hospitals this approach is not practical and would delay the treatment beyond what can be regarded as acceptable. MRI is most often requested after treatment has been started and clinical symptoms prevail or progress beyond what should be expected.

The presentation today, will concentrate on lesions masquerading acute stroke; how one might be able to detect and differentiate these from lesions caused by embolic ischemic stroke:

a) The first group will include patients with acute onset of neurologic deficit and suspected embolic stroke where a non-ischemic lesion sometimes can be detected/suspected on the initial CT before treatment is started, such as tumors, ADEM, multiple sclerosis etc.

b) The second group is represented by ischemic lesions of other origin than embolic stroke. These ischemic lesions are often the result of vascular involvement and arteritis thus needing a different treatment approach, such as some fungal infections, lesions secondary to intravenous drug abuse etc.

c) A third group represents lesions that have an appearance on imaging which can be misdiagnosed as an embolic stroke, such as changes caused by seizure, some malignant lesions etc.

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