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REVIEW

Therapeutic efficacy of brain imaging in acute ischemic stroke patients

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Summary This article reviews main pathological findings in ischemic stroke patients as imaged with CT, CTA, MRI, and MRA and discusses its clinical effectiveness on different levels: technical, diagnostic accuracy, impact on diagnosis and treatment decisions affecting patient clinical outcome. It emphasizes the importance of detecting ischemic brain tissue damage (infarction) early during a time period when reperfusion therapy may be beneficial and provides evidence that brain tissue hypoattenuation as displayed by non-enhanced CT represents net water uptake (ionic edema) that is highly accurate in defining brain tissue that will not recover with reperfusion whereas MRI is highly sensitive in detecting patterns of ischemic brain tissue even in stages that allow functional recovery.

Introduction

In theory, the clinical efficacy of brain imaging with computed tomography (CT) or magnetic resonance imaging (MRI) in patients with ischemic stroke, can be described at 6 different levels: (1) technical, (2) diagnostic accuracy, (3) diagnostic thinking, (4) therapeutic, (5) patient outcome, and (6) societal [1] (Table 1). In other words, image information may change treatment decisions if findings affect diagnostic and subsequently therapeutic concepts and if findings are reliably displayed with high accuracy. In stroke patients, brain and brain vessel imaging is required to differentiate between ischemic and hemorrhagic stroke, between arterial and venous disease, between macro- and microvessel disease and to assess the specific cause of arterial obstruction. Moreover, brain tissue and perfusion imaging

Abbreviations: ADC, apparent diffusion coefficient; ASPECTS, Alberta stroke program early CT score; CBF, cerebral blood flow; CBV, cerebral blood volume; CI, confidence interval; CT, computed tomography; CTA, CT angiography; CTP, CT perfusion imaging; DSA, digital subtraction angiography; DWI, diffusion-weighted MRI; ECASS, European Cooperative Acute Stroke Study; FLAIR, Fluid Attenuated Inversion Recovery; HU, Hounsfield Units; MCA, middle cerebral artery; MRA, MR angiography; MRI, magnetic resonance imaging; MRP, MR perfusion imaging; PROACT, intra-arterial prourokinase for acute ischemic stroke trial; SWI, susceptibility weighted imaging; tPA, tissue plasminogen activator.

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Table 1 The efficacy of diagnostic imaging according to Fryback and Thornbury [1].

Level	Assessment by
Technical efficacy	Intra-/inter-observer agreement
Diagnostic accuracy efficacy	Comparison with reference standard
Diagnostic thinking efficacy	Change in diagnostic concept
Therapeutic efficacy	Change in treatment
Patient outcome efficacy	Clinical outcome assessment
Societal efficacy	Measurement of health care costs

may help to differentiate between ischemic irreversibly injured brain tissue (brain infarction) and ischemic, but still viable brain tissue (penumbra) that will regain function with restoration of blood flow.

Diagnostic imaging has an impact on therapeutic decisions and clinical outcome only if an effective treatment is available [2]. The only proven effective treatment for ischemic stroke patients is intravenous or intra-arterial infusion of thrombolytics within 6 hours of stroke onset (''reperfusion therapy'') [3—6]. This article will review the impact of brain imaging findings on the effect of reperfusion therapy of ischemic stroke. It will show how brain infarction can be diagnosed and safely differentiated from viable ischemic brain tissue and will discuss whether cerebral angiography has a therapeutic impact. It will not discuss brain perfusion imaging, which has not yet shown patient outcome efficacy and lacks reliable standardization [7].

Cerebral vessel imaging

Cross sectional parenchymal imaging techniques like CT and MRI display cerebral arteries and veins as well. On non-enhanced CT, arteries and veins are hardly discernible from brain tissue, but become visible on thin slices (< 2.5 mm) if filled with thrombus (Fig. 1). Thrombo-embolic occlusions of major brain arteries appear as ''hyperdense artery sign'' in about 50% of patients examined with 4 to 8 mm thick slices [8]. It was recently shown that the reduction of CT slice thickness below the artery diameter of 2 to 3 mm dramatically increases the sensitivity of cerebral thrombus detection in major arteries to almost 100% [9]. It was further observed that intravenous tissue plasminogen (tPA) activator rarely recanalizes arteries occluded by thrombi exceeding 8 mm in length [10]. If this will be confirmed in prospective trials, thrombus detection and measurement will have therapeutic efficacy in ischemic stroke patients and may likely contribute to better clinical outcomes.

On MRI, arteries are identified by low signal (''flow void'') due to blood flow velocity and its typical tubular shape. Intra-arterial signal increases with slow or no flow. Disappearance of flow void signal loss thus indicates arterial obstruction. Moreover, gradient echo sequences or susceptibility weighted imaging (SWI) can directly display blood clots as tubular shaped structures with low

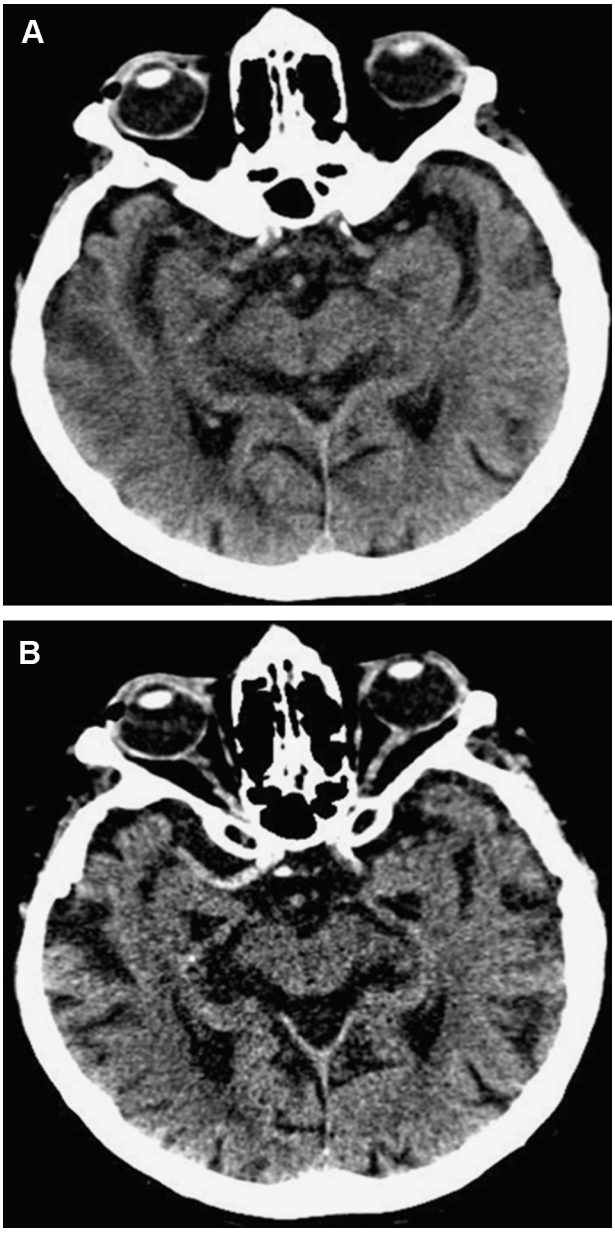

Figure 1 A. Unenhanced CT with 6 mm slice thickness of 78 years old woman with left-sided hemiparesis since 2 hours. B. A 1.5 mm slice of the CT in Fig. 1A showing thrombo-embolic occlusion of right middle cerebral artery trunk.

signal [11]. After contrast infusion, vascular enhancement is increased in territories with slow flow, e.g. beyond tight arterial stenosis (Fig. 2) [12]. Fresh thrombi within venous sinus appear as empty spaces with low signal whereas organized thrombi show contrast enhancement and cannot safely be differentiated from flowing blood. T2- and T1-Fluid Attenuated Inversion Recovery sequences before and after contrast infusion at 3 Tesla may be able to differentiate among intracranial atherosclerotic plaques (eccentric enhancement), inflammatory wall changes (concentric enhancement), and other wall pathologies [13]. MRI can detect subacute arterial wall hematoma due to dissection. Wall hematomas containing methemoglobin display a high signal on T2- and T1-weighted sequences. T1-weighted