Role of Imaging in the Diagnosis and Management of Acute Cerebral Infarction

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Goals

• Understand how imaging studies help in stroke diagnosis and management through examining findings in patients at different stages of acute ischemic stroke

• Know the advantages of different tests in stroke imaging
Background: Stroke

• Stroke is a lay term meaning a condition due to vascular lesions of the brain caused by hemorrhage, embolism, thrombosis, or rupturing aneurysm

• Primarily diagnosed clinically and confirmed and followed through imaging tests
Background: Stroke Types

Most common stroke etiologies:

1) Cerebral Infarction 80%
2) Primary Intracranial Hemorrhage 15%
3) Nontraumatic subarachnoid hemorrhage 5%

* FOCUS: Acute Cerebral Infarction
Menu of Radiological Tests

- Cerebral Angiogram
- CT: w/ or w/o contrast
  - CT angiogram (CTA)
- MR: w/ or w/o contrast
  - T1 or T2 weighted (T1WI, T2WI)
  - FLAIR
  - Diffusion weighted image (DWI)
  - Susceptibility
  - MR angiogram
Cerebral Angiogram

- Gold standard in the past
- Outdated and replaced by MRI/MRA
- High risk of producing further thrombus formation in brain and causing renal failure

Courtesy of Dr. Steve Reddy, BIDMC
Patient 1: CT w/o Contrast
Test of Choice in Emergencies

1) Distinguish between ischemic and hemorrhagic stroke
2) Normal CT in patient with <3 hrs of symptoms → can begin rtPA therapy if no other contraindications

*If abnormal findings are seen on CT, it’s too late to start rtPA

Normal findings
Left MCA Hemorrhage

Hyperdense area of hemorrhage seen clearly on CT

Courtesy of Dr. Nicole Nelson, BIDMC
CT Findings in Cerebral Infarction

Hyperacute: <12 hrs
- Normal 50-60%
- Hyperdense artery (dense MCA sign)
- Obscuration of the lenticular nucleus
- Loss of gray-white interfaces (insular ribbon sign)

Acute: 12-24 hrs
- Low density basal ganglia
- Sulcal effacement

1 to 3 Days:
- Increasing mass effect
- Wedge-shaped low density area involving gray and white matter
- Possible hemorrhagic transformation

Adapted from Osborn, 1994
MR Findings in Cerebral Infarction

Immediate

- Hyperintense on DWI (low apparent diffusion coefficient, ADC)
- IV contrast enhancement
- Perfusion alterations

<12 hrs
Sulcal effacement, gyral edema, loss of gray-white interfaces on T1

12 to 24 hrs

- Hyperintensity on T2
- Meningeal enhancement adjacent to infarct
- Mass effect

1 to 3 days

- IV and meningeal enhancement begin decline
- Signal abnormalities striking on T1WI, T2WI
- Possible hemorrhagic transformation

Adapted from Osborn, 1994
Treatment and Further Imaging

• After the initial CT scan, patient is treated with
  1) tPA
  2) anti-coagulants
  3) antiplatelet aggregating agents

• Further studies help in evaluating response to treatment and extent of brain damage not detected on the initial CT

• The following images presented will be of CT, CTA, MR, and MRA studies, which are most commonly used at large hospitals
The Ideal Early Patient

• Comes into the ER presenting with a recent onset of stroke symptoms
• CT scan shows no hemorrhage and no changes seen in ischemia
• Patient has no other contraindications for rtPA therapy \(\rightarrow\) rtPA is administered
• Follow progress with sensitive MR studies
Patient 2: DWI Hyperacute Infarction Stage

**HISTORY**
- 38 y.o. woman inpatient develops right hemispheric stroke symptoms while talking with physician
- MRI studies <1 hour after onset

**DWI**
- less anatomical detail
- detects cytotoxic edema minutes after ischemic event

**FINDINGS**
- an area of restricted diffusion within the right anterior MCA compatible with acute ischemia

Courtesy of Dr. Steve Reddy, BIDMC
Patient 3: Contrast-enhanced T1W

- 4 hrs after left MCA symptoms began
- Extensive Intravascular enhancement seen (an immediate finding)

Osborn, *Diagnostic Neuroradiology*, 1994
Our Patient: Ms. JB

79 y.o. brought by her son who found her sitting on a chair in her bedroom, unresponsive.

- **CC:** unable to move left arm/leg
- **no known cardiovascular disease**
- **PMH:** TB exposure s/p Rx, sinusitis, arthritis
- **DX:** Acute Stroke
JB: Early Stroke Findings on CT

Hypodensity and loss of gray and white matter differentiation

• right insula ➔ “Insular Ribbon Sign”

• Lenticular nucleus

• part of internal capsule

Normal Hyperdense Insular Cortex

-Internal Capsule

-Damaged region

Courtesy of Dr. Barbara Appignani, BIDMC
JB: Early Stroke Findings on CT

Hyperdensity in the Proximal Right MCA

NEXT:
- Patient started on aspirin, BP controlled
- MR studies 6 hrs later
JB: MRI-T2 Weighted Image

-T2W: good anatomical detail
-T2 signal hyperintensity in R MCA territory: R insula, basal ganglia and internal capsule
-no shift in midline structures

6 hrs after initial CT

Courtesy of Dr. Barbara Appignani, BIDMC
JB: MR-DWI

- Findings similar to those in T2WI→ hyperintensity in R MCA territory: R insula, basal ganglia and internal capsule
- Note less anatomical detail compared to T2WI, but more striking signal

6 hrs after initial CT

Courtesy of Dr. Barbara Appignani, BIDMC
JB: MRA 3-D Reconstruction

-KEY FINDING:
Absence of flow in R MCA branches

-Study pinpoints artery location with problem in flow through in R MCA territory

Circle of Willis
Internal Carotid Artery
Anterior Cerebral Artery
Middle Cerebral Artery (MCA)

Absence of flow
Patient 3: Ms. FL

- 86 y.o. with a history of atrial fibrillation who was at home walking towards her husband when she collapsed and became somulent
- Unable to speak and had difficulty moving her right side ➔ clinical dx of acute stroke
- CT scan w/o contrast 2 hrs after onset was normal ➔ Timing appropriate for rtPA
- rtPA contraindicated since INR>1.5
Ms. FL:
MR T2-Weighted 2 days later

FINDINGS:

• Striking enhancement of brain parenchyma

• Mass effect associated with infarct, mild midline shift

• Both commonly found 1-3 days post-stroke

Courtesy of Dr. Nicole Nelson, BIDMC
FL: MR FLAIR

FLAIR technique is similar to T2WI except that CSF signal is subtracted to more accurately represent hyperintense signal from fluid produced by damaged cells.

45 hrs after symptom onset

Courtesy of Dr. Nicole Nelson, BIDMC
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Often 4-6 hrs

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Adapted from Osborn, *Diagnostic Neuroradiology*, 1994
Summary: Goals in Imaging Patients Presenting with Clinical Signs of Acute Stroke

- Confirm clinical diagnosis → CT
- Distinguish between hemorrhagic and ischemic stroke, since treatment differs greatly → CT
- Assess the severity of brain damage and follow progression of damage → MR studies, plus CT to monitor new hemorrhage
Summary: Goals in Imaging Patients Presenting with Clinical Signs of Acute Stroke

1) CT
   - Hemorrhage
     - Signs of Ischemia? Severity?
   - No Hemorrhage
     - R/O Non-stroke causes of symptoms

2) MR
   - Assess severity, follow progress
     - T1WI
     - T2WI
     - DWI
     - FLAIR
     - Susceptibility
Summary

• Neuroradiology is important in confirming the diagnosis of acute cerebral infarction and monitoring progression

• Advances in MR techniques make early diagnosis and assessment possible, which are necessary in thrombolytic therapy to prevent irreversible brain damage
References


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The End