

DIFFUSION RESTRICTION

A Practical Guide for Radiological Differential Diagnosis

❖ IF THERE IS DIFFUSION RESTRICTION → HOW DO WE DETERMINE THE CAUSE?

DWI ↑ + ADC ↓ = TRUE DIFFUSION RESTRICTION

But this only tells us that restriction is present.

The real radiological questions are:

- 👉 WHERE is the restriction?
- 👉 HOW does the lesion enhance?
- 👉 WHAT do T2/FLAIR show?
- 👉 WHAT does SWI show?
- 👉 WHAT does perfusion show?
- 👉 WHAT is the distribution?
- 👉 WHAT does the clinical presentation suggest?

1. ACUTE INFARCTION

DWI ↑ + ADC ↓

The most important clue:

Vascular distribution

- Cortex + subcortical white matter
- Distribution corresponding to a specific arterial territory
- Gyriiform cortical restriction may be seen
- In large-vessel occlusion, associated vascular findings may be present
- Susceptibility vessel sign may be seen on SWI
- MRA/CTA may demonstrate arterial occlusion/stenosis

Pearl:

If the distribution follows a vascular territory → think infarction first.

2. PYOGENIC ABSCESS

The classic pattern:

CENTRAL RESTRICTION

- DWI ↑
- ADC ↓
- Restriction predominantly within the central purulent cavity •

Enhancing capsule surrounding the cavity

- Prominent perifocal vasogenic edema
- Usually a smooth or relatively smooth thin ring enhancement

- The center is usually T2 hyperintense, while the capsule may show relatively lower signal
- Usually low perfusion

💡 Why?

The high viscosity and cellular/inflammatory content of pus restrict water diffusion.

💡 Pearl:

Ring-enhancing lesion + central true diffusion restriction → strongly consider abscess.

⚠ However, this is not pathognomonic. Some metastases and other ring-enhancing lesions may also show central restriction.

💡 3. NECROTIC GLIOBLASTOMA / NECROTIC TUMOR

The classic pattern:

📌 THE CENTRAL NECROTIC COMPONENT USUALLY DOES NOT RESTRICT

- Necrotic center → ADC ↑ / facilitated diffusion
- The center is usually not markedly hyperintense on DWI
- Restriction is more commonly seen in the PERIPHERAL SOLID TUMOR COMPONENT
- Ring enhancement is often more irregular/nodular
- Thick and irregular enhancing wall
- Perfusion: increased rCBV in the solid component favors tumor
- Infiltrative T2/FLAIR abnormality may be present around the lesion

◆ Pearl:

Central facilitated diffusion + restriction in the peripheral solid component → think necrotic tumor.

But remember:

⚠ “If the center restricts, it must be an abscess” is incorrect.

Some necrotic tumors, particularly certain metastases, may also demonstrate diffusion restriction within the necrotic component.

◆ 4. LYMPHOMA

A very important distinction:

↑ THE RESTRICTED COMPONENT IS THE SOLID TUMOR ITSELF.

- DWI ↑
- ADC ↓↓
- Usually marked and relatively homogeneous diffusion restriction
- Usually strong and homogeneous enhancement
- Perfusion is often lower than in high-grade gliomas
- Periventricular location is common
- Corpus callosum involvement may occur
- Deep gray matter involvement may be seen

◆ Pearl:

If the solid tumor tissue—not a necrotic cavity—is restricting → think lymphoma.

❖ 5. HYPERCELLULAR TUMORS

As cellularity increases, the extracellular space decreases → water diffusion may become restricted.

Examples:

- Lymphoma
- Medulloblastoma
- Some high-grade gliomas
- Some metastases
- Other hypercellular tumors

📌 Do not rely on DWI alone.

Age + location + enhancement pattern + T2/FLAIR + perfusion + morphology should be assessed together.

❖ 6. ENCEPHALITIS

Especially: HSV encephalitis

📌 Typical distribution:

- Medial temporal lobe
- Insula
- Inferior frontal regions
- Often asymmetric

- DWI ↑
- ADC ↓
- T2/FLAIR ↑
- Cortical involvement
- Enhancement may develop over time
- Hemorrhagic components may be seen on SWI

Pearl:

Cortico-limbic diffusion restriction that does not follow a vascular territory → think encephalitis.

7. STATUS EPILEPTICUS / PERI-ICTAL CHANGES

DWI ↑ + ADC ↓ may be present.

But distribution is crucial:

- Cortical ribbon
- Hippocampus
- Pulvinar
- Caudate
- Sometimes cerebellum

 Does not follow a vascular territory.

An important additional finding:

🔥 Hyperperfusion on ASL/perfusion imaging

This can be a strong clue favoring status epilepticus, particularly in the acute setting.

💡 Pearl:

Diffusion restriction + nonvascular cortical distribution + hyperperfusion → think seizure/status epilepticus.

Regression of the abnormalities on follow-up MRI may also favor status epilepticus, whereas infarction usually results in persistent structural changes.

💡 8. HYPOXIC-ISCHEMIC INJURY

Again, distribution is the key.

- Cortical ribbon diffusion restriction
- Hippocampi
- Basal ganglia
- Cerebellum may be involved

In severe cases:

- 📌 Cortex + deep gray nuclei may be involved together.

 Pearl:

Global hypoxic clinical setting + bilateral/symmetric cortical and deep gray matter restriction → think hypoxic-ischemic injury.

 9. TOXIC / METABOLIC ENCEPHALOPATHIES

These can also cause diffusion restriction.

But again:

 DISTRIBUTION IS THE KEY TO THE DIAGNOSIS.

For example:

- Predominant basal ganglia involvement
- Cortical involvement
- Hippocampal involvement
- Cerebellar involvement
- Corpus callosum involvement

should be interpreted together with the clinical and laboratory findings.

 Pearl:

Bilateral + symmetric + nonvascular diffusion restriction → think toxic/metabolic causes.

THE MOST USEFUL RADIOLOGICAL DISTINCTION

IF THERE IS DIFFUSION RESTRICTION, FIRST ASK:

 WHERE IS THE RESTRICTION?

→ Within a vascular territory

 Acute infarction

→ In the CENTER of a ring-enhancing lesion

 Consider pyogenic abscess first

 But remember the exceptions.

→ In the PERIPHERAL SOLID COMPONENT of a ring-enhancing lesion

 Think necrotic tumor

→ Throughout the SOLID TUMOR / prominently within its solid component

 Think lymphoma / hypercellular tumor

→ Medial temporal + insular + cortical involvement, not following a vascular territory

 Think HSV encephalitis

→ Cortical + hippocampal + pulvinar/claustum involvement, not following a vascular territory + hyperperfusion

 Think status epilepticus

→ Bilateral/symmetric cortex + deep gray nuclei

→ Think hypoxic-ischemic / toxic-metabolic process

❓ THE FINAL FORMULA

DWI/ADC → “IS THERE RESTRICTION?”

Then:

DISTRIBUTION → “WHERE?”

ENHANCEMENT → “HOW?”

T2/FLAIR → “WHAT ASSOCIATED EDEMA/PATTERN IS PRESENT?”

SWI → “IS THERE HEMORRHAGE?”

PERFUSION → “IS THERE INCREASED TUMOR VASCULARITY/ACTIVITY?”

CLINICAL → “WHAT DOES THE CLINICAL PICTURE SAY?”

👉 The actual differential diagnosis is made at this second stage.

DWI tells us that there is “restriction.”

ADC confirms it.

Distribution + morphology + enhancement + additional sequences tell us “why.”

❖ DIFFUSION RESTRICTION — DIFFERENTIAL DIAGNOSIS TRAPS | PART 2

One of the most common mistakes when we see diffusion restriction:

❌ “ADC is low → this must be the diagnosis.”

The correct approach:

👉 LOCATION of restriction + ENHANCEMENT + MORPHOLOGY + DISTRIBUTION
+ ADDITIONAL SEQUENCES

❖ 1. ABSCESS vs NECROTIC TUMOR

Both can present as ring-enhancing lesions.

ABSCESS:

- 📍 Center → marked diffusion restriction
- 📍 ADC → low
- 📍 Usually smooth, thin ring enhancement
- 📍 Perifocal vasogenic edema
- 📍 Relatively regular capsule
- 📍 Perfusion → low

NECROTIC TUMOR:

- Necrotic center → usually no restriction
- Peripheral solid component → may restrict
- ADC → may be low in the solid component
- Ring → may be thicker, irregular, and/or nodular
- rCBV may be increased in the solid component
- Infiltrative T2/FLAIR abnormality may be present

💡 Pearl:

Center restricts → think abscess.

Solid wall restricts → think tumor.

⚠ However, this is not an absolute rule. Some metastases may also demonstrate diffusion restriction within their necrotic centers.

💡 2. LYMPHOMA vs HIGH-GRADE GLIOMA

Both can show diffusion restriction.

LYMPHOMA:

- Marked restriction within the solid tumor tissue
- ADC is usually markedly low

- Enhancement is often strong and homogeneous
- Periventricular location is common
- Corpus callosum/deep gray involvement may occur
- Perfusion is generally lower than in glioma

HIGH-GRADE GLIOMA:

- Restriction may be present in the solid component
- Necrotic center usually does not restrict
- Thick, irregular, and/or nodular ring enhancement
- Infiltrative T2/FLAIR abnormality
- rCBV is usually markedly increased in the solid component

Pearl:

Marked restriction + solid enhancing tissue + relatively low perfusion → think lymphoma.

Restriction + high perfusion + infiltrative/necrotic tumor pattern → think high-grade glioma.

❖ 3. EPIDERMOID vs ARACHNOID CYST

Both may appear CSF-like.

EPIDERMOID:

- DWI → markedly hyperintense
- ADC → low
- FLAIR → does not completely suppress like CSF
- “Dirty CSF” appearance
- Often located in cisternal spaces
- May insinuate around cranial nerves and vessels

ARACHNOID CYST:

- DWI → NO restriction
- ADC → high
- FLAIR → completely suppresses like CSF
- Usually well circumscribed
- May cause mass effect on adjacent structures

❖ Pearl:

CSF-like lesion + diffusion restriction → epidermoid until proven otherwise.

4. INFARCTION vs STATUS EPILEPTICUS

Both can show DWI ↑ + ADC ↓.

INFARCTION:

- Follows a vascular distribution
- Arterial territory pattern
- Cortical + subcortical involvement may occur
- Occlusion may be identified on MRA/CTA
- Persistent infarct-related structural changes may develop over time

STATUS EPILEPTICUS:

- Does not follow a vascular territory
- Cortical ribbon
- Hippocampus
- Pulvinar / claustrum
- Sometimes cerebellum
- Hyperperfusion may be seen on ASL/perfusion imaging
- May regress on follow-up imaging

Pearl:

Restriction + vascular territory → infarction.

Restriction + nonvascular distribution + hyperperfusion → think seizure/status epilepticus.

5. ABSCESS vs EPIDERMOID

Both can demonstrate marked diffusion restriction.

ABSCESS:

- Ring-enhancing lesion
- Central restriction
- Surrounding edema
- Capsule
- Clinical features of infection may be present

EPIDERMOID:

- Usually no enhancement
- Cisternal location
- CSF-like signal
- Marked DWI restriction
- Does not completely suppress like CSF on FLAIR

◆ Pearl:

Restriction + ring enhancement → think abscess.

Restriction + CSF-like cysternal lesion + no enhancement → think epidermoid.

◆ 6. METASTASIS vs GLIOBLASTOMA

Both may be necrotic and ring-enhancing.

METASTASIS:

- Often multiple
- Gray-white matter junction
- Necrotic center usually shows facilitated diffusion
- Peripheral solid component may restrict
- Prominent surrounding vasogenic edema
- Edema may extend beyond the tumor margins, but typical infiltrative tumor spread is less characteristic

GLIOBLASTOMA:

- Often solitary
- Thick, irregular, and/or nodular ring
- Necrosis + solid tumor component

- Infiltrative T2/FLAIR abnormality
- May extend along the corpus callosum
- Increased perfusion may be seen in the solid component

Pearl:

Multiple lesions at the gray-white junction → metastases strongly enter the differential.

Asolitary infiltrative lesion extending along white-matter pathways/corpus callosum → think glioblastoma.



FINAL “TRAP CHECKLIST”

When you see DWI ↑ + ADC ↓, do not jump directly to a diagnosis. Ask these 7 questions:

1 WHERE is the restriction?

Center? Solid wall? Cortex? Entire lesion?

2 HOW does it enhance?

Homogeneous? Thin ring? Thick irregular ring? Nodular?

3 WHAT is the distribution?

Vascular? Nonvascular? Bilateral/symmetric?

4 WHAT do T2/FLAIR show?

Vasogenic edema? Infiltration? “Dirty CSF”?

5 WHAT does SWI show?

Hemorrhage? Calcification? Susceptibility vessel sign?

6 WHAT does PERFUSION show?

High rCBV → favors tumor

Low perfusion → may support entities such as abscess/lymphoma

7 WHAT does the CLINICAL picture suggest?

Sudden deficit? Fever? Seizure? Immunosuppression? Known malignancy?

❖ THE GOLDEN RULE

“DWI restriction is a finding, not a diagnosis.”

First ask:

WHERE? → HOW? → DISTRIBUTION? → WHAT ELSE? → CLINICAL CONTEXT?

Then move toward the diagnosis.